FOURTH EDITION

Attention-Deficit Hyperactivity Disorder

A Handbook for Diagnosis & Treatment

edited by

Russell A. Barkley

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To my grandsons
Liam and Craig,
for yet another opportunity to experience
the joys of childhood

About the Editor

Russell A. Barkley, PhD, ABPP, ABCN, is Clinical Professor of Psychiatry at the Virginia Treatment Center for Children and Virginia Commonwealth University School of Medicine. Dr. Barkley has worked with children, adolescents, and families since the 1970s and is the author of numerous bestselling books for both professionals and the public, including *Taking Charge of ADHD* and *Your Defiant Child*. He has also published six assessment scales and more than 280 scientific articles and book chapters on ADHD, executive functioning, and childhood defiance, and is editor of the newsletter *The ADHD Report*. A frequent conference presenter and speaker who is widely cited in the national media, Dr. Barkley is past president of the Section on Clinical Child Psychology (the former Division 12) of the American Psychological Association (APA), and of the International Society for Research in Child and Adolescent Psychopathology. He is a recipient of awards from the American Academy of Pediatrics and the APA, among other honors. His website is *www.russellbarkley.org*.

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Preface

This fourth edition constitutes a nearly complete revision and update of the third edition, comprising at least 80% or more new material, a substantial expansion of topical areas covered on attention-deficit/hyperactivity disorder (ADHD), and at least triple the number of contributors in comparison to the prior edition. While it is essentially a new textbook, like its predecessors, this edition strives to extract from the mine of available scientific literature those nuggets of clinically important information regarding the nature, assessment, diagnosis, and management of ADHD in children, adolescents, and adults. The task of doing so has increased substantially with each new edition given the increasing frequency of journal articles relating to ADHD appearing in the scientific literature, now estimated to be at least 800–1,000 per year, a rate that seems to have doubled in just the past few years owing to a vast increase in the number of international contributors outside of Western Europe and North America. More than 6,000 studies on ADHD have been published since the publication of the 2006 edition of this work. So formidable an undertaking requires the assistance of many more contributors than in prior editions, for it is clear that no single individual can be an expert in all facets of this disorder and its management.

To help me with this endeavor, I have invited back many of the principal authors of chapters from the previous edition, along with many new contributors, each expert in the topics contained in their respective chapters. All were charged with incorporating new findings and new conclusions and clinical recommendations from the available research and related publications. I am truly grateful that all agreed to join in this endeavor. In order to draw out the messages essential for clinicians within each chapter, all chapters conclude with a checklist of "Key Clinical Points," most of which I prepared to help the reader quickly summarize the major conclusions and recommendations discussed in that chapter.

From time to time, media flare-ups have centered on ADHD, sometimes challenging its very existence. Taken in its totality, this book is a complete refutation of such assertions. It continues to show, as did previous editions, that ADHD is as valid a mental disorder as we are likely to find, with massive evidence from more than 10,000 studies dating back to medical descriptions in 1775. It clearly represents a serious deficiency in one or more psychological adaptations that harm the individuals so afflicted, which is the very definition of a mental disorder.

As in previous editions, I once again thank Kitty Moore, Seymour Weingarten, and Robert Matloff at The Guilford Press for supporting this book and providing a home for this and my other books. I also wish

to thank Carolyn Graham, Jacquelyn Coggin, and Laura Specht Patchkofsky at Guilford for helping to shepherd this book through the publication process in a professional and expeditious manner. My debt to them and the rest of Guilford's superbly capable staff for having assisted me for more than 33 years of publishing is incalculable, and I express my deep appreciation to all members of the Guilford "family" here. I am also extraordinarily appreciative of my wife, Patricia, who has stood by me for more than 45 years and provided a loving home for me, our two sons, Ken and Steve, now all grown up, and now our two grandsons, to whom this edition is dedicated. All provide me with a profound sense of family and their affection, for which I am exceptionally grateful. In such a home can this type of creative work be accomplished.

RUSSELL A. BARKLEY, PhD

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PART I. THE NATURE OF ADHD

PART I

The Nature of ADHD

CHAPTER 1

History of ADHD

Russell A. Barkley

Attention-deficit/hyperactivity disorder (ADHD) continues to be the current diagnostic label for children and adults presenting with significant problems with attention, and typically with impulsiveness and excessive activity as well. Children and adults with ADHD represent a rather heterogeneous population that displays considerable variation in the degree of members' symptoms, age of onset, cross-situational pervasiveness of those symptoms, and the extent to which other disorders occur in association with ADHD. The disorder represents one of the most common reasons children with behavioral problems are referred to medical and mental health practitioners in the United States and is one of the most prevalent childhood psychiatric disorders. Currently, referrals of adults for ADHD are also increasing at a rapid pace; until the 1990s and even to date, this age group has been a markedly underrecognized and underserved segment of the ADHD population.

This chapter presents an overview of ADHD's history—a history that spans more than two centuries in the medical and scientific literature. Whereas the previous edition noted that the medical history of ADHD began with Still's description of childhood cases in 1902, we now know that a number of earlier physicians described such cases dating back to the textbook by Melchior Adam Weikard published in German in 1775 (Barkley & Peters, 2012). This extends the history of ADHD in the medical literature back another 127 years. These new additions to the history of ADHD are described below. But given that the history of ADHD as understood from 1902 through 2006 has changed little since the preceding edition of this text (Barkley, 2006), little has been needed to update those sections of this chapter. In contrast, developments since that previous edition are described at the end of this chapter.

In the history of ADHD reside the nascent concepts that serve as the foundation for the current conceptualization of the disorder as largely involving self-regulation and executive functioning, as discussed here by Eric Willcutt (Chapter 15) and myself (Chapter 16). In this history also can be seen the emergence of current notions about its treatment. Such a history remains important for any serious student of ADHD, for it shows that many contemporary themes concerning its nature arose long ago. They have recurred throughout the subsequent history of ADHD to the present as clinicians and scientists strove for a clearer, more accurate understanding of the condition, its comorbid disorders, life course, impairments, and etiologies. Readers are directed to other and earlier sources for additional discussions of the history of this disorder (Accardo &

Blondis, 2000; Goldstein & Goldstein, 1998; Kessler, 1980; Ross & Ross, 1976, 1982; Schachar, 1986; Taylor, 2011; Warnke & Riederer, 2013; Werry, 1992).

THE HISTORICAL ORIGINS OF ADHD

The Late 1700s

One can find literary references to individuals having serious problems with inattention, hyperactivity, and poor impulse control in Shakespeare, who made mention of a malady of attention in *King Henry VIII*. But as of this writing, the medical history of ADHD-like descriptions traces back nearly 240 years to 1775. This early history has been expertly detailed in several sources (Taylor, 2011; Warnke & Riederer, 2013) but should be amended by more recent discoveries in that history, as discussed below.

It now appears that the first description of disorders of attention, at least as of this writing, occurred in the medical textbook by Melchior Adam Weikard in German in 1775 (or perhaps even 1770; see Barkley & Peters, 2012). Initially published anonymously, hence the difficulty with ascertaining the year of its initial publication, the medical textbook by Weikard described adults and children who were inattentive, distractible, lacking in persistence, overactive, and impulsive, which is quite similar to today's description of ADHD. Weikard implied that the disorder could result from poor childrearing but also suggests some biological predispositions as well. For treatment, he recommended sour milk, plant extracts, horseback riding, and even seclusion for severe cases.

This textbook would be followed in short order in 1798 with much more detailed descriptions of ADHD-like symptoms in the medical textbook by the Scottish physician Alexander Crichton (see Palmer & Finger, 2001), who may well have studied with Weikard in his medical training. Crichton described two types of attention disorders. The first was a disorder of distractibility, frequent shifting of attention or inconstancy, and lack of persistence or concentration, and aligns more closely with the attention disturbance evident in ADHD. The second was a disorder of diminished power or energy of attention that seems more like the attention problem evident in current descriptions of children and adults with sluggish cognitive tempo (SCT), which is briefly discussed in Chapter 2 on ADHD symptoms and subtypes and far more detailed coverage in Chapter 17, this volume. Crichton had little to say about this second disorder of attention other than it may be associated with debility or torpor of the body that weakens attention and results in individuals who are often characterized as retiring, unsocial, and having few friendships or attachments of any kind; even those few friendships seldom were of a durable nature. He argued that the faculty of attention can become sufficiently weakened that it may leave an individual insensible to external objects or to impressions that ordinarily would awaken social feelings.

The 1800s

In 1809, John Haslam described what may have been a case of ADHD in a 10-year-old boy who was uncontrollable, impulsive, and "a creature of volition and the terror of the family" (p. 199). Three years later, the famous American physician Benjamin Rush (1812) discussed three cases involving "the total perversion of moral faculties" (p. 359), which included the inability to focus attention. In the mid-1800s, the German pediatrician Heinrich Hoffman (1865) published a book of poems about psychological conditions of children based on observations from his clinical practice. He described both a very impulsive fidgety child he called "Fidgety Phil" and a very inattentive, daydreamy child he called "Johnny Head-in-Air" (see Stewart, 1970). Two years thereafter in England, Henry Maudsley (1867) published a report about a child who was driven by impulsiveness and was also quite destructive. In 1899, the Scottish psychiatrist, Thomas Clouston discussed cases of impulsive children who had learning problems. Much later in the United States, William James (1890/1950) noted in his *Principles of Psychology* a normal variant of character that he called the "explosive will," which may resemble the difficulties experienced by those who today are described as having ADHD.

In France the concept of ADHD may have originated in 1845 in the description of children and adults with attention problems by Jean-Etienne Dominique Esquirol, who believed that the insane no longer "enjoy the faculty of fixing, and directing their attention" (p. 28). Or perhaps the French history of ADHD began in the notion of "mental instability" that appears in the French medical literature in the 1885–1895 period under the leadership of Désiré-Magloire Bourneville (1885, 1895; see Bader & Hidjikhani, in press) at the Hospital Bicêtre in Paris. He observed children and adolescents who had been labeled "abnormal" and placed in medical and educational institutions, many

of whom were characterized by attention and other behavioral problems. Charles Baker, a student of Bourneville, wrote a clinical description of hyperactive and impulsive symptoms in 4 children in his 1892 thesis, according to Bourneville (1895). Attention problems were also mentioned in one case in this work.

THE PERIOD 1900 TO 1959

Still's Description in 1902

In the earlier editions of this text, credit for authoring the first medical description of cases resembling ADHD was awarded to George Still in 1902, owing to the lack of information on the earlier works of Weikard and Crichton. While this no longer remains the case, having been ousted from this credit by the discovery of Weikard's description noted earlier, Still did provide probably the most detailed account of the symptoms of these cases and the largest sample of such cases to that time. For these reasons, his observations deserve some recognition here. In a series of three published lectures to the Royal College of Physicians in 1902, Still described 43 children in his clinical practice who had serious problems with sustained attention; he agreed with William James (1890/1950) that such attention may be an important element in the "moral control of behavior." Most were also quite overactive. Many were often aggressive, defiant, resistant to discipline, and excessively emotional or "passionate." These children showed little "inhibitory volition" over their behavior, and they also manifested "lawlessness," spitefulness, cruelty, and dishonesty. Still proposed that the immediate gratification of the self was the "keynote" quality of these children, among other attributes. Passion (or heightened emotionality) was the most commonly observed attribute and the most noteworthy. Still noted further that such children had an insensitivity to punishment, for they would be punished (even physically) yet engage in the same infraction within a matter of hours.

Still believed that these children displayed a major "defect in moral control" over their behavior; a defect that was relatively chronic in most cases. He believed that in some cases, these children had acquired the defect secondary to an acute brain disease, and it might remit on recovery from the disease. He noted a higher risk for criminal acts in later development in some, though not all, of the chronic cases. Although this defect could be associated with intellectual retardation, as it was in 23 of the cases, it could also arise in children of near-normal intelligence, as it seemed to do in the remaining 20.

To Still (1902), the moral control of behavior meant "the control of action in conformity with the idea of the good of all" (p. 1008). Moral control was thought to arise out of a cognitive or conscious comparison of the individual's volitional activity with that of the good of all—a comparison he termed "moral consciousness." For purposes that will become evident later, it is important to realize here that to make such a comparison inherently involves the capacity to understand the consequences of one's actions over time and to hold in mind forms of information about oneself and one's actions, along with information on their context. Those forms of information involve the action being proposed by the individual, the context, and the moral principle or rule against which it must be compared. This notion may link Still's views with the contemporary concepts of self-awareness, working memory, and rule-governed behavior discussed later in this text. Still did not specifically identify these inherent aspects of the comparative process, but they are clearly implied in the manner in which he used the term "conscious" in describing this process. He stipulated that this process of comparison of proposed action to a rule concerning the greater good involved the critical element of the conscious or cognitive relation of individuals to their environment, or "self-awareness." Intellect was recognized as playing a part in moral consciousness, but equally or more important was the notion of volition or will. The latter is where Still believed the impairment arose in many of those with defective moral control who suffered no intellectual delay. Volition was viewed as being primarily inhibitory in nature, that a stimulus to act must be overpowered by the stimulus of the idea of the greater good of all.

Still concluded that a defect in moral control could arise as a function of three distinct impairments: "(1) defect of cognitive relation to the environment; (2) defect of moral consciousness; and (3) defect in inhibitory volition" (p. 1011). He placed these impairments in a hierarchical relation to each other in the order shown, arguing that impairments at a lower level would affect those levels above it and ultimately the moral control of behavior. Much as researchers do today, Still noted a greater proportion of males than females (3:1) in his sample, and he observed that the disorder appeared to arise in most cases before 8 years of age (typically in early childhood). Many of Still's cases displayed a

proneness to accidental injuries—an observation corroborated by numerous subsequent studies reviewed in a later chapter. And Still saw these youngsters as posing an increased threat to the safety of other children because of their aggressive or violent behavior. Alcoholism, criminality, and affective disorders such as depression and suicide were noted to be more common among their biological relatives—an observation once again buttressed by numerous studies published in recent years. Some of the children displayed a history of significant brain damage or convulsions, whereas others did not. A few had associated tic disorders, or "microkinesia"; this was perhaps the first time tic disorders and ADHD were noted to be comorbid conditions. We now recognize that while 10–15% of children with ADHD may manifest some form of tic disorder, as many as 50–70% of children with tic disorders and Tourette syndrome may have ADHD (Simpson, Jung, & Murphy, 2011).

Although many of Still's subjects were reported to have a chaotic family life, others came from households that provided a seemingly adequate upbringing. In fact, Still believed that when poor childrearing was clearly involved, the children should be exempt from the category of lack of moral control; he reserved it instead only for children who displayed a morbid (organic) failure of moral control despite adequate training. He proposed a biological predisposition to this behavioral condition that was probably hereditary in some children but the result of pre- or postnatal injury in others. In keeping with the theorizing of James (1890/1950), Still hypothesized that the deficits in inhibitory volition, moral control, and sustained attention were causally related to each other and to the same underlying neurological deficiency. He cautiously speculated on the possibility of either a decreased threshold for inhibition of responding to stimuli or a cortical disconnection syndrome, in which intellect was dissociated from "will" in a manner that might be due to neuronal cell modification. Any biologically compromising event that could cause significant brain damage ("cell modification") and retardation could, he conjectured, in its milder forms lead only to this defective moral control.

Also in England, Alfred Tredgold (1908) described children of low intelligence having abnormal behavior and limited powers of attention, impulse control, and willpower. He extended Still's theories and observations that early brain damage might present as behavioral and learning problems in later childhood. Foreshadowing current views of treatment, both Still (1902) and Tredgold found that temporary improvements in conduct might be achieved by alterations in the environment or by medications, but they stressed the relative permanence of the defect even in these cases. They emphasized the need for special educational environments for these children. We see here the origins of many later and even current notions about children with ADHD and oppositional defiant disorder (ODD), although it would take almost 70 years to return to many of them—owing in part to the ascendance in the interim of psychoanalytic, psychodynamic, and behavioral views that overemphasized childrearing as largely causing such behavioral disorders in children. The children described by Still and Tredgold would probably now be diagnosed as having not only ADHD but also ODD or conduct disorder (CD), and most likely a learning disability as well (see Chapters 5 and 6).

Around this same time, in Spain, the physician Rodriguez-Lafora (1917) wrote about his interests in childhood mental illness and described a group of children having psychopathic constitutions, a subset of which he called the "unstables." His description of them matches closely the modern view of ADHD (Bauermeister & Barkley, 2010), including inconstancy of attention, excessive activity, and impulsive behavior, as does his observation that such children get carried away by their adventurous temperament.

The Influence of the Encephalitis Epidemic

The history of interest in ADHD in North America can be traced to the outbreak of an encephalitis epidemic in 1917–1918, when clinicians were presented with a number of children who survived this brain infection but were left with significant behavioral and cognitive sequelae (Cantwell, 1981; Kessler, 1980; Stewart, 1970). Numerous articles that reported these sequelae (Ebaugh, 1923; Strecker & Ebaugh, 1924; Stryker, 1925) included many of the characteristics we now incorporate into the concept of ADHD. Such children were described as being impulsive and having impaired attention and regulation of activity, as well as impairments in other cognitive abilities, including memory; they were often noted to be socially disruptive as well. Symptoms of what is now called ODD, as well as delinquency and CD, also arose in some cases. "Postencephalitic behavior disorder," as it was called, was clearly the result of brain damage. The large number of affected

children resulted in significant professional and educational interest in this behavioral disorder. Its severity was such that many children were recommended for care and education outside the home and away from normal educational facilities. Despite a rather pessimistic view of the prognosis of these children, some facilities reported significant success in their treatment with simple behavior modification programs and increased supervision (Bender, 1942; Bond & Appel, 1931).

The Origins of a Brain Damage Syndrome

This association of a brain disease with behavioral pathology apparently led early investigators to study other, potential causes of brain injury in children and their behavioral manifestations, including birth trauma (Shirley, 1939); other infections besides encephalitis, such as measles (Meyer & Byers, 1952); lead toxicity (Byers & Lord, 1943); epilepsy (Levin, 1938); and head injury (Blau, 1936; Werner & Strauss, 1941). All were studied in children and found to be associated with numerous cognitive and behavioral impairments, including the triad of ADHD symptoms noted earlier. Other terms introduced during this era for children displaying these behavioral characteristics were "organic driveness" (Kahn & Cohen, 1934) and "restlessness" syndrome (Childers, 1935; Levin, 1938). Many of the children seen in these samples also had mental retardation or more serious behavioral disorders than what is today called ADHD. It would take investigators several decades to attempt to parse out the separate contributions of intellectual delay, learning disabilities, or other neuropsychological deficits from those of behavioral deficits in the maladjustment of these children. Even so, scientists at this time would discover that activity level was often inversely related to intelligence in children, increasing as intelligence declined in a sample—a finding supported in many subsequent studies (Rutter, 1989). It should also be noted that a large number of children in these older studies did in fact have brain damage or signs of such damage (epilepsy, hemiplegias, etc.).

Notable during this era was also recognition of the striking similarity between hyperactivity in children and the behavioral sequelae of frontal lobe lesions in primates (Blau, 1936; Levin, 1938). Frontal lobe ablation studies of monkeys had been done more than 60 years earlier (Ferrier, 1876), and the lesions were known to result in excessive restlessness, poor ability to sustain interest in activities, aimless wandering, and excessive appetite, among other behavioral changes. Several investigators, such as Levin (1938), used these similarities to postulate that severe restlessness in children might well be the result of pathological defects in the forebrain structures, although gross evidence of such was not always apparent in many of these children. Later, investigators (e.g., Barkley, 1997a; Chelune, Ferguson, Koon, & Dickey, 1986; Lou, Henriksen, & Bruhn, 1984; Lou, Henriksen, Bruhn, Borner, & Nielsen, 1989; Mattes, 1980) would return to this notion, but with greater evidence to substantiate their claims. Milder forms of hyperactivity, in contrast, were attributed in this era to psychological causes, such as "spoiled" child-rearing practices or delinquent family environments. This idea that poor or disrupted parenting causes ADHD would also be resurrected in the 1970s, and it continues even today among many laypeople and critics of ADHD.

Over the next decade, it became fashionable to consider most children hospitalized in psychiatric facilities with this symptom picture to have suffered from some type of brain damage (e.g., encephalitis or prenatal–perinatal trauma), whether or not there was evidence of such in the clinical history of the case. The concept of the "brain-injured child" was born in this era (Strauss & Lehtinen, 1947) and applied to children with these behavioral characteristics, many of whom had insufficient or no evidence of brain pathology. In fact, Strauss and Lehtinen argued that the psychological disturbances alone were de facto evidence of brain injury as the etiology. Owing in part to the absence of such evidence of brain damage, this term would later evolve into the concept of "minimal brain damage" and eventually "minimal brain dysfunction" (MBD) by the 1950s and 1960s. Even so, a few early investigators, such as Childers (1935), would raise serious questions about the notion of brain damage in these children when no historical documentation of damage existed. Substantial recommendations for educating these "brain-damaged" children were made in the earlier text by Tredgold (1908) and later in the classic text on special education by Strauss and Lehtinen (1947), which served as a forerunner to special educational services adopted much later in U.S. public schools. These recommendations included placing these children in smaller, more carefully regulated classrooms and reducing the amount of distracting stimulation in the environment. Strikingly austere classrooms were developed,

in which teachers avoided wearing jewelry or brightly colored clothing, and few pictures adorned the walls so as not to interfere unnecessarily with the education of these highly distractible students.

Although the population served by the Pennsylvania center in which Strauss, Werner, and Lehtinen worked principally contained children with mental retardation, the work of Cruickshank and his students (Dolphin & Cruickshank, 1951a, 1951b, 1951c) later extended these neuropsychological findings to children with cerebral palsy but near-normal or normal intelligence. This extension resulted in the extrapolation of the educational recommendations of Strauss to children without mental retardation who manifested behavioral or perceptual disturbances (Cruickshank & Dolphin, 1951; Strauss & Lehtinen, 1947). Echoes of these recommendations are still commonplace today in most educational plans for children with ADHD or learning disabilities, despite the utter lack of scientific support for their efficacy (Kessler, 1980; Routh, 1978; Zentall, 1985). These classrooms are historically significant because they were predecessors as well as instigators of the types of educational resources that would be incorporated into the initial Education for All Handicapped Children Act of 1975 (Public Law 94-142) mandating the special education of children with learning disabilities and behavioral disorders, and its later reauthorization, the Individuals with Disabilities Education Act of 1990 (IDEA; Public Law 101-476).

The Beginnings of Child Psychopharmacology for ADHD

Another significant series of articles on the treatment of hyperactive children appeared from 1937 to 1941. They marked the beginnings of medication therapy (particularly stimulants) for behaviorally disordered children in particular, as well as the field of child psychopharmacology in general (Bradley, 1937; Bradley & Bowen, 1940; Molitch & Eccles, 1937). Initiated originally to treat the headaches that resulted from conducting pneumoencephalograms during research studies of these disruptive youth, the administration of amphetamine resulted in a noticeable improvement in their behavioral problems and academic performance. Later studies would also confirm such a positive drug response in half or more of hyperactive hospitalized children (Laufer, Denhoff, & Solomons, 1957). As a result, by the 1970s, stimulant medications were gradually becoming the treatment of choice for the behavioral symptoms now associated with ADHD. And so they remain today (see Chapter 27).

The Emergence of a Hyperkinetic Impulse Syndrome

In the 1950s, researchers began a number of investigations into the neurological mechanisms underlying these behavioral symptoms, the most famous of which was probably that by Laufer and colleagues (1957). These writers referred to children with ADHD as having "hyperkinetic impulse disorder," and reasoned that the central nervous system (CNS) deficit occurred in the thalamic area. Here, poor filtering of stimulation occurred, allowing an excess of stimulation to reach the brain. The evidence was based on a study of the effects of the "photo-Metrozol" method, in which the drug metronidazole (Metrozol) is administered while flashes of light are presented to a child. The amount of drug required to induce a muscle jerk of the forearms, along with a spike wave pattern on the electroencephalogram (EEG), serves as the measure of interest. Laufer and colleagues found that inpatient children with hyperactivity required less Metrozol than those without hyperactivity to induce this pattern of response. This finding suggested that the hyperactive children had a lower threshold for stimulation, possibly in the thalamic area. No attempts to replicate this study have been done, and it is unlikely that such research would pass today's standards of ethical conduct in research required by institutional review boards on research with human subjects. Nevertheless, it remains a milestone in the history of the disorder for its delineation of a more specific mechanism that might give rise to hyperactivity (low cortical thresholds or overstimulation). Others at the time also conjectured that the existence of an imbalance between cortical and subcortical areas caused diminished control of subcortical areas responsible for sensory filtering that permitted excess stimulation to reach the cortex (Knobel, Wolman, & Mason, 1959).

By the end of this era, it seemed well accepted that hyperactivity was a brain damage syndrome, even when evidence of damage was lacking. The disorder was thought to be best treated through educational classrooms characterized by reduced stimulation or through residential centers. Its prognosis was considered fair to poor. The possibility that a relatively new class of medications, the stimulants, might hold promise for its treatment was beginning to be appreciated.

THE PERIOD 1960 TO 1969

The Decline of MBD and the Rise of Hyperactivity

In the late 1950s and early 1960s, critical reviews began to question the concept of a unitary syndrome of brain damage in children. They also pointed out the logical fallacy that if brain damage resulted in some of these behavioral symptoms, these symptoms could be pathognomonic of brain damage without any other corroborating evidence of CNS lesions. Chief among these critical reviews were those of Birch (1964), Herbert (1964), and Rapin (1964), who questioned the validity of applying the concept of brain damage to children who had only equivocal signs of neurological involvement, not necessarily damage. A plethora of research followed on children with MBD (see Rie & Rie, 1980, for reviews); in addition, a task force by the National Institute of Neurological Diseases and Blindness (Clements, 1966) recognized at least 99 symptoms for this disorder. The concept of MBD would die a slow death as it eventually was recognized to be vague, overinclusive, of little or no prescriptive value, and without much neurological evidence (Kirk, 1963). Its remaining value was its emphasis on neurological mechanisms over the often excessive, pedantic, and convoluted environmental mechanisms proposed at that time—particularly those etiological hypotheses stemming from psychoanalytical theory, which blamed parental and family factors entirely for these problems (Hertzig, Bortner, & Birch, 1969; Kessler, 1980; Taylor, 1983). The term "MBD" would eventually be replaced by more specific labels applying to somewhat more homogeneous cognitive, learning, and behavioral disorders, such as "dyslexia," "language disorders," "learning disabilities," and "hyperactivity." These new labels were based on children's observable and descriptive deficits rather than on some underlying, unobservable etiological mechanism in the brain.

The Hyperactivity Syndrome

As dissatisfaction with the term "MBD" was occurring, clinical investigators shifted their emphasis to the behavioral symptom thought to most characterize the disorder—that of hyperactivity. And so the concept of a hyperactivity syndrome arose, described in the classic articles by Laufer and Denhoff (1957), Chess (1960), and other reports of this era (Burks, 1960; Ounsted, 1955; Prechtl & Stemmer, 1962). Chess defined "hyperactivity" as follows: "The hyperactive child is one who carries out activities at a higher than normal rate of speed than the average child, or who is constantly in motion, or both" (p. 2379). Chess's article was historically significant for several reasons: (1) It emphasized activity as the defining feature of the disorder rather than speculation about underlying neurological causes, as other scientists of the time would also do; (2) it stressed the need to consider objective evidence of the symptom beyond the subjective reports of parents or teachers; (3) it took the blame for the child's problems away from the parents; and (4) it separated the syndrome of hyperactivity from the concept of a brain damage syndrome. Other scientists of this era would emphasize similar points (Werry & Sprague, 1970). Hyperactivity would now be recognized as a behavioral syndrome that could not only arise from organic pathology but also occur in its absence. Even so, it would continue to be viewed as the result of some biological difficulty rather than as being due solely to environmental causes.

Chess (1960) described the characteristics of 36 children diagnosed with "physiological hyperactivity" from a total of 881 children seen in a private practice. The ratio of males to females was approximately 4:1, and many children were referred prior to 6 years of age, intimating a relatively earlier age of onset than that for other childhood behavioral disorders. Educational difficulties were common in this group, particularly scholastic underachievement, and many displayed oppositional defiant behavior and poor peer relationships. Impulsive and aggressive behaviors, as well as poor attention span, were commonly associated characteristics. Chess believed that the hyperactivity could also be associated with mental retardation, organic brain damage, or serious mental illness (e.g., schizophrenia). Similar findings in later research would lead others to question the specificity and hence the utility of this symptom for the diagnosis of ADHD (Douglas, 1972). As with many of today's prescriptions, a multimodal treatment approach incorporating parent counseling, behavior modification, psychotherapy, medication, and special education was recommended. Unlike Still (1902), Chess and others writing in this era stressed the relatively benign nature of hyperactivity's symptoms and claimed that in most cases they resolved by puberty (Laufer & Denhoff, 1957; Solomons, 1965). Here, then, were the beginnings of a belief that would be widely held among clinicians well into the 1980s—that hyperactivity (ADHD) was outgrown by adolescence.

Also noteworthy in this era was the definition of hyperactivity given in the official diagnostic nomenclature at the time, the second edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-II; American Psychiatric Association, 1968). It employed only a single sentence describing the hyperkinetic reaction of childhood disorder and, following the lead of Chess (1960), stressed the view that the disorder was developmentally benign: "The disorder is characterized by overactivity, restlessness, distractibility, and short attention span, especially in young children; the behavior usually diminishes by adolescence" (p. 50).

Europe and North America Diverge in Viewpoints

It is likely that during this period (or even earlier), the perspective on hyperactivity in North America began to diverge from that in Europe, particularly Great Britain. In North America, hyperactivity would become a behavioral syndrome recognized chiefly by greater-than-normal levels of activity; would be viewed as a relatively common disturbance of childhood; would not necessarily be associated with demonstrable brain pathology or mental retardation; and would be regarded as more of an extreme degree in the normal variation of temperament in children. In Great Britain, the earlier and narrower view of a brain damage syndrome would continue into the 1970s: Hyperactivity or hyperkinesis was seen as an extreme state of excessive activity of an almost driven quality; was viewed as highly uncommon; and was usually thought to occur in conjunction with other signs of brain damage (e.g., epilepsy, hemiplegias, or mental retardation) or a clearer history of brain insult (e.g., trauma or infection) (Taylor, 1988). The divergence in views would lead to large discrepancies between North American and European estimations of the prevalence of the disorder, their diagnostic criteria, and their preferred treatment modalities. A rapprochement between these views would not occur until well into the 1980s (Rutter, 1988, 1989; Taylor, 1986, 1988).

The Prevailing View by 1969

As Ross and Ross (1976) noted in their exhaustive and scholarly review of the era, the perspective on hyperactivity in the 1960s was that it remained a brain dysfunction syndrome, although of a milder magnitude than previously believed. The disorder was no longer ascribed to brain damage; instead, a focus on brain mechanisms prevailed. The disorder was also viewed as having a predominant and relatively homogeneous set of symptoms, chief among which was excessive activity level or hyperactivity. Its prognosis was now felt to be relatively benign because it was believed to be often outgrown by puberty. The recommended treatments now consisted of short-term treatment with stimulant medication and psychotherapy, in addition to the minimum-stimulation types of classrooms recommended in earlier years.

THE PERIOD 1970 TO 1979

Research in the 1970s took a quantum leap forward, with more than 2,000 published studies by the time the decade ended (Weiss & Hechtman, 1979). Numerous clinical and scientific textbooks (Cantwell, 1975; Safer & Allen, 1976; Trites, 1979; Wender, 1971) appeared, along with a most thorough and scholarly review of the literature by Ross and Ross (1976). Special journal issues were devoted to the topic (Barkley, 1978; Douglas, 1976), along with numerous scientific gatherings (Knights & Bakker, 1976, 1980). Clearly, hyperactivity had become a subject that attracted serious professional, scientific, and popular attention.

By the early 1970s, the defining features of hyperactivity or hyperkinesis were broadened to include what investigators previously felt to be only associated characteristics, including impulsivity, short attention span, low frustration tolerance, distractibility, and aggressiveness (Marwitt & Stenner, 1972; Safer & Allen, 1976). Others (Wender, 1971, 1973) persisted with the excessively inclusive concept of MBD, in which even more features (e.g., motor clumsiness, cognitive impairments, and parent–child conflict) were viewed as hallmarks of the syndrome, and in which hyperactivity was unnecessary for the diagnosis. As noted earlier, the diagnostic term "MBD" would fade from clinical and scientific usage by the end of this decade—the result in no small part of the scholarly tome by Rie and Rie (1980) and critical reviews by Rutter (1977, 1982). These writings emphasized the lack of evidence for such a broad syndrome. The symptoms were not well defined, did not correlate significantly among themselves, had no well-specified etiology, and displayed no common course and outcome. The heterogeneity of the disorder was overwhelming, and more than a few commentators took note of the apparent hypocrisy in defin-

ing an MBD syndrome with the statement that there was often little or no evidence of neurological abnormality (Wender, 1971). Moreover, even in cases of well-established cerebral damage, the behavioral sequelae were not uniform across cases, and hyperactivity was seen in only a minority of individuals. Hence, contrary to 25 years of theorizing to this point, hyperactivity was not a common sequela of brain damage; children with true brain damage did not display a uniform pattern of behavioral deficits; and children with hyperactivity rarely had substantiated evidence of neurological damage (Rutter, 1989).

Wender's Theory of MBD

This decade was notable for two different models of the nature of ADHD (see also Barkley, 1998): Wender's theory of MBD (outlined here) and Douglas's model of attention and impulse control in hyperactive children (discussed in a later section). At the start of the decade, Wender (1971) described the essential psychological characteristics of children with MBD as comprising six clusters of symptoms: problems in (1) motor behavior, (2) attentional and perceptual–cognitive functioning, (3) learning, (4) impulse control, (5) interpersonal relations, and (6) emotion. Many of the characteristics first reported by Still (1902) were echoed by Wender (1971) within these six domains of functioning.

1. Within the realm of motor behavior, the essential features were noted to be hyperactivity and poor motor coordination. Excessive speech, colic, and sleeping difficulties were thought to be related to the hyperactivity. Foreshadowing the later official designation of a group of children with attentional problems but without hyperactivity (American Psychiatric Association, 1980), Wender (1971) expressed the opinion that some of these children who were hypoactive and listless still demonstrated attention disturbances. Such cases might now be considered to have the predominantly inattentive type of ADHD. He argued that they should be viewed as having this syndrome because of their manifestation of many of the other difficulties thought to characterize it.

2. Short attention span and poor concentration were described as the most striking deficits in the domain of attention and perceptual–cognitive functioning. Distractibility and daydreaming were also included with these attention disturbances, as was poor organization of ideas or percepts.

3. Learning difficulties were another domain of dysfunction, with most of these children observed to be doing poorly in their academic performance. A large percentage were described as having specific difficulties with learning to read, with handwriting, and with reading comprehension and arithmetic.

4. Impulse control problems, or a decreased ability to inhibit behavior, were identified as a characteristic of most children with MBD. Within this general category, Wender (1971) included low frustration tolerance; an inability to delay gratification; antisocial behavior; lack of planning, forethought, or judgment; and poor sphincter control, leading to enuresis and encopresis. Disorderliness or lack of organization and recklessness (particularly with regard to bodily safety) were also listed within this domain of dysfunction.

5. In the area of interpersonal relations, Wender (1971) singled out the unresponsiveness of these children to social demands as the most serious. Extraversion, excessive independence, obstinence, stubbornness, negativism, disobedience, noncompliance, sassiness, and imperviousness to discipline were some of the characteristics that instantiated the problem with interpersonal relations.

6. Finally, within the domain of emotional difficulties, Wender (1971) included increased lability of mood, altered reactivity, increased anger, aggressiveness, and temper outbursts, as well as dysphoria. The dysphoria of these children involved the specific difficulties of anhedonia, depression, low self-esteem, and anxiety. A diminished sensitivity to both pain and punishment was also felt to typify this area of dysfunction in children with MBD. All these symptoms bear a striking resemblance to the case descriptions that Still (1902) had provided in lectures to support his contention that a defect in moral control and volitional inhibition could exist in children apart from intellectual delay.

Wender (1971) theorized that these six domains of dysfunction could be best accounted for by three primary deficits: (1) a decreased experience of pleasure and pain, (2) a generally high and poorly modulated level of activation, and (3) extraversion. A consequence of the first deficit was that children with MBD would prove less sensitive to both reward and punishment, making them less susceptible to social influence. The generally high and poorly modulated level of activation was thought to be an aspect of poor inhibition. Hyperactivity, of course, was the consummate demon-

stration of this high level of activation. The problems with poor sustained attention and distractibility were conjectured to be secondary aspects of high activation. Emotional overreactivity, low frustration tolerance, quickness to anger, and temper outbursts resulted from the poor modulation of activation. These three primary deficits, then, created a cascade of effects into the larger social ecology of these children, resulting in numerous interpersonal problems and academic performance difficulties.

Like Still (1902), Wender (1971) gave a prominent role to the construct of poor inhibition. He believed that it explained the activation difficulties and the attention problems stemming from them, as well as the excessive emotionality, the low frustration tolerance, and the hot-temperedness of these children. It is therefore quite puzzling why deficient inhibition was not made a primary symptom in this theory, in place of high activation and poor modulation of activation.

Unlike Still (1902), who attempted to devise a theory, however, Wender (1971) did not say much about normal developmental processes with respect to the three primary areas of deficit, and therefore did not clarify more precisely what might be going awry in them to give rise to these characteristics of MBD. The exception was his discussion of a diminished sensitivity to the reasonably well-understood processes of reinforcement and punishment. A higher-than-normal threshold for pleasure and pain, as noted earlier, was thought to create these insensitivities to behavioral consequences.

From a present-day perspective, Wender (1971) is also unclear about a number of issues. For instance, how would the three primary deficits account for the difficulties with motor coordination that occurred alongside hyperactivity in his category of motor control problems? It is doubtful that the high level of activation that was said to cause the hyperactivity would also cause these motor deficits. Nor is it clear just how the academic achievement deficits in reading, math, and handwriting could arise from the three primary deficits in the model. It is also unclear why the construct of extraversion needed to be proposed at all, if what Wender meant by it was reduced social inhibition. This model might be just as parsimoniously explained by the deficit in behavioral inhibition already posited. And the meaning of the term "activation" as used by Wender is not very clearly specified. Did it refer to excessive behavior, in which case hyperactivity would have sufficed? Or did it refer to level of CNS arousal, in which case ample subsequent evidence has not found this to be the case (Hastings & Barkley, 1978; Rosenthal & Allen, 1978)? To his credit, Wender recognized the abstract nature of the term "activation" as he employed it in this theory but retained it because he felt it could be used to incorporate both hyperactivity and hypoactivity in children. It is never made clear just how this could be the case, however. Finally, Wender failed to distinguish symptoms from their consequences (impairments). The former would be the behavioral manifestations directly associated with or stemming from the disorder itself, such as impulsiveness, inattention, distractibility, and hyperactivity. The latter would be the effects of these behaviors on the social environment, such as interpersonal conflict within the family, poor educational performance, peer rejection, and accident proneness, to name just a few.

From the advantage of hindsight and subsequent research over the decades since the formulation of this theory, it is also evident that Wender (1971) was combining the symptoms of ODD (and even CD) with those of ADHD to form a single disorder. Still (1902) did very much the same thing. This was understandable given that clinic-referred cases were the starting point for both theories, and many clinic-referred cases are comorbid for both disorders (ADHD and ODD). However, sufficient accumulated evidence has subsequently shown that ADHD and ODD are not the same disorder (August & Stewart, 1983; Hinshaw, 1987; Stewart, deBlois, & Cummings, 1980).

The Emergence of a Central Place for Attention Deficits

At this time, disenchantment developed over the exclusive focus on hyperactivity as the sine qua non of this disorder (Werry & Sprague, 1970). Significant at this historical juncture would be the article based on the presidential address of Virginia Douglas (1972) to the Canadian Psychological Association. She argued that deficits in sustained attention and impulse control were more likely than just hyperactivity to account for the difficulties seen in these children. These other symptoms were also seen as the major areas on which the stimulant medications used to treat the disorder had their impact. Douglas's article was historically significant in other ways as well. Her extensive and thorough battery of objective measures of various behavioral and cognitive domains, heretofore unused in research on ADHD, allowed her to rule in or out

various characteristics felt to be typical for these children in earlier clinical and scientific lore. For instance, Douglas found that hyperactive children did not necessarily and uniformly have more reading or other learning disabilities than other children, did not perseverate on concept-learning tasks, did not manifest auditory or right–left discrimination problems, and had no difficulties with short-term memory. Most important, she and Susan Campbell (1973) demonstrated that children with hyperactivity were not always more distractible than children without it, and that the sustained attention problems could emerge in conditions in which no significant distractions existed.

The McGill University research team headed by Douglas repeatedly demonstrated that hyperactive children had some of their greatest difficulties on tasks assessing vigilance or sustained attention, such as the continuous-performance test (CPT). These findings would be repeatedly reconfirmed over the next 30 years of research using CPTs (Corkum & Siegel, 1993; Frazier, Demareem, & Youngstrom, 2004). Variations of this test would eventually be standardized and commercially marketed for diagnosis of the disorder (Conners, 1995; Gordon, 1983; Greenberg & Waldman, 1992). Douglas (1972) remarked on the extreme degree of variability demonstrated during task performances by these children—a characteristic that would later be advanced as one of the defining features of the disorder. The McGill team (Freibergs, 1965; Freibergs & Douglas, 1969; Parry & Douglas, 1976) also found that hyperactive children could perform at normal or near-normal levels of sustained attention under conditions of continuous and immediate reinforcement, but that their performance deteriorated dramatically when partial reinforcement was introduced, particularly at schedules below 50% reinforcement. Campbell, Douglas, and Morgenstern (1971) further demonstrated substantial problems with impulse control and field dependence in the cognitive styles of hyperactive children. Like Still (1902), roughly 70 years earlier, Douglas commented on the probable association between deficits in attention–impulse control and deficiencies in moral development that were plaguing her subjects, particularly in their adolescent years. The research of the McGill team showed dramatic improvements in these attention deficiencies during stimulant medication treatment, as did the research at other laboratories at the time (Conners & Rothschild, 1968; Sprague, Barnes, & Werry, 1970).

Finally, of substantial significance were the observations of Douglas's colleague, Gabrielle Weiss, from her follow-up studies (see Weiss & Hechtman, 1986) that although the hyperactivity of these children often diminished by adolescence, their problems with poor sustained attention and impulsivity persisted. This persistence of the disabilities and the risk for greater academic and social maladjustment would be identified by other research teams from their own follow-up investigations (Mendelson, Johnson, & Stewart, 1971), and would be better substantiated by more rigorous studies in the next two decades (see Barkley, Fischer, Edelbrock, & Smallish, 1990; Barkley, Fischer, Smallish, & Fletcher, 2002; Gittelman, Mannuzza, Shenker, & Bonagura, 1985).

Douglas's Model of Attention Deficits

Douglas (1980a, 1980b, 1983; Douglas & Peters, 1979) later elaborated, refined, and further substantiated her model of hyperactivity, which culminated in the view that four major deficits could account for symptoms of ADHD: (1) the investment, organization, and maintenance of attention and effort; (2) the inhibition of impulsive responding; (3) the modulation of arousal levels to meet situational demands; and (4) an unusually strong inclination to seek immediate reinforcement. This perspective initiated or guided a substantial amount of research over the next 15 years, including my own early studies (Barkley, 1977, 1989b; Barkley & Ullman, 1975). It constituted a model as close to a scientific paradigm as the field of hyperactivity was likely to have in its history to that point. Yet over the next 10 years results that emerged were somewhat at odds with this perspective. Scientists began seriously to question both the adequacy of an attention model in accounting for the varied behavioral deficits seen in children with ADHD and the effects of stimulant medications on them (Barkley, 1981, 1984; Draeger, Prior, & Sanson, 1986; Haenlein & Caul, 1987; van der Meere & Sergeant, 1988a, 1988b). Also deserving of mention is that such a description of deficiencies constitutes a pattern and not a theory, given that it stipulates no conditional relations among its parts or how they orchestrate to create the problems seen in the disorder. That is, it makes no testable or falsifiable predictions apart from those contained in the pattern so described.

Douglas's article and the subsequent research published by her team were so influential that they were probably the major reasons the disorder was renamed attention deficit disorder (ADD) in 1980 with the publication of DSM-III (American Psychiatric Associa-

tion, 1980). In this revised official taxonomy, deficits in sustained attention and impulse control were formally recognized as being of greater significance in the diagnosis than hyperactivity. The shift to attention deficits rather than hyperactivity as the major difficulty of these children was useful, at least for a time, because of the growing evidence (1) that hyperactivity was not specific to this particular condition but could be noted in other psychiatric disorders (anxiety, mania, autism, etc.); (2) that there was no clear delineation between "normal" and "abnormal" levels of activity; (3) that activity was in fact a multidimensional construct; and (4) that the symptoms of hyperactivity were quite situational in nature in many children (Rutter, 1989). But this approach corrected the problem of definition for little more than a decade before these same concerns were also raised about the construct of attention (multidimensional, situationally variable, etc.). Yet some research would show that at least deficits in vigilance or sustained attention could be used to discriminate this disorder from other psychiatric disorders (Werry, 1988).

Other Developments of the Era

A number of other historical developments during this period deserve mention.

The Rise of Medication Therapy

The first of these developments was the rapidly increasing use of stimulant medication with school-age hyperactive children. This use was no doubt spawned by the significant increase in research showing that stimulants often had dramatic effects on these children's hyperactive and inattentive behavior. A second development was the use of much more rigorous scientific methodology in drug studies, due in large measure to the early studies by C. Keith Conners (then working with Leon Eisenberg at Harvard University), and somewhat later to the research of Robert Sprague at the University of Illinois, Virginia Douglas at McGill University, and John Werry in New Zealand. This body of literature became voluminous (see Barkley, 1977; Ross & Ross, 1976), with more than 120 studies published through 1976 and more than twice this number by 1995 (Swanson, McBurnett, Christian, & Wigal, 1995), making this treatment approach the most well-studied therapy in child psychiatry.

Despite the proven efficacy of stimulant medication, public and professional misgivings about its increasingly widespread use with children emerged. For example, one news account (Maynard, 1970) reported that in Omaha, Nebraska, as much as 5–10% of the children in grade schools were receiving behavior-modifying drugs. This estimate of drug treatment would later be shown to be grossly exaggerated, as much as 10-fold, due to a misplaced decimal point in the story. And this would certainly not be the last instance of the mass media's penchant for hyperbole, sensation, and scandal in their accounts of stimulant medication treatments for ADHD—a penchant that seems only to have increased over subsequent years. Yet the public interest that was generated by the initial reports led to a congressional review of the use of psychotropic medications for schoolchildren. At this same time, the claim was being advanced that hyperactivity was a "myth" arising from intolerant teachers and parents, and an inadequate educational system (Conrad, 1975; Schrag & Divoky, 1975).

Environment as Etiology

Almost simultaneous with this backlash against "drugging" schoolchildren for behavior problems was another significant development in that decade: a growing belief that hyperactivity was a result of environmental causes. It is not just coincidental that this development occurred at the same time that the United States was experiencing a popular interest in natural foods, health consciousness, the extension of life expectancy via environmental manipulations, psychoanalytic theory, and behaviorism. An extremely popular view was that allergic or toxic reactions to food additives, such as dyes, preservatives, and salicylates (Feingold, 1975), caused hyperactive behavior. It was claimed that more than half of all hyperactive children had developed their difficulties because of diet. Effective treatment could be had if families of these children would buy or make foods that did not contain the offending substances. This view became so widespread that organized parent groups or "Feingold associations," comprised mainly of parents advocating Feingold's diet, were established in almost every U.S. state, and legislation was introduced (although not passed) in California requiring that all school cafeteria foods be prepared without these substances. A sizable number of research investigations was undertaken (see Conners, 1980, for a review), the more rigorous of which found these substances to have little, if any, effect on children's behavior. A National Advisory Committee on Hyperkinesis and Food Addi-

tives (1980) that was convened to review this literature concluded more strongly than Conners that the available evidence clearly refuted Feingold's claims. Nevertheless, it would be more than 10 years before this notion receded in popularity, to be replaced by the equally unsupported hypothesis that refined sugar was more to blame for hyperactivity than were food additives (for reviews, see Milich, Wolraich, & Lindgren, 1986; Wolraich, Wilson, & White, 1995).

The emphasis on environmental causes, however, spread to include possible sources other than diet. Block (1977) advanced the rather vague notion that technological development and more rapid cultural change would result in an increasing societal "tempo," causing growing excitation or environmental stimulation. This excitation or stimulation would interact with a predisposition in some children toward hyperactivity, making it manifest. It was felt that this theory explained the apparently increasing incidence of hyperactivity in developed cultures. Ross and Ross (1982) provided an excellent critique of the theory and concluded that there was insufficient evidence in support of it and some that would contradict it. Little evidence suggested that hyperactivity incidence was increasing, though its identification among children may well have been. Nor was there evidence that its prevalence varied as a function of societal development. Instead, Ross and Ross proposed that cultural effects on hyperactivity have more to do with whether important institutions of enculturation are consistent or inconsistent in the demands made and standards set for child behavior and development. These cultural views were said to determine the threshold for deviance that will be tolerated in children, as well as to exaggerate a predisposition to hyperactivity in some children. Consistent cultures will have fewer children diagnosed with hyperactivity, as they minimize individual differences among children and provide clear and consistent expectations and consequences for behavior that conforms to the expected norms. Inconsistent cultures, by contrast, will have more children diagnosed with hyperactivity, as they maximize or stress individual differences and provide ambiguous expectations and consequences to children regarding appropriate conduct. This intriguing hypothesis remains unstudied. However, on these grounds, an equally compelling case could be made for the opposite effects of cultural influences: In highly consistent, highly conforming cultures, hyperactive behavior may be considerably more obvious in children as they are unable to conform to these societal expectations, whereas inconsistent and low-conforming cultures may tolerate deviant behavior to a greater degree as part of the wider range of behavioral expression they encourage.

A different environmental view—that poor childrearing generally and poor child behavior management specifically lead to hyperactivity—was advanced by schools of psychology/psychiatry at diametrically opposite poles. Both psychoanalysts (Bettelheim, 1973; Harticollis, 1968) and behaviorists (Willis & Lovaas, 1977) promulgated this view, though for very different reasons. The psychoanalysts claimed that parents lacking tolerance for negative or hyperactive temperament in their infants would react with excessively negative, demanding parental responses, giving rise to clinical levels of hyperactivity. The behaviorists stressed poor conditioning of children to stimulus control by commands and instructions that would give rise to noncompliant and hyperactive behavior. Both groups singled out mothers as especially etiologically important in this causal connection, and both could derive some support from studies that found negative mother–child interactions in the preschool years to be associated with the continuation of hyperactivity into the late childhood (Campbell, 1987) and adolescent (Barkley, Fischer, et al., 1990) years.

However, such correlational data cannot prove a cause. They do not prove that poor childrearing or negative parent–child interactions cause hyperactivity; they only show that such factors are associated with its persistence. It could just as easily be that the severity of hyperactivity elicits greater maternal negative reactions, and that this severity is related to persistence of the disorder over time. Supporting this interpretation are the studies of stimulant drug effects on the interactions of mothers and their hyperactive children, which show that mothers' negative and directive behavior is greatly reduced when stimulant medication is used to reduce the hyperactivity in their children (Barkley, 1989b; Barkley & Cunningham, 1979; Barkley, Karlsson, Pollard, & Murphy, 1985; Danforth, Barkley, & Stokes, 1991). Moreover, follow-up studies show that the degree of hyperactivity in childhood is predictive of its own persistence into later childhood and adolescence, apart from its association with maternal behavior (Barkley, Fischer, et al., 1990; Campbell & Ewing, 1990). And given the dramatic hereditary contribution to ADHD, it is also just as likely that the more negative, impulsive, emotional, and inattentive behavior of mothers with their hyperactive children stems in

part from the mothers' own ADHD—a factor that has never been taken into account in the analysis of such data or in interpreting findings in this area. Nevertheless, family context would still prove to be important in predicting the outcome of hyperactive children, even though the mechanism of its action was not yet specified (Weiss & Hechtman, 1986). Parent training in child behavior management, furthermore, would be increasingly recommended as an important therapy in its own right (Dubey & Kaufman, 1978; Pelham, 1977), despite a paucity of studies concerning its actual efficacy at the time (Barkley, 1989c).

The Passage of Public Law 94-142

Another highly significant development was the passage of Public Law 94-142 in 1975, mandating special educational services for children with physical, learning, and behavioral disabilities, in addition to those services already available for mental retardation (see Henker & Whalen, 1980, for a review of the legal precedents leading up to this law). Although many of its recommendations were foreshadowed by Section 504 of the Rehabilitation Act of 1973 (Public Law 93-112), the financial incentives for the states associated with the adoption of Public Law 94-142 probably encouraged its immediate and widespread implementation by them all. Programs for learning disabilities, behavioral–emotional disturbance, language disorders, physical handicaps, and motor disabilities, among others, were now required to be provided to all eligible children in all public schools in the United States.

The full impact of these widely available educational treatment programs for hyperactive children has not yet been completely appreciated, for several reasons. First, hyperactivity, by itself, was overlooked in the initial criteria set forth for behavioral and learning disabilities warranting eligibility for these special classes. Children with such disabilities typically also had to have another condition, such as a learning disability, language delay, or emotional disorder, to receive exceptional educational services. The effects of special educational resources on the outcome of hyperactivity are difficult to assess given this confounding of multiple disorders. It was only after the passage of IDEA in 1990 (and a subsequent 1991 memorandum) that the U.S. Department of Education and its Office of Special Education chose to reinterpret these regulations, thereby allowing children with ADHD to receive special educational services for ADHD per se under the "Other Health Impaired" category of IDEA. Second, the mandated services had been in existence for only a little more than a decade when the long-term outcome studies begun in the late 1970s began to be reported. Those studies (e.g., Barkley, Fischer, et al., 1990) suggested that over 35% of children with ADHD received some type of special educational placement. Although the availability of these services seems to have reduced the percentage of children with ADHD retained in grade for their academic problems, compared to earlier follow-up studies, the rates of school suspensions and expulsions did not decline appreciably from pre-1977 rates. A more careful analysis of the effects of Public Law 94-142, and especially of its more recent reauthorization as the IDEA, is in order before its efficacy for children with ADHD can be judged.

The Rise of Behavior Modification

This growing emphasis on educational intervention for children with behavioral and learning disorders was accompanied by a plethora of research on the use of behavior modification techniques in the management of disruptive classroom behavior, particularly as an alternative to stimulant medication (Allyon, Layman, & Kandel, 1975; O'Leary, Pelham, Rosenbaum, & Price, 1976). Supported in large part by their successful use for children with mental retardation, behavioral technologies were now being extended to myriad childhood disorders—not only as potential treatments of symptoms but also as theoretical statements of their origins. Although the studies demonstrated considerable efficacy of these techniques in the management of inattentive and hyperactive behavior, they were not found to achieve the same degree of behavioral improvement as the stimulants (Gittelman-Klein et al., 1976), and so did not replace them as a treatment of choice. Nevertheless, opinion was growing that the stimulant drugs should never be used as a sole intervention, but should be combined with parent training and behavioral interventions in the classroom to provide the most comprehensive management approach for the disorder.

Developments in Assessment

Another hallmark of this era was the widespread adoption of the parent and teacher rating scales developed by C. Keith Conners (1969) for the assessment of symptoms of hyperactivity, particularly during trials on stimulant medication. For at least 20 years, these simply constructed ratings of behavioral items would be the "gold standard" for rating children's hyperac-

tivity for both research purposes and treatment with medication. The scales would also come to be used for monitoring treatment responses during clinical trials. Large-scale normative data were collected, particularly for the teacher scale, and epidemiological studies throughout the world relied on both scales for assessing the prevalence of hyperactivity in their populations. Their use moved the practice of diagnosis and the assessment of treatment effects from that of clinical impression alone to one in which at least some structured, semi-objective, and quantitative measure of behavioral deviance was employed. These scales would later be criticized for their confounding of hyperactivity with aggression. This confounding called into question whether the findings of research that relied on the scales were the result of oppositional, defiant, and hostile (aggressive) features of the population or of their hyperactivity (Ullmann, Sleator, & Sprague, 1984). Nevertheless, the widespread adoption of these rating scales in this era marks a historical turning point toward the use of quantitative assessment methods that can be empirically tested and assist in determining developmental patterns and deviance from norms.

Also significant during this decade was the effort to study the social-ecological impact of hyperactive–inattentive behavior. This line of research set about evaluating the effects on family interactions produced by a child with hyperactivity. Originally initiated by Campbell (1973, 1975), this line of inquiry dominated my own research over the next decade (Barkley & Cunningham, 1979; Cunningham & Barkley, 1978, 1979; Danforth et al., 1991), particularly evaluations of the effects of stimulant medication on these social exchanges. These studies showed that children with hyperactivity were much less compliant and more oppositional during parent–child exchanges than children without it, and that their mothers were more directive, commanding, and negative than mothers of nonhyperactive children. These difficulties would increase substantially when the situation changed from free-play to task-oriented demands. Studies also demonstrated that stimulant medication resulted in significant improvements in child compliance and decreases in maternal control and directiveness. Simultaneously, Humphries, Kinsbourne, and Swanson (1978) reported similar effects of stimulant medication, all of which suggested that much of parents' controlling and negative behavior toward hyperactive children was the result rather than the cause of the children's poor self-control and inattention. At the same time, Carol Whalen and Barbara Henker at the University of California–Irvine demonstrated similar interaction conflicts between hyperactive children and their teachers and peers, as well as similar effects of stimulant medication on these social interactions (Whalen & Henker, 1980; Whalen, Henker, & Dotemoto, 1980). This line of research would increase substantially in the next decade, and would be expanded by Charles Cunningham and others to include studies of peer interactions and the effects of stimulants on them (Cunningham, Siegel, & Offord, 1985).

A Focus on Psychophysiology

The decade of the 1970s was also noteworthy for a marked increase in the number of research studies on the psychophysiology of hyperactivity in children. There were numerous published studies measuring galvanic skin response, heart rate acceleration and deceleration, various parameters of the EEG, electropupillography, averaged evoked responses, and other aspects of electrophysiology. Many researchers were investigating the evidence for theories of over- or underarousal of the CNS in hyperactivity—theories that grew out of the speculations in the 1950s on cortical overstimulation and the ideas of both Wender (1971) and Douglas (1972; both discussed earlier) regarding abnormal arousal in the disorder. Most of these studies were seriously methodologically flawed, difficult to interpret, and often contradictory in their findings. Two influential reviews at the time (Hastings & Barkley, 1978; Rosenthal & Allen, 1978) were highly critical of most investigations but concluded that if there was any consistency across findings, it might be that hyperactive children showed a sluggish or underreactive electrophysiological response to stimulation. These reviews laid to rest the belief in an overstimulated cerebral cortex as the cause of the symptoms in hyperactivity, but they did little to suggest a specific neurophysiological mechanism for the observed underreactivity. Further advances in the contributions of psychophysiology to understanding hyperactivity would await further refinements in instrumentation and in definition and diagnosis of the disorder, along with advances in computer-assisted analysis of electrophysiological measures.

An Emerging Interest in Adult MBD or Hyperactivity

Finally, the 1970s should be credited with the emergence of clinical and research interests in the existence of MBD or hyperactivity in adult clinical patients. Ini-

tial interest in adult MBD can be traced to the latter part of the 1960s, seemingly arising as a result of two events. The first of these was the publication of several early follow-up studies demonstrating persistence of symptoms of hyperactivity or MBD into adulthood in many cases (Mendelson et al., 1971; Menkes, Rowe, & Menkes, 1967). The second was the publication by Harticollis (1968) of the results of neuropsychological and psychiatric assessments of 15 adolescent and young adult patients (ages 15–25) seen at the Menninger Clinic. The neuropsychological performance of these patients suggested evidence of moderate brain damage. Their behavioral profile suggested many of the symptoms that Still (1902) initially identified in the children he studied, particularly impulsiveness, overactivity, concreteness, mood lability, and proneness to aggressive behavior and depression. Some of the patients appeared to have demonstrated this behavior uniformly since childhood. Using psychoanalytic theory, Harticollis speculated that this condition arose from an early and possibly congenital defect in the ego apparatus, in interaction with busy, action-oriented, successful parents.

The following year, Quitkin and Klein (1969) reported on two behavioral syndromes in adults that might be related to MBD. The authors studied 105 patients at the Hillside Hospital in Glen Oaks, New York, for behavioral signs of "organicity" (brain damage); behavioral syndromes that might be considered neurological "soft signs" of CNS impairment; and any EEG findings, psychological testing results, or aspects of clinical presentation and history that might differentiate these patients from patients with other types of adult psychopathology. From the initial group of 105 patients, the authors selected those having a childhood history that suggested CNS damage, including early hyperactive and impulsive behavior. These subjects were further sorted into three groups based on current behavioral profiles: those having socially awkward and withdrawn behavior (n = 12), those having impulsive and destructive behavior (n = 19), and a "borderline" group that did not fit neatly into these other two groups (n = 11). The results indicated that nearly twice as many of the patients in these three "organic" groups as those in the control group had EEG abnormalities and impairments on psychological testing indicating organicity. Furthermore, early history of hyperactive–impulsive–inattentive behavior was highly predictive of placement in the adult impulsive–destructive group, implying a persistent course of this behavioral pattern from childhood to adulthood. Of the 19 patients in the impulsive–destructive group, 17 had received clinical diagnoses of character disorders (primarily emotionally unstable types), as compared to only five patients in the socially awkward group (who received diagnoses of the schizoid and passive–dependent types).

The results were interpreted as being in conflict with the beliefs widely held at the time that hyperactive–impulsive behavior tends to wane in adolescence. Instead, the authors argued that some of these children continued into young adulthood with this specific behavioral syndrome. Quitkin and Klein (1969) also took issue with Harticollis's (1968) psychoanalytic hypothesis that demanding and perfectionistic childrearing by parents caused or contributed to this syndrome given that their impulsive–destructive patients did not uniformly experience such an upbringing. In keeping with Still's (1902) original belief that family environment could not account for this syndrome, these authors hypothesized "that such parents would intensify the difficulty, but are not necessary to the formation of the impulsive–destructive syndrome" (Quitkin & Klein, 1969, p. 140) and that the "illness shaping role of the psycho-social environment may have been overemphasized by other authors" (p. 141). Treatment with a well-structured set of demands and educational procedures, as well as with phenothiazine medication, was thought to be indicated.

Later in this decade, Morrison and Minkoff (1975) similarly argued that explosive personality disorder or episodic dyscontrol syndrome in adulthood might well be the adult sequel to the hyperactivity syndrome in childhood. They also suggested that antidepressant medications might be useful in their management; this echoed a suggestion made earlier by Huessy (1974) in a letter to the editor of a journal that both antidepressants and stimulants might be the most useful medications for the treatment of adults with hyperkinesis or MBD. But the first truly scientific evaluation of the efficacy of stimulants for adults with MBD must be credited to Wood, Reimherr, Wender, and Johnson (1976), who used a double-blind, placebo-controlled method to assess response to methylphenidate in 11 of 15 adults with MBD, followed by an open trial of pemoline (another stimulant) and the antidepressants imipramine and amitriptyline. The authors found that eight of the 11 individuals tested on methylphenidate had a favorable response, whereas 10 of the 15 individuals tested in the open trial showed a positive response to either the stimulants or the antidepressants. Others in the 1970s and into the 1980s would also make the case for the existence of an adult equivalent of childhood hyperki-

nesis or MBD and the efficacy of using stimulants and antidepressants for its management (Gomez, Janowsky, Zetin, Huey, & Clopton, 1981; Mann & Greenspan, 1976; Packer, 1978; Pontius, 1973; Rybak, 1977; Shelley & Riester, 1972). Yet not until the 1990s would both the lay public and the professional field of adult psychiatry seriously begin to recognize the adult equivalent of childhood ADHD on a more widespread basis and to recommend stimulant or antidepressant treatment in these cases (Spencer et al., 1995; Wender, 1995) and even then the view was not without its critics (Shaffer, 1994).

The work of Pontius (1973) in this decade is historically notable for her proposition that many cases of MBD in adults demonstrating hyperactive and impulsive behavior may arise from frontal lobe and caudate dysfunction. Such dysfunction would lead to "an inability to construct plans of action ahead of the act, to sketch out a goal of action, to keep it in mind for some time (as an overriding idea) and to follow it through in actions under the constructive guidance of such planning" (p. 286). Moreover, if adult MBD arises from dysfunction in this frontal–caudate network, it should also be associated with an inability "to re-program an ongoing activity and to shift within *principles* of action whenever necessary" (p. 286, original emphasis). Pontius went on to show that, indeed, adults with MBD demonstrated deficits indicative of dysfunction in this brain network. Such observations would prove quite prophetic over 20 years later, when research demonstrated reduced size in the prefrontal–caudate network in children with ADHD (Castellanos et al., 1996; Filipek et al., 1997), and when ADHD theorists argued that the neuropsychological deficits associated with it involved the executive functions, such as planning; and the control of behavior by mentally represented information, rule-governed behavior, and response fluency and flexibility; among other deficits (Barkley, 1997a, 1997c).

The Prevailing View by 1979

The 1970s closed with the prevailing view that hyperactivity was not the only or most important behavioral deficit seen in hyperactive children, and that poor attention span and impulse control were equally (if not more) important in explaining their problems. Brain damage was relegated to an extremely minor role as a cause of the disorder, at least in the realm of childhood hyperactivity or MBD; however, other brain mechanisms, such as under-arousal or under-reactivity, brain neurotransmitter deficiencies (Wender, 1971), or neurological immaturity (Kinsbourne, 1977), were viewed as promising. Greater speculation about potential environmental causes or irritants emerged, particularly diet and childrearing. Thus, the most frequently recommended therapies for hyperactivity were not only stimulant medication but also widely available special education programs, classroom behavior modification, dietary management, and parent training in child management skills. A greater appreciation for the effects of hyperactive children on their immediate social ecology, and for the impact of stimulant medication in altering these social conflicts, was beginning to emerge.

However, the sizable discrepancy between North American and European views of the disorder remained: North American professionals continued to recognize the disorder as more common, in need of medication, and more likely to be an attention deficit, while those in Europe continued to view it as uncommon, defined by severe overactivity, and associated with brain damage. Those children in North America diagnosed as having hyperactivity or attention deficits would in Europe likely be diagnosed as having CD and be treated with psychotherapy, family therapy, and parent training in child management. Medication would be disparaged and little used. Nevertheless, the view that attention deficits were as important in the disorder as hyperactivity was beginning to make its way into European taxonomies (e.g., the *International Classification of Diseases*, ninth revision [ICD-9]; World Health Organization, 1978). Finally, in the 1970s there was some recognition that there were adult equivalents of childhood hyperactivity or MBD, that they might be indicative of frontal–caudate dysfunction, and that these cases responded to the same medication treatments suggested earlier for childhood ADHD (the stimulants and antidepressants).

THE PERIOD 1980 TO 1989

The exponential increase in research on hyperactivity characteristic of the 1970s continued unabated into the 1980s, making hyperactivity the most well-studied childhood psychiatric disorder in existence. More books were written, conferences convened, and scientific articles presented during this decade than in any previous historical period. This decade would become known for its emphasis on attempts to develop more specific diagnostic criteria; the differential conceptualization and diagnosis of hyperactivity versus other

psychiatric disorders; and, later in the decade, critical attacks on the notion that an inability to sustain attention was the core behavioral deficit in ADHD.

The Creation of an ADD Syndrome

Marking the beginning of this decade was the publication of DSM-III (American Psychiatric Association, 1980) and its radical reconceptualization (from that in DSM-II) of the hyperkinetic reaction of childhood diagnosis to that of ADD (with or without hyperactivity). The new diagnostic criteria were noteworthy for not only their greater emphasis on inattention and impulsivity as defining features of the disorder but also their creation of much more specific symptom lists, an explicit numerical cutoff score for symptoms, specific guidelines for age of onset and duration of symptoms, and the requirement of exclusion of other childhood psychiatric conditions as better explanations of the presenting symptoms. This was also a radical departure from the ICD-9 criteria set forth by the World Health Organization (1978) in its own taxonomy of child psychiatric disorders, which continued to emphasize pervasive hyperactivity as a hallmark of this disorder.

Even more controversial was the creation of subtypes of ADD, based on the presence or absence of hyperactivity (+ H/– H), in the DSM-III criteria. Little, if any, empirical research on this issue existed at the time these subtypes were formulated. Their creation in the official nomenclature of psychiatric disorders would, by the end of the 1980s, initiate numerous research studies into their existence, validity, and utility, along with a search for other potentially useful ways of subtyping ADD (situational pervasiveness, presence of aggression, stimulant drug response, etc.). Although the findings were at times conflicting, the trend in these studies was that children with ADD – H differed from those with ADD + H in some important domains of current adjustment. Those with ADD – H were characterized as more prone to daydreaming, hypoactive, lethargic, and disabled in academic achievement, but as substantially less aggressive and less rejected by their peers (Barkley, Grodzinsky, & DuPaul, 1992; Carlson, 1986; Goodyear & Hynd, 1992; Lahey & Carlson, 1992). Unfortunately, this research came too late to be considered in the subsequent revision of DSM-III.

In that revision (DSM-III-R; American Psychiatric Association, 1987), only the diagnostic criteria for ADD + H (now renamed ADHD; see "ADD Becomes ADHD," below) were stipulated. ADD – H was no longer officially recognized as a subtype of ADD, but was relegated to a minimally defined category, undifferentiated ADD. This reorganization was associated with an admonition that far more research on the utility of this subtyping approach was necessary before its place in this taxonomy could be identified. Despite the controversy that arose over the demotion of ADD – H in this fashion, it was actually a prudent gesture on the part of the committee asked to formulate these criteria. At the time, the committee (on which I served) had little available research to guide its deliberations in this matter. There was simply no indication whether ADD – H had a similar or qualitatively different type of attention deficit, which would make it a separate childhood psychiatric disorder in its own right. Rather than continue merely to conjecture about the nature of the subtype and how it should be diagnosed, the committee essentially placed the concept in abeyance until more research was available to its successor committee to guide its definition. Notable in the construction of DSM-III-R was its emphasis on the empirical validation of its diagnostic criteria through a field trial, which guided the selection of items for the symptom list and the recommended cutoff score on that list (Spitzer, Davies, & Barkley, 1990).

The Development of Research Diagnostic Criteria

At the same time that the DSM-III criteria for ADD + H and ADD – H were gaining recognition, others attempted to specify research diagnostic criteria (Barkley, 1982; Loney, 1983). My own efforts in this endeavor were motivated by the rather idiosyncratic and highly variable approach to diagnosis being used in clinical practice up to that time, the vague or often unspecified criteria used in published research studies, and the lack of specificity in current theoretical writings on the disorder up to 1980. There was also the more pragmatic consideration that, as a young scientist attempting to select hyperactive children for research studies, I had no operational or consensus-based criteria available for doing so. Therefore, I set forth a more operational definition of hyperactivity, or ADD + H. This definition not only required the usual parent and/or teacher complaints of inattention, impulsivity, and overactivity, but it also stipulated that these symptoms had to (1) be deviant for the child's mental age, as measured by well-standardized child behavior rating scales; (2) be relatively pervasive within the jurisdiction of the major

caregivers in the child's life (parent/home and teacher/school); (3) have developed by 6 years of age; and (4) have lasted at least 12 months (Barkley, 1982).

Concurrently, Loney (1983) and her colleagues had been engaged in a series of historically important studies that would differentiate the symptoms of hyperactivity or ADD + H from those of aggression or conduct problems (Loney, Langhorne, & Paternite, 1978; Loney & Milich, 1982). Following an empirical/statistical approach to developing research diagnostic criteria, Loney demonstrated that a relatively short list of symptoms of hyperactivity could be empirically separated from a similarly short list of aggression symptoms. Empirically derived cutoff scores on these symptom ratings by teachers could create these two semi-independent constructs. These constructs would prove highly useful in accounting for much of the heterogeneity and disagreement across studies. Among other things, it would become well established that many of the negative outcomes of hyperactivity in adolescence and young adulthood were actually due to the presence and degree of aggression coexisting with the hyperactivity. Purely hyperactive children would be shown to display substantial cognitive problems with attention and overactivity, whereas purely aggressive children would not. Previous findings of greater family psychopathology in hyperactive children would also be shown to be primarily a function of the degree of coexisting aggression or CD in the children (August & Stewart, 1983; Lahey et al., 1988). Furthermore, hyperactivity would be found to be associated with signs of developmental and neurological delay or immaturity, whereas aggression was more likely to be associated with environmental disadvantage and family dysfunction (Hinshaw, 1987; Milich & Loney, 1979; Paternite & Loney, 1980; Rutter, 1989; Werry, 1988; Weiss & Hechtman, 1986). The need for future studies to specify clearly the makeup of their samples along these two dimensions was now obvious. And the raging debate as to whether hyperactivity was separate from or merely synonymous with conduct problems would be settled by the important research discovery of the semi-independence of these two behavioral dimensions and their differing correlates (Ross & Ross, 1982). These findings would also lead to the demise of the commonplace use of the Conners's 10-item Hyperactivity Index to select children as hyperactive. It would now be shown that many of these items actually assessed aggression rather than hyperactivity, resulting in samples of children with mixed disorders (Ullmann et al., 1984).

The laudable drive toward greater clarity, specificity, and operational defining of diagnostic criteria would continue throughout this decade. Pressure would now be exerted from experts within the field (Quay, 1988b; Rutter, 1983, 1989; Werry, 1988) to demonstrate that the symptoms of ADHD could distinguish it from other childhood psychiatric disorders—a crucial test for the validity of a diagnostic entity—rather than continuing simply to demonstrate differences from nondisordered populations. The challenge would not be easily met. Eric Taylor (1986) and colleagues in Great Britain made notable advances in further refining the criteria and their measurement along more empirical lines. Taylor's (1989) statistical approach to studying clusters of behavioral disorders resulted in the recommendation that a syndrome of hyperactivity could be valid and distinct from other disorders, particularly conduct problems. This distinction required that the symptoms of hyperactivity and inattention be excessive and handicapping to the children; occur in two of three broadly defined settings (e.g., home, school, and clinic); be objectively measured rather than subjectively rated by parents and teachers; develop before age 6; last at least 6 months; and exclude children with autism, psychosis, anxiety, or affective/mood disorders (depression, mania, etc.).

Efforts to develop research diagnostic criteria for ADHD eventually led to an international symposium on the subject (Sergeant, 1988) and a general consensus that subjects selected for research on ADHD should at least meet the following criteria: (1) reports of problems with activity and attention by adults in at least two of three independent settings (home, school, clinic); (2) endorsement of at least three of four difficulties with activity and three of four with attention; (3) onset before 7 years of age; (4) duration of 2 years; (5) significantly elevated scores on parent–teacher ratings of these ADHD symptoms; and (6) exclusion of autism and psychosis. These proposed criteria were quite similar to others developed earlier in the decade (Barkley, 1982) but provided for greater specificity of symptoms of overactivity and inattention, and a longer duration of symptoms.

Subtyping of ADD

Also important in this era was the attempt to identify useful approaches to subtyping other than those just based on the degree of hyperactivity (+ H/– H) or aggression associated with ADD. A significant though underappreciated line of research by Roscoe Dykman

and Peggy Ackerman at the University of Arkansas distinguished between ADD with and ADD without learning disabilities, particularly reading impairments. Their research (Ackerman, Dykman, & Oglesby, 1983; Dykman, Ackerman, & Holcomb, 1985) and that of others (e.g., McGee, Williams, Moffit, & Anderson, 1989) showed that some of the cognitive deficits (verbal memory, intelligence, etc.) formerly attributed to ADHD were actually more a function of the presence and degree of language/reading difficulties than of ADHD. And although some studies showed that ADHD with reading disabilities is not a distinct subtype of ADHD (Halperin, Gittelman, Klein, & Rudel, 1984), the differential contributions of reading disorders to the cognitive test performance of children with ADHD required that subsequent researchers carefully select subjects with pure ADHD not associated with reading disability. If they did not, then they at least should identify the degree to which reading disorders exist in the sample and partial out the effects of these disorders on the cognitive test results.

Others in this era attempted to distinguish between "pervasive" and "situational" hyperactivity; the former was determined by the presence of hyperactivity at home and school, and the latter referred to hyperactivity in only one of these settings (Schachar, Rutter, & Smith, 1981). It would be shown that children with pervasive hyperactivity were likely to have more severe behavioral symptoms, greater aggression and peer relationship problems, and poor academic achievement. DSM-III-R (American Psychiatric Association, 1987) incorporated this concept into an index of severity of ADHD (see the last portion of Table 2.1). British scientists even viewed pervasiveness as an essential criterion for the diagnosis of a distinct syndrome of hyperactivity (as noted earlier). However, research appearing at the end of the decade (Costello, Loeber, & Stouthamer-Loeber, 1991) demonstrated that such group differences were more likely to be the results of differences in the source of the information used to classify the children (parents vs. teachers) than of actual behavioral differences between the situational and pervasive subgroups. This did not mean that symptom pervasiveness might not be a useful means of subtyping or diagnosing ADHD, but that more objective means of establishing it were needed than just comparing parent and teacher ratings on a questionnaire.

A different and relatively understudied approach to subtyping was created by the presence or absence of significant anxiety or affective disturbance. Several studies demonstrated that children with both ADHD and significant problems with anxiety or affective disturbance were likely to show poor or adverse responses to stimulant medication (Taylor, 1983; Voelker, Lachar, & Gdowski, 1983) and would perhaps respond better to antidepressant medications (Pliszka, 1987). The utility of this latter subtyping approach would be investigated and supported further in the next decade (DuPaul, Barkley, & McMurray, 1994; Tannock, 2000).

ADD Becomes ADHD

Later in the 1980s, in an effort to improve further the criteria for defining this disorder, the DSM was revised (DSM-III-R; American Psychiatric Association, 1987) as noted earlier, resulting in the renaming of the disorder to ADHD. The revisions were significant in several respects. First, a single list of symptoms and a single cutoff score replaced the three separate lists (inattention, impulsivity, and hyperactivity) and cutoff score in DSM-III. Second, the item list was now based more on empirically derived dimensions of child behavior from behavior rating scales, and the items and cutoff score underwent a large field trial to determine their sensitivity, specificity, and power to distinguish ADHD from other psychiatric disorders and from the absence of disorder (Spitzer et al., 1990). Third, the need was stressed that one had to establish the symptoms as developmentally inappropriate for the child's mental age. Fourth, the coexistence of mood disorders with ADHD no longer excluded the diagnosis of ADHD. And, more controversially, the subtype of ADD – H was removed as a subtype and relegated to a vaguely defined category, undifferentiated ADD, which was in need of greater research on its merits. ADHD was now classified with two other behavioral disorders (ODD and CD) in a supraordinate family or category known as the "disruptive behavior disorders," in view of their substantial overlap or comorbidity in clinic-referred populations of children.

ADHD as a Motivation Deficit Disorder

One of the more interesting conceptual developments only began to emerge in the latter half of the decade. This was the nascent and almost heretical view that ADHD was not actually a disorder of attention. Doubt about the central importance of attention to the disorder crept in late in the 1970s, as some researchers more fully plumbed the depths of the attention construct

with objective measures, while others took note of the striking situational variability of the symptoms (Douglas & Peters, 1979; Rosenthal & Allen, 1978; Routh, 1978; Sroufe, 1975). As more rigorous and technical studies of attention in children with ADHD appeared in the 1980s, an increasing number failed to find evidence of problems with attention under some experimental conditions, while observing them under others (for reviews, see Douglas, 1983, 1988; also see Barkley, 1984; Draeger et al., 1986; Sergeant, 1988; Sergeant & van der Meere, 1989; van der Meere & Sergeant, 1988a, 1988b). Moreover, if attention was conceptualized as involving the perception, filtering, and processing of information, no substantial evidence could be found in these studies for any such deficits. These findings, coupled with the realization that both instructional and motivational factors in an experiment played a strong role in determining the presence and degree of ADHD symptoms, led some investigators to hypothesize that deficits in motivation might be a better model for explaining the symptoms seen in ADHD (Glow & Glow, 1979; Rosenthal & Allen, 1978; Sroufe, 1975). Following this line of reasoning, others pursued a behavioral or functional analysis of these symptoms, resulting in hypothesized deficits in the stimulus control over behavior, particularly by rules and instructions. I argued that such deficits arose from neurological factors (Barkley, 1988a), whereas others argued that they arose from poor training of the child by parents (Willis & Lovaas, 1977).

I initially raised the possibility that rule-governed behavior might account for many of the deficits in ADHD but later amended this view to include the strong probability that response to behavioral consequences might also be impaired and could conceivably account for the problems with following rules (Barkley, 1981, 1984, 1990). Others independently advanced the notion that a deficit in responding to behavioral consequences, not attention, might be the difficulty in ADHD (Benninger, 1989; Haenlein & Caul, 1987; Quay, 1988a; Sagvolden, Wultz, Moser, Moser, & Morkrid, 1989; Sergeant, 1988; van der Meere & Sergeant, 1988a). That is, ADHD might arise out of an insensitivity to consequences (reinforcement, punishment, or both). This insensitivity was viewed as being neurological in origin. Yet this idea was not new, having been advanced some 10–20 years earlier by investigators in Australia (Glow & Glow, 1979), by those studying children with conduct problems (see Patterson, 1982, for a review), and by Wender (1971) in his classic text on MBD (discussed earlier). What was original in these more recent ideas is the greater specificity of their hypotheses and increasing evidence supporting them. Others continued to argue against the merits of a Skinnerian or functional analysis of the deficits in ADHD (Douglas, 1989), and for the continued explanatory value of cognitive models of attention in accounting for the deficits in ADHD.

The appeal of the motivational model came from several different sources: (1) its greater explanatory value in accounting for the more recent research findings on situational variability in attention in ADHD; (2) its consistency with neuroanatomical studies suggesting decreased activation of brain reward centers and their cortical–limbic regulating circuits (Lou et al., 1984, 1989); (3) its consistency with studies of the functions of dopamine pathways in regulating locomotor behavior and incentive or operant learning (Benninger, 1989); and (4) its greater prescriptive power in suggesting potential treatments for the ADHD symptoms. Whether or not ADHD would be labeled a motivational deficit, there was little doubt that these new theories based on the construct of motivation required altering the way in which this disorder was to be conceptualized. From here on, any attempts at theory construction would need to incorporate some components and processes dealing with motivation or effort.

Other Developments of the Era

The Increasing Importance of Social Ecology

The 1980s also witnessed considerably greater research into the social-ecological impact of ADHD symptoms on the children, their parents (Barkley, 1989b; Barkley, Karlsson, & Pollard, 1985; Mash & Johnston, 1982), teachers (Whalen, Henker, & Dotemoto, 1980, 1981), siblings (Mash & Johnston, 1983), and peers (Cunningham et al., 1985; Henker & Whalen, 1980). These investigations further explored the effects of stimulant medications on these social systems; they buttressed the conclusion that children with ADHD elicit significant negative, controlling, and hostile or rejecting interactions from others, which can be greatly reduced by stimulant medication. From these studies emerged the view that the disabilities associated with ADHD do not rest solely in a child, but in the interface between the child's capabilities and the environmental demands made within the social-ecological context in which that child must perform (Whalen & Henker, 1980). Changing the attitudes, behaviors, and expecta-

tions of caregivers, as well as the demands they make on children with ADHD in their care, should result in changes in the degree to which such children are disabled by their behavioral deficits.

Theoretical Advances

During this decade, Herbert Quay adopted the neuropsychological model of anxiety by Jeffrey Gray (1982, 1987, 1994) to explain the origin of the poor inhibition evident in ADHD (Quay, 1988a, 1988b, 1997). Gray identified both a behavioral inhibition system (BIS) and a behavioral activation system (BAS) as being critical to understanding emotion. He also stipulated mechanisms for basic nonspecific arousal and for the appraisal of incoming information that must be critical elements of any attempt to model the emotional functions of the brain. According to this theory, signals of reward serve to increase activity in the BAS, thus giving rise to approach behavior and the maintenance of such behavior. Active avoidance and escape from aversive consequences (negative reinforcement) likewise activate this system. Signals of impending punishment (particularly conditioned punishment), as well as frustrative nonreward (an absence of previously predictable reward), increase activity in the BIS. Another system is the fight–flight system, which reacts to unconditioned punitive stimuli.

Quay's use of this model for ADHD indicated that the impulsiveness characterizing the disorder could arise from diminished activity in the brain's BIS. This model predicted that those with ADHD should prove less sensitive to such signals, particularly in passive avoidance paradigms (Quay, 1988b). The theory also specifies predictions that can be used to test and even falsify the model as it applies to ADHD. For instance, Quay (1988a, 1988b) predicted that there should be greater resistance to extinction following periods of continuous reinforcement in those with ADHD, but less resistance when training conditions involve partial reward. They should also demonstrate a decreased ability to inhibit behavior in passive avoidance paradigms in which avoidance of the punishment is achieved through the inhibition of responding. And those with ADHD should also demonstrate diminished inhibition to signals of pain and novelty, as well as to conditioned signals of punishment. Finally, Quay predicted increased rates of responding by those with ADHD under fixed-interval or fixed-ratio schedules of consequences. Some of these predictions were supported by subsequent research; others either remain to be investigated more fully and rigorously, or have not been completely supported by the available evidence (see Milich, Hartung, Martin, & Haigler, 1994; Quay, 1997). Nevertheless, the theory remains a viable one for explaining the origin of the inhibitory deficits in ADHD and continues to deserve further research.

Further Developments in Nature, Etiology, and Course

Another noteworthy development in this decade was the greater sophistication of research designs in the attempt to explore the unique features of ADHD relative to other psychiatric conditions, rather than just in comparison to the absence of disorder. As Rutter (1983, 1989) noted repeatedly, the true test of the validity of a syndrome of ADHD is the ability to differentiate its features from other psychiatric disorders of children, such as mood or anxiety disorders, learning disorders, and particularly CD. Those studies that undertook such comparisons indicated that situational hyperactivity was not consistent in discriminating among psychiatric populations, but that difficulties with attention and pervasive (home and school) hyperactivity were more reliable in doing so and were often associated with patterns of neuropsychological immaturity (Firestone & Martin, 1979; Gittelman, 1988; McGee, Williams, & Silva, 1984a, 1984b; Rutter, 1989; Taylor, 1988; Werry, 1988).

The emerging interest in comparing children with ADD + H to those with ADD – H furthered this line of inquiry by demonstrating relatively unique features of each group in contrast to each other (see Chapters 2 and 17) and to groups of children with learning disabilities and no disability (Barkley, DuPaul, & McMurray, 1990, 1991). Further strengthening the position of ADHD as a psychiatric syndrome is evidence from family aggregation studies that relatives of children with ADHD have a different pattern of psychiatric disturbance than that of children with CD or mixed ADHD and CD (Biederman, Munir, & Knee, 1987; Lahey et al., 1988). Children with pure ADHD were more likely to have relatives with ADHD, academic achievement problems, and dysthymia, whereas those children with CD had a greater prevalence of relatives with CD, antisocial behavior, substance abuse, depression, and marital dysfunction. This finding led to speculation that ADHD has a different etiology than CD. The former was said to arise out of a biologically based disorder of

temperament or a neuropsychological delay; the latter, from inconsistent, coercive, and the dysfunctional childrearing and management frequently associated with parental psychiatric impairment (Hinshaw, 1987; Loeber, 1990; Patterson, 1982, 1986).

Equally elegant research examined potential etiologies of ADHD. Several studies on cerebral blood flow revealed patterns of underactivity in the prefrontal areas of the CNS and their rich connections to the limbic system via the striatum (Lou et al., 1984, 1989). Other studies (Hunt, Cohen, Anderson, & Minderaa, 1988; Rapoport & Zametkin, 1988; Shaywitz, Shaywitz, Cohen, & Young, 1983; Shekim, Glaser, Horwitz, Javaid, & Dylund, 1988; Zametkin & Rapoport, 1986) of brain neurotransmitters provided further evidence that deficiencies in dopamine, norepinephrine, or both, may be involved in explaining these patterns of brain underactivity—patterns arising in precisely those brain areas in which dopamine and norepinephrine are most involved. Drawing these lines of evidence together even further is the fact that these brain areas are critically involved in response inhibition, motivational learning, and response to reinforcement. More rigorous published studies on the hereditary transmission of ADHD (Goodman & Stevenson, 1989) indicated a strong heritability for ADHD symptoms.

Follow-up studies appearing in this decade were also more methodologically sophisticated, and hence more revealing of not only widespread maladjustment in children with ADHD as they reached adolescence and adulthood but also potential mechanisms involved in the differential courses shown within this population (Barkley, Fischer, et al., 1990, 1991; Fischer, Barkley, Edelbrock, & Smallish, 1990; Gittelman et al., 1985; Lambert, 1988; Weiss & Hechtman, 1993). These findings are discussed in Chapter 9. Again, neuropsychological delays, the presence and pervasiveness of early aggression, and mother–child conflict were associated with a different, and more negative, outcome in later childhood and adolescence than was ADHD alone (Campbell, 1987; Paternite & Loney, 1980).

There was also a movement during this decade away from the strict reliance on clinic-referred samples of children with ADHD toward the use of community-derived samples. This change was prompted by the widely acknowledged bias that occurs among clinic-referred samples of children with ADHD as a result of the process of referral itself. It is well known that children who are referred are often more (though not always the most) impaired, have more numerous comorbid conditions, are likely to have associated family difficulties, and are skewed toward those socioeconomic classes that value the utilization of mental health care resources. Such biases can create findings that are not representative of the nature of the disorder in its natural state. For instance, it has been shown that the ratio of boys to girls within clinic-referred samples of children with ADHD may range from 5:1 to 9:1, and that girls with ADHD within these samples are as likely to be as aggressive or oppositional as boys (see Chapter 2). By contrast, in samples of children with ADHD derived from community- or school-based samples, the ratio of boys to girls is only 2.5:1, and girls with ADHD are considerably less likely than boys to be aggressive. For these and other reasons, a greater emphasis on studying epidemiological samples of children, and the rates and nature of ADHD within them (Offord et al., 1987), arose toward the latter half of the 1980s.

Developments in Assessment

The 1980s also witnessed some advances in the tools of assessment, in addition to those for treatment. The Child Behavior Checklist (CBCL; Achenbach & Edelbrock, 1983, 1986) emerged as a more comprehensive, more rigorously developed and better-normed alternative to the Conners Rating Scales (Barkley, 1988c). It would become widely adopted in research on child psychopathology in general, not just in ADHD, by the end of the decade. Other rating scales more specific to ADHD were also developed, such as the ADD – H Comprehensive Teacher's Rating Scale (ACTeRS; Ullmann et al., 1984), the Home and School Situations Questionnaires (Barkley & Edelbrock, 1987; DuPaul & Barkley, 1992), the Child Attention Profile (see Barkley, 1988c), and the ADHD Rating Scale (DuPaul, 1991).

Gordon (1983) developed, normed, and commercially marketed a small, portable, computerized device that administered two tests believed to be sensitive to the deficits in ADHD. One was a CPT measuring vigilance and impulsivity, and the other was a direct reinforcement of low rates (DRL) test assessing impulse control. This device became the first commercially available objective means of assessment for children with ADHD. Although the DRL test showed some promise in early research (Gordon, 1979), it was subsequently shown to be insensitive to stimulant medication effects (Barkley, Fischer, Newby, & Breen, 1988) and was eventually deemphasized as useful in the diagnosis in ADHD. The

CPT, by contrast, showed satisfactory discrimination of children with ADHD from nondisabled groups and was sensitive to medication effects (Barkley et al., 1988; Gordon & Mettelman, 1988). Although cautionary statements indicated that more research evidence was needed to evaluate the utility of the instrument (Milich, Pelham, & Hinshaw, 1985), and that its false-negative rate (misses of children with legitimate ADHD) might be greater than that desired in a diagnostic tool, the device and others like it (Conners, 1995; Greenberg & Waldman, 1992) found a wide clinical following by the next decade.

Greater emphasis was also given to developing direct behavioral observation measures of ADHD symptoms that could be taken in the classroom or clinic, and that would be more objective and useful adjuncts to the parent and teacher rating scales in the diagnostic process. Abikoff, Gittelman-Klein, and Klein (1977) and O'Leary (1981) developed classroom observation codes with some promise for discriminating between children with ADHD and those with other or no disabilities (Gittelman, 1988). Roberts (1979), drawing on the earlier work of Routh and Schroeder (1976) and Kalverboer (1988), refined a laboratory playroom observation procedure that discriminated not only between children with ADHD and nondisabled children, but also between children with aggression or mixed aggression and ADHD. This coding system had excellent 2-year stability coefficients. Somewhat later, I streamlined the system (Barkley, 1988b) for more convenient clinical or classroom use and found it to be sensitive to stimulant medication effects (Barkley et al., 1988), to differentiate between children with ADD + H and ADD – H (Barkley, DuPaul, et al., 1991), and to correlate well with parent and teacher ratings of ADHD symptoms (Barkley, 1991). Nevertheless, problems with developing normative data and the practical implementation of such a procedure in busy clinic practices remained hindrances to its widespread adoption.

Developments in Treatment

Developments also continued in the realm of treatments for ADHD. Comparisons of single versus combined treatments were more common during the decade (Barkley, 1989a), as was the use of more sophisticated experimental designs (Hinshaw, Henker, & Whalen, 1984; Pelham, Schnedler, Bologna, & Contreras, 1980) and mixed interventions (Satterfield, Satterfield, & Cantwell, 1981). Several of these historical developments in treatment require mention. The first was the emergence of a new approach to the treatment of ADHD: cognitive-behavioral therapy, or CBT (Camp, 1980; Douglas, 1980a; Kendall & Braswell, 1985; Meichenbaum, 1988). Founded on the work of Russian neuropsychologists (Vygotsky and Luria), North American developmental and cognitive psychologists (Flavell, Beach, & Chinsky, 1966), and early cognitive-behavioral theories (Meichenbaum, 1977), the CBT approach stressed the need to develop self-directed speech in impulsive children to guide their definition of and attention to immediate problem situations, to generate solutions to these problems, and to guide their behavior as the solutions were performed. Self-evaluation, self-correction, and self-directed use of consequences were also viewed as important (Douglas, 1980a, 1980b). Although the first reports of the efficacy of this approach appeared in the late 1960s and the 1970s (Bornstein & Quevillon, 1976; Meichenbaum & Goodman, 1971), it was not until the 1980s that the initial claims of success with nonclinical populations of impulsive children were more fully tested in clinical populations of children with ADHD. The initial results were disappointing (Abikoff, 1987; Gittelman & Abikoff, 1989). Generally, they indicated some degree of improvement in impulsiveness on cognitive laboratory tasks; however, the improvement was insufficient to be detected in teacher or parent ratings of school and home ADHD behaviors, and CBT was certainly not as effective as stimulant medication (Brown, Wynne, & Medenis, 1985). Many continued to see some promise in these techniques (Barkley, 1981, 1989b; Meichenbaum, 1988; Whalen, Henker, & Hinshaw, 1985), particularly when they were implemented in natural environments by important caregivers (parents and teachers); others ended the decade with a challenge to those who persisted in their support of the CBT approach to provide further evidence for its efficacy (Gittelman & Abikoff, 1989). Such evidence would not be forthcoming. Later, even the conceptual basis for the treatment came under attack as being inconsistent with Vygotsky's theory of the internalization of language (Diaz & Berk, 1995).

A second development in treatment was the publication of a specific parent training format for families of children with ADHD and oppositional behavior. A specific set of steps for training parents of children with ADHD in child behavior management skills was developed (Barkley, 1981) and refined (Barkley, 1997b). The approach was founded on a substantial research literature (Barkley, 1997b; Forehand & McMahon,

1981; Patterson, 1982) demonstrating the efficacy of differential attention and time-out procedures for treating oppositional behavior in children—a behavior frequently associated with ADHD. These two procedures were coupled with additional components based on a theoretical formulation of ADHD as a developmental disorder that is typically chronic and associated with decreased rule-governed behavior and an insensitivity to certain consequences, particularly mild or social reinforcement. These components included counseling parents to conceptualize ADHD as a developmentally disabling condition; implementing more powerful home token economies to reinforce behavior, rather than relying on attention alone; using shaping techniques to develop nondisruptive, independent play; and training parents in cognitive-behavioral skills to teach their children during daily management encounters, particularly in managing disruptive behavior in public places (see Chapter 21). Because of the demonstrated impact of parental and family dysfunction on the severity of children's ADHD symptoms, on the children's risk for developing ODD and CD, and on the parents' responsiveness to treatments for the children, clinicians began to pay closer attention to intervention in family systems rather than just in child management skills. Noteworthy among these attempts were the modifications to the previously described parent training program by Charles Cunningham at McMaster University Medical Center (Cunningham, 1990, 2006). Arthur Robin at Wayne State University and the Children's Hospital of Michigan, and Sharon Foster at West Virginia University (Robin & Foster, 1989) also emphasized the need for work on family systems, as well as on problem-solving and communication skills in treating the parent–adolescent conflicts so common in families of teenagers with ADHD (see Chapter 22 for a discussion of this approach).

A similar increase in more sophisticated approaches to the classroom management of children with ADHD occurred in this era (Barkley, Copeland, & Sivage, 1980; Pelham et al., 1980; Pfiffner & O'Leary, 1987; Whalen & Henker, 1980). These developments were based on earlier promising studies in the 1970s of contingency management methods in hyperactive children (Allyon et al., 1975; see Chapter 24 for the details of such an approach). Although these methods may not produce the degree of behavioral change seen with the stimulant medications (Gittelman et al., 1980), they provide a more socially desirable intervention that can be a useful alternative when children have mild ADHD and cannot take stimulants or when their parents decline the prescription. More often, these methods serve as an adjunct to medication therapy to further enhance academic achievement.

The fourth area of treatment development was in social skills training for children with ADHD (see Chapter 23). Hinshaw and colleagues (1984) developed a program for training children with ADHD in anger control techniques. This program demonstrated some initial short-term effectiveness in assisting these children to deal with this common deficit in their social skills and emotional control (Barkley et al., 2000). Related approaches to social skills training for children with ADHD also showed initially promising results (Pfiffner & McBurnett, 1997), but subsequent research did not bear out this promise and suggested that some children with ADHD may even become more aggressive after participation in such group training formats (see Chapter 23).

Finally, medication treatments for children with ADHD expanded to include the use of the tricyclic antidepressants, particularly for those children with characteristics that contraindicated use of a stimulant medication (e.g., Tourette syndrome or other tic disorders) or for those with anxiety/depression (Pliszka, 1987). The work of Joseph Biederman and his colleagues at Massachusetts General Hospital (Biederman, Baldessarini, Wright, Knee, & Harmatz, 1989; Biederman, Gastfriend, & Jellinek, 1986) on the safety and efficacy of tricyclic antidepressants encouraged the rapid adoption of these drugs by many practitioners (see Ryan, 1990), particularly when the stimulants, such as methylphenidate (Ritalin), were receiving such negative publicity in the popular media (see the next section). There simultaneously appeared initially positive research reports on the use of the antihypertensive drug clonidine in the treatment of children with ADHD, particularly those with very high levels of hyperactive–impulsive behavior and aggression (Hunt, Caper, & O'Connell, 1990; Hunt, Minderaa, & Cohen, 1985; see Chapter 27).

Developments in Public Awareness

Several noteworthy developments also occurred in the public forum during this decade. Chief and most constructive among these was the blossoming of numerous parent support associations for families with ADHD. Although less than a handful of these existed in the early 1980s, within 9 years there would be well over

100 such associations throughout the United States alone. By the end of the decade, these would begin to organize into national networks and political action organizations known respectively as CHADD (originally Children with ADD, now Children and Adults with ADHD) and the Attention Deficit Disorder Association (ADDA). With this greater public/parent activism, initiatives were taken to have state and federal laws reevaluated and, it was hoped, changed to include ADHD as an educational disability in need of special educational services in public schools.

When it was passed in 1975, Public Law 94-142 included the concept of MBD under the category of learning disabilities that would be eligible for special educational services. But it did not include hyperactivity, ADD, or ADHD in its description of learning or behavioral disorders eligible for mandated special services in public school. This oversight would lead many public schools to deny access for children with ADD or ADHD to such services, and would cause much parental and teacher exasperation in trying to get educational recognition and assistance for this clearly academically disabling disorder. Other parents would initiate lawsuits against private schools for learning-disabled students for educational malpractice in failing to provide special services for children with ADHD (Skinner, 1988). By the early 1990s, these lobbying efforts would be partially successful in getting the U.S. Department of Education to reinterpret Public Law 94-142—and its 1990 reauthorization as IDEA—to include children with ADHD under the category of "Other Health Impaired" because of their difficulties in alertness and attention. Upon this reinterpretation, children with ADHD could now be considered eligible for special educational services, provided that the ADHD resulted in significant impairment in academic performance. Such efforts to obtain special educational resources for children and adolescents with ADHD stemmed from their tremendous risk for academic underachievement, failure, retention, suspension, and expulsion, not to mention negative social and occupational outcomes (Barkley, Fischer, et al., 1990, 1991; Cantwell & Satterfield, 1978; Weiss & Hechtman, 1986).

The Church of Scientology Campaign

Yet with this increased public activism also came a tremendously destructive trend in the United States, primarily fueled by the Church of Scientology and its Citizens Commission on Human Rights (CCHR). This campaign capitalized on the mass media's general tendency to publish alarming or sensational anecdotes uncritically, as well as the public's gullibility for such anecdotes. Drawing on evidence of an increase in stimulant medication use with schoolchildren, as well as the extant public concern over drug abuse, CCHR members effectively linked these events together to play on the public's general concern about using behavior-modifying drugs with children. In a campaign reminiscent of the gross overstatement seen in the earlier "Reefer Madness" campaign by the U.S. government against marijuana, members of CCHR selectively focused on the rare cases of adverse reactions to stimulants and greatly exaggerated both their number and degree to persuade the public that these reactions were commonplace. They also argued that massive overprescribing was posing a serious threat to schoolchildren, though actual evidence of such overprescribing was never presented. By picketing scientific and public conferences on ADHD, actively distributing leaflets to parents and students in many North American cities, seeking out appearances on many national television talk shows, and placing numerous letters to newspapers decrying the evils of Ritalin and the myth of ADHD (Bass, 1988; CCHR, 1987; Cowart, 1988; Dockx, 1988), CCHR members and others took this propaganda directly to the public. Ritalin, they claimed, was a dangerous and addictive drug often used by intolerant educators and parents and by money-hungry psychiatrists as a chemical straitjacket to subdue normally exuberant children (Clark, 1988; CCHR, 1987; Dockx, 1988). Dramatic, exaggerated, or unfounded claims were made that Ritalin use could frequently result in violence or murder, suicide, Tourette syndrome, permanent brain damage or emotional disturbance, seizures, high blood pressure, confusion, agitation, and depression (CCHR, 1987; Clark, 1988; Dockx, 1988; Laccetti, 1988; "Ritalin Linked," 1988; Toufexis, 1989; Williams, 1988). They also claimed that the increasing production and prescription of Ritalin were leading to increased abuse of such drugs by the general public (Associated Press, 1988; Cowart, 1988; "Rise in Ritalin Use," 1987). Great controversy was said to exist among the scientific and professional practice communities relative to this disorder and the use of medication. No evidence presented in these articles, however, demonstrated a rise in Ritalin abuse or linked it with the increased prescribing of the medication. Moreover, close inspection of professional journals and conferences revealed that no major or widespread controversy

ever existed within the professional or scientific fields over the nature of the disorder or the effectiveness of stimulant medication. Yet lawsuits were threatened, initiated, or assisted by the CCHR against practitioners for medical negligence and malpractice, and against schools for complicity in "pressuring" parents to have their children placed on these medicines (Bass, 1988; Cowart, 1988; Henig, 1988; Twyman, 1988; see also the 1988 segment on ABC's *Nightline*). A major lawsuit ($125 million) was also filed by the CCHR against the American Psychiatric Association for fraud in developing the criteria for ADHD (Henig, 1988; "Psychiatrist Sued," 1987), though the suit would later be dismissed.

So effective was this national campaign by the CCHR, so widespread were newspaper and television stories on adverse Ritalin reactions, and so easily could public sentiment be misled about a disorder and its treatment by a fringe political–religious group and overzealous, scandal-mongering journalists that within 1 year the public attitude toward Ritalin was dramatically altered. Ritalin was seen as a dangerous and overprescribed drug, and the public believed that there was tremendous professional controversy over its use. The minor benefits to come out of this distorted reporting were that some practitioners would become more rigorous in their assessments and more cautious in prescribing medication. Schools also became highly sensitized to the percentage of their enrollment receiving stimulant medication, and in some cases encouraged exploration of alternative behavioral means of managing children.

Yet even the few modestly positive effects of this campaign were greatly outweighed by the damaging effects on parents and children. Many parents were scared into unilaterally discontinuing the medication for their children without consulting their treating physicians. Others rigidly refused to consider the treatment, if recommended, as one part of their child's treatment plan or were harassed into such refusal by well-meaning relatives misled by the distorted church propaganda and media reports. Some adolescents with ADHD began refusing the treatment, even if it had been beneficial to them, after being alarmed by these stories. Some physicians stopped prescribing the medications altogether out of concern for the threats of litigation, thereby depriving many children in their care of the clear benefits of this treatment approach. Most frustrating to watch was the unnecessary anguish created for parents whose children were already on the medication or who were contemplating its use. The psychological damage done to those children whose lives could have been improved by this treatment was incalculable. The meager, poorly organized, and sporadically disseminated response of the mental health professions was primarily defensive in nature (Weiner, 1988) and (as usual) too little, too late to change the tide of public opinion. It would take years even to partially reverse this regression in public opinion toward ADHD and its treatment by medication, and the chilling effect all this had on physicians' prescribing of the medication. Public suspicion and concern over medication use for ADHD remains even today.

The Prevailing View at the End of the 1980s

This decade closed with the professional view of ADHD as a developmentally disabling condition with a generally chronic nature, a strong biological or hereditary predisposition, and a significant negative impact on academic and social outcomes for many children. However, its severity, comorbidity, and outcome were viewed as significantly affected by environmental (particularly familial) factors. Growing doubts about the central role of attention deficits in the disorder arose late in the decade, while increasing interest focused on possible motivational factors or reinforcement mechanisms as the core difficulty in ADHD. Effective treatment was now viewed as requiring multiple methods and professional disciplines working in concert over longer time intervals, with periodic reintervention as required, to improve the long-term prognosis for ADHD. The view that environmental causes were involved in the genesis of the disorder was weakened by increasing evidence for the heritability of the condition and its neuroanatomical localization. Even so, evidence that familial–environmental factors were associated with outcome was further strengthened. Developments in treatment would expand the focus of interventions to parental disturbances and family dysfunction, as well as to the children's anger control and social skills. A potentially effective role for the use of tricyclic antidepressants and antihypertensive medications was also demonstrated, expanding the armamentarium of symptomatic interventions for helping children with ADHD.

Despite these tremendous developments in the scientific and professional fields, the general public became overly sensitized to and excessively alarmed by the increasing use of stimulant medication as a treatment for this disorder. Fortunately, the explosive growth of parent support–political action associations

for ADHD arose almost simultaneously with this public controversy over Ritalin and held the promise of partially counteracting its effects and making the education of children with ADHD a national political priority at the start of the 1990s. These associations also offered the best hope that the general public could be provided with a more accurate depiction of ADHD and its treatment. Perhaps now the public could be made to understand that hyperactive, disruptive child behaviors could arise out of a biologically based disability that could be diminished or amplified by the social environment, rather than being entirely due to bad parenting and diet, as the simplistic yet pervasive societal view maintained.

THE PERIOD 1990 TO 1999

During the 1990s, a number of noteworthy developments occurred in the history of ADHD, chief among them being the increase in research on the neurological and genetic basis of the disorder and on ADHD as it occurs in clinic-referred adults.

Neuroimaging Research

Researchers had long suspected that ADHD was associated in some way with abnormalities or developmental delays in brain functioning. Supporting such an interpretation in the 1990s were numerous neuropsychological studies showing deficits in performance by children with ADHD on tests that were presumed to assess frontal lobe or executive functions (for reviews, see Barkley, 1997a; Barkley et al., 1992; Goodyear & Hynd, 1992). Moreover, psychophysiological research in earlier decades had suggested brain underactivity, particularly in functioning related to the frontal lobes (Hastings & Barkley, 1978; Klorman, 1992). Thus, there was good reason to suspect that delayed or disturbed functioning in the brain, and particularly the frontal lobes, might be involved in this disorder.

In 1990, Alan Zametkin and his colleagues at the National Institute of Mental Health (NIMH) published a landmark study. They evaluated brain metabolic activity in 25 adults with ADHD who had both a childhood history of the disorder and children with the disorder. The authors used positron emission tomography (PET), an exceptionally sensitive technique for detecting states of brain activity and its localization within the cerebral hemispheres. The results of this study indicated significantly reduced brain metabolic activity in adults with ADHD relative to a control group, primarily in frontal and striatal regions. Such results were certainly consistent in many, though not all, respects with the earlier demonstrations of reduced cerebral blood flow in the frontal and striatal regions in children with ADHD (Lou et al., 1984, 1989). Significant in the Zametkin and colleagues (1990) study, however, was its use of a much better defined sample of patients with ADHD and its focus on adults with ADHD. Although later attempts by this research team to replicate their original results with teenagers were consistent with these initial results for girls with ADHD, no differences were found in boys with ADHD (see Ernst, 1996, for a review). Sample sizes in these studies were quite small, however, almost ensuring some difficulties with the reliable demonstration of the original findings. Despite these difficulties, the original report stands out as one of the clearest demonstrations to date of reduced brain activity, particularly in the frontal regions, in ADHD.

At the same time as the NIMH research using PET scans appeared, other researchers were employing magnetic resonance imaging (MRI) to evaluate brain structures in children with ADHD. Hynd, Semrud-Clikeman, Lorys, Novey, and Eliopulos (1990) were the first to use this method, and they focused on total brain volume, as well as specific regions in the anterior and posterior brain sections. Children with ADHD were found to have abnormally smaller anterior cortical regions, especially on the right side, and they lacked the normal right–left frontal asymmetry. In subsequent research that focused on the size of the corpus callosum, this team found that both the anterior and posterior portions were smaller in children with ADHD (Hynd et al., 1991); however, in a later study, only the posterior region was found to be significantly smaller (Semrud-Clikeman et al., 1994). Additional studies were reported by Hynd and colleagues (1993), who found a smaller left caudate region in children with ADHD, and Giedd and colleagues (1994), who found smaller anterior regions of the corpus callosum (rostrum and rostral body).

More recently, two research teams published MRI studies with considerably larger samples of children with ADHD (Castellanos et al., 1994, 1996; Filipek et al., 1997). These studies documented significantly smaller right prefrontal lobe and striatal regions in these children. Castellanos and colleagues (1996) also found smaller right-sided regions of structures in the basal ganglia, such as the striatum, as well as the right

cerebellum. Filipek and colleagues (1997) observed the left striatal region to be smaller than the right. Despite some inconsistencies across these studies, most have implicated the prefrontal–striatal network as being smaller in children with ADHD, with the right prefrontal region being smaller than the left. Such studies have placed on a considerably firmer foundation the view that ADHD does indeed involve impairments in the development of the brain, particularly in the prefrontal–striatal regions, and that these impairments are likely to have originated in embryological development (Castellanos et al., 1996). Advances in neuroimaging technology continue to provide exciting and revealing new developments in the search for the structural differences in the brain that underlie this disorder (see Chapter 14). For instance, the advent of functional MRI (fMRI), with its greater sensitivity for localization of activity, has already resulted in a number of newly initiated investigations into possible impairments in these brain regions in children and adults with ADHD.

Genetic Research

Since the 1970s, studies have indicated that the parents of children with hyperactivity, ADD, or ADHD seem to have a greater frequency of psychiatric disorders, including ADHD. Cantwell (1975) and Morrison and Stewart (1973) both reported higher rates of hyperactivity in the biological parents of hyperactive children than in adoptive parents of such children. Yet both studies were retrospective, and both failed to study the biological parents of the adopted hyperactive children as a comparison group (Pauls, 1991). In the 1990s, a number of studies, particularly those by Biederman and colleagues, clarified and strengthened this evidence of the familial nature of ADHD. Between 10 and 35% of the immediate family members of children with ADHD were found to have the disorder; the risk to siblings of these children was approximately 32% (Biederman, Faraone, & Lapey, 1992; Biederman, Keenan, & Faraone, 1990; Pauls, 1991; Welner, Welner, Stewart, Palkes, & Wish, 1977). Even more striking, research has shown that if a parent has ADHD, the risk to the offspring is 57% (Biederman et al., 1995). Thus, family aggregation studies reveal that ADHD clusters among biological relatives of children or adults with the disorder, strongly implying a hereditary basis to this condition.

At the same time that these studies were appearing, several studies of twins were focusing on the heritability of the dimensions of behavior underlying ADHD (i.e., hyperactive–impulsive and inattentive) behavior, or on the clinical diagnosis of ADHD itself. Large-scale twin studies on this issue have quite consistently found a high heritability for ADHD symptoms or for the clinical diagnosis, with minimal or no contribution made by the shared environment (Edelbrock, Rende, Plomin, & Thompson, 1995; Levy & Hay, 1992). For instance, Gilger, Pennington, and DeFries (1992) found that if one twin was diagnosed with ADHD, the concordance for the disorder was 81% in monozygotic twins and 29% in dizygotic twins. Stevenson (1994) summarized the status of twin studies on symptoms of ADHD by stating that the average heritability is .80 for symptoms of this disorder (range .50–.98). More recent large-scale twin studies are remarkably consistent with this conclusion, demonstrating that the majority of variance (70–90%) in the trait of hyperactivity–impulsivity is due to genetic factors (averaging approximately 80%), and that such a genetic contribution may increase as scores for this trait become more extreme, although this latter point is debatable (Faraone, 1996; Gjone, Stevenson, & Sundet, 1996; Gjone, Stevenson, Sundet, & Eilertsen, 1996; Rhee, Waldman, Hay, & Levy, 1999; Silberg et al., 1996; Thapar, Hervas, & McGuffin, 1995; van den Oord, Verhulst, & Boomsma, 1996). Thus, twin studies added substantially more evidence to that already found in family aggregation studies supporting a strong genetic basis to ADHD and its behavioral symptoms. More recent twin studies have further buttressed the strong genetic contribution to ADHD (see Chapter 14). Equally important is the consistent evidence in such research that whatever environmental contributions may be made to ADHD symptoms fall more within the realm of unique (nonshared) environmental effects than within that of common or shared effects.

Also in this decade, a few researchers began using molecular genetic techniques to analyze DNA taken from children with ADHD and their family members to identify genes that may be associated with the disorder. The initial focus of this research was on the dopamine type 2 gene, given findings of its increased association with alcoholism, Tourette syndrome, and ADHD (Blum, Cull, Braverman, & Comings, 1996; Comings et al., 1991), but others failed to replicate this finding (Gelernter et al., 1991; Kelsoe et al., 1989). More recently, the dopamine transporter gene was implicated in ADHD (Cook et al., 1995; Cook, Stein, & Leventhal, 1997). Another gene related to dopamine,

the *D4RD* (repeater gene) was found to be overrepresented in the seven-repetition form of the gene in children with ADHD (LaHoste et al., 1996). The latter finding has been replicated in a number of additional studies and indicates that the presence of this allele increases the risk for ADHD by 1.5. Clearly, research into the molecular genetics involved in the transmission of ADHD across generations continues to be an exciting and fruitful area of research endeavor. Such research offers promise for the eventual development of not only genetic tests for ADHD and subtyping of ADHD into potentially more homogeneous and useful genotypes but also more specific pharmacological agents for treating ADHD.

ADHD in Adults

Although articles dealing with the adult equivalents of childhood hyperactivity or MBD date back to the late 1960s and 1970s (discussed earlier), they did not initiate widespread acceptance of these adult equivalents in the field of adult psychiatry and clinical psychology. It was not until the 1990s that the professional fields and the general public recognized ADHD in adults as a legitimate disorder. This was due in large part to a best-selling book by Edward Hallowell and John Ratey (1994), *Driven to Distraction*, which brought the disorder to the public's attention. More serious and more rigorous scientific research was also conducted on adults with ADHD across this decade. In addition, the greater clinical professional community began to consider the disorder a legitimate clinical condition worthy of differential diagnosis and treatment (Goldstein, 1997; Nadeau, 1995; Wender, 1995).

This broadening acceptance of ADHD in adults continues to the present time and is likely to increase further in the decades ahead. It seems to have been strengthened in some part throughout the 1990s by the repeated publication of follow-up studies that documented the persistence of the disorder into adolescence in up to 70% of cases, and into adulthood in up to as many as 66% of childhood cases (Barkley, Fischer, et al., 1990, 2002; Mannuzza, Gittelman-Klein, Bessler, Malloy, & LaPadula, 1993; Weiss & Hechtman, 1993). And it can be attributed as well to published studies on clinically referred adults diagnosed with the disorder (Biederman et al., 1993; Murphy & Barkley, 1996; Shekim, Asarnow, Hess, Zaucha, & Wheeler, 1990; Spencer, Biederman, Wilens, & Faraone, 1994). But it is probably in part a result of pressure from the general public, which was made more cognizant of this disorder in adults through various media, including the publication of other best-selling, popular books on the subject (Kelly & Ramundo, 1992; Murphy & LeVert, 1994; Weiss, 1992); numerous media accounts of the condition in adults; the efforts of large-scale parent support groups discussed earlier (e.g., CHADD) to promote greater public awareness of this issue; and the advent of Internet chat rooms, Web pages, and bulletin boards devoted to this topic (Gordon, 1997). Adults who obtain such information and seek out evaluation and treatment for their condition are simply not satisfied any longer with outdated opinions of some adult mental health specialists that the disorder does not exist in adults and is commonly outgrown by adolescence, a belief that was widespread in the 1960s.

Also notable in the 1990s was the publication of more rigorous studies demonstrating the efficacy of stimulants (Spencer et al., 1995) and antidepressants (Wilens et al., 1996) in the management of adult ADHD. Such studies confirmed the initial clinical speculations in the 1970s, as well as the conclusions from earlier, smaller studies by Paul Wender and his colleagues in the 1970s and 1980s (described earlier), that such medications were efficacious for this disorder in adults (Wender, Reimherr, & Wood, 1981; Wender, Reimherr, Wood, & Ward, 1985). Thus, the adult form of ADHD was found not only to share many patterns of symptoms and comorbid disorders with the childhood form, but also to respond just as well to the same medications that proved so useful in the management of childhood ADHD (see Chapter 35).

Other Developments of the Era

The 1990s were marked by other significant developments in the field of ADHD. In 1994, new diagnostic criteria for the disorder set forth in DSM-IV (American Psychiatric Association, 1994) included several improvements over those in the earlier DSM-III-R. But suffice it to say here that they reintroduced criteria for the diagnosis of a purely inattentive form of ADHD, similar to ADD – H in DSM-III. The diagnostic criteria also now require evidence of symptoms' pervasiveness across settings, as well as the demonstration of impairment in a major domain of life functioning (home, school, work). Based on a much larger field trial than any of their predecessors, DSM-IV contained the most empirically based criteria for ADHD in the history of this disorder (see Chapter 2).

A further development during this decade was the NIMH multisite study of ADHD that focused on various combinations of long-term treatments (Arnold et al., 1997; MTA Cooperative Group, 1999; see Chapter 28). This study (the Multimodal Treatment Study of ADHD [MTA]) determined what combinations of treatments were most effective for what subgroups of ADHD, based on those treatment strategies with the greatest empirical support in the prior treatment literature. Another long-term treatment study reported findings of great significance to the field: The Swedish government commissioned the longest treatment study of stimulant medication ever undertaken, the results of which indicated that amphetamine treatment remained effective for the entire 15 months of the investigation (see Gillberg et al., 1997). More sobering was the report that an intensive, yearlong treatment program using primarily CBT strategies produced no substantial treatment effects either at posttreatment or at follow-up (Braswell et al., 1997). Similarly, a yearlong, intensive early intervention program for hyperactive–aggressive children found no significant impact of parent training either at posttreatment or at 2-year follow-up (Barkley et al., 2000; Barkley, Fischer, et al., 2002); the school-based portion of this multimethod program produced some immediate treatment gains, but by 2-year follow-up, these had dissipated (Shelton et al., 2000). Finally, a multisite study of stimulant medication with and without intensive behavioral and psychosocial interventions revealed that the psychosocial interventions added little or nothing to treatment outcome beyond that achieved by stimulant medication alone (Abikoff & Hechtman, 1995). Its final results, not reported until 2004 (see Chapter 28), were in keeping with the findings of the MTA that the combined treatments were generally no substantially better than medication treatment alone. Although these studies do not entirely undermine earlier studies on the effectiveness of behavioral interventions for children with ADHD, they do suggest that some of those interventions produce minimal or no improvement when used on a large-scale basis; that the extent of improvement is difficult to detect when adjunctive stimulant medication is also used; and that treatment effects may not be maintained over time following treatment termination.

The 1990s also witnessed the emergence of trends that would be developed further over the next decade. These trends included a renewed interest in theory development related to ADHD (Barkley, 1997a, 1997c; Quay, 1988a, 1997; Sergeant & van der Meere, 1994), as well as an expanding recognition and treatment of the disorder in countries outside the United States and Canada (Fonseca et al., 1995; Shalev, Hartman, Stavsky, & Sergeant, 1995; Toone & van der Linden, 1997; Vermeersch & Fombonne, 1995). A new stimulant combination, Adderall, which appeared on the market in this decade, showed promise as being as effective for ADHD as the other stimulants (Swanson et al., 1998), and at least three new nonstimulant medications and an additional stimulant were in development or in Phase II clinical trials by several pharmaceutical companies during this decade. There also appeared to be an increasing interest in the use of peers as treatment agents in several new behavioral intervention programs for academic performance and peer conflict in school settings (DuPaul & Henningson, 1993; see Chapters 23 and 24).

The Prevailing View at the End of the 1990s

It seems clear that during the 1990s there was a shift back to viewing ADHD as far more influenced by neurological and genetic factors than by social or environmental ones. Clearly, the interaction of these sources of influence is generally well accepted by professionals at this time, but greater emphasis is now being placed on the former than on the latter in understanding the potential causation of the disorder. Moreover, evidence began accruing that the influence of the environment on the symptoms of the disorder fall chiefly in the realm of unique or nonshared factors rather than among the more frequently considered but now weakly supported common or shared family factors.

Over this decade, there was also a discernible shift toward the recognition that a deficit in behavioral inhibition may be the characteristic that most clearly distinguishes ADHD from other mental and developmental disorders (Barkley, 1997a; Nigg, 2001; Pennington & Ozonoff, 1996; Schachar, Tannock, & Logan, 1993), and that this deficit is associated with a significant disruption in the development of typical self-regulation. It is also noteworthy that the subtype of ADHD comprising chiefly inattention without hyperactive–impulsive behavior may possibly be a qualitatively distinct disorder from the subtype with hyperactive–impulsive behavior or the subtype with combined behavior (Barkley et al., 1992; Goodyear & Hynd, 1992; Lahey & Carlson, 1992). The issue of comorbidity became increasingly important in subgrouping children with ADHD, leading to greater understanding of the way disorders

that coexist with ADHD may influence family functioning, academic success, developmental course and outcome, and even treatment response. In contrast to the attitudes apparent in the middle of the 20th century, the view of ADHD at the close of the century was a less developmentally benign one, owing in large part to multiple follow-up studies that documented the pervasiveness of difficulties with adaptive functioning in the adult lives of many (though by no means all) persons clinically diagnosed with ADHD in childhood.

There is little doubt that the use of pharmacology in the management of the disorder continued its dramatic rise in popularity, owing in no small part to repeated demonstration of the efficacy of stimulants in the treatment of the disorder; the greater recognition of subtypes of ADHD, as well as girls and adults with ADHD; and the rather sobering results of multimethod intensive psychosocial intervention programs. Even so, combinations of medication with psychosocial and educational treatment programs remained the norm in recommendations for the management of the disorder across the 1990s, much as they were in the 1980s.

Across this decade, the expansion, solidification, and increased political activity and power of the patient and family support organizations, such as CHADD, were indeed a marvel to behold. They clearly led to far wider public recognition of the disorder, as well as to controversies over its existence, definition, and treatment with stimulant medications; still, the general trend toward greater public acceptance of ADHD as a developmental disability remained a largely optimistic one. Moreover, such political activity resulted in increased eligibility of those with ADHD for entitlements, under the IDEA, and legal protections, under the Americans with Disabilities Act of 1990 (Public Law 101-336).

THE NEW CENTURY: 2000 TO THE PRESENT

A number of developments arising in this period are covered in detail throughout the remaining chapters in this volume, so they receive only brief topical mention here because of their importance to the history of the disorder. For instance, the recently published DSM-5 diagnostic manual contains a few important adjustments to criteria for the clinical diagnosis of ADHD (American Psychiatric Association, 2013), as discussed in the next chapter. Those adjustments, and others that were recommended but not included, have spawned recent controversy that is also addressed in that chapter.

Trends from the 1990s have certainly continued into the 21st century, with far more published research on heredity, molecular genetics, and neuroimaging, along with some initial efforts to link these fields together. The result has been an explosion in the size of the ADHD literature, which has nearly doubled in 2013 alone, along with the publication of numerous meta-analyses of various segments of it, as referenced in various chapters in this volume. Not only has the hereditary basis of ADHD become firmly established by hundreds of recent studies, but numerous candidate genes for the disorder are also being identified, and new chromosomal regions deserving of greater investigation via scans of the entire human genome that involve hundreds, and soon thousands, of affected families. This area of research has revealed that not only are genes involved in the regulation of dopamine and norepinephrine networks in the brain involved in ADHD but also other, far evolutionary older genes involved in brain cell growth, endpoint termination, and neuronal sprouting may also be implicated (see Chapter 14).

Although no entirely new theories of ADHD have been proposed, existing theories have been expanded and clarified (Barkley, 2012b). There have also been tremendous advances in establishing the underlying neurological nature and mechanisms involved in ADHD in the field of neuroimaging, along with findings from developmental (longitudinal) neuroimaging studies documenting the delayed brain growth and altered growth trajectories associated with the disorder (see Chapter 14). There continues to be abundant research on the neuropsychology of ADHD and potential endophenotypes for use in genetic and neuroimaging investigations, discussed in several chapters in this volume.

Research efforts at subtyping ADHD have also increased since 2000 (see Chapters 2 and 17; also see Milich, Balentine, & Lynam, 2001). This research seems to suggest that the prior DSM-IV subtyping approach to ADHD has not proven useful, and that the disorder is likely to be a single condition varying in severity within the population while having two highly intercorrelated yet partially distinct symptom dimensions. Yet other research is leading to the possibility that perhaps a new attention disorder has been unearthed (Barkley, 2012a, 2012b, 2013). Known as SCT, this subset may have accounted for approximately 30–50% of those children previously placed in the DSM-IV predominantly inattentive type of ADHD, what clinicians began calling ADD. It is characterized

by a cognitive sluggishness and social passivity, in sharp contrast to the distractible, impulsive, overactive, and emotional difficulties so characteristic of the combined type of the disorder. Because of its derogatory and potentially offensive name, and the implication that the underlying cognitive dysfunction is known, I have suggested that the term SCT be renamed "concentration deficit disorder" (CDD; Barkley, 2014).

Although ADHD is a single and dimensional disorder in the human population, advances in molecular genetics may offer the possibility of genetically subtyping samples of individuals with ADHD into those who do and do not possess a particular candidate allele, so as to study over time the impact of the allele on the psychological and social phenotype of the disorder, and its developmental course and risk for future impairments. Such longitudinal studies are now under way.

Further work has also examined those disorders likely to be comorbid with ADHD in both children (see Angold, Costello, & Erkanli, 1999) and adults (Barkley, Murphy, & Fischer, 2008), and the impact they may have on risk for impairments, life course, and even treatment response in ADHD. It now appears, for instance, that the overlap of ADHD with the learning disorders (reading, spelling, math) may stem from both separate, distinct etiologies that arise together in particular cases and some small shared genetic contribution to both disorders, in contrast to the earlier, more simplistic view that one type of disorder may be causing the other. For now, existing evidence suggests that although the two sets of disorders are genetically linked to each other to a small extent, they also have a greater proportion of unshared etiologies. ADHD, however, may be a direct contributor to a progressive increase in problems with reading (and even story and video) comprehension, perhaps through its detrimental effects on working memory. The case for major depressive disorder gives us fairly substantial evidence that ADHD may create a genetic susceptibility to this disorder, albeit one that may require exposure to stress, social disruption, or traumatic events to become fully manifest. And the link of ADHD to ODD and CD, as well as later substance use and antisocial activity, continues to be supported by ongoing research.

The domain of treatment has seen several advances, not the least of which has been the continued reporting of results from the MTA (see Chapter 28), although there is controversy about how initial and especially follow-up results should be interpreted. No one doubts that this monumental study indicated that medication treatment is superior to psychosocial treatment or community care as usual in the initial results. Continuing disagreement appears to concern whether the combination of medication and psychosocial components resulted in important benefits that were not as evident in the medication-only condition. Although many professionals continue to adhere to the view that many cases require combined therapy and that it offers advantages for especially comorbid cases, some certainly concede the point that some cases may do sufficiently well on medications and require little additional psychosocial care.

Another advance in treatment was the development of sustained-release delivery systems for the previously extant stimulant medications (see Chapter 27). These new delivery systems are chemical engineering marvels (sustained-release pellets, osmotic pumps, skin patches, prodrugs, etc.); within the few years of their initial introduction to the marketplace, these extended-release formulations have become the standard of care for medication management, at least in the United States. Such delivery systems allow single doses of medication to manage ADHD symptoms effectively for periods of 8–12 hours. This has eliminated the need for school dosing and its numerous associated problems, not the least of which is stigmatization of children who required midday doses.

This decade also saw U.S. Food and Drug Administration (FDA) approval of two new nonstimulants for treating ADHD. The first of these new medications was the norepinephrine reuptake inhibitor, atomoxetine (Strattera). First approved for use in the United States in January 2003 by the FDA, atomoxetine was the first drug approved for management of ADHD in adults, along with use in children and teens. Over the next several years, the drug received approval for use in numerous other countries and is now prescribed for more than 4 million individuals worldwide. Attractive to many is the fact that this medication has no abuse potential and is therefore not a scheduled drug in the United States, which makes it far easier to prescribe than stimulants, which are Schedule II medications. As one of the most studied medications ever submitted for FDA approval for a neuroscience indication, atomoxetine has become the second-choice medication behind stimulants for management of ADHD in many professional association guidelines for ADHD management.

The second nonstimulant approved by the FDA in the United States was guanfacine XR (Intuniv) in

2009. For more on this medication, see Chapter 27. Guanfacine is an alpha$_2$ agonist that was originally used in the treatment of hypertension. A similar drug, clonidine, had been investigated for use in ADHD over the past 30 years with some success and was even used clinically off-label by some physicians in efforts to better manage the impulsive, hyperactive, and emotionally excitable aspects of ADHD apart from any benefits relative to attention. Some even combined clonidine with stimulants in an attempt to gain greater coverage of ADHD or some symptoms of comorbid disorders associated with anger or other emotion dysregulation. But its significant risks for cardiotoxicity limited its adoption on a more widespread basis, along with its lack of FDA approval for the management of ADHD. In contrast, guanfacine has been shown to present less risk for adverse cardiotoxic events. By reformulating the medication into an extended-release delivery system, guanfacine XR can be taken just once daily, providing treatment coverage of ADHD symptoms across much of the waking day. Like atomoxetine, guanfacine XR does not appear to improve ADHD symptoms as much as the stimulants, but both nonstimulants appear to benefit approximately 75% of individuals taking either medication. They may also be first-choice drugs when ADHD co-occurs with certain other psychiatric disorders or health conditions that might preclude the use of stimulants, or when stimulants may arguably produce some exacerbation, such as anxiety or tic disorders.

Few new psychosocial treatments for ADHD have been identified in nearly a decade since publication of the previous edition of this volume. Research continues to show that various formats of behavioral parent training can help parents manage children and teens with ADHD (see Chapters 21 and 22, this volume), as can training teachers in various behavior management strategies (see Chapter 24). But exciting developments in the alteration or combination of existing treatments may make them more effective for managing various impairments in ADHD. For instance, new CBTs for adults that focus on the executive function deficits that are so impairing in ADHD have been developed and evaluated in randomized trials with considerable success (see Chapter 32). A new variation in social skills training, known as Friendship Coaching, developed by Mikami and colleagues (Chapter 23, this volume) may offer a successful intervention for the social problems of children with ADHD. Prior studies have suggested that social skills training, at least as traditionally delivered in clinics by professionals, has not been especially effective. Mikami's approach uses parents as therapists (friendship coaches) to deliver the appropriate methods throughout the natural stream of social interactions with children in the natural social ecology. Initial promise was evident in the development of cognitive rehabilitation training programs relying on computer software game technology, such as those for working memory training. Yet subsequent efforts to replicate these initially promising findings have shown more limited, if any, positive effects (see Chapter 26). And controversy continues to surround the issue of the effectiveness of EEG biofeedback training (neurofeedback) developed more than 20 years ago (as discussed briefly in Chapter 11), with less rigorous studies showing clinical benefits, while more rigorous ones are less beneficial, if at all.

The international recognition of ADHD has grown sharply since 2000, owing in part to the emergence and expansion of parent support groups in many countries; the dramatic increase in research articles on ADHD in journals, especially from developing or non-Western countries; and the emergence of foreign professional societies dedicated to ADHD, such as Eunythydis in Europe and the World Federation for ADHD, both of which hold annual meetings that comprise numerous presentations on topics related to ADHD. Certainly, educational and advertising efforts by the pharmaceutical industry associated with the increasing number of countries approving the use of these medications has also contributed to greater international recognition of the disorder. But substantial credit must also be given to the increasing access people have to the Internet and the increasing amount of information on ADHD existing there. The Internet allows anyone with a computer, iPad, or smartphone to have nearly instantaneous access to websites such as those sponsored by CHADD (*www.chadd.org*) and ADDA (*www.add.org*) in the United States, the Center for ADD Awareness of Canada (*www.caddac.ca*) and its partner, the Canadian ADHD Resource Alliance (*www.caddra.ca*), among others. It also permits access to numerous videos on YouTube and similar forums.

There was a time when each country had its own view of mental disorders, their causes, and their management. Hence, the United States might view ADHD one way, Sweden in another, and Italy, France, Germany, or Spain might each view it in a different way. Such walls between different countries' understand-

ings of ADHD have now figuratively come crashing down, with the democratizing spread of information via the Internet and the scientific (and nonscientific!) information it can bring to any user. This means that there is no longer an Italian view of ADHD or a U.S. view, but an international view, founded on the most recent scientific advances as they become available on the Internet. Professionals, for instance, who may still practice a psychoanalytic view of childhood disorders as arising from early upbringing, can no longer count on this view going unchallenged by parents of children or adults with ADHD in their practices. These patients and families can readily discover on the Internet that such views have no scientific credibility; that long-term, analytically focused psychotherapy is not effective for ADHD; and that medications and more empirically based psychosocial accommodations are the cutting edge treatments. If they cannot obtain them in their country, they can quickly locate a neighboring one that is better informed and where such therapies may be accessible. We should expect to see more such developments on the international scene in the coming years. But as a consequence, we also continue to expect the same sort of media sensationalism and misrepresentation, baseless social criticism, and even Scientology's propaganda efforts periodically to erupt alongside this expanding international recognition of ADHD as a legitimate mental health and public health disorder.

ADHD has undoubtedly become a valid disorder and frequent topic of scientific study, widely accepted throughout the mental health and medical professions as a legitimate neurodevelopmental disability. At this time, it is unmistakably one of the most well-studied childhood disorders. That it is also the object of healthy, sustained research initiatives into its adult counterparts has led to far greater acceptance of adult ADHD than what occurred two decades earlier for the childhood version of the disorder. Further discoveries concerning the nature, causes, and developmental course of ADHD promise tremendous advances in our insight into not only this disorder but also the very nature and development of human self-regulation more generally, and its rather substantial neurological, genetic, and unique environmental underpinnings. Along with these advances will undoubtedly come new treatments and their combinations. These, let us hope, will greatly limit the impairments experienced by many who suffer from ADHD across their lifespan.

KEY CLINICAL POINTS

- ✓ ADHD has a long and exceptionally rich history of clinical and scientific publications, more than 10,000 since the initial descriptions of clinical patients by Weikard in 1775.
- ✓ Early conceptualizations of ADHD focused on inattention, impulsive behavior, and excessive activity, as well as defective moral control of behavior. Proponents of these views recognized that ADHD-like behavior could arise from brain injuries yet might also develop from flawed social environments. Later views emphasized ADHD's association with brain damage, particularly to the frontal lobes, followed by an emphasis on brain dysfunction, then hyperactivity. Current views of the etiologies of ADHD now emphasize its neurodevelopmental nature and the prominent roles played by genetic, as well as nongenetic, neurological factors.
- ✓ Advances in developing diagnostic criteria have resulted in more precise specification of symptoms, along with two symptom lists; an emphasis on childhood or early-adolescent onset of the disorder in most cases; and a requirement for both cross-setting pervasiveness of symptoms and evidence of impairment in one or more major life activities.
- ✓ More recent theories of ADHD have viewed deficits in self-regulation as central to the disorder, while also suggesting that deficits in executive functioning and biologically based motivational difficulties that undergird self-regulation are likely to account for most or all of the symptoms associated with the disorder.
- ✓ Efforts at subtyping ADHD, such as in the DSM-IV, did not prove successful. But a subset of inattentive children manifesting SCT or CDD, along with social passivity and other distinguishing clinical features, may yet come to be recognized as a second attention disorder that is distinct from yet partially overlaps ADHD.
- ✓ Research using neuroimaging techniques has served to isolate particular brain regions (especially the frontal–striatal–cerebellar network, and possibly other regions) as underlying the disorder, and particularly as involved in the difficulties with inhibition and executive functioning.
- ✓ Increasing research on heredity and genetics has clearly shown a striking hereditary basis to ADHD, along with the identification of numerous candidate

genes or chromosomal locations that hold some promise in explaining the disorder.

- ✓ Research into the neuropsychology of ADHD has also increased substantially in the past decade; it supports the view that ADHD is not only an inhibitory disorder but also one associated with deficits in the major executive functions that underlie self-regulation.
- ✓ Further research, especially on prenatal neurological hazards and postnatal injuries and environmental toxins, suggests that some cases of ADHD may arise from brain injury rather than, or in interaction with, genetic mechanisms.
- ✓ Numerous longitudinal studies now support the conclusion that ADHD is a relatively chronic disorder affecting many domains of major life activities from childhood through adolescence and into adulthood.
- ✓ Within the past decade, new medications and new delivery systems for older medications have been developed that both broaden the range of treatment options for managing the heterogeneity of clinical cases and sustain medication effects for longer periods across the day (with less need for in-school dosing).
- ✓ Advances in psychosocial treatment research have revealed specific subsets of individuals with ADHD who may be more or less likely to benefit from these empirically proven interventions. They have also revealed the limitations of these approaches for generalization and maintenance of treatment effects if they are not specifically programmed into the treatment protocol.
- ✓ ADHD is now recognized as a universal disorder, with an ever-growing international acceptance of both its existence and its status as a chronic disabling condition, for which combinations of medications and psychosocial treatments and accommodations may offer the most effective approach to management.

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CHAPTER 2

Primary Symptoms, Diagnostic Criteria, Subtyping, and Prevalence of ADHD

Walter Roberts, Richard Milich, and Russell A. Barkley

Attention-deficit/hyperactivity disorder (ADHD) is a common and well-recognized behavior disorder that affects millions of children, adolescents, and adults. A tremendous amount of research has been published on the primary characteristics and symptomatology of children and adults with ADHD. In this chapter, we briefly summarize this research to provide a concise overview of what is known about ADHD as a clinical construct. It is not our goal to review this sizable body of research critically. Instead, we have attempted to summarize information related to the phenomenology of ADHD that can help clinicians by providing the essence of what is known in this field of research to facilitate the diagnosis, assessment, and treatment of children and adults with ADHD.

The chapter begins with a discussion of the current diagnostic criteria for ADHD because these criteria define the clinical construct of ADHD. For this reason, it is important to understand the rationale behind *Diagnostic and Statistical Manual of Mental Disorders* (DSM) criteria, as well as some of the limitations of this system. We then review findings on the primary symptoms of this condition (i.e., inattention, hyperactivity–impulsivity) as they occur in children and adults, as well as sluggish cognitive tempo, a "secondary" symptom cluster that may be either a core component of at least some forms of ADHD or an entirely separate disorder that often overlaps with ADHD. What follows is a discussion of how these primary symptoms can change as a function of situational variables. We conclude the chapter with a discussion of the prevalence of ADHD.

DIAGNOSTIC CRITERIA FOR ADHD

Given the nature of our current classification system of psychopathology, it is necessary to begin our discussion of the nature of ADHD by reviewing the diagnostic criteria of the disorder. Simply put, these criteria define what is and is not ADHD. The current official diagnostic criteria for ADHD are described in the fifth edition of the DSM (DSM-5; American Psychiatric Association, 2013), which is used primarily in the United States. These criteria are similar to those in DSM-IV (American Psychiatric Association, 1994), although there are small changes that we describe throughout this chapter. Table 2.1 presents the DSM-5 criteria. DSM criteria are similar, although not identical, to

TABLE 2.1. DSM-5 Diagnostic Criteria for ADHD

A. Either (1) or (2):

(1) **Inattention:** Six (or more) of the following symptoms have persisted for at least 6 months to a degree that is inconsistent with developmental level and that negatively impacts directly on social and academic/occupational activities:

Note: For older adolescents and adults (age 17 and older), at least five symptoms are required. The symptoms are not solely a manifestation of oppositional behavior, defiance, hostility, or failure to understand task instructions.

(a) often fails to give close attention to details or makes careless mistakes in schoolwork, at work, or during other activities (e.g., overlooks or misses details, work is inaccurate)

(b) often has difficulty sustaining attention in tasks or play activities (e.g., has difficulty remaining focused during lectures, conversations, or lengthy reading)

(c) often does not seem to listen when spoken to directly (e.g., mind seems elsewhere, even in the absence of any obvious distraction)

(d) often does not follow through on instructions and fails to finish schoolwork, chores, or duties in the workplace (e.g., starts tasks but quickly loses focus and is easily sidetracked)

(e) often has difficulty organizing tasks and activities (e.g., difficulty managing sequential tasks; difficulty keeping materials and belongings in order; messy, disorganized work; has poor time management; fails to meet deadlines)

(f) often avoids, dislikes, or is reluctant to engage in tasks that require sustained mental effort (e.g., schoolwork or homework; for older adolescents and adults, preparing reports, completing forms, reviewing lengthy papers)

(g) often loses things necessary for tasks or activities (e.g., school materials, pencils, books, tools, wallets, keys, paperwork, eyeglasses, mobile telephones)

(h) is often easily distracted by extraneous stimuli (for older adolescents and adults, may include unrelated thoughts)

(i) is often forgetful in daily activities (e.g., doing chores, running errands; for older adolescents and adults, returning to class, paying bills, keeping appointments)

(2) **Hyperactivity and impulsivity:** Six (or more) of the following symptoms have persisted for at least 6 months to a degree that is inconsistent with developmental level and that negatively impacts directly on social and academic/occupational activities:

Note: For older adolescents and adults (age 17 and older), at least five symptoms are required. The symptoms are not solely a manifestation of oppositional behavior, defiance, hostility, or failure to understand task instructions.

Hyperactivity

(a) often fidgets with hands or feet or squirms in seat

(b) often leaves seat situations where remaining seated is expected (e.g., leaves his or her place in the classroom, in the office or other workplace, or in other situations that require remaining in place)

(c) often runs about or climbs excessively in situations in which it is inappropriate (**Note:** In adolescents or adults, may be limited to subjective feelings of restlessness)

(d) often unable to play or engage in leisure activities quietly

(e) is often "on the go," acting as if "driven by a motor" (e.g., is unable or uncomfortable being still for extended time, as in restaurants, meetings, etc.; may be experienced by others as being restless or difficult to keep up with)

(f) often talks excessively

Impulsivity

(g) often blurts out answers before questions have been completed (e.g., completes people's sentences; cannot wait for turn in conversation)

(h) often has difficulty awaiting turn (e.g., while waiting in line)

(i) often interrupts or intrudes on others (e.g., butts into conversations, games, or activities; may start using other people's things without asking or receiving permission; for adolescents and adults, may intrude into or take over what others are doing)

(continued)

TABLE 2.1. *(continued)*

B. Several hyperactive–impulsive or inattentive symptoms that caused impairment were present prior to age 12 years.

C. Several impairments from the symptoms are present in two or more settings (e.g., at home, school, or work; with friends or relatives; in other activities).

D. There is clear evidence that the symptoms interfere with, or reduce, the quality of social, academic, or occupational functioning.

E. The symptoms do not occur exclusively during the course of schizophrenia or another psychotic disorder and are not better accounted for by another mental disorder (e.g., mood disorder, anxiety disorder, dissociative disorder, or a personality disorder).

Specify whether:

314.01 (F90.2) combined presentation: If both Criteria A1 (inattention) and A2 (hyperactivity-impulsivity) are met for the past 6 months.

314.00 (F90.0) predominantly inattentive presentation: If Criterion A1 (inattention) is met but Criterion A2 (hyperactivity–impulsivity) is not met for the past 6 months.

314.01 (F90.1) Predominantly hyperactive–impulsive presentation: If Criterion A2 (hyperactivity–impulsivity) is met but Criterion A1 (inattention) is not met for the past 6 months.

Specify if:

In partial remission: When full criteria were met in the past, fewer than the full criteria have been met for the past 6 months, and the symptoms still result in impairment in social, academic, or occupational functioning.

Specify current severity:

Mild: Few, if any, symptoms in excess of those required to make the diagnosis are present, and symptoms result in no more than minor impairment in social or occupational functioning.

Moderate: Symptoms or functional impairment between "mild" and "severe" are present.

Severe: Many symptoms in excess of those required to make the diagnosis, or severe symptoms that are particularly severe, are present, or the symptoms result in marked impairment in social occupational functioning.

Note. From American Psychiatric Association (2013). Copyright 2013 by the American Psychiatric Association. Reprinted by permission.

the definition of the disorder in the 10th edition of the *International Classification of Diseases* (ICD-10; World Health Organization, 2008), which is used mainly in Europe.

DSM-5 criteria stipulate that people must have had their symptoms of ADHD for at least 6 months, and that these symptoms must occur to a degree that is developmentally deviant. The symptoms producing impairment must have developed by 12 years of age. In DSM-5, the number of symptoms required to meet criteria changes according to the individual's age. For children age 16 or younger, six or more of the nine items from at least one cluster of symptoms must be endorsed as developmentally inappropriate. For adolescents and adults age 17 or older, five or more symptoms from at least one of the symptom clusters must be endorsed. These symptoms must interfere with the individual's functioning in two or more settings. The presentation of ADHD to be diagnosed depends on whether criteria are met for inattention, hyperactivity–impulsivity, or both: the predominantly inattentive presentation (ADHD-PI), the predominantly hyperactive–impulsive presentation (ADHD-PHI), or the combined presentation (ADHD-C). Severity specifiers can be used to further specify a person's diagnosis (i.e., mild, moderate, or severe) based on his or her symptom profile and degree of functional impairment. For example, a child who shows six symptoms in both clusters and experiences minimal functional impairment in few settings might be diagnosed with ADHD-C mild, whereas a child with eight inattentive symptoms and two hyperactive–impulsive symptoms who shows considerable impairment across most settings might be diagnosed with ADHD-PI severe.

Critical Review of the DSM Criteria

Empirical Basis and Validity

Perhaps the strongest aspect of the current DSM criteria set for ADHD is its empirical basis. DSM-5 criteria, which are largely similar to their predecessors from DSM-IV, are some of the most rigorous and empirically derived criteria ever available in the history of ADHD. They were derived from a process in which (1) a committee of some of the leading experts in the field met to discuss its development; (2) a literature review of ADHD symptoms was conducted; (3) a survey of rating scales assessing the behavioral dimensions related to ADHD, along with their factor structure and psychometric properties, was undertaken; and (4) a field trial of the subsequently developed item pool was conducted with 380 children from 10 different sites in North America (Applegate et al., 1997; Lahey et al., 1994).

The pool of items from which the criteria were taken was derived primarily from factor analyses of items from parent and teacher rating scales in which the items already showed high intercorrelations with each other and the underlying dimension, as well as validity in distinguishing children with ADHD from other groups of children (Lahey et al., 1994; Spitzer, Davies, & Barkley, 1990). DSM-5 clusters these items underneath two symptom categories (i.e., inattention and hyperactivity–impulsivity); the factor structure of these items is supported by a multitude of factor analyses of behavioral ratings of over 60,000 children displaying these two symptom dimensions (Willcutt et al., 2012).

The cutoff points for the number of symptoms necessary for a diagnosis (six) were determined in a field trial (Lahey et al., 1994) as having the greatest interrater reliability and discrimination of children with ADHD from those without ADHD.[1] Thus, unlike previous iterations of the DSM, selection of the criteria put forth in the DSM-IV had an empirical basis. Other research has supported the stability of ADHD diagnoses over time (Willcutt et al., 2012), with the caveat that diagnoses are likely to shift between subtypes during subsequent assessments (Lahey, Pelham, Loney, Lee, & Willcutt, 2005). That is one reason why DSM-5 no longer allows for three subtypes and instead specifies three "presentations"; it is to emphasize that cases at certain times may have more of one symptom dimension than the other, without conveying the notion that they are distinct types. ADHD diagnoses predict future functional impairment, even when researchers control for confounding factors such as socioeconomic status, intelligence, and comorbid psychopathology (Lahey & Willcutt, 2010).

Situational Pervasiveness

The requirement that symptoms of ADHD should be present in two or more situations was introduced in DSM-IV and formalized as a diagnostic requirement in DSM-5. Given that ADHD appears to result from neurological dysfunction, it is expected that children with the disorder should show symptoms, and to a lesser extent, impairment, in more than one domain of functioning (e.g., at school and at home). If a person has symptoms only in a single setting, then it is likely that environmental influences in that setting rather than ADHD are eliciting the problematic behavior. As such, this requirement has important implications for correctly diagnosing and treating ADHD, but, unfortunately, the specifics of this criterion are not well developed in DSM-5.

An immediate problem with establishing impairment across settings is that information on each setting is often provided by a different informant(s) (e.g., parents, teachers), so ratings on each setting are typically confounded by the source. The degree of agreement between parents and teachers is modest for most dimensions of behavior; it often ranges between .30 and .50, depending on the behavioral dimension being rated (Achenbach, McConaughy, & Howell, 1987; Mitsis, McKay, Schulz, Newcorn, & Halperin, 2000). This relatively modest correlation between parent and teacher reports of behavior sets a low ceiling for the level of agreement that can be expected between two informants.

Possible causes of such disagreement between sources are numerous and difficult to establish on a case-by-case basis. Discrepancies may in part reflect real differences in the child's behavior in these different settings, probably as a function of true differences in situational demands. School, after all, is quite different from the home environment in its expectations, tasks, social context, and general demands for public self-regulation, and, as we describe later, these situational differences can have profound effects on the behavior of children with ADHD. The differences may also reflect the degree of historical knowledge the rater possesses concerning that child; at the beginning of the school year, teachers have far less information about a particular child they are asked to describe than will be the case later in the year. But the disagreements may

also reflect differences in the attitudes, experiences, and judgments of different people. Although it is likely that teachers have a larger "normative group" against which to compare a single child's behavior, parents likely have more behavioral observations of their child on which they can base their ratings. Indeed, parent and teacher ratings of symptoms predict unique components of functional impairment, suggesting that each source of information provides some valid information about the child's symptoms (Hart, Lahey, Loeber, & Hanson, 1994). As such, clinicians should interpret these assessment sources as providing information on the child *in that particular context* and nothing more, rather than as evidence as to whether or not the child really has the disorder. When agreement across parent, teacher, and clinician is a requirement for diagnosis, it severely reduces diagnosis (particularly for the ADHD-PI and ADHD-PHI presentations) within the childhood population (Mitsis et al., 2000).

In summary, the requirement that children show ADHD symptoms across settings ensures that those with behavioral problems secondary to specific environmental factors are not diagnosed with ADHD. However, there are problems with interrater agreement and situational variability in children's symptoms that can make establishing impairment across settings challenging. Clinicians do well to remember that no source of information is entirely infallible. There is also no empirical basis for requiring that reports from multiple informants be used to support a diagnosis of ADHD. Until more research is done to address this issue, even evidence for *a history* of symptoms across settings should probably be sufficient to satisfy this criterion, rather than the current implicit requirement of agreement between parents and teachers on the number and severity of symptoms—a requirement not actually stated in DSM-5.

Conceptual Considerations

The case has been made both in theoretical and empirical writings that ADHD is probably a disorder of executive functioning (EF; Barkley, 1997, 2012d). Reviews of psychometric tests of EF have typically concluded that only a minority of individuals with ADHD are impaired on these tests; thus, ADHD is not principally a disorder of EF (Willcutt, Doyle, Nigg, Faraone, & Pennington, 2005). EF deficits are therefore seen in only a subset of these patients, so other conceptualizations of the disorder must be considered. But as Barkley has argued (2011b, 2012d; Barkley & Fischer, 2011; Barkley & Murphy, 2011), this assumes that the premise of the assertion is correct, which is that psychometric tests of EF are the "gold standard" for assessing EF. This premise is highly questionable, especially since there is no relationship between EF tests and EF rating scales (Toplak, West, & Stanovich, 2013) or other measures of adaptive functioning, nor are those tests very adept at predicting impairment in major life activities with which EF ought to be involved (see Barkley, 2012d, for review and discussion). In contrast, studies using rating scales of EF indicate that the vast majority of children and adults with ADHD are so impaired, and that such scales are better at predicting impairment in major life activities (Barkley, 2011b, 2012a).

More to the point of this discussion, research has shown that rating scales of DSM symptoms are so highly correlated with EF rating scales that they approach or meet standards of colinearity (synonymity) (Barkley, 2011b, 2012a). If that continues to be the case, then it may well be that the current conceptualization of the two DSM symptom lists for ADHD should be broadened to include metacognition or EF as representing the conceptualization of the inattention dimension and behavioral disinhibition or self-restraint as representing the hyperactive–impulsive dimension. At the very least, future DSM editions may need to broaden these conceptualizations in the associated text with the criteria even if the names for the symptom dimensions remain unchanged.

Developmental Considerations

Developmental issues continue to pose significant problems for DSM ADHD criteria. The original DSM-IV field trials tested the use of ADHD criteria on children between ages 4 and 16. It is unclear at this time whether the current symptom set is appropriate for use outside of individuals in this age range. This concern arises from the finding that some symptoms, particularly those in the hyperactive–impulsive cluster, decline in frequency as people age (Hinshaw, Owens, Sami, & Fargeon, 2006; Larsson, Lichtenstein, & Larsson, 2006). This is problematic because applying symptoms that change in frequency across development may result in changing sensitivity and specificity across age groups. Given the age-dependent decline in hyperactive–impulsive symptoms, young preschool-age children (ages 2–3) may be inappropriately diagnosed as having ADHD (false positives), while a smaller than

expected percentage of adults would meet the criteria (false negatives).

This problem has been partially addressed in DSM-5. Instead of requiring six symptoms in either cluster for all age groups, older adolescents and adults are required to endorse five or more symptoms in at least one cluster to receive a diagnosis. Although this is a step in the right direction toward addressing the age-related reduction in hyperactivity–impulsivity symptoms, more could have been done. First, it is questionable whether a cutoff score of five items is most appropriate for adults. For instance, Murphy and Barkley (1996) initially found that endorsing four items placed older adults at the upper end of the distribution (i.e., > 93rd percentile). Furthermore, those adults who endorsed four or more symptoms continued to show functional impairment, which suggests that their symptoms continued to interfere with their functioning. These findings were later supported by analyses of both large samples of clinic-referred and community adults and children with ADHD followed to young adulthood (Barkley, Murphy, & Fischer, 2008). Subsequently, this cutoff was replicated using a large population sample representative of U.S. adults (Barkley, 2011a). Thus, the currently prescribed five-item cutoff for adults should probably be four symptoms on either list because the use of five items may continue to underidentify impaired adults. Others have also found this threshold to be valid for diagnosing adults with ADHD (see Barkley et al., 2008, for discussion). What this makes plain is that without some age-related adjustments to the thresholds, individuals can outgrow the DSM criteria without outgrowing their disorder, which is seen as developmentally deviant and impairing.

Second, DSM-5 does not adjust the required number of symptoms for preschool children. Unfortunately, there is little research on diagnostic thresholds in preschoolers, and none was done in support of DSM-5. For this reason it is difficult and potentially problematic to diagnose young children using DSM-5 criteria. As discussed earlier, preschool children show higher rates of hyperactive–impulsive symptoms than do school-age children. This may reflect normal developmental patterns rather than higher rates of ADHD in this age group. Indeed, it is in this age group that children are likely to be diagnosed with ADHD-PHI subtype—or "presentation" in DSM-5 terminology—a subtype of the disorder that often remits before adolescence (Willcutt et al., 2012). This may reflect typical developmental patterns rather than true ADHD in young children.

In addition to the necessity of age-dependent changes in the optimal cutoff score, one also must consider that the items themselves become more or less valid as a function of age. The items used to construct DSM-IV symptom lists were initially based entirely on research on children (Spitzer et al., 1990). To address the more egregious examples of this problem, DSM-5 now includes parenthetical clarifications of many symptoms to advise clinicians on how the symptoms might be expressed in teens or adults. This is because it is likely that a set of items developed specifically for children is not ideal for measuring the same construct in adolescents or adults. Problematic with this approach to adjusting item content is that the parenthetical clarifications were not themselves tested in any research projects as such to determine whether they in fact are clarifications of that item (are collinear with it) or actually may represent an additional symptom. These clarifications also have not been tested for their accuracy in discriminating teens or adults with ADHD, so while the intent here was commendable, the inclusion of these untested clarifications may pose problems for both reliability and validity of the item pool for diagnosis.

In a large-scale research project intended to identify the best items for the diagnosis of adult ADHD, Barkley and colleagues (2008) tested 99 new symptoms of ADHD, most of which were derived from either chart reviews of several hundred adults diagnosed with the disorder or rating scales and theories of executive functioning. Eight were from the symptom list for oppositional defiant disorder (ODD). These items were evaluated using both large samples of clinic-referred adults diagnosed with ADHD (via DSM-IV), adults seen at the same clinic but not meeting criteria for the disorder, and a community sample of adults. They were also evaluated in a study of children with ADHD who were followed to adulthood. Following the identification of the best item set from these 99 symptoms for discriminating between adults with ADHD and control groups, the resulting small set of items was then also tested against the DSM-IV 18-item set. The result was that of the nine items identified as being the most accurate at discriminating adults with ADHD, only three items were from DSM-IV and none were from the hyperactive–impulsive symptom list. These items, shown in Table 2.2, are listed in descending order of discriminative accuracy. The final two items on this list, taken from DSM-IV, were retained but contributed just 1% more accuracy to the group discriminations. Further analyses showed that a cutoff of 4 of the

TABLE 2.2. A List of Proposed Diagnostic Criteria for ADHD in Adults

Has **six** (or more) of the following nine symptoms or four or more of the first seven symptoms that have persisted for at least 6 months to a degree that is maladaptive and inconsistent with developmental level:

1. Often easily distracted by extraneous stimuli.
2. Often make decisions impulsively.
3. Often has difficulty stopping activities or behavior when they should do so.
4. Often starts a project or task without reading or listening to directions carefully.
5. Often shows poor follow through on promises or commitments they may make to others.
6. Often has trouble doing things in their proper order or sequence.
7. Often more likely to drive a motor vehicle much faster than others (excessive speeding). (If person has no driving history, substitute: "Often has difficulty engaging in leisure activities or doing fun things quietly.")
8. Often has difficulty sustaining attention in tasks or leisure activities.
9. Often has difficulty organizing tasks and activities.

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first seven items or six of the entire list of nine items would be useful for making a diagnosis of adult ADHD, and better at doing so than the DSM-IV. Other studies have also shown this reduced item set to be useful in identifying adult ADHD (Vergara-Moraques et al., 2011). Using this same original item set, Biederman and colleagues (Biederman et al., 2008) found that just eight items were as useful at identifying functional impairment in adults with ADHD as the entire item set, some of which overlap with those in Table 2.2. Later, Fedele, Hartung, Canu, and Wilkowski (2010) studied this same item pool for its utility in predicting impairment in ADHD in a large sample of college students and found that 17 items were sufficient to account for such impairment accurately. Note that Barkley and colleagues (2008) developed their items from those that best discriminated between adults with ADHD and non-ADHD groups; that is a different undertaking than identifying items best at predicting functional impairment, as these latter two studies attempted to do. So it is not surprising that the resulting reduced item sets found in these studies are not identical. All of this is to say that better items than those in DSM-5 exist as symptoms for diagnosing adults with ADHD, whether for discriminating them from other conditions or for predicting impairment. Although this information was shared with the DSM-5 committee developing the criteria for ADHD, no new items were eventually included in the final DSM-5 for use with adults.

Another issue raised from an inspection of the two clusters or dimensions of items suggests that the items for inattention may have a wider developmental applicability across the school-age ranges of childhood and possibly into adolescence and young adulthood (e.g., "Is often easily distracted"). Those for hyperactivity–impulsivity, in contrast, seem much more applicable to young children and less appropriate or not at all applicable to older teens and adults (e.g., "Often leaves seat in situations when remaining seated is expected"). This hypothesis is supported by the previously discussed findings that people show reductions in hyperactive–impulsive symptoms, but not inattentive symptoms, as they age. In other words, it may be that hyperactivity–impulsivity does not actually decline over time, but that instead DSM criteria become less sensitive to these constructs throughout development.

How, then, can clinicians handle these limitations of the DSM criteria related to developmental changes? Although only limited effort is made in DSM-5 to define "developmental inappropriateness," the ubiquity of well-normed behavior rating scales for ADHD symptoms argues for the use of such instruments to determine the extent of developmental deviance in a particular case (see Chapter 18 for a discussion of these scales). Although not wholly objective, such instruments do provide a means of quantifying parent and teacher opinions in the case of children, and self-report and other-report of symptoms in the case of adults being evaluated for ADHD (Barkley, 2011a; Conners, Erhardt, & Sparrow, 1999). Moreover, national norms are now available for the parent and teacher versions of these instruments for children (DuPaul et al., 1997; DuPaul, Power, McGoey, Ikeda, & Anastopoulos, 1998), and self and other ratings for adults (Barkley, 2011a). The use of such scales automatically provides a means for establishing deviance relative to both age and gender membership of an individual given that norms are provided separately for males and females by age groups. With such norms available, we must then specify a recommended threshold that is considered "inappropriate." It would seem prudent to establish a cutoff score

on these scales of at least the 90th percentile, and preferably the 93rd percentile, as the demarcation for clinical significance, given that the 93rd percentile (+1.5 standard deviations above the mean) is a traditionally employed cutoff point for this purpose (DuPaul, Power, McGoey, et al., 1998). This recommendation should be taken not as gospel but instead as a guideline for circumventing some of the limitations of DSM criteria.

The age-of-onset criterion in DSM-5 specifies that to be diagnosed with ADHD, a person must have shown evidence of symptoms producing impairment when he or she was 12 years of age or younger. In DSM-IV, this age restriction required evidence of impairment prior to age 7. This criterion was challenged by the results of its own field trial (Applegate et al., 1997), and a subsequent review confirmed that strict application of the DSM-IV age-of-onset requirement resulted in underidentification of people showing impairment (Kieling, Kieling, & Rohde, 2010). Although people with early-onset ADHD (i.e., prior to age 7) appear to have more severe and persistent conditions, with more problems with reading and school performance generally (McGee, Williams, & Feehan, 1992), those with later onset still demonstrate considerable functional impairment related to their symptoms (Faraone et al., 2006). In the original DSM-IV field trials (Applegate et al., 1997), the age 7 age-of-onset criterion resulted in underidentification of children with ADHD-PI, but it performed reasonably well for children with ADHD-C, perhaps because hyperactivity–impulsivity symptoms can result in disruptive behavior that is more salient than symptoms of inattention to teachers and parents. Overall, that DSM-5 requires symptom onset before age 12 rather than age 7 will likely result in fewer false negatives, although clinicians should be cautious if children present with symptoms of hyperactivity that were not present earlier in their development.

Clinicians may also want to view age of onset as a guideline that requires onset of symptoms in childhood or adolescence rather than as an empirically supported demarcation between real and false cases of disorder. We advise this for two reasons. First, research shows that at least 7–10% of children and adults meeting all other criteria for ADHD have an onset of symptoms after age 12 (Barkley et al., 2008). Second, self- and parental recall of childhood onset of these symptoms is quite unreliable among teens and adults, on average occurring 4–5 years later than what was established when they were children with ADHD entering a longitudinal study (Barkley et al., 2008).

Subtyping

Beginning with DSM-III, diagnostic specifiers have been used to distinguish among people whose symptoms are primarily in the inattentive cluster, those whose symptoms are primarily hyperactive–impulsive, and those who show symptoms in both clusters. There are differences, however, in how these symptom specifiers are described in each DSM edition. In DSM-IV, as previously mentioned, diagnostic subtypes were determined based on the pattern of symptoms endorsed. In DSM-5, the "subtype" terminology was dropped in favor of a more fluid "presentation" system. This change was a reaction to the finding that often children would be diagnosed with one subtype of ADHD, then meet criteria for another subtype of the disorder at follow-up (Lahey et al., 2005; Willcutt et al., 2012). Although this presentation system is clinically useful in that it allows clinicians to distinguish between children whose primary problems are attention-related and those who struggle with hyperactivity–impulsivity, the structure of this system has created numerous problems.

First and foremost is the concern that the diagnostic category ADHD-PI contains at least two diagnostic subgroups (Diamond, 2005; Milich, Balentine, & Lynam, 2001). One group includes children who demonstrate clinically significant symptoms of inattention and subclinical, but still considerable, levels of hyperactivity–impulsivity. Another group comprises children whose inattentive symptoms are linked to problems with arousal and sluggish cognitive tempo (SCT; McBurnett, Pfiffner, & Frick, 2001). Although we describe the SCT dimension later in this chapter and it is discussed more thoroughly in Chapter 17, suffice to say that there are a number of qualitative differences between individuals with the SCT subset of ADHD-PI and those with "subthreshold" ADHD-C; emerging research suggests that these groups suffer from qualitatively different disorders (Barkley, 2012c, 2013, 2014). Another subgroup that meets criteria for ADHD-PI is those people who originally met criteria for ADHD-C but experienced the previously described age-related reduction in hyperactive–impulsive symptoms. These individuals likely suffer from a disorder that is qualitatively distinct disorder from that of persons whose symptoms were always consistent with ADHD-PI but due to partial symptom remission in the hyperactive–impulsive cluster do not meet criteria for ADHD-C. One way to address this issue may be to develop a separate set of criteria for individuals with inattentive symptoms but no

hyperactive–impulsive symptoms (Adams, Milich, & Fillmore, 2010). This may improve classification accuracy and reduce heterogeneity among people diagnosed with ADHD-PI.

Another problem with the subtyping system is that very little is known about differences among the subtypes in terms of developmental trajectory, associated problems, and treatment response. Most of the research on children and adults with ADHD does not differentiate between the groups, so by default most of the participants show a considerable number of hyperactive–impulsive symptoms. There are, of course, studies that have specifically recruited an ADHD-PI group for comparison with children with ADHD-C (Nigg, Blaskey, Huang-Pollock, & Rappley, 2002); however, these studies are the exception rather than the rule, and even these studies may have limitation in their findings because the ADHD-PI groups typically include both children with "true" ADHD-PI and those with "subthreshold" ADHD-C. Indeed, studies that have attempted to identify groups of children with "pure" inattentive symptoms often report intriguing differences between the subtypes (Adams, Derefinko, Milich, & Fillmore, 2008; Carr, Henderson, & Nigg, 2010; Fillmore, Milich, & Lorch, 2009).

As a counterpoint to our view on the importance of subtypes, several studies have shown that children diagnosed with one subtype of ADHD are very likely to meet criteria for another subtype during a later assessment (Willcutt et al., 2012). This subtype instability calls into question the importance of research on the differences between subtypes of the disorder because if subtypes do not persist, then the differences among them are unlikely to be consequential in the long run. However, it should be noted that some of this subtype instability may result from small changes in symptom count (e.g., six hyperactive–impulsive symptoms during an initial assessment and five hyperactive–impulsive symptoms at follow-up). As discussed earlier, a way to improve subtype stability may be to develop a separate set of criteria for those with heightened inattentive symptoms and few or no hyperactive–impulsive symptoms.

A final issue concerns the validity of the ADHD-PHI subtype. This is by far the most uncommon subtype of this disorder; most children who show symptoms of hyperactivity–impulsivity also meet criteria in the inattentive symptom cluster. Those individuals who only meet criteria in the hyperactive–impulsive symptom cluster are mostly young and show the highest level of diagnostic instability over time (Lahey et al., 2005). One source of this diagnostic instability may be that ADHD-PHI is an earlier developmental stage of ADHD-C. Indeed, many children originally diagnosed with ADHD-PHI are diagnosed with ADHD-C during follow-up assessment. Another possible source of this diagnostic instability is that young children diagnosed with ADHD-PHI show symptom remission as they age from early to middle childhood. By far the largest proportion of these children do not meet criteria for ADHD at follow-up (Willcutt et al., 2012), which suggests that the original diagnosis reflected a typical developmental phase or even a time-limited behavior disorder.

To summarize, children and adults with ADHD-PI are a mixed group. Some of them (perhaps 30–50%) have an SCT form of attention disturbance, which may constitute a qualitatively unique disorder from the attention disturbance in ADHD-C. Others are older children and adults who were once diagnosed as having ADHD-C but have shown a decrease in the number and severity of their symptoms of hyperactivity with age, such that they now fall below the critical number of six such symptoms required for the ADHD-C diagnosis. Although the DSM decision rules would reclassify these individuals as having ADHD-PI, clinicians may wish to continue conceptualizing and treating them as having ADHD-C. Despite these limitations, whether people are diagnosed with ADHD-PI or ADHD-C is not entirely consequential at this time given the dearth of research on subtype differences in treatment response, developmental trajectory, and associated problems.

Functional Impairment as a Requirement for Diagnosing ADHD

The addition of a requirement of impairment as a criterion for diagnosis of a mental disorder is crucial, and its importance cannot be overemphasized. Simply because a child or adult may show a higher frequency or severity of symptoms related to ADHD than is typical of others does not by itself warrant a diagnosis of ADHD. Efforts to define the nature of a mental disorder typically incorporate such a requirement to distinguish a mental disorder from the wide range of normal human behavior and problems in living that do not necessarily lead to a harmful dysfunction or impairment (Wakefield, 1997). In treatment, it is aspects of functional impairment, rather than symptoms per se, that are tar-

geted for intervention (Pelham, 2001). As such, clinicians should remember that while symptom counts are means to an end for diagnostic purposes, clinical interventions should emphasize functional impairments that result from the disorder.

While the inclusion of an impairment criterion in ADHD diagnostic criteria is commendable for various reasons, defining the comparison standard for making such a determination has been left ambiguous. As discussed later in Chapter 33 on workplace accommodations for adults with ADHD, clinicians have sometimes used IQ as the standard against which to judgment impairment in high-IQ individuals, as if IQ were the "gold standard" against which all human abilities are to be compared for deviation. Such disparities are then seen as evidence that the person is not functioning up to his or her expected potential. Others have done the same thing by using high-functioning local peer groups as an index of what a person with ADHD should be expected to do. Any significant variation by an adult from the performance of this peer group is then viewed as evidence that the person is impaired (e.g., a medical student with ADHD who is doing poorly in organic chemistry relative to other medical students at that college). In neither of these cases does the individual have to place below the average of the population, which makes these standards questionable, if not laughable, for determining impairment. Inherent in that determination is that the individual must be functioning below the average, normal, or typical person in the population of that age group. To say otherwise mocks the very meaning of the terms "impairment" and "disorder." Future DSM editions need to do a better job of making explicit what is meant by "impairment" (e.g., functional ineffectiveness that leads to adverse consequences; Barkley, 2011c, 2012b) and against what standard it is to be judged (e.g., average person).

Sex Differences in ADHD

Sex differences in ADHD are covered in depth later in Chapter 9, but we should briefly mention here the (lack of) consideration of sex differences in DSM-5. Research evaluating gender differences in psychopathology using the DSM-IV criteria sets demonstrates that males in the general population display more symptoms of ADHD than do females (Polanczyk, de Lima, Horta, Biederman, & Rohde, 2007), more so in childhood than in adulthood. This gender difference results in females having to meet a higher threshold relative to other females to be diagnosed with ADHD than do males relative to other males. The problem is further accentuated by the fact that the majority of individuals in the DSM-IV field trial were males, making the DSM criteria primarily male-referenced. Nonetheless, a series of studies by Hinshaw (Miller, Sheridan, Cardoos, & Hinshaw, 2013) has shown that females with ADHD show equally if not more negative functional outcomes compared to their male counterparts (see Chapter 9). In light of these findings, an important future goal is for researchers to examine whether female-specific cutoffs or even a separate set of criteria better captures the disorder as it presents in girls. In the meantime, clinicians should be aware of these important sex differences in the nature of ADHD.

Symptom Persistence

A final issue concerns the requirement that people display symptoms of the disorder for at least 6 months to receive the diagnosis. This number was chosen mainly in keeping with the criteria set forth in earlier DSM editions and consistency with criteria used for other disorders; there is little or no research support for selecting this particular length of time for symptom presence in the case of ADHD. Nonetheless, given that ADHD is a lifelong disorder apparently resulting from neurological dysfunction, it is reasonable to expect the symptoms to persist. Yet specifying a precise duration is difficult in the absence of research to guide the issue. Research on preschool-age children might prove helpful here. Such research shows that many children age 3 years or younger may have parents or preschool teachers who report concerns about their activity level or attention, but that these concerns have a high likelihood of remission within 12 months (Campbell, 1987). It would seem that, at least among preschoolers, the 6-month duration specified in DSM-5 may be too brief, resulting in the possibility of overidentification of ADHD in children at this age (false positives). However, this same body of research found that for those children whose problems lasted at least 12 months or beyond age 4 years, a persistent pattern of behavior was established that was highly predictive of its continuance into the school-age range. The finding suggests that the duration of symptoms might be better set at 12 months or longer, to improve the rigor of diagnosis in detecting true cases of disorder.

Other Changes in DSM-5

The recent release of DSM-5 has presented an opportunity to address some of the ongoing problems with ADHD as a diagnostic category. Unfortunately, the changes in this DSM edition may have created more problems than they solve. Although we described many of the more pervasive problems in the structure of ADHD in DSM earlier in this section, there are changes in the DSM-5 that may further complicate the diagnosis and treatment of ADHD (Roberts & Milich, 2013).

One major change in DSM-5 is that the section of the manual containing ADHD was changed. In DSM-IV, ADHD was included in the section "Disorders Usually Diagnosed in Infancy, Childhood, or Adolescence," which included all of the disruptive behavior disorders of childhood. This section was omitted from DSM-5, and the disorders that previously were in this section have been reassigned to "Neurodevelopmental Disorders" and "Disruptive, Impulsive Control, and Conduct Disorders," based on the nature of the disorder in question. Although ADHD was included as a "Neurodevelopmental Disorder," this may not have been the most appropriate classification method. An immediate problem with this decision is that different subtypes of the disorder are appropriate for different sections. On the one hand, children with ADHD-C and ADHD-PHI clearly present with disruptive and impulsive behavior that often leads to conduct problems—making this subtype an excellent fit for a section entitled "Disruptive, Impulsive Control, and Conduct Disorders." Furthermore, ADHD-C is highly comorbid with other externalizing disorders in this section, such as ODD and conduct disorder (Lahey & Willcutt, 2002). ADHD-PI, on the other hand, does not fit well in this section because children with primarily inattentive symptoms rarely show hyperactivity or impulsivity. ADHD-PI is better categorized in the "Neurodevelopmental Disorders" section with other childhood disorders not on the externalizing spectrum. Clearly, ADHD-C combined with ADHD-PHI and ADHD-PI are best suited for different sections of DSM-5. Because the three subtypes are linked together under a single diagnostic category, however, at least one must be categorized in an inappropriate diagnostic section.

Another series of changes in DSM-5 will likely affect the prevalence of the disorder (Sibley et al., 2012). DSM-IV required that children show symptoms and impairment when they were age 7 years or younger to receive a diagnosis. As previously mentioned, DSM-5 raised the age-of-onset criteria to age 12 years. Additionally, DSM-5 requires a history of symptoms— evidence that this history of symptoms caused impairment is no longer necessary. A similarly subtle change was made to the requirement that the child show problems across multiple settings. In DSM-IV, a diagnosis required that children show symptoms and impairment across more than one setting. In DSM-5, however, it is only necessary that children show "several" symptoms across more than one setting, so impairment in one setting is sufficient for a diagnosis. Finally, in DSM-5, autism spectrum disorders are no longer exclusionary conditions for diagnosing ADHD. This change will result in increased prevalence of ADHD as children with autism spectrum disorders are diagnosed with comorbid ADHD.

THE NATURE OF ADHD

Is ADHD a Mental Disorder?

Social critics (e.g., McGinnis, 1997), some non-expert professionals (e.g., Timimi, 2004), and fringe political–religious groups (e.g., the Church of Scientology and affiliated groups) charge that ADHD is a myth—or, more specifically, that professionals have been too quick to label energetic and exuberant children as having a mental disorder, and that educators also may be using these labels as an excuse for simply poor educational environments. In other words, children diagnosed with ADHD are actually normal but are being labeled "mentally disordered" because of parent and teacher intolerance (Kohn, 1989), parental and cultural anxiety surrounding childrearing (Timimi, 2004), or some unspecified and undocumented conspiracy between the mental health community and pharmaceutical companies (Timimi, 2004).

If this claim of ADHD as myth were actually true, and not just the propaganda it often turns out to represent, we should find no differences of any cognitive, behavioral, or social significance between children with and without the ADHD label. We should also find that being diagnosed with ADHD is not associated with any significant later risks in development for maladjustment (impairments) within any domains of adaptive functioning or social or school performance. Furthermore, research on potential etiologies for the disorder should likewise come up empty-handed. This is hardly

the case. The first 12 chapters of this volume constitute a direct and monumental refutation of such claims. Differences between children and adults with and without ADHD are numerous. And, as shown later, numerous developmental risks await the children meeting clinical diagnostic criteria for the disorder. Moreover, certain potential etiological factors are consistently noted in the research literature as being associated with ADHD. Therefore, any claims that ADHD is a myth reflect either a stunning level of scientific illiteracy or outright attempts to misrepresent the science of ADHD so as to mislead the public with propaganda (Barkley, 2004).

The fact that ADHD is a valid psychological trait, however, does not necessarily mean that it is also a mental disorder. Determining whether ADHD is a valid disorder requires that some standards for defining "disorder" be available. Jerome Wakefield (1997) has provided the field with the best available criteria to date for doing so, in our opinion. He has argued that mental disorders must meet two criteria to be viewed as such: (1) They must involve the dysfunction of universal mental mechanisms (adaptations) that have been selected in an evolutionary sense (have survival value); and (2) they must engender substantial harm to the individual (mortality, morbidity, or impaired major life activities). It should be clear from the totality of information on ADHD presented in this text that the disorder handily meets both criteria. Those with ADHD, as described previously, can show dysfunction in adaptive cognitive mechanisms (e.g., inhibitory control, working memory; Castellanos, Sonuga-Barke, Milham, & Tannock, 2006). These functions are evolutionarily adaptive, helping individuals to organize their behavior relative to time and the social future, and thereby to prioritize long-term over short-term social consequences (Ardila, 2008). Moreover, these neurocognitive deficits result in harm to the individual (see Chapters 3–8). In summary, ADHD meets the criteria of a valid mental disorder because it includes harmful dysfunction in a set of cognitive mechanisms evolved to have a survival advantage and confers functional impairment on those with the disorder.

Is ADHD a Categorical or Dimensional Trait?

Whether psychopathology is best conceptualized as categorical diagnoses or dimensional traits received an increasing amount of attention prior to the release of DSM-5. The notion of applying categories for psychopathologies of children seems to derive from the medical model, in which such categories constitute disease states (Edelbrock & Costello, 1984). From this perspective, an individual either has a disorder or does not. The DSM, in one sense, uses this categorical approach (all or none) by requiring that a person meet certain thresholds to be diagnosed with ADHD. The view of psychopathologies as representing dimensions of behavior, or even typologies (profiles) of these dimensions, arises from the perspective of developmental psychopathology (Achenbach & Edelbrock, 1983). In this view, ADHD constitutes the extreme end of a dimension, or dimensions, of behavior that falls along a continuum with the behavior of typical children. The dimensional view regards ADHD not necessarily as a disease entity but as a matter of degree in what is otherwise a characteristic of typical children.

As with most psychological constructs, ADHD is most accurately understood as a dimension. Many of the items described in DSM that constitute the ADHD symptom set were derived from behavioral rating scales that measure attention problems and hyperactivity (Spitzer et al., 1990). These scales and their item pools are dimensional. The underlying dimensional nature of ADHD is also supported by genetics studies suggesting that ADHD represents a dimensional condition rather than a pathological category (Hay, Bennett, Levy, Sergeant, & Swanson, 2007; Sharp, McQuillin, & Gurling, 2009).

Nonetheless, ADHD is typically described as and understood among the lay public as a categorical condition. This misrepresentation of the true nature of ADHD is not without merit. With our current system of mental health and disability services, it is important that categorical decisions be made, so that corresponding yes or no decisions can be made regarding the disbursement of services. With this is mind, an immediate question is whether assigning categories to a continuous variable results in a significant loss of validity when measuring that construct, and if so, can this loss of validity be justified by the utility of artificially dichotomizing the condition? In one study addressing this issue, Lahey and Willcutt (2010) compared categorical diagnosis to a continuous classification method based on symptom count to determine which performed better in predicting functional impairment and remaining stable over time. They found that the continuous classification approach performed better than the categorical system on both accounts.

Overall, the dimensional approach to ADHD seems most consistent with the available evidence, whereas

the categorical approach remains one of convenience, parsimony, and tradition (Hinshaw, 1994). Clinicians would do well to be flexible in how they conceptualize the disorder because different scenarios likely call for different treatment of the subject. Along these lines, DSM-5 takes a mixed approach by including diagnostic modifiers (i.e., mild, moderate, severe) that can be used in addition to the categorical diagnosis. This provides clinicians with more latitude to describe patients' symptoms on a dimension. Whether these dimensional diagnostic modifiers are beneficial to clinical practice remains to be seen.

PRIMARY SYMPTOMS

An important distinction should be made at the outset. The term "symptom" as used here refers to a behavior (e.g., skipping from one uncompleted activity to another) or a set of behaviors that significantly covary together (e.g., inattention) and are believed to represent a dimension of a mental disorder. The term "symptom" must be distinguished from that of "impairment," because the two are often confused in clinical discussions of disorders. "Impairments" are the consequences or outcomes of symptoms or symptom classes, such as retention in grade, failure to graduate from high school, vehicular crashes, license suspensions, teen pregnancy, or criminal arrests. They reflect functional ineffectiveness in these various domains of major life activity and serve as signs of the environment "kicking back" in response to the display of the symptom(s). The various impairments associated with the disorder are discussed in later chapters of this volume.

Those with ADHD are commonly observed by others as having chronic difficulties with inattention and/or impulsivity–hyperactivity. They display these characteristics early in development and to a degree that is excessive and inappropriate for their age or developmental level. Their symptoms are most evident in situations that tax their capacity to pay attention, restrain their movement, inhibit their impulses, and regulate their own behavior relative to rules, time, and the future. As noted in Chapter 1, definitions have varied considerably throughout the history of this disorder, as have the recommended criteria for obtaining a diagnosis. In this section we first review the dimensions of behavior at the core of the DSM ADHD symptom clusters: inattention and hyperactivity–impulsivity. We then briefly discuss SCT, a construct that is closely related to and may overlap with ADHD. That condition receives far more consideration in Chapter 17.

Two dimensions of behavior are almost uniformly found when the symptoms of ADHD as rated by parents and teachers are factor-analyzed (Willcutt et al., 2012). These two dimensions are used to create and diagnose the disorder and to construct its subtypes, at least within the DSM. The first dimension comprises impulsive and hyperactive behaviors, and the second encompasses behaviors related to poor control of attention. These dimensions are found across ethnic and cultural groups (Rohde et al., 2005), supporting the notion that ADHD comprises two separate but correlated symptom clusters.

Hyperactivity–Impulsivity

The first dimension of symptoms that emerges from factor analyses of symptom ratings in both children and adults is that of poor inhibition and associated hyperactivity (Willcutt et al., 2012). Clinically, those with ADHD are often noted to respond quickly to situations, without waiting for instructions to be completed or adequately appreciating what is required in the setting, resulting in impulsive errors. These individuals may also fail to consider the potentially negative, destructive, or even dangerous consequences that may be associated with particular situations or behaviors. Taking chances on a whim or dare, especially from a peer, may occur more often than is typical. They may carelessly damage or destroy others' property considerably more frequently than do children without ADHD.

Waiting for their turn in a game or in a group lineup before going to an activity is often problematic for children with ADHD; indeed, waiting in general may be problematic for people of all ages with the disorder. When faced with tasks or situations in which they are encouraged to delay seeking gratification and to work toward a longer-term goal and larger reward, they often opt for the immediate, smaller reward that requires less work to achieve. They are notorious for taking "shortcuts" in their work performance, applying the least amount of effort and taking the least amount of time in performing tasks they find boring or aversive. When they desire something to which others control access and they must wait a while to obtain it, as in a parent's promise to eventually take them shopping or to a movie, they may badger the parent excessively during the waiting interval, appearing to others as incessantly demanding and self-centered. Situations or games that

involve sharing, cooperation, and restraint with peers are particularly problematic for these impulsive children. Verbally, they often say things indiscreetly, without regard for the feelings of others or for the social consequences to themselves. Blurting out answers to questions prematurely and interrupting the conversations of others are commonplace.

Impulsivity is multidimensional in nature (Nigg, 2000). Discussions of impulsivity often include constructs such as executive control, delay of gratification, effort, and even compliance (Olson, Schilling, & Bates, 1999). Others reorganize inhibition into executive (volitional), motivational (precipitated by fear or anxiety), and automatic attentional inhibitory processes (Nigg, 2000). Those forms of impulsivity often associated with ADHD involve the undercontrol of behavior (poor executive functioning); poor sustained inhibition; and the inability to delay a response, defer gratification, or inhibit dominant or prepotent responses (Nigg, 2001). But there is also evidence that children with ADHD have an equal or greater problem with delay aversion: They find waiting to be aversive and therefore act impulsively to terminate the delay more quickly (Johansen, Aase, Meyer, & Sagvolden, 2002).

Evidence that behavioral disinhibition, or poor effortful regulation and inhibition of behavior, is in fact a hallmark of the disorder is substantial (Barkley, 1997; Nigg, 2001). First, whereas heightened rates of inattentive behavior characterize a broad range of clinical groups, hyperactive–impulsive behaviors are relatively specific to ADHD and distinguish people with ADHD from other clinical groups much more consistently than do inattentive behaviors (Kofler et al., 2013; Lipszyc & Schachar, 2010). Second, when objective measures of the three sets of symptoms of ADHD are subjected to a discriminant function analysis (a statistical method of examining the variables that contribute most to group discrimination), it is routinely the symptoms of impulsive errors, typically on vigilance tasks or those assessing response inhibition, and excessive activity level that best discriminate between children with and without ADHD (Losier, McGrath, & Klein, 1996). A third source of evidence is derived from the field trial of the DSM-III-R symptom list (Spitzer et al., 1990), which tested these symptoms' sensitivity and specificity. These descriptors were rank-ordered by their discriminating power and presented in DSM-III-R in descending order. Careful inspection of this rank ordering revealed that, again, symptoms characteristic of disinhibition, such as poorly regulated activity and impulsivity, were more likely to discriminate children with ADHD from those with other psychiatric disorders or no disorder. These observations inform disinhibition-based theories of ADHD, which are described later in this volume.

Recent meta-analyses of studies using stop-signal tasks and continuous performance tasks confirm that adults with ADHD show inhibitory deficits similar to those of children with the disorder (Hervey, Epstein, & Curry, 2004; Lijffijt, Kenemans, Verbaten, & van Engeland, 2005). Murphy and Barkley (1996) indicated that adults diagnosed with ADHD report difficulties with impulse control, such as "blurting out answers" and "interrupting or intruding on others" (see Barkley et al., 2008, for more evidence). Overall the symptoms reported by these adults were consistent with those reported by children with the disorder. Additionally, these impulse control problems can interfere with adult-specific activities, such as driving (Barkley, 2004; see Chapters 11 and 29). Thus, it appears that the symptoms characterizing childhood ADHD are likely to be associated with its adult equivalent.

Related to the difficulties with impulse control in those with ADHD are symptoms of excessive or developmentally inappropriate levels of activity, whether motor or vocal. Restlessness, fidgeting, and generally unnecessary gross bodily movements are commonplace, both in the complaints received from parents and teachers and in objective measures (Wood, Asherson, Rijsdijk, & Kuntsi, 2009). These movements are often irrelevant to the task or situation and at times seem purposeless. Parents often describe such a child as "always up and on the go," "acts as if driven by a motor," "climbs excessively," "can't sit still," "talks excessively," "often hums or makes odd noises," and "is squirmy" (DuPaul, Power, McGoey, et al., 1998). Observers of such children at school or while working on independent tasks find them out of their seats, moving around the classroom without permission, restlessly moving their arms and legs while working, playing with objects not related to the task, talking out of turn to others, and making unusual vocal noises (Barkley, DuPaul, & McMurray, 1990). The restlessness is likely to be more problematic in boring or low-stimulation situations than in ones where greater stimulation is available (Antrop, Roeyers, Van Oost, & Buysse, 2000; Zentall, Falkenberg, & Smith, 1985). It is not uncommon for these children to narrate their activity, consistent with their generally excessive patterns of speech and movement (Berk & Potts, 1991).

Numerous scientific studies using objective measures of activity level support observations that children with

ADHD are more active, restless, and fidgety than nonclinical children (McGrath, Handwerk, Armstrong, Lucas, & Friman, 2004). This heightened activity level is not just secondary to impulsivity: Children with ADHD are even more active during sleep (Cohen-Zion & Ancoli-Israel, 2004). Their activity levels in early morning hours may not be different from those of nondisabled children, but they may become so by the afternoon (Dane, Schachar, & Tannock, 2000). As with poor sustained attention, however, there are many different types of hyperactivity (Barkley & Ullman, 1975), and it is not always clear exactly which types are the most deviant for children with ADHD. Measures of ankle movement and locomotion seem to differentiate them most reliably from nondisabled children (Barkley & Cunningham, 1979), but even some studies of wrist activity and total body motion found them to be different as well (Teicher, Ito, Glod, & Barber, 1996). Additionally, objective measurement of their activity level during tasks demanding sustained attention reveals them to move their heads and bodies more than others, to move further away from their chairs than others, to cover a greater spatial area in doing so, and to show more simplified or less complex movement patterns in doing so (Teicher et al., 1996). Their heightened activity level is most pronounced in low-stimulation environments, suggesting that this hyperactivity is a form of stimulation seeking (Antrop et al., 2000).

Although hyperactivity is *conceptually* distinct from impulsivity, there is a large enough correlation between hyperactive and impulsive symptoms to conclude that they form a single symptom factor, at least in children. Typically, studies that factor-analyze behavioral ratings find that items of restlessness or other types of overactivity load on a factor constituting impulsive or disinhibited behavior (Willcutt et al., 2012). Likewise, studies of objective measures or behavioral measures of hyperactivity load onto the same factor as do similar measures of impulsivity (Berlin & Bohlin, 2002). These findings mean that overactivity as a dimension of behavioral impairment is closely associated with the disinhibition factor for children with ADHD.

In adults with ADHD, on the other hand, symptoms of hyperactive or restless behavior are often present but appear to involve more difficulties with fidgeting, a more subjective sense of restlessness, and excessive speech than the more gross motor overactivity characteristic of young children with ADHD. Researchers (Barkley et al., 2008; Murphy & Barkley, 1996) have found that nearly 60% of adults with ADHD reported that they often talked excessively. This excessive talking, however, did not distinguish the adults with ADHD from a clinical control group, suggesting that excessive talking may be a general characteristic of adults with psychopathology rather than a specific symptom of ADHD. Furthermore, whereas among children hyperactivity and impulsivity load onto a single symptom dimension, hyperactivity appears to form its own symptom dimension in adults (Barkley, 2011a; Kooij et al., 2005; Murphy & Barkley, 1996). It is clear that hyperactivity in adults functions differently than that in children, and this hyperactivity dimension in adults may be less important than impulsivity (mainly verbal). Clinicians should place more emphasis on behavioral impulsivity in adults with ADHD because hyperactivity may be of relatively less importance for diagnosing the disorder in adults.

Inattention

Another hallmark that is characteristic of children and adults with ADHD is impaired control of attention. Parents and teachers often describe these attention problems in terms such as the following: "Doesn't seem to listen," "Fails to finish assigned tasks," "Daydreams," "Often loses things," "Can't concentrate," "Easily distracted," "Can't work independently of supervision," "Requires more redirection," "Shifts from one uncompleted activity to another," and "Confused or seems to be in a fog" (Barkley et al., 1990). Many of these terms are the most frequently endorsed items from rating scales completed by the caregivers of these children (DuPaul, Power, Anastopoulos, & Reid, 1998; Mahone et al., 2002), and this trait of children with ADHD has been confirmed by behavioral observation studies indicating that children with ADHD spend more time off task or not paying attention than do their nonclinical peers (Barkley et al., 1990; DuPaul & Stoner, 2003).

Although the behaviors underlying the construct of inattention are well documented, less is known about the source of cognitive dysfunction that contributes to inattention at the behavioral level. Attention as it is studied by neurocognitive scientists is multifaceted and can refer to many different cognitive processes that support the control of attention (Chun, Golomb, & Turk-Browne, 2011; Petersen & Posner, 2012). "Attention" is a broad term encompassing many different cognitive mechanisms that support information processing, and inattentive behavior can result from failure of one or more of any of these cognitive processes.

People with ADHD are often characterized by poor attentional vigilance (i.e., remaining focused by sustaining attention on a certain task), particularly when they are required to attend to a dull or repetitive task (Boonstra, Oosterlaan, Sergeant, & Buitelaar, 2005; Hoza, Pelham, Waschbusch, Kipp, & Owens, 2001). This is consistent with the general observation that children and adults with ADHD have trouble persisting in tasks that are not inherently stimulating or interesting. People with ADHD also are more distractible than their nonclinical peers. Laboratory studies using eye-tracking tasks have shown that people with ADHD are more likely than their nonclinical peers to shift their attention toward task-irrelevant stimuli that they are instructed to ignore (Roberts, Fillmore, & Milich, 2011; Ross, Harris, Olincy, & Radant, 2000). This distractibility is also well documented by studies of response-time distributions using simple and choice response-time tasks. These almost uniformly show that those with ADHD have a slower and more variable response style than controls (Lijffijt et al., 2005), and this increased variability in response time occurs because those with ADHD become distracted and disengaged from the task (Adams, Roberts, Milich, & Fillmore, 2011; Spencer et al., 2009).

Whether distractibility, poor vigilance, a combination thereof, or even another trait contributes to inattention in those with ADHD is not entirely clear and may vary on a case-by-case basis. For example, the neuropsychological problem underlying poor attention in children with ADHD-C is most often one of diminished persistence of effort or sustained responding to tasks that have little intrinsic appeal or minimal immediate consequences upon completion (Johansen et al., 2002). Thus, although casual observation of these children would suggest high susceptibility to distraction, their poor task persistence may result from a motivational problem given their inability to persist in tasks that are not immediately reinforcing. Other children may have trouble attending due to an inability to inhibit shifts in attention toward distracting stimuli. A third viable mechanism is that children and adults with ADHD have impaired working memory (holding information in mind that guides behavior toward goals or tasks). Distractions may so interfere with working memory that the individual forgets the original task or goal he or she was pursuing and switches to a new task, one that is often provided by the distraction. In this sense, the underlying cognitive dysfunction is quite different, but the resulting behavioral symptoms are similar. Multiple pathway models of ADHD have garnered substantial empirical support, and future conceptualizations of the disorder will likely acknowledge that no single cognitive impairment is sufficient to explain all cases of ADHD (Fair, Bathula, Nikolas, & Nigg, 2012; Sonuga-Barke, Bitsakou, & Thompson, 2010).

Research indicates that adults with ADHD experience many of the same attention problems that are seen in children with the disorder. Much of this work is based on studies using the same neuropsychological measures of attention, such as manual response tasks and eye-tracking tasks (Lijffijt et al., 2005; Roberts et al., 2011). Other researchers have examined how inattention manifests in adult-specific tasks, such as driving, and have shown that adults with ADHD have more lapses in attention during these activities, particularly under high-distraction conditions (Reimer, Mehler, D'Ambrosio, & Fried, 2010). Finally, studies (Barkley et al., 2008; Murphy & Barkley, 1996) indicate that adults with ADHD report many of the same attention problems seen in children with ADHD, and these self-reports were corroborated by parents and spouses. In summary, there is ample justification for believing that adults and children with ADHD suffer from many of the same attention problems.

Secondary Symptom Cluster: Sluggish Cognitive Tempo

SCT is a dimension of symptoms that are related but empirically distinct from the inattentive symptom cluster of ADHD (McBurnett et al., 2001). Emergence of this symptom cluster was based on comparisons of the attention deficit disorder (ADD) subtypes in DSM-III, which included ADD with and without hyperactivity. The ADD with hyperactivity group was described as inattentive by way of distractibility and sloppiness, whereas those with ADD without hyperactivity showed a qualitatively different form of inattention characterized by daydreaming, sluggishness, slow processing, lethargy, and physical hypoactivity (Carlson & Mann, 2000). Subsequent factor analyses of items derived from DSM-III criteria revealed that these items formed a separate factor than the inattentive items included in DSM-IV (Milich et al., 2001). Items derived from this SCT symptom dimension were included in the DSM-IV field trials, but they were not included in the final diagnostic criteria (Lahey et al., 1994). Although these items were removed from the official diagnostic system, in recent years there has been a renewed interest in

the role of SCT symptoms in ADHD and how these symptoms fit into the ADHD nosology (Barkley, 2012c, 2013, 2014).

One reason for the renewed interest is that SCT may explain diagnostic heterogeneity among people with ADHD-PI. As we previously argued, people diagnosed with ADHD-PI fall into one of two diagnostic categories: (1) those who endorse six or more inattentive items but are marginally below the diagnostic threshold in the hyperactive–impulsive symptom cluster, and actually represent just subthreshold ADHD-C-type cases, and (2) those who endorse six or more inattentive items and have few or no hyperactive–impulsive items. We have argued that the former group suffers from the same underlying pathology as individuals with ADHD-C, whereas those in the latter group have a qualitatively distinct disorder that appears to be characterized by SCT (Milich et al., 2001). It follows that a set of criteria including SCT symptoms would better differentiate between the subtypes and improve subtype stability.

Although there is a relative dearth of research on SCT compared to inattentive and hyperactive–impulsive symptom dimensions in ADHD, emerging evidence indicates that people with SCT only and ADHD + SCT show different patterns of impairment and comorbidities than those with ADHD only (Barkley, 2012c, 2013, 2014). For example, Carlson and Mann (2002) compared children with ADHD-PI, with and without SCT, and found that those with SCT had fewer externalizing problems and were rated by teachers as being more anxious and depressed, and as having more social dysfunction than those without SCT. Other studies have shown that the SCT symptom dimension is more strongly associated with internalizing symptoms than are the DSM-IV ADHD symptom clusters (Bauermeister, Barkley, Bauermeister, Martinez, & McBurnett, 2012; Garner, Marceaux, Mrug, Patterson, & Hodgens, 2010). In summary, there is substantial empirical evidence that SCT is a statistically valid clinical entity with different external correlates than DSM-IV ADHD, at least in children (Barkley, 2014; Becker, 2013). There is very little research comparing long-term outcomes of people with SCT symptoms and those with ADHD; however, Barkley (2012c) recently examined adults with ADHD only, with SCT only, and with ADHD + SCT. He found that that SCT was associated with more psychosocial impairment, particularly in the areas of employment and education. He also found that, unlike ADHD, SCT was not associated significantly with EF deficits in everyday life (ratings) once the overlap of SCT with ADHD symptoms was statistically controlled (Barkley, 2012c, 2013). This suggests that whereas ADHD is linked to substantial and pervasive EF deficits in everyday life, SCT is not. An important future direction for research will be to characterize SCT in terms of course, outcome, and treatment response in comparison to inattention and hyperactivity–impulsivity. For more on SCT, see Chapter 17.

SITUATIONAL AND TEMPORAL VARIATION IN ADHD SYMPTOMS

People's ADHD symptoms can become more or less pronounced as a function of their environment and/or the demands placed on them. As a clinical example, consider a hyperactive boy during an afternoon at school. During recess, his high level of activity would be appropriate for the situation and not unlike the activity level of his classmates. A casual observer may not be able to differentiate the boy with ADHD from his typically developing peers in this setting. Consider the same child when he returns to class. As the situation changes and his classmates begin to reign in their activity levels, the boy with ADHD may continue to exhibit hyperactivity. In this setting, his symptoms become more apparent and, more importantly, disruptive to himself and his classmates. This example describes a common observation that the behavioral problems demonstrated by children with ADHD are not equally present across different settings (Landau & Milich, 1988). When children are playing alone, when they are washing and bathing, and when the father is at home are less troublesome situations for children with ADHD, whereas instances when they are asked to do chores, when a parent is on the telephone, when visitors are in the home, or when they are in public places may be times of peak symptom severity (Porrino et al., 1983). In this section we review how common situational factors can influence the severity of ADHD symptoms in affected individuals.

Level of Environmental Demand for Inhibition

Some of the factors determining this variation have been delineated. One of these—the extent to which caregivers demand that children with ADHD restrict their behavior—appears to affect the degree of devi-

ance of these children's behavior compared to that of nondisabled children. In free-play or low-demand settings, children with ADHD are less distinguishable from typical children than they are in highly restrictive ones (Barkley, 1985). Related to this issue of setting demands is the effect of task complexity on children with ADHD. The more complicated a task, and hence its greater demands for planning, organization, and executive regulation of behavior, the greater the likelihood that children with ADHD will perform more poorly than nondisabled children (Marzocchi, Lucangeli, De Meo, Fini, & Cornoldi, 2002). Clearly, the symptoms of ADHD are most disabling when the demands of the environment or task exceed a child's capacity to sustain attention, resist distractions, regulate activity, and restrain impulses. Children with ADHD appear less deviant and certainly are viewed by others as less troublesome in environments that place few or no demands on their behavioral faculties than in settings or tasks that place high demands on these abilities.

Novelty and Task Stimulation

Children with ADHD display fewer behavioral problems in novel or unfamiliar surroundings or when tasks are unusually novel, but their level of deviant behavior increases as familiarity with the setting increases (Zentall et al., 1985). We would expect to find that the behavior of these children is rated as far better at the beginning of the academic year, when they are presented with new teachers, classmates, classrooms, and sometimes even school facilities. However, we would expect their behavioral control to deteriorate over the initial weeks of school. Even academic performance among children with ADHD can be improved by novel material. Beike and Zentall (2012) demonstrated that story novelty improved attention and recall of children with ADHD. In fact, although these children performed much more poorly than nonclinical children when reading a familiar story, their performance was indistinguishable from the nonclinical group when the story was novel. Situational novelty also can influence children's behavior outside of school. A common clinical example is when children with ADHD visit grandparents they see infrequently, who are likely to provide them with considerable one-to-one attention. In this type of novel and stimulating environment, one can expect a temporary reduction in children's symptomatology.

The degree of stimulation in the task also seems to be a factor in the performance of children with ADHD. Research suggests that highly stimulating educational materials are more likely than relatively low stimulation to improve the attention of these children (Lee & Zentall, 2002). Likewise, children with ADHD make fewer impulsive choices in a high-stimulation environment (Antrop et al., 2006) and are better able to inhibit behavioral impulses while performing a stimulating task (Shaw, Grayson, & Lewis, 2005). It should be noted, however, that the beneficial effects of stimulation only occur when the task itself is highly engaging. If environmental features outside of the task are stimulating, then these stimuli can compete for children's attention and degrade their performance, perhaps because the children are distracted by these highly salient objects (Landau et al., 1992; Lee & Zentall, 2002).

An important clinical application of this finding is that children with ADHD may not misbehave in novel settings, such as a physician's or psychologist's office. It is important to consider that such a setting is relatively new for the child, so his or her symptoms may not manifest until the novelty of the new situation wears off.

Caregiver Effects

Children who have ADHD appear to be more compliant and less disruptive with their fathers than with their mothers (Tallmadge & Barkley, 1983). They are certainly rated routinely as manifesting lower levels of symptoms by their fathers than by their mothers (DuPaul et al., 1998). There are several possible reasons for this. For one, mothers are still the primary custodians of children within the family, even when they are employed outside the home; therefore, they may be the ones who are most likely to make demands that tax or exceed the children's limitations in the areas of persistence of attention, activity regulation, impulse control, and rule-governed behavior. Getting children to do chores and schoolwork, perform self-care routines, and control their behavior in public remain predominantly maternal responsibilities. As such, mothers may be more likely than fathers to witness their children's ADHD symptoms (but see Webster-Stratton, 1988).

Another reason may be that mothers and fathers tend to view and respond to inappropriate child behavior somewhat differently. Mothers may be more likely to reason with children, to repeat their instructions, and to use affection as a means of governing child compli-

ance. Fathers seem to repeat their commands less, to reason less, and to be quicker to discipline children for misconduct or noncompliance. The larger size of fathers and their consequently greater strength, among other characteristics, may also be perceived as more threatening by children and are therefore more likely to elicit compliance in response to commands given by fathers. For whatever reason, the greater obedience of children with ADHD to their fathers than to their mothers is now well established. This should not necessarily be construed as a sign that a child does not actually have ADHD or that the child's problems are entirely the result of maternal mismanagement.

Timing and Magnitude of Consequences

Settings or tasks that involve a high rate of immediate reinforcement for compliance with instructions result in significant reductions in, or in some cases amelioration of, attention deficits (reviewed in Luman, Oosterlaan, & Sergeant, 2005). Differences in activity level between groups with and without hyperactivity while watching television may be less than in other activities, whereas such differences are substantially evident during reading and math classes at school (Porrino et al., 1983). Children with ADHD seem to prefer immediate to delayed rewards (Sagvolden, Aase, Zeiner, & Berger, 1998). It seems that when children with ADHD are engaged in highly reinforcing activities, they may even perform at levels close to those of typical children; however, when the schedule and magnitude of reinforcement are decreased, the behavior of these children may become readily distinguishable from that of typical children (Barkley, Copeland, & Sivage, 1980). Such dramatic changes in the degree of deviant behavior as a function of motivational parameters in the setting have led several scientists to suggest that ADHD involves a problem in the manner in which behavior is influenced by environmental contingencies (Johansen et al., 2002).

A situational factor related to motivation appears to involve the degree of individualized attention provided to the children with ADHD. During one-on-one situations, these children may appear less active, inattentive, and impulsive, whereas in group situations, where there is little such attention, the children may appear at their worst. For instanceresearchers (Draeger, Prior, & Sanson, 1986; Steinkamp, 1980) found that children with ADHD perform better on cognitive and academic tasks when they perform the tasks with an adult in the room. Both factors (response consequences and individualized attention) are often incorporated as treatment recommendations into home and school management programs (see Part III).

Fatigue

Fatigue or time of day (or both) may have an impact on the degree of deviance of ADHD symptoms. Zagar and Bowers (1983) studied children with ADHD in their classrooms and found that they performed significantly better on various problem-solving tasks in the morning, whereas their classroom behavior was significantly worse in the afternoon. These changes in behavior with time of day did not appear to be a function of boredom or fatigue with the task, as efforts were made to counterbalance the order of administration of the tests across mornings and afternoons. Performance in the afternoon was routinely worse, whether it was the first or second administration of the task. However, general fatigue (defined simply as time since the last resting or sleeping period) might still explain these results.

This is not to say that differences between children with and without hyperactivity do not exist in the early morning but emerge only as time of day advances. Antrop and colleagues (2005) compared time-of-day effects on noisiness and other disruptive behaviors in a group of children with ADHD and a group of comparison children. Both groups showed similar increases in out-of-seat behavior during the afternoon, and the hyperactive children were more often out of their seats than controls, regardless of time of day. Given their normal susceptibility to time-of-day effects, however, clinicians might expect children with ADHD to show increased rates of disruptive behavior during the afternoon. Likewise, educators would do well to schedule overlearned, repetitive, or difficult tasks that require the greatest powers of attention and behavioral restraint for morning periods, while reserving recreational, entertaining, or physical activities for the afternoon (Zagar & Bowers, 1983). Such findings challenge the seemingly common practice of scheduling homework periods for children with ADHD in the late afternoon or early evening. Furthermore, they highlight the importance of sleep hygiene in children and adolescents with ADHD, because these individuals often report sleep-related problems that may increase their problems associated with fatigue (Konofal, Lecendreux, & Cortese, 2010).

PREVALENCE

The current consensus of expert opinion is that approximately 5.0% of children and 2.5% of adults have ADHD (American Psychiatric Association, 2013). On what is this based? A multitude of published studies has attempted to estimate the prevalence of ADHD in the general population. The answer to this question, however, depends largely on the methods used in each study. Factors affecting estimates include diagnostic method, how ADHD is defined, whether or not the functional impairment criterion is considered, and geographic location of the survey (Polanczyk et al., 2007). We begin this section with a discussion of how different methodological decisions in studies can affect prevalence estimates and conclude with a review of studies estimating the prevalence of ADHD in the population.

Defining Deviance

A problem in establishing prevalence has always been deciding what cutoff point is needed along the dimension or distribution of ADHD symptoms to determine that a person's behavior is sufficiently deviant from the norm to justify a diagnosis. Some have used the criterion of 1.5 standard deviations above the mean for nondisabled children on parent or teacher rating scales of ADHD symptoms; however, this cutoff may overestimate the number of people who meet criteria for the disorder. One study using this definition classified 14% of the population as hyperactive (Trites, Dugas, Lynch, & Ferguson, 1979). Other investigators have used the cutoff of 2 standard deviations above the mean and provided a more realistic prevalence estimate of 2–9%.

Applying such a stringent statistical criterion as 2 standard deviations is consistent with tradition in defining other conditions (e.g., learning disabilities and intellectual disability) as deviant. It also ensures that an excessive number of children are not being given a psychiatric diagnosis and reserves the diagnosis for the most severely afflicted. When the 2 standard deviations cutoff is used, children who are identified are extremely impaired, and this impairment is stable over time (Barkley, Fischer, Smallish, & Fletcher, 2002). Although it may be preferable to utilize a conservative definition of a disorder in a research setting, it would be prudent to use a lower cutoff (i.e., 1.5 standard deviations above the mean) in a clinical setting, where it may be better to err on the side of caution.

The real issue, though, is the following: At what level of developmental deviance is impairment in one or more major life activities likely to be evident? In short, disorder begins where impairment begins. If all or nearly all of the children exceeding the 93rd percentile have evidence of impairment, we can rest assured that this cutoff score is diagnostically meaningful. If not, then the threshold needs to be set either higher or lower, until we find a threshold that achieves this purpose. And so, while the actual number chosen as the diagnostic threshold may be a bit arbitrary, it is hardly meaningless. The further above this threshold a child or adult scores, the greater the likelihood that he or she will experience impairment in major life activities and the more such activities are likely to be impaired. Establishing the presence or absence of functional impairment should be the main focus of the diagnostic process. It is fine to view cutoff scores with flexibility when patients show functional impairment alongside their symptoms.

Prevalence of ADHD in Children

Estimates from Symptom Rating Scales and Clinical Diagnostic Criteria

There is an enormous literature on the attempt to estimate the prevalence of ADHD, using various research methodologies, in children in different geographical regions. We provide in this section an overview of this literature by reviewing meta-analyses as well as selected high-quality, individual studies on the prevalence of ADHD in children.

First it is important to consider how cases are identified. Perhaps the most common method for estimating the prevalence of ADHD is to use a parent or teacher rating scale of the symptoms of the disorder, then survey large populations of children. In these studies, children who score above the DSM (or ICD) diagnostic threshold are considered to meet criteria for the disorder. An advantage to this approach is that data for large samples of children can be collected quickly. Because such an approach does not incorporate other important criteria relevant to a diagnosis, however, the prevalence figures it may yield are probably overestimates, if only because the approach does not invoke an impairment criterion. Such scales are useful in screening for disorder and suggesting an upper limit to prevalence, but alone they do not define the true prevalence of ADHD.

Other studies use clinical interviews in addition to parent- and teacher-report measures. Although these studies are typically more difficult to conduct, they are able to more rigorously apply the full diagnostic criteria by assessing for functional impairment and interviewing respondents. Because DSM-5 criteria also require early onset of symptoms (before age 13), pervasiveness across settings, the exclusion of other disorders, and impairment in one or more major domains of life functioning, prevalence estimates produced by these studies are likely closer to the "true" prevalence of ADHD in the general population than are estimates produced by other methods.

Several meta-analyses have tested how much diagnostic method (e.g., rating scale vs. clinical interview) influences prevalence estimates. Polanczyk and colleagues (2007) conducted a meta-analysis of 102 studies and found that one of the best predictors of heterogeneity in prevalence estimates is whether the impairment criterion was included in the diagnostic decision. Other individual studies have shown that drastic changes in prevalence emerge depending on whether the presence of clinical impairment is required for a diagnosis. Wolraich, Hannah, Baumgaertel, and Feurer (1998) found a prevalence rate of 16.1% in a group of schoolchildren by using symptom count criteria, but the reported prevalence rate dropped to 6.8% when functional impairment was required for a diagnosis. Other authors found significant, albeit less pronounced, drops in prevalence estimates when functional impairment is required for diagnosis (Jensen et al., 1995; Simonoff et al., 1997). Clearly, assessing for functional impairment is an important step in producing accurate prevalence estimates.

Another methodological factor that can influence prevalence estimates is the source of information upon which diagnostic decisions are made. Many studies gather information on children's behavior from only a single caregiver; other, more comprehensive studies use ratings from multiple sources (e.g., parent and teacher reports). Other studies that estimate prevalence in older children, adolescents, and adults do so by gathering self-reported symptom counts. Not surprisingly, the source of information influences prevalence estimates (Polanczyk et al., 2007). Prevalence estimates based on parent and teacher ratings are generally comparable (Magnusson, Smari, Gretarsdottir, & Prandardottir, 1999), although this is not always true. For example, Breton and colleagues (1999) reported prevalence estimates of 5.0% based on parent report and 8.9% based on teacher report. Prevalence estimates based on self-reported symptoms from older children and adolescents are typically lower than those based on caregiver report. Romano and colleagues (2001) reported a prevalence of 3.3% in a sample of adolescents based on parent report, but this prevalence rate dropped to 0.6% when the estimate was based on participants' self-reported symptoms. This is consistent with the finding that children and adolescents have trouble accurately reporting on their own behavior (Owens, Goldfine, Evangelista, Hoza, & Kaiser, 2007).

The prevalence of ADHD also appears to differ significantly as a function of how many people must agree on the diagnosis. This is not entirely surprising; it is almost always more likely for a single condition to be satisfied (i.e., meeting criteria according to a single rater) than for two independent conditions to be satisfied (i.e., meeting criteria according to two raters). This is especially true given that ratings of children's behavior from different caregivers are only modestly correlated. Lambert, Sandoval, and Sassone (1978) asked parents, teachers, and physicians of 5,000 children in elementary school to identify the children they considered to be hyperactive. Approximately 5% of these children were defined as hyperactive when the opinion of only one of these caregivers (parent, teacher, physician) was required—a prevalence figure close to official population estimates (Polanczyk et al., 2007). However, this prevalence figure dropped to approximately 1% when agreement among all three sources was required.

There are similar differences in prevalence estimates in studies that conduct evaluations in a single setting and those performing evaluations across multiple settings (e.g., school, home). Perhaps unsurprisingly, studies that require symptoms across more than one setting typically produce lower prevalence estimates than those assessing impairment in a single setting (Skounti, Philalithis, & Galanakis, 2007). This finding may be influenced by the fact that gathering evaluations across multiple settings is often operationalized as gathering data from different caregivers (e.g., parents report on behavior at home, and teachers report on behavior at school). As previously discussed, the poor agreement among caregivers sets a modest upward limit of prevalence when diagnosis is contingent on both sources.

The age range of the sample can influence prevalence estimates, with older samples producing lower prevalence estimates. Polanczyk and colleagues' (2007)

meta-analysis examining differences between children and adolescents reported estimates of approximately 3% for adolescents and 6% for children. As previously discussed, the declining prevalence of ADHD with age may be partly an artifact of the DSM items' being chiefly applicable to young children, although this may change with the age-related adjustments to the DSM-5 set of criteria.

Geographic location appears to have an effect on prevalence estimates (Faraone, Sergeant, Gillberg, & Biederman, 2003). Although the majority of prevalence estimates were conducted in the United States and Europe, there has recently been a surge of studies from other parts of the world. Meta-analysis suggests that there is no significant difference in prevalence rates among North America (6%), Europe (5%), South America (12%),[2] Asia (4%), or Oceania (5%). Estimates from the Middle East (3%) are significantly lower than those in the United States, and estimates from Africa (8%) are higher than those in the United States (Polanczyk et al., 2007).

Finally, characteristics of the community from which the sample was drawn can influence prevalence estimates. Although more representative sampling techniques (e.g., multistage sampling; Kessler et al., 2006) produce more generalizable prevalence estimates, such studies are logistically challenging. Many studies sample from limited geographic areas, such as a single metropolitan area or even a single school. There is mixed evidence regarding the relation between socioeconomic status (SES) and ADHD: Some studies report that children in lower SES settings are more likely to show hyperactivity (Boyle & Lipman, 2002), whereas other studies show no such relation (Szatmari, 1992). One reason for these discrepant findings may be that whereas some studies control for comorbid disorders, others do not. For example, Szatmari (1992) found that a relation between ADHD symptoms and SES disappeared when he controlled for comorbid conduct disorder (CD) symptoms. Whether or not it is appropriate to control for CD symptoms in this type of analysis is a debatable point (Beauchaine, Hinshaw, & Pang, 2010), so the nature of the relation among SES and ADHD and CD prevalence rates remains an unresolved issue.

Prevalence Determined by Cases Identified in the Community

A somewhat different approach to estimating the prevalence of ADHD is to review school records to determine the percentage of children identified to schools as having a clinical diagnosis of ADHD. There are serious flaws in such an approach, not the least of which is that schools may not be told by either parents or professionals that children have received the diagnosis. It is also quite possible that a child may have the disorder but never have been referred or diagnosed. In either scenario, the school records would miss detecting such cases. As such, studies that examine the number of cases in schools should not be considered measures of the true population prevalence of ADHD. These data, however, can be used to answer a different question: How often are children and adolescents being diagnosed with ADHD in the community? The number of children identified as having ADHD is more fluid and sensitive to social forces than is the "true" prevalence rate of the disorder. For example, in 1992 the U.S. Department of Education released a memorandum stating that students with ADHD should be provided with access to special education services. Although this policy had no effect on the actual prevalence of ADHD in the population, it almost certainly increased the number of children being identified as more parents sought evaluations for their children so they could receive additional services.

Given recent concerns about overdiagnosis of ADHD among school-aged children, these studies are important in their own right. In an initial study that raised concern about overdiagnosis of ADHD, LeFever, Dawson, and Morrow (1999) reported prevalence rates of ADHD in two southeastern Virginia school districts that were two to three times higher than the DSM-IV-based cited average prevalence range of 3–5% (American Psychiatric Association, 1994). LeFever and colleagues examined school-identified cases to estimate prevalence of ADHD in grades 2–5. In addition to these higher rates than those based on DSM-IV criteria, the researchers found a disproportionate percentage of white males (18–20%) diagnosed with ADHD in both districts. The results of the LeFever and colleagues study were touted by its authors and others (see Timimi, 2004) as evidence of ADHD overdiagnosis. The LeFever and colleagues study was also touted as evidence of overuse of psychostimulant medication to manage behavioral concerns (see Chapter 27). The researchers found a prevalence for stimulant treatment, as identified in school records, ranging from 7 to 10%, among the highest found anywhere in the United States.

The results of the LeFever and colleagues (1999) study are questionable, and a subsequent study attempting to replicate these findings failed to do so. In an un-

published study, Tjersland, Grabowski, Hathaway, and Holley (2005) attempted to replicate and extend the work of LeFever and colleagues in the same region of Virginia. They also used cases of ADHD identified in school records to determine prevalence of disorder and of psychostimulant treatment in a school district adjoining the two studied by LeFever and her colleagues. This district is highly similar, if not identical, demographically to those studied by LeFever and colleagues. Several sources of information were reviewed, including student information cards that contained medical history and listed ADHD as a known condition, if applicable. Information was also collected from an inspection of physician forms authorizing medication treatment at school. A second phase of the data collection involved a review of student cumulative files. Record reviews of student files were conducted for students with Section 504 plans and for students receiving special education services for learning disabilities, emotional disturbances, developmental delays, or the "other health impaired" category. As a consequence, this study was a more comprehensive review of school records than that undertaken by LeFever and colleagues; therefore, Tjersland and colleagues should have identified as many, if not more, cases of ADHD and stimulant treatment than did LeFever and colleagues.

The results of this second study completely contradicted the LeFever and colleagues (1999) results. The study found a prevalence rate for ADHD of just 4.4%, closely matching the DSM-IV-based estimated prevalence, as well as the average of studies using clinical diagnosis (to be reviewed below). The study also found that 4% of the children were being treated with psychostimulant medication—well below the 7–10% figure cited by LeFever and colleagues. Data on prevalence at each grade level from 1 through 12 indicated that the highest prevalence rates were evident in fourth (7.1%) and fifth (6.3%) grades. LeFever and colleagues claimed that national averages obscure the "clear overdiagnosis" of ADHD in some groups. They suggest that one in every three white elementary school boys is being diagnosed with the disorder in southeastern Virginia. The data from the Tjersland and colleagues (2005) study did not replicate this finding; it found that only 8.1% of white males received the ADHD diagnosis, based on the school records.

Although the LeFever and colleagues (1999) study probably overestimates the rate of ADHD diagnosis in school-age children, subsequent large-scale studies have confirmed that in some cases the rates of ADHD diagnosis observed in schools is higher than would be expected given the estimated population prevalence of the disorder. The Centers for Disease Control and Prevention estimated that 9.5% of children had at some point been diagnosed with ADHD, as reported by parents(Centers for Disease Control and Prevention, 2010). Boys (13.2%) were more likely to have received a diagnosis than girls (5.6%). Children from Southern states (e.g., Kentucky, 12.8%) are diagnosed at a much higher rate than children in other regions (e.g., California, 6.2%). More recent data collected by the Centers for Disease Control and Prevention analyzed and released in the *New York Times* estimated that 11% of high school-age children had received a diagnosis of ADHD at some point in their lives (Schwarz & Cohen, 2013). Among high school–age boys (ages 14–17) the rate of diagnosis was 19%. It is difficult to judge the veracity of these claims without knowing methods used by the *New York Times* to produce these figures, but their results are nonetheless provocative. Clearly the rate at which children are being diagnosed with ADHD exceeds the population prevalence of the disorder, particularly among high school-age boys. The gap between the true population prevalence of ADHD and diagnostic rates appears to be increasing over time (Centers for Disease Control and Prevention, 2010; Getahun et al., 2013). The forces driving the increasing rates of ADHD diagnosis are not entirely understood, but they should become more apparent as research is done to address this problem. In the meantime, clinicians should use well-validated assessment techniques to ensure that children meet criteria for ADHD before diagnosing them.

Prevalence of Adult ADHD

Since publication of the previous edition of this book there has been a large amount of research examining the prevalence of ADHD among adults. The most comprehensive study to date was conducted by Kessler and colleagues (2006), using a sample of 3,199 adults. This study combined a well-controlled population sampling approach (i.e., the National Comorbidity Survey Replication) and in-person clinical interviews to establish functional impairment among those who screened positive during a telephone interview. This study estimated a population prevalence of 4.4% among adults ages 18–44 years. Barkley (2012c) recently found a prevalence of approximately 5% in his large representative sample of U.S. adults. A meta-analysis

of smaller studies produced a lower estimate of 2.5% (Simon, Czobor, Balint, Meszaros, & Bitter, 2009), although the Kessler colleagues (2006) study was not included in their estimate due to methodological issues. A related meta-analysis examined the cross-national prevalence of adult ADHD (Fayyad et al., 2007) using methodology similar to that just described to estimate prevalence rates in Belgium (4.1%), Colombia (1.9%), France (7.3%), Germany (3.1%), Italy (2.8%), Lebanon (1.8%), Mexico (1.9%), Netherlands (5.0%), and Spain (1.2%). This study shows that the prevalence of ADHD in adults is relatively similar across cultures.

CONCLUSION

We have demonstrated in this chapter that ADHD is a legitimate mental health disorder whose diagnostic criteria are based on a substantial body of empirical evidence from hundreds of scientific studies. The fact that debate will always exist concerning the precise criteria that should be used to diagnose the condition reflects not so much a mistaken view that ADHD is some vague or invalid condition or myth as an evolving consequence of the scientific process itself applied to ADHD as earlier findings are further tested and revised as a function of later findings that produce an increasingly refined and rigorous set of criteria for disorder recognition. The symptoms of ADHD cohere into two highly related dimensions of behavior, and those symptoms are substantiated by various studies employing objective measures that demonstrate that children with ADHD do, in fact, display more such behavior than do children without ADHD. While most cases of ADHD are evident by age 12, some continue to emerge thereafter, in part as a consequence of the unreliability of retrospective recall of symptom onset, and because of the creation of new cases of ADHD in the population as a result of the accrual of new cases of neurological injuries that may give rise to new cases of ADHD over time. The symptoms of ADHD, particularly the ADHD-HI dimension, decline with age but remain present across the life course (Barkley, 2011a) and support the view that it is a lifelong disorder. And while boys are significantly more likely to manifest ADHD than girls by an average ratio of 3:1, this sex difference declines by adulthood to a nearly equal representation in both sexes. ADHD is a universal condition found in all countries and cultures studied to date with relatively consistent prevalence.

KEY CLINICAL POINTS

- ✓ ADHD is a common and well-recognized behavior disorder that affects millions of children, adolescents, and adults.
- ✓ The diagnostic criteria used to recognize ADHD (e.g., in DSM-5) are the most scientifically validated to date and are based on hundreds of studies, as well as expert consensus opinion.
- ✓ DSM-5 no longer conceptualizes ADHD as comprising three separate types; instead, it is presented as a single disorder that can vary in the population in each of its two symptom dimensions, and those relative severities can change over time, leading to the creation of three kinds of "presentations" of ADHD rather than subtypes.
- ✓ Although the symptom lists for ADHD in DSM-5 remain the same, qualifier symptoms have been added to help clinicians understand the expression of that symptom at older ages beyond childhood.
- ✓ The age of onset for ADHD has been adjusted upward to age 12 in DSM-5 but remains primarily a rough guideline to follow rather than a firm demarcation in what is otherwise the "shifting sands" of development. So long as cases meet all other criteria for the disorder, clinicians should be flexible in imposing an age of onset, recognizing that recall of onset is unreliable.
- ✓ The symptom threshold for diagnosing ADHD has now been reduced from 6 to 5 in DSM-5 for application to adults so as to retain the sensitivity of the diagnostic criteria to the detection of the disorder. Other research suggests that a threshold of 4 is even better.
- ✓ The disorder manifests itself across multiple settings and leads to impairment in various domains of major life activities.
- ✓ The behavioral symptoms used for diagnosis are not only substantiated in parent and teacher reports of these symptoms in children and self- and other-reports in adults, they are further confirmed by direct observational methods that demonstrate them to be excessive in people diagnosed with the disorder.
- ✓ ADHD severity may fluctuate somewhat across settings and time of day, and as a consequence of various factors in the situation, such as the schedule of consequences for behavior, novelty, adult supervision, and other factors.

- ✓ Although ADHD is presented as a categorical condition in DSM taxonomy, it is actually a dimensional disorder whose symptoms vary in severity across the entire human population and may become a disorder (produce impairment) at excessive levels of severity.
- ✓ The prevalence of ADHD in children appears to be on average between 5 and 7%, whereas in adults it ranges from 3 to 5%.
- ✓ The disorder is universal, having been identified in every country and culture in which it has been studied.

NOTES

1. Data from the DSM-IV field trials indicated that a diagnostic threshold of five, rather than six, hyperactive–impulsive symptoms best differentiated between impaired and nonimpaired children. However, the work group decided to use the more conservative threshold of six symptoms in order to "minimize the identification of normally active children as exhibiting attention deficit hyperactivity disorder" (Lahey et al., 1994, pp. 1676–1677), perhaps to make the resulting diagnostic rates more palatable for the general public. This decision documents how the DSM criteria are not infallible and are partly political in nature, so clinicians should approach diagnosis with some flexibility and avoid viewing these criteria as dogma.
2. Although the prevalence estimate for South America appears to be much higher than the estimates for other regions, Polanczyk and colleagues' meta-analysis found that the difference was not significant. Prevalence estimates from this region were highly variable, so the accuracy of the 12% estimate is suspect.

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CHAPTER 3

Emotional Dysregulation Is a Core Component of ADHD

Russell A. Barkley

As the previous chapter has nicely documented, for over 40 years, clinical descriptions of the core nature of attention-deficit/hyperactivity disorder (ADHD) have focused exclusively on the two-dimensional structure of the disorder cited in clinical diagnostic criteria (inattention, hyperactivity–impulsivity), such as those in the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-III, DSM-III-R, DSM-IV, DSM-5; American Psychiatric Association, 1980, 1987, 1994, 2013). As noted in DSM-5, the essential feature of ADHD is a persistent pattern of inattention and/or hyperactivity–impulsivity that is more frequent and severe than is typically observed in individuals at a comparable level of development. Such official descriptions concerning the core nature of the disorder and the related symptom lists focus exclusively on a two-dimensional structure as being the central features of the disorder. Here I argue that this overemphasis on the most observable and objectively measurable features of the disorder has led to the exclusion or a deemphasis of another feature of the disorder that is just as central to its understanding yet considerably more difficult to quantify. This largely neglected element is a deficiency in both the effortful (executive or cognitive) inhibition and the top-down self-control of emotions in general and particularly those pertaining to the self-regulation of frustration, impatience, and anger. The most noticeable and initial consequence of this deficiency in people with ADHD is a striking propensity for failure to inhibit emotions, or emotional impulsivity (EI).

EI refers to the quickness or speed with which, and the greater likelihood that an individual will react with primary (particularly negative) emotions in response to events compared to others of the same developmental level or age. It is not the same as emotional intensity, which itself can vary across individuals and is not thought to be a problem in those with ADHD. The primary emotional reactions of those with ADHD are not so much more intense initially as they are less moderated by conscious, effortful executive self-regulation of those emotions. Others may have felt the same emotionally intense reaction to an event but are less likely to display the primary emotional behavior associated with it before moderating its expression and even generating secondary emotional states to counteract or supplant the initial primary ones.

The related component to emotional inhibition is emotional self-regulation, which represents a conscious, "top-down" and effortful (executive) moderation of the initial emotional reaction. Like Melnick and Hinshaw (2000), I defer to Gottman and Katz (1989) for their helpful definition of emotion regulation as the

ability to "(a) inhibit inappropriate behavior related to strong negative or positive emotion, (b) self-soothe any physiological arousal that the strong affect has induced, (c) refocus attention, and (d) organize for coordinated action in the service of an external goal" (p. 373). Its opposite is referred to here as deficient emotional self-regulation (DESR). Notice that the definition incorporates emotional inhibition as the first step in emotional self-regulation, which is consistent with the view to be taken here and that of my theory of executive functioning in ADHD (Barkley, 1997a, 1997c, 2012b). Yet it will prove helpful in the following discussion to identify them separately as EI and DESR, with the understanding that the former is the initial step contained within the latter. That is because one cannot self-soothe or otherwise moderate one's initial emotional reactions to events if he or she has not first inhibited the impulsive expression of those initial reactions. EI therefore interferes with subsequent efforts to engage in emotional self-regulation. It is also helpful to separate them because most of the available research in ADHD and emotions seems to be directed at documenting the first (EI) more than the second (DESR).

This chapter examines the importance of EI–DESR in understanding the nature of ADHD. It also seeks to explain why the explicit recognition of EI–DESR as a central feature of the disorder may prove useful in broadening our understanding of the emotional and social maladjustment evident in cases of the disorder. Apart from providing a more accurate clinical portrayal of the disorder for both greater professional and public understanding, this argument stresses that formally acknowledging the place of EI–DESR has a sound basis in (1) the history of ADHD; (2) the neuropsychological nature of the disorder; (3) the neuroanatomy of ADHD; (4) the empirical research on the symptoms of EI–DESR in ADHD; (5) the linkage of ADHD with oppositional defiant disorder (ODD), among other comorbidities; and (6) the concurrent and later impairments often seen in ADHD, especially in the domains of social functioning. Others have also argued that emotional dysregulation is an important component of ADHD based on lines of evidence other than those I summarize here (Martel, 2009; Skirrow, McLoughlin, Kuntsi, & Asherson, 2009).

Before addressing these issues, it is crucial for the arguments to be made here that the reader understand that this chapter addresses the nature of ADHD (previously, the combined type in DSM-IV) and not that group of inattentive subjects who have come to be characterized in research articles as having sluggish cognitive tempo (SCT; see Chapter 17; also see Barkley, 2012b, 2013, 2014; Milich, Balentine, & Lynam, 2001; special issue of the *Journal of Abnormal Child Psychology*, 2014) or what I have recently renamed as concentration deficit disorder (CDD; Barkley, 2014; Saxbe & Barkley, 2014). Individuals having SCT/CDD differ in important ways from those with ADHD (Milich et al., 2001; Saxbe & Barkley, 2014). This has led some researchers in the field to view it as a separate disorder from ADHD (Barkley, 2001, 2014; Diamond, 2005; Milich et al., 2001; see Chapters 2 and 17) or at least as a qualitatively different type of the disorder. And it has led theorists to exclude those with SCT from their efforts to build working models of ADHD and its underlying neuropsychology (Barkley, 1997c, 2012b, 2013, 2014; Castellanos, Sonuga-Barke, Milham, & Tannock, 2006; Nigg & Casey, 2005; Sagvolden, Johansen, Aase, & Russell, 2005). I do not focus on SCT/CDD in this review because it is the focus of Chapter 17. That is because SCT/CDD appears to include a distinct attention problem but no affiliation with the impulsive and poorly inhibited behavior inherent in ADHD (Barkley, 2012b, 2013, 2014; Diamond, 2005; Milich et al., 2001).

As I discuss below, it is especially this impulsive aspect of ADHD that provides a strong link between ADHD and EI–DESR through the disruption of emotional inhibition, item (a) in the definition of DESR, as noted earlier. As Martel (2009) reasoned, the hyperactive–impulsive (HI) dimension of ADHD is associated with a breakdown in the emotionality component (EI) whereas inattention is associated with a breakdown in the regulatory side (DESR). Martel further argued that breakdowns in regulatory control of emotion are a specific signature of ADHD, whereas increased negative emotionality may be nonspecific and shared across the disruptive disorders (ADHD, ODD, and conduct disorder [CD]) accounting for their comorbidity—a point with which I agree, as will be shown later.

THE IMPORTANCE OF EI–DESR IN THE HISTORY OF ADHD

Historical accounts of ADHD (Accardo & Blondis, 2000; Barkley, 1998, 2006, Chapter 1; Goldstein & Goldstein, 1998; Kessler, 1980; Ross & Ross, 1976, 1982; Schachar, 1986; Werry, 1992) have traditionally cited the three published lectures by George Still (1902) as the beginning of medical or scientific interest

in the disorder. However, Barkley and Peters (2012) located and translated a chapter on attention deficits in a German medical textbook by Melchior Adam Weikard in 1775. In his description of this attention disorder that closely resembles ADHD, Weikard noted on several occasions the problem subjects had with emotional dysregulation. Later, a chapter in a medical textbook by the Scottish physician Alexander Crichton (1798) was published on the subject of "diseases of attention" (Palmer & Finger, 2001).

Crichton (1798) saw attention as representing the central feature of one's awareness. It is what one has chosen to concentrate on, at least for the moment, be it an external stimulus or internal thought. This process was seen to be effortful, not automatic, requiring that one actively initiate the action of concentrating one's attention on something. And it was seen as being a volitional or willful activity. Crichton identified several different components of attention, one of which was inconstancy of attention. By this, he seems to have meant the inability to sustain one's attention for an adequate period of time on a particular object of attention, which results in skipping across various things to which one attends, spending little time with each. This component resembles the present concepts of sustained attention and, related to it, resistance to distractibility, both of which are believed to be central to attention deficits in ADHD. Chrichton (1798, p. 272) wrote that "in this disease of attention, if it can with propriety be called so, every imperfection seems to agitate the person, and gives him or her an unnatural degree of mental restlessness."

Crichton's second component of inattention involved the energy or power of the capacity to attend. This likely corresponds to modern notions of arousal and alertness because Crichton (1798) felt that attention could become fatigued or be affected by inadequate mental energy. Perhaps this component corresponds more closely to the nature of the attention problem believed to be involved in SCT/CDD, that of a more passive, sluggish, daydreamy, confused, and lethargic form of inattentiveness. More to the point of this discussion, Crichton observed that individuals prone to inordinate or excessive levels of distractibility often reacted to the myriad distractions in a situation "with a degree of anger that borders on insanity" (p. 272).

Later, Still (1902) noted the connection between problems with attention and difficulties with the control of emotions in his descriptions of subjects thought to have defective moral control of behavior. Still defined the moral regulation of behavior as the voluntary inhibition of behavior and subsequent consideration of one's actions for the good of one's self and the good of others. He described 43 children in his clinical practice who had serious problems with defective moral control, 23 of which were also believed to have impaired intellect (mental retardation). Still reasoned that the 20 cases that could not be attributed to impaired intellect or physical disease arose from a deficit in the voluntary inhibition of behavior, or impulsiveness. He also commented on their impaired capacity for sustained attention. Common to most of these subjects as well was that they were also quite overactive. These are the cases that most closely resemble the ADHD of today. Many were often aggressive, defiant, resistant to discipline, and showed little "inhibitory volition" over their behavior. Still proposed that the immediate gratification of the self was the "keynote" quality underlying all of the attributes of the children. Among all of those attributes, "an extreme degree of passionateness was the most common feature" (p. 1080), or being easily aroused to emotion. This "morbid exaggeration of emotional excitability" (p. 1165) was thought to arise from the same defect in volitional inhibition that characterized the other behavioral attributes of these children. Thus, if Still's cases represent ADHD, which many believe they do, then he was clearly linking the behavior to deficits in the volitional regulation of emotions, or EI–DESR. Indeed, he viewed EI–DESR as the most noteworthy attribute of these cases, not impaired attention or overactivity. Just as important, as I demonstrate later, Still attributed the EI features of cases to subjects' larger problem of volitional or executive (willful, effortful) inhibition rather than to some impaired feature of attention.

In the interim period from the beginning of the 1900s to 1950, little published medical or research interest in children resembled today's notions of ADHD. Certainly articles appeared on the striking deviations in behavior and personality that followed in children who survived the encephalitis epidemic of 1917–1918 and were labeled as having "post-encephalitic behavior disorder" (Ebaugh, 1923; Strecker & Ebaugh, 1924; Stryker, 1925). Most could be characterized as hyperactive, inattentive, impulsive, and easily aroused to emotion, but such children often experienced substantial brain damage, and loss of intelligence and adaptive functioning, to the extent that many of them would today be considered to be mentally retarded. This association of a brain disease with behavioral pathology ap-

parently led early investigators to study other potential causes of brain injury in children and their behavioral manifestations. Birth trauma (Shirley, 1939), lead toxicity (Byers & Lord, 1943), epilepsy (Levin, 1938), and head injury (Blau, 1936; Werner & Strauss, 1941) were studied in children and found to be associated with numerous cognitive and behavioral impairments, including ADHD symptoms. Other terms introduced during this era for children displaying these behavioral characteristics were "organic driveness" (Kahn & Cohen, 1934) and "restlessness" syndrome (Childers, 1935; Levin, 1938). Many of the children in these samples were also mentally retarded or more seriously behaviorally disordered than are children who today are considered to have ADHD. While these children manifested symptoms of ADHD as it is currently conceptualized, they clearly represent an acquired form of the disorder and therefore more closely resemble cases Still spoke about as arising from defective intelligence or as being secondary to physical disease. This is certainly true for a minority of cases of ADHD diagnosed today (Nigg, 2006). Such acquired cases of ADHD-like symptoms underscore the point that those symptoms can arise as a result of damage to particular brain regions, yet they shed little light on just how ADHD could arise in the absence of clear-cut evidence of brain damage.

Notable during this era was also the recognition of the striking similarity between hyperactive children and the behavioral sequelae of frontal lobe lesions in primates (Blau, 1936; Levin, 1938). In frontal lobe ablation studies of monkeys more than 60 years earlier (Ferrier, 1876), the lesions were known to result in excessive restlessness, poor ability to sustain interest in activities, aimless wandering, heightened emotional excitability or reactivity, and excessive appetite, among other behavioral changes. Several investigators, such as Levin (1938), would use these similarities to postulate that severe restlessness in children was likely the result of pathological defects in the forebrain structures, although gross evidence of such was not always apparent in many of these children.

Nevertheless, in the absence of such evidence, the concept of a brain-injured child syndrome was born (Strauss & Lehtinen, 1947) in which the behavioral symptoms of ADHD alone were taken to imply an underlying neurological etiology. The concept would evolve into that of minimal brain damage and further to minimal brain dysfunction (MBD) by the 1960s and 1970s (see Rie & Rie, 1980, for the most thorough treatment of MBD published at that time). The behavioral symptoms of MBD were thought to involve "(1) hyperactivity; (2) impulsiveness; (3) short attention span; (4) perseveration; and (5) *emotional lability*" (Schwalb, 1967, p. 2320, emphasis added). All were thought to be central to the diagnosis of the disorder and characterized the majority of children so diagnosed.

The diagnosis, however, came under severe criticism for being too inclusive (some monographs at the time attributed up to 99 symptoms to the disorder; Clements, 1966) and for lacking sufficient evidence of actual neurological etiologies. Chief among these early critics were Birch (1964), Herbert (1964), and Rapin (1964), who questioned the validity of applying the concept of brain damage to children who had only equivocal signs of neurological involvement, not necessarily damage. Rutter (1977) reviewed the evidence of psychiatric disorders in children with established brain injuries and the inverse evidence of brain damage among children with various psychiatric disorders, concluding "that the behavioural stereotype of the brain damaged child must be firmly rejected" (p. 11). Even so, he went on to state that the hyperactive child syndrome, while not a syndrome of brain damage, was associated with biological factors that "probably often play an important part in its aetiology" (p.12).

The totality of such criticisms led to an increasing preference for the more circumscribed diagnostic term of "hyperactive" or "hyperkinetic child syndrome," an increased focus on identifying and objectively measuring the behavioral features of the disorder, a move away from seeing brain damage as the sole etiology, and hence a decline in the use of the diagnosis of MBD. Despite this shift, the central behavioral tenets of the disorder remained much the same. In a classic article, Laufer, Denhoff, and Solomons (1957) described "hyperkinetic impulse disorder" as involving not only hyperactivity but also short attention span, poor concentration, impulsivity, unpredictable and explosive behavior, and low frustration tolerance.

Similarly, writing about hyperactive children for the widely read popular science magazine, *Scientific American*, Mark Stewart (1970, p. 94, emphasis added) stated that "a child with this syndrome is continually in motion, cannot concentrate for more than a moment, acts and speaks on impulse, *is impatient and easily upset*. At home he is constantly in trouble because of his restlessness, noisiness, and disobedience. In school he is readily distracted, rarely finishes his work, tends to clown and talk out of turn in class and becomes labeled a discipline problem." Citing one of his own studies of the

behavioral symptoms most common to the disorder, Stewart noted that up to 59% manifested difficulties that could be considered signs of EI–DESR including temper tantrums or being defiant, irritable, or impatient. His follow-up study of these children as teenagers indicated that they had changed little in terms of these behavioral characteristics.

Other researchers during this era preferred to focus almost exclusively on hyperactivity as the central feature of the disorder and went about studying the extent and kind of motor activity involved, relegating the other behavioral (attention, impulsiveness) and emotional features of the disorder to associated problems (Keogh, 1971). These included well-known investigators such as Stella Chess (1960), John Werry (1968), and Virginia Douglas (Werry, Weiss, & Douglas, 1964; Werry, Weiss, Douglas, & Martin, 1966), and Robert Sprague (Werry & Sprague, 1970). A plethora of studies in this era objectively measured various aspects of motor activity level (for reviews, see Barkley & Cunningham, 1979; Luk, 1985; Zentall, 1985) and attention span (Barkley & Ullman, 1975; Campbell, 1976; Douglas, 1972). Debates arose over whether the attention problems, distractibility, impulsiveness, and emotional excitability believed to have been involved in earlier notions of hyperkinetic impulse disorder were central to this hyperactive child syndrome or just associated features seen in some cases (Keogh, 1971). Despite this effort of clinical researchers to curtail the concept of hyperactivity to just an excessive degree of movement relative to one's developmental level or age (Chess, 1960), clinicians were not following suit. A survey of pediatricians, teachers, psychologists, psychiatrists, and social workers at the time by Schrager, Lindy, Harrison McDermott, and Wilson (1966) indicated that they continued to identify six features (not one) as characteristic of hyperactive children (fidgety and restless, inattentive, hard to manage, cannot sit still, easily distracted) including *cannot take frustration* (p. 103, emphasis added).

By the 1970s, this effort to isolate the nature of the hyperactive child syndrome to just a single central symptom involving activity level had failed. Reviews at the time continued to emphasize the central features as involving restlessness, distractibility, short attention span, and impulsivity (Barkley, 1981, 1982; Campbell, 1976; Pelham, 1982) in line with the diagnostic criteria espoused in DSM-II and DSM-III (American Psychiatric Association, 1968, 1980). Others, however, continued to include low frustration tolerance, emotional lability, and aggression with these other core features (Cantwell, 1975; Weiss & Hechtman, 1979; Wender, 1971). As Jan Loney (1980, p. 265, emphasis added) memorably phrased it in her own review, "Numerous experts . . . have painted the diagnostic picture of a child in perpetual motion; a child who flits around and blurts out but doesn't finish assignments or chores; *a child with a short and highly flammable fuse*; a child of the present, who neither benefits from the past nor plans for the future."

Even so, a bifurcation was becoming evident during this historical period 1970–1980. There were some clinical researchers, principally psychologists, who saw the disorder as involving mainly problems with hyperactivity, inattention, and poor impulse control (Barkley, 1981; Campbell, 1976; Douglas, 1972; Pelham, 1982). These experts typically placed little emphasis on the problems with emotional self-regulation in their concept of the disorder, seeing it as only an associated feature and evident in only some cases. In contrast, other experts, mainly child psychiatrists, continued to view symptoms of EI–DESR as being just as central to the disorder as the holy trinity noted earlier, including Paul Wender (1971, 1973). Wender saw the symptoms of EI–DESR as involving four forms: increased lability, altered reactivity, increased aggressiveness, and dysphoria. Wender's colleague, David Woods, also include symptoms of EI–DESR as part of the core features of adults with ADHD (Woods, 1986). Likewise, Dennis Cantwell (1975) wrote in his classic book, *The Hyperactive Child*, that the disorder involved emotional excitability as a "core" feature in addition to the emerging customary triad of inattention, hyperactivity, and impulsivity. He stated that "the excitability of the hyperactive child is manifested by temper tantrums and fights over trivial matters, low frustration tolerance and a tendency to become overexcited" (p. 6). He also noted that the majority of these children manifested depression and low self-esteem. And in the United Kingdom, other psychiatrists continued to write about the main features of the disorder as including poor toleration of frustration, temper tantrums, and lability of mood (Sandberg, Rutter, & Taylor, 1978).

As one who began studying ADHD during this era, it has become evident to me in retrospect that several reasons may account for this growing abandonment of symptoms of EI–DESR as being a central feature of ADHD. One reason was its greater difficulty of measurement in laboratory studies attempting to clarify the psychology of ADHD. Research at the time was replete

with various methods for objectively recording activity level, attention, and impulsive behavior. Far less effort was given to measuring the emotional manifestations of the disorder, most likely owing to the fact that emotions are less easily recorded than movement. One need only scan the historically important theoretical research paper by Virginia Douglas (1972) to see this nearly complete abandonment of EI–DESR in ADHD. In an article based on her Presidential Address to the Canadian Psychological Association, Douglas exemplified the largely commendable preoccupation at the time to use objective laboratory measures to document symptoms in this disorder. Her paper contributed heavily to the view that ADHD involves a triad of symptoms (inattention, impulsivity, and hyperactivity), one that did not include emotional excitability, low frustration tolerance, or quickness to anger—encapsulated here as EI–DESR.

The focus on symptom measurement at the time was understandable and laudable. Investigators were striving to put ADHD research on a solid footing of scientific facts that could be objectively recorded and statistically analyzed. One uses what one has measured, and at the time no one had much in the way of measures of emotional self-regulation. Clearly, symptoms of EI–DESR do not lend themselves as well to being measured and analyzed as do activity level, attention, and behavioral impulse control. One exception to this rule was the symptom of aggression, which could be observed and quantified in social interactions and subsequently was found to be a major characteristic of the peer social interactions of ADHD children (Whalen & Henker, 1985). It, along with dimensions of negative temperament (Prior, Leonard, & Wood, 1983), came to be quantified initially via parent and teacher rating scales (Conners, 1969) and later self-ratings (Milich & Okazaki, 1991). Studies using such ratings clearly showed the marked differences between ADHD and control groups in measures of social aggression, task frustration, and mood and intensity. A few investigators succeeded at objectively counting statements of negative emotion during task performance, though often as mere incidental measures in an array of laboratory tasks that focused on the ADHD symptom triad. Yet they were not disappointed to find the difficulties with emotion to be quite divergent between ADHD and control children (Rosenbaum & Baker, 1984). Others used laboratory paradigms involving delay of gratification to index signs of low frustration tolerance, as suggested by Mischel (see Mischel, Shoda, & Rodriguez, 1989). They likewise found success in distinguishing ADHD and control groups (Homatidis & Konstantareas, 1981; Rapport, Tucker, DuPaul, Merlo, & Stoner, 1986). But by then, it was too late to reintroduce problems with emotional self-regulation back into the core features of ADHD, and no one really seemed to have tried to do so. Why?

That answer comes from a second likely reason for the splintering of EI–DESR from the core features of ADHD at the time, and that was clearly the publication and promotion of diagnostic criteria for ADHD in the DSM. Beginning with DSM-II's definition of hyperkinetic reaction of childhood and continuing through to the present, the disorder has been conceptualized as involving the three core features of hyperactivity, inattention, and poor impulse control. Symptoms of EI–DESR are excluded as being such a central feature, though some DSM editions, such as DSM-IV-TR (American Psychiatric Association, 2000, p. 80), do mention such symptoms as "associated features" including low frustration tolerance, temper outbursts, mood lability, demoralization, and dysphoria. If anything, these symptoms of EI–DESR were used to create in DSM-III an entirely new disorder—oppositional defiant disorder (ODD)—that even then was recognized as having a robust association with ADHD. Nevertheless, for the past 35 years, part of this psychiatric disorder's official taxonomic doctrine has been that problems with emotional regulation were not a central feature of ADHD, though they could be construed as associated features occurring some of the time. I believe that this taxonomic neglect of EI–DESR and its relegation to a secondary or associated status in conceptualizing ADHD may have been driven in large part by the first reason cited earlier—the problem of measurement of poor emotion regulation. As far as most clinicians and researchers have been concerned, this official taxonomic neglect sealed the fate of symptoms of EI–DESR as being merely an associated feature of ADHD, even though historically it was been viewed otherwise even back to the earliest reports of the disorder.

One of the purposes of this chapter is to question the wisdom of having disconnected EI–DESR from the core triad of ADHD symptoms, despite its close involvement in one of them—impulsive and poorly inhibited behavior. After all, if emotions are behavior, then emotional expressions should be as impulsive as gestural and verbal behavior in those afflicted with ADHD given that one of its central deficits is "volitional inhibition," which even Still (1902) argued was

the case. However, the musings of clinicians and theorists on the essential features of ADHD, while suggestive of the central nature of in the conceptualization of ADHD and its precursor disorders, are by themselves not sufficient evidence that this is actually so. Historical commentaries aside, what other reasons are there to consider EI–DESR as a central feature of ADHD?

THE IMPORTANCE OF EI–DESR IN THE NEUROPSYCHOLOGY OF ADHD

The past 16 years have witnessed a renewed interest in the difficulties with emotion regulation in ADHD among theorists grappling with model building. I have previously attempted to set forth a more comprehensive neuropsychological theory of ADHD (Barkley, 1997a, 1997c, 2012a, 2012c) that went beyond those at the time that stipulated the central feature of ADHD to be a problem with behavioral inhibition (Quay, 1987, 1997) or of an energetic pool of motivation or arousal (Sergeant, 1988; van der Meere & Sergeant, 1988). Five executive functions (EFs) were identified in my initial theory that could be implicated in ADHD: behavioral inhibition, nonverbal working memory (chiefly visual imagery and private audition), verbal working memory (primarily private self-speech), the self-regulation of emotion–motivation–arousal (using these working memory systems), and reconstitution (generating multiple response options through a process of mental play [analysis and synthesis]). To this I have more recently added self-awareness (Barkley, 2012c).

These executive functions provide for human "self-regulation," defined as (1) any action one directs at oneself (2) in order to change subsequent behavior (3) so as to alter a distant or delayed consequence and thereby maximize those consequences. Each EF, in fact, was argued to constitute a specific form of self-control or self-directed activities (e.g., self-directed attention, self-restraint, self-imaging, self-speech, self-emoting, and self-play). All functioned in concert for the overarching purpose of guiding behavior across time delays toward future goals that served to maximize delayed rather than immediate consequences. The executive deficits associated with ADHD led to a seventh nonexecutive deficit in the control of motor behavior via the EFs that disrupted its coordination, temporal sequencing, and hierarchical structure during goal-directed activities.

This EF-based theory of ADHD reinserted EI–DESR into a central theoretical position in understanding the nature of the disorder. It viewed emotional inhibition and the subsequent self-regulation of emotion as being one among the five (now six; Barkley, 2012a, 2012c) major EFs and argued that ADHD involved a disruption in it. In essence, this EF operated in conjunction with that of behavioral inhibition chiefly to accomplish four purposes very similar to the components of the earlier definition of emotional regulation by Gottman and Katz (1989). First, the inhibitory function served to delay (inhibit) the initial prepotent or dominant responses to an event, including their emotional tone and other emotional behavior, so that both the motor response *and* related emotional behavior were deferred. This set the stage for the second purpose of this EF, which was the modification of the initial emotional state to make it more congruent with and supportive of the individual's long-term goals. In part, this involved the use of the working memory systems (self-directed visual imagery and self-speech) to assist with self-soothing of emotional arousal and to create hindsight. The latter allowed the individual to consider consciously what had previously been experienced in similar situations and so guide the construction of the eventual response to the event informed by such prior information. This retrospective function of the working memory systems led to foresight or the prospective function. That function was the construction of an expectation about the situation and a consideration of potential future consequences that would arise for responding in various ways. This led to a conscious anticipation of that hypothetical future, and the selection and construction of a preparatory set of responses of what to do and when that would be more consistent with the longer-term welfare (goals) of the individual. In using the working memory systems in this way, the individual automatically has the additional capacity to delay the prepotent *emotional* response to that same event and use visual imagery and private speech to create alternative, competing secondary emotional states to it. This is because covert visual imagery and covert self-speech, among other forms of covert self-directed behavior, produce not only private images and verbalizations but also the private emotional charges associated with them (Damasio, 1994, 1995; Fuster, 1997). Such use of private action to countermand or counterbalance the initial emotional charge of external events contributes to self-soothing of emotional arousal and the formation of more socially appropriate emotional responses. They also contribute to the development of *emotional self-control* (Kopp, 1982). In short, the indi-

vidual possesses not only a means to consider and construct alternative motor responses to an event that will better maximize future consequences but also a capacity to construct alternative *emotional* responses joined to those behavioral actions that serve to achieve the same ends.

To summarize, this theory predicts two interrelated emotional problems for those with ADHD: one inhibitory and the other self-regulatory. The first problem is likely to be far more related to the HI symptom dimension and less so to the inattention dimension, whereas the second may be more related to the inattention dimension given its close connection to working memory systems and the manipulation of their content. Those with ADHD will be as impulsive with their primary emotions as they are with their motor or behavioral responses because they are essentially a single unitary event: Action and emotion are united in the response. And the person with ADHD will not be able to generate the countervailing secondary emotion needed to quell the primary one provoked by the event or to self-soothe primary emotional arousal to moderate the expression of his or her emotions so that he or she is more socially acceptable, much less to provide the emotional support needed for the alternative goal-directed behavior. This is the self-regulatory aspect of the executive system that is contingent on the inhibitory action and is also viewed as deficient in ADHD.

According to this theory, to the extent that an individual with ADHD displays a deficiency in *behavioral* inhibition or impulsiveness, he or she will automatically display an equivalent degree of deficiency in *emotional* inhibition or emotional impulsiveness. From this perspective, emotional impulsiveness, or EI, and the associated subsequent deficiency in the self-regulation of emotions, are just as much a part of ADHD as behavioral impulsiveness, and the associated problems with behavioral self-regulation. Still (1902) had reached the same conclusion for similar reasons.

But there are two other purposes to the EF of emotional inhibition/self-regulation. To understand them we must first dissect the concept of emotion. Emotions are motivational states. They can be regarded as arising from a two-dimensional process (Gray, 1994; Lang, 1995): One dimension is motivational and comprises approach–avoidance valences concerning any analysis of our actions and their attendant consequences (reinforcement–punishment); the other, a dimension of arousal that is orthogonal to the motivational one, contributes to the degree of activation, force, or intensity of the motivational state. Largely unappreciated to date by many researchers in the field of ADHD is that this view of emotion would lead to the prediction of both motivational and arousal problems inherent in those with ADHD. If one's emotional responses to events are as impulsive as his or her behavioral ones, then so will be his or her motivational and arousal responses. Given that these features of emotion form a unity (emotion–motivation–arousal), the more impulsively one's emotions are directly elicited by environmental events, the more one's motivation and arousal will be determined by and dependent on those events. As argued in this theory, the more one is able to manage, moderate, and manipulate one's emotional states via self-regulation in the service of their future goals, the more one automatically is capable of manipulating one's own motivational and arousal states to achieve those same ends. This is because the latter states (motivation and arousal) are the basis for the former one (emotion). ADHD must therefore involve a deficit in self-motivation and the self-regulation of arousal in support of future, goal-directed behavior as surely as it involves a deficit in the regulation of emotion for this self-same end. It is the capacity to self-motivate that serves to bridge gaps across delays and longer periods of time in the absence of consequences and therefore motivates delayed gratification and future-directed behavior (Fuster, 1997; Mischel et al., 1989). The evidence for such motivational deficits in ADHD is long-standing (Glow & Glow, 1979) and substantial (Luman, Oosterlaan, & Sergeant, 2005), and is supported below in the neuroanatomical network(s) underlying ADHD, especially the role of the anterior cingulate cortex in governing the amygdala specifically and the limbic system more generally. But the existence of and theoretical basis for this emotional–motivational component of ADHD goes largely unnoticed in current diagnostic conceptualizations of the disorder.

My theory of ADHD is not the only one that posits that difficulties with emotion regulation are likely to arise in ADHD. More recently, Nigg and Casey (2005) suggested that three distinguishable neural networks are to be found in the neuroanatomical regions implicated in ADHD, and that these cortical–subcortical loops or networks account for both the cognitive *and affective* response regulation problems seen in ADHD. Nigg and Casey posit that disruption of the frontal–limbic pathway would give rise to difficulties with emotion regulation in ADHD, while the frontal–striatal (working memory and response selection) and frontal–cerebellar

pathways (temporal organizing and timing of behavior) would be associated with the more cognitive deficits of knowing the "what" and "when," respectively, of preparing for and responding to events in various situations. They argued that affect regulation, motivation, and emotionally reactive responding are important in ADHD, and that dysfunction in the frontal–limbic–amygdala circuit probably accounts for it. A short time later, Sagvolden, Johansen, Aase, and Russell (2005) published their dopaminergic theory of ADHD, which predicted difficulties with impulsive emotion and low frustration tolerance as a consequence of hypodopaminergic activation in at least one of the three dopamine networks in this model (the mesolimbic pathway). This pathway is similar to the frontal–limbic pathway in the Nigg and Casey model, and is said to produce much the same emotional consequences in ADHD. This frontal–limbic pathway has more recently been identified as that mediating the "hot" or emotional–impulsive, hyperactive, and motivational features of ADHD (and EF) as distinguished from the "cool" pathways (frontal–striatal, frontal–cerebellar) believed to mediate the attention, disorganization, working memory, and timing problems in ADHD (Castellanos, Sonuga-Barke, Milham, & Tannock, 2006).

The central dogma on the nature of ADHD prevailing at the moment, and captured in DSM descriptions and criteria (see Chapter 2), does not acknowledge such deficits in emotion regulation as an inherent part of ADHD. It views problems with emotional control as merely associated features. But there are not only good historical grounds for correcting this abandonment of EI–DESR in the nature of ADHD, as shown earlier, but as the previous discussion attests, there are also good theoretical reasons for doing so.

THE IMPORTANCE OF EI–DESR IN THE NEUROANATOMY OF ADHD

Regardless of, or apart from, theoretical predictions about the role of EI–DESR in the central nature of ADHD, the growing body of evidence on the neuroanatomical networks that underlie the disorder provides further justification for the inclusion of EI–DESR as a core feature of this disorder. The networks believed to give rise to the inattentive, hyperactive, and impulsive behavior of those with ADHD, reflecting their deficient self-regulation, are also the networks that govern the inhibition of reactive emotion and the subsequent cognitive, effortful, top-down, or executive control of emotion. Specifically, these are the lateral prefrontal cortex (LPFC) and the anterior cingulate cortex (ACC). It is not my purpose in this chapter to give a detailed and critical review of the results of neuroimaging studies of ADHD. Suffice to say here, detailed reviews and meta-analyses of the research appear to support the conclusion that at least five brain regions are involved in ADHD (Bush, Valera, & Seidman, 2005; Cortese et al., 2012; Hart, Radua, Nakao, Mataix-Cols, & Rubia, 2013; Hutchinson, Mathias, & Banich, 2008; Mackie et al., 2007; Paloyelis, Mehta, Kuntsi, & Asherson, 2007; Valera, Faraone, Murray, & Seidman, 2007): (1) the LPFC, (2) the ACC, (3) the basal ganglia (and particularly the caudate of the striatum), (4) the splenium of the corpus callosum, and (5) the cerebellum (especially the vermis). Where asymmetries are found in these regions, evidence suggests greater involvement of the right hemisphere than left in the disorder (Valera et al., 2007). Noteworthy is that differences in the size and functioning of these regions have been found in the unaffected first-degree relatives of people with ADHD, which suggests that they may be part of a familial endophenotype for the disorder. The deficits found in relatives often fall intermediate between affected people with ADHD and control groups (Mulder et al., 2008). Several of these affected brain regions are also found to distinguish among people with ADHD, those with CD (Rubia et al., 2009), and those with bipolar disorder (Biederman, Makris, et al., 2008). Also of importance is that the size and level of activity within these regions are correlated with the degree of ADHD symptoms (Casey et al., 1997).

Studies of the cognitive or conscious regulation of emotion have implicated the LPFC, medial PFC, and ACC, along with its connections to the amygdala (and limbic system more generally) in this executive function (Ochsner & Gross, 2005). Of interest to this analysis is that the little research that exists has found that the generation of emotion from an aversive external stimulus was largely driven by activation in the amygdala in a bottom-up fashion of brain activation. In contrast the conscious and volitional generation of an emotion to a mentally represented event held in mind (working memory) revealed a more top-down activation of the LPFC, the medial PFC, the ACC, then the amygdala. Except for the amygdala, the remaining brain regions have also been implicated in ADHD, as noted earlier.

From this vantage point, studies indicate that the neuroanatomical network involved in conscious emo-

tional self-regulation in normal brain functioning would be disturbed in those with ADHD given the association of ADHD with deviations in most of the same regions involved in that network. This disturbance would constitute a top-down problem in the conscious regulation of emotions and not a bottom-up problem of an overactive amygdala–limbic system. In essence, ADHD creates a state in which the normal emotion-generating properties of the limbic system and particularly the anger-, frustration-, and aggression-generating properties of the amygdala are inadequately regulated by higher cortical regions and intermediate structures (nucleus acumbens, etc.). These higher level regions provide for self-control and take over the emotions in the service of longer-term goal-directed, hierarchically organized, and socially acceptable behavior.

One key to understanding how EI–DESR may arise in ADHD is the ACC. As reviews have noted, this brain region traditionally has been associated with the limbic system, and it is likely through the ACC, in part, that a top-down cortical (conscious or volitional) regulation of the emotional system (limbic system) is achieved (Bush, Luu, & Posner, 2000). Such a top-down hierarchical regulation of conscious, complex, goal-directed behavior has been attributed to a caudal-to-rostral, anterior-to-posterior organization of the PFC (Badre, 2008). This makes clear why the smaller size and functional deficits in the PFC in ADHD would give rise to a breakdown in self-regulation and in the hierarchical organization of goal-directed actions. Concerning the specific thesis of this chapter, these reviews suggest that it may be via the connections to the ACC that this top-down control by the PFC is exercised over emotional behavior and its support of goal-directed actions (Etkin, Egner, Peraza, Kandel, & Hirsch, 2006; Ochsner & Gross, 2005).

The ACC can be subdivided into two regions, dorsal (upper) and rostral–ventral (lower), that may distinguish between the cognitive–executive activities and the emotion regulation activities of the ACC, respectively (Bush et al., 2000). Neuroimaging studies of ADHD have not made much of an effort to distinguish between these two functional zones of the ACC. But a few studies suggest that both zones of the ACC are involved in ADHD (Bush et al., 2000; Krauel et al., 2007). This helps to explain partially both the difficulties with behavioral and emotional self-regulation associated with this disorder.

As some reviews have noted, it is also likely that it is through the ACC that drive and motivation have an influence over motor functioning (Paus, 2001). This would be consistent with the theoretical positions staked out earlier that ADHD, which seems to arise in part from deficits in the ACC, would involve impaired drive and motivation along with those of EI–DESR. For the time being, the reliable finding of ACC involvement in ADHD, in addition to that of the LPFC, suggests that this may be one of the neuroanatomical bases for anticipating that EI–DESR would be as much of a core deficit associated with ADHD as would the other EFs regulated by this network. This hypothesis of problematic top-down management of the amygdala–limbic system by the executive brain (PFC) in ADHD gained recent support in findings of dysfunctional connectivity in amygdala–LPFC networks in ADHD that were directly associated with the degree of emotional lability (Hulvershorn et al., 2014).

EVIDENCE FOR EI–DESR IN ADHD

So far, it has been shown that there are sound historical, theoretical (neuropsychological), and neuroanatomical reasons for believing that EI–DESR should be a central deficit in ADHD. But is it? What is the evidence for such emotion regulation problems apart from the clinical observations of historically significant figures, the theoretical propositions, or the neuroanatomical findings in the field of ADHD? The growing body of evidence that has begun to accumulate on this topic suggests that ADHD is associated with emotional dysregulation. Until the past few years, very few studies bothered to focus on this issue. And, of course, this seems to be due in part to difficulties involved in the measurement of emotional behavior relative to the more easily assessed motor and attention behaviors involved in the disorder, as noted earlier. It may also be yet another indication that removing these emotional aspects of the disorder from clinical diagnostic criteria beginning with DSM-II and relegating them to the status of associated features in DSM-III onward may have led to relatively less interest among researchers in their involvement in ADHD.

Rating Scales

Certainly the largest body of evidence comprises studies that used ratings scales of emotional problems in children with ADHD relative to control groups. Since it is not so much anxiety or depression that seems to

be problematic in most cases of ADHD, apart from existing as comorbid disorders in a minority of cases (Barkley, 2006; Brown, 2000), I am ignoring evidence for the involvement of these problems in ADHD. The evidence reviewed so far suggests that the inhibition of frustration, impatience, anger, hostility, and even reactive social aggression, as well as more general emotional arousability, excitability, or lability, is more closely involved in the emotional self-regulation problems associated with ADHD. It is clear from reviews of research on the commonly available behavior rating scales employed in the field of ADHD, such as the Child Behavior Checklist, Conners' Parent and Teacher Rating Scales, Behavior Problem Checklist, IOWA Conners Rating Scales, and Personality Inventory for Children (Barkley, 1990, 1998), that the disorder is nearly uniformly associated with elevations in parent and teacher ratings of these negative emotions. Typically, items assessing these emotions are found on subscales that are labeled as conduct problems or aggression and usually together comprise a broad-band externalizing dimension, along with subscales assessing hyperactivity and inattention (Achenbach & Edelbrock, 1986). The relationships among these scales are quite high, as demonstrated by correlations of .60–.83 and higher between Aggression and Hyperactivity scales and loadings on the Externalizing broad-band factor of .70–.76 (Achenbach, 1986). Ratings of inattention often share a much lower relationship with aggression (.20–.56; Achenbach & Edelbrock, 1986). It is also telling from the standpoint of earlier arguments that items assessing impulsiveness load as high or higher on factors measuring aggression and the negative emotions of interest here (frustration, hostility, temper, anger) than they do on factors assessing hyperactivity or inattention. In short, the more severe one's ADHD symptoms are, particularly those related to hyperactivity and impulsivity, the more severe are ratings of frustration, temper, anger, hostility, and aggression. It is certainly possible that this simply reflects comorbidity between ADHD and disorders such as ODD and CD, so this evidence could be dismissed as not necessarily showing an inherent core problem in ADHD with EI–DESR. But this line of argument can just as easily be turned around and used to explain why there is such strong comorbidity between ADHD and especially ODD: It is the overlapping shared variance between these disorders in their problems with EI–DESR that provide the explanatory linkage, as I discuss below. This is not merely conjecture. Recent research shows that the emotional lability often seen in ADHD is linked to the same shared genetic variance that underlies the traditional ADHD symptom dimensions (Merwood et al., 2014). In other words, there is a common genetic liability to ADHD inattention, HI symptoms, and emotional lability.

Far more evidence than this exists on the link between ADHD symptoms and emotion regulation difficulties. The Behavior Rating Inventory of Executive Functioning (BRIEF; Gioia, Isquith, Guy, & Kenworthy, 2000) is an 86-item rating scale developed to provide parent and teacher ratings of children's executive abilities. It contains eight narrow-band scales, three of which are pertinent to the present discussion, those being Inhibit, Working Memory, and Emotional Control. The Inhibit scale contains items reflecting not only impulsiveness but also hyperactivity. The Working Memory scale includes items related to sustained attention and distractibility, among other ADHD-like symptoms, such as disorganization and forgetfulness. The Emotional Control scale contains the following items, most of which bear directly on the argument being made here: Overreacts to small problems; Has explosive, angry outbursts; Becomes tearful easily; Has outbursts for little reason; Mood changes frequently; Reacts more strongly to situations than other children; Mood is easily influenced by the situation; Angry or tearful outbursts are intense but end suddenly; Small events trigger big reactions; and Becomes upset too easily. The Working Memory and Inhibit scales correlate highly with the respective ADHD scales of Inattention (r = .60) and Hyperactive–Impulsive (r = .73) using the DSM-IV items (Gioia et al., 2000). The Emotional Control scale is also significantly correlated with the HI items from DSM-IV (.56). Indeed, when these three scores from the BRIEF are factor-analyzed with parent and teacher ratings of ADHD symptoms, they comprise just two factors, reflecting the DSM symptom dimensions. ADHD inattention scores load highly on the same factor, as do Working Memory scores, while ADHD Hyperactive–Impulsive scores load highly on the same factor as items from the Inhibit scale. More pertinent to my point here, the Emotion Control scale of the BRIEF loads nearly as highly (.78) on the factor containing the BRIEF Inhibit (.87) and ADHD Hyperactive–Impulsive items (.76) as do the latter two scales, indicating that it is clearly as much an inherent part of this aspect of ADHD as hyperactive and impulsive behavior. Noteworthy as well is that the Emotional Control scale shows a substantially lower relationship to Working Memory and ADHD Inatten-

tion items, supporting the point made earlier that it is the impulsivity dimension of ADHD that is involved in its EI problems. Not surprisingly, research by the developers indicates that children with ADHD combined type are rated significantly more highly on all three of these BRIEF scales (Working Memory, Inhibit, and Emotional Control) relative to control children. Others have found similar results (Mahone et al., 2002). Both children with ADHD and those with ADHD + Tourette syndrome were found to have significantly elevated scores on the Emotional Control scale, along with those for Inhibition and Working Memory, compared to a control group. And this study also found, once again, that the broad-band ratings of Behavioral Regulation (Inhibition and Emotional Control) were substantially correlated with ratings of hyperactive and impulsive ADHD symptoms (*r*'s = .70) and less so with ratings of ADHD inattention (.57).

The BRIEF narrow-band scales have been found to comprise two larger, broad-band dimensions or factors labeled as Metacognition (containing the Working Memory scale, among others related to planning, shifting, organization, etc.) and Behavioral Regulation (containing the Inhibit and Emotional Control scales). The latter is the evidence with a bearing on the issue of this chapter. Problems with emotional inhibition and its self-regulation are part of the same dimension as problems with HI behavior. In summary, problems with inhibition of behavior are highly associated with problems with inhibition and self-regulation of emotion.

The same pattern of findings is evident in the research used to develop the Conners Adult ADHD Rating Scales (Conners, Erhardt, & Sparrow, 1998). Symptoms assessing emotional dysregulation were found to load on the same factor as symptoms of impulsiveness, supporting the argument made here for their coexistence. And this factor is strongly related to DSM-IV symptoms of ADHD on this scale and especially the HI dimension of the disorder.

More evidence of emotional dysregulation comes from a study that followed children diagnosed with ADHD into adolescence using multiple self-report measures of anger, hostility, verbal, and physical aggression at the follow-up evaluation (Harty, Miller, Newcorn, & Halperin, 2009). The initial sample was classified as having ADHD only, ADHD with ODD, and ADHD with CD in childhood. At follow-up, the groups did not differ in hostility. Only the ADHD with ODD and ADHD with CD groups demonstrated more anger and verbal and physical aggression relative to a non-ADHD control group at follow-up, with those with CD showing the highest levels, particularly for physical aggression. This might suggest that symptoms of EI in ADHD are a consequence of comorbid ODD and CD. However, the authors found that all of their group differences were accounted for by the severity of ADHD in childhood and by its persistence into adolescence. When these were statistically controlled, the only remaining group difference was greater physical aggression at adolescence in the subgroup that had CD in childhood. These results indicate that it is severity of ADHD in childhood and its persistence into adolescence that is associated with high levels of anger, hostility, and verbal aggression in adolescence, while CD makes an additional contribution to later risk for physical aggression. Such results are in keeping with the hypothesis here that ADHD accounts for the emotional component of ODD and for the persistence of emotions such as anger, hostility, and verbal aggression over time (discussed further below).

Research on adults with ADHD using items reflecting EI–DESR has likewise shown that such adults manifest significant problems in this domain (Barkley, Murphy, & Fischer, 2008). In comparison to a community control group, clinic-referred adults diagnosed with ADHD were significantly more likely to endorse problems with EI symptoms, as shown in Figure 3.1 (also see Barkley & Murphy, 2010). These same symptoms were also significantly more common in adults with ADHD than in a clinical control group not diagnosed with ADHD. Just as telling, the frequency of these symptoms was just as common in the adults with ADHD as those for DSM-IV inattention (74–97%) and often occurred more frequently than the HI symptoms in DSM-IV (30–79%). More recent studies also found that adults with ADHD had higher rates of emotional impulsivity and dysregulation, similarly assessed, than did control adults, and that these were not a function of comorbidity with depression or ODD (Mitchell, Robertson, Anastopoulos, Nelson-Gray, & Kollins, 2012; Surman et al., 2011, 2013). Important to note is that these symptoms of EI–DESR are not redundant with the cold cognitive executive deficits (typically working memory) often associated with ADHD and in fact are not associated with those EF deficits at all (Surman et al., in press). Thus, they serve as an additional component to the nature of ADHD in adults not represented in other neuropsychological deficits traditionally associated with the disorder.

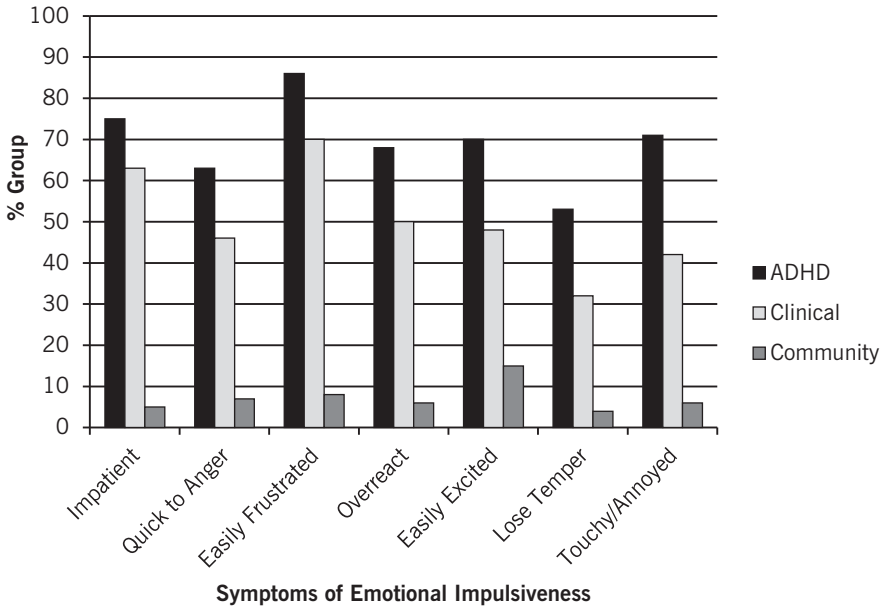

FIGURE 3.1. Percentage of adults with ADHD compared to clinical and community control adults showing each of seven symptoms of emotional impulsivity. Data used to construct the graph are adapted from Barkley, Murphy, and Fischer (2008).

In a follow-up study in that same book that reported on children with ADHD to young adulthood (Barkley et al., 2008), we found that children whose ADHD had persisted to a mean age of 27 years were significantly more likely to endorse these same items of EI–DESR than were those whose ADHD had not persisted or those in a control group followed to adulthood as well, as shown in Figure 3.2 (also see Barkley & Fischer, 2011). Again, these symptoms of impulsive emotion were as common as the DSM-IV symptoms of inattention and as common or more so than those for HI behavior in the group with persistent ADHD. We also found that these items loaded onto the same dimension as symptoms of hyperactivity–impulsivity from the DSM-IV, but not on those symptoms of inattention. This demonstrates that symptoms of EI–DESR are just as common if not more so than the DSM-IV symptoms that represent the disorder.

More evidence of a link between ADHD and emotional dysregulation comes from the development of my own scales evaluating EF for children (Barkley 2012b) and adults (Barkley, 2011), which contain five dimensions. The three that are most pertinent to this review are Self-Restraint (inhibition), Emotional Self-Regulation, and Self-Motivation. Results of comparisons between ADHD and control cases in the population normative samples routinely revealed that the vast majority of children and adults with ADHD score in the impaired range on these scales (Barkley, 2011; Barkley, 2012b). And the majority of children with ADHD that persisted to young adulthood (Barkley & Fischer, 2011), and clinic-referred adults with ADHD (Barkley, 2011; Barkley & Murphy, 2011) also scored in the impaired range on the Self-Restraint and Self-Motivation scales (the Self-Control of Emotion dimension was not yet available for use in these studies). Evidence from the development of these scales also indicated that impulsive emotions loaded on the Self-Restraint scale, as hypothesized earlier, while difficulties with self-regulation of emotions formed their own separate but related dimension.

Other recent studies have likewise found that children with ADHD are rated by parents as having significantly more problems with emotional functioning

than control cases, and that these problems are distinct from those EF deficits associated with the "cool" EFs, such as working memory (Sjöwall, Roth, Lindqvist, & Thorell, 2013).

In keeping with these results, higher rates of "road rage" (anger, hostility, and aggression while using a motor vehicle) have been found in adults with ADHD or many ADHD symptoms than in control groups (Richards, Deffenbacher, Rosen, Barkley, & Rodricks, 2006). Also, Ramirez and colleagues (1997) found that adults with many ADHD symptoms expressed more anger, did so in more dysfunctional ways, and were more labile in terms of anxious/depressed moods than those with fewer ADHD symptoms. Likewise, Able, Johnston, Adler, and Swindle (2007) found similar results among nonreferred adults who met criteria for ADHD in a population screen of 21,000 managed care plan members. These adults reported significantly greater interpersonal anger and conflict toward others they considered close to them and greater frequency with which their emotional symptoms interfered with the work, social, or family/home life than adults who did not have ADHD (Able et al., 2007). Dowson and Blackwell (2010) similarly found ADHD in adults to be linked to hot temperedness and impulsive aggression. The evidence to date therefore demonstrates that both children and adults with ADHD have considerable difficulties with frustration, anger, hostility, and aggression.

Research using rating scales of emotional reactivity, lability, and regulation likewise finds that these behaviors are problematic in both children and adults with ADHD (Jensen & Rosén, 2004). Kitchens, Rosén, and Braaten (1999) reported that children with ADHD rated themselves and their mothers rated them as more angry and depressed than did mothers of children without ADHD; the greater depression ratings were more typical of males than of females with ADHD. In a subsequent study, Braaten and Rosén (2000) reported that boys with ADHD expressed lower levels of empathy and exhibited more sadness, anger, and guilt than control boys. Similarly, Maedgen and Carlson (2000) found that children with ADHD have significantly more problems with emotional self-regulation than control children. And in several recent studies based on children's self-reports, children with ADHD had

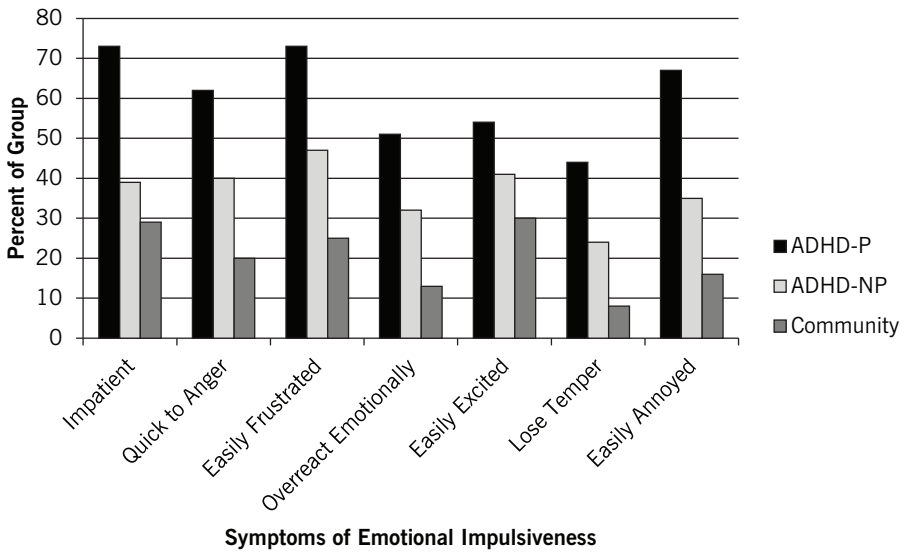

FIGURE 3.2. Percentage of children followed to adulthood (mean age 27 years) showing each of seven symptoms of emotional impulsivity for children with ADHD that persists to adulthood (ADHD-P), for those whose ADHD did not persist to adulthood (ADHD-NP), and for the control group. Data used to construct the graph are adapted from Barkley, Murphy, and Fischer (2008).

more difficulties with emotion regulation and emotional instability and less emotional maturity than control children (Ciuluvica, Mitrofan, & Grilli, 2013; Jogsan, 2013). Once more, such problems with emotion reactivity and regulation were associated more with the HI dimension and far less with inattention in ADHD (Ciuluvica et al., 2013) as has been found in preschoolers with ADHD as well (Miller, Miller, Healey, Marshall, & Halperin, 2013). The largest study to date that used ratings of emotional lability in children with ADHD, involving 1,186 children with the disorder and 1,827 siblings, revealed that children with ADHD on average place at or above 1.5 standard deviations above age and sex comparisons groups in their emotional lability (Sobanski et al., 2010). Emotional lability above the 75th percentile for age and sex norms significantly increased the risk that children with ADHD would also manifest ODD, affective disorders, and substance use disorders. Similar results are evident in adults with ADHD. Skirrow and Asherson (2013) recently studied 500 adults referred to a clinic for adult ADHD and found that emotional lability was significantly greater in those diagnosed with ADHD. Yet again, in both studies, the problems of emotional lability were associated with core ADHD symptoms, primarily the HI dimension (Skirrow & Asherson, 2013; Sobanski et al., 2010). As noted earlier, research on 1,920 child twins found that a common or shared genetic pathway exists between traditional ADHD symptom dimensions and the dimension of emotional lability (Merwood et al., 2014).

Other evidence for the centrality of EI–DESR in ADHD comes from the small but growing body of research on the link between early childhood temperament and ADHD (Nigg, Goldsmith, & Sachek, 2004). "Temperament" is the global term for a set of early emerging traits in development that form a basis for later personality. Most of the constituent traits are thought to be affective in nature (Goldsmith et al., 1987) and can be distinguished as involving either reactivity or regulation. In the area of reactivity, most theories distinguish between withdrawal (fearfulness) and approach (hostile–angry) components of negative affectivity and between approach and nonapproach positive affectivity (i.e., exuberance, contentment). Within the regulatory domain of temperament lie processes that attenuate, amplify, or sustain elicited emotions, such as comforting behavior, self-distraction, cognitive self-statements and imagery, and other instrumental voluntary and effortful behaviors that serve to modify or moderate elicited emotions (Nigg et al., 2004). These self-modifying activities of this domain are often subsumed under the term "emotional regulation." It should now be evident how extremes of early childhood temperament in both the reactivity (hostile–angry) and emotional regulatory domains may serve to link research on temperament to ADHD, as Nigg et al. have argued.

Nigg et al. (2004) cited several studies of the overlap or link between negative temperament in early development and risk for the psychopathological dimension of externalizing behavior more generally and ADHD specifically. First, studies have revealed significant relationships among negative affectivity, poor self-regulation, and impulsivity as correlates of this broad-band externalizing dimension (Campbell, Pierce, March, Ewing, & Szumowski, 1994; Huey & Weisz, 1997; Sanson, Stuart, Prior, & Oberklaid, 1993). Longitudinal studies have likewise found that early ratings by mothers of children's temperamental difficultness were significantly predictive of degree of externalizing behavior at ages 5, 6, and 8 years (Bates & Bayles, 1988; Bates, Bayles, Bennett, Ridge, & Brown, 1991). A later longitudinal study of pregnant women that followed women through their children's deliveries and early development to first grade found that both maternal ratings and direct observations of child behavior involving early temperament were linked to later risk for ADHD (Goldsmith, Lemery, & Essex, 2004). Specifically, they found that the areas of hostile and aggressive behavior, anger, and difficulties with inhibitory control were significantly predictive of severity of children's ADHD symptoms by entry into kindergarten. Of relevance to the thesis of this chapter is that early maternal ratings of children's anger and irritability during the first 3.5 years of development were significantly predictive of later ratings of severity of ADHD symptoms, as well as inhibitory control at age 4.5. The relationship of anger and irritability to ADHD was found to be mediated by their link to poor inhibitory control and its relationship to ADHD. Indeed, the cross-sectional correlations of ADHD symptoms with relevant temperament ratings were .6 to .7 (Lemery, Essex, & Smider, 2002). The inverse has also been demonstrated: Children with ADHD manifest significant elevations on measures of several temperamental traits, including negative affectivity (Foley, McClowry, & Castellanos, 2008; Martel & Nigg, 2006).

Such research provides supporting evidence for the thesis presented here that problems with inhibiting emotion and its subsequent self-regulation are strongly associated with concurrent ADHD and are predictive

of later ADHD severity. They further illustrate that it is chiefly through the symptom dimension of impulsivity that this link between emotional reactivity and poor emotion regulation and ADHD is likely to occur, as evidence reviewed earlier has already suggested (Martel, 2009). And it shows that these constructs of emotional control are important in understanding or more clearly illustrating the relationship of ADHD to other psychological problems and even comorbid psychiatric disorders. The two symptom dimensions of ADHD in DSM-IV simply do not make such linkages evident or understandable without the explicit acknowledgment that EI–DESR is related to them and is a central component of ADHD.

Direct Observations

Direct evidence of problems with EI–DESR comes from observational studies of emotional behavior in children with ADHD. Rosenbaum and Baker (1984) found that greater negative affect was expressed by children with ADHD during a concept learning task involving noncontingent negative feedback. Cole, Zahn-Waxler, and Smith (1994) found that levels of negative affect were significantly and positively correlated with symptoms of and risk for ADHD, but only in boys. Greater emotional responsiveness has been reported as well in the social interactions of children with ADHD. Johnston and Mash (2001) found that children with ADHD displayed greater emotional intonation in their verbal interactions with their mothers. Studies of peer interactions also revealed children with ADHD to be more negative and emotional in their social communications with peers. This greater level of expressed negative emotion in children with ADHD is most salient in the subgroup that has high levels of comorbid aggression (Hinshaw & Melnick, 1995). Consistent with such findings, Keltner, Moffitt, and Stouthamer-Loeber (1995) recorded the facial expressions of adolescent boys during a structured social interaction. Four groups of boys were created; the first was rated as having high levels of externalizing symptoms (hyperactive–impulsive–inattentive–aggressive behavior); the second comprised boys rated as having more internalizing symptoms (anxiety, depression, etc.); the third group comprised boys having elevations in ratings of both types of symptoms; and the fourth group comprised nondisordered adolescent boys. Boys showing high levels of externalizing symptoms were found to demonstrate significantly more facial expressions of anger than the other groups, which were low in externalizing symptoms. These results suggest the possibility that the commonly noted association of ADHD with defiant and hostile behavior (see Hinshaw, 1987, for a review) may, at least in part, stem from a deficiency in emotional self-regulation in those with ADHD. Again, however, these findings merely suggest rather than confirm a link between ADHD and EI–DESR, and tend to imply that the poorest emotion modulation may be within the aggressive subgroup of children with ADHD.

Several studies have specifically evaluated frustration tolerance and anger responses in children with ADHD. Milich and Okazaki (1991) presented children with ADHD and control children with both solvable and unsolvable puzzles, and noted that children with ADHD were more likely to give up on solving either set of puzzles, especially those that were unsolvable, presumably because they became more frustrated with the latter puzzles. As evidence of this, they noted that the children with ADHD reported higher levels of frustration during these tasks than did the control children. In a separate study, Milich, Carlson, Pelham, and Licht (1991) found that these difficulties with frustration were ameliorated by the stimulant methylphenidate. Walcott and Landau (2004) evaluated boys with ADHD during a frustrating peer competition task in which half of the boys in each group were instructed to try to hide their feelings if they became upset. Boys with ADHD were more likely than control boys to become upset and were less effective at hiding their feelings, even when instructed to do so, in keeping with the EI and DESR components hypothesized to exist in theories of ADHD discussed earlier (Barkley, 1997b, 2012a). Similarly, Waschbusch and colleagues (2002) found that children with ADHD manifested more anger in response to behavioral provocation than did children without ADHD, regardless of comorbidity with ODD and CD (Waschbusch et al., 2002). As noted earlier, Rapport et al. (1986) also studied children with ADHD and control children, using a delayed gratification task presumed to assess frustration tolerance. They found the children with ADHD to be markedly less able to handle the frustration associated with delayed rewards. This evidence that children with ADHD quit or fail to persist at activities that demand problem solving is also evident in studies of their academic performance (Hoza, Pelham, Waschbusch, Kipp, & Owens, 2001). Such studies provide evidence that low frustration tolerance and quickness to anger seem to be associated with ADHD.

However, Melnick and Hinshaw (2000) observed children with ADHD during a family problem-solving task that elicited frustration. They found that only a highly aggressive subgroup of boys with ADHD demonstrated greater emotional responding to the emotional induction of the task and a less constructive pattern of emotional coping compared to nonaggressive boys with ADHD or controls. The extent to which the involvement of family members as part of this task added extra elements of either parental control or opportunities for family conflict (and aggression) might explain the discrepancy between this finding and those from the three studies discussed earlier. Also problematic in the study was the lack of information on the subtypes of ADHD enrolled and how they related to the subgrouping of children into aggressive and nonaggressive ADHD groups. As noted earlier, children with ADHD predominantly inattentive type (ADHD-PI) and especially those with SCT may not have symptoms of EI or DESR given that the latter are highly associated with the HI dimension in DSM-IV, a dimension on which such children with ADHD-PI-type would be low or even normal. A surprising finding of the study was that boys with ADHD assigned to the nonaggressive group fell within the normal range on all measures of emotion regulation. This is highly unlikely given evidence reviewed earlier if that group contained an equal percentage of children with ADHD combined presentation (ADHD-C) of equal severity to the children in the aggressive ADHD group. Nevertheless, the weight of the evidence suggests a link between ADHD and impulsive and poorly regulated emotion, specifically that of frustration.

Some evidence also demonstrates that this problem with emotional self-regulation arises from difficulties in the management of one's own emotions and not in its perception in others. Shapiro, Hughes, August, and Bloomquist (1993) found that children with ADHD did not differ from a normal control group in their ability to identify and process emotional cues in others. This may be true, however, only in "cool" situations, such as identifying facial expressions in pictures in storybooks or photos. This has certainly been observed in other studies of emotion recognition and understanding in which children with ADHD do not differ from controls, whereas children with Asperger syndrome or nonverbal learning disabilities are deficient (Semrud-Clikeman, Walkowiak, Wilkinson, & Minne, 2010). When in actual "hot" or live situations involving emotions of others, children with ADHD are more likely to show "emotional contagion" (adopting the emotional expressions of others) and more emotion in response to provocation, and are less accurate in identifying both their own emotions and those of others (Casey, 1996; Norvilitis, Casey, Brooklier, & Bonello, 2000). It is possible that this distinction between hot and cool situations involving emotional evaluation stems from live emotions generating more emotional reactions in those with ADHD, which may overwhelm their already deficient executive or cognitive ability to focus attention on their own emotions or the emotional cues of others when they are emotionally aroused. Regardless, whether those with ADHD have problems perceiving the emotional states of others is unimportant to the present argument because this chapter focuses on the capacity for emotional inhibition and its subsequent self-regulation as being central to ADHD, and not the perception of emotions in others.

Psychophysiological Research

Very few studies have focused on the use of psychophysiological measures of the sympathetic and parasympathetic nervous systems to evaluate emotion regulation problems in children with ADHD. One study that did so during tasks involving negative emotion induction and suppression reported that children with ADHD show ineffective and high levels of parasympathetic activity, and reduced flexibility or adaptation in this activity across these tasks than do control children, who show greater ability to adapt their parasympathetic activity to setting demands (Musser et al., 2011).

The foregoing evidence from rating scales, direct observation, and psychophysiological studies largely demonstrates that the link between ADHD and poor emotion regulation is not hypothetical but actual. The vast majority of the prevailing evidence pertains to the EI component of the emotional dysregulation associated with ADHD, and shows that it is more related to the HI dimension of ADHD than to its inattention dimension, as expected in the earlier theoretical review. There has been far less research on the capacity of those with ADHD to self-regulate their emotional states actively and consciously, which is the more executive aspect, or the DESR component, of ADHD. I have hypothesized that this executive element of emotion regulation may well be associated with the inattention dimension of ADHD. For instance, Knouse and associates (2008) found the inattention dimension of ADHD symptoms to be more highly predictive of

lower positive and higher negative mood on their measures of emotional well-being than were symptoms of hyperactivity–impulsivity in their adult samples. As Hinshaw (2003) concluded in his review of poor emotion regulation in ADHD, the problems with EI appear to be largely independent of the EF deficits often associated with the disorder, at least when EF is psychometrically tested (indexing of the "cool" EF components). But the problems with DESR have rarely been studied in those with ADHD and may well be associated with working memory difficulties and the EFs more generally that are often found to be more closely linked to the inattention dimension of ADHD symptoms.

In summary, we see here objective evidence of clinical observations dating back through history to Crichton (1798), showing that disorders of attention and inhibition also involve problems with low frustration tolerance, anger, and negative emotional reactivity. While more research on this issue is clearly warranted, it is obvious even from this limited array of research findings that ADHD is associated with symptoms of EI–DESR. The argument that this merely reflects comorbidity with other disorders, particularly ODD, is not persuasive given that the symptoms of EI correlate highly with and load on the same behavioral dimensions as the DSM symptoms of hyperactivity–impulsivity, while being substantially less associated (though not unassociated) with the inattention–working memory symptoms of ADHD. If mere comorbidity were the explanation for these findings, the problems with EI should have formed their own separate dimension reflecting a distinct yet overlapping disorder with ADHD, as is the case with CD, depression, or anxiety. That has not typically been the case in the research reviewed here. Even so, this evidence suggests that future research would do well to examine the role of comorbidity in EI–DESR symptoms, if only to clearly rule out comorbid disorders such as CD, depression, and anxiety. ODD, on the other hand, may not be just an overlapping disorder with ADHD; it may be a direct consequence of it. This issue is explored next.

THE IMPORTANCE OF EI–DESR SYMPTOMS IN UNDERSTANDING COMORBIDITY WITH ODD

ODD is characterized in DSM-5 as "a frequent and persistent pattern of angry/irritable mood, argumentative/defiant behavior, or vindictiveness . . ." (American Psychiatric Association, 2013, p. 463). It comprises a set of eight symptoms, at least three of which reflect negative affect (loses temper, touchy or easily annoyed, angry and resentful), while four reflect social conflict with others (argues with adults, defies or refuses to comply, deliberately annoys others, blames others for his or her own mistakes). The final symptom likely relates to both an affective and a social component of the disorder (acts spiteful or vindictive) and may be more related to CD. ODD has a point prevalence of 1.4% of girls and 3.2% of boys in children ages 5–16 years, with 36% of girls and 43% of boys with ODD having another disorder (Maughan, Rowe, Messer, Goodman, & Meltzer, 2004). Lifetime prevalence of ODD is 10.2%, with over 92% of such cases having another comorbid disorder (Nock, Kazdin, Hiripi, & Kessler, 2007). The most common comorbidity is ADHD or impulse control disorders (Maughan et al., 2004; Nock et al., 2007; Speltz, McClellan, DeKlyen, & Jones, 1999).

The inverse is also true. ODD is the most common comorbid psychiatric disorder seen in conjunction with ADHD, occurring on average in 65% of cases and in as many as 84% of clinic-referred cases in childhood (see Chapter 5). It is possible that this may even be an underestimate given that such studies may not distinguish among ADHD subtypes. It is ADHD-C with which ODD is likely to have a far greater affiliation than with ADHD-PI or SCT/CDD (Milich et al., 2001; see also Chapter 17). ODD is 11 times more likely to coexist with ADHD than to occur at its base rate in the general population even in epidemiological samples (Angold, Costello, & Erkanli, 1999). ODD has also been found to be a common comorbidity in children with ADHD followed to adulthood (50% of those with persistent ADHD) and in clinic-referred adults with ADHD (Barkley et al., 2008), occurring in up to 35–53% of these adult cases according to self-reported information. This source of information may lead to underestimates of disorder among patients with ADHD (Barkley et al., 2008).

It is certainly possible that ADHD is one of several causes or contributing factors to the risk for ODD. Suggestive evidence for such a causal or contributory relationship comes from several lines of research findings. First, the severity of ADHD is certainly significantly and substantially correlated with the risk for and severity of ODD, with correlations between the two dimensions rising to as high as .91 in teenagers (Barkley, Guevremont, Anastopoulos, DuPaul, & Shelton, 1993) and ranging from .68 to .86 in younger children (Burns & Walsh, 2002; Gadow & Nolan, 2002; Harvey,

Friedman-Weieneth, Goldstein, & Sherman, 2007). Using data from my various studies, I analyzed available data for this review and found correlations between ADHD and ODD symptoms to be .72 in our study of kindergarten children with high levels of disruptive behavior and a control group (Shelton et al., 1998), .71 in self-reports of retrospective childhood symptoms in our follow-up study of hyperactive children to age 21 (Fischer, Barkley, Smallish, & Fletcher, 2002), .70 in the employer ratings of those participants' current symptoms at work, and .77 in participants' own ratings of those same symptoms in the workplace. All of these results show a robust relationship between the severity of symptoms of these two disorders. Others have also found that the severity of ADHD is predictive of the severity of later anger and verbal aggression in adolescence among children diagnosed with ADHD (Harty et al., 2009). And it is the persistence of ADHD from childhood to adolescence that explains these difficulties with anger and verbal aggression in adolescence (Harty et al., 2009).

Second, the prevalence of ODD alone is most common in the preschool age group and becomes relatively less common by school age and onward, occurring in just 1–3% of children after age 5 (Maughan et al., 2004; Bauermeister, 1992; Lavigne et al., 2001). After this age, approximately half or more cases of ODD are comorbid with ADHD. ADHD is therefore associated with the persistence of ODD over development, which may further imply a causal connection between the two disorders.

Third, some longitudinal research using preschool children shows that initial ADHD symptoms, particularly the HI dimension, are predictive of higher ODD scores 1 and 2 years later, whereas initial ODD scores were not predictive of later HI or inattention symptoms (Burns & Walsh, 2002). These findings hold even after statistically accounting for the ability of each symptom dimension to predict itself over time. As those authors noted, this suggests that the HI dimension of ADHD influences the development of ODD behavior in some way. This is consistent with substantial research showing that impulsivity or response inhibition, not hyperactivity, is linked to risk for and severity of conduct problems or externalizing behavior generally, both concurrently and longitudinally into adolescence (Berlin & Bohlin, 2002; Olson, Schilling, & Bates, 1999). Any initial link of hyperactivity to conduct problems seems to be accounted for by its association with poor inhibition (Berlin & Bohlin, 2002). Other follow-up research with this age group suggests that early-onset ODD *is* predictive of both later ODD and later ODD with ADHD, but that its stability over development when occurring alone was quite low, with approximately half of all cases remitting every 1–2 years (from 24% in preschool to 5% at 5-year follow-up; Lavigne et al., 2001). Speltz and colleagues (1999) found that early-onset ODD predicted later risk for ADHD as well. With development, ODD shows an increasing affiliation with ADHD, and especially persistent ADHD (Lavigne et al., 2001). And when ODD occurs with ADHD, it is significantly persistent over the next 4- to 10-year period (August, Realmuto, Joyce, & Hektner, 1999; Biederman, Petty, Dolan, et al., 2008; Speltz et al., 1999).

Fourth, studies of preschool children with hyperactivity (HYP) alone, ODD alone, and both disorders find that only the HYP groups are associated with greater pre- and perinatal risk factors and with a family history of both ADHD and ODD, whereas ODD alone is not (Harvey et al., 2007). Noteworthy as well is that children with HYP alone still had relatives with higher rates of ODD, whereas children with ODD alone did not. Further study of these preschool groups showed that both the children with HYP and HYP/ODD had greater levels of parental ADHD symptoms, maternal Axis I psychiatric disorders, negative life events, paternal Axis II psychiatric disorders, marital status, couple conflict, or the use of low intensity couples' conflict tactics (Goldstein, Harvey, Freidman-Weieneth, Pierce, et al., 2007). (Only ODD was not further studied in this subsequent research because it was not found initially to be assosciated with biogenetic risk factors.) Yet these two groups did differ in the degree of maternal Axis II psychopathology, paternal Axis I psychopathology, and the use of more severe couple conflict tactics. This suggests that genetic and biological contributors to disorder are chiefly linked with ADHD (HYP) whether it occurs with or without ODD, and not with ODD alone. It further suggests that there exists a genetic predisposition to ADHD that also seems to create a risk factor for ODD in relatives. It further implies that social-environmental factors may be more responsible for preschool ODD when it occurs alone than is the case with ADHD or ADHD with ODD.

Obviously this suggests that ADHD is not the only contributor or cause of ODD, especially in the preschool age group in which ADHD can exist alone (Bauermeister, 1992; Gadow & Nolan, 2002). But it does suggest that when ODD exists alone, it is not an especially developmentally persistent disorder unless it

is linked to ADHD. It also intimates that the nature of ODD when it occurs alone in the preschool age group may have more to do with both the behavioral or social conflict component of the disorder and its greater situation specificity toward parents than its EI component, as the more detailed investigation on the nature of ODD by Hoffenaar and Hoeksma (2002) suggests. And that social component of ODD alone may explain why it is associated with disrupted parenting and family stress both concurrently (Cunningham & Boyle, 2002) and over development (Burke, Pardini, & Loeber, 2008), whereas ADHD alone shows no such reciprocal effects of parent–child interactions over development (Burke et al., 2008). All this could explain the very low level of concurrent validity (interjudge agreement and discrimination on observational measures) of ODD only relative to ADHD and especially their combination, which has substantially greater concurrent validity (Harvey et al., 2007). Research on ODD rarely separates these two components (emotional, social conflict) out for an examination of their development, correlates, stability, and contributors/causes. The behavioral–social component of ODD may be more likely to arise from disrupted parenting and family stressors, along with associated mismanagement of the gambits of preschool children's sporadically defiant behavior (Barkley, 1997c; Burke et al., 2008; Cunningham & Boyle, 2002; Lahey & Waldman, 2003; Moffitt, 1993; Patterson, DeGarmo, & Knutson, 2000). The emotional component of ODD may involve an underlying neuropsychological deficit in emotional impulsiveness and perhaps to a lesser extent its deficient self-regulation, as is argued here and by others (Martel, 2009) to be the case in ADHD.

In my opinion, it is the underappreciated EI–DESR symptoms of ADHD that are most likely to explain this high level of comorbidity of ADHD with ODD as categories of disorders and their strong relationship when assessed dimensionally. This is evident in the fact that the three symptoms reflecting negative emotion in the ODD symptom list are very similar to those believed to comprise EI noted earlier (low frustration tolerance, quickness to anger, impatience, and emotional excitability). As shown earlier, these EI symptoms and related problems with DESR are more common in children with ADHD. Research on the construct of oppositionality using a larger range of items identifies two large trait factors as more likely representing this construct or disorder, along with a set of situation-specific factors (parents, other authorities, peers) (Hoffenaar & Hoeksma, 2002). As noted earlier, these two trait factors are emotional and behavioral (social conflict). DSM-5 now reflects this dichotomy of the ODD symptoms. Others have also argued for viewing ODD as having two components (emotional and social behavioral; Burke, Loeber, & Pardini, 2009). They seem most likely to represent the components of negative affectivity and social behavioral conflict discussed earlier for DSM-5 symptoms. Supporting this distinction between these components are recent findings that the emotional symptoms of ODD are more predictive of later internalizing disorders, and particularly depression, whereas social conflict symptoms are more related to the developmental risk for CD (Burke, 2009).

Given that three of the symptoms of ODD appear to involve EI–DESR, children with ADHD-C could be considered to be virtually borderline or subthreshold cases of ODD as a consequence. Their low frustration tolerance, impatience, and quickness to anger set them up for patterns of reactive verbal aggression during provocative encounters with parents and peers, perhaps explaining why reactively aggressive children often show significantly higher rates of ADHD symptoms than do proactively aggressive children or control groups (Vitaro, Brendgen, & Tremblay, 2002). Indeed, as Martel and Nigg (2006) found, it is the temperamental trait of negative affectivity associated with ADHD in children that is most closely linked to their risk for ODD. As Ambrosini, Bennett, and Elia (2013) also observed, children with ADHD who also manifest irritable mood are those most likely to be at risk for ODD and mood disorders, and such irritable mood is closely linked to having ADHD-C. Children with ADHD would therefore require the development of just one or two additional symptoms from the social interaction conflict component before crossing the requisite diagnostic threshold of four symptoms for ODD. Indeed, given that verbal aggression has been shown to be part of the greater anger evident in ADHD (Harty et al., 2009), a fourth symptom of ODD may already exist in those with ADHD (stubbornness, defiance, refusal to obey). Further supporting this view that it is the negative emotionality dimension of ADHD that links it to ODD, genetic studies using twins indicate that a substantial portion of the additive genetic, nonadditive genetic, and nonshared environmental contributions to ADHD, ODD, and CD are in fact shared with negative emotionality (Singh & Waldman, 2010). This rise in the occurrence and severity of the social-interactional component of ODD in children with ADHD and its

persistence over time may have as much to do with the severity of ADHD and parenting stress, parental psychopathology, its relationship to disrupted parenting, and severity of parent conflict as with biogenetic contributors associated with ADHD and the EI–DESR component of ODD (August et al., 1999; Burke et al., 2008; Goldstein, Harvey, & Friedman-Weieneth, 2007; Goldstein, Harvey, Friedman-Weieneth, et al., 2007; Johnston & Mash, 2001).

It may well be that this rise in the social conflict component of ODD further accounts for its developmental linkage to concurrent and later CD, and the reason that ADHD is often seen as a precursor to CD. The link between ADHD and CD appears to be largely mediated by the development of ODD in that developmental pathway (Angold et al., 1999; Biederman, Petty, Dolan, et al., 2008; Biederman, Petty, Monuteaux, et al., 2008; van Lier, van der Ende, Koot, & Verhulst, 2007; Rowe, Maughan, Pickles, Costello, & Angold, 2002; Whittinger, Langley, Fowler, Thomas, & Thapar, 2007), at least in males, and between ADHD and major depression–anxiety in both males and females with ADHD and ODD (Biederman, Petty, Dolan, et al., 2008; Biederman, Petty, Monuteaux, et al., 2008; van Lier et al., 2007; Rowe et al., 2002).

The causal or at least contributory link of ADHD to ODD is also evident in the earlier findings that it is largely the poor inhibition symptoms (impulsivity) of ADHD that show the strongest link to risk for ODD specifically and its reflection in the more general ratings of conduct problems (Martel & Nigg, 2006). Symptoms of EI–DESR have been repeatedly shown to be an inherent part of the impulsive dimension of ADHD (see earlier discussion) and therefore may easily provide the explanatory link of this dimension to ODD symptoms. In fact, some factor-analytic studies of both ADHD and ODD symptoms reveal that the impulsive symptoms in ADHD also gross-load substantially onto the factor representing ODD nearly as much as they do on the HI factor of ADHD (Burns, Boe, Walsh, Sommers-Flanagan, & Teegarden, 2001). Intervention studies likewise show that improvements in ADHD symptoms are closely associated with improvements in ODD symptoms (Biederman et al., 2007; MTA Cooperative Group, 1999). These findings are consistent with the hypothesis presented here that some ODD symptoms are a consequence of ADHD itself and especially its EI–DESR component. Thus, reducing ADHD symptoms using medications should result in nearly comparable reductions in symptoms of ODD.

The available evidence can at least be taken to suggest that the high comorbidity between ADHD and ODD is most likely driven in large part by the EI–DESR component of ADHD as part of the larger impulsivity dimension of ADHD. This problem with emotional impulsiveness and poor self-regulation of elicited emotions is especially problematic for negative affectivity, and specifically for low frustration tolerance, quickness to anger, emotional excitability or reactivity, and hostility or reactive aggression. Thus, ADHD strongly predisposes children toward the emotional component of ODD requiring that they simply need to develop just one symptom from the behavioral social conflict component of ODD to become fully diagnosable as having ODD. It seems likely that this evolution of ADHD with subthreshold emotional ODD to full ODD with the additional social conflict component may be associated with or a consequence of the family environment (specifically, disrupted parenting and related parental psychopathology, family stressors, and more severe marital conflict tactics). The point here is that this link would be far less obvious if the role of EI–DESR as a core element of ADHD were not made explicit.

This pathway by which ADHD contributes to risk for ODD through the former's EI–DESR component not only helps us better to understand the comorbidity of ADHD with ODD across development, but it also suggests one pathway through which EI–DESR then contributes to the risk for depression and anxiety later in adolescence. As noted earlier, research indicates that it is the emotional dysregulation component of ODD that is associated with those downstream developmental risks, and that component is largely a function of the coexistence of ADHD with ODD. As Martel (2009) also noted, the EI component of ADHD is part of a general liability for all three externalizing disorders. As I argue here, it may also be an indirect pathway for later risks for other emotion-related comorbidities evident in ADHD.

THE IMPORTANCE OF EI–DESR IN IMPAIRMENTS OF ADHD

So far, I have argued in this chapter that EI-DESR is a core feature of ADHD, and that this creates a significant overlap with the negative emotional component of ODD, thus predisposing those with ADHD to a high risk for developing ODD, among other disorders. This emotional feature of ADHD, however, and its link

to ODD, may also provide a clearer understanding of some of the social impairments that are likely to arise in conjunction with ADHD. My purpose here is not to review the extensive literature on social impairment in ADHD but simply to suggest that one contributor to it is likely to be the EI–DESR component of ADHD.

Within the family, the impulsive emotions and their poor self-regulation of children with ADHD would be expected to increase parental controlling responses and parental expressed emotion as part of the reciprocity involved in parent-child interactions (Danforth, Barkley, & Stokes, 1991; Johnston & Mash, 2001; Mash & Johnston, 1990). As discussed earlier, these interactions, if not dealt with constructively by parents, may foster children's transition from just having the EI–DESR and associated emotional component of ADHD to the behavioral or social conflict component and therefore the likelihood of receiving a formal diagnosis of ODD. Certainly, the excessive activity, poor sustained attention, and verbal and motor impulsiveness in ADHD would by themselves be expected to result in increased controlling responses from parents, as they clearly seem to do, with a commensurate diminution in such responses when the child is treated with stimulants (Danforth et al., 1991). Longitudinal studies likewise are in keeping with a greater effect of child ADHD on parental behavior than of parental behavior on child ADHD (Burke et al., 2008; Lifford, Harold, & Thapar, 2008). The evidence is substantial that ADHD in children is associated with significantly elevated reports of parenting stress and family conflict, poorer parenting practices, greater parental commands and punishment, vacillation between lax and harsh punishment, and a reduced sense of parenting competence (Fischer, 1990; Johnston & Mash, 2001; Mash & Johnston, 1990). Observational studies of parent–child interactions find that increased levels of parental commands and instructions, reduced parental responsiveness to child-initiated interactions, greater parental negativity, and greater levels of parental rejection and coercion yield poorer child compliance (Johnston & Mash, 2001). While these findings for ratings and observations are often more severe in families having children with both ADHD and ODD (or conduct problems), they are higher in families with children who have ADHD only than in control groups. This illustrates the point that ADHD alone is sufficient to increase parent–child conflict and stress.

It is quite possible that the degree of child EI–DESR is one contributor to such conflicts, along with the more traditional dyad of ADHD symptoms, and that ADHD medication management that results in changes in the EI–DESR component contributes to improved parent–child interactions. Suggestive evidence of this is found in the further increases in parent–child conflict and parenting stress that are often found when ODD symptoms are also elevated (Barkley et al., 2008; Johnston & Mash, 2001). The effect of ADHD medications on parent–child interactions is largely to reduce both the child's negative behavior and noncompliance, and it is these changes that result in reduced parental controlling behavior and negativity toward the child (Danforth et al., 1991; Johnston & Mash, 2001). Although far less studied, the same appears to be true of the teacher–child interactions of children with ADHD and the effects of medication treatment (Whalen, Henker, & Dotemoto, 1980). It is not unreasonable to assume that the EI–DESR component of ADHD contributes partially to children's interaction problems with adult authorities and that it is the resulting changes in this component by ADHD medications that partially result in improvements in these interactions.

Also supportive of the argument made here are findings that children with ADHD-C have higher levels of family conflict and parenting stress than do children with ADHD-PI (Lewis, 1992; Paternite, Loney, & Roberts, 1996). Since the EI-DESR element of ADHD has been shown to be most closely linked to the HI symptom dimension and this is more severe in children with ADHD-C than in those with ADHD-PI, this would imply that such EI–DESR problems may be a contributor to these problems with family functioning. Unfortunately, studies of both ADHD and its comorbidity with ODD and their impact on parenting and parent–child relations have not usually singled out the EI–DESR component of ADHD for its contribution to these problems, so its specific role in these family conflicts remains conjectural at the moment. It may remain so if the EI–DESR component of ADHD is not made more explicit, so as to encourage greater care in future research to examine it relative to the other well-known symptom dyads of ADHD.

The majority of children with ADHD are likely to experience peer rejection (52-80%; Hoza, 2007). They are often rated as scoring lower in terms of peer social preferences, higher in their negative impact on peer relations, and they are less well liked and more likely to have fewer close friends. These problems are not simply a function of comorbidity with other disorders; they appear to arise from ADHD specifically (Hoza et al., 2005; Nijmeijer et al., 2008). And they do not appear

to result from overall lower rates of positive behavior exhibited by children with ADHD because these rates are often close to those found in control children. It is the higher rates of negative interactions they initiate that distinguish between ADHD and control groups (Abikoff et al., 2004; Whalen & Henker, 1985). As noted by Whalen and Henker (1992), the emotional dysregulation seen in children with ADHD is a major contributor to disrupting the smoothness, reciprocity, and cooperative activities involved in peer relationships. Recent evidence suggests that peer relationship problems in children with ADHD are best predicted specifically by their difficulties with following rules, their reduced helping behavior, their *whining*, and their inattention (Mrug, Hoza, Pelham, Gnagy, & Greiner, 2007). Other studies have also found both the level of child impulsiveness and verbal and physical anger and aggression to be important predictors of peer interaction problems and rejection (Buhrmester, Camparo, Christensen, Gonzalez, & Hinshaw, 1992; Melnick & Hinshaw, 2000; Hinshaw, 2003). Such findings support Whalen and Henker's (1992) conclusions about the source of peer relationship problems in children with ADHD and specifically imply that EI–DESR is at least one of the contributing factors to peer relationship difficulties associated with ADHD. This is also the case for typically developing children. Negative emotionality is a major predictor of peer likability (Dougherty, 2006; Schultz, Izard, Stapleton, Buckingham-Howes, & Bear, 2009). As with parent–child and teacher–child interactions, the effects of ADHD medications are largely to reduce such negative emotions, thereby resulting in improved peer relations, often with little or no added benefits to be gained from adding social skills training (Abikoff et al., 2004; Cunningham, Siegel, & Offord, 1985; MTA Cooperative Group, 1999). But as with studies of parent–child relations, the link here between EI–DESR and peer relationships problems in ADHD needs more study, but it is likely to be proven correct given the evidence that in typical children negative emotionality is a major predictor of social status (Dougherty, 2006).

Other domains of functional impairment, particularly in adults, may also be related at least in part to the degree of EI–DESR present in cases of ADHD. Skirrow and Asherson (2013) found that emotional lability in adults with ADHD is a significant predictor of impairment in daily life independent of the other two ADHD symptom dimensions. Later research by Surman and colleagues (2013) found similar results, and that EI–DESR predicted lower quality of life and social adjustment. My colleagues and I also examined predictors of being fired or dismissed from employment and those of poor work performance as rated by supervisors in adults with ADHD. We found that the degree of ADHD symptoms significantly predicted the latter work performance problems, but level of emotional impulsiveness was predictive of occupational impairment generally and the percentage of jobs from which the adult had been fired specifically (Barkley & Fischer, 2010; Barkley & Murphy, 2010). Others have also found emotional problems to be a major pathway from ADHD to impaired occupational outcomes (Gjervan, Hjemdal, & Nordahl, in press).

Just as symptoms of EI–DESR may impair the peer and cohabiting relationships of adults with ADHD, they may also interfere with their cohabiting/marital relationships. There are only a few studies of the dating and marital relations of teens and adults with ADHD, but they indicate greater levels of conflict and dissatisfaction relative to control samples (Barkley et al., 2008). It seems quite likely that problems with EI–DESR may be one factor in such relationship strife, as might persistent ODD that is related to it. This was indeed found to be the case in subsequent analyses of these two large databases of adults with ADHD and children growing up with ADHD followed as adults (Barkley & Fischer, 2010; Barkley & Murphy, 2010). Surman and colleagues (2013) later replicated these findings in their large study of adults with ADHD.

Parenting may also be adversely affected by the problems with EI–DESR in adults with ADHD. Studies of parents with ADHD have revealed higher levels of family conflict more generally and lower levels of cohesion (Biederman, Faraone, & Monuteaux, 2002). Research using more specific measures of parenting find greater negative parenting, higher ratings of expressed negative emotion, and lower levels of positive parenting in mothers with high levels of ADHD symptoms (Chronis-Toscano et al., 2008; Psychogiou, Daley, Thompson, & Sonuga-Barke, 2008) and more negative, critical, overreactive, and authoritarian parenting in fathers with higher symptom levels of ADHD (Arnold, O'Leary, & Edwards, 1997). Adult self-reports of EI symptoms on the Conners Adult ADHD Rating Scale were found to be significantly associated with the findings for both negative and positive parenting (Chronis-Tuscano et al., 2008), supporting the argument here that EI–DESR is one factor contributing to the parenting problems of adults with ADHD. While one factor in such disrupted

parenting is certainly the fact that 40–60% of the children of adults with ADHD have the same disorder, as well as ODD, or at least more symptoms of both than do control groups (Barkley et al., 2008), problematic parenting remains evident even after controlling for related disorders in the children (Murray & Johnston, 2006), suggesting that EI–DESR may be one contributor to impaired parenting in adults with ADHD. It is certainly a predictor of both parenting stress among adults with ADHD and the degree of oppositionality in their offspring (Barkley & Murphy, 2010) and siblings (Sobanski et al., 2010; Surman et al., 2011).

Research on the driving of people with ADHD indicates not only numerous deficits and adverse outcomes in this domain of major life activity (Barkley & Cox, 2007) but also has specifically noted elevated rates of driving anger, hostility, and aggression (road rage) among both adults with ADHD and college students showing elevated rates of ADHD (Richards et al., 2006). Road rage is a major contributor to risk for traffic citations and car crashes (see Richards et al., 2006) above and beyond factors such as driver inattention. This implies that problems with EI–DESR may make a specific contribution to the driving problems of adults with ADHD, above and beyond just their problems with attention. Subsequent research indeed has found this to be the case, with EI making unique contributions to crash risk (Barkley & Fischer, 2010; Surman et al., 2013) and to driving while intoxicated, above and beyond contributions made by traditional ADHD symptoms.

Likewise, a few studies have found a link between EI–DESR in adults with ADHD and their higher risk for arrest rates (Barkley & Fischer, 2010; Barkley & Murphy, 2010; Surman et al., 2013). Important to note, once again, is that these contributions are over and above those made by the traditional ADHD symptom dimensions in predicting these adverse outcomes.

Although all this evidence is suggestive of a link between EI–DESR problems inherent in ADHD and these various areas of impairment, such a link has not been established directly. This is largely because the EI–DESR aspects of ADHD have not been considered to be a central component of ADHD and are therefore not likely to be evaluated directly in research in these and other areas of impairment arising from the disorder. So long as a central role of emotional dysregulation is unacknowledged or at least underappreciated as being a part of ADHD, as Harty and colleagues (2009) suggested, its importance in research on impairment may remain understudied.

CONCLUSIONS AND CLINICAL IMPLICATIONS

In this chapter I have argued that EI–DESR is a core feature of ADHD that deserves to be represented in its own right both in conceptualizations of the disorder, as it is in current theories, and in diagnostic criteria for the disorder, as it has not been since DSM-II. A similar stance has been taken by other reviewers as well (Corbisiero, Stieglitz, Retz, & Roster, 2013; Martel, 2009; Skirrow et al., 2009). This argument is based on the historical record over its initial 175-year history, until the 1960s and 1970s, which considered problems with emotion regulation to be part of the disorder. At that time, this component of the disorder was split off from its conceptualization and relegated to the status of an associated feature, if mentioned at all. However, current theories of the disorder have resurrected its place in the nature of ADHD itself and in doing so is supported by findings on the neuroanatomical basis of ADHD and the association of that neural network with EI–DESR. This EI–DESR element of ADHD is probably a specific consequence of the neurodevelopmental abnormalities evident in the frontal–limbic pathway (dorsolateral PFC and ACC) of the brain and particularly the top-down governing (cognitive–effortful) influence of these structures (via the ACC) over the amygdala specifically and the limbic system more generally(emotional brain). As shown here, there is a growing body of evidence that both children and adults with ADHD actually do have significant EI symptoms. Problems with DESR also seem evident but have been far less studied in research on ADHD.

Acknowledging the place of EI–DESR in ADHD also contributes to our understanding of the high comorbidity of ADHD and ODD, and the social impairments associated with the disorder. Having ADHD-C virtually creates a borderline case of ODD in children because these EI symptoms constitute at least three to four of the eight symptoms on the diagnostic symptom list for ODD (DSM-5). I have attempted to clarify that EI–DESR is not just an associated feature of ADHD or a mere function of comorbidity, that it is inherent in the disorder itself. Making its presence explicit in our conceptualizations of ADHD and its diagnostic criteria can serve to better illustrate just why ADHD shows such a high rate of comorbidity with ODD and with reactive aggression. ADHD is surely not the sole or exclusive cause of ODD, but it does strongly predispose those with the disorder to at least the three to four emotion-based symptoms of ODD (its emotional component).

When combined with disrupted parenting and other social and situational factors, it is but a small step (one symptom) to developing the behavioral or social conflict component of ODD, and thereby crossing the diagnostic threshold into clinically diagnosable ODD.

Research on the family, teacher, and peer relations of individuals with ADHD suggests that the EI–DESR aspects of ADHD are major contributors to the problems experienced in these relationships. Therefore, as noted in Chapter 23 on social skills training, one major component of such intervention must target the emotional dysregulation that so often drives the social rejection of these children and adults, in addition to more traditional social behaviors such as sharing, cooperation, taking turns, and so forth.

Adding EI–DESR back into ADHD also helps one to understand the impact ADHD medications may have on this domain. If ADHD includes this EI component, then medical treatments that succeed in reducing ADHD symptoms should likely impact the emotional ones as well. Research on the medications for ADHD has long suggested just that; the reduction of ADHD symptoms by medications is also associated with a reduction in associated ODD symptoms; the two reductions are highly correlated with each other. This, as I have argued, likely occurs through the impact of medications on the "hot" EI–DESR executive network component of ADHD. But the different ADHD medication types may well achieve this effect on EI–DESR via different routes. The foregoing neuroimaging research implies that stimulants may act on emotion largely by dampening or even suppressing limbic system activity, an effect that does not seem to occur with the nonstimulants. Hence, high dosing with stimulants could lead to emotional blunting or constriction of normal affect. In contrast, nonstimulants such as atomoxetine may work by activating anterior cingulate and frontal executive networks, thus facilitating the executive management of emotions generated via the limbic system rather than suppressing them. Guanfacine XR (extended release) and related drugs may achieve their emotion-regulating properties through finer tuning of the $alpha_2$ receptors in the frontal cortex and therefore achieve clearer neuronal signaling that results in executive control over emotional states. All this is admittedly speculative, based on a very limited set of findings on the differences in effects of ADHD medications evident in functional neuroimaging studies. But for now it may help clinicians better understand the differing impacts of ADHD medication types on emotion regulation.

Future revisions to the diagnostic criteria for ADHD (i.e., DSM-6) would do well to list explicitly at least the EI symptoms evident in ADHD, most likely on the impulsivity dimension, in order to underscore the importance of this aspect of ADHD beyond just the inattention and HI symptoms now showcased in these criteria. Along with reducing the overemphasis on hyperactivity and verbal impulsiveness on this symptom dimension and explicitly adding impulsive behavior and decision making to it, the inclusion of symptoms of EI–DESR would serve to represent better our current conceptualization of the disorder. This would also encourage investigators to study this aspect of ADHD explicitly in their research on comorbidity, impairments, and treatment response.

Regardless of what the next DSM may include, clinicians need to be cognizant of the EI–DESR symptoms inherent in ADHD and evaluate them as much as they evaluate the traditional ADHD symptoms during their initial assessment of a patient for ADHD. Doing so can provide not only a clearer and more comprehensive account of the patient's current status but also a richer understanding of the basis for many of the impairments the patient may be experiencing that are partly or largely a consequence of this emotional component of ADHD. That ADHD includes such a component likewise needs to be explained by clinicians to their patients with ADHD and their families, so that they, too, gain such a better, more complete understanding of the condition and why patients may emote as they do. Interventions need to target this component of ADHD in addition to the ongoing efforts to develop both psychosocial and medical interventions that focus on the traditional symptom complex of ADHD and its related "cold" cognitive executive deficits, and how best to help family members cope with and assist the patient with ADHD in the effective management of their emotional dysregulation.

Yet none of this is to suggest that all of the emotional difficulties seen in a patient with ADHD can be written off to the emotional dysregulation component championed in this chapter. As subsequent chapters make plain, ADHD is certainly associated with an elevated risk for various mood and anxiety disorders. How, then, are we to distinguish which affective disturbances belong to ADHD and which require the search for a comorbid affective disorder to account for them? There is little direct research on the issue, but the findings to date suggest some tentative clinical guidelines for such differential diagnosis. First, con-

sider that the emotional disturbances in ADHD are just that—emotions, and not moods. Emotions are of short duration, are provoked, and often are situation-specific to the setting of the provocation. They are also largely rational, which is to say understandable, to others given that typical people would have had the same subjective reaction to the provocation. But the difference is that the typical person would have acted to suppress the voluntary aspects of the emotion over which they have some volitional control rather than express it publicly. They would then have engaged in the self-regulatory steps to down-regulate or otherwise alter the emotion to make it more compatible with the situation, others, and the person's longer-term goals and welfare. Recovery from such a provoked change in emotional state can be relatively quick compared to a change in mood, though perhaps not as easily as is seen in typical people given that those with ADHD have more difficulties down-regulating strong emotions using executive self-control. In contrast, a mood is just that—a long-duration change in emotional state that is often cross-situational and may arise without provocation or from trivial events that would often not have led others to react in this fashion. It can be described as capricious, as well as extreme. Consequently, it is not rational in the sense that other people would have the same emotional state under these circumstances over such an extended period of time and across settings. Admittedly, the dividing line between an emotion and a mood is not as crisp as is portrayed here. But the previous guidelines seem sensible at this time to guide clinicians in sorting out what affective symptoms of a patient with ADHD belong to that disorder and its EI–DESR problems, and what symptoms are likely to be attributable to a comorbid disorder.

In summary, it is time to return EI–DESR to its rightful place in the core or central components of ADHD.

KEY CLINICAL POINTS

- ✓ During the first 170 years of its medical history, ADHD and its precursor disorders were believed to involve deficits in emotional inhibition and self-regulation, along with the core problems with attention and HI behavior.
- ✓ Beginning in the 1960s, especially with DSM-II, the symptoms of EI and DESR were divorced from the core deficits of ADHD, and treated as merely associated problems that may arise in some cases.
- ✓ Compelling evidence now argues for the return of EI–DESR to the status of a core component of ADHD in its conceptualization and DSM diagnostic criteria. The argument is based on six lines of reasoning and evidence:
  1. EI–DESR has a long history of being a central feature of ADHD in its clinical conceptualization.
  2. Current neuropsychological theories of ADHD consider EI–DESR to be just such a central component.
  3. The neuroanatomical findings associated with ADHD would have to give rise to commensurate symptoms of EI–DESR.
  4. Ample evidence now exists that children and adults with ADHD are highly likely to manifest EI–DESR (low frustration tolerance, impatience, quickness to anger, and being easily excited to emotional reactions more generally).
  5. Returning EI-DESR to a central place in ADHD would more clearly show the basis for its high comorbidity with ODD and probably several related disorders.
  6. Promoting EI–DESR back to the status of a core component would also clarify one basis for the frequent social interaction problems and impairments in several other domains of major life activities (work, driving, marriage/cohabiting, and parenting) seen in ADHD.
- ✓ Understanding the role of EI–DESR in ADHD would assist with differential diagnosis and reduce misdiagnosing emotional problems in ADHD as entirely arising from comorbidity.
- ✓ ADHD medications appear to reduce the EI–DESR component of ADHD as much as they do traditional ADHD symptom dimensions, yet they may do so through different neural mechanisms and networks.
- ✓ Psychosocial interventions for ADHD should include programs targeted at helping patients with EI–DESR specifically rather than just traditional ADHD symptom dimensions.

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CHAPTER 4

Developmental and Neuropsychological Deficits in Children with ADHD

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As discussed in Chapter 2, attention-deficit/hyperactivity disorder (ADHD) is characterized by developmentally inappropriate levels of inattention, impulsivity, and/or hyperactivity, and the degree to which these symptoms are manifested varies by presentation. In addition to deficits in these core areas, children with ADHD often have developmental and neuropsychologically related difficulties. Our purpose in this chapter is to discuss developmental and neuropsychological findings, and to address implications of this body of literature for clinicians. It is important to note that the material covered is a selective rather than a systematic or exhaustive review, and references are provided for additional information. The developmental areas we cover include general adaptive functioning, motor coordination, deficits associated with language and learning, self-regulation, and self-perceptions. Neuropsychological topics include findings from intellectual and executive function studies. Last, we review methodological limitations of the current literature and advance suggestions for future research.

DEVELOPMENTAL FUNCTIONING

In terms of developmental functioning, summarized in Table 4.1, ADHD is commonly conceptualized as a dynamic disorder as opposed to a static condition that unfolds across the lifespan (Halperin & Healey, 2011). It emerges in early childhood and in many cases continues into adulthood (see Chapter 9; also see Barkley, Fischer, Smallish, & Fletcher, 2006; Barkley, Murphy, & Fischer, 2008; Faraone, Biederman, & Mick, 2006; Kessler et al., 2006). Due to its dynamic nature, researchers and clinicians have argued that ADHD is best viewed within the context of a developmental trajectory, wherein many of the factors contributing to the diverse outcomes of individuals with ADHD may be identified (Halperin & Healey, 2011). Throughout the years, research has identified numerous developmental deficits associated with ADHD that can greatly impact the outcomes of individuals with the disorder (Barkley, 2006).

Adaptive Functioning

As children progress through the various stages of development into adolescence and adulthood, they are expected gradually to attain "adaptive skills," typically defined as the ability to function in everyday activities. Adaptive skills include, for example, independence and self-help skills, self-knowledge, motor skills, and social and communication skills (Sparrow, Balla, & Cicchetti, 1984). Given the functional impairment associated

TABLE 4.1. Summary of Developmental Functioning

Adaptive functioning

- Adaptive functioning is generally poorer in children with ADHD than in typically developing children.
- Deficits are common in the areas of daily living and social communication (e.g., organizing information, effective communication).
- Difficulties appear to be related to ADHD symptomatology and not limited to externalizing symptoms of ODD and CD.

Motor coordination

- A large percentage of children with ADHD exhibit some type of motor coordination problems.
- Impairments often appear in the motor skills domains of strength, visual–motor coordination, adjusting speed, and dexterity.
- Evidence suggests that ADHD inattentive or ADHD combined subtypes are more likely to display motor coordination difficulties compared to those with the ADHD hyperactive–impulsive subtype.
- Manual dexterity difficulties are often the most impaired domain of motor coordination (e.g., writing, drawing, and playing a musical instrument).
- Stimulant medication and physical therapy are often effective at improving motor deficits in children with ADHD.

Language ability

- Linguistic difficulties are prevalent in children with ADHD and may be identified as early as during the preschool years.
- Difficulties are often evident with receptive, expressive, and pragmatic language skills
- Linguistic deficits may cause difficulty comprehending instructions, making inferences about social context, and initiating, maintaining, and ending a conversation.
- Language ability may have an impact on the social functioning of children with ADHD.
- Social difficulties may further exacerbate the language deficits associated with ADHD.
- Subtypes of ADHD may be differentially associated with difficulties in different domains of language ability.

Learning difficulties

- Learning disabilities and academic underachievement are more common in children with ADHD than in the general population.
- Comorbidity rate of LDs and ADHD may be as high as 45%.
- Children with ADHD who do not meet diagnostic criteria for an LD often have some degree of learning difficulties.
- Common coexisting learning problems include lower academic achievement, use of special education services, grade retention, higher rates of high school dropout, and lower rates of postsecondary education.
- Evidence suggests behavior-based strategies are the most effective in addressing the academic difficulties of children with ADHD.

Self-perceptions

- Studies suggest that children with ADHD often display an inflated self-esteem.
- Internalization of speech may be an important component of self-regulation.
- Children with ADHD appear to demonstrate a different, potentially less-developed pattern of private speech than children without ADHD.

with ADHD (American Psychiatric Association, 2013) numerous investigations have explored the extent to which ADHD affects adaptive functioning. In general, studies have found that the adaptive skills of children with ADHD are poorer than those of their typically developing peers (Jarratt, Riccio, & Siekierski, 2005; Reynolds & Kamphaus, 2002; Stein, Szumowski, Blondis, & Roizen, 1995). Specifically, children with ADHD have been found to have difficulties in multiple areas of daily living and in social communication (Stein et al., 1995). Manifestations of such difficulties include deficiencies in organizing information and communicating it effectively, responding inappropriately to others, and excessive talking (Roizen, Blondis, Irwin, & Stein, 1994).

For instance, Stein and colleagues (1995) compared children with ADHD to those with a mild intellectual disability or pervasive developmental disorder and found that the discrepancy between intelligence and adaptive functioning was *greater* for children with ADHD in the areas of communication and daily living, even after they controlled for externalizing symptoms of oppositional defiant disorder (ODD) and conduct disorder (CD). With regard to the socialization domain of adaptive functioning, however, group differences disappeared after the authors accounted for symptoms of ODD and CD. In contrast, Clark, Prior, and Kinsella (2002) reported that in adolescents with ADHD, socialization was impaired not only in those with ADHD and ODD/CD combined, but also in those with ADHD without externalizing problems. The findings by Clark and colleagues corroborate results from an earlier longitudinal epidemiological study by Taylor, Chadwick, Heptinstall, and Danckaerts (1996) suggesting that adolescents both with and without ODD/CD experience difficulties with social adjustment. Furthermore, Clark and colleagues found that executive functions (EFs) significantly predicted adaptive functioning in the domains of communication and socialization, and compared to measures of verbal intelligence, EFs (see discussion of EFs as they relate to ADHD later in this chapter) were a stronger predictor of socialization skills. Stavro, Ettenhofer, and Nigg (2007) further explored the relationship between EFs and adaptive behavior, and found that symptoms of inattention accounted for more variance in adaptive functioning than hyperactive–impulsive symptoms. Although EF was initially found to be associated with adaptive functioning, the relationship disappeared after researchers controlled for ADHD symptoms. In addition, when adult participants retrospectively rated ADHD symptoms in childhood, the findings revealed that only inattention was related to EF or adaptive functioning. Stavro and colleagues concluded that EF may exert its effects on adaptive functioning through the behavioral path between inattention and adaptive functioning. Alternatively, ADHD symptoms can be viewed as a subset of and are highly correlated with EF behaviors in daily life, as detected by EF rating scales (Barkley, 2012a, 2013). In that case, controlling for ADHD symptoms would remove much of the variance in adaptive functioning owing to EF deficits. Recently, Ware and colleagues (2012) compared adaptive and EF functioning in children with ADHD and children exposed to prenatal alcohol, and found that both groups had adaptive behavior deficits. The adaptive behavior deficits in children with ADHD, however, were more general than those found in children with prenatal exposure to alcohol.

A number of other studies also found children with ADHD to have diminished overall adaptive functioning in comparison to normal or control groups of children (Barkley, Fischer, Edelbrock, & Smallish, 1990; Greene et al., 1996; Shelton et al., 1998; Ware et al., 2012). Collectively, these studies suggest that children with ADHD are more likely to display poorer adaptive functioning, especially in the areas of communication and socialization, than their same age peers. These findings remain robust even after researchers control for IQ (Stein et al., 1995). Furthermore, the disparity between cognitive functioning and adaptive skills appears to be greater for children with ADHD than for those with other developmental disabilities, suggesting that ADHD symptoms may have a specific detrimental effect on adaptive skills. Additionally, the greater the disparity between adaptive functioning and IQ, the more severe the ADHD, risk for comorbidity, and deficits in academic functioning (Shelton et al., 1998). Clinicians should therefore be especially attentive to possible adaptive behavior deficits when working with children with ADHD and include interventions to address these deficits in their treatment protocols.

Motor Coordination

One of the most prominent theories of ADHD, proposed by Barkley (1997, 2012b), stated that a primary impairment of ADHD involves diminished behavioral inhibition that leads to deficits in four domains: (1) nonverbal working memory, (2) self-regulation of

affect, motivation and arousal, (3) internalization of speech, and (4) reconstitution. Barkley (1997) further asserted that impairment in these four domains adversely affects motor control. Indeed, impaired motor control is a common co-occurring condition in ADHD, with studies suggesting that more than 50% of children with ADHD manifest some type of motor coordination deficit. In addition, research suggests that a large percentage of children with ADHD also meet criteria for developmental coordination disorder (DCD), perhaps up to 50% (Brossard-Racine, Shevell, Snider, Bélanger, & Majnemer, 2012; Watemberg, Waiserberg, Zuk, & Lerman-Sagie, 2007). Several studies have documented that even in the absence of DCD, children with ADHD demonstrate deficient motor coordination compared to their peers, although not all studies substantiate this finding (Brossard-Racine et al., 2012). In a review of 49 studies, Harvey and Reid (2003) concluded that many children with ADHD are at a risk for compromised motor skills, including strength, visual–motor coordination, speed, and dexterity; in many cases these impairments may be lifelong. Harvey and Reid also noted, however, that some children with ADHD may demonstrate excellent motor coordination and/or skills comparable to those of their peers. Interestingly, limited evidence indicates that those with inattentive or combined DSM-IV subtypes were more likely to display poor motor coordination than those of the hyperactive–impulsive subtype (Pitcher, Piek, & Hay, 2003; Watemberg et al., 2007).

Manual dexterity, including writing, drawing, and playing a musical instrument, is frequently cited as the most impaired domain of motor coordination in children with ADHD (Brossard-Racine et al., 2012; Flapper, Houwen, & Schoemaker, 2006; Pitcher et al., 2003). In addition, time perception, which frequently is impaired in individuals with ADHD, can affect motor coordination in various tasks, for example, when playing sports or driving (Barkley, Murphy, & Bush, 2001; Yang et al., 2007). For example, Chen and colleagues (2013) compared small groups of children with and without ADHD on a jump rope task and found that children with ADHD tended to have poorer hand–foot coordination, greater difficulty modifying their pace, and more trouble adjusting to the speed of the task than the comparison children.

Fliers and colleagues (2008) investigated whether the co-occurrence of ADHD and motor problems was comparable across age, ADHD DSM-IV subtype, and gender, using teacher and parent ratings of motor coordination skills of children and adolescents with and without ADHD. Results suggested that males and females were equally affected by motor coordination deficits, although females without ADHD were rated as having fewer motor problems than males without ADHD, resulting in a larger discrepancy between females with and without ADHD than between males with and without ADHD. Additionally, inattention was a stronger predictor of motor deficits than hyperactivity–impulsivity, and, as stated previously, this finding has been reported in other studies (e.g., Martin, Piek, & Hay, 2006). It is important to note, however, that some studies have also found that hyperactivity/impulsivity predicts motor skills (Fliers et al., 2008). For example, Piek, Pitcher, and Hay (1999) found that inattention was a better predictor of fine motor skills deficits, but there was also evidence to suggest that hyperactivity–impulsivity symptoms were a better predictor of gross motor skills. Although preliminary, findings suggest that stimulant medication (Bart, Daniel, Dan, Bar-Haim, 2013; Brossard-Racine et al., 2012; Flapper et al., 2006) and physical therapy (Watemberg et al., 2007) may ameliorate the motor coordination difficulties commonly found in ADHD.

In summary, studies suggest that the motor skills of children with ADHD are often poorer than those of their peers, and in many cases these children meet diagnostic criteria for DCD. Of course, variation exists, and some children with ADHD do not have motor coordination difficulties, or they may even have excellent motor coordination. The contribution of different DSM-5 subtypes or symptom dimensions of ADHD to motor deficits is unclear, although preliminary evidence suggests that the association between inattention, rather than hyperactivity–impulsivity, and motor difficulties is stronger. Clinicians should be aware of the co-occurrence of ADHD and motor coordination deficits, and consider this information when assessing children for ADHD and addressing treatment interventions. Last, preliminary evidence supports the possibility that stimulant medication and physical therapy may be helpful interventions for improving the motor skills of individuals with ADHD.

Language Ability

Language ability is one of the foundations for effective social communication and, overall, successful development and research have found that linguistic difficulties are prevalent in children with ADHD (Peterson et al.,

2013). For example, Tirosh and Cohen (1998) reported a 45% rate of language deficits among children in a community sample identified as having ADHD, using "nonconservative criteria" (p. 495). In a clinical sample of children with ADHD, Bruce, Thernlund, and Nettelbladt (2006) reported ratings indicating that compared to typically developing peers, 67% of participants had linguistic problems above the 90th percentile. Both receptive language (Bruce et al., 2006; McInnes, Humphries, Hogg-Johnson, & Tannock, 2003; Wassenberg et al., 2010) and expressive language (Humphries, Koltun, Malone, & Roberts, 1994; Kim & Kaiser, 2000) appear to be affected. "Receptive language" concerns the understanding of language or messages produced by others, whereas "expressive language" refers to the production of messages sent to others (Wicks-Nelson & Israel, 2009). In addition, findings reported by McInnes and colleagues (2003) suggest that children with ADHD *without* any apparent language impairment also demonstrate subtle listening comprehension deficits. Specifically, McInnes and colleagues found that despite having adequate language abilities, as measured by a standardized language test, the children demonstrated minor comprehension deficits while listening to spoken passages. For example, the children with ADHD had more difficulty comprehending instructions and making inferences from the passages, even when the researchers controlled for decoding and sentence formulation abilities, although they comprehended factual information as well as typically developing children. In another study, Staikova, Gomes, Tartter, McCabe, and Halperin (2013) examined "pragmatic" use of language in children with ADHD, which can be defined as the conversational use of language, including both verbal and nonverbal skills, and found that the children had a number of pragmatic language deficits. More importantly, these deficits were evident when researchers controlled for general language functioning, and they were observed in the areas of discourse management (initiating, maintaining, and ending a conversation), presupposition (i.e., theory of mind, assumptions about the social context of conversation), and narration. Additionally, Staikova and colleagues found that discourse management mediated the relationship between ADHD and social difficulties, suggesting that ADHD symptoms lead to difficulties with language communication, particularly conversation management, which in turn lead to social difficulties.

Language difficulties associated with ADHD may be detected as early as the preschool years. Specifically, Gremillion and Martel (in press) recently compared preschoolers with ADHD to those with comorbid ODD and to preschoolers with no disability and found that preschoolers with ADHD had significantly poorer expressive and pragmatic language compared to both typically developing children and children with ODD only. Symptoms of hyperactivity–impulsivity were found to contribute primarily to poor language skills, while symptoms of inattention were more strongly associated with weaker receptive and expressive vocabulary.

Collectively, the literature supports the notion that language difficulties often co-occur with ADHD, particularly in the areas of expressive and receptive language, and the pragmatic use of language. Although the language skills of many children with ADHD may be considered age-appropriate according to results of standardized tests, they may nevertheless have slight linguistic difficulties, such as with understanding instructions and making inferences. In addition, difficulties with the pragmatic use of language, such as conversation management, are likely to lead to social difficulties. This relationship is likely to be bidirectional, however, as social communication can significantly contribute to language proficiency (Gremillion & Martel, in press). These findings suggest that initial linguistic problems of children with ADHD can lead to social difficulties, which in turn can limit their opportunities for advancing their language skills, thereby compromising their social skills even further. Clinicians need to be aware of the different types of language difficulties that often beset children with ADHD and to consider this information during assessment and when designing interventions.

Learning Difficulties

It has been well established that language ability is strongly associated with reading ability (Catts, Fey, Tomblin, & Zhang, 2002; Snowling, Bishop, & Stothard, 2000). Not surprisingly, reading disabilities, as well as other types of learning disabilities, are more common in individuals with ADHD than in the general population (Sexton, Gelhorn, Bell, & Classi, 2012). In a recent review of 17 studies assessing the comorbidity of ADHD and learning disabilities (LDs), DuPaul, Gormley, and Laracy (2013) concluded that the comorbidity rate is as high as 45%, which is higher than previous estimates have indicated. DuPaul and colleagues noted, however, that changes to DSM-5 criteria for both

ADHD and LDs may affect these numbers, although it is unclear in which direction. In a population-based sample, Carroll, Maughan, Goodman, and Meltzer (2005) reported that 18.9% of children with ADHD had a comorbid reading disability, whereas Pastor and Reuben (2008) using a population-based sample found reading disability prevalence rates of 44% in children with ADHD. Mayes and Calhoun (2006a) examined the reading abilities of clinic-referred children with ADHD and reported that 33% scored significantly lower on a reading test than predictions based on their Full-Scale IQ. In a similar study investigating LDs in general (not focusing specifically on reading ability), Mayes, Calhoun, and Crowell (2000) found that 70% of clinic-referred children with ADHD had an LD in reading, spelling, writing, or math and that children with both ADHD and an LD had more severe learning problems than children with a learning disability only. Furthermore, the results suggested that many children with ADHD who did not meet diagnostic criteria for an LD had some degree of learning difficulties (Mayes et al., 2000). Conversely, in a prospective, longitudinal study, Washbrook, Propper, and Sayal (2013) found that preschool children with elevated symptoms of inattention and hyperactivity–impulsivity who did not meet diagnostic criteria for ADHD were still at a heightened risk for academic underachievement in adolescence. In a longitudinal study by Scholtens, Rydell, and Yang-Wallentin (2013), ADHD symptoms measured in grades 6, 11, and 12, were stable over time and negatively associated with *both* concurrent and future academic outcomes.

The totality of the findings in the voluminous literature on the overlap of ADHD with LDs documents the overall diminished academic functioning of individuals with ADHD (Weyandt, 2007). Studies have indeed shown that children with ADHD are more likely to demonstrate lower academic achievement, require special education services, experience grade retention, have higher rates of high school dropout, and lower rates of postsecondary education than their peers without ADHD (Chapters 6 and 12; also see Barkley, Fischer, Edelbrock, & Smallish, 1990; Mannuzza, Gittlman-Klein, Bessler, Malloy, & LaPadula, 1993). With regard to academic interventions for children with ADHD, DuPaul and Weyandt (2006) reviewed studies on school-based interventions for students with ADHD and concluded that there is substantial evidence for the effectiveness of behavior-based strategies, and potential support for peer-tutoring and certain recent social skills training strategies to enhance the academic outcomes of children with the disorder (see also Chapters 23 and 24). The implications of this body of work for clinicians are that many children with ADHD have comorbid learning difficulties and/or disabilities, and thorough assessments of both are required. In addition to interventions that target reducing ADHD symptomatology, clinicians should be sensitive to the unique educational needs of children with ADHD and particularly their comorbid LDs.

Self-Perception

Previously, it was generally believed that children with ADHD have low self-esteem due to the numerous challenges they face in everyday activities, and some studies have supported this notion (e.g., Dumas & Pelletier, 1999; Edbom, Lichtenstein, Granlund, & Larsson, 2006). In contrast, many other studies indicate that children with ADHD display an inflated self-esteem or a *positive illusory bias* in perceptions of their own competence, although the evidence regarding this issue has at times been inconclusive (Hoza, Pelham, Dobbs, Owens, & Pillow, 2002). Hoza and colleagues (2002) compared the self-perceptions of boys with ADHD and teacher ratings in academic, behavioral, and social domains, and found that the boys tended to overestimate their ability in all areas. Specifically, boys with ADHD with more externalizing behaviors overestimated their social and behavioral functioning, whereas boys with ADHD who demonstrated academic underachievement overestimated their academic competence in comparison to controls and other boys with ADHD who were functioning well academically. In addition, boys with ADHD and comorbid depressive symptoms had significantly lower ratings of self-worth than control boys except in the behavioral domain, where self-ratings were higher than those of controls. Hoza and colleagues speculated that divergent findings on self-perceptions in children with ADHD may in part stem from whether or not the effects of internalizing disorders, such as depression, on self-perceptions are taken into account. In a more recent study, Hoza and colleagues (2004) found further evidence for the positive illusory bias among children with ADHD who grossly overestimated their competence, particularly in areas where they were the most deficient, regardless of who was used as a comparison rater of competence (i.e., mother, father, or teacher). Both boys and girls overestimated their ability, although girls with and with-

out ADHD had less inflated perceptions compared to teacher ratings in the domains of behavior and physical appearance. It is not so much that children with ADHD view themselves as far more competent than others (grandiosity) as that they report themselves as being as competent as others when they are clearly deficient in that task or activity. Some theorists have speculated that the positive illusory bias serves a protective function, allowing children with ADHD to cope better with the difficulties they experience (Diener & Milich, 1997). Ohan and Johnston (2002), however, only found partial support for this hypothesis, suggesting that it holds true only for perceptions of social competence, as opposed to academic competence.

Internalization of Speech

Factors that may mediate self-regulation and promote problem solving have been explored, and several theorists have argued that the internalization of speech is an important factor in this process (Barkley, 1997; Berk & Landau, 1993; Corkum, Humphries, Mullane, & Theriault, 2008; Ostad & Sorensen, 2007; Vygotsky, 1978). Berk and Landau's (1993) observation that children with LD and ADHD symptoms actively used task-relevant private speech does not support previous ideas regarding a lack of private speech in children with learning difficulties. More recent studies have also corroborated such findings among children with ADHD (e.g., Kopecky, Chang, Klorman, Thatcher, & Borgstedt, 2005). However, Kopecky and colleagues noted that children with ADHD produced more private speech during failure than success than did comparison children. Similarly, in the study by Corkum and colleagues (2008), children with ADHD demonstrated a different pattern of private speech than comparison children in problem-solving and inhibition tasks. On the problem-solving tasks, children with ADHD displayed more task-irrelevant private speech, whereas on the inhibition task, they produced more task-relevant private speech, although their performance on the inhibition tasks was poorer than that of peers. Corkum and colleagues concluded that children with ADHD possess less developed private speech and self-regulation strategies than do children without ADHD, which leads to impaired self-regulation. What remains unclear, however, is whether private speech is a causal factor in the development of successful self-regulation or simply one of several indicators of self-regulation.

In summary, studies reveal that children with ADHD may have inaccurate and positively biased perceptions of self-competence, especially in areas in which they experience the most significant problems, although findings regarding this issue have been inconclusive. The internalization of speech has been proposed as a significant contributing factor to self-regulation, and indeed, children with ADHD demonstrate a different pattern of private speech during problem-solving tasks, although causal inferences regarding the impact of private speech on self-regulation are unwarranted at this time.

Conclusions and Clinical Implications of Developmental Findings

Many children with ADHD demonstrate developmental impairments in the domains of adaptive functioning, motor coordination, language skills, learning ability, and self-regulation of emotion (see Chapter 3), often meeting diagnostic criteria for a disorder in the area in question (e.g., DCD). The severity of ADHD symptoms is positively associated with such impairments, and evidence suggests that ADHD subtypes (or presentations, as in DSM-5) may be differentially associated with adaptive behavior deficits. (e.g., inattention has been more strongly associated with poor motor coordination than with hyperactivity–impulsivity). Research clearly indicates that developmental deficits can worsen the functional impairment of children with ADHD and subsequently lead to increased use of interventions and support services such as medication and individual therapy.

Despite substantial evidence of poorer adaptive functioning among individuals with ADHD compared to normal controls, adaptive skills are generally not an area targeted by ADHD assessment or intervention (Jarratt et al., 2005). Researchers, however, have called for a greater emphasis on addressing adaptive functioning (e.g., Roizen et al., 1994; Stein et al., 1995) when designing intervention programs for individuals with ADHD given that poor adaptive functioning is likely to exacerbate the functional impairment inherent in ADHD. Assessing adaptive functioning specifically may inform interventions and shed light on the strengths and weaknesses of individuals with ADHD. Similarly, motor coordination may be an important prognostic indicator that warrants the attention of clinicians and others working with children and adolescents with ADHD (Fliers et al., 2008). With regard

to ADHD and LDs in particular, evidence-based strategies that address *both* ADHD and LD symptoms should be implemented in school and home settings (DuPaul et al., 2013).

NEUROPSYCHOLOGICAL AND COGNITIVE FUNCTIONING

A summary of neuropsychological and cognitive functioning is presented in Table 4.2.

Intellectual Development

Over the years, authors have touted in books and more recently on blogs and websites that it is advantageous to have ADHD. Some have claimed that individuals with this disorder have more intelligence than their nondisabled peers, or have suggested that children with ADHD are more creative and gifted than their peers (e.g., Hartmann, 1997). Research, however, presents a different picture. Numerous studies have indicated that children with ADHD often obtain significantly lower IQs than children without the disorder. For example, McConaughy, Ivanova, Antshel, and Eiraldi (2009) evaluated over 177 children ages 6–11 using the Wechsler Intelligence Scale for Children—Fourth Edition (WISC-IV) and found that those with ADHD performed significantly lower on the Full-Scale IQ and composite scores than children without ADHD. The difference on standardized intelligence tests is often substantial, with an average difference of approximately 9 points (Barkley, Karlsson, & Pollard, 1985; Faraone et al., 1993; Fischer, Barkley, Fletcher, & Smallish, 1990; Frazier, Demaree, & Youngstrom, 2004; Mariani & Barkley, 1997; McGee, Williams, Moffitt, & Anderson, 1989; Moffitt, 1990; Prior, Leonard, & Wood, 1983; Stewart, Pitts, Craig, & Dieruf, 1966; Tarver-Behring, Barkley, & Karlsson, 1985; Werry, Elkind, & Reeves, 1987). It is important to note, however, that although children with ADHD obtained lower IQ scores than comparison students in a variety of studies, their IQs were generally within the average range and not substantially below average. These findings support the view that, as a group, individuals with ADHD are *not* more intelligent than the general population and often score lower on standardized IQ tests than their nondisabled peers. The bottom line is that levels of intelligence vary among individuals with ADHD just as they do in the general population.

The reasons for the lower levels of intellectual performance often found in children with ADHD relative to their nondisabled peers (and even their siblings) are unclear. Some have suggested that coexisting LDs may account for the difference (Bohline, 1985), or familial factors may be involved (Biederman, Fried, Petty, Mahoney, & Faraone, 2012), whereas others have attempted to determine the influence of attention problems and EF deficits on IQ scores. For example, during administration of the WISC-III, children with ADHD displayed significantly higher levels of aberrant test behaviors, including inattention and externalizing behaviors (McConaughy et al., 2009). To help determine whether ADHD symptoms were related to IQ performance, Sharp and colleagues (2003) studied a sample of 25 monozygotic twins, 10 pairs of whom were discordant for ADHD. Contrary to expectations, the Full-Scale IQ of the discordant pairs was similar, suggesting that attention deficits do not substantially impair IQ test performance. Other studies have also found weak to poor correlations between severity of inattention and impulsivity–hyperactivity symptoms and IQ scores (e.g., Chae, 1999; Naglieri, Goldstein, Delauder, & Schwebach, 2005). The literature is mixed, however, as earlier studies did report a relationship between impulsive–hyperactive behavior and lower IQ scores (Halperin & Gittelman, 1982; Hinshaw, 1992; McGee, Williams, & Feehan, 1992; Sonuga-Barke, Lamparelli, Stevenson, Thompson, & Henry, 1994; Werry et al., 1987). More recently, researchers at the University of Copenhagen reviewed the literature and concluded that the mean influence of attention deficits and ADHD on IQ amounts to 2–5 IQ points (Jepsen, Fagerlund, & Mortensen, 2009). Interestingly, these findings are similar to those found in the general population when ADHD symptoms, not the disorder, are correlated with IQ, suggesting that symptoms of inattention and impulsivity–hyperactivity may affect intellectual test performance in all children, not simply those with ADHD (Goodman, Simonoff, & Stevenson, 1995).

It is also important to note that children with ADHD as a group do not typically perform *better* than their same-age peers on IQ tests (i.e., in the gifted range). An IQ of 120 or higher on the Wechsler scales is considered to be superior and is sometimes used as a cutoff score for giftedness. Operationalization of the construct of giftedness varies, however, and IQ is often only one of many criteria used to determine eligibility for gifted programs. As mentioned previously, many studies have indicated that the performance of children

TABLE 4.2. Summary of Cognitive and Neuropsychological Functioning

Intellectual functioning

- Children with ADHD often perform lower on IQ tests than children without the disorder, and the difference can be as much as 9 points.
- Children with ADHD, however, generally have IQ scores in the average range and not substantially below average.
- Levels of intelligence vary among individuals with ADHD just as they do in the general population.
- Many children with ADHD obtain lower scores on IQ tests; however, some may score above average, below average, or in the gifted range of intelligence.
- Symptoms of inattention and hyperactivity–impulsivity may affect cognitive test performance in all children, not just those with ADHD.
- Profile analysis, although common practice among school psychologists, is controversial and largely criticized by psychometric theory.
- The FDI and other forms of profile analysis of IQ are neither reliable nor valid methods for diagnosing ADHD.

Executive function

- EF encompasses a variety of cognitive abilities that allow for impulse control, strategic planning, cognitive flexibility, and goal-directed behavior.
- EF deficits are characteristic of many, but not all, individuals with ADHD and are present in other childhood disorders.
- Evidence suggests that children with EF deficits have lower academic achievement.
- EF deficits are likely to emerge early in life in children with ADHD, and the impairments tend to persist into adolescence and young adulthood.
- EF deficits are routinely more common and more severe on rating scales of EF than on psychometric EF tests.

Planning

- The EF construct of planning has been conceived in a number of ways, including visual–spatial and strategic planning.
- Deficits in planning are characteristic of some, but not all, children with ADHD.
- Planning deficits are also found in children with other types of clinical disorders and are neither unique to nor diagnostic of ADHD.

Inhibition

- The literature indicates that a variety of EF tasks have been used to measure behavioral inhibition, and the psychometric properties of these tasks vary.
- Many, but not all, children with ADHD perform more poorly on inhibition tasks than do children without the disorder.
- Inhibition tasks do not differentiate reliably between different subtypes of ADHD, although findings are inconclusive, and they are not diagnostic of ADHD.

Working memory

- Many studies have found that children with ADHD perform more poorly than their nondisabled peers on working memory tasks that require memory for digits forward and backward.
- Differences between children with and without ADHD in working memory are even larger when tasks are complex.
- Verbal working memory problems are not unique to ADHD; children with other disorders show similar difficulties. Some studies suggest that nonverbal working memory of children with ADHD may be substantially more impaired than verbal working memory.
- Individuals with ADHD may have deficient time perception (e.g., estimating passage of time processing time, and time discrimination).
- Findings relative to time perception in individuals with ADHD have been mixed, however, and studies are often characterized by methodological problems.

with ADHD is often lower than that of comparison children on IQ tests. However, this is not to say that some children with ADHD are not also gifted (sometimes referred to as "twice exceptional") (Budding & Chidekel, 2012). In fact, Antshel and colleagues (2007) studied 49 children with ADHD and an IQ of 120 or higher and 92 children without ADHD and an IQ of 120 or higher. Results indicated that the performance of children with ADHD who were "gifted" in terms of intelligence was similar on behavioral and academic measures to that of children of average intelligence. For example, both groups repeated more grades, required more academic supports, and were rated more poorly on parent rating scales compared to children without ADHD. Grizenko, Zhang, Polotskaia, and Joober (2012) found that children with Full-Scale IQs of 120 or higher had lower severity of ADHD symptoms compared to those with average (80–119) or borderline (70–79) IQs. Interestingly, Grizenko and colleagues found that all three groups of children responded equally to stimulant medication, with improvements on parent, teacher, and laboratory ratings.

In addition to composite score differences, researchers have explored whether children with ADHD perform differently than controls on specific subtests. This practice, known as "profile analysis," is controversial and largely criticized by psychometric theory (McDermott, Fantuzzo, & Glutting, 1990), yet it remains a common practice among clinicians, including school psychologists, clinical psychologists, and neuropsychologists (Pfeiffer, Reddy, Kletzel, Schmelzler, & Boyer, 2000). In 1980 and again in 1994, Kaufman suggested that the Freedom from Distractibility Factor (FFD) on the WISC-III, which comprised Coding, Arithmetic and Digit Span subtests and later was known as the Freedom from Distractibility Index (FDI) on the WISC-III, which comprised Arithmetic and Digit Span subtest scores, reflected attention problems. Subsequently, clinicians used the FDI as a diagnostic indicator for ADHD and researchers continue to explore its diagnostic utility. Collectively, these studies indicate that most children with ADHD do not show selective deficits on the FDI. For example, Egeland Sundberg, Andreassen, and Stensli (2006) studied a group of Norwegian children, with and without ADHD, and found no FDI difference between the groups. Anastopoulos, Spisto, and Maher (1994) reported similar findings with the WISC-III, as have others. It is important to note that lower FDI scores among children with ADHD are not unique to these children; in fact, nondisabled children and/or children with other disorders may perform poorly on the FDI. Likewise, as stated previously, many children with ADHD do not perform poorly on the FDI.

Additional IQ profiles have been studied and include comparisons using Processing Speed, Working Memory, Perceptual Organization, and Perceptual Reasoning Indices, as well as composite scores. Findings have been inconsistent across studies, with some reporting differences between children with and without ADHD, and others reporting no difference. For example, Ek, Westerlund, and Fernell (2013) compared 198 children classified as meeting full ADHD criteria and those who did not meet the criteria, and found that both groups scored higher on the General Cognitive Index than on Working Memory and Processing Speed. The groups' similar performance on the FDI is further evidence that the FDI is not a reliable indicator of ADHD. Alternatively, Mayes and Calhoun (2006b) compared the performance of children on the WISC-III and WISC-IV, and found that 100% of the children with ADHD in the sample scored lowest on the WISC-IV Working Memory Index or the Processing Speed Index. In 2004, Mayes and Calhoun reported that children with ADHD had lower Coding scores compared to children with mood disorders or brain injuries and that the "low FDI profile was two times more common in ADHD than other diagnoses" (p. 563). A score that is lower but still within the average range is not necessarily clinically meaningful, and certainly it is not diagnostic. The implication for clinicians from this body of work is that the FDI and other forms of profile analysis of IQ tests are neither reliable nor valid indicators for ADHD, and no configuration of subtests is diagnostic or specific to ADHD. Rather, clinicians should rely on a multimethod assessment approach involving objective, empirically supported techniques, as discussed in subsequent chapters.

Executive Functioning

Beyond intellectual functioning, a plethora of neuropsychological studies has been conducted with children with ADHD. These studies have included general neuropsychological tests batteries (e.g., Halstead–Reitan Battery, Luria Nebraska Neuropsychological Battery) and individual neuropsychological tasks. With regard to test batteries, Rajendran and colleagues (2013) recently explored whether severity of ADHD symptoms was associated with changes in neuropsychological

functioning in a group of preschoolers followed longitudinally using the Developmental Neuropsychological Assessment battery (NEPSY; Korkman, Kirk, & Kemp, 1998). Results indicated that baseline neuropsychological functioning was *not* associated with the slope of change of severity of ADHD symptoms. Similarly, Schaughency and colleagues (1989) failed to find support for the hypothesis that ADHD is associated with neuropsychological dysfunction on the Luria–Nebraska Neuropsychological Battery (Children's Revision). These findings, as well as others, led Gordon, Barkley, and Lovett (2006) to conclude that "in a review of the literature, we can establish no basis for suggesting routine administration of neuropsychological batteries within an ADHD evaluation" (p. 374). The take-away message for clinicians is that the use of neuropsychological test batteries with children for the purpose of an ADHD evaluation and diagnosis is unwarranted. Neuropsychological test batteries may be useful, of course, when a child has a suspected or known neurological history, or a more complicated medical history that may impact neuropsychological functioning.

Separate from test batteries, the performance of groups of children with ADHD on individual neuropsychological tasks has been studied extensively. Tasks that have received particular attention include those that measure verbal working memory, nonverbal working memory, self-regulation and inhibition, planning, perceptual motor speed, reaction time, perseveration, and sustained attention. Collectively, these cognitive processes are known as EFs. EF is a complex construct that encompasses a variety of cognitive abilities that allow for impulse control, strategic planning, cognitive flexibility, and goal-directed behavior (Barkley, 2012b). Approximately 25 years ago Welsh and Pennington (1988) defined EF as the ability to maintain an appropriate problem-solving set for attainment of future goals that may involve one or more of the following: "(1) an intention to inhibit a response or defer it to a later more appropriate time; (2) a strategic plan of action sequences; and (3) a mental representation of the task, including the relevant stimulus information encoded into memory and the desired future goal-state" (pp. 201–202).

The study of EFs and ADHD originated from research with primates that had undergone lesions to the frontal regions and neuropsychological studies of adults who sustained frontal lobe damage. In both primates and humans, injury to the frontal lobes was associated with deficits in planning ability, impulse control, and behavioral inhibition (Blau, 1936; Luria, 1966, 1972). Levin (1938) was among the first to note the striking similarity of these deficits and the symptoms associated with hyperactive children. Since that time, various perspectives concerning the role of EFs in ADHD have emerged. For example, Barkley (1997, 2012b), who perhaps has advanced the most comprehensive theory of ADHD to date, proposed that EFs are an integral component of a unifying theory of ADHD that stems from underlying deficits in behavioral inhibition and, more recently, working memory (Barkley, 2012b). Others (e.g., Brown, 2008) have also suggested that rather than being a behavioral disorder, due to the developmental impairment of EFs, ADHD is a cognitive disorder. Weyandt (2005, 2009; Weyandt et al., 2013) has emphasized that although EF deficits are characteristic of many individuals with ADHD, they are not characteristic of many if not most individuals with ADHD (Willcutt, Doyle, Nigg, Faraone, & Pennington, 2005), at least as assessed by psychometric tests, and are present in other childhood disorders. This has led some reviewers to question the role of EF in ADHD (Weyandt, 2005, 2009) and others to conclude that in most cases ADHD is not a disorder of EF (Boonstra, Oosterlaan, Sergeant, & Buitelaar, 2005; Jonsdottir, Bouma, Sergeant, & Scherder, 2006; Marchetta, Hurks, Krabbendam, & Jolles, 2008; Nigg, Willcutt, Doyle, & Sonuga-Barke, 2005; Willcutt, et al., 2005). The veracity of this conclusion, however, is based on the premise that psychometric tests are the most valid measure for evaluating EF.

In contrast, on rating scales of EF, the vast majority of children with ADHD, children with ADHD followed to adulthood, and adults with ADHD are impaired across all dimensions typically captured by these scales (Barkley, 2012a, 2012b; Barkley & Fischer, 2011; Barkley & Murphy, 2010, 2011; Mahone & Hoffman, 2007; Reddy, Hale, & Brodzinsky, 2011; Thorell, Eninger, Brocki, & Bohlin, 2010; Weyandt et al., in press). Surprisingly, a meta-analysis of numerous studies of the issue found that EF tests and EF ratings do not correlate significantly with each other and therefore cannot be viewed as evaluating the same construct (Toplak, West, & Stanovich, 2013). The results of these studies typically reveal that any single EF task shares very little (0–10%) of its variance with EF ratings. Furthermore, the relationships among these tasks are frequently not statistically significant. Even the best

combination of EF tests shares approximately 12–20% of the variance with EF ratings or observations as reflected in these studies. If IQ is statistically removed from the results, the few significant relationships found in studies between EF tests and EF ratings often become nonsignificant (Mahone et al., 2002). Based on empirical evidence, Barkley (2012a, 2012b; Barkley & Fischer, 2011; Barkley & Murphy, 2011) has argued that EF rating scales are likely more valid indicators of EF because they are substantially more predictive of adaptive functioning and domains of impairment in daily life than are traditional EF tasks. Therefore, inferences and conclusions about the relationship between EF and ADHD will vary markedly depending on the types of methods used to evaluate EF.

The study of EFs in ADHD is critical because evidence suggests that children with EF deficits have lower achievement and are more likely to repeat a grade (Biederman et al., 2004). They are also at risk for various impairments in major life activities (Barkley & Fischer, 2011). Developmentally, research suggests that children with ADHD may demonstrate EF difficulties early in life. For example, Arnett, MacDonald, and Pennington (2013) reported that third-grade children with high versus low ADHD symptomology could be distinguished at age 15 months using behavioral and cognitive measures. Ghassabian and colleagues (2013) found an association between a smaller corpus callosum in infancy and impaired EF performance at age 4. Hutchinson, Mathias, and Banich (2008) also studied morphology of the corpus callosum and reported a relationship with ADHD in children and adolescents. Studies also suggest that EF problems in preschool may be predictive of EF deficits and ADHD in later childhood (Berlin, Bohlin, & Rydell, 2003; Friedman et al., 2007). With regard to long-term outcome, Fischer, Barkley, Smallish, and Fletcher (2005) found that children with ADHD continued to display EF difficulties into young adulthood. Biederman and colleagues (2007) reported that the majority (69%) of males with ADHD participating in a 7-year follow-up study maintained EF deficits into adulthood. Others (e.g., Hinshaw, Carte, Fan, Jassy, & Owens, 2007; Rinsky & Hinshaw, 2011) have conducted longitudinal studies of children with ADHD into adolescence and adulthood and have reported similar findings. Sciberras and colleagues (in press) have launched a community-based longitudinal study in Australia to study EFs (and other outcomes) of children with ADHD compared to those without the disorder. Collectively, these findings suggest that EF deficits are likely to emerge early in life in children with ADHD, and the impairments tend to persist into adolescence and young adulthood.

Although it is beyond the scope of this chapter to review the extensive literature concerning EF deficits and ADHD, core findings in the areas of planning, inhibition, and working memory are discussed. For more extensive discussions, readers should see the various meta-analyses of studies using EF tests with ADHD (Boonstra et al., 2005; Frazier et al., 2004; Willcutt et al., 2005).

Planning

The EF construct of planning has been conceived in a number of ways including visual–spatial and strategic planning. Although a variety of neuropsychological instruments has been used to assess planning, the Tower of London (TOL) test and its variants (e.g., Tower of Hanoi) are often used in the literature. In addition to the TOL, the Rey–Osterrieth Complex Figure/or Rey Complex Figure Test (ROCF/RCFT), and Trail Making tests are the most commonly used planning tasks reported in the literature according to a recent review of 141 studies employing EF tasks (Weyandt et al., 2013). In general, the findings across studies that compare planning task performance of children with and without ADHD are inconsistent, with some studies reporting poorer performance on these tasks in children with ADHD, while others have not found group differences. In 2005, Willcutt and colleagues conducted a meta-analysis of 83 EF studies and found that the majority of studies (59% of 27 studies) reported significant group differences on measures of planning. The most consistent differences were found with Tower of Hanoi and Porteus Maze tests rather than the TOL or ROCF. Similarly, Weyandt and colleagues (2013) also reported between-group differences among children with and without ADHD on planning tasks. However, they also found that children with other clinical disorders performed more poorly than control children on EF tasks. Although many studies do not differentiate between ADHD subtypes, Gau and Chiang (2013) recently found that children with ADHD combined subtype and ADHD inattentive subtype demonstrated poorer visual–spatial planning performance, as measured by the Stockings of Cambridge subtest of the Cambridge Neuropsychological Test Automated Battery, com-

pared to children with ADHD hyperactive–impulsive subtype and children without ADHD. Dolan and Lennox (2013) compared performance on the Stockings of Cambridge planning task in adolescents with comorbid conduct disorder (CD) and ADHD. Results revealed that children with ADHD performed more poorly on the planning task compared to controls and the CD-only group. In contrast, Skogli, Egeland, Andersen, Hovik, and Øie (2014) compared children with all three subtypes of ADHD to children without the disorder on a number of EF tasks and found no difference between groups in planning task performance.

Additional studies have used a variety of planning tasks, and the reader is referred to meta-analytic studies for additional information. It is important to remember, as noted earlier, that although meta-analytic studies and review studies have found group differences between children with and without ADHD, these differences are not found in all studies or in most cases of ADHD. In fact, nearly 30% of the studies reviewed by Willcutt and colleagues (2005) did not report significant differences between groups and, of those that did report differences, the effect sizes were not large. Similarly, Weyandt and colleagues (2013) found that planning task sensitivity to group differences ranged from 4% (TOL and variants) to 36% (Trail Making Test and variants). Collectively, these findings indicate that deficits in planning, at least as psychometrically assessed, are characteristic of some but not all children with ADHD. On rating scales of EF, in contrast, planning difficulties are nearly ubiquitous in children with ADHD (Barkley, 2012a) as well as adults (Barkley, 2011; Barkley & Murphy, 2010). Findings suggest that planning deficits are also found in children with other types of clinical disorders and are not unique to ADHD (Barkley, 2012a; Salcedo-Marin, Moreno-Grandos, Ruiz-Veguilla & Ferrin, 2013; Weyandt et al., 2013). The implication for clinicians is that planning tasks are useful in differentiating between children with and without planning difficulties, and many children with ADHD do exhibit planning deficits; however, these tasks are not diagnostic of the disorder.

Inhibition

Barkley (2006, p. 301) defined the construct of "behavioral inhibition" as (1) inhibiting the initial prepotent response to an event, (2) stopping an ongoing response or response pattern, thereby permitting a delay in the decision to respond or to continue responding, and (3) protecting this period of delay and the self-directed responses that occur within it from disruption by competing events and responses (interference control). Similarly, others, such as Nigg (2005), have argued that inhibition is a multidimensional construct and should be assessed accordingly. Over the years, a significant number of studies have reported differences in EF inhibition tasks between groups of children with and without ADHD (e.g., Barkley, Grodzinsky, & DuPaul, 1992; Ikeda, Okuzumi, & Kokubun, 2013; Metin et al., 2013; Pineda, Ardila, Rosselli, Cadavid, Mancheno, & Mejia, 1998; Thissen et al., 2013; Weyandt & Willis, 1994). It is important to note that a variety of EF tasks designed to measure behavioral inhibition have been used in the literature, and the psychometric properties of these tasks vary as well. In a recent review, Weyandt and colleagues (2013) found the most commonly used executive function tasks to assess inhibition included the Stroop Color–Word Test (and variants), the Wisconsin Card Sorting Test, the continuous-performance test, and the go/no-go task. Results revealed that these tasks varied in their sensitivity to group differences, and *all* of the tasks were better able to differentiate between children with and without ADHD compared to children with other clinical disorders (e.g., anxiety, depression). Research by Corbett and Constantine (2006) also indicated that inhibition deficits are found not only between children with and without ADHD but also in other clinical groups. Specifically, their findings revealed that children with ADHD performed poorly on an inhibition task (integrated visual and auditory (IVA) continuous-performance test; Sandford & Turner, 2000); however, children with autism performed even more poorly than children with ADHD on this task. Similarly, Schoemaker and colleagues (2012) found that preschool children with ADHD performed more poorly on inhibition measures (e.g., the go/no-go task) than children without the disorder; however, children with disruptive behavior disorders also showed inhibition deficits, comparable to those of children with ADHD. Interestingly, Skogli and colleagues (2014) in Norway recently reported that although they found differences between children with and without ADHD on a variety of EF measures, including inhibition measures, the tasks did not differentiate between children with different subtypes of ADHD. This finding was in contrast to studies that proposed distinctly different underlying pathologies for the DSM-IV subtypes of ADHD based on inhibitory function (e.g., Adams, Derefinko, Milich, & Fillmore, 2008; Randall, Brocki,

& Kerns, 2009). In 2005, Van Mourik, Oosterlaan, and Sergeant conducted a meta-analysis of 17 studies using the Stroop Color–Word Test, and concluded that "the results obtained with the Stroop Colour and Word Task do not provide strong evidence for a core deficit in interference control in AD/HD" (p. 162). The authors acknowledged, however, that the task itself might not be a valid measure of an inhibition deficit. In contrast, when EF rating scales contain subscales assessing inhibition, the vast majority of children, and perhaps young adults, with ADHD are impaired (Barkley, 2012a; Mahone & Hoffman, 2007; Reddy et al., 2011; Thorell et al., 2010; Weyandt et al., 2013). Theoretically, these findings are not surprising because children with ADHD are chosen to participate in research in part due to significantly elevated symptoms of hyperactivity–impulsivity, which index problems with behavioral inhibition.

What we can conclude from this body of work is that many, but not all, children with ADHD perform more poorly on psychometric or behavioral inhibition tasks than do children without the disorder. EF deficits in behavioral inhibition are not unique to ADHD, however; children with other types of clinical disorders have been found to perform poorly on these tasks. The clinical relevance is that although these tasks may be useful in elucidating the types of EF deficits a particular child may have, the tasks are not diagnostic and should be used as part of a larger assessment protocol. Moreover, recent findings suggest that different conclusions about the relationship between EF and ADHD may be reached if EF rating scales are employed along with traditional EF tasks.

Working Memory

"Working memory" has been defined by Becker and Morris (1999) as "a system of interacting components that maintain newly acquired and reactivated stored information, both verbal and nonverbal, and make it available for further information processing" (p. 1). Working memory has been studied rather extensively in children with ADHD. With regard to nonverbal working memory, Barkley (2006) subdivided nonverbal working memory into visual–spatial memory, sequential working memory, and sense of time. Visual–spatial memory tasks typically involve memory for location of designs or patterns of designs. Relative to verbal working memory, relatively few studies of nonverbal working memory have been conducted. Earlier studies (e.g., Douglas & Benezra, 1990; Mariani & Barkley, 1997; Reader, Harris, Schuerholz, & Denckla, 1994; Weyandt & Willis, 1994) produced conflicting results, with some, but not others, reporting visual–spatial working memory deficits in children with ADHD. Frazier and colleagues (2004) conducted a meta-analysis of six studies and did not find evidence of nonverbal working memory deficits in children with ADHD relative to control children. More recently, Martinussen and Tannock (2006) explored nonverbal working memory performance of children with ADHD compared to (1) children with ADHD and language disorder, (2) children with language disorders, and (3) control children. Results revealed that children with ADHD without comorbid language disorders exhibited deficits in visual–spatial storage and verbal and visual–spatial functions that were independent of comorbid psychiatric disorders. Children with language disorders, regardless of comorbidity with ADHD, exhibited impairments in both verbal and spatial storage. Symptoms of inattention, but not symptoms of hyperactivity–impulsivity, predicted performance on verbal and visual–spatial measures independent of age, verbal cognitive ability, and reading and language performance. Consistent with these findings were results by Gau and Chiang (2013), who found that inattention symptoms during early childhood (ages 6–8) were associated with impairments in nonverbal spatial working memory later in development. However, Thissen and colleagues (2013) recently reported that EF deficits, including nonverbal working memory, found in childhood were no longer evident during adolescence. In contrast, laboratory studies using very sensitive tasks of nonverbal and verbal working memory on repeated occasions do find that children with ADHD have substantial deficits in this domain and that the effect size (group difference) is nearly double that seen in the domain of verbal working memory and dramatically impaired relative to control children (Rapport et al., 2008), and that working memory deficits may adversely affect the social functioning of children with ADHD (Kofler et al., 2011). Thus, whether nonverbal working memory is impaired in children with ADHD is partly task- or test-related. Interestingly, preliminary findings suggest that stimulant medication may improve nonverbal working memory in children with ADHD (Shang & Gau, 2012) and alterations of the dopamine transporter gene (*DAT1*) may be associated with deficits in nonverbal working memory in children with ADHD (Shang & Gau, 2014).

Studies of verbal working memory in children with ADHD are plentiful and typically involve memory for verbally presented information (e.g., digits) and mental computation. Verbal working memory purportedly plays a critical role in reading and math achievement (Swanson, Jerman, & Zheng, 2008), and others have suggested that verbal working memory underlies more complex EFs (Rapport, Chung, Shore, & Isaacs, 2001). A number of studies indicate that children with ADHD perform more poorly than their nondisabled peers on tasks that require memory for digits forward and backward (Asberg, Kopp, & Gillberg, in press; Frazier et al., 2004; Rapport et al., 2008). In a recent longitudinal study of children with ADHD, Tillman, Brocki, Sorensen, and Lundervold (in press) found that deficits in working memory (for digits) during adolescence was predicted by inhibition deficits earlier in childhood. Differences between children with and without ADHD in working memory are even larger when tasks require more complex information and when this information must be held for longer periods of time (Seidman, Biederman, Faraone, Weber, & Oullette, 1997). Similar to other types of EF difficulties, however, research suggests that verbal working memory problems are not unique to ADHD. For example, Rhodes, Park, Seth, and Coghill (2012) recently reported that children with oppositional defiant disorder (ODD) and children with ADHD and comorbid ODD performed poorly on verbal working memory tasks; however, children with ADHD alone did not show impairment (all three groups performed poorly on visual–spatial working memory). Skogan and colleagues (in press) reported similar working memory findings in preschoolers with ADHD and ODD relative to preschoolers without these disorders. In addition, Willcutt and colleagues (2013) reported that children with reading disability and those with math disability both demonstrated weaknesses in working memory. What can be concluded from these studies is that children with ADHD often have difficulties with verbal working memory; however, children with other disorders often show similar deficits.

Sense of Time

With regard to sense of time, a substantial number of studies indicate that individuals with ADHD have alterations in time perception, for example, estimating passage of time, processing time, and time discrimination (Plummer & Humphrey, 2009; Smith et al., 2013; Walg, Oepen, & Prior, in press; Wilson, Heinrichs-Graham, White, Knott, & Wetzel, 2013). As Barkley (2006) noted, sense of time involves a number of dimensions, including time perception, motor timing, time estimation, time production–reproduction, and use of time in natural settings. Findings across these dimensions have been mixed, and studies are often characterized by methodological problems. Nevertheless, problems with time sense have been postulated to underlie the impulsivity difficulties characteristic of ADHD (e.g., Barkley, Koplowitz, Anderson, & McMurray, 1997; West et al., 2000). Factors that likely contribute to these, albeit inconsistent, alterations in time include impulsivity, difficulty sustaining attention, distractors, and perhaps, as more recently suggested, reduced activation in specific brain regions (anterior cingulate, prefrontal cortices, habenula, cerebellum) may underlie time-related deficits sometimes found in ADHD (Lee & Goto, 2013; Weyandt, 2005; Wilson et al., 2013). Perhaps most troublesome to parents and educators is the difficulty children with ADHD often have in completing tasks on time, as well as planning and organizational difficulties (Langberg, Dvorsky & Evans, 2013).

Likewise, studies using rating EF scales that include subscales related to time sense and time management find robust differences between ADHD groups and control children. Research has consistently found that the vast majority of children with ADHD, those followed to adulthood, and adults with ADHD fall in the impaired range on such scales (Barkley, 2012a; Barkley & Fischer, 2011; Barkley & Murphy, 2010, 2011; Barkley et al., 1997; Houghton, Durkin, Ang, Taylor, & Brandtman, 2011).

METHODOLOGICAL CONSIDERATIONS AND CONCLUSION

Several methodological issues should be considered relative to findings on developmental and neuropsychological deficits associated with ADHD. Many of the studies rely on small samples, and effect sizes, although rarely reported, are often small. This combination of small sample and effect sizes leads to low statistical power and reduced ability to detect differences that might exist between children with and without ADHD. Subtype information is also infrequently reported, and heterogeneity of symptoms may obfuscate differences. However, DSM-5 has eliminated the subtypes of

ADHD, and this may no longer be much of an issue. Also researchers are now looking more closely at sluggish cognitive tempo, a second attention disorder often lumped in with the old ADHD inattentive type, as the likely reason why ADHD inattentive types were sometimes noted to differ on various psychological tests (see Chapter 17). Demographic variables also likely play a role in the inconsistencies across studies. For example, Thissen and colleagues (2013) reported that age is an important moderator in the relationship between EFs and ADHD. Additionally, different criteria are used across studies to define a deficit (e.g., studies assessing language/learning difficulties) and can make overall conclusions or generalizations difficult to achieve. This issue is particularly salient regarding the definition of LDs, language disabilities, and EFs.

Perhaps a more serious methodological problem concerns the questionable psychometric properties of the EF tasks employed across studies. For example, tasks are referred to in the literature as "planning," "inhibition," or working memory tests; however, given the nature of the tasks, it is unlikely that these are "pure" measures of planning, inhibition, or working memory. The ROFC, for example, is often referred to as a planning task; however, aspects of working memory and perhaps inhibition are required to complete the task. Similarly, the types of tasks included vary widely across studies (e.g., multiple planning and/or working memory tasks are used), and it is presumptuous to assume the tasks are measuring the same construct. Indeed, confirmatory factor-analytic studies suggest that EFs are moderately correlated with one another, and "it is important to recognize both the unity and diversity of executive functions" (Miyake, Friedman, Emerson, Witzki, & Howerter, 2000, p. 49). In addition, despite their widespread usage, EF tests are not necessarily effective at discriminating between different clinical and control groups (Weyandt et al., 2013).

It is important to note, however, that the use of ratings scales designed to measure EF can often yield markedly different results than traditional EF tests; hence, the nature of EF and ADHD warrants further investigation. Notably, EF rating scales are better able to discriminate between cases of ADHD and other disorders, as well as typical control cases, and recent findings indicate that EF rating scales are better predictors of impairment in major life activities. Collectively, these findings suggest that a more comprehensive picture of the nature of EF will be elucidated if EF rating scales are used in conjunction with traditional EF tests.

Although the developmental and neuropsychological deficits reviewed herein appear frequently to co-occur with ADHD, it is critical that a causal relationship not be inferred because some children with ADHD do not have developmental or neuropsychological deficits. Likewise, children without ADHD have been found to have developmental and neuropsychological deficits; hence, deficits in these areas are not unique to ADHD and are in no way diagnostic of the disorder. Further longitudinal studies are needed to identify how ADHD manifests across the lifespan, and to identify variables that contribute to the heterogeneity of the disorder. Empirical studies are also needed to establish effective interventions to address the adaptive behavior and neuropsychological deficits often found in children with ADHD.

KEY CLINICAL POINTS

- ✓ Children with ADHD have significant deficits in self-care and adaptive functioning that are more disparate from their level of intelligence than do children with mild developmental delay.
- ✓ The deficits due to ADHD are primarily in the domains of communication, daily living, and socialization, and are closely associated with the degree of executive dysfunction.
- ✓ ADHD is also associated with significant delays in motor development and coordination, with up to half or more children qualifying for a diagnosis of DCD. Fine motor coordination, particularly for writing and drawing, may be the most impaired areas of motor performance. These difficulties may be lifelong.
- ✓ ADHD medications as well as physical therapy may serve to improve motor problems.
- ✓ A sizable minority of children with ADHD may experience problems in language development, primarily in expressive language, and especially the executive or pragmatic aspects of language expression and organization. These language problems can contribute additional risk for social problems beyond the risks posed by the primary symptoms of ADHD and comorbidity.
- ✓ Up to 45% or more of children with ADHD may have LDs (see Chapter 6).
- ✓ Although the evidence is somewhat mixed, ADHD is associated with a positive illusory bias in children's self-perception of competence in various domains of

task performance. It is not so much that children with ADHD report being significantly more competent than others but that they perceive themselves as doing as well as most others, when their actual performance in that domain is significantly worse than that of others.

- ✓ Expressions of low self-worth or low self-esteem are primarily a function of comorbidity with depression.
- ✓ ADHD is routinely found to be linked to a delay in the internationalization of language. This may account for the greater verbosity of children with ADHD compared to children of similar age who have already progressed to using private self-speech. And the self-speech they do employ is less organized, less efficient, and less likely to guide their task performance effectively.
- ✓ Children with ADHD, on average, are likely to score slightly lower in general intelligence than do typically developing children. Contrary to popular belief, ADHD is not more likely to be associated with intellectual giftedness.
- ✓ Substantial research has demonstrated that ADHD is associated with significant deficits in various EFs, including response inhibition, nonverbal and verbal working memory, and planning abilities. While these differences are commonly seen in group-level comparisons with controls, only a minority of children with ADHD score in the impaired range on traditional EF tests.
- ✓ In contrast, the majority of children and adults with ADHD score in the impaired range on rating scales of EF in everyday life. Since rating scales and EF tests are not significantly correlated with each other, and rating scales have greater utility in predicting impairment, clinical evaluations of EF should include rating scales of EF and not rely exclusively on EF tests to evaluate this domain of functioning.
- ✓ ADHD is associated with impairments in one's sense of time, particularly in time reproduction and on measures of the timing and timeliness of actions. These differences are even more evident on rating scales that measure sense of time and time management.

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CHAPTER 5

Comorbid Psychiatric Disorders in Children with ADHD

Steven R. Pliszka

There is no doubt that a diagnosis of attention-deficit/hyperactive disorder (ADHD) conveys a significant risk for other, coexisting psychiatric disorders. Indeed, as many as 67–80% of clinic-referred children and 80% or more of clinic-referred adults with ADHD have at least one other disorder, and up to half have two other disorders (Barkley, Murphy, & Fischer, 2008). Such findings refute not only the naive claims that ADHD is a myth but also that ADHD is otherwise a benign condition about which one need not be concerned or seek treatment. This chapter focuses on each of the major comorbidities found with ADHD (disruptive behavior; mood, anxiety, and substance use, and tic disorders; intellectual disability; and autism spectrum disorders). There is a particular an emphasis on new paradigms from DSM 5 regarding mood dysregulation and bipolar disorder. This chapter has been further influenced by the growing awareness of the interrelatedness of many classic psychiatric disorders (Faraone, 2013). Angold, Costello, and Erkanli (1999) reviewed the reasons why disorders may coexist with each other. Common underlying etiologies (genetics, family environment, etc.) may lead to both disorders, or two disorders may be correlated with a third disorder that can account for their relationship. Apparent comorbidity also can be an artifact of methodological problems in research, such as referral bias, ascertainment bias, overlap in symptoms on diagnostic symptom lists, and other factors. Based on their meta-analysis of community samples in 21 studies, Angold and colleagues computed odds ratios (ORs) reflecting the likelihood that two disorders will coexist with each other. Use of community samples is important; clinic-referred samples can demonstrate an overlap of disorders that is based mainly, if not entirely, on referral biases in how those samples were obtained from those particular clinics. They found that, relative to the general population, patients with ADHD had greater than expected prevalence of oppositional defiant disorder (ODD)/conduct disorder (CD) (10 times), depression (5.5 times) and anxiety (3 times).

Yoshimasu and colleagues (2012) identified 379 ADHD cases in a large community sample of 5,718 children in a birth cohort (1976–1982) who were followed to age 19 years. Hazard ratios (HRs) estimated for a broad range of disorders revealed that ADHD was associated with a higher risk for adjustment disorders (HR = 3.88), ODD/CD (HR = 3.67), anxiety disorders (HR = 2.94), tic disorders (HR = 6.53), eating disorders (HR = 5.68), personality disorders (HR = 5.8), and substance-related disorders (HR = 4.03). Typically, comorbid disorders have an onset after that of the ADHD, indicating that the clinician must be alert to

the development of comorbidity over the course of the patient's life.

ODD AND CD

Studies suggest that from 45 to 84% of children and adolescents with ADHD will meet full diagnostic criteria for ODD, either alone or with CD (Barkley, DuPaul, & McMurray, 1990; Fischer, Barkley, Edelbrock, & Smallish, 1990; Pfiffner et al., 1999; Pliszka, Carlson, & Swanson, 1999; Wilens et al., 2002). The Multimodal Treatment Study of ADHD (MTA) found that 40% of children with ADHD met criteria for ODD, while 14.3% met criteria for CD (Swanson et al., 2008). Wilens and colleagues (2002) reported that 62% of their preschool-age children with ADHD and 59% of their school-age sample also had ODD. These same studies indicate that among those with ADHD, as many as 15–56% of children and 44–50% of adolescents will be diagnosed with CD (Szatmari, Boyle, & Offord, 1989; Wilens et al., 2002). In their meta-analysis, Angold and colleagues (1999) reported a median OR of 10.7 (range 7.7 to 14.8) between ADHD and CD/ODD, making it the most likely comorbidity between ADHD and any other set of disorders. This relationship does not appear to be an artifact of the coexistence of a third type of disorder with these two. As Barkley (2010) has argued (Chapter 3), this comorbidity may be partly or largely a function of the difficulties with emotional dysregulation in ADHD that make such cases highly likely to progress into ODD.

A review (Pliszka, 2009) and a meta-analysis (Waschbusch, 2002) have examined the following differences between children with ADHD and those with CD/ODD.

Family Environment and History

Children with ADHD and ODD/CD have parents with far greater rates of psychopathology than do children with ADHD alone. This higher rate of mental illness in families includes not only antisocial problems but also depressive and anxiety disorders as well (Biederman et al., 1992; Faraone, Biederman, Jetton, & Tsuang, 1997; Faraone, Biederman, Keenan, & Tsuang, 1991; Goldstein, Harvey, & Friedman-Weieneth, 2007; Lahey et al., 1988; Pfiffner, McBurnett, Rathouz, & Judice, 2005). Pfiffner and colleagues (2005) demonstrated that the family histories of children with ADHD and CD were significantly more likely to include maternal depression and paternal antisocial behavior than those of children with ADHD alone, or those with only ADHD and ODD. Parents of 3-year olds with both hyperactivity and oppositional behavior had significantly more antisocial behavior (fathers only), depression, and anxiety than those parents of preschoolers who were only hyperactive (Goldstein, Harvey, Friedman-Weieneth, Pierce, et al., 2007). In contrast, they found that adult ADHD was equally elevated in the parents of both hyperactive and hyperactive–oppositional preschoolers. Parents of hyperactive–oppositional preschoolers were in conflict with each other significantly more than parents of control or hyperactive-only children.

Compared to children with ADHD alone, children with ADHD and ODD/CD are more likely to experience divorce (August & Stewart, 1983) or be placed in foster care (Reeves, Werry, Elkind, & Zametkin, 1987). Mothers of hyperactive–oppositional preschoolers experienced more parental distress, had more dysfunctional interactions with their child, and rated the child as being more difficult than did parents of hyperactive-only preschoolers (Goldstein, Harvey, & Friedman-Weieneth, 2007). In a study of older children with ADHD, this pattern of negative parent–child interactions in those with ODD/CD was even more apparent. Pfiffner and colleagues (2005) obtained measures of parenting in three groups of children with ADHD: ADHD only (n = 66), ADHD/ODD (n = 48) and ADHD/CD (n = 35). Both ODD and CD were associated with maternal negative/ineffective discipline, while only CD was associated with lack of maternal warmth, ineffective parental discipline, and parental antisocial personality. In the MTA, a codiagnosis of ODD/CD was associated with more deviant scores on the Wells Parenting Index and more negative parent–child interactions (Jensen et al., 2001).

Cognition

Children with ADHD and comorbid CD (or delinquency) were far more likely than those with ADHD alone to have reading and learning disabilities (McGee, Williams, & Silva, 1984; Moffitt & Silva, 1988). Children with ADHD and CD had lower reading scores than did children with CD alone (who were not different from controls) (Schachar & Tannock, 1995). Moffitt (1990) followed an epidemiological sample of children with ADHD and controls from ages 3 to 13 years. Children with lower Verbal IQ, ADHD, *and* high family adver-

sity at age 3 were more likely to become delinquent later in life. Compared to children with ADHD alone, those with comorbid ODD/CD showed more severe hyperactive–impulsive symptoms on both parent and teacher ratings (Jensen et al., 2001; Newcorn et al., 2001). The comorbid group also made more impulsive errors on a laboratory measure of impulsivity (Newcorn et al., 2001).

Long-Term Outcome

Children with ADHD and comorbid disruptive behavior disorders (particularly CD) have a much higher likelihood of developing antisocial behaviors as adults than do those with ADHD alone. Both hyperactivity and aggression independently contribute to the prediction of criminal behavior in childhood and adolescence. Satterfield and colleagues (2007) examined the criminal histories of 179 children with ADHD and 75 controls over a 30-year period. Almost 30% of the children with ADHD were convicted of an offense (17% for a felony); the comparable rates in controls were 8.0 and 2.7%, respectively. Felony convictions, in particular, were much higher among the subjects with ADHD who had higher CD ratings in childhood. Kindergartners who are rated high in hyperactivity, antisocial behavior, and fearlessness are far more likely to be involved in deviant peer activities 12 years later than kindergartners who are only hyperactive (Lacourse et al., 2006).

The path to substance abuse for children with ADHD is mediated by CD. Adolescent drug use outcomes were compared between ADHD-only (n = 27), ADHD externalizing (mostly ODD) (n = 82), and normal control (n = 91) groups; only the externalizing group had a higher rate of substance and alcohol use disorders than the ADHD-only group or controls (August et al., 2006). Conduct, not attentional problems, predicted drug abuse in a 25-year follow-up of 1,265 adolescents in New Zealand. Any association between early attentional problems and later substance abuse was mediated by the association between conduct and attentional problems (Fergusson, Horwood, & Ridder, 2007).

Response to Pharmacological Treatment

Early studies showed that the ADHD core symptoms in children with ADHD and ODD/CD respond as well to psychopharmacological treatment as those in children with ADHD alone (Barkley, McMurray, Edelbrock, & Robbins, 1989; Pliszka, 1989). In addition, stimulants have been shown to reduce oppositional behavior, as well as covert and overt antisocial behavior (Hinshaw, Heller, & McHale, 1992; Klein et al., 1997). In the MTA, the stimulant response of children with ADHD and ODD/CD was just as good as that of the noncomorbid ADHD group (Jensen et al., 2001). Recent studies of both long-acting stimulants and atomoxetine have robust effects on symptoms of ODD (Newcorn, Spencer, Biederman, Milton, & Michelson, 2005; Spencer et al., 2006).

Genetics

Biederman and colleagues (1992) first showed that ADHD/CD "breeds true" in families; that is, children with ADHD without CD do not have higher than expected rates of relatives with CD. The increase is confined to relatives of children with the combined condition. Examining a genetic isolate of families, Jain and colleagues (2007) found genetic markers that cosegregated with ADHD/ODD and ADHD/CD, suggesting that these are distinct genetic subtypes. Silberg, Maes and Eaves (2012) used an extended children-of-twins design to test whether genetic and/or family environmental factors accounted for the association between parental antisocial behavior and children's behavioral problems. The relationship between parental antisocial personality and childhood depression was solely environmental, whereas genetic and family environmental factors accounted for the resemblance between parents' antisocial personality and children's conduct disturbance. The association between parental antisocial personality and ADHD was entirely genetic. Polygenetic risk (greater number of ADHD risk alleles) was significantly higher in subjects with ADHD and CD than in those without CD (Hamshere et al., 2013).

Catecholamine-O-methyltransferase (COMT) degrades dopamine and norepinephrine. The human COMT gene is located on chromosome 22q11 and contains a valine/methionine (Val/Met) polymorphism at codon 158. The Met allele is associated with a 40% reduction in enzymatic activity in the prefrontal cortex (PFC) and possibly a higher level of PFC dopamine (Chen et al., 2004). Multiple studies have shown that persons with ADHD who carry the Met allele have higher rates of antisocial, disruptive, and aggressive behavior than Val/Val carriers (Caspi et al., 2008; DeYoung et al., 2010; Langley, Heron, O'Donovan, Owen, & Thapar, 2010; Monuteaux, Biederman, Doyle, Mick,

& Faraone, 2009; Thapar et al., 2005). There is an interaction between being a Met allele carrier with a history of child abuse and future antisocial behavior (Kim-Cohen et al., 2006).

Neuroimaging

Rubia (2011) reviewed neuroimaging studies that compared children with ADHD to those with CD. The review was limited by the fact that, in most of the studies, the CD sample was highly comorbid with ADHD. The review highlighted four functional magnetic resonance imaging (fMRI) studies (Rubia et al., 2008, 2010; Rubia, Halari, et al., 2009; Rubia, Smith, et al., 2009) that compared children with ADHD to those with CD without ADHD. They found that relative to pure CD, children with ADHD alone showed underactivation in the inferior frontal cortex and overactivation of the cerebellum during tasks of motor inhibition and attention. In contrast, underactivation of limbic regions was noted in children with pure CD relative to those with ADHD alone during reward tasks. Rubia hypothesized that "direct comparisons in functional imaging show that these associations of cool inferior fronto-striato-cerebellar dysfunction in ADHD and of hot orbitofrontal-paralimbic dysfunction in CD are disorder-specific" (p. e69). There is, however, growing evidence that dysfunction in such "hot" ventromedial orbitofrontal–limbic circuits is present in ADHD even without comorbid aggression or CD.

EMOTIONAL IMPULSIVITY

Highly relevant to my discussion of comorbidity of ADHD and mood disorders is the concept of emotional impulsivity (EI), which is expanded on at length elsewhere (Barkley, 2010; see also Chapter 3). EI comprises impatience, low frustration tolerance, hot temper, irritability, anger, and emotional overreactivity. Difficulties with emotional regulation, not surprisingly, affect children with ADHD and comorbid disruptive behavior much more than they affect those with ADHD alone. Melnick and Hinshaw (2000) placed children with ADHD who scored high or low in aggression (as well as controls) in a playroom with their parents. The family had to build a Lego model that had two pieces missing. The children's emotional reactions were assessed by observers blind to the children's diagnostic status. Relative to children with ADHD alone, ADHD/aggressive boys vented more loudly, showed less overall emotional regulation, accommodated less, and had more negative responses. All the groups were then observed in a naturalistic summer camp. Controlling for core ADHD symptomatology, Melnick and Hinshaw found that noncompliance in the naturalistic setting was predicted by boys' overall emotion regulation and three specific strategies (emotional accommodation, problem solving, negative responses) during the task. Over the long term, EI is found at much higher rates in adults with ADHD than in controls and adults who had childhood ADHD but do not currently meet criteria for the disorder. EI also predicts impairments in a variety of activities (driving, finances, dating, marriage) beyond the ADHD symptoms themselves (Barkley & Fischer, 2010).

fMRI was used to assess neural activity in adolescents with and without ADHD while they performed a task involving the subliminal presentation of fearful faces (Posner et al., 2011). Adolescents with ADHD had greater right amygdala activation in response to fearful faces than controls, as well as increased connectivity between the amygdala and the lateral prefrontal cortex (LPFC). Intriguingly, in controls, presentation of the faces appeared to inhibit the amygdala–LPFC connections, whereas they enhanced the connections in the subjects with ADHD. Stimulant treatment normalized these findings in those with ADHD, both reducing amygdala activation and decreasing amygdala and LPFC connectivity. Presence or absence of ODD did not affect the imaging findings. As this discussion proceeds, a key question will relate to the difference between the "irritability" of EI and that of a major depressive or manic episode based on the fifth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-5; American Psychiatric Association, 2013).

AGGRESSION AND IRRITABILITY

Children with ADHD who meet criteria for ODD appear to come in two "subtypes": irritable and defiant (Kuny et al., 2013). There are those with ADHD who talk back, argue, and tease their siblings and peers yet rarely lose their temper (for any length of time) or do spiteful things. Others with ADHD/ODD throw prolonged tantrums (yelling, screaming, saying "I hate you!") yet do not become physically aggressive. More problematic individuals with ADHD do indeed engage in verbal ("I'm gonna kill you") or physical aggres-

sion (hitting people, putting holes in walls) and may or may not engage in other CD behaviors. Which of these children has a comorbid mood disorder? Parents often do not distinguish between this array of symptoms when they come for their child's evaluation ("He's just mad!"), and even experienced clinicians may lump all of these symptoms into a heterogeneous category of mood disorder. It is best to begin by carefully parsing the symptom clusters of aggression, irritability, depression, and mania.

Mick, Spencer, Wozniak, and Biederman (2005) looked at the specific questions that assess irritability in the Kiddie Schedule for Affective Disorders and Schizophrenia (K-SADS). For depression, the KSADS asks, "Has there ever been a period of 2 weeks or longer in which you were feeling mad (or cranky) most of the day, nearly every day?" In contrast, in the mania section, the irritability question is phrased differently: "Have you ever had a period of 1 week or longer when you felt super-angry, grouchy (or cranky) all of the time?" Mick and colleagues noted that in DSM-IV, items about depression and mania followed a "two-tier" format. That is, the question about irritability was asked first. Only if the abnormal mood was present 50% of the time did the clinician query the parent or patient about other symptoms of either depression or mania. Manic irritability was characterized by extreme ("super-angry") and pervasiveness ("all the time"), while depressive irritability was characterized by primarily negative mood and attitude, which may be punctuated by some good days. Explosive outbursts were far less likely to be a part of the picture in depression, but they were in mania. In contrast, ODD followed a one-tier format in that the parent or patient was queried about all nine symptoms. While there was no specific "irritability" item, three of the symptoms of ODD clearly tapped into this construct: "Do you often lose your temper?"; "Are you often angry or resentful?"; and "Is it easy to make you mad or to annoy you?" In the case of ODD, the symptoms had to be present "more often than not" for a period of 6 months or more.

Mick and colleagues (2005) next examined data from 274 children with ADHD, ages 6–17 years, who were studied using the K-SADS. Many (n = 144) had no mood disorder diagnosis, while 100 had unipolar depression and 30 had bipolar disorder. ODD-only-type irritability (easily annoyed, loses one's temper, angry or resentful) was highly prevalent in all three groups. In contrast, children with ADHD and comorbid depression were more likely to endorse the mad–cranky item than those without a mood disorder. Those with bipolar disorder endorsed the "super-mad" criteria in addition to the "mad–cranky" item in the depressive module. Even if the angry mood was extreme, this was not sufficient to diagnose a mood disorder. Only 46% of the children met criteria for bipolar disorder because it was also necessary that they show other symptoms: inflated self-esteem, decreased need for sleep, pressured speech, and flight of ideas or excessive involvement in pleasurable/dangerous activities.

It is clear from this that disentangling irritability and aggression is not straightforward. Aggression itself was subdivided between "reactive" and "proactive," with the former having a strong irritable/angry component and the latter perceived as "instrumental" (i.e., coldly grabbing a purse from a victim solely to obtain the contents) (Dodge, 1991). Studies of aggression in ADHD have not always clearly distinguished the two subtypes, although it is clear from a review of these studies that reactive aggression is more often the focus (King & Waschbusch, 2010). Other reviews indicated that children with ADHD show elevated levels of aggressive behavior even when researchers control for cooccurring ODD/CD. Dodge, Harnish, Lochman, and Bates (1997) found that children with high levels of reactive aggression also tend to have more inattention and impulsivity–hyperactivity problems. Children with ADHD, ODD/CD, *and* reactive aggression are more impaired in terms of peer relationships and classroom behavior than children with ADHD + ODD/CD who are not aggressive (Waschbusch, Willoughby, & Pelham, 1998). Jensen and colleagues (2007) reexamined data from the MTA and found that 267 of the original sample of 579 children (46%) exhibited clinically significant aggression. Of even greater concern, 44% of the aggressive subgroup remained so even after medication treatment of ADHD.

Social information-processing theory (Dodge, 2006; Dodge & Schwartz, 1997) provides a mechanism to better understand how aggressive children think. Dodge and Schwartz reviewed the stages of Dodge's social information-processing model: encoding, interpretation of social cues, goal selection, response access and construction, and response decision. When looking at social situations, aggressive children encode fewer relevant cues and do not seek out additional information when the situation is ambiguous. They are more likely to interpret ambiguous cues as hostile. Dodge (2006) suggested that recognizing hostile intent may be a "hardwired" function of the amygdala, while at-

tributing benign intent to an ambiguous situation is a learned process. As for goal selection, aggressive children seek dominance and control, and generate fewer potential responses to a problematic social situation. There is a negative correlation between the number of responses and the child's rate of aggression. In terms of response decision, aggressive children view aggression as producing more desirable outcomes. They are more likely to see aggression as leading to tangible rewards and peer group approval, and they do not see their victims as suffering any real harm. The encoding and the response decision phases differ in children with different subtypes of aggression (Dodge et al., 1997). Children with ADHD as a whole make more social information-processing errors than do controls (King & Waschbusch, 2010). However, children with ADHD and aggression, relative to controls and nonaggressive children with ADHD, utilize social cues poorly (Milich & Dodge, 1984), have more difficulty anticipating the consequences of their aggressive actions (Bloomquist, August, Cohen, Doyle, & Everhart, 1997), and generate aggressive solutions to problems (Matthys, Cuperus, & van Engeland, 1999).

Both pharmacological and psychosocial methods have been utilized in the treatment of ADHD and aggression. Two large-scale reviews of the effects of stimulant medication on aggression have been undertaken. Connor, Glatt, Lopez, Jackson, and Melloni (2002) reviewed 28 published studies from 1970 to 2001 that measured stimulant effect on aggression; mean effect sizes were 0.84 for overt aggression and 0.69 for covert aggression. Similarly, Pappadopoulos and colleagues (2006) reviewed 19 stimulant studies involving over 1,000 subjects and found stimulants to have an effect size of 0.78 in the treatment of aggression. These effect sizes are equivalent to those of stimulants on the core symptoms of ADHD. There has been a long-standing belief that stimulants *cause* aggression, so much so that the U.S. Food and Drug Administration convened a special panel to study this issue (Mosholder, 2006). No significant difference was found between placebo and stimulant in the prevalence of psychotic, manic, and suicidal events or aggression. Only very rarely do patients have an idiosyncratic reaction of this sort to stimulant. Elliott, Malecki, and Demaray (2001) reviewed social skills interventions for children and noted that they are most effective for withdrawn children and tend to be less effective for aggressive children, while parent training is effective for children with ADHD and comorbid ODD/CD (Fabiano et al., 2009; Patterson, Chamberlain, & Reid, 1982). Combining psychosocial treatment with pharmacological treatment of the child's ADHD is a desirable first-line intervention before moving to more aggressive psychopharmacology (i.e., mood stabilizers, antipsychotics) (Pliszka, 2009).

How much of a benefit is provided in terms of reducing aggression by adding an antipsychotic to a stimulant? Children with ADHD and aggressive behavior were treated with stimulants and parent training for 3 weeks, then either placebo or risperidone was added in a randomized, double-blind fashion (Aman et al., 2014). Disruptive behavior scores declined from a mean of 45 at baseline to 25 at Week 3 (before addition of risperidone). At Week 9, the risperidone group was showed a further mean drop of 14 in disruptive behavior ratings, compared to an 8.1 mean drop in the placebo group ($p = .0143$, effect size = 0.5). There were no differences between the groups at Week 9 in terms of clinician ratings of overall improvement. Thus, while adding risperidone was beneficial in reducing severe disruptive behavior, the effect was modest when combined psychosocial and stimulant treatment of ADHD were implemented first.

DISRUPTIVE MOOD DYSREGULATION DISORDER

In DSM-5, the American Psychiatric Association (2013) has added the diagnosis of disruptive mood dysregulation disorder (DMDD; see Table 5.1). This change in name illustrates a fundamental dilemma: Is this severely problematic mix of mood lability and aggression a "disruptive" or a "mood" disorder? The need for such a new category was driven in large part by the debate as to whether bipolar disorder (BP) in youth exists in either a classic episodic form or a chronic irritable form (Biederman, 1998; Carlson, 2007; Klein, Pine, & Klein, 1998). Furthermore, in the last decade there has been a 40-fold increase in outpatient visits (Moreno et al., 2007) and a sixfold increase in hospital admissions for youth diagnosed with BP (Blader & Carlson, 2007). During the same period, there has been a marked increase in the use of mood stabilizers and antipsychotics in children and youth (Moreno et al., 2007; Olfson, Blanco, Liu, Moreno, & Laje, 2006), often for predominant symptoms of aggression. The debate has moved beyond the scientific literature into the lay media, where the approach to the topic is often ambivalent: a great deal of sympathy for children with

TABLE 5.1. DSM-5 Diagnostic Criteria for Disruptive Mood Dysregulation Disorder (DMDD)

A. Severe recurrent temper outbursts manifested verbally (e.g., verbal rages) and/or behaviorally (e.g., physical aggression toward people or property) that are grossly out of proportion in intensity or duration to the situation or provocation.

B. The temper outbursts are inconsistent with developmental level.

C. The temper outbursts occur, on average, three or more times per week

D. The mood between temper outbursts is persistently irritable or angry most of the day, nearly every day, and is observable by others (e.g., parents, teachers, peers).

E. Criteria A–D have been present for 12 or more months. Throughout that time, the individual has not had a period lasting 3 or more consecutive months without all of the symptoms in Criteria A–D.

F. Criteria A and D are present in at least two of three settings (i.e., at home, at school, with peers) and are severe in at least one of these.

G. The diagnosis should not be made for the first time before age 6 years or after age 18 years. Age of onset by 10 years.

H. By history or observation, the age at onset of Criteria A–E is before 10 years.

I. There has never been a distinct period lasting more than 1 day during which the full symptom criteria, except duration, for a manic or hypomanic episode have been met. **Note:** Developmentally appropriate mood elevation, such as occurs in the context of a highly positive event or its anticipation, should not be considered as a symptom of mania or hypomania.

J. The behaviors do not occur exclusively during an episode of major depressive disorder and are not better explained by another mental disorder (e.g., autism spectrum disorder, posttraumatic stress disorder, separation anxiety disorder, persistent depressive disorder [dysthymia]). **Note**: This diagnosis cannot coexist with oppositional defiant disorder, intermittent explosive disorder, or bipolar disorder, though it can coexist with others, including major depressive disorder, attention deficit/hyperactivity disorder, conduct disorder, and substance use disorders. Individuals whose symptoms meet criteria for both disruptive mood dysregulation disorder and oppositional defiant disorder should only be given the diagnosis of disruptive mood dysregulation disorder. If an individual has ever experienced a manic or hypomanic episode, the diagnosis of disruptive mood dysregulation disorder should not be assigned.

K. The symptoms are not attributable to the physiological effects of a substance or to another medical or neurological condition.

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BP or severe mood issues (Egan, 2008) versus skepticism regarding the psychopharmacology of the disorder (Carey, 2007).

The debate began when Wozniak and colleagues (1995) identified 43 children in a psychiatric outpatient clinic who met their criteria (K-SADS) for mania; all but one of these children also met criteria for ADHD. They were compared to 164 nonmanic children with ADHD and 84 controls. The total of 206 (42 manic and 164 nonmanic) subjects with ADHD yielded a 20% prevalence of mania of 20% in the ADHD sample in this study. There were only two children with euphoric mania, while 77% showed "extreme and persistent mania." That is, they did not cycle or have any prolonged periods of euthymia. Eighty-four percent showed "mixed mania," in which symptoms of mania and depression co-occurred. Biederman and colleagues (1995) studied a second sample of 120 children with ADHD and found 29 children (21%) who met criteria for BP. Child Behavior Checklist (CBCL) scores of the children with ADHD/mania were elevated compared to those of children with ADHD only on nearly all of the subscales. Of note, it was the Aggression subscale that most differentiated the manic/ADHD group from the ADHD-only group (Biederman et al., 1995).

At the time, Biederman and colleagues (1998) and Klein and colleagues (1998) had an extensive debate in the *Journal of the American Academy of Child and Ado-*

lescent Psychiatry regarding the validity of the broader diagnosis. In particular, Klein and colleagues pointed to the fact that the concept of chronic irritable mania violated the DSM-IV requirement for a "distinct period" of abnormal mood. That is, there is a period of euthymic mood and a specific point at which the manic or depressive episode can be said to begin. However, given that virtually all studies indicated that children with BP have very high rates (40–90%) of comorbid ADHD (Kowatch, Youngstrom, Danielyan, & Findling, 2005; Singh, DelBello, Kowatch, & Strakowski, 2006), how does one mark the beginning or end of a manic episode when the child is chronically hyperactive and talkative? For instance, B. Geller and colleagues (1995) noted that one child in a study had 104 episodes in a year, lasting from as short as 4 hours to a whole day. Findling and colleagues (2001) followed 90 youth with BP using prospective life charting and found that only two subjects ever showed "interepisode recovery," defined as being euthymic for a 2-month period.

In 2003, Leibenluft, Charney, Towbin, Bhangoo, and Pine (2003) published a very influential article that proposed a research strategy for resolving these issues. They suggested that the diagnosis of BP be limited to those youth who show elation as the mood change and whose abnormal mood state was clearly distinct from baseline. These youth also would have to meet the other first-rank symptoms of mania (pressured speech, grandiosity, etc.) and for the required periods of time. They proposed severe mood dysregulation (SMD) as an alternative diagnosis for those with chronic irritability. SMD formed the template for DMDD, but there are significant differences. In DSM-5, both entities require persistent abnormal mood (anger/sadness but *not* elation) and temper outbursts, with severe temper problems in at least one setting. SMD permits the presence of manic symptoms in the hyperarousal category (insomnia, agitation, pressured speech, etc.) as long as the youth does not have elevated mood, grandiosity, or inflated self-esteem. In contrast, in DSM-5, DMDD specifically excludes children with hyperarousal (see Table 5.1). A substantial body of data has emerged with respect to SMD, but the reader should bear in mind that it might not apply entirely to DMDD due to this important difference in their criteria.

Brotman and colleagues (2006) examined data from the Great Smoky Mountains epidemiology study and used items from the structured interview in that project to diagnose SMD retrospectively; about 3% of the cohort met criteria for this diagnosis. Not surprisingly, there was a high degree of comorbidity in members of this sample: ADHD (27%), ODD (50%), anxiety disorder (15%), and depression (13.4%). The rate of BP in this study was extremely low and did not differ between those with and without SMD. Recently, data from three large epidemiological studies were combined to examine the prevalence of DMDD based on constructing the criteria from the specific questions asked in structured interviews (Copeland, Angold, Costello, & Egger, 2013). The prevalence rates ranged from 0.8 to 3.3%, with the highest rate in preschoolers (who may not be given the DMDD diagnosis). The highest level of comorbidity were with depressive disorders (ORs 9.9–23.5) and ODD (ORs 52.9–103.0). DMDD *cannot* be comorbid with ODD, however, so if these two exclusionary criteria are applied, the prevalence falls to about 1% (Axelson, 2013). Axelson further points out that the diagnosis of DMDD requires children and parents to recall the frequency, persistence, and duration of aggressive outbursts and irritability, which can be very difficult and perhaps lead to instability of the diagnosis and its poor test–retest reliability (kappa = 0.25; Regier et al., 2013).

Does DMDD belong in the bipolar or unipolar spectrum? Stringaris and colleagues (2010) followed 93 youth with narrow phenotype ("classic") BP and 84 youth with SMD. At follow-up (median = 28.7 months), 63% of youth with BP had a full manic episode, compared to only a handful of such episodes in youth with SMD, who were more likely to have depressive episodes, though not dramatically so. A number of long-term follow-up studies suggest that youth with chronic irritability (although these subjects were not necessarily diagnosed with SMD) are more likely to show depression rather than BP on follow-up (Sobanski et al., 2010; Stringaris, Cohen, Pine, & Leibenluft, 2009; Stringaris & Goodman, 2009). This underlies the DSM-5 decision to include this disorder in the depressive disorders category (American Psychiatric Association, 2013, p. 157).

As noted, Biederman and colleagues (1995) published a report of a distinct behavioral profile of children with BP, as measured by the CBCL, with marked elevations on the Attention, Aggression, and Anxious/Depression subscales. This profile was found in about 0.8% of a large twin population and had a different pattern of inheritance from those with ADHD alone (Hudziak, Althoff, Derks, Faraone, & Boomsma, 2005). Thus, there is evidence that this dysregulated group is a distinct genetic subpopulation. Althoff,

Verhulst, Rettew, Hudziak, and van der Ende (2010) followed over 2,000 children who had CBCL's conducted at baseline and subjected to latent class analysis (LCA). LCA clearly identified a subgroup of children who showed the "BP profile" on the CBCL. However, when they were followed up 14 years later, none had developed BP. Rather, this dysregulated profile was a risk factor for a wide range of psychiatric disorders. It should be noted, however, that in this entire sample of over 1,500 subjects who were followed, there were only six cases of BP. This does raise some concerns that the interview procedures either underidentified BP or the sample was not completely representative of the general population. Nonetheless, it certainly showed that elevations on the Attention, Aggression, and Anxiety/Depression subscales of the CBCL are not diagnostic or predictive of BP.

Imaging studies have shown differences between youth with BP (narrowly defined) and those with SMD (Brotman et al., 2010). Children with ADHD alone, BP, SMD, and typically developing controls observed faces while undergoing fMRI. The participants had to rate either the fearfulness of the faces or the nose width of the individual (the latter was a control condition for contrast). Surprisingly, youth with BP did not differ from controls on these measures. Children with ADHD alone showed a marked increase in left amygdala activation relative to controls, while those with SMD showed deactivation of the amygdala compared to both controls and those with ADHD alone. This does suggests that the SMD and narrow BP groups are physiologically distinct, as was implied by an earlier event-related potential study (Rich et al., 2010). The picture remains unclear, however. In contrast to Brotman and colleagues (2010), a recent study from the same group found that children with BP and SMD both had reduced amygdala activation to angry faces relative to healthy controls (Thomas et al., 2012). Relative to healthy controls, children with SMD show decreased activation of the left posterior cingulate and precuneus during negative feedback (losing money in a game) but no differences during positive feedback (Deveney et al., 2013). No differences were found in the amygdala or prefrontal areas that were predicted to differentiate these groups.

What are the pharmacological treatment implications for DMDD? A diagnosis of BP is an indication for treatment with mood stabilizers and second-generation antipsychotics (SGAs; Kowatch, Fristad, et al., 2005). Given that these agents also are effective for aggression (Pappadopulos et al., 2003, 2006; Schur et al., 2003), it not surprising that that there has been a marked increase in the clinical use of these agents in children and adolescents (Patel et al., 2005; Patel, Crismon, & Shafer, 2006). This too has been a source of controversy in the lay media (Harris, Carey, & Roberts, 2007), and pharmaceutical companies have paid large fines for off-label marketing for use of these agents in children and older adults. SGAs are associated with significant weight gain and change in lipid metabolism (Correll et al., 2009). Will the addition of DMDD to DSM-5 lead to changes in prescribing practices? If the chronically irritable and aggressive child currently diagnosed as "BP not otherwise specified (NOS)" has his or her diagnosis changed to DMDD, will the physician be less likely to prescribe less medication? At present this does not seem likely. Brotman and colleagues (2010) found that equal numbers of the BP and SMD groups were taking mood stabilizers and SGAs. One study suggests that it is possible to avoid placing some aggressive children on SGAs. Blader, Schooler, Jensen, Pliszka, and Kafantaris (2009) treated 74 children with ADHD and significant aggression (though not necessarily diagnosed with SMD) with stimulants and a behavior management program; the aggression remitted in 31 of the participants. Thirty subjects were randomized to either placebo or valproate for 6 weeks; those subjects on valproate had a significant decrease in aggression. Although this group of children with ADHD had high levels of reactive aggression, they were not formally diagnosed with SMD or DMDD. Dickstein and colleagues (2009) admitted 45 youth with SMD to an inpatient unit; 20 of these had reduced aggression after a week on placebo and were excluded. The remaining 25 subjects whose mood dysregulation remained problematic were randomized to either lithium or placebo for 6 weeks. There was not even a trend for lithium to be effective relative to placebo, and there were few responders in either group, suggesting that SMD is very difficult to treat. A study of citalopram in SMD is under way (Dickstein et al., 2009). The results of such a study will be informative. If SMD is indeed part of the BP spectrum, one would expect high rates of "switching" to mania. If this does not occur, then this would be evidence that SMD is not related to BP.

The addition of DMDD provides a challenge for the clinician. It will need to be considered whenever a child with ADHD and aggression *also* exhibits chronic irritability between aggressive episodes. This is likely to encompass a large group of children who often

have been diagnosed with "mood disorder, not otherwise specified." Thus, the differential diagnosis will be among mania, DMDD, and ADHD + ODD. Note in Table 5.1 that DMDD *cannot* be comorbid with ODD or a manic episode, while it *can* be comorbid with ADHD or a major depressive episode.

MANIA

DSM-5 (American Psychiatric Association, 2013, p. 157) mentions "some researchers" (no citation) who "view severe, non-episodic irritability as characteristic of bipolar disorder." This may be an oblique reference to the Massachusetts General group's earlier work, although it should be noted that the full mania criteria (not just chronic irritability) were used in their studies (Biederman et al., 1995; Wozniak et al., 1995). It is further stated in DSM-5 that "the term bipolar disorder is explicitly reserved for episodic presentations of bipolar symptoms" (p. 157). Thus, DSM-5 has "doubled-down" on the 19th-century definition of Emil Kraepelin of the manic episode. But what is an "episode?" A patient must have an elevated, expansive or irritable mood (1 week for mania, 4 days for hypomania) with three or more of the associated symptoms (grandiosity, decreased need for sleep, increased talkativeness, flight of ideas, increased goal-directed activity or risky behaviors). Rather than diagnose children and adolescents with ADHD who have chronic irritability/aggression alone with BP, clinicians should begin using DMDD for this group. This section focuses on those with ADHD who meet the classic criteria for a manic episode, although we must accept the word of the researchers that their samples contain patients who have distinct cycles of mood disturbance as identified by structured interviews, such as the K-SADS.

Pataki and Carlson (2013) reviewed a number of major issues concerning the comorbidity of ADHD and BP. They noted that community samples show very low rates of BP in children with ADHD (0–2%), while studies in tertiary-care clinics show comorbidity rates of 11–30%. In contrast, preadolescent children with BP have ADHD at rates of 75–95%. In the Longitudinal Assessment of Manic Symptoms (LAMS; Arnold et al., 2011) study, a Mania Screening scale was administered to over 2,600 parents of patients (ages 6–12) presenting to several academic child psychiatry clinics; 1,124 screened high for mania, while about 1,500 did not. Children from the high (n = 621) and low (n = 86) mania screening groups underwent an in-depth psychiatric interview, and BP was diagnosed only if the manic episodes were episodic. The authors found that 60% of the sample met criteria for ADHD without BP, only 6.3% met criteria for BP without ADHD, 16.5% had both ADHD and BP, and the remaining 17.5% did not meet criteria for either diagnosis. Parent K-SADS established the diagnosis in this study; it was interesting that teacher ratings of mania were elevated only in the two ADHD groups and that the ADHD + BP and ADHD – BP groups did not differ from each other. More striking, the BP-only group had lower mania ratings than the ADHD-only group. Pataki and Carlson asked how mania could only present at home. This shows the lack of clear research or clinical consensus on how to aggregate data from different sources to make a diagnosis.

Table 5.2 suggests ways for the clinician to distinguish between the various comorbid mood diagnoses that can present with ADHD. As Carlson (2007) pointed out, the differential diagnosis is not *between* ADHD and BP; the question is whether the aggression and mood lability meet the criteria for BP. As shown in Table 5.2, severe ADHD, ADHD with ODD/ intermittent explosive disorder (IED), and ADHD/BP are all characterized by increases in motor activity, distractibility, and excessive talking, so these factors cannot be used to distinguish the conditions. Caretakers often view ADHD (combined type) children as energetic, but those with ADHD/BP show marked increased in energy ("They never wear out," "Everyone else in the family is exhausted"). In DSM-5, it is now imperative to separate the chronic irritability of DMDD from the episodic manic episodes of BP. Sleep can be disturbed in all children with ADHD (trouble going to bed and a tendency to sleep late), but only those with ADHD/BP show a true *decreased need* for sleep. It is the core symptoms of mania that truly distinguish BP from ODD or mood dysregulation (B. Geller, Tillman, Craney, & Bolhofner, 2004; B. Geller, Warner, Williams, & Zimmerman, 1998; B. Geller, Williams, et al., 1998). What is the difference between excessive talking and pressured speech? Children with ADHD often engage in more random speech at inappropriate times (talking in class or in church). Yet when they must engage in goal-directed speech, they often are less talkative (e.g., during the clinician interview). In contrast, children with BP talk over the examiner, use more words, and clearly have an increased *rate and volume* of speech. Patients with ADHD/ODD/IED are often perceived as

TABLE 5.2. Differential Diagnosis of Mood Lability and Aggression in ADHD

	"Severe" ADHD	ADHD + IED or ODD/CD	ADHD + DMDD	ADHD and BP
Motor activity	Increased	Increased	Increased	Increased
Distractibility	Increased	Increased	Increased	Increased
Excessive talking	Increased	Increased	Increased	Increased
Energy	Increased	Increased	Increased	Markedly Increased
Anger outbursts	None	Intense, but time limited, interoutburst mood within normal limits	Severe outbursts at least three times a week in at least two settings	Prolonged, affective storms, "walking on egg shells"
General mood	Euthymic	Irritable only when frustrated	Chronic irritability/ sadness	Pervasive abnormal mood over weeks, but distinct episodes
Sleep	Erratic bedtime and wake up time, sleeps 8-10 hours	"Night owl"—particularly in adolescents; sleeps 8–10 hours	"Night owl"—particularly in adolescents; sleeps 8–10 hours	Sleeps 6 hours a night or less
Core mania symptoms				
Pressured speech	Absent	Absent	Absent	Present
Flight of ideas	Absent	Absent	Absent	Present
Grandiosity	Absent	Absent	Absent	"Full of self"
Sexuality	Absent	Early sexual activity	Early sexual activity	Abnormal sex acts
Judgment/risks	Immature	Antisocial risks	Absent	Dangerous acts
Elation/silliness	Limited	Limited	Limited	Present
Psychosis	Absent	Absent	Absent	Often present

arrogant and as lacking remorse, but the patient with BP lacks any realistic awareness of his or her capabilities. Although frank delusions of grandeur are rare in children and teens, beliefs that one can do whatever one wants are often a key symptom. A child with ODD knows he or she will fail a test (and does not care), whereas a child with BP may be fully convinced that he or she will make an "A" even without having studied or going to class. Children and teens with BP often hold completely unrealistic fantasies about what will happen in the future and plan their lives around them. Children with ADHD often tend to initiate sexual activity earlier than non-ADHD peers, but there is nothing hypersexual about their behavior. Children with BP engage in age-inappropriate sex play and obsession with pornography (especially on the Internet). Psychosis, particularly if the delusions or hallucinations have a grandiose character, strongly suggests mania. Psychosis is absent in ODD, CD, and DMDD.

A major concern for many clinicians is that treatment of ADHD will make a child with BP worse. Will stimulant treatment of ADHD in a child at risk for BP (due to either family history of mood disorder or the presence of mood dysregulation) induce a manic episode? Galanter and colleagues (2003) examined the stimulant response of children in the MTA who showed high levels of manic-like symptoms (but were not diagnosed with BP). They found that in this subgroup, the response of ADHD symptoms to stimulants was just as robust as that in the "euthymic" children with ADHD, with no evidence of mood destabilization. After reviewing four studies that showed no evidence of emerging manic symptoms in children with ADHD treated with stimulants, Goldsmith, Singh, and Chang (2011) concluded, "Collectively, these findings suggest that psychostimulant exposure is not instrumental in the development of BD. Moreover, ADHD symptom severity at the time of presentation may be the pri-

mary predictor of psychostimulant treatment, which also does not predict a greater chance of BD outcome" (p. 228). There is widespread consensus that a patient with acute mania should undergo mood stabilization treatment before attempting treatment of ADHD (Pataki & Carlson, 2013; Pliszka, 2009). Three placebo-controlled studies (Findling et al., 2007; Scheffer, Kowatch, Carmody, & Rush, 2005; Zeni, Tramontina, Ketzer, Pheula, & Rohde, 2009) found that once mood is stable, stimulants can be combined with mood stabilizers without causing relapse of mania.

DEPRESSION

Regarding the comorbidity of depression and ADHD, in this review I move beyond irritability and aggression to focus on those individuals who have ADHD and full DSM-5 criteria for major depressive disorder (MDD) or persistent depressive disorder (as dysthymia has been renamed). DSM-5 continues to allow clinicians to diagnose MDD in youth using irritable mood as well as depression as criteria; the abnormal mood must be persistent and associated with four of these other criteria (loss of interest, weight loss, sleep disturbance, agitation/retardation, fatigue, low self-esteem, diminished concentration, suicidal ideation/attempts). When a patient has ADHD, the concentration problems related to depression must be above and beyond those attributable to the ADHD. Up to one-third of children with ADHD may meet criteria for depression, while one-fourth to one-half of children with depression have ADHD (Angold & Costello, 1993; Pliszka et al., 1999). On the other hand, some studies show no overlap at all between ADHD and depression (McGee et al., 1990). This variation in prevalence is related to sample type (community vs. clinical), different methods of eliciting depressive symptoms, and type of informant (parent vs. child).

Daviss (2008) reviewed the literature regarding the comorbidity of depression and ADHD. Compared to those with depression alone, youth with comorbid ADHD and depression show greater impairments in social functioning, earlier age of onset of depression (in females), and higher rates of suicidality and reoccurrence of depression. Adolescents with ADHD and depression have higher levels of family conflict, more negative life events, and more trauma exposure than do those with ADHD and no depression. Blackman, Ostrander, and Herman (2005) compared their sample of depressed and nondepressed children with ADHD to healthy controls on a variety of measures. Depressed children with ADHD were not different from nondepressed subjects in terms of hyperactivity, conduct problems, or aggression, but they did have greater problems with social competence. The relationship between ADHD, depression, and suicidality is complex. Youth with ADHD do have higher rates of suicidal ideation and deliberate self-harm than controls, even after researchers control for gender, substance abuse, and behavioral problems (Hurtig, Taanila, Moilanen, Nordstrom, & Ebeling, 2012). However, the depressed youth with both ADHD and disruptive behavior disorder remains at the highest risk for suicide (James, Lai, & Dahl, 2004; also see Chapter 11).

Is there any etiological relationship between ADHD and depression? Many children with ADHD in fact have positive illusory biases that "protect" their self-esteem from the consequences of their behavior and predict poor response to psychosocial intervention (Mikami, Calhoun, & Abikoff, 2010; see Chapter 4). Thus, it is unlikely that the high rate of depression in ADHD is related to the "demoralization" of living with ADHD (Biederman et al., 1998). Family studies reveal a pattern in which patients with ADHD alone have a higher than expected rate of depressive disorders in their relatives; conversely, patients with depression alone have a higher than expected rate of ADHD (Faraone & Biederman, 1997). This suggests that the disorders share genetic factors.

There have been only limited post hoc analyses of antidepressant clinical trial data as to whether the presence of ADHD affects treatment outcome, and the results are contradictory (Daviss, 2008). In contrast, there is no evidence that early treatment of ADHD with stimulants predisposes a patient to future depressive episodes (Daviss, Birmaher, Diler, & Mintz, 2008; Staikova, Marks, Miller, Newcorn, & Halperin, 2010). Studies of the treatment of comorbid depression and ADHD are also very limited. Findling (1996) found that a combination of stimulants and specific serotonin reuptake inhibitors were well tolerated in seven patients, ages 10–16 years, with comorbid ADHD and MDD. An open-label study of bupropion in 24 adolescents with comorbid ADHD and MDD revealed an 88% response rate for depression and a 63% response rate for ADHD (Daviss et al., 2001). Kratochvil and colleagues (2005) randomized 173 youth with comorbid ADHD and depression to either placebo or fluoxetine for 8 weeks, with both groups receiving atomoxetine during the final 5 weeks of the study. Depression and ADHD symptoms were reduced in both groups, with no clinically mean-

ingful differences between them at endpoint. A large group (n = 142) of adolescents with ADHD and MDD were randomized to either placebo or atomoxetine for 9 weeks in a double-blind design. Symptoms of ADHD were significantly improved on atomoxetine relative to placebo at endpoint, but both groups improved over the study in terms of depression (Bangs et al., 2007). It has become standard clinical practice to combine stimulants and antidepressants in the treatment of comorbid ADHD and MDD, with the more severe disorder being treated first (Pliszka et al., 2006).

ANXIETY DISORDER AND POSTTRAUMATIC STRESS DISORDERS

Estimates of the prevalence of anxiety disorders in children with ADHD have ranged from 25 to 50%, compared to the prevalence of about 6–20% in the general pediatric population (Costello, Egger, & Angold, 2004). Thus, the OR of having an anxiety disorder is 2.1–4.3 greater in children with ADHD relative to the general population (Angold et al., 1999). Children with anxiety disorder also have higher than expected rates of ADHD (Last, Hersen, Kazdin, Finkelstein, & Strauss, 1987), as do adults with panic disorder (Fones, Pollack, Susswein, & Otto, 2000; Safren, Lanka, Otto, & Pollack, 2001). Adults with ADHD also show high rates of anxiety disorders relative to the general population (Biederman et al., 1993).

When children with ADHD and anxiety disorders are compared to those with anxiety disorder alone, no differences emerge in subtype or severity of the anxiety itself. That is, ADHD does not have any moderating effect on the anxiety disorders (Hammerness et al., 2010). Sleep disorders are increased in people with ADHD and anxiety relative to those with ADHD without anxiety; the latter in turn have more sleep problems than do controls (Accardo et al., 2012; Hansen, Skirbekk, Oerbeck, Richter, & Kristensen, 2011). Studies have been inconsistent as to whether ADHD with and without anxiety differs in terms of the presence of other disruptive behavior disorders. One study found a lower rate of CD (Pliszka, 1989), while most studies have found no difference in the rate of ODD or CD (Becker, Luebbe, Stoppelbein, Greening, & Fite, 2012; Biederman, Faraone, Keenan, Steingard, & Tsuang, 1991; Pliszka, 1992; Vloet, Konrad, Herpertz-Dahlmann, Polier, & Gunther, 2010). Still other studies have shown increased rates of ODD/CD in the ADHD with anxiety group relative to those with ADHD alone (Humphreys, Aguirre, & Lee, 2012; Newcorn et al., 2001; Tannock, 2000). Differences in these studies may relate to both sample source (community vs. clinical) and interview method. In highly structured interviews, parents of those with ADHD and ODD/CD may interpret irritability as anxiety, whereas clinical interview methods that include child self-report rule out the anxiety disorder in ODD/CD because the child denies anxiety having when asked directly.

How anxiety affects the cognitive and behavioral aspects of ADHD may well depend on whether the child has an additional comorbid disruptive behavior disorder. Newcorn and colleagues (2001) examined continuous performance test errors in children with ADHD in the MTA, stratified by comorbidity. The ADHD/anxiety group showed decreased impulsivity and dyscontrol errors relative to the other ADHD groups. This effect was moderated by gender: Only girls with ADHD/anxiety showed this pattern. Cases with ADHD/anxiety *and* ODD/CD were as impulsive on this measure as those with ADHD alone. Teachers also rated children with ADHD/anxiety as less impulsive than either the ADHD-only or dual-comorbid group. On the other hand, parents rated children with ADHD/anxiety + ODD/CD as *more* impulsive and hyperactive than those with ADHD alone. One recent study has shown that anxiety aggravates behavioral dysregulation as assessed by parent report (Sørensen, Plessen, Nicholas, & Lundervold, 2011), while another showed no effect of anxiety on a computerized measure of impulse control in children with ADHD (Vloet et al., 2010) Vloet and colleagues (2010) also found, however, that performance on attentional measures was *improved* in those with ADHD and anxiety relative to the ADHD only group. Both of these studies included both ADHD/anxiety and ADHD/anxiety/ODD/CD groups and did not examine the triple-comorbid groups separately.

Stimulant treatment does not worsen anxiety in the comorbid group (Pliszka, 1989), and one study in adults with ADHD indicates that it may actually reduce anxiety (Gabriel, 2010). Children with ADHD and anxiety were more likely to have a placebo response (Pliszka, 1989). This was not confirmed by the MTA (MTA Cooperative Group, 1999b), in which children with ADHD/anxiety showed a positive response to methylphenidate, as did those without anxiety. Interestingly, those subjects with ADHD/anxiety (but not ODD/CD) had a more robust response to the behavioral intervention by itself relative to nonanxious children with ADHD. The subgroup with triple comorbidity (ADHD + ODD/CD + anxiety) was more likely to benefit from the combination treatment of the behavioral

intervention and medication relative to the children with ADHD alone. In a further analysis of the MTA treatment outcome data, March and colleagues (2000) found that parent-reported anxiety alone was a moderator; there was no relationship between the children's self-report of anxiety and treatment outcome. Parent-reported anxiety was strongly related to the comorbidity of ODD/CD and to the phenomena of "negative affectivity," rather than fears and phobia. Thus, March and colleagues hypothesized that the strong behavioral management focus of the MTA psychosocial intervention was helpful for children with ADHD/anxiety and ODD/CD because it helped the parent manage such negativity.

How and when should anxiety in the presence of ADHD be treated pharmacologically? Numerous studies have established the efficacy of specific serotonin reuptake inhibitors in the treatment of a variety of child and adolescent anxiety disorders (Pliszka, 2011). But since these agents do not treat ADHD, they must be combined with a stimulant. Abikoff and colleagues (2005) treated 32 children with ADHD/anxiety with methylphenidate and found that 26 (81%) responded with respect to ADHD but remained anxious. Twenty-five children were then randomized to either placebo or fluvoxamine (while remaining on methylphenidate) for 8 weeks. At the end of the study period, there was no difference between the treatment groups in terms of anxiety. Thus, the efficacy of this approach remains to be established. Alternatively, atomoxetine may treat both ADHD and anxiety. A large sample (n = 162) of children with ADHD/anxiety were randomized to receive either atomoxetine or placebo for 12 weeks in a double-blind, placebo-controlled, parallel-groups design (D. A. Geller et al., 2007). An ADHD rating scale and an anxiety scale were used to assess the different comorbid symptoms over the course of the trial. *Both* ADHD and anxiety symptoms were reduced significantly on atomoxetine relative to placebo; clinicians also rated those children on atomoxetine as showing greater global improvement. Cognitive-behavioral therapy is a highly effective treatment for a variety of childhood anxiety disorders (Pliszka, 2011), so it also may be combined with pharmacological treatment of ADHD in order to address both problems.

The relationship between ADHD and posttraumatic stress disorder (PTSD) is complex. Wozniak and colleagues (1999) found no statistically significant difference in the rate of trauma exposure between an ADHD sample (12%) and controls (7%). While MDD was associated with exposure to trauma, ADHD was not, and exposure to trauma was rare in this sample. Ford and colleagues (Ford et al., 1999, 2000) examined the problem from a different angle, looking at a large sample of children treated in an outpatient clinic for trauma exposure. Once they controlled for a variety of family factors associated with trauma (parental psychopathology, social adversity, poor parenting), they found PTSD to be associated primarily with ODD rather than with ADHD itself. Only 6% of the children with ADHD alone had PTSD, compared to 22% of the children with ADHD and ODD. Nonetheless, recent studies have shown an increased risk of PTSD in adults with ADHD (Adler, Kunz, Chua, Rotrosen, & Resnick, 2004; Kessler et al., 2006). Biederman and colleagues (2013) obtained a large battery of psychiatric and neuropsychological measures on 271 youth with ADHD and 230 controls and their siblings both at baseline and at follow up 4–11 years later. Rates of PTSD were significantly higher in those with ADHD (5.2%) than in controls (1.7%). Comorbidity of PTSD did not affect the age of onset, severity, or nature of symptoms of ADHD; mean age of onset of PTSD was significantly later than that of the ADHD symptoms. Those with ADHD and PTSD also had higher rates of other disruptive, anxiety, and mood disorders. PTSD was not elevated in the siblings of those with ADHD alone, whereas ADHD was increased in the siblings of those with both ADHD and ADHD + PTSD. The latter finding (together with the age of onset of PTSD vs. ADHD) indicates that it is unlikely that ADHD is in fact an underlying or masked form of PTSD. ADHD is a risk factor for PTSD, however, and the two disorders may share familial etiological factors.

TIC DISORDER AND OBSESSIVE–COMPULSIVE DISORDER

There is a genetic link between tic disorder and obsessive–compulsive disorder (OCD) because individuals with OCD have a higher than expected number of relatives with tic disorder and vice versa (Pauls et al., 1995; Pauls, Leckman, & Cohen, 1993). Roughly 13–26% of patients with OCD have tic disorders (D. A. Geller, 2006), while up to 50% of patients with full-blown Tourette syndrome have symptoms of OCD (Swain, Scahill, Lombroso, King, & Leckman, 2007). Simple and transient tics are quite common in the pediatric population, affecting 6–20% of all children (Khalifa & von Knorring, 2005), while prevalence of chronic motor tics and Tourette syndrome combined is 1.2% (Kraft et al., 2012). Onset of tic disorder is gener-

ally between ages 3 and 5 years, with peak prevalence at 9–12 years. Only about 20% of children continue to experience tics in adulthood (Bloch et al., 2006; Swain et al., 2007). Whereas 50 to 60% of children with tic disorders have ADHD (Rothenberger, Roessner, Banaschewski, & Leckman, 2007), only about 10% of children with ADHD have a tic disorder (MTA Cooperative Group, 1999a).

Reviewing the extensive clinical and neuropsychological literature on the overlap of ADHD and tic disorders, Rothenberger and colleagues (2007) concluded the following:

- Tic disorders and ADHD are not alternative phenotypes of a single underlying genetic cause; ADHD with tic disorder may be a separate etiological entity.
- In comorbid ADHD and tic disorder, ADHD is associated not with tic severity but with learning problems, disruptive behavior disorders, and social dysfunction.
- The presence of tic disorder does not have any impact on the symptoms or severity of the ADHD itself. Greimel, Herpertz-Dahlmann, Gunther, Vitt, and Konrad (2008) did not find any differences between children with ADHD with and without tics on several laboratory measures of impulsivity, executive function, and sustained attention.

The natural course of tic disorder parallels the time that children with ADHD are likely to start and stop stimulant treatment, and this may have led to the false belief that stimulants "unmask" or "cause" tics. Ninety-one children with ADHD, with and without comorbid tics (but who did not have a formal tic disorder), were randomly assigned to receive stimulant or placebo in a 1-year prospective study (Law & Schachar, 1999). New tics developed in 19.6% of the children without preexisting tics who received methylphendiate and in 16.7% of those receiving placebo. Deterioration of tics was observed in 33% of children with preexisting tics who received methylphenidate and in 33% of those who received placebo. Thus, contrary to common clinical lore, stimulant treatment did not lead to an increase in tics. Perhaps even more surprising, multiple controlled studies of patients with comorbid ADHD and tic disorders have failed to show any increase of tics when on stimulant relative to placebo (Bloch, Panza, Landeros-Weisenberger, & Leckman, 2009). Tricyclic antidepressants and atomoxetine reduced tics relative to placebo (Allen et al., 2005; Spencer, Biederman, Kerman, Steingard, & Wilens, 1993). In a multicenter, randomized, double-blind clinical trial, 136 children with ADHD and chronic tic disorder were randomly administered clonidine alone, methylphenidate alone, a combination of the two, or placebo in a 2×2 factorial design for 16 weeks (Tourette's Syndrome Study Group, 2002). For the primary outcome measure of ADHD, significant improvement occurred for subjects assigned to clonidine and those assigned to methylphenidate. Compared with placebo, the greatest benefit occurred with the combination of clonidine and methylphenidate. Worsening of tics was no higher in those treated with methylphenidate (20%) than in those administered clonidine alone (26%) or placebo (22%). Remarkably, when compared with placebo, measured tic severity lessened in all active treatment groups, with the greatest reduction of tics found in those treated with the combination of clonidine and methylphenidate. In those patients whose tics remain problematic when the ADHD is treated with stimulant, adding an alpha agonist such as clonidine or guanfacine is an effective strategy.

While OCD is not found at elevated rates in patients with ADHD, 51% of children and 36% of adolescents with OCD also meet criteria for ADHD. The ADHD in these cases is not accounted for as an epiphenomena of the OCD itself (i.e., as obsessions distracting the patient) (D. Geller et al., 1998; D. A. Geller, 2006; D. A. Geller et al., 2002). Examining the triple comorbidity of ADHD, tic disorder, and OCD adds a layer of complexity. Using a worldwide database on patients with tic disorders, Freeman (2007) found that 21% of children with ADHD and Tourette syndrome also had OCD relative to 16% of those with Tourette syndrome who did not have ADHD. Masi and colleagues (2006) compared children with ADHD, ADHD/OCD, and controls in terms of OCD symptoms, psychiatric diagnoses, and social functioning. Children with and without ADHD did not differ in prevalence of ordering, aggressive, contamination, or hoarding obsessions. Those with ADHD/OCD had generally poorer social functioning compared to those with OCD only; they also had a higher prevalence of bipolar, tic, and oppositional disorders, but a lower prevalence of depression. Similarly, Sukhodolsky and colleagues (2005) found that children with ADHD/OCD have poorer social skills both at home and school, less family cohesion, and higher ratings of internalizing symptoms than children with OCD only (who in turn were more impaired on these measures than controls). As with tic disorders, the greater impairment of the ADHD/OCD

was driven primarily by the presence of the ADHD and other externalizing diagnoses associated with it, rather than any unique interaction of the ADHD and OCD symptomatology. Family studies have looked at the rate of both OCD and ADHD in the relatives of children with OCD alone, ADHD alone, and those with ADHD/OCD compared to controls (D. Geller et al., 2007a, 2007b). Again, as with tics, the rate of ADHD is elevated in the relatives of patients with ADHD, and OCD is elevated in the relatives of patients with OCD, but ADHD/OCD combined is only found in relatives of those patients with ADHD/OCD (cosegregation).

SUBSTANCE ABUSE

A more complete discussion of substance use and abuse in ADHD can be found in Chapter 11. A meta-analysis of longitudinal studies shows clearly that ADHD is a risk factor for future nicotine (OR = 2.36), alcohol (OR = 1.35), and cannabis (OR = 1.51) abuse, with less precise but still elevated risk of abuse of the other psychoactive substances (Charach, Yeung, Climans, & Lillie, 2011). As noted in Chapter 11, the comorbidity of CD in childhood is the strongest predictor of future substance use/abuse. Of interest, a number of other clinical features (family history of substance use disorder, executive function defects) have not been found to predict substance use (Wilens et al., 2011; Wilens & Morrison, 2011). Exposure to maternal substance abuse increases a youth's later risk for substance abuse, but ADHD does not further increase this risk (Yule, Wilens, Martelon, Simon, & Biederman, 2013). Prenatal exposure to nicotine increases the risk for neurodevelopmental disorders and future substance abuse (Ernst, Moolchan, & Robinson, 2001). In a large community sample in Europe (which excluded subjects with any psychiatric disorder, including ADHD), smoking during pregnancy was associated with greater inattention and increased smoking in the adolescent. During a reward task, subjects exposed to prenatal nicotine had reduced activation of the ventral striatum on fMRI (Muller et al., 2013), a pattern also seen in adolescents with ADHD (Scheres, Milham, Knutson, & Castellanos, 2007).

A meta-analysis indicated that treatment of ADHD with stimulants in childhood does not increase the risk of later substance use, though it also may not provide a protective factor (Humphreys, Eng, & Lee, 2013). What about the acute treatment of youth who have comorbid ADHD and substance abuse? Riggs, Hall, Mikulich-Gilbertson, Lohman, and Kayser (2004) randomized 69 adolescents with CD, substance use, and ADHD to a 12-week clinical trial of pemoline (n = 35) or placebo (n = 34). Pemoline was more effective than placebo for ADHD symptoms, but it had no effect on substance use. Over 300 youth with ADHD and substance use were randomized to either placebo or osmotic, controlled-release oral delivery system (OROS) methylphenidate; all subjects received cognitive-behavioral therapy aimed at the substance use. The stimulant reduced parent- but not youth-rated ADHD symptoms relative to placebo, but both groups showed similar declines in substance use. Similar results were found in a placebo-controlled trial of atomoxetine for comorbid ADHD and substance use (Thurstone, Riggs, Salomonsen-Sautel, & Mikulich-Gilbertson, 2010). Thus, ADHD that is comorbid with substance abuse can be treated pharmacologically without further increasing or decreasing substance use.

INTELLECTUAL DISABILITY

ADHD occurs in children with intellectual disability (ID) at prevalence rates of 18–40%, compared to 7–10% in the general population (Epstein, Cullinan, & Polloway, 1986; Koller, Richardson, Katz, & McLaren, 1983; Pearson & Aman, 1994). Several large-scale studies that examined the prevalence of ADHD symptoms in those with ID have concluded that the increased rate cannot be accounted for by rater bias or by confounding associations with other psychiatric conditions (Hastings, Beck, Daley, & Hill, 2005; Simonoff, Pickles, Wood, Gringras, & Chadwick, 2007). The presence or absence of ADHD is not related to the underlying etiology of the ID (Pliszka, 2009).

Over the last 15 years, at least 20 randomized controlled trials have examined the effects of methylphenidate in children with ADHD who have comorbid ID (see Handen & Gilchrist, 2006, for a review). The response rate to methylphenidate (45–66%) significantly exceeds that of placebo and is only slightly below the response rate for typically developing children with ADHD. Some studies have shown that an IQ above 50 predicts a better response to stimulant medication (Aman, Kern, McGhee, & Arnold, 1993; Aman, Marks, Turbott, Wilsher, & Merry, 1991) and that very low IQ levels (severe, profound) predict a poorer response (Aman, Buican, & Arnold, 2003). There is evidence that children with ADHD and ID are at a higher risk for both tics and social withdrawal than are typically developing children (Handen, Feldman, Gosling,

Breaux, & McAuliffe, 1991). While somewhat more conservative dose titration would be wise in this population, ADHD should not be left untreated in the child with ID; clinicians and families should never dismiss the symptoms of ADHD as "normal" for a child with ID because of a reduced mental age.

AUTISM SPECTRUM DISORDERS

DSM-5 combines autistic disorder, Asperger's syndrome, and pervasive developmental disorder into the single category autism spectrum disorder (ASD). It also removed ASD as an exclusionary criterion for the diagnosis of ADHD—a criterion that both clinicians and researchers have long ignored. Reviews of prevalence studies have shown that 30–80% of individuals with ASD meet criteria for ADHD, and those with ADHD have ASD traits above the levels in the general population (Rommelse, Franke, Geurts, Hartman, & Buitelaar, 2010). The study of ADHD and ASD is of particular interest because these two disorders, once thought exclusionary of each other, are increasingly thought to share clinical and etiological factors (Rommelse, Geurts, Franke, Buitelaar, & Hartman, 2011). This review notes that ADHD and ASD both have early onset (though the onset of ADHD is earlier), high heritability, comorbidity with learning/language problems, and shared genes. In twin studies, ADHD and ASD traits can be assessed dimensionally; the latter is typically done with an instrument such as the Social Communication Questionnaire (SCQ). In children, correlations between ASD and ADHD traits are .54 for parent ratings and .51 for teacher ratings, with estimates of genetic correlations of greater than .5 (Constantino, Hudziak, & Todd, 2003). In young adult twins, the genetic correlation between ADHD and ASD traits on self-report scales was .72 (Reiersen & Todd, 2008). Some caution is required, however, since some items on social questionnaire scales (e.g., "Does not understand others' feelings") may be rated higher in children with ADHD due to the comorbidity of ODD or CD rather than to autistic lack of empathy.

The relationship between ASD and ADHD was explored by performing LCA on data from the Conners Parent Rating Scale—Revised, Long version (CPRS-R:L) and the SCQ obtained from a community sample of 644 participants ages 6–17 years (van der Meer et al., 2012). The participants did not undergo a clinical interview to formally make either diagnosis. The LCA study can show that different symptom ratings group together to form identifiable subsets of diagnoses. About 65% of the sample fell into the normal range on both scales. Sixteen percent of the sample had scores on the CPRS-R:L in the clinical range for ADHD but minimal ASD symptoms. *No* class of children that emerged had only ASD symptoms without ADHD symptoms. Those with elevated ASD symptoms fell into two classes: those with predominately ADHD symptoms with significant ASD problems (ADHD + ASD, 9.2%) and those with more prominent ASD symptoms with problematic ADHD symptoms (ASD + ADHD, 9.0%). Many of these children were unlikely to meet criteria for a formal diagnosis of ADHD or ASD (i.e., the study should *not* be interpreted to mean that 35% of the population meet criteria for either ASD or ADHD!). The study does show that many children have significant levels of both traits. Membership in each class was related to performance on neurocognitive tasks. The ADHD class was impaired only on tasks of motor inhibition and working memory; those with ADHD/ASD were also impaired on a measure of facial emotion recognition.

Over the last several decades, multiple candidate gene, linkage, and genomewide association studies have been performed on thousands of subjects in an attempt to discover genes for both ADHD and ASD. These studies increasingly show an overlap of ADHD and ASD (Nijmeijer et al., 2009). Nijmeijer and colleagues (2009) obtained the SQCs for over 1,000 children with ADHD and their siblings (none of the subjects met formal criteria for ASD). DNA was obtained from the subjects and their parents, and multivariate quantitative trait locus (QTL) linkage was examined for ADHD and ASD symptoms to identify loci in the genome related to ADHD, ASD-like traits, or both. While none of the loci identified reached genomewide significance, the study suggested separate loci underlying ASD symptoms on chromosomes 7q, 12q, 15q, 16p, and 18p. The loci on chromosome 12, 16, and 18 were also related to ADHD, suggesting a possible genetic cause for the comorbidity. In an extensive review, Rommelse and colleagues (2010) identified 16 single-nucleotide polymorphisms (SNPs) from ADHD studies that might be involved in ASD, as well as 25 SNPs from ASD studies that could be related to ADHD.

The pharmacological treatment of ADHD in those with ASD is becoming increasingly well established. A large-scale stimulant trial has established the efficacy and safety of methylphenidate in this population (Research Units on Pediatric Psychopharmacology [RUPP]

Autism Network, 2005). Children with ASD (n = 72) received a test dose of methylphenidate; 66 of those who tolerated it were enrolled in a 4-week, double-blind crossover study of placebo and three different doses of methylphenidate. Adverse events (though none were serious or life-threatening) led to discontinuation of medication in 18% of the participants. Overall, 49% of the subjects were considered methylphenidate responders. Thus, while stimulants are helpful in reducing inattention and hyperactivity in children with ASD, one must expect more treatment failures in these children than in more typically developing children with ADHD. In an 8-week, double-blind, placebo-controlled trial of atomoxetine in 97 patients with ADHD/ASD, 21% were judged "very much improved" on active drug, compared to only 9% on placebo (p = .014) (Harfterkamp et al., 2012). Adverse events were typical of atomoxetine, but none were serious. Guanfacine significantly reduced symptoms of ADHD in patients with ASD in an open trial (Scahill et al., 2006) and was superior to placebo in a small (n = 11) controlled crossover study (Handen, Sahl, & Hardan, 2008). ADHD in the patient with ASD should be treated just as it is in the patient without ASD, with the caveat that there are likely to be more nonresponders. However, there is no evidence that severe, adverse psychiatric events are to be anticipated.

THE END OF COMORBIDITY?

Pliszka (2009) reviewed the diagnoses of 1,035 patients ages 3–18 years in a university-based child and adolescent psychiatry clinic (see Figure 5.1). Whereas only 27% had no comorbidity, many children had more than one comorbid diagnosis. Figure 5.1 shows only those with up to three diagnoses. Pliszka also noted the presence of about 40 children who had four or more diagnoses. This challenges the view of examining co-

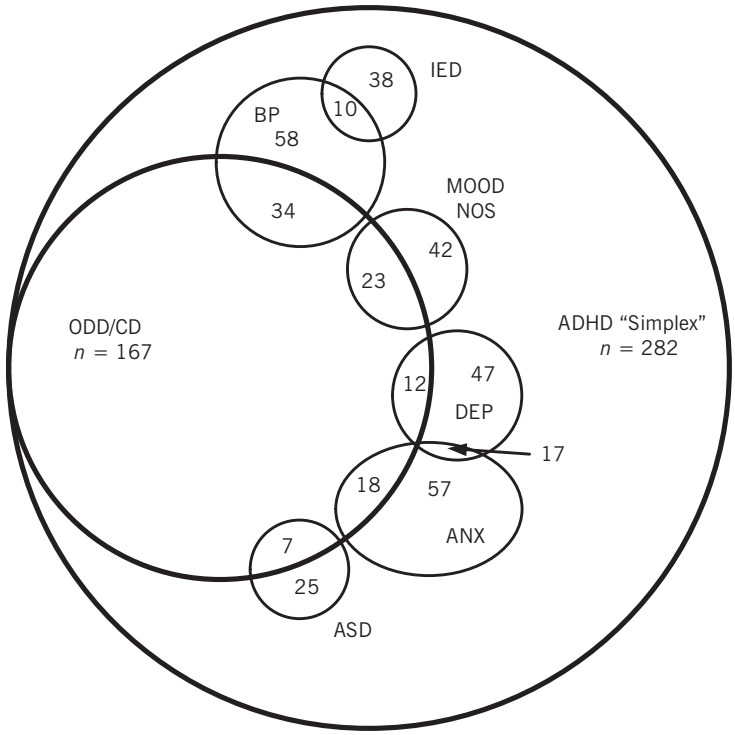

FIGURE 5.1. Overlap of diagnoses in children and adolescents with ADHD with more than one comorbid diagnosis. From Pliszka (2009). Copyright 2009 by The Guilford Press. Reprinted by permission.

morbidity from the standpoint of just one other comorbid diagnosis (i.e., ADHD + ODD or ADHD + BP). Genetic and imaging studies increasingly challenge the categorical approach to the problem of comorbidity (Faraone, 2013). Larsson and colleagues (2013) studied over 60,000 persons with ADHD and their first- and second-degree relatives, who were matched with a control group of people without ADHD and their relatives. Conditional logistic regression was used to determine the risks of bipolar disorder and schizophrenia in the relatives of the two groups. First-degree relatives of the ADHD group were at increased risk of both BP and schizophrenia. The authors suggested that the co-occurrence of ADHD and BP, as well as ADHD and schizophrenia, is due to shared genetic factors. Another large-scale study found four genetic markers that surpassed the statistical cutoff for genomewide significance for *all* five disorders: ADHD, ASD, BP, MDD, and schizophrenia (Cross-Disorder Group of the Psychiatric Genomics Consortium, 2013). It is no longer a matter of one gene–one disorder or even separate genes for separate disorders. It would be tempting to believe that comorbidity emerges when an individual has both "ADHD genes" and "bipolar genes," but this is not case. Rather, there are risk genes that affect underlying neurodevelopment from which any of the major psychiatric disorders may emerge. Dimensional measures of ADHD are as heritable as the categorical diagnosis (70–90%) (Sherman, Iacono, & McGue, 1997). Disorders that share behavioral or cognitive features (impulsivity, working memory deficits) also may show similar abnormalities on neuroimaging studies (Pliszka, 2012). A new paradigm is needed that focuses on the principal dimensional impairments, such as inattention, impulsivity, mood regulation, social reciprocity, anxiety, negative cognitions and learning/language deficits. Each patient may vary along each dimension, with the "comorbid" individuals being impaired on multiple dimensions. These dimensions are also more likely to be related to underlying "endophenotypes," that is, neurobiological substrates which in turn are more directly related to genetic and environmental etiological factors.

KEY CLINICAL POINTS

- ✓ Between 67 and 80% or more of children and adults with ADHD have a second disorder, and many have a third disorder that coexists with their ADHD (comorbidity).
- ✓ The presence of the comorbid disorder(s) can impact current functioning, life course risks, and treatment planning, among other important clinical concerns.
- ✓ Among the most common comorbidities are ODD (45–84%) followed by CD (15–56%) and related substance use disorders, anxiety (25–50%), depression (up to 30%), disruptive mood dysregulation disorder, and to a much lesser extent BP (0–30%), PTSD (5–6%), tic disorders (10%), and OCDs.
- ✓ Among other factors, the component of dysregulation of emotion in ADHD may be a predisposing factor to and shared trait of comorbidity with ODD, anxiety, depression, and BP, among others.
- ✓ Likewise, irritability may also be a substantial risk factor for mood and anxiety disorders, as well as reactive aggression. The new diagnosis of disruptive mood disorder appears to capture this propensity for irritability and aggression, and is designed for children who previously were thought to have BP without mania. Little is known about the overlap of DMDD with ADHD and its impact on impairments, life course, and treatment planning.
- ✓ By adolescence or adulthood, some personality disorders are also more likely to be linked to ADHD.
- ✓ Children with comorbid ADHD + ODD/CD often have families with greater psychopathology and social adversity, and these children are at increased risk for antisocial activities, drug use, peer rejection, and school failure, among other adverse outcomes. They are also more likely to develop depression and anxiety disorders by adolescence.
- ✓ Children with ADHD in community samples have no higher risk for BP (0–2%) than the base rate in the population, which may also be the case for clinic-referred children with ADHD followed to adulthood. But some studies of clinic-referred children with ADHD show significantly higher risks (11–30%). Among children with BP, however, the risk for comorbid ADHD is quite high (60%+).
- ✓ The presence of BP with ADHD warrants management of the mood disorder first, before enacting ADHD-specific treatments, such as ADHD medications.
- ✓ Evidence is mixed on whether ADHD is associated with a higher risk for depression, with such risk being mediated more by comorbidity with ODD/CD, adverse life events, and parental history of depression. But 25–50% of depressed children and teens may have

comorbid ADHD, which increases their risk for suicide attempts.

- ✓ Children with ADHD and anxiety disorders may have a somewhat lower level of impulsivity, although results are mixed. Some research suggests that this comorbidity may increase positive responding to psychosocial interventions and may not be related to a reduced ADHD medication response, as was earlier believed. Nonetheless, stimulants are unlikely to manage comorbid anxiety; nonstimulants, such as atomoxetine, may be beneficial in managing anxiety in such cases. The addition of cognitive-behavioral therapy to target the anxiety symptoms might also be beneficial.
- ✓ While only a small percentage of children with ADHD have tic disorders or OCD, 50–60% of those with tic disorders and 36–51% of those with OCD may have ADHD. In this comorbidity, it is typically the ADHD that creates most of the impairments in major life activities.
- ✓ ADHD alone is a risk factor for future nicotine, alcohol, and cannabis use, while the presence of ODD/CD may further increase the risk for use and abuse of other substances. Treatment with ADHD medications does not predispose children to any risks for later substance use or abuse.
- ✓ Although children with ADHD have only a slightly greater risk for ID, cases of the latter do show elevated rates of ADHD (18–40%). The response rate of these comorbid individuals to ADHD medications is only slightly lower than that of individuals with ADHD only, unless IQ is below 50, in which case even a lower response rate may be expected.
- ✓ About 20–30% of children with ADHD may show elevated rates of ASD symptoms, while up to 30–50% of ASD cases may have comorbid ADHD. Comorbid individuals are somewhat less likely than individuals with ADHD only to respond positively to ADHD medications.

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CHAPTER 6

Educational Impairments in Children with ADHD

George J. DuPaul and Joshua M. Langberg

Children and adolescents with attention-deficit/hyperactivity disorder (ADHD) frequently exhibit clinically significant educational impairment. Prevalence rates reported for learning and/or achievement problems in samples of youth with ADHD range from 50 to 80%, depending on the definition of the problem (DuPaul & Stoner, 2014). The educational impairment of children with ADHD persists into adolescence and adulthood in the vast majority of cases (e.g., 75%; Hechtman, 2000; see also Barkley, Murphy, & Fischer, 2008, Chapter 12) and is one of the main reasons individuals with ADHD are referred for assessment and treatment (Loe & Feldman, 2007). Furthermore, the long-term connection between ADHD symptoms and delinquency is largely mediated by low academic achievement (Defoe, Farrington, & Loeber, 2013), thus highlighting the critical importance of educational impairment as an assessment and intervention target for this population. As a group, children and adolescents with ADHD experience significantly lower standardized achievement scores and school grades, and higher rates of grade retention and school dropout in comparison to their same-age peers (DuPaul & Stoner, 2014). Longitudinal research demonstrates that the educational impairments of youth with ADHD are best attributed to core characteristics of the disorder, such as deficits in executive function (EF) and symptoms of inattention, rather than to comorbid conditions such as oppositional defiant disorder (ODD) or conduct disorder (CD) (Langberg, Molina, et al., 2011; Massetti et al., 2008; Miller, Nevado-Montenegro, & Hinshaw, 2012).

Although a majority of youth with ADHD experience educational impairment, there is significant variability in the presentation, severity, and causes of this impairment. For some youth with ADHD, educational impairment is largely rooted in behavioral issues, such as inattention, distractibility, and restlessness, that interfere with the ability to be productive with work completion. Youth with ADHD may also exhibit specific skills deficits that interfere with learning, such as deficits in reading or mathematics ability. Many youth with ADHD also exhibit deficits in aspects of EF, such as self-regulation, decision making, and engaging in planned sequences of goal-directed behaviors (see Chapter 4). Youth with ADHD and EF deficits often experience problems with the normal behaviors that facilitate learning and performance, such as organization of school materials and time management (Langberg, Epstein, & Graham, 2008). Each of these problems alone, or in combination, can lead to the occurrence of negative educational outcomes. Importantly, the presentation of these problems is linked to development; the

behaviors that are most salient and impairing vary with the child's age/grade and contextual demands. Given the diversity of educational impairments experienced by youth with ADHD, and the fact that they manifest differently across development, evidence-based assessment and treatment can be complex.

Accordingly, the primary goals of this chapter are to (1) identify the main educational impairments experienced by youth with ADHD; (2) describe how to measure/assess each distinct aspect of impairment; and (3) briefly review the evidence for interventions that target educational impairment (see Chapter 24 for more details regarding school-based interventions). For our purposes in this chapter, "educational impairment" is defined as *a failure to learn, progress, or perform academically at a level that could reasonably be expected given the child's age, intellectual ability, and family–school context*. Importantly, this definition takes an idiographic perspective to defining impairment, whereby if full academic potential is not being realized, then educational impairment is considered to be present, regardless of the child's standing relative to peers. This definition also limits educational impairment to the realm of academic functioning rather than broadly defining educational impairment as difficulties in school. For this reason, we do not review the assessment/treatment of problems with school-based peer relationships or interpersonal functioning except as directly related to academics (e.g., see the section "Educational Impairment Exhibited by Preschoolers").

In this chapter, we use the terms "skills deficit," "performance deficit," and "academic enablers" to describe factors related to educational impairment. The term "skills deficit" refers to a lack or absence of specific skills/abilities, such as reading or mathematics skills, at a point in time when mastery of those skills would be expected. Skills deficits are often characterized as learning disabilities (LD). In contrast, the term "performance deficit" refers to problems in demonstrating already acquired knowledge or attained skills. Specifically, the child understands the material and has the necessary skills but has difficulty demonstrating this knowledge on tests, through homework, or verbally. Finally, the term "academic enabler" encompasses a range of behaviors that facilitate the process of learning and performance, such as managing and organizing homework assignments and studying effectively (DiPerna & Elliott, 2000). It is worth noting that the distinction between performance and enabler behaviors is not always clear, and some behaviors may fit into both categories. For example, making careless mistakes is covered under performance deficits in this chapter (i.e., careless mistakes prevent the student from demonstrating knowledge) but it may also be considered an enabler (i.e., test taking skills/strategies to review and check work for accuracy). Nevertheless, when describing educational impairments in this chapter, we differentiate among skills, performance, and enablers because each may require a different assessment tool and intervention approach. Furthermore, given that educational impairments often manifest differently across development, information is presented by grade in school (i.e., preschool, elementary school, and secondary school). Impairments, assessment, and intervention for elementary and secondary school students with ADHD are discussed separately for academic skills, performance, and enablers.

DEFICITS IN EDUCATIONAL FUNCTIONING ASSOCIATED WITH ADHD

Educational Impairment Exhibited by Preschoolers

Despite the many difficulties associated with clinically significant ADHD symptoms in young children, the vast majority of research studies on this disorder have been conducted with elementary school-age children. Although issues such as rapid developmental changes between ages 2 and 6 years make diagnosis of preschool-age children appear somewhat tenuous (Lahey et al., 1998), there is ample evidence that symptoms of ADHD emerge at a very young age (e.g., Egger, Kondo, & Angold, 2006; Sterba, Egger, & Angold, 2007; Strickland et al., 2011) in many, though by no means all, cases and are associated with significant deviations in brain structure (Mahone et al., 2011). Furthermore, ADHD-related characteristics observed in young children mirror those of older children with respect to prevalence, subtypes, and gender differences, offering support for an accurate nosology.

ADHD in preschool-age children is associated with significant impairment in behavioral, social, and preacademic functioning, with affected children approximately two standard deviations below their typically developing peers in all three areas (DuPaul, McGoey, Eckert, & VanBrakle, 2001). Because ADHD tends to be chronic, at least 70–80% of preschool-age children with this disorder continue to exhibit significant ADHD symptoms during elementary school (Lahey et

al., 2004; Riddle et al., 2013). Children exhibiting high levels of hyperactive and impulsive behaviors (i.e., combined or predominantly hyperactive–impulsive presentations of ADHD) are at higher than average risk for developing other disruptive behavior disorders (i.e., ODD and CD, along with academic and social deficits; Campbell & Ewing, 1990). In addition, 59–67% of children with ADHD whose difficulties are persistent at school entry continue to show significant disruptive behavior disorder symptoms during middle childhood and early adolescence (Pierce, Ewing, & Campbell, 1999) and nearly 90% will fall short of being considered well adjusted as adolescents (Lee, Lahey, Owens, & Hinshaw, 2008).

Children's early experiences with literacy and numeracy have a significant influence on later academic skills. Specifically, children's experience with early literacy activities, such as those that increase phonemic awareness (Snow, Burns, & Griffin, 1998) and early numeracy activities (Gersten, Jordan, & Flojo, 2005), make a significant difference in their later language and literacy skills and mathematics achievement. Unfortunately, preschoolers with ADHD experience significant difficulties with early literacy and numeracy skills. For example, DuPaul and colleagues (2001) found that young children meeting diagnostic criteria for ADHD obtained significantly lower scores on a test of cognitive, developmental, and academic functioning compared to a sample of typically developing peers. On average, relative to mean scores obtained by typically developing peers, children with ADHD received scores one standard deviation below the expected mean for their age. This academic achievement gap is similar to that found for older children and adolescents with ADHD (Frazier, Youngstrom, Glutting, & Watkins, 2007) and suggests that many young children with ADHD enter kindergarten significantly behind their typically developing classmates in basic math and prereading skills (Spira & Fischel, 2005). Not surprisingly, 3- and 4-year-old children with ADHD are significantly more likely to receive special education services than their peers without ADHD (Marks et al., 2009). In fact, Marks and colleagues (2009) found that approximately 25% of their sample of children with ADHD received special education services relative to about 5% of control sample children. Of greatest concern, the educational impairments exhibited by young children with ADHD are associated with chronic underachievement in reading, math, and spelling throughout the school years (Massetti et al., 2008).

Although very few research studies have examined deficits in specific preacademic skills among preschoolers with ADHD, there appear to be at least three areas of educational functioning that may be affected: early literacy skills, early numeracy skills, and school-readiness skills and behaviors (DuPaul & Kern, 2011). Early or emergent literacy skills include three factors correlated with later reading achievement: oral language, phonological awareness and processing, and knowledge of print (Whitehurst & Lonigan, 1998). The latter two factors are particularly important in the development of early reading skills such as decoding (Lonigan et al., 1999). Several studies have shown statistically significant relationships between ADHD symptoms and emergent literacy skills (e.g., phonological sensitivity, print knowledge) in young children such that higher severity/frequency of ADHD symptoms is associated with lower performance on measures of early literacy (e.g., Lonigan et al., 1999; Sims & Lonigan, 2013). This relationship appears to be primarily accounted for by inattention symptoms as statistically significant correlations of low to moderate magnitude are typically found for measures of inattention, whereas correlations of early literacy with impulsivity (Sims & Lonigan, 2013) or hyperactivity (Lonigan et al., 1999) are near zero and not statistically significant.

Early numeracy skills include quantity comparison, oral counting, one-to-one correspondence, and number naming (Floyd, Hojnoski, & Key, 2006), as well as early geometry skills (e.g., shape naming) (Polignano & Hojnoski, 2012). The development of preschool math abilities has received much less attention relative to emergent literacy skills. In fact, we were unable to locate any studies examining the degree to which young children with ADHD exhibited deficits in specific numeracy or geometry skills beyond aforementioned lags in general math achievement scores. Based on research regarding early literacy skills, we presume that ADHD symptoms are associated with deficits in all areas of early numeracy development and that this association is particularly more pronounced in children with inattention rather than impulsivity and hyperactivity symptoms. Of course, this assumption requires greater empirical scrutiny.

The concept of school readiness includes the development of age-appropriate early academic skills (e.g., letter, number, and color recognition) as described previously, as well as self-regulatory behaviors including self- and social awareness (Bracken, 1998). Self-regulatory behaviors are very similar to academic en-

ablers (e.g., executive and organizational functioning) discussed later in this chapter in reference to elementary- and secondary-level students. Kindergarten teachers expect children to be able to follow directives, comply with rules, pay attention to instruction and assigned work, and organize classroom materials. Research studies have consistently demonstrated that young children with ADHD are approximately one standard deviation below their typically developing peers with respect to school readiness skills and behaviors (e.g., DuPaul et al., 2001). This is not surprising because ADHD symptoms, particularly in the inattention domain, overlap considerably with school readiness behaviors.

Educational Impairment Exhibited by Elementary School Students

Academic Skills

Elementary-age children with ADHD sometimes exhibit deficits in specific academic skills, such as reading or math. Meta-analyses, which combine information from all available studies, suggest that, on average, children with ADHD perform significantly below their peers in reading, math, and spelling (Frazier et al., 2007). Meta-analyses use effect sizes to quantify the magnitude of differences between groups. An effect size below 0.30 is considered a small difference, one in the 0.50 range is considered a medium difference, and an effect size around 0.80 is be considered a large effect/difference between groups (Cohen, 1988). In comparison to their peers, children with ADHD score on average 0.71 standard deviation units (i.e., a medium to large effect) lower on standardized achievements tests, which measure academic skills (Frazier et al., 2007). The largest differences appear to be associated with reading skills (d = 0.73), with moderate effect sizes reported for math (d = 0.67) and spelling (d = 0.55). There is also accumulating evidence to suggest that many youth with ADHD have significant difficulties with written expression, perhaps even to a greater extent than with reading (Mayes, Calhoun, & Crowell, 2000). Deficits in reading, math, spelling, and writing have been documented on standardized achievement tests, as well as assessed through the use of parent and teacher rating scales (Frazier et al., 2007).

It is important to note that meta-analytic studies report group-level differences, and not all elementary-age children with ADHD exhibit academic skills deficits. Furthermore, the Frazier and colleagues (2007) meta-analysis did not exclude youth with LD. Approximately, 1 in 3 children with ADHD also meet criteria for an LD (DuPaul & Stoner, 2014), with some studies reporting higher rates of overlap when problems with written expression are also considered (DuPaul, Gormley, & Laracy, 2013; Mayes et al., 2000). This relatively high comorbidity rate is important because it may be that group-level differences in academic skills are primarily driven by the one-third of the sample with ADHD that also has a comorbid LD (i.e., rather than skills deficits inherent to ADHD).

The relationship between ADHD and LD is complex, and the directionality is not entirely clear. Specifically, it may be that youth with ADHD do not acquire academic skills, such as reading and math, at the expected rate because their symptoms of inattention and distractibility interfere with learning (i.e., ADHD causes LD). Alternatively, it could be that an LD leads to a diagnosis of ADHD because children who cannot understand the subject matter being presented are likely to appear inattentive and distracted. Indeed, in samples of youth with LD, approximately 38% also meet criteria for ADHD (DuPaul et al., 2013). Likely, both scenarios occur because youth with ADHD and LD appear to share a common biological etiology and a genetic predisposition to both disorders (Willcut, Pennington, Olson, Chhabildas, & Hulslander, 2005). Regardless of the directionality of the relationship, it is clear that with 30– 45% of youth with ADHD also exhibiting sufficient skills deficits to warrant being classified as having an LD, clinicians need to be prepared to assess for skills deficits when seeking to identify causes of educational impairment.

In terms of development, early indicators of a skills deficit often include delays in speech and language development, and other prerequisites to academic achievement. For example, a young child might have a limited expressive vocabulary, "talk late," and be slow with naming objects or colors (Swanson, Harris, & Graham, 2013). In early elementary school, children with ADHD and skills deficits are often identified because they are struggling to learn to read. Specifically, children may display difficulty with phonemic awareness and other phonological processing skills, such as sound–symbol relationships or "phonics." Elementary-age children may also have difficulty with other aspects of phonological awareness, such as rhyming or syllable blending (Shanahan & Lonigan, 2010). As children progress with reading, they must learn to apply phonological and decoding skills fluently and automati-

cally. Children who are not able to apply these skills fluently often exhibit deficits with reading comprehension by middle to late elementary school (Swanson et al., 2013). In early to middle elementary school, children may also begin to exhibit difficulties acquiring math skills, such as learning math facts (e.g., addition and subtraction) and understanding concepts such as measurement (Anderson, 2010). For all academic skills (reading, math, and written language), it is important to acknowledge that students learn at different paces, and it can take some children more time than others to be able to apply skills fluently.

Academic Performance

Elementary-age children with ADHD frequently experience difficulties with accurately and efficiently completing classwork, homework, and tests. Specifically, when provided with an academic task such as a worksheet or a quiz to complete independently in a set amount of time, on average, youth with ADHD complete fewer problems and fewer problems correctly than do their peers (e.g., Atkins, Pelham, & Licht, 1985). Problems with productivity can largely be attributed to the core symptoms of ADHD, including high rates of off-task behaviors. Research has consistently demonstrated that students with ADHD exhibit significantly lower rates of on-task behavior relative to their elementary school peers (for meta-analytic review, see Kofler, Rapport, & Alderson, 2008). Differences in frequency and length of on-task behavior are evident across classroom activities (e.g., higher during small-group instruction relative to individual seatwork) and academic content (e.g., higher during less academic subjects like art and music relative to core academic subjects) (Imeraj et al., 2013). Presumably, because children with ADHD are easily distracted and frequently off-task, they are less efficient with work completion (Cantwell & Baker, 1991). Furthermore, a core symptom of ADHD is "fails to give close attention to details or makes careless mistakes in schoolwork" (American Psychiatric Association, 2013, p. 59). As such, even when children with ADHD are able to stay on-task, they often fail to pay attention to instructions, rush through assignments, and fail to check the accuracy of their work.

In terms of the relationship between ADHD symptoms and educational impairment, it is largely symptoms of inattention that drive problems with academic outcomes (i.e., as opposed to symptoms of hyperactivity and impulsivity). Multiple longitudinal studies have demonstrated that symptoms of inattention in early childhood predict academic performance throughout elementary school and into middle and high school (e.g., Langberg, Molina, et al., 2011; Massetti et al., 2008; Rapport, Scanlan, & Denney, 1999). Furthermore, the negative influence of inattention appears to increase (i.e., become more powerful) as youth move through elementary school and into middle school (Rowe & Rowe, 1992). It is also important to consider that approximately 60% of elementary-age children with ADHD meet criteria for ODD. ODD is characterized by the presence of noncompliant and defiant behaviors that can compound problems with academic productivity. Children with ADHD and ODD may actively defy parents and teachers, and refuse to start or to complete assignments.

Increasing evidence suggests that dysfunction in motivation and reward processing may also play a significant role in problems with academic productivity (e.g., Volkow et al., 2010). Specifically, children with ADHD appear to be particularly sensitive to immediate rewards and to have a difficult time getting motivated to work for rewards available in the future, even if those rewards are larger than those that are immediately available (Barkley, 1997; Sonuga-Barke, 2003). This has significant implications both for academic productivity and for engaging in academic enabler behaviors (see the next section). In terms of academic productivity, elementary-age children with ADHD frequently choose to engage in immediately rewarding behaviors (e.g., drawing, daydreaming, and talking with friends) over work completion, which is associated with delayed reward (i.e., later receiving a good grade). Stated differently, youth with ADHD seem to lack the motivation to devote sufficient time and effort to being productive with assignment/test completion because doing so is not immediately reinforcing (Barkley, 1997). The tendency of youth with ADHD to choose immediately available rewards over larger, long-term rewards has significant implications for interventions targeting educational impairment.

Academic Enablers

Even when academic skills and performance are not problematic, youth with ADHD may still experience educational impairment because of problems with academic enablers. As noted earlier, many youth with ADHD exhibit deficits in aspects of EF and have significant difficulty engaging in goal-directed behaviors

over prolonged periods of time (i.e., organization and planning behaviors). Academically, EF deficits in children with ADHD manifest as lost or misplaced homework assignments; disorganized desks and book bags; failure to record homework assignments accurately, if at all; and inefficient planning ahead to complete work (Langberg, Epstein, & Graham, 2008). The ability to organize behaviors and school materials effectively across time (i.e., plan out steps necessary to complete work) appears be particularly important for academic functioning (Abikoff & Gallagher, 2008; Langberg, Dvorsky, & Evans, 2013). Specifically, difficulties with organization of homework materials have been shown to predict the academic outcomes of youth with ADHD, above and beyond the traditional symptoms of ADHD (e.g., Langberg, Epstein, et al., 2011; Langberg, Molina, et al., 2011).

For youth with ADHD, the prominence of difficulties with academic enablers varies considerably across development, primarily as a function of changes in academic demands (e.g., workload) and in the types tasks the child is expected to manage independently. Often, by the time children reach third or fourth grade, they are expected to take some responsibility for organizing and managing their own school materials and for recording homework assignments accurately (Abikoff & Gallagher, 2008). Specifically, teachers provide fewer prompts about homework recording and are less likely to help students organize desks and book bags. Furthermore, around the same time, parents may begin to encourage their children to be more autonomous with homework management and completion. Typically, problems with materials organization and homework management (i.e., losing materials and assignments) become apparent and impairing in middle to late elementary school, whereas problems with planning, time management, note taking, and studying become more apparent in middle and high school, in which long-term assignments and cumulative exams are common (Evans, Serpell, & White, 2005).

Educational Impairment Exhibited by Secondary School Students

Academic Skills

Adolescents with ADHD continue to display significant deficits in reading, math, and spelling in comparison to their peers (d = 0.60; Frazier et al., 2007). In the Frazier and colleagues (2007) meta-analysis, the magnitude of skills deficits appeared to decrease from childhood (d = 0.75) to adolescence (d = 0.60). However, there were only seven studies of adolescents included in the meta-analysis. More recent longitudinal work confirms that skills deficits persist into adolescence and suggests that the magnitude of these deficits may actually increase. In the Multimodal Treatment Study of Children with ADHD (MTA), 436 children diagnosed with ADHD were followed into adolescence (M age = 16.6 at 8-year follow-up; Molina et al., 2009). Participants with ADHD performed significantly lower on math (d = 0.50) and reading (d = 0.65) standardized achievement tests in adolescence than did a group of matched peers without ADHD (N = 232). The magnitude of these differences is similar to what was reported in the Frazier and colleagues (2007) meta-analysis. In comparison, Massetti and colleagues (2008) compared the academic achievement of 125 children with ADHD, followed into adolescence, and 130 demographically matched comparison children, and examined the impact of ADHD subtype. Participants diagnosed with ADHD inattentive subtype (ADHD-I) in childhood exhibited significant skills deficits in adolescence in comparison to the control group, and the magnitude of the effect was large (d = 1.16 for reading; d = 1.30 for math). In contrast, comparisons between children diagnosed with ADHD combined subtype (ADHD-C) or ADHD hyperactive–impulsive subtype (ADHD-HI) and controls revealed small or nonexistent differences in academic skills in adolescence. These findings suggest that ADHD presentation/type may play an important role in predicting the persistence of skills deficits into adolescence.

In summary, academic skills deficits persist into adolescence, as measured by performance on standardized achievement tests, and these deficits are clinically significant and moderate to large in magnitude. Furthermore, inattentive symptoms appear to be the driving force in terms of predicting the maintenance of skills deficits over time (Massetti et al., 2008). It is important to note that in the longitudinal studies reviewed previously, only a small percentage of the samples continued to meet full diagnostic criteria for ADHD in adolescence (e.g., MTA = 30%; Molina et al., 2009). Therefore, it is clear that a childhood diagnosis of ADHD confers significant risk for the presence of academic skills deficits in adolescence regardless of whether the child continues to meet full diagnostic criteria for ADHD. This has also been found to be the case when these children with ADHD continue to be followed

into adulthood (Barkley et al., 2008; also see Chapter 12).

Academic Performance

Although symptoms of hyperactivity and impulsivity often decline–improve during adolescence, symptoms of inattention remain relatively stable (Hart, Lahey, Loeber, Applegate, & Frick, 1995; Martel, von Eye, & Nigg, 2012) and may actually worsen during developmental transitions (Langberg, Epstein, Altaye, et al., 2008). As such, it is not surprising that adolescents with ADHD continue to experience difficulties completing school work efficiently and effectively. In elementary school, difficulties with productivity are primarily seen in children's classwork and homework, such as while completing a math worksheet. Following the transition to middle school, grades become more heavily influenced by writing assignments, quizzes, and a few large exams, spaced-out across the academic semester (e.g., midterms/finals). Furthermore, teachers are more likely to "lecture" during class (Evans, Pelham, & Grudberg, 1995). Accordingly, adolescents with ADHD frequently exhibit problems with academic performance across a wider range of activities, including examination completion, studying for tests, homework completion, and note taking in the classroom (Evans et al., 2001). For example, an adolescent with ADHD may know how to study for tests (i.e., have the knowledge/skills set) but have significant difficulty independently remaining on-task and studying in an efficient manner. Clinically, this often presents as parents expressing concern that their adolescent is spending 2 or 3 hours per night studying and completing homework when it should take no longer than 1 hour. Similarly, adolescents with ADHD may not be productive with note taking during class and may fail to retain material from lectures adequately (Evans et al., 1995). In summary, the academic performance difficulties exhibited by children with ADHD frequently persist into middle school and high school, and become apparent across broader range of tasks.

Academic Enablers

As noted in the section "Educational Impairment Exhibited by Elementary School Students," many youth with ADHD exhibit deficits in EF, and these deficits are associated with educational impairment (Biederman et al., 2004; Miller et al., 2012). The period of early adolescence is thought to be when development of more complex EF skills, such as planning, organization, and self-regulation of these actions, takes place (Best, Miller, & Naglieri, 2011). At the same time that these skills are developing, they are becoming increasingly important for academic success. Specifically, surrounding the elementary to middle school transition, academic demands significantly increase as students are expected manage classwork, homework, and test preparation for multiple teachers (Eccles, 2004; Jacobson, Williford, & Pianta, 2011). Furthermore, students are asked to complete more long-term projects and homework assignments (e.g., book reports). These changes increase the importance of EF abilities such as goal-setting, organizing, and planning out actions in advance over extended periods of time. At the same time, parental and teacher support decline, and students are forced to self-regulate their own goal-directed organization and planning behaviors (Steinberg, 2005). Accordingly, during the period of adolescence, the EF deficits exhibited by students with ADHD often lead to significant problems with academic enablers and to additional educational impairment (Jacobson et al., 2011). In fact, academic enablers, such as homework management abilities, have been shown to mediate the relationship between ADHD symptoms of inattention and educational impairment (Langberg, Molina, et al., 2011). The meditational role for academic enablers is important because it suggests that interventions that improve ADHD symptoms will not necessarily impact educational impairment unless academic enabler behaviors also improve, thus possibly necessitating direct targeting of enablers (e.g., homework support strategies).

Academic Outcomes

Given that children diagnosed with ADHD continue to exhibit significant difficulties with academic skills, performance, and enablers into adolescence, it is not surprising that a diagnosis of ADHD is associated with numerous negative educational outcomes in adolescence and young adulthood. For example, in comparison to their peers, high school students with ADHD experience significantly higher rates of course failure, lower overall levels of class placement (Kent et al., 2011), and are more likely to drop out of school (Barkley et al., 2006). Adolescents with ADHD are also far less likely than their peers to enroll in a 4-year college (29.5% of the ADHD sample and 76.8% of the comparison sample; Kuriyan et al., 2013), and instead

are more likely to enroll in junior/community colleges (55% of the ADHD sample and 18% of the comparison sample; Kuriyan et al., 2013). Furthermore, recent research demonstrates that problems with educational impairment continue at the college level; college students with ADHD frequently struggle academically and fail to graduate (Weyandt & DuPaul, 2013; also see Chapter 12). Importantly, academic problems identified as early as elementary school have been shown to predict educational outcomes in late adolescence (Langberg, Molina, et al., 2011). As such, clinicians need to assess for problems with academic skills, productivity, and enablers accurately and comprehensively, so that interventions can be implemented and the occurrence of these negative outcomes is reduced.

MEASUREMENT OF EDUCATIONAL FUNCTIONING

Given the ubiquitous and potentially chronic educational impairment experienced by many children and adolescents with ADHD, clinicians must use psychometrically sound measures both to assess academic functioning as part of the diagnostic process and to document changes as a function of intervention. These measures and methods vary according to the purpose and focus of assessment. For example, measures used to establish impairment for the purpose of diagnosis differ from those used to assess progress with treatment. Also, assessments vary based on whether academic skills or enablers are being targeted. Finally, assessment types and content differ across age groups (preschool, elementary, and secondary). For a more general discussion of assessment of ADHD in children, see Chapter 16.

Measurement of Educational Functioning in Preschoolers

Direct assessment of early educational functioning involves both norm-referenced tests of academic achievement and school readiness, and criterion-referenced (curriculum-based) measurement of early literacy and numeracy skills (DuPaul & Kern, 2011). With respect to norm-referenced assessment, one option is to use standardized achievement tests that include normative data from preschool through high school and beyond. For example, the Woodcock–Johnson Tests of Achievement, Third Edition (WJ-III; Woodcock, McGrew, & Mather, 2001) includes 12 subtests that assess reading, math, and written expression for individuals ranging in age from 2 to 90 years. Of course, the primary limitation of the WJ-III and similar achievement tests for assessing young children is the high probability of floor effects given that most skills assessed by these measures are advanced for the preschool age group. A more developmentally appropriate option is to use norm-referenced tests of early language, math, and reading abilities such as the Bracken Basic Concepts Scale, Third Edition: Receptive (BBCS-3:R; Bracken, 2006b) and the Bracken Basic Concept Scale, Third Edition: Expressive (BBCS-3:E; Bracken, 2006a). These tests provide age-appropriate information about early academic skills, including school readiness. Furthermore, these measures provide specific assessment of school readiness, and receptive and expressive skills with normative data for children from age 3 years to age 6 years, 11 months.

Criterion-referenced tests specific to early academic skills may be of even greater value than norm-referenced achievement tests given that obtained data may translate more directly to instructional strategies and directly assess progress toward individual academic goals with treatment. For reading and language, Phonological Awareness Literacy Screening (Invernizzi, Sullivan, & Meier,, 2001) and Dynamic Indicators of Basic Early Literacy Skills (DIBELS; Kaminski & Good, 1996) have good psychometric properties with the early childhood population. Fewer measures are available for early math skills, but instruments include the Early Numeracy Skills Assessment (ENSA; Sokol, 2002) and the Preschool Numeracy Indicators (Floyd, Hojnoski, & Key, 2006; Hojnoski, Silberglitt, & Floyd, 2009).

Measurement of Educational Functioning in Elementary School Students

Assessment of educational functioning in elementary school students with ADHD may include norm-referenced achievement tests (i.e., for diagnostic purposes, to measure academic impairment relative to typically developing peers) and curriculum-based measurement (CBM) probes (i.e., for assessing treatment effects on academic skills). In addition, several methods and measures can be used to assess academic enablers and productivity, including behavior rating scales and collection of permanent products.

Norm-Referenced Achievement Tests

As described previously, on average, students with ADHD score about 0.71 standard deviation units below their typically developing peers on standardized achievement tests (Frazier et al., 2007). Thus, one way to establish whether there is educational impairment secondary to ADHD symptoms is to use a published, norm-referenced achievement test covering the basic skills areas of reading, math, and writing. Some of the more prominent standardized achievement tests include the aforementioned WJ-III (Woodcock et al., 2001), the Wechsler Individual Achievement Test, Second Edition (WIAT-II; Wechsler, 2001), and the Kaufman Test of Educational Achievement, Second Edition (K-TEA-II; Kaufman & Kaufman, 2004). All of these tests provide normative data across a wide age range with respect to reading, math, and writing skills. As such, they provide reliable and valid data regarding how a student's achievement in these areas compares with others of the same age or grade level. Specifically, clinicians can examine obtained standard scores to determine the degree to which a child is exhibiting academic impairment relative to grade-level expectations. The primary disadvantages of norm-referenced achievement tests is that they require more time and resources to administer than do other academic indices (e.g., report card grades, CBM probes) and typically are insensitive to treatment effects, at least over short periods of time, such as several weeks or months (Shapiro, 2011).

CBM Probes for Math, Reading, and Writing

Brief probes of a child's acquisition of skills being taught in the curriculum can be very helpful in several ways (Shinn, 1998). First, CBM data can pinpoint the instructional level of a child within a given subject area. In fact, one of the reasons that CBM methods were developed was to aid teachers in making instructional decisions. Thus, CBM can help determine how individual students are performing relative to expected benchmarks within the curriculum. Second, for exploring the possible diagnosis of ADHD for a given child, CBM data can help a clinician to ascertain whether a child's attention and behavior difficulties may result from the frustration of being asked to do academic work that is beyond his or her capabilities. Stated differently, it is possible that the child is being instructed at a *frustrational* rather than at an *instructional* level. It may be that a child is consistently being asked to complete work that is too easy (e.g., mastery-level material), resulting in attention problems due to boredom. This information can be very helpful in deciding whether inattention and/or hyperactivity–impulsivity difficulties are due to ADHD or are secondary to inappropriate educational placement and instruction (DuPaul & Stoner, 2014).

A third assessment purpose is to use CBM probes on a repeated basis over time to determine whether changes in instruction or implementation of intervention strategies lead to concomitant improvements in academic skills acquisition. Because CBM probes are relatively brief (2–3 minutes), it is feasible to collect these data periodically prior to and following implementation of academic or behavioral interventions, or even following initiation of pharmacotherapy. The primary index used to evaluate skills acquisition (i.e., growth) is slope; therefore, multiple data points are necessary to calculate slope reliably in each intervention phase (Shapiro, 2011). Typically, CBM data are collected two to three times per week over several weeks in order to establish slope under specific treatment conditions.

Measures of Academic Enablers and Productivity

Academic enablers and productivity are most commonly measured through parent- and teacher-completed rating scales and the collection of permanent products; best-practice procedures include a combination of these methods. Parent and teacher rating scales have been developed to evaluate a wide range of academic enabler behaviors, including homework management, homework completion, organization of materials, time management and planning, and study skills. Collection of rating scales from parents and teachers is considered best-practice because perspectives can vary considerably across informants–settings (Pelham, Fabiano, & Massetti, 2005). For example, a parent should be able to rate problems accurately during homework completion (e.g., distractibility and off-task behaviors while completing work at home), specific behaviors related to organization of materials (bringing the necessary books/materials home), and procrastination (waiting until the last minute to complete homework). In contrast, teachers may be less aware of these behaviors because they do not have the opportunity to observe the home setting (Pendergast, Watkins, & Canivez, in press),

but may be better equipped than the parent to identify problems with desk organization, being prepared for class (materials and homework), and work productivity in the classroom.

Two brief measures that focus on the ability to manage and complete homework include the Homework Problems Checklist (HPC; Anesko, Schoiock, Ramirez, & Levine, 1987) and the Homework Performance Questionnaire (HPQ; Mautone, Marshall, Costigan, Clarke, & Power, 2012; Power et al., 2006). Both of these measures include items related to the organization, management, and completion of homework materials, and have been shown to have adequate psychometric properties. Notably, only the HPQ has both parent and teacher versions (i.e., HPC is for parents only). The Children's Organizational Skills Scale (COSS; Abikoff & Gallagher, 2009) is a measure of organization, planning, and time-management skills that has parent, teacher, and child versions. Scoring the COSS yields three subscale scores that have been validated through factor analysis: Task Planning, Organized Actions, and Memory and Materials Management. Normative data are available for the COSS, and psychometric properties are adequate. The Classroom Performance Survey (CPS) is 22-item, teacher-completed measure that assesses problem behaviors commonly exhibited in the classroom by students with ADHD, including difficulties with being prepared for class, with materials organization, interacting effectively with teachers, and turning in homework assignments. A recent study of the CPS with an adolescent sample validated psychometric properties and provided normative data (Brady et al., 2012). Finally, the Academic Competence Evaluation Scales (ACES; DiPerna & Elliot, 2000) is a norm-referenced measure with student and teacher versions that measure a wide range of academic enabler behaviors, including academic motivation, study skills, and classroom engagement. The ACES has been used in multiple studies evaluating the impact of educational interventions and has good psychometric properties.

Regardless of the specific rating scales administered, permanent products should also be collected. Specifically, clinicians are encouraged to supplement rating scales with objective data, such as the percentage of homework assignments turned in daily/weekly, the percentage of classwork completed and/or completed accurately, school grades earned each semester and overall grade point average (GPA), and objective measures of binder/locker organization. The collection of these permanent products is especially important in the context of establishing treatment goals/targets and evaluating response to intervention. This is because permanent products, such as the percentage of assignments turned in daily, are going to be more sensitive to change/improvement and easier to collect repeatedly. Furthermore, permanent products, such as grades on examinations, tend to be more meaningful to stakeholders (i.e., parents and teachers).

Measurement of Educational Functioning in Secondary School Students

As was the case for elementary school students, assessment of educational functioning in secondary school students includes norm-referenced achievement tests, measures of academic enablers and productivity, and, possibly, CBM probes of academic skills.

Norm-Referenced Achievement Tests

Norm-referenced academic achievement tests can be used to assess the degree to which ADHD symptoms in adolescents are associated with academic impairment. Given the wide age and grade range for these instruments, clinicians can use the same tests that were recommended for younger children (i.e., WJ-III, WIAT, K-TEA). Obtained standard scores in reading, math, and writing can then be compared with age and grade norms to ascertain whether or not students are meeting expectations (i.e., possible impairment in academic achievement). The disadvantages of these measures are the same as for younger students, in that individual achievement testing requires time and resources. Furthermore, scores on these measures may not be sensitive to short-term treatment effects.

Other Measures of Academic Skill and Progress

Although CBM probes for secondary schools are available (e.g., Espin, Wallace, Lembke, Campbell, & Long, 2010), these measures are more limited at the secondary level because most students have progressed beyond basic skills acquisition in reading and math. Thus, other assessment techniques are necessary, particularly when documenting academic progress following intervention. One relatively crude measure of educational functioning is report card grades. Although grades have substantial face validity and are used to make high-stakes decisions (e.g., whether students pass a par-

ticular course), they are limited as measures of intervention effects because grades are typically issued on a quarterly (i.e., infrequent) basis. Another possibility is to obtain ratings of academic progress from teachers in a student's primary classes (e.g., English, math, science, and social studies). For example, the CPS was designed to assess the unique academic performance demands of secondary schools (Children and Adults with Attention Deficit Disorders [CHADD], 1996). The original CPS was a 20-item, Likert-type response measure that asked teachers to rate students' classroom behaviors pertaining to areas of strengths and weakness. A 15-item revised version of the CPS is available that comprises two factors: Academic Skills and Interpersonal Skills (Brady, Evans, Berlin, Bunford, & Kern, 2012). Both factors have been shown to have adequate reliability and validity. In particular, the Academic Skills factor can be helpful in obtaining teacher judgments regarding student educational functioning over a specific period of time.

Measures of Academic Enablers and Productivity

Each of the measures described in the previous section (e.g., HPQ, ACES) can also be utilized with secondary school students. However, as noted previously, additional enablers (e.g., time management, note-taking skills) become more relevant in middle and high school, and may need to be assessed. Furthermore, the aspects of materials organization that are most relevant often shift from organization of desks and book bags in elementary school to organization of lockers and binders in secondary school. Objective skills checklists that measure organization of binder, book bag, and locker, and time-management and planning skills have been developed and used in multiple outcome studies (e.g., Evans, Schultz, DeMars, & Davis, 2011), and are publically available (Langberg, 2011). Tools designed to evaluate the productivity and accuracy of note taking from lectures have also been developed for adolescents with ADHD (Evans et al., 1995).

In addition to the rating scales described in the previous section, the Learning and Study Strategies Inventory (LASSI; Weinstein & Palmer, 2002) is a student-completed rating scale that assesses multiple academic enablers, including time-management and study skills, use of study aids, and academic motivation. The LASSI is commonly used to evaluate academic enablers at the college level. Rating scales designed to measure EF can also be used to evaluate the academic enablers of adolescents with ADHD. For example, the Behavior Rating Inventory of Executive Function (BRIEF; Gioia, Isquith, Guy, & Kenworthy 2000) comprises 86 items rated on a 3-point scale (*never*, *sometimes*, *often*), with higher ratings indicating greater EF impairment. Several subscales on the BRIEF measure academic enabler behaviors, including the Planning and Organization and the Organization of Materials subscales. The BRIEF is psychometrically valid, and the Planning and Organization and Organization of Materials subscales have been shown to predict the school grades and homework problems of adolescents with ADHD (Langberg et al., 2013). As with the assessment of elementary-age students, permanent products, such as the percentage of homework turned in on time and/or hours spent completing homework, should also be collected.

INTERVENTIONS TARGETING EDUCATIONAL IMPAIRMENT

Because educational impairment is one of the most common and chronic difficulties associated with ADHD, children and adolescents with this disorder typically require early and ongoing academic support and intervention. At the very least, the effects of treatments used to address ADHD symptoms (e.g., stimulant medication, behavioral intervention) on academic skills and enablers should be assessed, so that the data can be used to make appropriate treatment decisions. Although methods commonly used to treat the educational impairments of youth ADHD are described briefly below, additional details regarding these treatment strategies are provided in other chapters (e.g., Chapter 24). Because very few research studies have specifically examined academic intervention effects for students with ADHD, we sometimes draw on empirical findings from other at-risk populations (e.g., students with LD).

Interventions to Support Development of Preacademic Skills in Preschoolers

Given the importance of reading for short-term and long-term academic achievement, many intervention approaches have been developed to enhance skills fundamental to reading development. It is important to note that no extant studies have directly assessed the effects of academic intervention strategies specifically for young children with ADHD. Thus, we have iden-

tified strategies that have been examined empirically with at-risk groups (e.g., children from low socioeconomic status [SES] families) or with the general population of preschoolers. A universal intervention that benefits all young children is for parents and teachers to embed strategies that promote early literacy into household and preschool routines and activities, respectively. For example, *Ladders to Literacy: A Preschool Activity Book* (Notari-Syverson, O'Connor, & Vadasy, 1998), can be used in preschool settings. The program offers multiple activity choices and opportunities for practice, which makes it appropriate for children at a variety of developmental levels. Activities and experiences contained in *Ladders to Literacy* fall into three broad areas that have been identified to influence children's literacy development, including print/book awareness, metalinguistic awareness, and oral language. *Ladders to Literacy* contains simple activities that are also feasible for parents and can be completed in the context of other activities (e.g., while washing the dishes or driving the car). An example of an activity is for parents to ask children to predict what will happen next when reading a familiar story.

For young children with ADHD who exhibit severe or refractory literacy deficits, more intensive early reading intervention strategies may be used, such as shared book reading (Justice, Kaderavek, Xitao, Sofka, & Hunt, 2009), phonological awareness training (Koutsoftas, Harmon, & Gray, 2009), explicit emergent literacy intervention (Justice, Chow, Capellini, Flanigan, & Colton, 2003), and computer-assisted instruction for phonological sensitivity (Lonigan et al., 2003).

One of the simplest yet most effective methods for building early reading skills is for parents and other adults to read books to children. In fact, shared book reading, without directly targeting oral language abilities, has been found to improve children's vocabulary, grammar skills, and letter-sound awareness (van Kleeck, 2008). The value of shared book reading is further enhanced when adults and children discuss story content in an interactive way. This is sometimes referred to as "dialogic reading" (Zevenbergen & Whitehurst, 2003). With interactive dialogue, shared book reading can address both decoding and comprehension skills. Comprehension skills are directly targeted by having adults ask literal (e.g., "What did the main character do?") and inferential (e.g., "How do you think the main character feels?") questions about stories and embedding scripted questions in storybooks before sharing them. Scripted questions are particularly important for adults who are less comfortable or experienced in reading to children and can structure the interaction in a way that best addresses comprehension skills. Family literacy workshops can be used to encourage parents to engage in effective book sharing techniques. In fact, Primavera (2000) found that book-sharing workshops led to significant increases in parents of low-income preschoolers reading to their children, as well as significant enhancement of children's language skills and interest in reading. Similar findings were obtained for a sample of Head Start preschoolers with language impairments, exposed to a book-sharing with embedded questions intervention twice per week over 8 weeks (van Kleeck, Vander Woude, & Hammett, 2006). In fact, moderate to large effect sizes in literal and inferential comprehension skills were found between the intervention and control groups in this study.

Comparatively little research has examined specific early intervention strategies for the development of mathematics skills, and no studies have focused specifically on effects for preschoolers with ADHD. Thus, recommended approaches have limited empirical support. The Number Worlds program (Griffin, 2007) is a promising intervention protocol that addresses early mathematics skills through direct instruction from prekindergarten through sixth grade. Mathematics is viewed as comprising three "worlds," including the world of real quantities that exist in space and time, the world of counting numbers and iconic symbols (e.g., spoken language), and the world of formal symbols (e.g., written numerals and operation signs). Number Worlds leads children through a developmental sequence that is consistent with the putative natural progression of math knowledge. The Number Worlds program has been evaluated longitudinally with several samples of children. Significant improvements in various math skills (e.g., number knowledge, computational skills) have been observed in children receiving instruction in the Number Worlds program relative to control participants (Griffin, 2007).

The early intervention for ADHD program described by DuPaul and Kern (2011) takes a similar approach to the Number Worlds program in promoting early numeracy skills. One session of group-based parent education is devoted to promoting numeracy skills, wherein parents are urged to take an active approach to incorporating numeracy learning into everyday activities. Furthermore, activities require children to manipulate objects in real-world tasks in order to make math concepts as concrete as possible. The primary

objective of home-based activities is for children to develop number sense. A child who has a well-developed number sense (1) is able to think about numbers in a variety of ways, (2) has a sense of what numbers mean, (3) is able to make comparisons, and (4) has the ability to perform mental math. Nine related number sense skills are addressed, including rote counting, quantity concepts, counting using one-to-one correspondence, representations, number identification, number naming, number writing, adding and subtracting, and fractions. In the final portion of the parent education session, parents pair up to generate ideas about possible activities for each math concept, then share these ideas with the larger group. Parents are given a homework assignment to write down four different ways that they will teach number sense skills to their children before the next parent education session. They are also asked to note how these activities worked, so that they can report back to the group at the beginning of the next session. Again, the specific effects of this strategy await controlled empirical evaluation.

Direct Interventions for Skills Deficits

As reviewed in the next section, most classroom interventions for elementary school students with ADHD have used behavioral strategies to improve academic performance and/or enablers. Over the past two decades, interventions directly targeting academic skills and abilities have been increasingly evaluated with ADHD samples. Exemplifying this point, DuPaul and Eckert (1997) conducted a meta-analysis of the school-based intervention for ADHD literature from 1971 to 1995, and found only eight studies of academic interventions over that time period (representing 12.7% of the intervention studies located for that meta-analytic review). Conversely, a meta-analysis of the more recent school-based ADHD intervention literature found 15 studies of academic intervention over the period 1996–2010, representing 25% of the available studies (DuPaul, Eckert, & Vilardo, 2012). In both meta-analyses, interventions directly targeting academic skills (e.g., peer tutoring, computer-assisted instruction) were associated with moderate to large effects on academic enabling behaviors (e.g., staying on-task, assignment completion). In fact, effect sizes associated with academic interventions were of similar magnitude to those obtained for behavioral interventions (e.g., contingent reinforcement). Academic interventions were associated with relatively small effects on reading and math skills in the earlier meta-analysis (DuPaul & Eckert, 1997), but with effect sizes of moderate magnitude in the more recent meta-analytic review (DuPaul et al., 2012). Alternatively, effects of behavioral interventions on academic skills were small in magnitude in both meta-analyses. Thus, it appears that academic interventions are valuable in terms of not only directly improving achievement but also impacting classroom behavior, perhaps to the same degree as more traditional behavioral strategies.

Academic interventions that have successfully improved reading, math, and other subject area skills of students with ADHD and related behavior disorders include peer tutoring (e.g., DuPaul, Ervin, Hook, & McGoey, 1998), computer-assisted instruction (e.g., Clarfield & Stoner, 2005), a self-regulated strategy for written expression (e.g., Lienemann & Reid, 2008), and explicit instruction (Nelson, Benner, & Boharty, 2014). These interventions are discussed in greater detail in Chapter 24.

We discuss two academic interventions in detail to illustrate the use of skills-oriented strategies in treating students with ADHD, and because these interventions provide a great example of using resources other than the classroom teacher to deliver treatment. Classwide peer tutoring (CWPT; Greenwood, Maheady, & Delquadri, 2002) involves splitting a classroom of students into two teams, then into tutoring pairs. Students in each pair take turns in the tutor and tutee roles. The tutor presents specified material (e.g., math problems), monitors tutee responses, provides praise and points for correct answers, corrects incorrect responses, and arranges for additional practice on missed items. During the 20-minute CWPT session, the teacher monitors pairs and provides bonus points for adherence to prescribed procedures and cooperative behavior. After the session is complete, students record their progress; the scores for each team are tallied, and one team is declared the winner. The CWPT procedure has been shown to enhance academic performance and reduce behavioral difficulties among students with ADHD, as well as their typically developing classmates (e.g., DuPaul et al., 1998).

Computer-assisted instruction (CAI) includes several features that may be helpful to students with ADHD. CAI can provide clear instructions, breaking measurable objectives into manageable goals that lead to immediate performance feedback. CAI can also present material with an interesting, interactive, game-like display using both visual and auditory stimuli

that highlight key information and limits distraction. CAI is particularly valuable in providing students with ADHD the opportunity to practice learned skills. Specifically, CAI has been found to increase time on-task, completion of coursework, and higher accuracy on assignments relative to typical paper-and-pencil classwork (e.g., Mautone et al., 2005). These effects have also been observed in children with comorbid ADHD and LD (Ota & DuPaul, 2002), suggesting that CAI may be helpful for students with ADHD and academic skills deficits.

Behavioral Interventions Targeting Academic Performance and Enablers

Behavior therapy/modification is the most commonly utilized evidence-based psychosocial approach for treating problems with academic performance and enablers (Pelham & Fabiano, 2008). Interventions grounded in behavior theory apply operant procedures, such as the manipulation of antecedents and consequences, to increase the occurrence of desired behaviors (Fabiano et al., 2009). Behavior modification has been used to treat children with ADHD for many years. Initially, behavior modification primarily addressed problems such as noncompliance, defiance, and off-task/productivity behaviors. Evidence-based interventions targeting these behaviors include behavioral parent training and classroom contingency management (Pelham & Fabiano, 2008). More recently, the principles of behavior modification have also been applied to problems with academic enablers, such as organization and time-management skills (see Storer, Evans, & Langberg, 2013, for a review). In general, the impact of behavior modification is large and significant for improving behavioral targets (e.g., disruptive behaviors; Fabiano et al., 2009) and small to moderate for addressing specific academic behaviors (e.g., assignment completion or reading fluency; DuPaul et al., 2012). Below, we review a few interventions that use the principles of behavior modification to address problems with work productivity and enablers (see also Chapter 24).

One of the most widely used behavior modification interventions is a Daily Report Card (DRC), which is designed to address the fact that specific, immediate, and frequent feedback is needed to facilitate behavior change in youth with ADHD. A DRC typically comprises three or four specifically defined target behaviors that a teacher monitors and tracks during the school day. Ideally, the teacher provides frequent feedback regarding the student's performance (e.g., praise and/or redirection) and sends the DRC home to parents each day after school for additional rewards/consequences to be applied (e.g., increased television time for meeting a DRC target). Disruptive classroom behaviors and academic productivity are common targets on DRCs. For example, a DRC target might be that the student completes all of the assigned classwork within the designated time and with at least 80% accuracy. Academic enabler behaviors can also be targeted on the DRC. For example, the DRC might involve the student coming prepared to class with all of the necessary materials, including a writing utensil, school binder, and completed homework. The efficacy of DRCs has been evaluated for children with ADHD as part of large, multimodal interventions (e.g., MTA; Arnold et al., 1997), as a stand-alone intervention (Owens et al., 2012), and as implemented through individualized education plans (IEPs; Fabiano et al., 2010). DRCs have been shown to improve the academic productivity and reduce disruptive classroom behaviors of children with ADHD (Fabiano et al., 2010; Owens et al., 2012), and they require minimal time and effort to implement.

The principles of behavior modification can also be used to teach and encourage students to apply academic enabler behaviors consistently. Most interventions that target academic enablers begin with a period of skills training, in which the student is taught specific strategies for note taking, organization, time management, and study skills, or some combination of these (e.g., Abikoff et al., 2013; Evans et al., 1995, 2011; Langberg, Epstein, Becker, Girio-Herrera, & Vaughn, 2012; Pfiffner, Villodas, Kaiser, Rooney, & McBurnett, 2013; Power et al., 2012). For example, a student might be taught a specific and structured way to organize a school binder. Similar to a DRC, specific targets/goals are set relative to implementation of skills, and monitoring/tracking is frequent. For example, an academic enabler target might include the following: The student brings his or her school binder to class at least 4 out of 5 days and all worksheets/papers are be filed in the appropriate section of the binder. Rewards and consequences are then applied as frequently and immediately as possible (often though the use of a point system) based the child's progress toward achieving the goal. Behavioral interventions targeting academic enablers have been shown to lead to large improvements in both parent and teacher ratings of academic impairment and

in more objective measures, such as school grades (e.g., Pfiffner et al., 2013).

Strategies to Prevent High School Dropout

Adolescents with ADHD are at higher than average risk for dropping out of school and not completing their high school education (Barkley et al., 2008; also see Chapter 12). Presumably, risk for dropout may be reduced when evidence-based treatments for ADHD are used on a consistent basis. Alternatively, rather than assuming that ADHD treatment will lessen educational risk, clinicians are advised to consider the use of dropout prevention strategies. A prominent example of an effective dropout prevention program is Check and Connect (Anderson et al., 2004). This program includes two components: (1) *Check*: systematically evaluate the student's engagement and functioning in school by monitoring classroom performance, behavior and attendance, and (2) *Connect*: establish a constructive mentoring relationship between the student and school-based coach (e.g., teacher, counselor, or administrator). Studies have demonstrated that the quality of staff–student relationships involved in *Check and Connect* predicts increased attendance, homework completion and interest in school for students with learning disabilities and behavior disorders (Anderson, Christenson, Sinclair, & Lehr, 2004). In *Check and Connect*, the coach regularly collects data related to key school functioning behaviors (e.g., attendance, tardiness, assignment completion) and records this information on tracking sheets. If behaviors exceed the established Check and Connect thresholds indicating risk, then the coach and student use problem-solving techniques to generate and implement a solution. For this strategy to be successful, it is critical that coaches establish a relationship with the students to keep the latter engaged and motivated to achieve at school.

IMPLICATIONS FOR FUTURE RESEARCH

Although increased clinical and research attention has been paid to the identification, assessment, and treatment of educational impairment in children and adolescents with ADHD, the empirical literature in this area is still in its infancy compared to the abundant research focused on ADHD symptoms, associated behaviors, and comorbid disorders. Given the ubiquitous, chronic impact of ADHD on school and academic outcomes, there is a critical need to increase the number and complexity of empirical studies focused on educational impairment. Three major directions are recommended for research in this area include comprehensive longitudinal investigations of educational impairment, identification of predictors of educational impairment, and further development of intervention targeting educational functioning.

Comprehensive Longitudinal Investigations of Educational Impairment

Most studies examining educational functioning in children and adolescents with ADHD have been cross-sectional (i.e., academic performance or achievement data collected on one occasion) (Frazier et al., 2007). This is problematic because the functional impairments associated with ADHD are chronic and dynamic, not static. Thus, cross-sectional studies provide a limited snapshot of the challenges that students with ADHD encounter across their school years. Another limitation of the extant literature is a relative preponderance of studies with elementary school-age children and scant empirical evaluation of academic difficulties experienced by preschoolers and adolescents with ADHD. Even when academic achievement is examined in longitudinal investigations, currently available studies typically employ relatively limited measurement of educational functioning. Most often, studies only use one or two measures, and many investigations rely exclusively on a single, norm-referenced achievement test. As discussed in this chapter, educational functioning is multifaceted and comprises skills and performance components. Therefore, a more comprehensive approach to outcome measurement is needed.

To address these important limitations, longitudinal investigations of educational functioning in children with ADHD should begin in preschool and extend through at least high school, if not postsecondary schooling. Furthermore, outcomes should be assessed using multiple methods, including norm-referenced achievement tests, criterion-referenced or CBMs, teacher ratings of academic skills and enablers, report card grades, and perhaps permanent products and/or direct observations of academic-related behaviors. Stated differently, educational functioning should receive the same emphasis as has been accorded ADHD symptoms, behavioral functioning, and psychological/psychiatric

comorbidities in prior longitudinal studies. The recommended longitudinal approach should yield data that will be critical in helping clinicians and researchers to advance our understanding of (1) the nature of educational impairments associated with ADHD, (2) the dynamic changes that may occur with respect to scholastic functioning, and (3) the critical time periods when intervention may be most needed (e.g., entry to elementary school, transition to middle school).

Identification of Predictors of Educational Impairment

Similar to the study of educational functioning in general, very few studies have sought to identify variables that may moderate or predict academic and school outcomes in children and adolescents with ADHD. Those studies that have been conducted have focused on generic, demographic characteristics such as gender, SES, and IQ or on broad ADHD symptom dimensions as predictors. As reviewed previously, there is ample evidence that inattention symptoms rather than hyperactivity–impulsivity are statistically significant predictors of educational outcome in this population (e.g., Massetti et al., 2008). Although such findings certainly advance our understanding of factors related to educational impairment, future studies should take a more comprehensive, multivariate approach to identifying predictors. The examination of a varied set of predictors, along with multiple measures of educational functioning, may not only increase the number of known moderators of outcome but may also indicate more specific predictor–outcome relationships. For example, it is possible that variables that predict outcomes for academic enablers may be different from factors that predict achievement in specific academic areas (e.g., Langberg, Molina, et al., 2011). The identification of predictors is critically important, particularly if these variables are malleable because these findings may help clinicians and researchers identify possible targets for prevention and early intervention programs.

Further Development of Interventions Targeting Educational Impairment

Rapid progress has been made over the last 10 years with respect to the development of interventions targeting educational impairment; however, a number of notable limitations remain. In comparison to research with elementary-age students with ADHD, there has been minimal research on psychosocial interventions that address the educational impairment of secondary-age students with ADHD (DuPaul et al., 2012). In particular, there has been almost no research evaluating educational interventions for high school and college students with ADHD. This is important because the types of interventions applied to elementary-age students with ADHD are unlikely to be effective with high school and college-age students. One reason for this is that high school and college-age students are expected to operate autonomously and receive minimal support from parents and teachers. Behavioral interventions for elementary-age students rely heavily on parents and teachers to provide structure, monitoring, and rewards. This is unlikely to be feasible or acceptable as a major component of interventions for high school and college age students, and alternate strategies will need to be developed and evaluated (Fleming & McMahon, 2012). These interventions are clearly needed; although the number of college students with ADHD is rapidly increasing, many are struggling academically and may fail to graduate (Weyandt & DuPaul, 2013).

Another limitation is the relative lack of interventions targeting educational impairment that are truly feasible for schools to implement. Delivering such interventions in school settings is associated with improved outcomes and greater prospects for generalization (DuPaul et al., 2012, Evans, Langberg, & Williams, 2003). However, many of the interventions that have been developed for youth with ADHD are multicomponent interventions that are staff- and resource-intensive. Even if these interventions are highly effective, it is not clear that schools have the expertise or resources needed to carry them out. Additional research is needed on interventions such as DRCs that can be implemented with minimal staff effort and resources. Research is also needed to understand better the efficacy of services currently provided in schools for youth with ADHD. For example, academic accommodations are commonly used by schools to address educational impairment even though very little is known about their efficacy (Harrison, Bunford, Evans, & Owens, in press). Furthermore, research is needed to evaluate different strategies for deploying and disseminating ADHD interventions into school settings and factors that predict schools adoption of evidence-based interventions. Without an increased focus on feasibility, acceptability, and deployment/dissemination, it is unlikely that the interventions being developed for educational impairment will reach the youth for whom they were designed.

CONCLUSIONS

The majority of children and adolescents with ADHD exhibit clinically significant impairment in educational functioning that is multifaceted and chronic. In particular, students with this disorder are at higher than average risk for underachievement, grade retention, referral for special education services, failing report card grades, school dropout, and lower completion of postsecondary education. Impairment can be experienced in the development of academic skills, performance, and/or enablers. Deficits in one or more of these areas not only hinder scholastic progress but also have important implications for individuals' eventual financial status and psychological well-being. Academic deficits can be identified at an early age (i.e., prior to school entry) and require multimodal, ongoing assessment throughout a student's educational career. Intervention strategies can be directed towards skills acquisition and fluency, consistent scholastic performance, improved academic achievement, and enhanced academic enablers. Ultimately, multiple areas of functioning may require intervention in order to optimize both short- and long-term progress. It is hoped that the next wave of empirical research will prioritize comprehensive assessment and treatment of educational functioning, preferably in the context of longitudinal designs that include multiple measures of outcome. Anything short of a more comprehensive approach to research, assessment, and intervention of academic functioning will limit our ability to promote successful outcomes for students with ADHD.

KEY CLINICAL POINTS

- ✓ The vast majority of children and teens with ADHD experience impairment in the educational setting.
- ✓ They manifest lower preschool academic readiness skills, lower academic achievement skills in formal school settings, skills deficits, performance deficits, and poor academic enabler behaviors. These are in addition to their ADHD symptoms, EF deficits, peer relationship problems, comorbid psychiatric disorders, and health problems, as identified in other chapters in this section.
- ✓ In academic skills, children and teens with ADHD show significant deficits in reading, math, spelling, and handwriting competencies, as well as a higher probability of qualifying for a learning disability (33–45%+).
- ✓ Performance deficits include high rates of off-task behavior, variable on-task behavior, less efficient approaches to work performance, careless work behavior, inability to sustain motivation to work as long as typically developing children, and reduced self-monitoring and self-correction of work.
- ✓ Poorer competencies in academic enablers include deficits in EF in daily life, such as time management, self-organization, problem solving, self-motivation, and emotional self-regulation, all of which further contribute to risk for academic failure.
- ✓ Deficits in academic functioning are more a function of the degree of ADHD inattention symptoms than of the hyperactive–impulsive symptom dimension.
- ✓ Significant transitions in academic settings over the course of development, such as the shift from elementary to middle school or from the latter to high school, may be associated with a worsening of symptoms and further impairments due in part to a reduction in external structure and increased emphasis on self-regulation associated with such transitions.
- ✓ Follow-up studies also show higher than typical rates of adverse academic outcomes, such as grade retention and failure to complete compulsory education, among others.
- ✓ Assessing the educational problems of children and teens with ADHD is complex and should include not only tests of basic achievement skills but also rating scales of academic performance and school enablers, and curriculum-based assessment and direct observations of in-school functioning, among other, more ethological approaches to documenting difficulties.
- ✓ Interventions for the educational impairments of children and teens with ADHD need to target not only the modification of ADHD symptoms and related problematic behaviors but also academic skills, performance-related behavior, and academic enablers, if improvements in more than just behavior are to be achieved. Additional interventions may be needed to target directly the increased risk for dropping out of school.

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CHAPTER 7

Families and ADHD

Charlotte Johnston and Andrea Chronis-Tuscano

Despite recognition that genetics and biological characteristics such as brain structure and function are central to the etiology of attention-deficit/hyperactivity disorder (ADHD) (Scassellati, Bonvicini, Faraone, & Gennarelli, 2010; also see Chapter 14), research convincingly indicates that parenting and parental psychopathology play an important role in determining the developmental course of ADHD and the occurrence of comorbid conditions (e.g., Faraone et al., 2005; Nigg, Hinshaw, & Huang-Pollock, 2006). We argue in this chapter that the family context that surrounds children with ADHD is crucial for both a complete understanding of the disorder at a nomothetic level and the idiographic understanding necessary to guide treatment planning for an individual child and his or her family. Family factors are seen as contributing to ADHD partly in a direct etiological fashion, and partly as important moderators and mediators of child outcomes and treatment effects. We place parent–child interactions at the nexus of the many family factors that impinge on and interact with child ADHD, recognizing the proximal nature of these interactions to the child and their potential as either powerful protective or risk factors. Our understanding of this central place of parent–child interactions in families of children with ADHD is grounded in a developmental–transactional framework, which recognizes that parents and children each contribute to the quality of family interactions, and that individual characteristics and relationship properties evolve dynamically over time.

We begin the chapter with an outline of this developmental–transactional framework and its implications for understanding, assessing, and treating ADHD within the family context. Using this framework as our reference point, we then review current knowledge regarding the centerpiece of the framework, parent–child interactions. In the following sections, we consider evidence addressing the impact of parent and family characteristics, including parent psychopathologies and marital and sibling relationships, on parent–child interactions and child ADHD. Although our theoretical model incorporates important extrafamilial influences, such as the school environment, cultural perspectives, and the impact of social disadvantage, we focus on the family context, and this precludes extensive consideration of these broader contextual factors. We conclude with a discussion of emerging directions and challenges in our understanding of the families of children with ADHD. Throughout the chapter, we strive to highlight research findings that may have par-

ticular relevance within the clinical context and hold promise for improving the lives and outcomes of these children and their families.

THE DEVELOPMENTAL–TRANSACTIONAL MODEL OF ADHD AND FAMILY FUNCTIONING

We believe that the most useful framework for understanding families of children with ADHD is one that acknowledges biological and social influences, recognizes the dynamic and interactive nature of influences among family members, and incorporates contextual factors both within and outside of the family (Johnston & Mash, 2001; Sonuga-Barke & Halperin, 2010). A simplified schematic representation of such a model is provided in Figure 7.1. Unpacking the model, we first highlight the strong heritability of ADHD symptoms (e.g., Nikolas & Burt, 2010; Waldman & Gizer, 2006) and the resultant recognition that an individual, typically a child diagnosed with ADHD, may well reside within a family in which other members also show high levels of ADHD symptomatology. We provide a more complete discussion of this possibility in the following section on parental psychopathology, including ADHD. In addition to the biological links in ADHD symptomatology among family members, research also suggests genetic overlap of ADHD with other disorders (e.g., oppositional defiant disorder [ODD] or conduct disorder [CD], referred to collectively as disruptive behavior disorders in this chapter; learning disorders, autism spectrum disorders, depression, substance use disorders, bipolar disorder) and with personality or temperament dimensions of neuroticism and low conscientiousness (e.g., Faraone, Biederman, & Wozniak, 2012; Lichtenstein, Carlström, Råstam, Gillberg, & Anckarsäter, 2010; Martel, Nikolas, Jernigan, Friderici, & Nigg, 2010; Taylor, Allan, Mikolajewski, & Hart, 2013; see Child Characteristics in Figure 7.1). An awareness of these possible shared biological underpinnings informs a more comprehensive understanding of the difficulties families of children with ADHD may be facing.

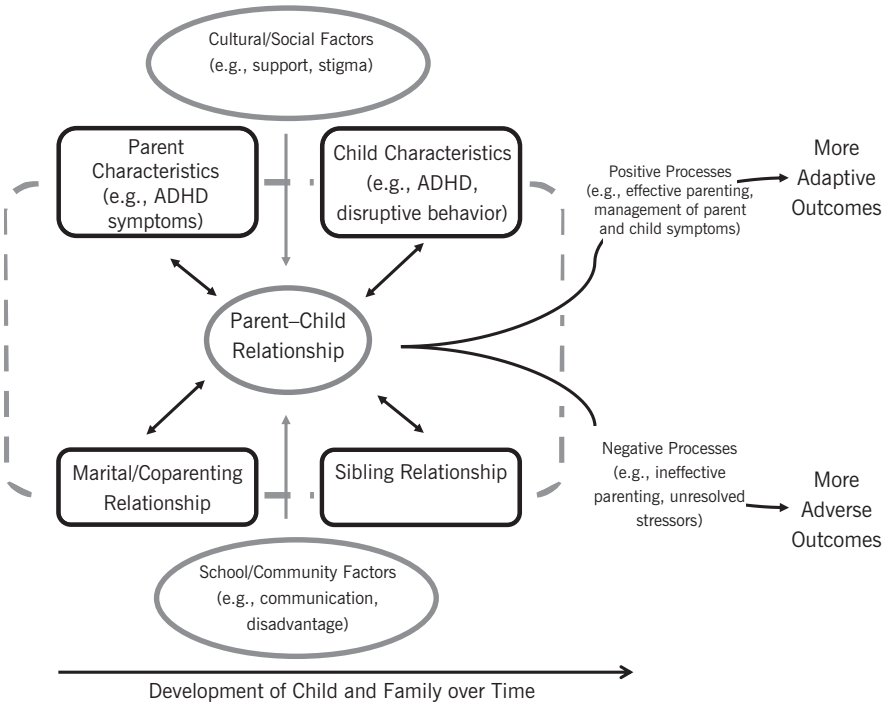

FIGURE 7.1. Developmental–transactional model of ADHD within the family context.

Sharing influence and often interacting with the child's genetic vulnerabilities to ADHD are the important and proximal impacts of functioning within the parent–child dyad (the Parent–Child Relationship at the center of Figure 7.1). We use the term "parent–child" to encompass the child's relationships with any and all relevant parenting figures in the family. Not only does parenting interact with the child's genetic endowment to modulate risk, but environmental factors such as parenting also appear to have epigenetic functions, whereby these environmental factors alter the functioning of genes in a way that contributes to stable changes in the expression of the child's genetic makeup (Nigg, 2012). Although direct effects on the child are acknowledged, other family variables, including parents' cognitions and characteristics and marital and sibling relationships, are seen as exerting their influence on the trajectory of child functioning primarily through the filter of parent–child interactions (e.g., Li & Lee, 2013; Nikolas, Friderici, Waldman, Jernigan, & Nigg, 2010; Pennington et al., 2009; see Parent Characteristics, Marital/Coparenting Relationship, and Sibling Relationship in Figure 7.1). Critically, within the developmental–transactional model, the characteristics and behaviors of family members and family subsystems are reciprocally influential, with each acting to change the other over the course of time and development (these influences are partially captured by the numerous recursive and continuous arrows in Figure 7.1). This fact necessitates an understanding of family functioning that resists a focus on unilateral effects (e.g., parent or child blaming) and places current functioning within the family's own historical context. Finally, related to the dynamic, ever-changing nature of the influences among family members is a recognition that patterns of functioning are tied to the developmental stage of the child, or of the family, and that a true understanding of the child only arises from knowledge of the developmental pathway that has led to the current picture of functioning.

In summary, at the center of understanding child ADHD is recognition of the constant flow of influences from child to parent and back again. Interacting with a child with ADHD is a stress-generating experience that can negatively alter parental cognitive, emotional, and behavioral functioning, especially if ADHD is comorbid with ODD (e.g., Fischer, 1990; Theule, Wiener, Tannock, & Jenkins, 2013). At the same time, parent factors, such as depression or poor self-regulation, serve to influence the quality of parenting and parent–child interactions, and ultimately adjustment and outcomes for children with ADHD (e.g., Johnston, Mash, Miller, & Ninowski, 2012). Both parent and child functioning are intertwined with other aspects of the family, such as functioning within sibling or interparental systems. Finally, consideration must be given to not only the mutual influences within the family but also the broader social context outside the family. ADHD does not exist in isolation from the influences of friends and relatives, the child's school, the neighborhood, and the family's cultural and/or religious affiliations. For example, over three-fourths of parents of children with ADHD report stigmatizing experiences as a result of their child's ADHD, including feelings of social isolation and dismissive attitudes by health professionals (e.g., dosReis, Barksdale, Sherman, Maloney, & Charach, 2010; Mikami, Chong, Saporito, & Na, 2013).

This theoretical framework not only serves to guide research questions and methods in the study of ADHD, but it also is intended to highlight how clinicians can best approach assessment and treatment of the disorder. Assessment should focus on not only the child with ADHD but also the familial and social-contextual factors that may serve to exacerbate or ameliorate the child's symptoms and level of functioning. In turn, treatment must recognize and incorporate the parental, family, and social resources surrounding the child with ADHD and work to maximize the strengths of the child and family, while minimizing the impact of existing individual, familial, and social risk factors.

PARENT–CHILD RELATIONSHIPS AND ADHD

Using the framework of this developmental–transactional model as a backdrop, we now turn to a discussion of what is known about the centerpiece of the model, parent–child interactions, within families of children diagnosed with ADHD. Reviews of research characterizing parent–child interactions in families of children with ADHD have consistently yielded a picture of high levels of parenting stress and conflicted parent–child interactions characterized by both increased directiveness or authoritarian parenting and reduced warmth or positivity (e.g., Deault, 2010; Johnston & Mash, 2001; Theule et al., 2013). As would be expected, the specific nature of the interactions and stressors varies with development; however, the fundamental nature of the impairment is revealed in the fact that these disruptions in parent–child interactions

have been found across children, ranging in age from toddlerhood to adolescence (Johnston & Lee-Flynn, 2011). Variations in the interactions of parents with boys versus girls with ADHD, or in mother–child versus father–child relationships are not well understood because the majority of studies have focused on mothers and sons. However, a sufficient number of studies suggest possibly important differences related to both parent and child gender (e.g., Lifford, Harold, & Thapar, 2009; Peris & Hinshaw, 2003; Pfiffner, McBurnett, Rathouz, & Judice, 2005) that we highlight the need for further research in this area.

Although a developmental–transactional model emphasizes bidirectionality and reciprocity in parent–child interactions, this view must accommodate evidence of differential strength in parent-to-child versus child-to-parent effects, and of differential pathways of influence of the child's ADHD symptoms compared to other aspects of his or her functioning. That is, while some studies support specific links between early parenting and the development or severity of child ADHD symptoms (e.g., Ellis & Nigg, 2009; Harold et al., 2013; Hawes, Dadds, Frost, & Russell, 2013; Keown, 2012; Thorell, Rydell, & Bohlin, 2012), more studies point to effects of child ADHD on parenting. Early research manipulating the child's medication status (e.g., Barkley & Cunningham, 1979; Danforth, Barkley, & Stokes, 1991) converges with more recent, sophisticated longitudinal studies testing bidirectional influences (e.g., Burke, Pardini, & Loeber, 2008) to support child ADHD symptoms as primary drivers of parenting difficulties such as overreactivity or inconsistency. An illuminating demonstration of these effects is provided in experimental studies that indicate interaction with child actors displaying ADHD and disruptive symptoms elicits feelings of inadequacy, stress, depression, and hostility in parents, as well as corresponding increases in physiological stress measures (e.g., heart rate) and subsequent alcohol consumption (Pelham & Lang, 1999). Moving beyond simple unidirectional mechanisms of causality, evidence increasingly points to more complex, interactive patterns of influence between parenting and child ADHD symptoms. Specifically, ineffective parenting styles, in combination or interaction with the child's inattentive or impulsive nature, appear to set the stage for the development of child disruptive behaviors (i.e., oppositional or conduct problems) and perhaps emotional problems such as anxiety or depression (Harold et al., 2013; Johnston & Mash, 2001).

Most studies of parent–child interactions in families of children with ADHD are quite clear in showing that parenting and parent–child relationship problems are more closely linked to child disruptive behaviors rather than to ADHD symptoms (e.g., Burke et al., 2008; Johnston & Mash, 2001). This pattern is found not only in cross-sectional studies but also in more convincing longitudinal studies, in which initial levels of both child ADHD and disruptive behavior problems, as well as a variety of other family and parent risk factors, are controlled. Such longitudinal studies consistently demonstrate that parenting difficulties (e.g., low responsiveness, low positivity, overreactivity, inconsistency) are predictive of increases in or maintenance of disruptive behavior over time in children with ADHD (e.g., Biederman, Mick, Faraone, & Burback, 2001; Chronis et al., 2007; Lifford et al., 2008). Supporting a causal chain of events consistent with the transactional model, longitudinal studies, exemplified by that of Harvey, Metcalfe, Herbert, and Fanton (2011), indicate that parenting difficulties such as overreactive discipline play a mediating role between initial levels of child ADHD symptoms and subsequent disruptive behavior problems.

The pattern of results showing that disruptions in parenting, in combination with youth ADHD symptoms, are particularly strongly tied to comorbid disruptive behaviors has been demonstrated across the developmental spectrum from preschoolers (e.g., Cunningham & Boyle, 2002) to adolescents (Edwards, Barkley, Laneir, Fletcher, & Metevia, 2001; Gau & Chang, 2013). Parents' setting of firm, consistent, and reasonable limits on behavior and encouraging appropriate behavior are crucial in protecting against the development of disruptive problems in young children with ADHD (Harvey et al., 2011); recent studies of adolescents with ADHD show that similar parenting skills are relevant in preventing serious negative outcomes for these youth. For example, youth ADHD symptoms have been shown to interact with poor parental monitoring in predicting problems such as youth alcohol use or delinquency (e.g., Molina et al., 2012; Walther et al., 2012). Thus, although the family environment does not appear critical in the origin of child ADHD symptoms, family factors such as parent–child interactions do emerge as crucial in predicting the development or sparing of serious child outcomes such as conduct problems or substance abuse. The personal, familial, and societal costs of these negative consequences for children with ADHD are more than sufficient to motivate

careful and continued attention to the role of family context in this disorder.

Some of the most exciting recent work that focuses on parenting in families of children with ADHD incorporates potential genetic risks, such as variations on the *DAT1* or *DRD4* gene, and examines the interactions of these child genetic risks with parenting behaviors as determinants of both child ADHD symptoms and comorbid emotional or disruptive behaviors (e.g., Li & Lee, 2013; Martel et al., 2011; Martel, Nikolas, Jernigan, Friderici, & Nigg, 2012; Nikolas, Klump, & Burt, 2012; Sonuga-Barke et al., 2009). Consistent with the bidirectional and reciprocal relations expected by a transactional model, other work in this area has focused on how genetic risk for ADHD in parents interacts with child disruptive behavior problems to predict negative parenting (e.g., Lee et al., 2010). Although studies in this domain are still relatively rare and the results are not entirely consistent, they do point to an emergent understanding of how genetic and behavioral influences may combine in determining the developmental trajectories of children with ADHD, and they highlight potential differences in both parents' and children's genetic susceptibility to environmental influences, such as parenting styles or child problems (Belsky & Pluess, 2009).

Despite considerable current and past research in relation to parent–child interactions and child ADHD, much remains unknown. For example, there is a need for further specification of how individual parent or child characteristics, such as gender or developmental stage, as well as broader contextual factors, such as culture, may moderate the pattern of relations between parenting and child ADHD. Similarly, in contrast to the well-established associations of parenting and comorbid disruptive behavior problems, relatively little work has examined how parenting and parent–child interactions may be related to other common ADHD comorbidities such as anxiety or depression, academic and learning problems, or social problems with peers.

PARENTAL COGNITIONS

In understanding parent–child interactions in families of children with ADHD, research has focused on not only behavioral interactions between parents and child but also parents' cognitions about their children and about themselves as parents, and how these cognitions may shape the parent–child relationship (e.g., Hoza, Johnston, Pillow, & Ascough, 2006; Johnston & Ohan, 2005). This work is grounded in a cognitive-behavioral framework and posits that in order to understand fully how parents behave with their children we must examine parents' cognitions, such as expectations for child behavior, attributions regarding child intentionality, and attitudes toward childrearing that may underlie parental actions. Consistent with the patterns of association found among parenting behaviors, child ADHD, and comorbid disruptive problems, studies focused on parenting cognitions typically indicate that there are some differences between the cognitions of parents of children with ADHD and parents of typically developing children (e.g., Gerdes & Hoza, 2006; Johnston & Freeman, 1997). However, these differences may well be driven by child characteristics. In contrast, consistent and unique predictive linkages have been discovered between maladaptive parental cognitions, such as child-blaming attributions, and disruptive behavior problems in children with ADHD. For example, studies have shown that negative parental attributions for child behavior are uniquely predictive of greater child disruptive behavior over time, even accounting for initial levels of child ADHD, disruptive problems, and other family risk factors (e.g., Johnston, Hommersen, & Seipp, 2009; Williamson & Johnston, in press). It appears that child ADHD symptoms may set the stage for parents to adopt problematic cognitions about the child, and these parental cognitions then function to impair parenting and to fuel the development of more problematic child disruptive behavior.

In addition to attributions for child behavior, other research on parental cognitions has sought to understand how parents of children with ADHD think about their role as parents. Not surprisingly, an individual's sense of parenting efficacy seems to be negatively impacted by the experience of parenting a child with ADHD (Mash & Johnston, 1983) and may mediate between parental factors such as depression and parenting problems (Gerdes et al., 2007). Although a reduced sense of parenting efficacy seems to mark a reluctance to adopt effective parenting strategies (Johnston, Mah, & Regambal, 2010), parenting interventions, when used, serve to improve parents' sense of competence and also their satisfaction or enjoyment in the parenting role (Daley & O'Brien, 2013; Pisterman et al., 1992).

Cognitions, such as parents' sense of their own ability to parent a child with ADHD effectively or the causal explanations they develop for their children's

behavior, hold promise as potentially modifiable characteristics. These may be useful in promoting parents' ability to cope with and manage a child's ADHD symptoms in a way that disrupts the negative cycle leading from difficult child ADHD symptoms to less effective parenting choices, and ultimately to more serious child disruptive behavior problems. For example, a recent study by Chronis-Tuscano and colleagues (2013) illustrates the success of an intervention that targeted not only parenting skills but also depressive symptoms in mothers of children with ADHD, with a focus on strategies designed to address directly maladaptive cognitions about the child and one's ability as a parent, as well as the feelings and parenting behaviors associated with these thoughts. Other converging evidence supporting the importance and potential impact of parental cognitions comes from a study by Lench, Levine, and Whalen (2013), who demonstrated that in families of children diagnosed with ADHD, in which parents continued to see the child's characteristics in a positive light, there were fewer negative parent–child interactions, less parental frustration, and greater optimism regarding the child's future than in families in which parents did not hold these positive views of the child. Importantly, these positive differences persisted despite there being no difference between the two types of families in the levels of child ADHD or comorbid symptom severity.

We note one caveat relative to the previous comments about the importance of parental cognitions in families of children with ADHD. Although we believe that parents of children with ADHD would be well advised to avoid adopting child-blaming explanations that can lead to both parent and child hostility, and to increase their own sense of parenting efficacy, these more positive cognitions must be balanced with, and grounded in, reality. For example, adopting explanations for child behavior that overemphasize the child's ADHD status may lead to permissive or overprotective approaches to parenting that do not serve the child's best interests (Hinshaw, 2005). Instead, it may be most useful for parents to endorse attitudes that, while not blaming the child for ADHD behaviors, do, within reason, hold the child accountable for developing (with help and guidance from parents, teachers or other mentors) skills and strategies that will minimize the negative impact of the symptoms over time. The power of parenting attitudes that communicate this expectation of the child's ability to function independently was recently demonstrated in a study by Thomassin and Suveg (2012), who showed that high levels of parental support for the child's autonomy served as a buffer to reduce the association between child ADHD symptoms and poor task persistence. Similarly, just as feeling defeated may reduce parents' willingness to learn and try new parenting strategies, holding an extreme or illusory positive view of one's parenting ability may also serve as a barrier to developing and evolving as an effective parent. We believe that finding this balance between overly positive or negative versus realistic cognitions about the child and one's parenting ability represents one of the greatest challenges facing parents of children with ADHD. Interventions geared to assist parents in thinking critically, but optimistically, about both their own and their children's behavior (e.g., Chronis-Tuscano et al., 2013) hold promise as tools for reducing parental stress and frustration, and enhancing the positive reciprocity of parent–child interactions over time.

INDIVIDUAL PARENTING FUNCTIONING

ADHD

The strong heritability of ADHD symptoms implies that many parents of children with the disorder also are affected. Estimates indicate that over half of adults with ADHD have children with the disorder, and up to half of children with ADHD have a parent with high levels of ADHD symptoms (e.g., Biederman, Faraone, Mick, & Spencer, 1995; Chronis et al., 2003; Kessler et al., 2006). With the growing recognition of ADHD as a disorder common among multiple family members, accumulating studies indicate that ADHD symptoms in parents are associated with a range of both parent and child difficulties, including difficulties in parenting (Johnston et al., 2012), increased severity of child ADHD symptoms and disruptive behaviors (Agha, Zammit, Thapar, & Langley, 2013; Zisser & Eyberg, 2012), peer difficulties (Griggs & Mikami, 2011), and diminished response to treatment (Chronis-Tuscano et al., 2011; Sonuga-Barke, Daley, & Thompson, 2002). A major challenge in understanding the links between child outcomes and parental ADHD symptoms is parsing the direct and indirect contributions of both genes and behaviors to these relations. Although the links between parental ADHD and child problems may reflect the passive shared genetic associations of biological children and parents (e.g., Lifford, Harold, & Thapar, 2009), it is likely that the effects of parental ADHD on children also are mediated by the behav-

iors that transpire between parents and children (e.g., Herbert, Harvey, Lugo-Candelas, & Breaux, 2013), as well as more complex patterns of moderation of the child's genetic vulnerabilities by exposure to variations in parent–child interactions (e.g., Sonuga-Barke et al., 2009). Indeed, a recent report using data from two adoption-based studies demonstrated the importance of (biologically unrelated) mothers' hostility for the course of child ADHD symptoms, as well as the role of early child impulsivity as an evocative influence on the hostility of biologically unrelated mothers. This then predicted the course of child ADHD symptoms (Harold et al., 2013). Additionally, in this study, biological mothers' ADHD symptoms were significantly related to child impulsivity–activation; and adoptive mothers' ADHD symptoms were significantly correlated with their hostile parenting. This study was not only able to control for the effects shared genes may have on both child ADHD and parenting but it also examined the effects of child behavior on parenting in a genetically controlled fashion. Studies such as this provide very strong evidence for the reciprocal–transactional influence of child and parent on one another, and suggest convincingly that despite the strong influence of genes on ADHD behavior, parenting behavior also exerts an important influence on the course of child ADHD.

Whether measured dimensionally or categorically, higher levels of ADHD symptoms in parents consistently have been associated with increased parent–child conflict, and with elevations in difficulties in parental control, including harsh or overreactive discipline, lax or inconsistent discipline, poor monitoring of child behavior, chaotic homes with poor routines and structure, and harsh responses to children's expressions of emotions (Johnston et al., 2012; Mazursky-Horowitz et al., in press). These associations between ADHD symptoms in parents and problems with the negative or control aspects of parenting have been documented by both self-report and observational measures of parenting, and within samples of children as young as infants and as old as adolescents or young adults. In addition, the associations often appear even when researchers control for other aspects of parent and/or child psychopathology (e.g., parental depression, child disruptive behaviors).

In contrast to the consistent associations between parental ADHD and negative or control-related parenting behaviors, the associations between parental ADHD and positive, emotionally responsive aspects of parenting are somewhat less consistent (Johnston et al., 2012). Some studies indicate that ADHD symptoms are associated with reduced parental warmth/responsiveness (e.g., Chronis-Tuscano et al., 2008; Landau, Amiel-Laviad, Berger, Naama, & Auerbach, 2009; Semple, Mash, Ninowski, & Benzies, 2011), whereas others have not found these negative associations between parental ADHD and more positive parenting (e.g., Mokrova, O'Brien, Calkins & Keane, 2010; Murray & Johnston, 2006), or have found associations only for inattentive and not for hyperactive-impulsive symptoms (e.g., Chen & Johnston, 2007). This pattern of inconsistent findings alerts us to nuances in how parental ADHD symptoms are related to parenting, and suggest differences that may be tied to the dimension of ADHD symptoms measured and/or the aspects of parenting under consideration.

Psychogiou, Daley, Thompson, and Sonuga-Barke (2007, 2008) have posed the intriguing possibility that parental ADHD may interact with child ADHD to produce, in some instances, a buffering effect. In these studies, when mothers had higher levels of ADHD symptoms, child ADHD symptoms were associated with more positive, rather than more problematic, parenting, an effect that has been theorized to indicate increased empathy or tolerance for children with ADHD among parents who themselves experience the symptoms. However, these findings were unique to mothers in the Psychogiou and colleagues studies, and have not always been replicated in other samples (Johnston & Lee-Flynn, 2011). Other recent evidence (Lui, Johnston, Lee, & Lee-Flynn, 2013) suggests that the interactions may reflect a combination of actual increased positivity among parents with ADHD symptoms and potential overreporting of positive parenting by parents with high levels of ADHD symptoms, consistent with a general positive bias in the self-reports of individuals with ADHD (Owens, Goldfine, Evangelista, Hoza, & Kaiser, 2007).

In summary, parents with high levels of ADHD symptoms clearly struggle with maintaining a consistent, calm, and organized approach to managing child behavior. In contrast, the relations between parental ADHD symptoms and the positive aspects of parent–child relationships are less consistent. There is even the possibility that, under some circumstances, parental ADHD symptoms may contribute in a positive fashion to parent–child interactions. It is obvious that much further research with respect to adult ADHD and parenting is needed. The field awaits replications and extensions of existing findings before firm conclu-

sions can be drawn, and before the multiple pathways through which parental ADHD symptoms may exert their impact on child development are understood.

Depression

A wealth of evidence suggests that mothers of children with ADHD experience elevated levels of stress and depressive symptoms (Deault, 2010; Johnston & Mash, 2001). Approximately half of all mothers of children with ADHD have experienced at least one major depressive episode in their lifetime (Chronis et al., 2003). Importantly, a history of maternal depression uniquely predicts negative long-term outcomes for children with ADHD, including the development of later conduct problems, depression, and suicidal behavior (Chronis et al., 2007; Chronis-Tuscano et al., 2010), making maternal depression an important consideration for clinicians who treat children with ADHD.

Within our overarching model, it is important to acknowledge possible genetic associations between parental depression symptoms and child ADHD. For instance, depression and ADHD share some common genetic underpinnings given that the serotonergic and dopaminergic systems have been implicated in both disorders (Eley et al., 2004; Faraone & Mick, 2010; Hawi et al., 2002; Levinson, 2006; Li, Sham, Owen, & He, 2006; Nestler & Carlezon, 2006). However, it also is useful for clinicians to understand the link between ADHD and maternal depression within a developmental–transactional framework whereby parental depressive symptoms and child ADHD and disruptive behavior problems reciprocally influence one another at a behavioral level (e.g., Nicholson, Deboeck, Farris, Boker, & Borkowski, 2011).

In terms of child effects, parents of children with ADHD may experience depressive symptoms in part as a result of decreased environmental reinforcement associated with child ADHD and disruptive problems (Chronis-Tuscano & Clarke, 2008; Lewinsohn, Hoberman, Teri, & Hautzinger, 1985). As we have discussed, compared to parents of typically developing children, parents of children with ADHD often experience fewer positive interactions with their children, lower parenting efficacy, more parenting stress, and have fewer opportunities to engage in activities they enjoy due to the amount of time and effort they invest in parenting a child who requires high levels of supervision and assistance.

As one example, our ongoing work suggests that mothers of children with ADHD often feel the need to leave their jobs to be more available to attend school meetings and appointments during the day as well as to provide needed supervision and homework support for their children during after school hours. Such decisions may yield benefits for the child, but perhaps at the cost of leaving mothers more vulnerable to depression. Indeed, across several studies, employment has been shown to be a protective factor for women who have children with special needs (Lewis, Kagan, Heaton & Cranshaw, 1999) and may be a source of competency experiences, which can contribute to overall well-being (Lewinsohn, Hoberman, Teri, & Hautzinger, 1985). Specifically among parents of children with ADHD, mothers' working hours are related to less time devoted to child care, which is linked to more effective parenting and fewer conduct problems in the children (Harvey, 1998). Thus, due to the demands of parenting a child with ADHD, mothers may have fewer opportunities to participate in activities that are pleasurable or that contribute to feelings of competence in other domains and may, as a result, experience more stress and depression (Chronis-Tuscano & Clarke, 2008; O'Brien, Merson, Sauber, & Chronis-Tuscano, 2013).

Just as child ADHD may increase the risk of parental depression, a wealth of evidence from studies of families of children with ADHD and those of typically developing children demonstrates that depression can negatively impact the manner in which parents interact with their children (Johnston & Mash, 2001; Lovejoy, Graczyk, O'Hare, & Neuman, 2000; Wilson & Durbin, 2010). In particular, parents who are depressed may have difficulty noticing the child's efforts and positive behavior, have a low threshold for and react emotionally to child misbehavior, be disengaged and less interactive with the child, or be less motivated to participate in treatments that required effort and persistence. Within our transactional framework, these parenting behaviors can fuel future child disruptive behavior problems, which then contribute further to parental stress and depressive symptoms in a cyclical manner. Parental depression also can contribute to interparental conflict, which may exacerbate inconsistent parenting and poor child adjustment.

Perhaps because of the gender divergence in the prevalence of depression, with women having significantly higher rates than men, much less is known about depression that may occur in fathers of children with ADHD. However, recent studies are beginning to shed light on the relationship between paternal depression and child ADHD symptoms. The few studies that have examined the effects of paternal depression on offspring

outcomes have found links to both child ADHD symptoms and externalizing (i.e., disruptive behavior) and internalizing symptoms more broadly (Kane & Garber, 2004). For example, in a sample of 3-year-old children, paternal depression was related to increased child hyperactivity, even after researchers controlled for the effects of maternal depression (Ramchandani, Stein, Evans, & O'Connor, 2005). Similar links are found in school-age samples (Harvey et al., 2011; Ramchandani et al., 2008). Importantly, a recent study revealed that paternal (but not maternal) depression predicted the persistence of child ADHD into adulthood, highlighting the need for further research and clinical attention regarding depression in fathers (Lara et al., 2009). Finally, a recent meta-analysis focusing on depression in fathers reported small, but significant, effect sizes of paternal depression on both positive and negative parenting (Wilson & Durbin, 2010). Interestingly, these effect sizes mirrored those found in studies that examined parenting in depressed mothers. Thus, although studies are beginning to show that paternal depression may have important and unique effects on child adjustment, the pathways through which maternal and paternal depression might differentially be linked to various child outcomes, such as the persistence of ADHD and development of comorbid conditions, warrant further investigation.

Antisocial Behavior

Some parents of children with ADHD (approximately 3–13%; Chronis et al., 2003) have a history of or current antisocial behavior and criminal involvement, although, in contrast to the research on depression, which has included predominantly mothers, here the focus has been mostly on fathers. There is substantial evidence of links between parental antisocial behavior and both child ADHD and disruptive behavior problems (Chronis et al., 2003). A recent study identified parental antisocial behavior as a key predictor of the persistence of child ADHD into adulthood, highlighting its importance as a prognostic indicator (Lara et al., 2009).

Research has begun to clarify the extent to which the associations between parent antisocial behavior and child ADHD symptoms can better be attributed to genetic and/or environmental factors. Child ADHD symptoms have been related to a history of arrests and/or alcohol use disorders in biological, rather than adoptive, parents—suggestive of a genetic link (Beaver, Nedelec, Rowland & Schwartz, 2012) and consistent with a general genetic liability from parent antisocial behavior to both child ADHD and disruptive behavior disorders (e.g., Bornovalova, Hicks, Iacono & McGue, 2010). At the same time, genetically informed studies have revealed the strongest evidence for disorder-specific risk transmission for psychopathology from parent to child (e.g., Clark, Cornelius, Wood & Vanyukov, 2004). In other words, when multiple forms of parental psychopathology are considered, child ADHD seems to be best predicted by ADHD in either parent, while, in contrast, it is child disruptive behavior (particularly CD) that seems to be most closely related to parental antisocial behavior.

In addition, the genetic contribution to ADHD appears to be much stronger than the genetic contribution to antisocial/disruptive behavior problems. For instance, in a recent study, whereas both genetic and environmental factors accounted for the resemblance between parental antisocial behavior and child disruptive behavior problems, the resemblance between parental antisocial behavior and child ADHD was attributed entirely to genetic factors (Silberg, Maes & Eaves, 2012). Together, these studies point to the likelihood of the genetic associations between parental antisocial behavior and child ADHD being mostly caused by the co-occurrence of child ADHD and disruptive disorders, and the co-occurrence of parent antisocial behavior and ADHD.

Antisocial behavior in parents constitutes a risk for not only maladaptive parenting but also many other environmental disadvantages, such as poverty, interparental conflict, or the child's exposure to violence. Consistent with the central role of parent–child interactions as contributors to the functioning of children with ADHD, at least some studies have found evidence of the mediating role of parental negativity and/or poor supervision in the pathway from parental antisocial behavior to the development of conduct problems in children with ADHD (e.g., Conger, Neppl, Kim, & Scaramella, 2003; Pfiffner et al., 2005). In summary, in line with a developmental–transactional model, both direct and indirect pathways tie antisocial behavior in a parent to maladaptive family interactions, poor child outcomes, and disturbances in the other parent's functioning or the interparental dyad.

Alcohol/Substance Abuse

As with research on antisocial behavior, studies have documented alcohol- and drug-related problems in parents of children with ADHD, as well as an increased

risk for ADHD and disruptive behavior problems in children of fathers with substance use disorders (many of whom had ADHD and CD as children; Chronis et al., 2003; Clark et al., 2004). Risk for child behavioral problems is greatest when both parents have substance abuse problems (Osborne & Berger, 2009). Also in line with the links between child ADHD and other forms of parental psychopathology, genetic transmission forms at least part of the explanation for this association. For example, Knopik and colleagues (2006) found support for a genetic link between maternal alcohol use disorders and offspring risk for ADHD. However, as we discussed previously, consistent with disorder-specific risk transmission, studies that have assessed a broad range of parental psychopathology have found that child ADHD is best predicted by ADHD in the mother and/or father, more so than parental alcohol/substance use (e.g., Clark et al., 2004).

Viewed within a transactional framework, substance use in the parent may be seen as both cause and effect of child ADHD/disruptive behavior problems. It likely interacts with other family variables, such as parenting or other forms of parental psychopathology, in complex ways. As one illustration of the possible directions of effect between child ADHD and parental substance abuse, we consider a series of experimental studies by Lang, Pelham, and colleagues (e.g., Pelham & Lang, 1999). These studies have clearly documented "child effects" on the amount that adults drink following difficult interactions with deviant children. In the first of these studies, college student participants interacted with child confederates who were well behaved or who displayed behaviors characteristic of a child with ADHD and disruptive behavior problems. Interestingly, even though interacting with the deviant child confederate produced distress in both males and females, only male students drank more following these interactions (Lang, Pelham, Johnston & Gelernter, 1989). However, in later studies with parents, they found that not all parents drink more following difficult interactions with children showing symptoms of ADHD and/or disruptive behavior problems. The effects of stressful parent–child interactions on alcohol consumption seem to be most pronounced in parents with a family history of alcoholism (Pelham et al., 1998) and/or parents with higher levels of anxiety and depressive symptoms (Kashdan, Adams, Kleiman, Pelham, & Lang, 2013). In summary, these experimental studies suggest that, while not all parents of children with ADHD drink more in response to stressful interactions with their children, some are at risk for these child-induced effects. It is therefore important for clinicians to assess parents' level of stress reactivity and tendency to engage in stress-induced drinking.

Turning to the effects of parental substance use on parenting, this same series of experimental studies also examined the effects of drinking on parents' perceptions of their children and parenting behaviors while interacting with the child confederates. Intoxicated parents perceived fewer behavioral problems in deviant confederates, although alcohol consumption did not affect ratings of the pleasantness of the interaction or the effects of the interaction on parent mood and stress levels (Lang, Pelham, Atkeson & Murphy, 1999). Importantly, however, intoxicated parents were observed to be more permissive, off-task, and directive, and less attentive in interactions with child confederates (Lang et al., 1999)—behaviors suggesting that alcohol impaired their ability to provide proper support, supervision, and feedback to the child.

Similarly, although rarely studied in relation to child ADHD, the broader literature suggests that illicit drug use in parents is associated with observed ineffective parenting, which in turn is associated with child disruptive behavior problems (Bailey et al., 2013). Moreover, parental neglect (Dunn et al., 2002) and punitiveness (Miller, Smyth, & Mudar, 1999) are more often seen in drug- and alcohol-abusing parents than in other parents. The possibility that children with ADHD may be particularly susceptible to the negative effects of less effective parenting within families of alcohol/drug-abusing parents highlights the need for further study in this area.

In summary, the extant literature demonstrates effects of child ADHD and disruptive behavior on parental alcohol consumption, as well as effects of alcohol/substance use on parents' perceptions of child behavior and observed parenting behavior. These processes are likely to be most pronounced in families with a history of alcohol/substance abuse and dependence, and among parents who experience depression or anxiety in addition to stress-induced drinking.

Interparental Conflict and Divorce

The clinical presentation of children with ADHD is often compounded by a family history of marital instability, exposure to interparental conflict, and divorce. Parents of children with ADHD are at least three times more likely to be separated or divorced compared to

parents of children without ADHD (Barkley, Fischer, Edelbrock, & Smallish, 1991). Not only are parents of children with ADHD more likely to divorce, but they also have a shorter latency to divorce compared to parents of children without the disorder (Wymbs et al., 2008b). Strikingly, results of a Danish study suggested that 10 years after birth of a child with ADHD, parents had a 75% greater probability of ending their relationship than did parents of a child without ADHD (Kvist, Nielsen, & Simonsen, 2013).

Risk for interparental conflict and divorce in families of children with ADHD may be exacerbated in part by parents' own mental health problems, such as those considered in previous sections (e.g., antisocial behavior in fathers; Wymbs et al., 2008b). For instance, adults with ADHD are more than 5 times more likely to be divorced currently and 15 times more likely to have ever been divorced than adults in the general population (Klein et al., 2012). At the same time, recent twin studies have suggested little role for parental genetic factors as an explanation for the higher divorce rate in parents of children with ADHD (Schermerhorn et al., 2012). In a study of genetically identical adult twins, in which one of the adults was parent to a child with ADHD and the other was not, ADHD in the child predicted parental divorce and interparental conflict. This association held when researchers controlled for genetic risk, parent psychopathology, timing of parental divorce, and forms of offspring psychopathology other than ADHD. Together, these studies provide convincing evidence for "child effects" on interparental conflict and divorce.

Whether parents of children with ADHD are married, separated, or divorced, they often experience disagreements regarding the severity of the child's symptoms, the extent to which the ADHD symptoms impair the child's functioning, and what treatment approach, if any, to take (Stein, Diller, & Resnikoff, 2001). Parents of children with ADHD also are more likely to disagree about how to manage the child's difficult behavior (Johnston & Behrenz, 1993). In particular, disagreements related to the child, relative to other sources of interparental conflict, have been linked to child behavior problems (Nikolas et al., 2012). Furthermore, these childrearing disagreements are likely to be reciprocally linked to not only interparent inconsistencies in responses to the child but also parental distress and marital discord.

As with parent–child interactions, there is growing evidence that coexistence of child and parent disruptive or antisocial behavior can exacerbate conflict between parents of children with ADHD. Within an ADHD sample, paternal antisocial behavior and the severity of child disruptive behavior symptoms predicted the timing of divorce (Wymbs et al., 2008b). In another study, adolescents with ADHD and comorbid CD reported witnessing more frequent and unresolved interparental conflict compared to adolescents without ADHD, adolescents with ADHD only, or adolescents with ADHD and oppositional defiant disorder (Wymbs, Pelham, Molina, & Gnagy, 2008a). Providing the strongest evidence of child effects are experimental studies indicating that both during and after interacting with a child confederate who is behaving like a child with ADHD, parents communicated more negatively with one another (Wymbs & Pelham, 2010). And these child effects were stronger for parents of children with comorbid disruptive behavior problems. Moreover, negative parenting partially mediated the causal pathway between disruptive child behavior and negative communication between parents (Wymbs, 2011). In summary, these studies suggest that child disruptive behavior contributes to both negative parenting and increased interparental conflict, in line with our transactional model. Thus, as with other aspects of family functioning, we see that co-occurring disruptive behavior problems compound the associations between child ADHD and interparental conflict and divorce.

Turning to the effects of interparental discord on children with ADHD, we know from years of developmental research that interparental discord, particularly when it occurs in the child's presence, can contribute to poor child outcomes. Confirming these effects in the context of child ADHD, the child's perceptions of interparental conflict (self-blame in particular) are associated with parent and teacher ratings of the child's inattention and hyperactivity– impulsivity, even after researchers control for a number of other risk factors (Counts, Nigg, Stawicki, Rappley & Von Eye, 2005). Importantly, developmental research in this area demonstrates that the child's perspective is critical because the child's perception of interparental conflict is more predictive of child behavior problems than the parents' reports of conflict (Grych & Fincham, 1990). Clinicians are thus advised to assess inter-parent discord using both parent and child reports in the formulation.

Providing further evidence for reciprocal effects among child ADHD, disruptive behavior problems, and interparental conflict, prospective longitudinal research indicates that over a 1-year period, marital discord predicts child emotional and behavioral dysregu-

lation; at the same time, child behavioral dysregulation is linked to subsequent higher interparental conflict (Schermerhorn, Cummings, DeCarlo, & Davies, 2007). Not only is there evidence for reciprocal interparental and child effects, but recent work also has focused on how interparental discord may interact with child genetic risk in determining outcomes for children with ADHD. For example, in recent studies by Nikolas and colleagues (2012, 2013), the child's tendency to blame him- or herself for interparental conflict moderated genetic and environmental (i.e., interparental conflict) influences on child ADHD.

Together, work in understanding interparental discord in families of children with ADHD fits within a pattern of bidirectional and reciprocal influences operating between the child and parents. In other words, child ADHD and disruptive behavior problems both contribute to, and are influenced by, interparental discord, often via inconsistencies in parenting behavior and disruptions in the parent–child relationship. In addition, parental psychopathology and other family stressors contribute to these interparental processes to produce a cumulative picture of influences on the child's developmental trajectory.

SIBLING RELATIONSHIPS AND ADHD

Sibling relationships play a critical role in the family (Feinberg, Solmeyer, & McHale, 2012). Interactions with brothers and sisters are often proposed as the first testing grounds for children's development of important social competencies (Brody, 2004), and children's perceptions of differential parental treatment of siblings are strong predictors of adjustment outcomes (Buist, Deković, & Prinzie, 2013). The strong heritability of ADHD combined with shared environmental influences (e.g., Hicks, Foster, Iacono, & McGue, 2013), place siblings of children with ADHD at elevated risk for both ADHD and a range of other behavioral and emotional problems (e.g., Listug-Lunde, Zevenbergen, & Petros, 2008; Steinhausen et al., 2012). This collection of facts—including the potential of siblings to steer children toward more or less socially skilled developmental trajectories, the elevated likelihood of problems among siblings of children with ADHD, and the higher likelihood of differential allocation of parental resources or affection when one sibling has ADHD—might lead one to expect that sibling relationships of children with ADHD have been the target of considerable research. However, this is not the case. What little research there has been confirms increased conflict in the sibling relationships of children with ADHD, particularly if there are comorbid disruptive behavior problems along with ADHD (e.g., Mikami & Pfiffner, 2008). Much less is known about the mechanisms underlying this conflict or its implications for children's outcomes.

Future research focused on sibling relationships of individuals with ADHD, across the lifespan, may provide insight into growth-promoting or detrimental factors in these relationships. It is possible that poor sibling relationships may deprive the child with ADHD of the chance to develop even rudimentary social skills, thus setting the stage for increased interpersonal problems as the child enters the world outside of the family. At the same time, a supportive and accepting sibling relationship may provide a crucial buffer or place of safety throughout the life of the individual with ADHD. Research in this area is challenged by the need to disentangle genetic and environmental influences on sibling relationships, to account for the complexity in sibling relationships due to birth order and gender variations, and to examine these relationships as they develop and exist in parallel to the individual developmental pathways and parent–child relationships of each of the siblings.

CONCLUSIONS

As the research reviewed in this chapter illustrates, our understanding of the role of families in child ADHD has evolved considerably over the past 35 years. From early incorrect and harmful views of bad parenting as the cause of child ADHD through a period of research in which family contributions were limited to genetic influences on the neurobiology of ADHD, we have emerged to a place where the role of family in child ADHD can be seen as more complex and nuanced, with multiple reciprocal and dynamic interactions among child and parent genetic vulnerabilities, parenting and family context, and extrafamilial influences. Although we have considered different aspects of family functioning in separate sections of this chapter, we hasten in this final section to point out the many interrelationships and shared influences among these factors. As outlined in the transactional model, each aspect of child, parent, and family functioning is by definition part of a network of mutual and associated influences. And these multiple linkages mean that the presence

of risk factors in one area (e.g., parental depression) heralds elevated risks in other areas (e.g., interparental discord) as well. On the other hand, strengths within the child or the family, such as positive interactions or effective monitoring, may exert a protective influence.

We conclude with thoughts regarding how clinicians can best utilize the information presented in this chapter. The evidence points clearly to the necessity of careful and full consideration of family characteristics (including both risk and protective factors) when evaluating and treating children with ADHD. On the one hand, clinicians must appreciate the multiple difficulties that may characterize families of children with ADHD, and the unfortunate potential of these parental and family challenges to place children with ADHD on a pathway to the development of more serious disruptive behavioral and emotional problems.

On the other hand, to the extent that these specific risk factors are modifiable, we believe that there are substantial opportunities for clinicians to work collaboratively with families to alter the child's environment in a way that minimizes the negative impact of the ADHD symptoms on the family, prevents the development of more serious problems, and functions to optimize the functioning of all family members. Thus, the interrelated nature of child and parent functioning in families of children with ADHD presents both a challenge and an opportunity. While there is the risk of multiplying and escalating problems, the mutual and reciprocal influences also mean that positive changes in any aspect of child, parent, or family functioning have the potential to contribute positively to development in other domains.

KEY CLINICAL POINTS

- ✓ Parent–child interactions form the core of a set of larger family and social experiences that impact the development of children with ADHD.
- ✓ Child ADHD symptoms contribute to parenting difficulties, and child ADHD and parenting problems together contribute to the development of more serious disruptive behavior problems.
- ✓ Parents who have a low sense of parenting efficacy or who endorse child-blaming explanations for child misbehavior are at greater risk for ineffective parenting, and their children are at increased risk for the development of disruptive behavior problems.
- ✓ Clinicians should be aware of the potential of interventions aimed at parenting behavior and cognitions because these provide avenues to the prevention of serious disruptive behavior problems in children with ADHD.
- ✓ ADHD in both parents and children within a family is relatively common.
- ✓ Parental ADHD symptoms are predictive of difficulties with providing calm and consistent discipline, consistent structure/organization, and supervision. Parental ADHD symptoms are less consistently associated with problems with positive parenting or responsiveness.
- ✓ Clinicians should always consider the possibility of parental ADHD when children are referred with the disorder, and assess the extent to which parental ADHD symptoms may be impacting parenting or treatment progress.
- ✓ Approximately half of all mothers of children with ADHD have experienced clinical depression, and even more experienced heightened parenting stress and subclinical levels of depressive symptoms. Depression in fathers also is associated with child ADHD and disruptive behavior problems.
- ✓ Maternal depression is associated with the later development of depression and suicidal behavior, as well as the maintenance or exacerbation of conduct problems, in children with ADHD.
- ✓ Consistent with a transactional framework, exposure to difficult child behavior may contribute to the development of depression in parents. Likewise, parental depression can impact the quality of parenting and contribute to interparental conflict—both of which influence child adjustment.
- ✓ Clinicians should be keenly aware of parent stress and depression in families of children with ADHD, particularly as these factors relate to parenting and treatment participation/outcome.
- ✓ Antisocial behavior is present in some parents of children with ADHD. Antisocial behavior in parents is more closely associated with child disruptive behaviors (particularly CD) than with ADHD per se, and is often comorbid with adult ADHD.
- ✓ Parent antisocial behavior can have direct and indirect influences on the child via parenting, interparental conflict, exposure to violence, and other environmental risks (e.g., poverty).

- ✓ In families of children with ADHD, alcohol/substance use problems may be present in one or both parents.
- ✓ A subset of parents with anxiety and depressive symptoms may respond to challenging interactions with their children by engaging in stress-induced drinking.
- ✓ Given that alcohol consumption and drug use can influence parents' perceptions of their children and their behavior during parent–child interactions, clinicians should be aware of a family history of alcohol and drug problems, as well as the parent's tendency to engage in stress-induced drinking.
- ✓ Parents of children with ADHD are more likely to experience interparental conflict and divorce. Child disruptive behavior problems and parental psychopathology exacerbate risk for interparental conflict and divorce.
- ✓ There is evidence of "child effects" on interparental conflict. At the same time, exposure to interparental discord has a negative impact on child adjustment.
- ✓ Children's perceptions of interparental conflict, and their tendency to blame themselves for this conflict, are important for clinicians to consider because both predict poor child adjustment.
- ✓ Clinicians should strive to help parents to work collaboratively to provide consistent structure and discipline, while minimizing the extent to which the child is exposed to interparental conflict.
- ✓ Siblings of children with ADHD are at elevated risk for both ADHD and other problems.
- ✓ Little research has addressed sibling relationships, but clinicians may find that sibling relationships have the potential either to exacerbate the difficulties of children with ADHD or to provide helpful support throughout development.

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CHAPTER 8

Peer Relationships of Children with ADHD

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A substantial portion of children with attention-deficit/hyperactivity disorder (ADHD) demonstrate significant struggles in their social relationships and behavior. In fact, revisions to the description of ADHD in DSM-5 now include social activities as a domain of functioning that can be negatively affected by the symptoms of inattention, hyperactivity, or impulsivity (American Psychiatric Association, 2013). It is not hard to see how these symptoms may interfere with social interactions. Interrupting, talking excessively, noisiness, and difficulty waiting for their turn are likely to make children with ADHD appear intrusive and annoying to peers during play activities (Pelham & Bender, 1982). Similarly, distraction and failure to listen are likely to interfere with the ability to attend to peers effectively, and to notice and respond to social cues (Cadesky, Mota, & Schachar, 2000). Effective assessment and treatment of youth with ADHD must involve consideration of the child's social functioning not only within the broader peer group but also in terms of their dyadic friendships, social behaviors, and social-cognitive skills. In addition, unique challenges emerge during adolescence, when peer interactions can involve risky behaviors (e.g., substance use, sexual behavior) and may also take electronic and online forms (e.g., texting, social media). In this chapter we review what is currently known about the social impairments of children with ADHD across these contexts. When available, evidence of differences in presentation across genders and DSM-5 presentations of ADHD (formally referred to as "subtypes" in DSM-IV) is noted.

PEER RELATIONSHIPS

Peer Acceptance and Rejection

When peers are asked to nominate the classmates they like and dislike as friends, children with ADHD are likely to be nominated by the majority of their classmates as being disliked and by few as being liked (Gresham, MacMillian, Bocian, Ward, & Forness, 1998). In fact, in a review, Hoza (2007) estimated that between 50 and 80% of children with ADHD could be classified as rejected by their broader peer group. Children with all three presentations of ADHD (combined, predominantly inattentive, and predominantly hyperactive/impulsive) were at increased risk for peer rejection, though risk may have been highest among those with the combined presentation of ADHD (Gaub & Carlson, 1997). Children with ADHD and comorbid oppositional defiant disorder (ODD) or conduct disorder (CD) also have higher rates of peer rejection than

children with ADHD alone (Mrug et al., 2009). However, there did not appear to be gender differences in rates of peer rejection and lack of acceptance in youth with ADHD (Hoza, Mrug, et al., 2005). In general, children with ADHD reported liking their peers more than their peers reported liking them (Hoza, Mrug, et al., 2005; Mrug et al., 2009); this suggests that although children with ADHD may see many children within their peer group as desirable friends, this viewpoint is unlikely to be reciprocated.

Of significant concern were findings that peer rejection develops rapidly and is resistant to change (Hoza, 2007). For instance, as early as the first day of a summer academic program, campers rated children with ADHD as less desirable as friends than control children (Erhardt & Hinshaw, 1994). Pelham and Bender (1982) observed nonhyperactive peers' responses to hyperactive children when placed in a playgroup together for 90 minutes. They found that the nonhyperactive peers often began complaining about the hyperactive child's behavior within minutes. In addition, when asked how much they liked playing with the hyperactive child at the end of the play session, nonhyperactive peers reported their extreme dislike of the hyperactive children (Pelham & Bender, 1982). Thus, it appears that, almost immediately, peers find the social interaction style of children with ADHD aversive. What is more, these results suggest that children with ADHD may quickly re-create their rejected status when entering new peer groups.

Other evidence suggests that negative peer regard is not easily reversed, even with intensive evidence-based treatments that reduce the symptoms of ADHD (Hoza, Gerdes, et al., 2005). This may in part be due to the intractable nature of children's social reputations and peers' expectations of children with ADHD (Hoza, 2007). For example Harris, Milich, Corbitt, Hoover, and Brady (1992) found that if children expected to interact with a child who displayed symptoms of ADHD, then they were less friendly toward that child and more likely to describe that child's behavior as being hyperactive than if they had no expectations about the child. Most importantly, these differences were found regardless of whether the child did indeed have ADHD. This suggests that even if negative social behaviors improve, peers may continue to view the child with ADHD in a negative light. Hoza (2007) highlighted these findings and argued that in order to address the peer rejection of children with ADHD, their social reputation within their broader peer group must be changed.

Rejection by the broader peer group also appears to be a marker of risk for both internalizing and externalizing problems in children with ADHD. Among males and females with and without ADHD, childhood peer rejection is found to predict increased risk for adolescent substance use, delinquency, anxiety, and global impairments over and above the risk associated with an ADHD diagnosis (Mrug et al., 2012). Results from a female-only sample suggested that childhood peer rejection may act as an additive risk factor above and beyond the risk incurred by a diagnosis of ADHD. Specifically, peer rejection predicted increased risk for adolescent externalizing problems, internalizing problems, eating pathology, and poor academic achievement (Mikami & Hinshaw, 2006). Studies that used nonclinical samples provide insight into how peer rejection may increase risk for later maladjustment. Specifically, children who are rejected by peers are likely to be socially isolated, which may limit social support and subsequently increase risk for internalizing problems such as depression (Bell-Dolan, Reaven, & Peterson, 1993). Children who are rejected by peers are also more likely to turn to other rejected peers as friends; such peers may be more likely to model and encourage delinquent behaviors, thus increasing risk for externalizing problems (Berndt, 1999). Indeed one study revealed that among a broad range of risk factors, peer rejection was the strongest predictor of later psychological maladjustment in a nonclinical sample of children (Cowen, Pederson, Babigian, Izzo, & Trost, 1973).

Dyadic Friendships

In addition to rejection by the broader peer group, children with ADHD also are less likely to have reciprocated friendships (i.e., when two children mutually nominate one another as a friend). For instance, using a subsample of children from the Multimodal Treatment Study of Children with ADHD (MTA; MTA Cooperative Group, 1999), Hoza, Mrug, and colleagues (2005) found that 56% of children with ADHD had no reciprocated friendships, 33% had one reciprocated friendship, and only 9% had two reciprocated friendships. In contrast, 32% of comparison children had no reciprocated friendships, 39% had one reciprocated friendship, and 22% had two. Other evidence suggests that a lack of any dyadic friendships may be even greater among children with clinically elevated symptoms of inattention and hyperactivity that co-occur with conduct problems. Gresham and colleagues (1998) found that

more than 70% of children with this symptom profile had no reciprocated friendships as compared to less than 25% of control children. Though evidence is limited, it appears that males and females with ADHD are equally likely to lack dyadic friendships (Hoza, Mrug, et al., 2005).

Children with ADHD also appear to differ in whom they befriend. Although children with ADHD and comparison children are equally likely to identify socially accepted children as desired friends, these socially accepted children are less likely to name a child with ADHD as someone they want as a friend (Hoza, Mrug, et al., 2005). This may deny children with ADHD the opportunity to interact and learn from more socially skilled children, and limit the options of whom they can befriend. Research examining the characteristics of the mutual friends of children with ADHD revealed that their friends are more likely also to demonstrate symptoms of ADHD, noncompliance, and deviance (Bagwell, Molina, Pelham, & Hoza, 2001; Blachman & Hinshaw 2002; Normand et al., 2011; Whalen & Henker, 1985). It may be that peers with similar behavioral problems are more willing to befriend children with ADHD or, as Normand and colleagues (2011) suggested, children with ADHD are specifically attracted to peers with similar social goals (e.g., sensation seeking, deviance).

Despite the likelihood that children with ADHD seek out friends whose symptom profiles are similar to theirs, this does not translate into better friendship quality. In fact, when Normand and colleagues (2011) examined the friendships of children with and without ADHD, they found that both children with ADHD and their identified friend mutually perceived less positive features (e.g., validation, companionship, help, intimacy, and conflict resolution) and were less satisfied in their dyadic friendship relative to comparison children and their friend. Children with ADHD also perceived more negative features (e.g., aggression, exclusivity, and conflict) in these friendships, though this perception was not shared by the friend. During social interaction tasks with their identified friend, children with ADHD also were more likely to violate rules when the task was competitive and were more insensitive, self-centered, and dominant in their negotiation style when the task involved sharing (Normand et al., 2011). Thus, it may be the behavior of the child with ADHD, rather than the friend's behavior, that increases negativity within the friendship. Other evidence suggests that the friendships of girls with ADHD may specifically be characterized by higher levels of relational aggression (e.g., gossiping, spreading rumors, and intentional exclusion) both within the friendship and directed toward others (Blachman & Hinshaw, 2002).

Given the higher incidence of negative behaviors within the friendships of children with ADHD, it may not be surprising that these children also have less stable friendships over time (Blachman & Hinshaw, 2002; Normand et al., 2013). Normand and colleagues found that at 6-month follow-up, 25% of children with ADHD were no longer mutually friends with their friend from 6 months earlier. In contrast, only 9% of control children lost their mutual friendship. They also found evidence to suggest that children with ADHD may be more likely to lose friends because their friends' dissatisfaction with the friendship increases over time. At 6-month follow-up, the friends of children with ADHD were more dissatisfied and perceived less positive features in the friendship than they did at baseline. Importantly, children with ADHD did not perceive these same declines in their friendship quality, suggesting that they may fail to notice problems within the friendship as they emerge.

Preserving friendships may be especially important for children with ADHD. Evidence suggests that the presence of a best friend is protective against more global peer rejection (Blachman & Hinshaw, 2002) and a greater number of friendships is protective against peer victimization (Cardoos & Hinshaw, 2011). Greater intimacy with a best friend (e.g., self-disclosure of secrets and problems) also may buffer risk for increased social problems over time in children with elevated ADHD symptoms (Becker, Fite, Luebbe, Stoppelbein, & Greening, 2013). Despite the more short-term benefits of dyadic friendships, long-term studies suggest that lack of a best friend did not predict behavioral and emotional maladjustment or global impairment 6 to 8 years later, although greater peer rejection did (Mrug et al., 2012). Even if dyadic friendships do not predict long-term outcomes, Mikami (2010) argued that it may be more effective to focus on cultivating or improving specific friendships for children with ADHD rather than attempting to shift the perception of an entire peer group.

Bullying and Victimization

The peer relationships of children with ADHD also may be characterized by higher incidences of both vic-

timization and bullying. Evidence suggests that children with ADHD are more likely to be victimized by peers in verbal (e.g., teasing), physical (e.g., pushing or shoving), and relational (e.g., damaging social relationships) ways relative to comparison children (Wiener & Mak, 2009). For instance among 9- to 14-year-old children, 58% of those with ADHD were victimized several times a week by peers, compared to 14% of comparison children (Wiener & Mak, 2009). Not only are children with ADHD more likely to be victimized, they also are at risk for engaging in bullying behaviors themselves. For instance, Unnever and Cornell (2003) found that middle school students with ADHD were more likely to be victimized two to three times a month relative to comparison children (34 vs. 22%), but they were also more likely to bully peers (13 vs. 8%). Some evidence suggests that rather than ADHD symptoms, it is the presence of comorbid ODD that predicts engagement in bullying (Wiener & Mak, 2009). The experience of victimization or engagement in bullying also has been found to increase risk for social and emotional maladjustment in children with ADHD. For instance, Taylor, Saylor, Twyman, and Macias (2010) found that relative to youth with ADHD who were not victimized, those who were victimized had greater co-occurring parent-rated internalizing and externalizing problems and more self-reported depression symptoms. In addition, youth with ADHD who were victimized and also engaged in bullying were found to have the highest levels of internalizing problems and depression symptoms within the ADHD sample.

SOCIAL BEHAVIORS

Peer difficulties among children with ADHD may partly stem from their intrusive, annoying, and disruptive social interaction style (Pelham & Bender, 1982; Whalen & Henker, 1992). For example, in small playgroups, children with ADHD were found to display high frequencies of negative verbalizations (e.g., teasing, name calling), negative nonverbal behaviors (e.g., hitting, noncompliance, intruding on others' activities), and loud or disruptive behaviors (e.g., running around, loud or repeated yelling; Pelham & Bender, 1982). During play and structured classroom activities, children with ADHD were rated as more noncompliant, disruptive, and aggressive than control children (Erhardt & Hinshaw, 1994). Also, during a social leadership task with younger unknown peers, children with ADHD displayed more hostile affect, disruptiveness, criticism, and overinvolvement than did comparison children (Buhrmester, Whalen, Henker, MacDonald, & Hinshaw, 1992). Peers may be especially intolerant of the disruptive and noncompliant behavior that children with ADHD tend to exhibit (Erhardt & Hinshaw, 1994; Mrug, Hoza, Pelham, Gnagy, & Greiner, 2007). Mrug and colleagues (2007) found that in a summer camp program, rule breaking, failure to pay attention during activities, and complaints were the strongest predictors of peer rejection in children with and without ADHD.

Some children with ADHD may also engage in aggressive behaviors. Risk for aggression appeared greatest among children with the combined presentation of ADHD (Hodgens, Cole, & Boldizar, 2000; Maedgen & Carlson, 2000) and children with comorbid ODD or CD symptoms (Abikoff et al., 2002; Ohan & Johnston, 2007). The type of aggressive behaviors displayed may also differ between genders. Relative to comparison children of the same gender, males with ADHD were more likely to engage in both physical and verbal aggression, whereas females with ADHD were more likely to engage only in verbal aggression (Abikoff et al., 2002). In female samples, it has been found that girls with ADHD are at higher risk for engaging in both overt and relational forms of aggression relative to girls without ADHD (Zalecki & Hinshaw, 2004). Relational aggression appears to be highest among girls with ADHD and comorbid ODD, though girls with ADHD alone still demonstrate higher rates than controls (Ohan & Johnston, 2007). The quality of relationally aggressive behaviors also may differ in girls with and without ADHD. Ohan and Johnston used a simulated chat room task to examine relationally aggressive behaviors in girls with and without ADHD. Results indicated that girls with ADHD were more likely to use overt and less skillful forms of relational aggression (e.g., stating in posted messages that they were ignoring or did not like another player). In contrast, control girls used more planned and covert forms of relational aggression (e.g., divulging one player's secrets to strengthen their relationship with a different player; Ohan & Johnston, 2007). Covert and skillful forms of relational aggression have been found to predict greater popularity and social power in nonclinical samples (Heilbron & Prinstein, 2008); however, girls with ADHD may be unlikely to accrue this social benefit from their more overt relationally aggressive gestures.

Importantly, children with ADHD typically do not differ from comparison children in their use of positive prosocial behaviors (Buhrmester et al., 1992; Mikami, Huang-Pollock, Pfiffner, McBurnett, & Hangai, 2007; Pelham & Bender, 1982) suggesting that it is not a lack of positive social interaction that drives negative peer relationships. Instead, children with ADHD often are described as being "socially busy" and may be more likely to initiate social interactions relative to peers without the disorder (Whalen & Henker, 1985, 1992). They also may present as more emotionally intense. Evidence suggests that those with combined type ADHD demonstrate more intense emotional displays of both positive and negative affect relative to comparison children (Maedgen & Carlson, 2000). Although not necessarily negative in nature, the fact that children with ADHD engage in a higher frequency of social interactions and are more emotionally intense may nonetheless increase their visibility by peers; this may create more opportunities for unwanted or negative peer interactions (Whalen & Henker, 1985).

Subtypes of ADHD

It is important to note that the previous descriptions best characterize the social behaviors of children with the combined and predominantly hyperactive–impulsive presentations of ADHD. Studies that have differentiated between the social behaviors of children diagnosed with DSM-IV combined type and inattentive type ADHD find that those with the predominantly inattentive type are more likely to appear as socially withdrawn, passive, and shy (Hodgens et al., 2000; Lahey, Schaughency, Strauss, & Frame, 1984; Maedgen & Carlson, 2000). For instance, during a playgroup with newly acquainted peers, children with inattentive type ADHD were rated by observers as high in social withdrawal relative to children with combined type ADHD and control children (Hodgens et al., 2000). Children with inattentive type ADHD also were rated by teachers as less assertive than children with combined type and comparison children (Solanto, Pope-Boyd, Tryon, & Stepak, 2009). Although the social behaviors of children with predominantly inattentive type ADHD may be qualitatively different, research indicates that these behaviors are still associated with peer dislike (Hodgens et al., 2000) and suggests that the subtypes do not differ in global social impairment (Solanto et al., 2009). Of course, these subtypes are now cast as "presentations" in DSM-5. These presentations may differ based on the severity of the predominant ADHD symptom dimension but do not represent distinct or static diagnostic categories.

SOCIAL-COGNITIVE SKILLS

Social-cognitive deficits also may contribute to the social impairments of children with ADHD. Although researchers have noted that some children with ADHD may lack appropriate social knowledge and skills (Grenell, Glass, & Katz, 1987; Wheeler & Carlson, 1994) this is not necessarily always the case. It is possible that many children with ADHD have social skills in their repertoires but primarily demonstrate social performance deficits; in other words, they have difficulty *applying* social skills effectively (de Boo & Prins, 2007; Wheeler & Carlson, 1994). In support of a social performance deficit, studies reveal that children with ADHD are poor monitors of their social behavior and have difficulty modulating behavior in accordance with shifting environmental demands (Landau & Milich, 1988; Whalen, Henker, Collins, McAuliffe, & Vaux, 1979). For example, Landau and Milich (1988) observed the social behaviors of children with and without ADHD when they were asked to shift between the roles of "host" and "guest" during a TV talk show game with another child. They found that control children were able to change their behavior in accordance with the two roles (e.g., answering more questions as the guest than as the host). However, children with ADHD failed to modulate their behavior between the two roles; instead they tended to adopt one interaction style and to use that style regardless of the shifting environmental demands or the response of the other child (Landau & Milich, 1988). The social performance deficits that children with ADHD display may result in part from deficits in key social-cognitive skills, including social information-processing, emotion recognition, and perceptions of social performance.

Social Information Processing

According to Crick and Dodge's (1994) reformulated social information-processing (SIP) model, individuals process social information in multiple stages. This includes encoding relevant social cues, interpretation of social information, setting a social goal, generating

and evaluating potential responses, and selecting a response. Evidence suggests that children with ADHD demonstrate a unique pattern of SIP, which may help explain their social performance deficits. For example, relative to comparison children, children with ADHD encode fewer social cues when presented with hypothetical social vignettes (Andrade et al., 2012; Matthys, Cuperus, & van Engeland, 1999). When children with ADHD do not have comorbid ODD or CD, their errors in encoding tend to be nonsystematic (Andrade et al., 2012; Cadesky et al., 2000; Sibley, Evans, & Serpell, 2010), suggesting that they are primarily a result of inattention to social information rather than biased attending. Children with ADHD also have greater difficulty integrating and organizing social cues coherently and are more likely to base their interpretation of social situations on the most recently supplied social information (Milch-Reich, Campbell, Pelham, Connelly, & Geva 1999). This is likely to affect their interpretation of social situations and their ability to make informed decisions about how they should respond. In addition, when asked to generate possible responses to social dilemmas, children with ADHD think of fewer responses than do comparison children (Matthys et al., 1999; Zentall, Cassady, & Javorsky, 2001). Thus, children with ADHD may fail to encode key social cues, have difficulty using those cues effectively to understand a social situation, and have greater difficulty identifying appropriate social responses. As a result, children with ADHD are likely to miss important social information and may persist in their use of negative or inappropriate behaviors.

Children with ADHD and comorbid ODD or CD may demonstrate a different pattern of SIP that is more consistent with aggressive children (Crick & Dodge, 1994). Matthys and colleagues (1999) found that boys with ADHD and comorbid ODD or CD were more likely to generate aggressive responses to hypothetical social dilemmas and to report greater confidence in their ability to use aggression relative to comparison children; this pattern of responding was not found in children with ADHD without this comorbidity (Matthys et al., 1999). However, in a sample of girls with ADHD, a diagnosis of ODD or CD was not associated with the generation of more aggressive responses or biased interpretations of hostility (i.e., a hostile attributional bias). In addition, SIP patterns only weakly predicted aggressive behavior in girls with ADHD, though associations were stronger for comparison girls (Mikami, Lee, Hinshaw, & Mullin, 2008). This may suggest that, at least among girls with ADHD, aggressive behavior results more from impulsivity and poor emotion regulation than from deficient SIP. Given that few studies have examined the implications of SIP for behavior in children with ADHD, additional research is needed.

Emotion Recognition

More limited evidence suggests that children with ADHD also may have specific difficulty identifying emotions. Incorrect identification of emotion has been found both when children are presented with spoken sentences and when they view pictures of faces (see Uekermann et al., 2010, for a review). Some evidence suggests that the errors in emotion identification made by children with ADHD are random in nature (Cadesky et al., 2000), suggesting that they primarily result from inattention. However, other evidence suggests that children with ADHD may have specific difficulty identifying anger or fear in facial expressions, and may be more likely to perceive these expressions as neutral or sad (Williams et al., 2008). Failure to read emotions in others may directly impair the ability of children with ADHD to monitor peer feedback. Given the tendency of children with ADHD to behave in emotionally intense ways (see Chapter 3), failure to notice anger or fear may be especially problematic. In these situations, children with ADHD may not realize that their intensity is making peers uncomfortable, and they may therefore persist in these behaviors.

Positively Biased Self-Perceptions

When children with ADHD rate their social competence on self-report measures, on average, they tend to overestimate their competence relative to how their teacher or parent rates their competence (Hoza, Pelham, Dobbs, Owens, & Pillow, 2002; Hoza et al., 2004). This overestimation of competence, termed a "positive bias" (or, in earlier work, a "positive illusory bias"), is found when examining self-perceptions of social competence, as well as competence in other domains (e.g., academic competence, behavioral conduct). This positive bias also is more common and more extreme in children with ADHD than in comparison children (see Owens, Goldfine, Evangelista, Hoza, & Kaiser, 2007, for a review) and does not result merely from

more impaired competence (McQuade et al., 2011). Yet not all children with ADHD exhibit this bias. A substantial subset of children with the combined or hyperactive/impulsive presentations of ADHD demonstrate a positive bias; however, children with the inattentive presentation of ADHD generally to not show a positive bias (Owens & Hoza, 2003).

In addition to evidence of inflated self-perceptions of competence as compared to parent or teacher ratings, children with ADHD, on average, also demonstrate a positive bias when asked to evaluate their *actual* social performance in other contexts. For instance, Hoza, Waschbusch, Pelham, Molina and Milich (2000) asked children with and without ADHD to complete a social interaction task with a child confederate and manipulated their success or failure during the task. On average, children with ADHD rated their social performance during the task as more favorable than did comparison children, despite being rated as less socially effective by objective observers. Importantly, this overly positive self-evaluation was present even in the failure condition, when social failure was blatant (e.g., when the confederate gave clear verbal and nonverbal cues of disinterest; Hoza et al., 2000). Failure of children with ADHD to acknowledge or perceive their social impairments also is found in other areas of social functioning. For instance, children with ADHD failed to report declines in their dyadic friendship that were reported by their friend (Normand et al., 2013). They also failed to report that they engaged in bullying despite endorsement of these behaviors by parents and teachers (Wiener & Mak, 2009).

It is not clear whether the presence of a positive bias in children with ADHD is a result of self-protective motivations or impaired self-monitoring (Owens et al., 2007), and there is evidence to support both possibilities. For instance, Diener and Milich (1997) found that during a social interaction task, children with ADHD overestimated how much their partner liked them only when they received no feedback about their performance during the task. However, when children with ADHD were provided with positive feedback, they did not overestimate their partner's liking. This would suggest that children with ADHD demonstrate a positive bias primarily as a defensive strategy that they may use to counter feelings of inadequacy. If such feelings are assuaged, children with ADHD may have no reason to overestimate their performance. Yet other evidence suggests that a positive bias may result from impairments in executive functioning (discussed in Chapter 4), which may limit social reasoning and perspective-taking ability. Specifically, McQuade and colleagues (2011) compared the executive functioning skills of control children and children with ADHD, with and without a positive bias. Results indicated that children with ADHD and a positive bias in social competence demonstrated greater deficits in a range of executive functioning skills compared to the other groups. This may suggest that children with ADHD with a positive bias have cognitive deficits that impair their ability to engage in effective self-evaluation.

Findings from a study by Hoza, Vaughn, Waschbusch, Murray-Close, and McCabe (2012) suggest that children with ADHD may have an especially difficult time accurately reporting on their social competence relative to other domains of functioning. Specifically, Hoza and colleagues examined how children with and without ADHD rated their competence when told that they could earn a dollar for each item that was rated the same as their teacher. They found that with this potent motivator, children with ADHD significantly reduced their overestimation of academic and behavioral conduct relative to teacher report but continued to demonstrate a positive bias in social competence. One possible reason for this finding is that children with ADHD have greater difficulty accurately judging their social competence, perhaps because social feedback is more subtle, or because of cognitive impairments that affect SIP. An alternative possibility, however, is that admitting to impairments in social functioning is especially difficult for some children with ADHD and that, even with motivation, they continue to deny their social struggles.

Although the reason for a positive bias is still not fully understood, failure to acknowledge or perceive social impairments is likely to have negative implications for the social functioning of children with ADHD. Researchers have proposed that accurate self-perceptions may be necessary for individuals to adjust their behavior and to learn from prior mistakes (Colvin & Block, 1994). Thus, children with a positive bias would be expected to be more likely to persist in social impairments over time. Indeed, Mikami, Calhoun, and Abikoff (2010) found that over the course of a behavioral summer treatment program, children with ADHD with a social positive bias showed a decline in peer liking at the end of the summer, whereas those without a positive bias became better liked. The researchers also found

that children with ADHD acquired a greater number of friends at the end of treatment only when they did not have a positive bias in behavioral conduct; those that did have this bias showed no improvement in friendships. It is concerning that this pattern of results was found in the context of a therapeutic summer program in which explicit feedback regarding behavior and instruction in social skills was provided. Those children with a positive bias may have failed to acknowledge the social feedback supplied and may therefore have persisted in their use of maladaptive social strategies.

Other evidence suggests that children with ADHD with a positive social bias may demonstrate a distinct pattern of negative social behaviors. Specifically, Linnea, Hoza, Tomb, and Kaiser (2012) compared the social behaviors of children with ADHD, with and without a positive social bias, and comparison children during a laboratory social interaction task. Children with ADHD with a positive bias were rated by coders as less friendly, responsive, and engaged than both comparison children and children with ADHD without a positive bias. Children with ADHD and a positive bias also exhibited higher rates of odd behaviors, were more likely to appear inattentive, and were more helpless than comparison children; those with ADHD without this bias did not show impairments on these factors (Linnea et al., 2012). A positive bias in behavioral conduct also may specifically increase risk for aggressive behavior among children with ADHD (Hoza, Murray-Close, Arnold, Hinshaw, & Hechtman, 2010; Murray-Close et al., 2010); this may result from greater defensiveness when they receive negative social feedback (Baumeister, Smart, & Boden, 1996). Thus, it appears that children with ADHD and a positive social or behavioral bias are more likely to demonstrate persistent social impairment and greater negative behaviors. Given that acknowledgment of social impairments is likely a necessary prerequisite for adjusting behavior and improving over time, it makes sense that we would see more impaired social functioning among children with ADHD when they demonstrate a positive bias.

UNIQUE SOCIAL CHALLENGES IN ADOLESCENCE

For most children with ADHD, the symptoms of the disorder (Barkley, Fischer, Edelbrock, & Smallish, 1990) and their social impairments (Bagwell et al., 2001) persist into adolescence. However, the developmental period of adolescence may introduce unique social challenges for youth with ADHD. For instance, adolescents with ADHD are more likely to engage in social behaviors that specifically promote deviancy and vulnerability to risky behaviors (Whalen, Jamner, Henker, Delfino, & Lozano, 2002). Whalen and colleagues found that adolescents with high levels of ADHD symptoms were more likely to drink alcohol or smoke cigarettes, to spend more time socializing with friends, and to spend less time with family. The researchers proposed that this pattern of behaviors may suggest that adolescents with ADHD are less well monitored by parents and may therefore have greater opportunity to be exposed to socially risky situations. Comorbid CD may further increase socially risky behavior in adolescents with ADHD. One study found that relative to comparison adolescents and adolescents with ADHD without comorbid CD, adolescents with ADHD and comorbid CD were less likely to be involved in conventional adolescent activities, such as school or community activities. In addition, these adolescents also were more likely to have friends who were using drugs and alcohol. In fact, 64% of parents of adolescents with ADHD and comorbid CD disapproved of their child's friends, compared to 38% of adolescents with ADHD without comorbid CD and 28% of parents of adolescents without ADHD (Bagwell et al., 2001). Other evidence suggests that association with deviant peers increases the likelihood that adolescents with ADHD will engage in substance use themselves (Marshal & Molina, 2006; Marshal, Molina, & Pelham, 2003). Furthermore, deviant peer behavior may more strongly influence the likelihood of substance use in adolescents with ADHD relative to comparison children (Marshal et al., 2003). This risk may be greatest when these adolescents have comorbid ODD or elevated CD symptoms (Marshal & Molina, 2006). Thus, not only are adolescents with ADHD more likely to be drawn to deviant peers, but also they may be particularly impressionable and more likely to mimic their undesirable behaviors.

In addition to substance use, adolescents with ADHD also are more likely to engage in risky sexual behaviors. One study revealed that relative to comparison males, males with a childhood diagnosis of ADHD engaged in sexual activity and intercourse at an earlier age, had more sexual partners, had more sex with newly acquainted peers, had more casual sex without condom use, and had a greater frequency of partner pregnancies

by early adulthood (Flory, Molina, Pelham, Gnagy, & Smith, 2006). Males with ADHD may have increased engagement in these risky sexual behaviors regardless of comorbid disorders (Flory et al., 2006); however, in females, CD, rather than ADHD, may predict greater risk (Monuteaux, Faraone, Gross, & Biederman, 2007). Some evidence suggests that a positive bias in behavioral or social competence may partially explain (i.e., mediate) the association between childhood ADHD and risky sexual behavior in young adulthood (Hoza et al., 2013). This could suggest that adolescents with ADHD are likely to engage in risky sexual behavior because of impaired self-monitoring or poor insight (see Chapter 11 for more on this topic).

The landscape of social interaction for adolescents also has undergone drastic changes in recent years, with increased use of electronic and online social interactions using e-mail, texting, chat rooms, instant messaging, and social media pages such as Facebook. Among a sample of high school students, 90% reported having an e-mail account, 65% reported using instant messaging, and 88% had a profile on at least one social networking site (Reich, Subrahmanyam, & Espinoza, 2012). Research has not directly examined social media use among adolescents with ADHD. However, evidence does indicate that youth with ADHD demonstrate a pattern of aversive and negative social behaviors during online chat room tasks that is similar to their behaviors in live social interactions (Mikami et al., 2007; Ohan & Johnston, 2007). Thus, it is likely that their social impairments also manifest in online interactions with peers. Given that youth with ADHD experience peer rejection and bullying in face-to-face interactions, they may also be at higher risk for victimization by cyberbullying (e.g., bullying through e-mail, instant messaging, chat room, Web, or text interactions). Indeed, in a sample of 10- to 20-year-olds with a diagnosis of either ADHD or an autism spectrum disorder, 21% reported being cyberbullied in the past 2 months but only about 6% reported engaging in cyberbullying (Kowalski & Fedina, 2011). Because a comparison group was not included, it is unclear how these rates compare to normative samples of children and adolescents. Although additional research is sorely needed in this area, the social profile outlined thus far suggests that adolescents with ADHD are likely to demonstrate problems in their peer relationships when online or when using a cell phone. Given their engagement in other risky social behaviors, it also is possible that adolescents with ADHD may engage in risky online behaviors such as posting inappropriate pictures, befriending people they do not know, or posting private or potentially damaging personal information.

CONCLUSION

Peer problems are common and often enduring associated impairments for youth with ADHD. Not only are these children more rejected and less accepted by peers (Hoza, Mrug, et al., 2005), but they also have fewer reciprocal friendships (Mikami, 2010), and when they do form friendships, these friendships are less enduring and of lower quality (Normand et al., 2011, 2013). Unfortunately these patterns of peer dysfunction form quickly in new peer groups (e.g., Pelham & Bender, 1982) and are hard to alter (Hoza, Gerdes, et al., 2005).

The reasons for these peer difficulties are not fully understood, although the very behaviors that characterize ADHD (e.g., impulsivity, hyperactivity, disruptiveness) are likely to be noxious to peers. Children with ADHD are found to be annoying, intrusive, and disruptive in their social interaction style (Pelham & Bender, 1982). They also may be socially eager, attempting to interact as much as (or more than) typical peers, and often with greater emotional intensity (Whalen & Henker, 1992). Despite interest and desire for friendship, their attempts may often be unsuccessful. Overtly negative social behaviors are more typical of children with combined type and hyperactive–impulsive type ADHD, whereas those with inattentive type ADHD may present as socially withdrawn and shy (Hodgens et al., 2000). Although these social behavior profiles differ, all three subtypes are more likely to be disliked by their peers (Hodgens et al., 2000).

Research on the underpinnings of peer dysfunction in this population suggests that even those youth with ADHD who possess appropriate social skills may still have difficulty performing them appropriately in social situations (de Boo & Prins, 2007). Whether this performance deficit stems from inattention to social cues, poor emotion recognition and regulation, impaired SIP, faulty self-monitoring in ongoing social situations, or a combination of these factors has not been definitively determined (McQuade & Hoza, 2008). However, what is certain is that peer dysfunction is an impairing problem, more often than not affecting the happiness and well-being of these youth and placing them at risk for long-term maladjustment (Mrug et al., 2012). Thus, whenever present, peer relationship problems should

be considered a critical target of intervention for youth with ADHD.

KEY CLINICAL POINTS

- ✓ Children with ADHD are more likely to be rejected and less likely to be accepted by the broader peer group.
- ✓ Peer rejection in children with ADHD develops quickly and is resistant to change. Persistence of peer rejection may partly be influenced by the social reputation of the child with ADHD.
- ✓ Children with ADHD are less likely to have reciprocal friendships and are more likely to befriend children with similar behavioral challenges.
- ✓ The friendships of children with ADHD are more negative and less positive in quality, and less stable over time.
- ✓ Children with ADHD are at higher risk for not only being victimized by peers but also engaging in bullying behaviors.
- ✓ Peer rejection, fewer or poorer quality friendships, victimization, and bullying predict greater emotional and behavioral problems in children with ADHD.
- ✓ Children with ADHD, especially the combined and predominantly hyperactive–impulsive presentations, display high rates of intrusive, annoying, and disruptive behavior during social interactions with peers.
- ✓ Aggressive behavior is more common among children with the combined presentation of ADHD, especially with comorbid ODD or CD. Studies using female samples suggest that they may be more relationally aggressive in addition to overtly aggressive.
- ✓ There is a lack of evidence suggesting differences in use of positive social behaviors among children with and without ADHD.
- ✓ Children with ADHD are more socially busy and emotionally intense, which may create more opportunities for negative social interactions with peers.
- ✓ Children with the predominantly inattentive presentation of ADHD are more likely to be shy, withdrawn, and passive in their social interactions with peers; despite differences in social behavior, children with this subtype are still rejected by peers and socially impaired.
- ✓ Children with ADHD demonstrate a distinct pattern of SIP impairments; they encode fewer social cues, have greater difficulty interpreting social situations, and generate fewer effective social responses.
- ✓ The SIP pattern of children with ADHD and comorbid ODD and CD may be specifically characterized by greater ability to generate aggressive responses and greater confidence in using them.
- ✓ Some evidence suggests that children with ADHD may have specific impairments in identifying emotions in others' faces or in spoken sentences.
- ✓ A subset of children with ADHD demonstrate positively biased self-perceptions of social competence and performance.
- ✓ Children with ADHD and a positive bias are less likely to show improvements in their peer relationships, demonstrate a greater frequency of negative social behaviors, and are more aggressive.
- ✓ Adolescents with ADHD may be less well monitored by parents, placing them at increased risk for engaging in socially risky behaviors.
- ✓ Adolescents with ADHD and comorbid CD are more likely to associate with deviant peers and to have friends who use substances; such association may increase their own engagement in substance use, especially if they have comorbid ODD or CD.
- ✓ Adolescents with ADHD are at higher risk for engaging in sexually risky behaviors.
- ✓ Youth with ADHD are likely at higher risk of experiencing social impairments when using online social media and cell phones, though research investigating online behaviors is lacking.

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CHAPTER 9

Developmental Progression and Gender Differences among Individuals with ADHD

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It is widely known that attention-deficit/hyperactivity disorder (ADHD) is a highly heritable, neurodevelopmental condition affecting many individuals around the world. In the United States, between 6.0 and 9.5% of children and youth have a current diagnosis of ADHD (Barkley, 2006; Visser, Danielson, Bitsko, Perou, & Blumberg, 2013). These estimates, along with U.S. Census data (*www.census.gov/prod/cen2010/briefs/c2010br-03.pdf*), indicate that in 2010, between 3.2 and 5.1 million school-age youth were affected. Among these millions of children and adolescents with ADHD, concurrent psychiatric comorbidities and functional impairments in a wide range of domains—academic, social, psychiatric, and personal well-being—are well documented and widely acknowledged. However, much of what is known about ADHD applies mainly to school-age boys. As ADHD researchers have broadened their focus in recent years, it has become clear that the presentation and nature of ADHD changes over time and perhaps across genders. Our chapter concerns just this variability in presentation. What happens over time to children and adolescents with ADHD? What is the typical developmental trajectory of the disorder and its symptoms, and what are the typical outcomes during adolescence and adulthood? Furthermore, are there important differences between the sexes, especially in terms of developmental progressions or long-term outcomes?

Beyond the scientific relevance of these questions is a more fundamental rationale. Parents of children with ADHD, and the clinicians involved, want to know as much as they can about their childrens' likely futures. Families need to know what to expect. They may wonder whether their child's course will be similar to or different from what is typical and whether his or her future depends on gender, co-occurring psychiatric problems, learning disabilities, or treatment efforts. Thus, we begin our chapter with a discussion of long-term, prospective, longitudinal studies of children who have received a diagnosis of ADHD (or its equivalent) during childhood. We review relatively recent studies with a thorough baseline assessment of ADHD diagnostic status, a control group, and a follow-up assessment at least 4 years later. More distal projects and articles (e.g., Weiss, Hechtman, Milroy, & Perlman, 1985) have produced important findings, but these have been well reviewed elsewhere and are not included here. We include only findings from samples of children and youth who received a categorical designation, DSM-based or otherwise, of ADHD. We do not include studies that assessed only dimensionally measured symptoms related to ADHD, such as "attentional difficulties."

From these prospective longitudinal studies we derive evidence regarding the typical changes in ADHD diagnoses and symptoms over time, from childhood through adulthood. We also derive information concerning outcomes for children and adolescents with ADHD across various domains of psychopathology and impairment, including externalizing and internalizing problems, substance use and abuse, educational and occupational attainment, social functioning, and driving. We do not consider neuropsychological outcomes (although we do consider neuropsychological functioning in the section "Gender Differences") because the focus of our chapter is psychosocial and adaptive functioning, and because a review of cognitive and neuropsychological features would take us far beyond our page limits.

We highlight at the outset that many outcomes for which children with ADHD are at risk are not obtained by all (or even most) children with this condition. Clearly, what is typical for the group does not apply to every member of the group. *Multifinality*, whereby a specific risk factor is associated with a range of outcomes, is the rule with ADHD (Cicchetti & Rogosch, 1996). Moderators and mediators that can explain varyious developmental outcomes include comorbidities, ADHD subtype, neuropsychological functioning, and treatment utilization. We briefly review evidence for these, as well as gender.

Except in the section "Gender Differences," we do not include studies of adults who have self-referred for ADHD treatment or who have otherwise been ascertained as adults. The population of individuals who present with ADHD symptoms in adulthood may be rather different from the population that is identified and diagnosed during childhood, even though many of those adults recall ADHD symptoms as children (Barkley, Murphy, & Fischer, 2008). Furthermore, Mannuzza, Klein, Bessler, and Shrout (2002) estimate that only 27% of adults who recall having ADHD as a child would have met strict diagnostic criteria during that developmental time frame. Whereas data from these individuals can provide important information about ADHD in adults, they are not particularly relevant to questions regarding developmental progression.

After our review and discussion of the longitudinal course, typical outcomes, and moderators of outcomes, we discuss gender differences across the age span. In this section a number of investigations (many cross-sectional) are available for review. We consider gender differences in prevalence, manifestations (e.g., comorbidities, social functioning, neuropsychological functioning), and treatment response. Of particular note, given the developmental theme of our chapter, we focus on whether long-term outcomes are different for girls and boys, and whether ADHD manifests differently between the sexes over time.

THE LAY OF THE LAND: THE PRIMARY LONGITUDINAL STUDIES

Long-term longitudinal studies of clinical child populations and control participants are expensive and difficult to execute. Thus, the information on which we base this first section of our chapter is drawn from a limited number of research projects, although the number of articles produced by these research projects is large. Below are overviews of participants and procedures employed by the major longitudinal projects, in roughly chronological order.

From consecutive referrals by parents, teachers, and pediatricians to a Los Angeles-based outpatient mental health clinic in the early 1970s, James Satterfield and his colleagues recruited European American boys (ages 6–12) for a study of delinquency outcomes and preventive treatments. Using standardized measures administered to parents and teachers, 180 boys were diagnosed as hyperactive. Had the criteria existed at the time, all participants probably would have met criteria for the DSM-III (American Psychiatric Association, 1980) diagnosis of attention deficit disorder (ADD) with hyperactivity. The 110 boys recruited first received 2.1 years of medication treatment, and 70 subsequently recruited boys received 1.8 years of medication plus individualized psychosocial treatment. Eighty-eight control children, matched for sex, age, IQ, and social class, were recruited from local schools. Nine years later, official arrest records for 81 of the medication-treated group and 50 of the multimodal-treated group were obtained when the children averaged 17 years of age. These same records were obtained in early adulthood (ages 19–25) and middle adulthood (ages 32–42), at which point arrest records were available on over 90% of the participants.

Salvatore Mannuzza, Rachel Klein, and their colleagues ascertained a sample of children with hyperactivity in New York City in the early 1970s. From over 1,000 children referred to a psychiatric clinic, 115 were identified as hyperactive based on teacher report and a DSM-II (American Psychiatric Association, 1968) diagnosis of hyperkinetic reaction (for Cohort 2, below,

hyperactivity at home was also required). All children were European American and middle class. The sample was 92% male; only 19 participants were girls. A control group without childhood behavior problems, n = 100, was ascertained at the adolescent follow-up from nonpsychiatric departments of the same medical center to which the probands were referred. Additionally, a replication sample of 111 children with ADHD and 78 controls was obtained shortly thereafter using the same screening and recruitment strategies as the first cohort and with a highly similar follow-up schedule. These children have been followed during adolescence (mean age 18), early adulthood (mean age 25), and most recently during adulthood, 33 years after baseline (in their early 40s). At adolescent follow-up, 98% of Cohort 1 and 89% of Cohort 2 were assessed; at young adult follow-up, 90% of Cohort 1 and 83% of Cohort 2 were assessed; and at the 33-year follow-up, the retention rate was 65% for the participants with ADHD and 76% for the controls.

Importantly, in both the original and the replication samples, children referred primarily for aggression or antisocial behavior were screened out prior to participation. The investigators' intent was to study a group of "pure" hyperactive children without co-occurring externalizing problems. The authors contend that all children in the study would have met the more modern criteria for ADHD combined presentation (ADHD-C). Unlike some prior investigations and like most of the investigations since, follow-up assessors were blind to group status.

Russell Barkley and Mariellen Fischer began the next large, longitudinal study of children with ADHD in Milwaukee, WI, by recruiting and enrolling 158 children in 1979 and 1980. Children, ages 4–12, were referred to a university medical clinic and considered to meet research diagnostic criteria for ADHD when (1) scores on hyperactivity rating scales exceeded two standard deviations above the mean; (2) the parent reported significant behavior problems at home; (3) the parent or teacher complained of inattention, impulsivity, and hyperactivity; and (4) symptom duration was at least 12 months. DSM criteria were not used, but it is quite likely that all participants would have met DSM-III-R criteria for ADHD. Although cross-setting symptomatology was not required, most children displayed problematic behavior in multiple settings, and all had an early age of symptom onset. Notably, given the requirement of elevated scores on a parent-rated hyperactivity index, most children were also aggressive or defiant. This sample was 91% male and 94% European American. They, and 81 control participants who were referred by participants, have been assessed four times: at recruitment during childhood, during adolescence (age range 12–17), during young adulthood (age range 19–25), then during adulthood (mean age 27). Retention rates of participants with ADHD have ranged across age, with average retention rates across publications reported to be between 78 and 93%.

Joseph Biederman and his colleagues have conducted two long-term longitudinal studies of children and adolescents with ADHD using highly similar methodologies in each. The first study involved boys ages 6–17 who were recruited and enrolled in the 1980s. Probands were referred from a pediatric psychopharmacology clinic and from primary care pediatric practices. One hundred forty boys who met DSM-III-R (American Psychiatric Association, 1987) criteria for ADHD according to a structured diagnostic interview administered to parents and children older than 12 were enrolled, as were 120 control boys recruited from the same pediatric practices that referred the ADHD probands. All boys were European American and middle class. Initial follow-up occurred 4 years later and retention was very high (91%). At the 10-year follow-up (mean age 22) 80% of participants with ADHD and 88% of controls were retained.

In the subsequent girls' study, parallel recruitment and assessment strategies were used. Ultimately, 140 probands and 122 controls ages 6–18 were enrolled. Unlike the boys' study, a small percentage of nonwhite (6% of the ADHD group and 11% of the control group) and lower-income children were included. After their initial assessment, these girls were followed up 5 years later at the mean age of 17, at which time 88% of the participants with ADHD and 92% of the controls were reassessed. These girls were also reassessed during young adulthood (mean age 22), approximately 11 years (range 8–14) after their baseline assessment. At that point, retention of the participants with ADHD and controls was fair (69 and 75%, respectively). Of note, in both the boys' and girls' studies, subtypes (combined vs. predominantly inattentive) were not diagnosed because DSM-III-R (American Psychiatric Association, 1987) diagnostic criteria were used, but it is believed that the vast majority of participants would have met current criteria for ADHD-C.

In New York City between 1990 and 1997, Jeffrey Halperin, Jeffrey Newcorn, and colleagues recruited 169 children ages 7–11 from local schools and medi-

cal practices. The children were referred for behavior problems and assessed using the Parent version of the Diagnostic Interview Schedule for Children (DISC), either version 2.1 (Fisher et al., 1993) or version 2.3 (Shaffer, Fisher, Dulcan, & Davies, 1996). Enrolled children met criteria for ADHD according to DSM-III-R (American Psychiatric Association, 1987), and most would have met DSM-IV (American Psychiatric Association, 1994) criteria for ADHD-C. The sample was ethnically diverse (24% European American, 26% African American, 35% Hispanic), mostly low income, and mostly male (88%). A control group of 85 children was recruited through direct advertisement in the probands' neighborhoods. Fifty-one percent of these children were followed up once, 9 years later, when they were ages 16–21 (mean age 18.2).

The Multimodal Treatment Study of Children with ADHD (the MTA) was the largest child clinical trial ever sponsored by the National Institute of Mental Health. Originally, 579 school-age children with ADHD were recruited at six sites in the United States and Canada from mental health settings, pediatricians, advertisements, and schools. They were carefully diagnosed using DSM-IV criteria for ADHD-C, according to the Parent version of the DISC, version 2.3 (Shaffer et al., 1996), supplemented with up to two symptoms identified by teachers for children falling just below the DISC diagnostic threshold. Children averaged 8.5 years old (range 7.0–9.9 years), were 80% male, and 61% European American. They were randomized to one of four 14-month-long treatment conditions: medication only, behavioral treatment only, medication plus behavioral treatment, and community control. Treatment occurred between 1994 and 1997, with initial follow-ups at 10- and 22-months posttreatment. Since a 6-year postbaseline follow-up, participants have been assessed every 2 years, with data collection coming to an end in 2013, 16 years after baseline. At each follow-up, priority was placed on obtaining multiinformant and multimethod data across domains of functioning, with a particular emphasis on substance use and disorder outcomes.

William Pelham, Brooke Molina, and colleagues conducted the Pittsburgh ADHD Longitudinal Study (PALS), in which 364 probands were recruited between 1999 and 2003 as adolescents and young adults (ages 11–28) for a study of predictors of alcohol and drug abuse. Between 1987 and 1996, when they were ages 5–17 years, all had been diagnosed with ADHD according to DSM-III-R or DSM-IV criteria as part of their participation in a summer treatment program. Additionally, 240 controls recruited primarily through pediatric practices matched the probands in terms of age, sex, ethnicity, and parental education level. The total PALS sample was 89% male and 82% European American. After adolescent ascertainment, these participants were followed yearly five times, with excellent retention (over 90%).

Benjamin Lahey and William Pelham recruited preschoolers (ages 3–6 years) in consecutive years at two sites. In Chicago, children were recruited from a psychiatry clinic. In Pittsburgh, half of the children were recruited from a psychiatry clinic and half from an advertisement. Two hundred fifty-five children who met symptom criteria for ADHD based on parent–teacher rating were enrolled. Given that the children had not yet entered the formal school environment, the cross-situational impairment was not required for eligibility, but all children showed impairment in at least one setting, and most showed impairment in two. Controls, who matched probands in terms of sex, ethnicity, and age, were recruited from schools similar to those attended by the probands. Eighteen percent of the sample was female, and 36% were nonwhite. Children were followed up yearly for 14 years, with the last assessment occurring during late adolescence. Retention rates were high (80–92% across assessments 6–14 years after baseline); measures emphasized psychiatric symptoms and impairment.

The Berkeley Girls with ADHD Longitudinal Study (BGALS) began in 1997 and is ongoing; a fourth wave of data collection began in the Fall of 2013. Girls ages 6–12 were recruited and enrolled in a naturalistic, observational, 5-week summer program that operated for three summers: 1997 (n = 79); 1998 (n = 77) and 1999 (n = 72). Through a multigated screening and diagnostic process, a total of 140 girls diagnosed with ADHD according to DSM-IV criteria, and 88 age- and ethnicity-matched comparison girls without ADHD, were assessed at baseline. Diagnostic status was established using the Parent versions of the DISC-IV (Shaffer, Fisher, Lucas, Dulcan, & Schwab-Stone, 2000) and the Swanson, Nolan, and Pelham–IV (SNAP-IV; Swanson, 1992) ratings. Ninety-three of the girls were diagnosed with ADHD-C, and 47 with predominantly inattentive type ADHD (ADHD-PI). The sample, representative of the San Francisco East Bay area, is 53% white and socioeconomically diverse. Girls were first seen during childhood, then followed up during adolescence (mean age = 14.2 years) and young adulthood

(mean age = 19.6 years). Retention at the first follow-up was 92%, and that at the second was 95%. At each assessment point, data were collected from multiple informants (parents, teachers, peers, self, objective testing) regarding multiple domains of symptoms and impairments.

Of note, girls with ADHD-PI, as well as ADHD-C, participated in the BGALS study, allowing comparison of subtype differences not possible in previous studies that used DSM-III or DSM-III-R criteria, or focused only on children with ADHD-C. This study, the MTA, and Biederman's girls' study, are to our knowledge the only three long-term longitudinal studies that include a large number of girls with ADHD. Additionally, although not an epidemiological or true community sample, girls in the BGALS study were recruited from multiple sources, including schools and direct advertisements. Some children were referred from physicians, but unlike the previous longitudinal studies reviewed here, many were not. As noted below, participant recruitment strategies are an important consideration when interpreting and generalizing findings from studies of children with ADHD.

Across these studies, a few characteristics are noteworthy. With few exceptions, most children with ADHD followed longitudinally have been boys. Nearly all participants have been from the United States. The majority have been white and middle class, although the Newcorn/Halperin participants were primarily nonwhite and the BGALS sample was only 53% white. For various reasons, including the lack of established ADHD subtype diagnoses at the time of ascertainment in most samples, ADHD subtypes have not been documented, except in the BGALS sample. In most cases, participant presentation was consistent with the ADHD-C subtype. Importantly, the majority of children followed prospectively have been identified through referral to psychiatric or mental health facilities, although a sizable minority have been referred by teachers and schools or recruited through direct advertisement. To our knowledge, only two prospective longitudinal studies of children with and without ADHD have employed true community samples: August and colleagues (2006) and Bussing, Mason, Bell, Porter, and Garvan (2010). August and colleagues screened over 7,000 children in grades 1–4 at 22 schools in Minnesota and ultimately identified 205 children who met full DSM-III-R criteria for ADHD. Some of these children (n = 131) were followed up in late adolescence (36% attrition), at a mean age of 18. Bussing and colleagues randomly sampled 1,615 children from over 12,000 public school children in kindergarten through fifth grade in North Florida. Girls were oversampled, and the sample was demographically diverse. Four hundred seventy-six were identified as high risk for ADHD, but ultimately only 169 were diagnosed with ADHD and agreed to participate. These children, and a control group, were followed up, ultimately until age 17.

THE DEVELOPMENTAL PROGRESSION OF ADHD

Diagnostic Status and Symptoms over Time

ADHD was once believed to be a transient childhood problem that typically resolved by adolescence or adulthood. The sum total of evidence from longitudinal research reveals that this is clearly not the case. Some earlier reports showed ADHD diagnostic persistence, when self-reported, to be quite low. For example, only 8–11% of probands showed continuing self-reported ADHD at adolescent follow-up, with rates depending on definition of persistence (Mannuzza, Klein, Bessler, Malloy, & LaPadula, 1993). Similarly, Barkley, Fischer, Smallish, and Fletcher (2002) documented that only 5% of hyperactive probands self-reported persistence into young adulthood. According to Hill and Schoener's (1996) meta-analysis of nine studies utilizing mostly self-report at follow-up, the diagnostic rates of ADHD decline 50% every 5 years. Their findings suggest that, by adulthood, very few people diagnosed as children continue to meet criteria for ADHD (0.05% of adults at age 40), implying that most children with ADHD will "grow out of it."

However, answers to the question about developmental progression depend on how persistence is defined and whom one asks (Barkley et al., 2002; Mannuzza, Klein, & Moulton, 2003). When parents are queried about their children's continuing symptoms and impairment, the picture is vastly different from that portrayed by the previously cited findings. For example, in Barkley's sample, using DSM-III-R diagnostic criteria, parent report produced a persistence rate of 72% at adolescence (Barkley, Fischer, Edelbrock, & Smallish, 1991). In young adulthood, this rate was 46%, compared to the 5% reported previously (Barkley et al., 2002). When developmentally referenced criteria were used (i.e., when normative declines in inattention, hyperactivity, and impulsivity were considered), the rate of parent-reported ADHD persistence increased to

66%, and the rate of self-reported ADHD persistence increased to 12%. Importantly, evidence suggests that parent report has more predictive validity than self-report, especially in terms of its concurrent and longitudinal associations with impairment (Barkley et al., 2002). A diagnostic lesson is that exclusive reliance on self-reported symptomatology is highly likely to lead to false-negative designations in the world of ADHD assessment.

Similarly, in the Mannuzza samples, when based on parent report, rates of ADHD persistence into adolescence increased from 8 and 11% (depending on cohort) to 40 and 43%, respectively (Gittelman, Mannuzza, Shenker, & Bonagura, 1985; Mannuzza et al., 1991). Of note, this rate of persistence into adolescence, even when parent-reported, is somewhat lower than findings from the Barkley, Biederman, and Hinshaw samples, probably because children with aggression and antisocial behavior were screened out of the recruitment process. These lower rates may obtain in a "pure" ADHD sample, whereas higher rates obtain in more representative samples when typical comorbidities are allowed. Similarly, Molina and colleagues (2009) reported that even according to parents, only 30% of their original sample retained the diagnosis 8 years later during adolescence, probably because most of the children in their sample received intensive treatment for ADHD.

Other, more recent studies clearly show high rates of parent-reported ADHD persistence into adolescence and adulthood, whether narrowly defined by DSM diagnostic criteria or more broadly defined based on symptom level and impairment. August, Braswell, and Thuras (1998) showed that during early to midadolescence, 69% of their participants still met criteria for ADHD. In the BGALS study, only 39 of the 128 girls with childhood ADHD assessed at the 5-year follow-up during adolescence no longer met full diagnostic criteria. Of those with an original ADHD-PI diagnosis, 76% continued to meet full ADHD diagnostic criteria during adolescence, according to parents, as did 66% of the girls with ADHD-C (Hinshaw, Owens, Sami, & Fargeon, 2006). In contrast, only 10% met diagnostic criteria on the DISC according to self-report during adolescence. At the 10-year follow-up (Hinshaw et al., 2012), rates of full ADHD diagnostic persistence were somewhat lower. Of those with a childhood diagnosis of ADHD-PI, 61% retained an ADHD diagnosis at young adulthood, as did 56% of those with ADHD-C. Only 15% of probands met criteria on the self-report DISC. Of note, these persistence levels do not account for changes in subtype; they reflect how many girls with a particular childhood diagnosis had any type of ADHD at follow-up.

In estimating the persistence versus remission of ADHD over time, Biederman and colleagues not only examined the rates at which children continued to meet full diagnostic criteria for ADHD (syndromatic persistence) but also included girls and boys who fell just short of the symptom criteria and demonstrated significant functional impairment (symptomatic persistence) in estimates of persistence versus remission of ADHD over time. Accordingly, in the female sample during adolescence, 82% showed either type of ADHD persistence (Biederman et al., 2006a). As young adults, 62% of these girls showed symptomatic or syndromatic persistence (Biederman, Petty, Fried, et al., 2010). Among the boys, as adolescents, 85% retained full or subthreshold ADHD and full recovery was rare (Biederman, Faraone, Milberger, & Guite, 1996; Biederman, Mick, & Faraone, 2000). During young adulthood, 63% continued to have either syndromatic or symptomatic ADHD, and an additional 15% had low symptom levels but were functionally impaired (Biederman, Petty, Evans, Small, & Faraone, 2010).

It follows that because, at a diagnostic level, childhood ADHD tends to persist, ADHD symptoms also tend to persist across time. At the same time, ADHD symptom presence should be judged in relation to what is expected given one's age. As a function of development, ADHD symptoms of inattention, hyperactivity, and impulsivity can and do decline over time (e.g., 18-year-olds are not as physically active, forgetful, or disorganized as 6-year-olds), but generally remain much higher in individuals with childhood ADHD compared to those without (Biederman et al., 2000; Hart, Lahey, Loeber, Applegate, & Frick, 1995; Loya & Hinshaw, 2013). For example, during both childhood to adolescence (Fischer, Barkley, Edelbrock, & Smallish, 1990) and adolescence to adulthood (Fischer, Barkley, Smallish, & Fletcher, 2005), all children show normative improvements on objective measures of inattention and impulsivity, but children diagnosed with ADHD still remain significantly more inattentive and impulsive than those without a childhood ADHD diagnosis. Loya and Hinshaw (2013) showed the same for parent-reported symptoms of inattention, impulsivity, and hyperactivity among girls. All decline significantly from childhood until early adulthood, but they remain four to 11 times as high in girls with childhood ADHD compared to girls without. Among girls and boys, this

overall decline in symptoms occurs gradually across childhood and adolescence, then accelerates by early adulthood (Monuteaux, Mick, Faraone, & Biederman, 2010). It appears that symptoms of hyperactivity–impulsivity show less stability, in the form of greater declines, than symptoms of inattention (August et al., 1998; Biederman et al., 2000; Fischer, Barkley, Fletcher, & Smallish, 1993b; Hart et al., 1995), at least until young adulthood, after which time the symptom types are equivalently stable (Biederman, Petty, Evans, et al., 2010). Thus, at the symptom level, children with ADHD typically continue to exhibit and to be impaired by inattention and disorganization as they grow into adolescents and adults, even as the level of their symptoms (particularly hyperactive–impulsive symptoms) declines with normative development.

In summary, data obtained primarily from clinically referred children (both boys and girls) suggest that according to parents, rather than participants, ADHD persists at high levels into adolescence and at moderate levels into adulthood. Wilens, Biederman, and Spencer (2002) estimated that half of children rigorously diagnosed with ADHD will continue to meet diagnostic criteria for the disorder as adults. In line with this conclusion, and using a community-based sample of children with ADHD, Bussing and colleagues (2010) showed that 44% continued to meet full diagnostic criteria during adolescence. When persistence of ADHD is defined by continuing symptoms and significant impairment (rather than meeting full diagnostic criteria), as many as two-thirds of children diagnosed with ADHD show persistence into adulthood (Faraone, Biederman, & Mick, 2006). This is true despite normative declines in symptoms of inattention, hyperactivity, and impulsivity. It is important to remember that data about the developmental progression of ADHD are obtained primarily from referred children rather than from undetected children with ADHD in the community. We know less about what happens to children who have ADHD but are not identified as needing help. Additionally, most children in the relevant investigations met criteria for what is now called ADHD-C. Persistence rates for children with ADHD-PI may be lower (as suggested by Hart et al., 1995). However, the limited data collected from children with ADHD-PI followed long term (Hinshaw et al., 2006, 2012), as well as the observation that inattentive symptoms seem to more stable than hyperactive–impulsive ones, suggest that this may not be the case. Moreover, although rates of diagnostic persistence are high—and continued ADHD symptoms and impairment over the long term are expected—there are exceptions. A minority recover or at least improve substantially over time. This may be encouraging news for parents and clinicians.

The latter finding prompts questions about what predicts remission versus persistence. Biederman and colleagues (Biederman, Faraone, Milberger, & Guite, 1996; Biederman, Petty, Evans, et al., 2010) have suggested that increased symptom severity, psychiatric comorbidities, greater impairment, and family history of ADHD are all factors that moderate outcome in the sense that they are associated with increased likelihood of ADHD persistence across time. As noted below, the majority of investigations assessing moderators of outcome have concerned prediction of functional impairments (e.g., Barkley & Fischer 2010, 2011; Fischer, Barkley, Fletcher, & Smallish, 1993a; Miller & Hinshaw, 2010), substance abuse outcomes (e.g., DeSanctis et al., 2008; Marshal, Molina, & Pelham, 2003), or externalizing behavior such as criminality (e.g., Babinski, Hartsough, & Lambert, 1999; DeSanctis, Nomura, Newcorn, & Halperin, 2012; Satterfield & Schell, 1997; Satterfield, Swanson, Schell, & Lee, 1994; Satterfield et al., 2007; Sibley et al., 2011). Continued investigation of which children with ADHD are more or less likely to show persistence versus remission is an important additional step in our understanding of the developmental progression of ADHD. It is also necessary for targeting intervention efforts toward those most likely to show persistence and therefore need treatment.

Expected Outcomes and Moderators of Outcome

Beyond persistence of ADHD symptoms and diagnoses, what are the expected outcomes for children diagnosed with ADHD? The outcome domains most often investigated include externalizing problems (conduct disorder [CD], oppositional defiant disorder [ODD], criminality, antisocial personality disorder [ASPD]), internalizing problems (anxiety and depression), substance use and abuse or dependence, academic functioning, occupational functioning, driving problems, social functioning, and overall impairment. A plethora of longitudinal research over the past 25 years shows that childhood ADHD is a statistical, but not necessarily causal, risk factor for poor outcome in all of these domains. However, there are important caveats. As noted earlier, symptoms and impairment at follow-up depend on who is queried. In many instances, children with

ADHD do not report symptoms and impairment when their parents or others who know them well clearly do. In addition, whether outcome is compromised depends on how it is conceptualized and measured. For example, in the substance use and abuse domain, whether or not ADHD is a risk factor depends on the substance in question and whether the dependent variable reflects trying the substance, frequency of using the substance, or developing a diagnosable substance-related disorder.

When ADHD does clearly portend poor outcome, one must ask whether the finding is related to ADHD specifically or to some correlate of ADHD, such as psychiatric comorbidity at baseline. In particular, when adolescent or adult psychiatric comorbidity is found to be more common among children with ADHD than among controls, is this because baseline comorbidities are simply continuing later in life? When prospective associations between ADHD status and adolescent or adult outcome are statistically significant, are the children with ADHD doing poorly in an absolute and clinically significant sense, or are they simply doing somewhat more poorly than controls? Crucially, when ADHD does predict a poor outcome, it means that the chance of a poor outcome is heightened but not inevitable. In other words, (1) not everyone with ADHD will show the negative outcome, and (2) ADHD itself may not be causal; comorbidities or associated impairments at baseline may instead be the actual cause. Furthermore, the range of outcomes within a given domain, even among children all diagnosed with ADHD, is wide. Therefore, we must ask which baseline variables predict or moderate—or which intervening variables mediate or explain—the particular outcome in question.[1]

Externalizing Domain

Adolescent and adult outcomes in the externalizing domain have been well researched, with a clear take-home message: Childhood ADHD is a risk factor for later externalizing problems at all ages for both boys and girls. Risk for 1-year prevalence of antisocial outcomes (ODD, CD, ASP) was increased in girls at adolescence, and the difference between ADHD and control groups remained significant after researchers controlled for non-antisocial baseline psychopathology (Biederman et al., 2006a), with very similar results obtained for boys (Biederman, Faraone, Milberger, Curtis, et al., 1996). According to Hinshaw and colleagues (2006), parent-reported rates of ODD and CD diagnoses, and the level of externalizing problems (according to both parents and teachers), were higher for girls with ADHD-C or ADHD-PI relative to controls, and these differences survived statistical adjustment for child age and IQ, socioeconomic status (SES), and baseline anxiety and depression (but not baseline CD/ODD), with effect sizes moderate to very large. However, the variety of self-reported delinquent acts was not different for girls with ADHD and controls. Also using data from this sample, Owens, Hinshaw, Lee, and Lahey (2009) showed that 58% of those girls with childhood ADHD had poor adjustment in the externalizing domain during adolescence (as measured by parent and teacher report of ODD symptoms) compared with only 9% of the control girls. Using parallel methodology in a separate, mostly male sample, Lee, Lahey, Owens, and Hinshaw (2008) showed 66% of the ADHD group to be poorly adjusted in the externalizing domain during adolescence versus 21% of comparison children. Using the PALS sample, Babinski, Pelham, Molina, Gnagy, and colleagues (2011) also found higher levels of delinquency severity by young adulthood, with a moderate effect size (d = 0.54), although group differences were not adjusted for baseline demographics or psychopathology.

In line with these findings, among their boys with childhood ADHD (and in contrast to comparison participants), Gittelman and colleagues (1985) found increased rates of CD at adolescence, with those demonstrating persistent ADHD most likely to show antisocial outcomes. Mannuzza, Klein, Abikoff, and Moulton (2004) reported increased rates of CD during adolescence among children with ADHD. The risk for antisocial outcomes persisted when they statistically controlled for age and SES (with baseline antisocial behavior controlled by design); it was more likely when ADHD did not remit. Still, the risk did not depend on the continued presence of ADHD; a childhood diagnosis of ADHD without comorbid conduct problems was enough to increase risk. Also using this sample, Mannuzza, Klein, Konig, and Giampino (1989) reported more arrests, convictions, and incarcerations by late adolescence among the boys with ADHD versus comparison boys. Similarly, Barkley and colleagues (1990) reported that at adolescent follow-up, 59% of their participants with childhood ADHD had CD or ODD. The participants with childhood ADHD also exhibited more criminal behavior. In a more recent report, Barkley, Fischer, Smallish, and Fletcher (2004) indicated that the increased adolescent antisocial behavior

among children with ADHD is primarily drug-related, especially among those with a CD diagnosis.

Virtually every other report of adolescent outcome among children with ADHD reveals similar findings. Satterfield and colleagues used official arrest records to document the long-term criminality outcomes of their samples of hyperactive boys, most of whom had comorbid externalizing problems at baseline. At follow-up in adolescence, these hyperactive boys showed higher rates of felony offenses, but not minor offenses, than controls. Using the PALS sample followed to age 17, Sibley and colleagues (2011) found all children with ADHD to be at risk for earlier age of delinquency and increased severity and variety of delinquency, but the children with ADHD and CD had worse delinquency outcomes than the children with ADHD and ODD or ADHD alone. Furthermore, there were no differences between children with and without ADHD in mild offending prevalence during adolescence. Lahey and colleagues (2007) found higher levels of CD among adolescent boys and girls, with differences maintained when controlling for baseline symptoms of CD. Extending such findings from primarily clinic-referred children, in a community sample, Bussing and colleagues (2010) found increased risks for CD, ODD, and juvenile delinquency among both girls and boys with ADHD followed into adolescence. Similarly, Yoshimasu and colleagues (2012) used archived data to examine a large birth cohort of all children born between 1976 and 1982 in Rochester, MN. Through a multigated screening procedure, 379 children were identified as having ADHD according to DSM-IV (American Psychiatric Association, 1994) criteria. Their increased risk for adolescent CD or ODD was large (hazard ratio = 9.54) and significant, with the risk equivalent for girls and boys. This finding held when the researchers statistically adjusted for maternal education, but they did not control for baseline psychiatric comorbidities.

Adult outcome of children with ADHD in the externalizing domain is highly similar to what is reported among adolescents. Hinshaw and colleagues (2012) found increased rates of parent-reported CD and ODD diagnoses, as well as higher levels of parent- and self-reported externalizing problems among girls with ADHD followed into young adulthood. Yet the variety of self-reported delinquent acts was not different across ADHD and comparison participants. All differences survived control of baseline demographics, internalizing (but not externalizing) comorbidities, and child IQ. Similarly, Biederman, Petty, Evans, and colleagues (2010) documented increased risk for self-reported CD, ODD, and ASP in their female sample. These ADHD versus comparison differences were obtained using both lifetime and one-year prevalence rates and survived control of baseline demographics and all types of psychopathology. Results for boys followed into young adulthood were quite similar, except that the significant difference in one-year prevalence of antisocial disorders did not survive statistical control for baseline antisocial disorders (Biederman et al., 2006b). Across these highly comparable samples of girls and boys with ADHD, the hazard ratio for lifetime antisocial disorders was 7.2 among girls (Biederman, Petty, Evans, et al., 2010) and 5.9 among boys (Biederman et al., 2006b).

By young adult follow-up, hyperactive boys followed by Satterfield and colleagues had earlier arrests, more arrests for violent crimes, and higher rates of arrest and incarceration than control boys, but the number of children who subsequently were arrested only once did not differ across the groups (Satterfield & Schell, 1997). Importantly, SES, IQ, and family type were not controlled. However, at the follow-up in midadulthood, those with childhood hyperactivity showed greater arrest rates (44 vs. 15%), conviction rates (29 vs. 8%), and incarceration rates (26 vs. 8%) than controls, with IQ and SES covaried. Importantly, most children with ADHD did not go on to develop criminal records, and no child with ADHD alone became a chronic offender. Thus, a history of aggression or antisocial behavior appears crucial for the prediction. In other words, the risk for later criminality was not due to childhood ADHD per se but appeared to result from ADHD's increasing the risk for subsequent antisocial psychopathology (CD/ODD), which increased risk for adult offending.

Mannuzza, Klein, Bessler, Malloy, and LaPadula (1998) found higher rates of ASP during early adulthood among their ADHD versus control participants (12 vs. 3%). Well into adulthood, Klein and colleagues (2012) found more ASP (16 vs. 0%) among their participants followed to age 41. At age 38, official records showed that in contrast to comparison boys, those with ADHD had more arrests (47 vs. 24%), convictions (42 vs. 14%), incarcerations (15 vs. 1%), felonies (14 vs. 1%), and violent offenses (19 vs. 3%). Importantly, these differences were entirely accounted for by ASP or substance use disorder in adolescence, which were predicted by earlier ADHD (Mannuzza, Klein, & Moulton, 2008). As in the Satterfield sample, the majority of children with ADHD did not become criminals, and the large majority did not develop ASP. Similarly, Wymbs and

colleagues (2012) found that intimate partner aggression in adults who had ADHD as children was partially accounted for by the development of ASP.

In summary, during adolescence and adulthood according to parent and official reports, and sometimes self-report, a childhood diagnosis of ADHD is statistically associated with increased risk for externalizing-related psychiatric problems (CD, ODD, ASP), as well as antisocial and criminal behavior. Often but not always, the increased risk for antisocial outcomes during adolescence is related to the presence of comorbid externalizing problems during childhood. Antisocial outcomes during adulthood seem to depend almost exclusively on the development or presence of externalizing problems during adolescence and not directly to the presence of ADHD during childhood. Although Gaub and Carlson (1997) reported that rates of co-occurring externalizing problems are lower in girls than in boys (more on this issue, below), the risks for developing externalizing problems appear equivalent across gender. In other words, girls in general are less likely to exhibit externalizing behavior than boys, regardless of ADHD status, but when girls with ADHD are compared to girls without ADHD, their risk of developing externalizing problems is equal to that of boys with ADHD compared to boys without ADHD. Despite increased risk, many, if not most, children with ADHD do *not* go onto to develop externalizing problems or engage in criminal behavior. Finally, because the vast majority of children studied thus far had or would have met criteria for ADHD-C, it is not known whether or not ADHD-PI confers the same longitudinal risk. Among females, Hinshaw and colleagues (2006, 2012) did find that that on five of seven measures across adolescence and young adulthood, the risk for externalizing outcomes was equivalent across ADHD subtype. Exceptions were greater frequency of parent-rated CD diagnosis during adolescence, and higher levels of parent-rated externalizing problems during young adulthood among girls with ADHD-C than among those with ADHD-PI.

Internalizing Domain

Although many have reported increased risks for later internalizing problems among children with ADHD, the evidence for this association is not as robust or uniform as it is for the relation between childhood ADHD and later externalizing problems. The caveats discussed earlier (e.g., considering whether ADHD is a statistical or causal risk factor, considering whether internalizing problems develop over time or are a continuation of previously existing psychopathology) also apply to the assessment of internalizing outcomes among children with ADHD. However, as noted below, gender may be a relevant factor here.

Most recently, Biederman, Petty, Fried, and colleagues (2010) reported increased risk of mood (hazard ratio = 6.8) and anxiety (hazard ratio = 2.1) disorders among girls with ADHD followed to young adulthood, with differences between girls with ADHD and comparison girls surviving control of baseline demographics and all types of psychopathology. At the adolescent follow-up of the same sample, 1-year prevalence of major psychopathology—defined as depression with severe impairment or multiple anxiety disorders or bipolar disorder or psychosis—was increased among girls with childhood diagnoses of ADHD, and differences survived statistical control of baseline psychopathology. Differences in the presence of less severe but clinically significant forms of depression and anxiety were not tested. Although the elevated risk for severe internalizing problems was similar in Biederman's sample of boys during adolescence (Biederman, Faraone, Milberger, Curtis, et al., 1996), it did not obtain in his sample of boys during young adulthood (see below). Lee and colleagues (2008) demonstrated that 36% of children with ADHD are poorly adjusted in the internalizing domain at adolescence compared to only 11% of controls (a significant difference). Likewise, using a parallel methodology with the BGALS sample, Owens and colleagues (2009) found 51% of girls with childhood ADHD to have relatively high levels of parent-reported symptoms of depression and anxiety during adolescence compared to 15% of the comparison girls. Chronis-Tuscano and colleagues (2010) reported increased risk through age 18 years for major depressive disorder or dysthymia (hazard ratio = 4.32) among 125 participants with early childhood ADHD, especially among the girls. Lahey and colleagues (2007) found that the higher rates of adolescent major depression and anxiety disorders for boys and girls with early diagnoses of ADHD remained significant when they controlled for baseline internalizing symptoms. Fischer, Barkley, Smallish, and Fletcher (2002) found that children with ADHD were at high risk for major depressive disorder as young adults, especially those who developed comorbid CD during adolescence. Babinski, Pelman, Molina, Waschbusch, and colleagues (2011) found increased levels of parent-reported, but not self-reported, internalizing problems for men and women. In their community-

identified sample of children with and without ADHD, Bussing and colleagues (2010) found increased risk for anxiety and depressive disorders by adolescence. All girls, with and without ADHD, were at greater risk for anxiety and depression than boys. Yet Yoshimasu and colleagues (2012) reported increased risk for adolescent mood (hazard ratio = 3.67) and anxiety (hazard ratio = 2.94) disorders to be equivalent across genders. These results survived statistical adjustment for maternal education, but baseline comorbidities were not covaried.

Others have failed to find an association between childhood ADHD and later internalizing problems. In none of the reports from Mannuzza and Klein were boys with ADHD found to be at current or lifetime risk for later internalizing problems at any age (Klein et al., 2012; Mannuzza et al., 1991, 1993, 1998). At the follow-up in adolescence of the Hinshaw and colleagues (2006) sample, only some measures of internalizing problems differentiated between girls with ADHD and comparison girls. There were no differences on parent-reported comorbid anxiety or depression; teacher-rated internalizing problems and self-reported depressive symptoms were no longer significant after statistical adjustment of child IQ and age, SES, and baseline ODD/CD. Only parent-reported internalizing symptoms differentiated the groups, with both ADHD-C and ADHD-PI subtypes showing far higher levels than comparisons (effect sizes close to 0.9). At follow-up in young adulthood (Hinshaw et al., 2012), girls with ADHD and comparison girls did not self-report differences in internalizing problems; parent-reported differences in internalizing comorbidities and levels of internalizing symptoms did not survive control of baseline demographic variables and comorbidities. Similarly, Babinski, Pelman, Molina, Waschbusch, and colleagues (2011) did not find self-reported differences in internalizing symptoms for adult men or women. Although Biederman and colleagues (2006b) did find that boys, by young adulthood, were at increased lifetime (but not 1-year) risk for anxiety disorders, the differences between boys with ADHD and comparison boys did not remain after controlling for baseline demographics and comorbidities. These investigators also found increased risk for lifetime and 1-year major psychopathology (including depression), but only lifetime risk remained significant after statistical controls were applied.

In summary, childhood ADHD does appear to increase risk for later internalizing problems, especially during adolescence, but evidence is mixed, and associations often do not hold once baseline demographics and psychiatric comorbidities are considered. On balance, the link between childhood ADHD and later internalizing problems seems to be somewhat stronger for girls than for boys, but this association is not unique to children with ADHD. By adolescence, girls are at higher risk for internalizing problems than are boys, regardless of ADHD status (Hinshaw, 2009).

Substance Use and Abuse

Substance use and abuse are widely studied outcomes of childhood ADHD. Generally, the empirical evidence suggests that ADHD is a risk factor in this domain. However, not all specific outcomes receive consistent support, and some studies fail to find a link between childhood ADHD and later substance use-related outcomes. Results vary depending on the substance studied and whether the variable at hand is "ever used," age at first use, frequency of use, rate of progression to abuse or dependence, or the presence or absence of a substance use disorder. Even across significant findings for similar outcome variables, effect sizes vary.

NICOTINE

Typically, cigarette smoking frequency is used as a measure of nicotine use and dependence. Molina and colleagues (2013), Barkley, Fischer, Edelbrock, and Smallish (1990), Elkins, McGue, and Iacono (2007), Milberger, Biederman, Faraone, Chen, and Jones (1997), and Molina and Pelham (2003) each demonstrated that childhood ADHD is positively associated with cigarette use during adolescence. The latter two studies also documented earlier initiation of smoking among children with ADHD, and Elkins and colleagues demonstrated that ADHD symptoms predict early onset of smoking, with consistent results for girls and boys. Flory, Malone, and Lamis, (2011) found that childhood ADHD symptoms predict cigarette smoking frequency in 10th grade for both boys and girls, controlling for race, SES, and early externalizing problems. However, Bussing and colleagues (2010) reported no differences in tobacco use among community-identified girls and boys at age 16. Similarly, Babinski, Pelman, Molina, Gnagy, and colleagues (2011) did not find that girls with ADHD smoke more frequently than comparison subjects during adolescence or young adulthood. Furthermore, some investigators document a significant role of comorbid conduct or externalizing problems in the genesis of frequent cigarette use

(Burke, Loeber, & Lahey, 2001). For example, according to Barkley and colleagues, 32% of boys with childhood hyperactivity alone smoked during adolescence, whereas 65% of those with hyperactivity and conduct problems did so. Milberger and colleagues found that smoking rates were highest among children with ADHD and comorbid baseline psychopathology. Similarly, Molina and Pelham found that persistent ADHD, especially in concert with CD, was much more strongly associated with later cigarette use than was nonpersistent ADHD.

ALCOHOL

The evidence for increased later use of alcohol among children with ADHD is not as compelling as it is for other psychoactive substances (Charach, Yeung, Climans, & Lillie, 2011; Lee, Humphreys, Flory, Liu, & Glass, 2011), perhaps because alcohol use and abuse occur at high rates among adolescents and adults in general. Molina, Pelham, Cheong, Marshal, and Gnagy (2012) found no increased risk for alcohol use frequency at age 17 among children with ADHD, although ADHD plus low parental monitoring did predict alcohol use. Babinski, Pelham, Molina, Gnagy, and colleagues (2011) found no self-reported problems with binge drinking in adolescence or young adulthood among women. Barkley and colleagues (1990) did not find increased rates of alcohol use among boys during adolescence, based on both mother and child report. Gittelman and colleagues (1985) and Mannuzza and colleagues (1991) found no increased risk for alcohol use disorder in boys with ADHD followed into adolescence; their results were paralleled by Klein and colleagues (2012) when the same boys were followed to mean age 41. Elkins and colleagues (2007) did not find that ADHD predicts alcohol use during adolescence, although symptoms of hyperactivity and impulsivity did predict age of initiation (controlling for CD), with results consistent across gender. Molina and Pelham (2003) found no increased risk for trying alcohol or for alcohol disorders in their follow-up of children with ADHD, although there was an association with increased frequency of drunkenness, with increased risk for children with persistent ADHD or with comorbid CD. Molina and colleagues (2013) found increased rates of ever using alcohol among children with ADHD-C in the MTA followed into adolescence, but rates of alcohol disorders were not different between children with ADHD and comparison children.

ILLICIT SUBSTANCES

In contrast to the results regarding alcohol use and abuse, there is more evidence suggesting an increased risk for later use and abuse of illicit psychoactive substances among children with ADHD (Charach et al., 2011), although, again, the evidence is not uniform. Molina and Pelham (2003) reported increased frequency and earlier initiation of illicit drug use among children with ADHD followed into adolescence, with large effects, especially for children with persistent ADHD. Children with ADHD also used marijuana more frequently than children without ADHD, although the risk for ever trying marijuana was not elevated. Molina and colleagues (2013), in the most definitive study to date, reported more repeat use of marijuana by adolescence among children in the MTA (all with ADHD-C) compared to controls. Mannuzza and colleagues (1991) reported increased drug use at adolescence in boys with ADHD. Although the differences were not significant after they controlled for SES, the effect size was very large. Among the same boys followed to mean age 41, Klein and colleagues (2012) found increased risk for illicit drug use disorders. Similarly, Elkins and colleagues (2007) found childhood ADHD to predict illicit drug use, but not marijuana use, during adolescence, with findings consistent across gender and moderated by CD. Controlling for conduct problems, race, and SES, Malone, Van Eck, Flory, and Lamis (2010) found that ADHD symptom trajectory predicted onset of illicit drug use. Babinski, Pelham, Molina, Waschbusch, and colleagues (2011) found increased parent-reported use of marijuana among boys, but not girls, with ADHD followed to young adulthood. However, they found no self-reported differences in marijuana use at young adulthood for either sex. Barkley and colleagues (1990) found no evidence of illicit substance use differences among boys with and without hyperactivity. Bussing and colleagues (2010) showed no differences in reports of ever having used marijuana for girls or boys, with and without ADHD, but the sample was followed only until age 16, and differences might emerge later in development.

ANY PSYCHOACTIVE SUBSTANCE

Rather than computing risk for use of particular substances, a number of researchers have analyzed composite variables indexing use or abuse of a range of psychoactive substances. Not surprisingly, given the

varying patterns reported earlier, the empirical literature is mixed regarding whether childhood ADHD is associated with generalized risk for later substance use or abuse. Biederman and colleagues (2006a; Biederman, Petty, Fried, et al., 2010) did find that girls with ADHD have higher lifetime and 1-year prevalence of substance use disorders of any kind than comparison girls during both adolescence and young adulthood. However, differences during adolescence did not survive control of baseline psychopathology (thus, they were not specific to ADHD), and neither did the 1-year prevalence differences during early adulthood. Among boys with and without ADHD, Biederman's team did not find any differences in substance use disorders during adolescence (Biederman, Faraone, Milberger, Curtis, et al., 1996) and the differences found during young adulthood (Biederman et al., 2006b) did not hold with statistical adjustment for baseline psychopathology. However, during early adolescence, Biederman and colleagues (1997) found that progression from use to abuse, and from abuse to dependence, was accelerated in boys with ADHD. Also, psychoactive substance use disorders were related to the presence of CD but not ODD.

Molina and colleagues (2013) found elevated rates of any substance use (beyond a single drink or cigarette), any substance use disorder, and number of substances used among the MTA children with ADHD-C followed into adolescence. Gittelman and colleagues (1985) also found increased rates of substance use disorder at adolescence in boys with ADHD, with rates especially high when ADHD was persistent and when CD was comorbid. Similar findings emerged when this sample was followed into adulthood (Mannuzza et al., 1993, 1998). For example, during adulthood, 16% of children with ADHD had a drug abuse disorder compared to 4% of controls. Note that although the odds ratio was large (4.6), the absolute likelihood of developing a drug use disorder in this sample, in which participants with comorbid antisocial problems were excluded, was small. Results held with statistical control of SES.

Among community samples, risks for illicit drug use and abuse are documented. In August and colleagues (2006), only children with ADHD plus an externalizing disorder were at risk; individuals with ADHD only were not. In Yoshimasu and colleagues (2012), the hazard ratio, with controls for maternal education, was 4.03 and equivalent between genders. Of note, Hinshaw and colleagues (2006, 2012) reported no differences in overall substance use severity (a composite measure of variety of substances used and frequency of use) among girls with ADHD followed to adolescence and to adulthood.

SUMMARY

The literature regarding risk for substance use and abuse posed by a childhood ADHD diagnosis is so varied as to almost preclude a summary. At a general level, childhood ADHD does pose some risk for substance use problems during adolescence and possibly during adulthood. In particular, early age of initiation and increased use and abuse of nicotine and illicit substances (but not necessarily marijuana) are predictable from childhood ADHD status. However, empirical support for the association between childhood ADHD and later use and abuse of alcohol is weak. Gender does not reliably moderate any longitudinal, predictive associations. Sometimes associations between ADHD and later substance use are found for girls and sometimes they are not, as is also true for boys. It may be that results depend specifically (1) on the type of substance studied and/or (2) how use or abuse is operationalized and measured. It may also be that there is wide variability in outcome, so that ADHD is associated with risk for later substance use and abuse for some youth but not others (i.e., there is heterogeneity of risk). Thus, identifying moderators of outcome (i.e., factors on which the association between ADHD and later substance use and abuse depends) may be more fruitful than continued assessment of whether at a group-level ADHD is associated with later substance use or abuse.

One such moderator (or mediator, if measured during longitudinal assessments) of the relation between childhood ADHD and later substance use and abuse is comorbid or subsequent psychopathology, especially CD (August et al., 2006; Barkley et al., 2004; Burke et al., 2001; Chilcoat & Breslau, 1999; Looby, 2008; Mannuzza & Klein, 2000; Molina & Pelham, 2003; Nigg, 2013). Realmuto and colleagues (2009) found that risk for later drug was heightened only among those who during childhood had comorbid externalizing problems, even if those problems remitted by adolescence. However, the risk incurred by CD does not absolve the risk for substance use problems associated with earlier ADHD because in many cases ADHD leads to CD. In other words, CD may be the proximal risk factor and statistically accounts for the association between ADHD and substance use problems, but ADHD may be the original, distal risk factor (Molina & Pelham 2014).

Others factors, including maltreatment (DeSanctis et al., 2008) and deviant peer association (Marshal et al., 2003), may moderate or mediate the link between childhood ADHD and later substance use and abuse. Recent theoretical models articulating hypothesized pathways to substance use and abuse enumerate these factors and their putative mediating/moderating roles (Molina & Pelham, 2014). Substance use and abuse among children with ADHD is not as widespread as commonly believed. For example, using Biederman's samples of girls and boys, Wilens and colleagues (2011) estimated the hazard ratio for any substance use disorder to be 1.47. A childhood diagnosis of ADHD, if uncomplicated by concurrent or subsequent externalizing problems or other psychopathology, does not pose a notable risk for most later substance use problems among girls or boys, with the probable exception of increased nicotine use.

A discussion of risk for substance abuse in children with ADHD would not be complete without a comment on whether stimulant treatment moderates this risk. First of all, when observational studies show any association between an intervention (in this case, stimulant medication) and either a positive or negative outcome, the association could be related to the intervention selection bias (Larzelere, Kuhn, & Johnson, 2004). That is, factors driving who decides to seek or accept medication and who does not may be substantially responsible for the outcome, rather than the intervention per se. If children who take stimulants appear to have poor outcomes, it may be because their ADHD was more severe, driving parents to seek medical treatment. Alternatively, more positive outcomes may be related to parental education or income, concern for the child's welfare, or access to other interventions rather than to the medication per se.

Some researchers fear that treating ADHD with stimulant medication might increase risk for psychoactive substance use and abuse as children grow. They postulate that early exposure increases sensitization to psychoactive drugs (Lambert, 2002). Even some who recognize that treatment with stimulants is clearly effective and indicated for many children nevertheless express concern about later increased drug use (Vitiello, 2001). However, at this point, little evidence supports the sensitization hypothesis. Most studies report no association between stimulant medication use during childhood and later substance use or abuse (e.g., Biederman et al., 2008; Chilcoat & Breslau, 1999; Winters et al., 2011). Molina and colleagues (2013) showed no increased—or decreased—risk for adolescent substance use or abuse/dependence associated with stimulant medication treatment in the MTA sample. This conclusion was also reached by Humphreys, Eng, and Lee (2013) in their meta-analysis of studies examining the relation between stimulant medication use and later psychoactive substance use.

Furthermore, some find stimulant treatment to be negatively associated with risk for substance use *disorders*, although this relation has not been tested or established for more general substance use outcomes. For example, Biederman (2003) found risk of substance use disorder to be three to four times more likely in children with ADHD not treated with stimulants than in those treated with stimulants. Katusic and colleagues (2005) also found stimulant therapy to be associated with reduced risk for later substance abuse among boys, controlling for SES. Mannuzza, Klein, Truong, and colleagues (2008) found that earlier use of stimulant therapy protects against substance disorder outcomes.

There is also evidence that stimulant treatment is associated with reduced risk for other undesirable outcomes, including major depression during adolescence (Daviss, Birmaher, Diler, & Mintz, 2008) and psychopathology during young adulthood (Biederman, Monuteaux, Spencer, Wilens, & Faraone, 2009). It is also associated with higher achievement scores and high school grade point average (GPA), when researchers control for initial ADHD symptom severity (Powers, Marks, Miller, Newcorn, & Halperin, 2008). Barbaresi, Katusic, Colligan, Weaver, and Jacobsen (2007) also found that stimulant treatment is associated with positive long-term school outcomes (decreased absenteeism, fewer grade retentions, and higher reading scores) among children with ADHD, as did Scheffler and colleagues (2009), who found that initial stimulant use was associated with increases in reading and math scores among a representative national sample of elementary schoolchildren with ADHD. In all, the bulk of the empirical evidence suggests that stimulant medication does not increase or decrease risk for later substance use and abuse. However, some evidence suggests it may function as a protective factor by reducing risks for poor outcome by ameliorating the long-term effects of childhood ADHD.

Academic Functioning

Not surprisingly, children who by definition have trouble paying attention are likely to have problems learning in a traditional school environment (see Chapters 6 and 12), but the picture across development bears

closer scrutiny. Hinshaw and colleagues (2006) reported lower standardized achievement scores in math and reading, as well as lower teacher ratings of academic performance among girls with ADHD followed into adolescence, compared to controls, with deficits equivalent for girls with ADHD-C and ADHD-PI. However, only the differences in math achievement withheld control for baseline demographics, child IQ, and comorbidities. At young adulthood follow-up, math and reading scores were also equally compromised for girls with ADHD-C and ADHD-PI, and the total years of education obtained was lower for girls with ADHD. Yet only the reading score deficit survived the same statistical controls. Using the same sample, Owens and colleagues (2009) found that 35% of the girls with ADHD have both reading and math achievement scores that are one or more standard deviations below the mean during adolescence, compared to only 4% of controls. Lee and colleagues (2008) demonstrated highly similar results (18 vs. 6%) in a sample of mostly boys.

Hyperactive boys followed to adolescence (Fischer et al., 1990) had lower scores than comparison boys on standardized tests of spelling, reading, and arithmetic when researchers controlled for child IQ and maternal education. They also had more school-related disciplinary problems (e.g., 46% had been suspended and 11% expelled vs. 15 and 2%, respectively, for controls), but these differences were primarily accounted for by comorbid CD (Barkley et al., 1990). Among the grown-up hyperactive group, 32% did not graduate from high school, compared to 0% of the control participants, with differences remaining when researchers controlled for IQ (Barkley, Fischer, Smallish, & Fletcher, 2006). Mannuzza, Klein, Bessler, Malloy, and Hynes (1997) also documented poor educational outcomes over the long-term for boys with ADHD, who in young adulthood had completed two fewer years of education than controls, a difference that held when researchers controlled for IQ. Mannuzza and colleagues (1993) documented the same: 12.1 years versus 14.6 years of education for the participants with ADHD and controls, respectively. Of the boys with ADHD, 20% did not graduate from high school, compared to 2% of the controls. Accordingly, at their midadulthood follow-up, Klein and colleagues (2012) found that 17% of participants with ADHD had not graduated from high school versus 1% of controls. In terms of college graduation, the rate for participants with ADHD was 21%, and that for those without ADHD was 47% (without controls for IQ or SES). As in the Barkley and colleagues (2006) sample, poorer educational outcomes were related to ASP. Babinski, Pelham, Molina, Gnagy, and colleagues (2011) documented in women with a history of childhood ADHD greater academic impairment (according to parent- but not self-ratings); more frequent grade retention and need of academic support; and lower spelling, math, and reading achievement scores. These differences, however, did not withstand adjustment for IQ (although one could argue that covarying IQ is an example of overcontrol; see Miller & Chapman, 2001). Utilizing PALS participants, Kuriyan and colleagues (2013) found children (mostly boys) with ADHD to be worse off on almost all educational variables assessed, including achievement scores, disciplinary problems at school, and level of post–high school education. However, they noted that results for individual children were wide ranging. Children with ADHD who did not have academic or disciplinary problems at school were not at risk for lower educational attainment. Extending the findings to a community sample, Bussing and colleagues (2010) found increased use of special services, more grade retention, lower graduation rates, and lower test scores among children with ADHD, with no differences between girls and boys.

In summary, childhood ADHD predicts significant academic and educational attainment problems for girls and boys during adolescence and young adulthood, with some attenuation in findings when IQ is controlled. Furthermore, two studies suggest that the risk associated with childhood ADHD is contingent on the presence of a learning disability (Faraone, Biederman, Monuteaux, Doyle, & Seidman, 2001) or other features sometimes associated with ADHD (lower IQ, reading ability, and SES; Trampush, Miller, Newcorn, & Halperin, 2009). Thus, although childhood ADHD is associated with risk for later academic and educational problems, it is likely to be part of a multifaceted causal pathway.

Occupational Functioning

The relatively few studies that have followed children with ADHD through adulthood have all reported that they have more employment problems than do children without ADHD. Still, deficits do not obtain on every measure of employment, and participants do not report occupational impairment as often as their parents. Barkley and Fischer (2011) reported lower Hollingshead Job Index scores and fewer hours worked among participants with ADHD by adulthood; those with persistent ADHD also had lower work quality, more trouble on the job (i.e., being fired or disciplined), more hostility

with supervisors, and more job turnover compared to controls and to those with nonpersistent ADHD (Barkley & Fischer, 2011). Barkley, Fischer, Smallish, and Fletcher (2006) reported that boys with both persistent and nonpersistent hyperactivity were more often fired from jobs and had lower job performance, and were more likely to have experienced homelessness (Fischer & Barkley, 2006) by young adulthood than were boys who were not hyperactive as children. Similarly, Mannuzza and colleagues (1993, 1997) found that their participants with ADHD had lower occupational rank in early adulthood but were not more likely to be unemployed than participants without childhood ADHD. Results were partly, but not exclusively, determined by concomitant ASP. Klein and colleagues (2012) found lower employment rates, salaries, and SES among their boys with ADHD at age 41, but effects dissipated after they controlled for ongoing mental disorder. Babinski, Pelham, Molina, Gnagy, and colleagues (2011) found parent-reported, but not self-reported, problems with job performance among women with childhood ADHD. Both men and women self-reported lower SES (Babinski, Pelham, Molina, Waschbusch, et al., 2011). Kuriyan and colleagues (2013) reported lower occupational status, lower maximum hourly salary, and more job loss among those with childhood ADHD versus those without.

Clearly, childhood ADHD signals increased risk for poor occupational functioning and attainment in adulthood. At this point, however, the mechanisms of the association are not completely illuminated. It may be that continuing symptoms of ADHD have a negative impact on success in the workplace, but is also may be that the link is mediated by lower educational attainment or the development of other mental health problems prior to adulthood, as suggested by Klein and colleagues (2012) and Kuriyan and colleagues (2013). It is also unknown whether employment problems resulting from childhood ADHD are present in women to the same extent as in men, although the small-sample Babinski, Pelham, Molina, Waschbusch, and colleagues (2011) investigation suggests that this may be the case.

Driving Problems

Investigators have begun to examine the driving behavior and consequences associated with a childhood diagnosis of ADHD. Fischer, Barkley, Smallish, and Fletcher (2007) have carefully studied these outcomes using official records, self-report, actual driving tests, and a driving simulator. When hyperactive children become drivers, their performance is worse on almost every measure of driving behavior and related outcomes measured during young adulthood, including more traffic citations (e.g., reckless driving and driving without a license), greater frequency of license suspensions, more damage during crashes, less safe driving behavior, more impulsive errors, and slower reaction times. Similarly, Olazagasti and colleagues (2013) reported more at-fault accidents and more accidents involving injury among their ADHD participants followed to age 41. Hinshaw and colleagues (2012) did not find differences in driving offenses and violations at young adulthood follow-up when based on self-report, although significant differences did emerge based on parent report. Although the literature base thus far is small, it is reasonable to conclude that childhood ADHD is a significant risk factor for unsafe and illegal driving behavior (see Chapters 11 and 29).

Social Skills and Relationships

During adolescent follow-up, Hinshaw and colleagues (2006) found significant teacher- and parent-reported social skills deficits among girls with ADHD-C and ADHD-PI, with large effects that survived stringent statistical controls. Teachers rated girls with ADHD-C, but not those with ADHD-PI, as less preferred by peers than those without ADHD. There was also more parent-rated peer conflict for all girls with ADHD, and the effect size for controls versus those with ADHD-C was larger than for those with ADHD-PI (1.07 vs. 0.62). These differences survived statistical control of child age and IQ, SES, and importantly, all baseline comorbidities. In the same sample, Owens and colleagues (2009) found 60% of girls with ADHD to be poorly adjusted in the social skills domain during adolescence according to parents and teachers, versus only 17% of the comparison girls. Lee and colleagues (2008) found 63% of adolescents with childhood ADHD to be poorly adjusted in the social skills domain versus 29% of the control children. In the peer domain, Owens and colleagues found 40% of girls with ADHD to be unpreferred according to teachers versus only 12% of the control girls. The comparable figures according to Lee and colleagues were 49% (ADHD) and 16% (controls).

In adulthood, Barkley and colleagues (2006) found that boys who had been hyperactive as children had fewer closer friends, trouble keeping friends, and according to parents, more social problems than compari-

son boys. Fischer and Barkley (2006) found no differences in number of social relationships in young adults with hyperactivity, but relationship quality was poorer (e.g., fewer close friends, more arguing, trouble keeping friends). Klein (2012) reported more divorces by age 41 among adults who had had childhood ADHD. Finally, on five of seven measures, parents reported that their adult daughters had problems with family and friends (Babinski, Pelham, Molina, Gnagy, et al., 2011). However, paralleling other investigations, the women self-reported relationship problems on only two of 10 measures (romantic relationships and conflict with mother). Clearly, childhood ADHD portends social and relationship problems later in life, although the database supporting this conclusion is not large.

Overall Impairment

Two studies, Lee and colleagues (2008), using the Chicago–Pittsburgh preschool sample, and Owens and colleagues (2009), using the BGALS sample, used parallel methodology to address this question of overall impairment via person-centered analyses. Rates of positive adjustment across the domains of ADHD symptoms, externalizing problems, internalizing problems, peer acceptance, social skills, and academic functioning (only in Owens et al., 2009), were calculated. In both, few children with ADHD showed cross-domain positive adjustment during early adolescence. In Lee et al. only 15% of the children, who were mostly boys, showed positive adjustment in at least four of five domains compared to 64% of the comparison children. In Owens and colleagues the comparable figures were 16% (girls with ADHD) and 86% (comparisons). Others who have looked at overall impairment dimensionally also report much poorer functioning over the long term in children with ADHD. For example, Hinshaw and colleagues (2006) reported effect sizes of 1.07 (ADHD-PI vs. controls) and 1.20 (ADHD-C vs. controls) during adolescence on their parent-reported overall impairment measure. These differences were not accounted for by child age or IQ, SES, or baseline comorbidities. At young adulthood follow-up, results were nearly identical (Hinshaw et al., 2012). In Barkley and Fischer (2011), parents rated participants with persistent and nonpersistent ADHD as more impaired than control boys in all 10 domains assessed; according to self-report, only those with persistent ADHD were impaired across domains. Similarly, Klein and colleagues (2012) found that children with ADHD followed into adulthood (age 41) showed deficits on 11 of 12 measures of adult functioning. In their community sample, Bussing and colleagues (2010) found higher levels of parent- and self-reported overall impairment, and lower self-reported quality of life, among children with ADHD followed into adolescence compared to control participants. Undoubtedly, both girls and boys with ADHD typically experience significant and pervasive impairment in adolescents and into adulthood.

Other Domains

Other negative outcomes of childhood ADHD have been documented, although replication is needed. Regarding self-harm, Hinshaw and colleagues (2012) reported greatly increased risk for self-injury and suicide attempts by young adulthood among girls with ADHD-C but not ADHD-PI. Similarly, Chronis-Tuscano and colleagues (2010) reported increased risk for attempting suicide through age 18 years, especially among girls. Reports of risky behavior during adolescence and adulthood are also emerging. Olazagasti and colleagues (2013) reported more head injuries, emergency department admissions, and sexually transmitted diseases by middle adulthood in boys with ADHD. Barkley and colleagues (2006) documented risks for earlier sexual intercourse and increased likelihood of earlier parenthood, both of which were related to severity of lifetime CD. Flory, Molina, Pelham, Gnagy, and Smith (2006) also reported increased risky sexual behavior (earlier sexual activity, more partners, more casual sex, more partner pregnancies) among boys with ADHD as young adults. Results were moderated by CD, but ADHD alone also posed a risk.

Although parents of children with ADHD often appear to be concerned about their children's self-esteem, evidence for the association between ADHD and low self-esteem is mixed. Slomkowski, Klein, and Mannuzza (1995) did find lower self-esteem during adolescence among children with ADHD (d = 0.5), but self-esteem did not moderate or mediate adult outcomes. Babinski, Pelham, Molina, Gnagy, and colleagues (2011) found parent- but not youth-reported low levels of self-esteem among grown-ups who as children had ADHD. Hinshaw and colleagues (2006) found small ADHD versus control group differences in adolescents on self-perceptions of social and scholastic competence that did not survive control of baseline comorbidities, IQ, or SES, and found no differences on any measure of self-esteem during young adulthood (Hinshaw et al., 2012).

Finally, a small body of literature does consistently support the relation between childhood ADHD and later personality disorders for both men and women (Fischer et al., 2002; Miller, Miller, Newcorn, & Halperin, 2008; Yoshimasu et al., 2012), especially among those with persistent ADHD or comorbid CD.

Summary

Clearly, for both boys and girls, childhood ADHD is associated with increased risk for poor outcome in adolescence and adulthood in the externalizing, academic, occupational, driving, social skills, and overall impairment domains. In most cases ADHD continues to be associated with negative outcomes when researchers adjust for key baseline covariates (especially IQ and comorbid psychopathology), suggesting that risk is related to ADHD per se and not to features associated with the diagnosis. There is also substantial evidence that childhood ADHD increases risk for later substance use and abuse, although findings regarding particular substances and measures of use and abuse are highly variable. The evidence that suggests childhood ADHD increases risk for later internalizing problems is weakest, but it does exist.

Importantly, evidence suggests increased statistical risk, meaning that, on average, certain negative outcomes are more likely in a group of children with ADHD than in a group of children without ADHD. However, outcomes of children with ADHD are variable, and conclusions about what is typical do not extend to all individuals. Whether a particular child with ADHD will experience negative outcomes always depends on baseline or co-occurring characteristics (moderators) and intervening variables (mediators). For example, as noted repeatedly in this chapter, comorbid externalizing problems—especially CD—or the development of externalizing problems during adolescence are clearly factors on which outcome for a particular child would depend with respect to many domains of outcome. Maltreatment (DeSanctis et al., 2008, 2012; Guendelman, Owens, Galan, Gard, & Hinshaw, 2013), ADHD subtype (Hinshaw et al., 2012), executive functioning deficits (Barkley & Fischer, 2011; Miller & Hinshaw, 2010; Miller, Nevado-Montenegro, & Hinshaw, 2012), social disability (Greene, Biederman, Faraone, Sienna, & Garcia-Jetton, 1997), family instability and adversity (Fischer et al., 1993a), and emotional impulsiveness (Barkley & Fischer, 2010) have each been identified as moderating particular outcomes in children with ADHD. At the same time, deviant peer association (e.g., Marshal et al., 2003), impulsivity (Swanson, Owens, & Hinshaw, in press), and low levels of emotional well-being (Latimer et al., 2003) have been identified as mediators of certain outcomes. Not surprisingly, each of these factors predicts poorer adjustment later on, and all essentially reflect additional risks or challenges at the level of the child, peer group, or family.

Treatment, of course, is also a potential mediator. A plethora of evidence suggests that in the short term, both pharmacological and psychosocial treatments are beneficial (Hinshaw, Klein, & Abikoff, 2007). But long-term prospective studies suggest that longer term outcomes of treated and untreated children are equivalent (e.g., Molina et al., 2009). This discouraging finding may occur because factors associated with treatment seeking or treatment continuation after a randomized trial, such as symptom severity or impairment, may be responsible for outcome rather than the treatment itself. It is also important to remember that ADHD is a chronic condition requiring long-term management, yet it is not clear that current, evidence-based, time-limited interventions necessarily persist in their effectiveness over the long haul. Thus, short-term treatment does not appear to mediate long-term outcome.

Unfortunately, a full discussion of the extant literature regarding mediators and moderators of outcome in children with ADHD is beyond the scope of this chapter. Still, what is known about moderators and mediators of outcome for children with ADHD is sparse and is a key area for future research. For now, it should be understood that when attempting to predict outcome or prognosis for an individual child, the child's and the family's particular strengths and weaknesses, as well as those at the level of the school, need to be fully considered.

GENDER DIFFERENCES[2]

We begin by noting the uphill battle that has been fought for clinical and research recognition of the presence and impact of ADHD in females. Believed to be a male disorder for much of the 20th century, only in the past few decades has recognition been given to the reality of ADHD in girls and women, with mounting evidence for the substantial impairments incurred by females with this condition (seminal meta-analyses: Gaub & Carlson, 1997; Gershon, 2002; long-term

follow-up investigations: Biederman, Petty, Fried, et al., 2010; Hinshaw et al., 2012). The clinical reality of ADHD in girls and women, paired with the official wisdom for many years that females could not really "have" ADHD, led to countless instances of underrecognition of the presence of ADHD, internalization of negative sequelae of symptomatology, and inaccessibility of relevant services (see Nadeau, Littman, & Quinn, 1999, for poignant case examples).

Along these lines, once it became recognized that girls and women can and do experience symptoms and attendant problems linked to ADHD, there was considerable effort to demonstrate substantial differences in the ways that relevant symptoms are expressed in males and females (e.g., hyperverbal behaviors in girls/women vs. hyperactive behaviors in boys/men). Moreover, some have contended that the optimal means of evaluating ADHD would be to compare appropriate symptoms to sex-specific norms as opposed to sex-neutral norms (e.g., McGee & Feehan, 1991; Waschbusch & King, 2006). The objective was to acknowledge that a substantial number of females, with extreme symptomatology compared to other females (if not to males), show clear impairment.

Overall, despite some evidence that sex-specific norms may be recommended, there are difficulties with the argument that simply altering relevant symptomatology to the point that sex differences are minimized or eliminated is valid. Some might argue, for example, that men are underrecognized and underdiagnosed with major depression, relative to women, in part because "male equivalents" (e.g., antisocial behavior, substance abuse) are not counted as part of depressive symptomatology. However, making the assumption that these symptom domains are automatically linked to depression is tantamount to asserting that there are no meaningful differences or divergent validity across various patterns of symptoms. Clearly, clinicians should be alert to the possibility of sex-specific expression of ADHD-related symptoms, but we cannot agree that changing the diagnostic criteria to encourage sex-parity in diagnosis is warranted. Indeed, it is well known that most neurodevelopmental disorders show a male predominance (e.g., autism spectrum disorders; early-onset conduct disorder; Tourette syndrome). ADHD, at least during childhood, is no exception (see American Psychiatric Association, 2013). In most, if not all, of the research reviewed below, the diagnostic criteria have been gender/sex-neutral rather than specific.

Despite efforts to distinguish male-like and female-like expressions of ADHD, there are few known gender differences in the developmental progression of ADHD and its core symptoms from childhood to adulthood, and there are few reliable gender differences in expected developmental outcomes of childhood ADHD. A possible exception with respect to the latter is heightened risk for internalizing problems among girls, which is true for all girls, not just those with ADHD. In this section we consider the information available about gender differences from cross-sectional studies. Most study participants are children and adolescents, and findings from these two populations are considered together. When information about gender differences in adults with ADHD is available, we consider it separately. Because the main focus of this section of the chapter is gender differences, the database for the review below comprises investigations that directly compare males and females with ADHD (in childhood, adolescence, and adulthood) with respect to a number of important symptomatic and impairment-related domains of outcome.

However, before we continue, we emphasize that the study of girls with ADHD as they develop into teens and young women—even without direct comparisons to boys—is important in its own right. Indeed, the relative paucity of data on girls and women with ADHD until relatively recently means that the overwhelming majority of inferences about developmental progressions concern male pathways. Thus, we believe that a brief review of issues salient for females with ADHD is warranted.

First, girls with ADHD, as they head toward adolescence and adulthood, maintain difficulties, relative to matched comparison females, in the essential outcome domain of psychiatric comorbidity. Indeed, Biederman, Petty, Fried, and colleagues (2010) found significant differences in lifetime rates of antisocial disorders, mood disorders, anxiety disorders, eating disorders, substance dependence, and developmental disorders in a 10-year follow-up of girls with ADHD. Hinshaw and colleagues (2012) yielded similar findings with respect to externalizing and internalizing disorders by follow-up in young adulthood. Yet significant differences with respect to rates of eating disorders or substance abuse/dependence were not found (see also Babinski, Pelham, Molina, Gnagy, et al., 2011). Note that in the BGALS investigation, significant differences between ADHD and comparison groups with respect to eating-related symptomatology at follow-up in adolescence (Hinshaw

et al., 2006) were no longer significant by the follow-up in young adulthood (Hinshaw et al., 2012), in large measure because of "catch-up" in eating disorder symptoms in the comparison sample by late adolescence.

Second, in terms of functional impairment, it is evident that girls with ADHD, relative to matched comparison girls, maintain significant deficits in academic achievement, social/interpersonal relationships, and need for service utilization (Hinshaw et al., 2012). Such persisting impairments often survive statistical control of demographic, comorbidity-related, and cognitive (i.e., IQ) covariates, revealing some specificity of ADHD–impairment linkages. Even though, in some cases, symptom levels had dropped below the DSM thresholds for continued ADHD diagnoses, clear impairments typically remained years after initial childhood diagnosis.

We also note provocative findings from the past several years with respect to the outcome domain of self-harm. Chronis-Tuscano and colleagues (2010) found that by adolescence, suicidal ideation and suicide attempts were far more prevalent in youth with ADHD than in comparison youth, particularly those with impulsive as opposed to purely inattentive symptomatology, with the small female subsample exhibiting particularly high risk for suicide attempts, significantly greater than that of the male subsample. Moreover, Hinshaw and colleagues (2012) found that by young adulthood, girls with ADHD-C, but not ADHD-PI or matched comparison girls, were at markedly higher risk for both suicide attempts (22 vs. 8 vs. 6%, respectively) and nonsuicidal self-injurious behavior (51 vs. 29 vs. 19%, respectively).

In short, girls with ADHD display clear comorbidities and impairments as they develop. It may also be the case that the gender-atypicality of many symptoms of ADHD (particularly extreme levels of impulsivity) place girls and women with ADHD at particularly high risk for continuing impairments. We now examine whether and how ADHD and its correlates manifest differently in boys and girls.

Prevalence, Subtypes, Core Symptoms, and Impairment

Like the early assumption that children outgrow ADHD by adulthood, it was formerly believed that ADHD in girls was extremely rare. Some estimates, based on clinical samples (i.e., those presenting for treatment or psychiatric referral), put the gender ratio as high as 9:1 (American Psychiatric Association, 1994). However, in the general population, things are different. At least during childhood and adolescence, males do predominate, but at a far lower ratio. In fact, two large community samples found the gender ratio to be 2.3:1.0 (Bauermeister et al., 2007; Ramtekkar, Reiersen, Todorov, & Todd, 2010), which is consistent with Arnold's (1996) gender ratio estimate of 2:1 to 3:1. This same ratio was reported in a meta-analysis of epidemiological studies of adults (Hooman & Ganji, 2012), although others have reported that the prevalence of ADHD in adults is equivalent for males and females (de Zwaan et al., 2012; Debjani, Cherbuin, Butterworth, Anstey, & Easteal, 2012). Thus, in the general population, ADHD is clearly less common in girls than in boys (though not necessarily in women vs. men), but it still occurs much more frequently in females than would be anticipated given reported gender ratios in clinical samples.

In the MTA study, for example, where recruitment was performed through school, pediatric, and mental health sources, as well as advertisements, there was a 4:1 boy-to-girl ratio among 7- to 9-year-olds (MTA Cooperative Group, 1999). Only ADHD-C was sampled, however, leading to questions about gender differences in subtype prevalence. Some investigations suggest that among girls, the ratio of ADHD-C to ADHD-PI is smaller than it is for boys. In other words, girls with ADHD may be more likely than boys to have the ADHD-PI subtype (Biederman et al., 2002; Hartung et al., 2002; Milich, Balentine, & Lynam, 2001), even though ADHD-C is more prevalent (at least in clinically referred samples).[3] However, in a large sample of siblings of children with ADHD, Biederman and colleagues (2005) found no gender differences in subtype (58% of ADHD females and 61% of males had ADHD-C; 25% of ADHD females and 27% of males had ADHD-PI). Similarly, Graetz, Sawyer, and Baghurst (2005) found no differences in subtype prevalence rates across girls and boys, nor did O'Brien, Dowell, Mostofsky, Denckla, and Mahone (2010), Ghanizadeh (2009), or Nolan, Volpe, Gadow, and Sprafkin (1999). Gender equivalence in subtype rates has been found among men and women with ADHD as well (Rasmussen & Levander, 2009). As is true for general prevalence rates, it may be that the ratio of ADHD-C to ADHD-PI for males and females is more similar in community as opposed to clinical samples.

Related to this issue is whether the core symptoms of ADHD vary across gender. Some studies suggest that boys with ADHD, on average, have higher levels

of hyperactive–impulsive symptoms than girls (Elkins, Malone, Keyes, Iacono, & McGue, 2011; Hartung et al., 2002; Hasson & Fine, 2012; Newcorn et al., 2001). Therefore, because a diagnosis of ADHD-C requires the presence of hyperactivity–impulsivity symptoms in addition to inattentive symptoms, boys also have higher levels of total ADHD symptoms (Monuteaux et al., 2010; Newcorn et al., 2001; Thorell & Rydell, 2008). In addition, one study reported higher levels of inattention in preschool-age boys with ADHD than in girls (Hartung et al., 2002), but only according to teachers, not parents.

On the other hand, some investigators report higher levels of hyperactive–impulsive symptoms (Rucklidge & Tannock, 2001) or total ADHD symptoms (Seidman et al., 2005) in diagnosed girls than in boys. Others do not find significant gender differences in hyperactive–impulsive symptom levels (Graetz et al., 2005; Owens, Pfiffner, & Hinshaw, 2013; Yang, Jong, Chung, & Chen, 2004), total symptom levels (Elkins et al., 2011, Graetz et al., 2005; Owens et al., 2013), or inattentive symptoms, whether objectively measured (Hasson & Fine, 2012; Newcorn et al., 2001; Yang et al., 2004) or according to parent and/or teacher report (Arcia & Conners, 1998; Brown, Madan-Swain, & Baldwin, 1991; Elkins et al., 2011; Graetz et al., 2005; Hartung et al., 2002 [parent report only]; Owens et al., 2013; Yang et al., 2004). In addition, the gender differences in reported symptom levels are usually small. Although boys with ADHD *may* typically have slightly higher levels of hyperactivity–impulsivity than girls, most evidence suggests that levels of inattention and total symptoms are quite similar between genders. This conclusion is somewhat different from earlier meta-analyses (Gaub & Carlson, 1997; Gershon, 2002) relying mostly on clinically referred samples, which suggest that girls have lower levels of most symptoms. More recent, large, community-based studies suggest that this is not the case.

Among adults, ADHD symptom levels appear to be comparable between men and women (Biederman, Faraone, Monuteaux, Bober, & Cadogen, 2004) or higher among women (Arcia & Conners, 1998; Fedele, Lefler, Hartung, & Canu, 2012; Robinson et al., 2008). It is important to remember that these reports of increased symptoms in women with ADHD may be due to a change from parent and teacher reports of children's symptoms to self-reports among adults. Women may have lower thresholds for what they consider symptomatic behavior or may be more likely than men to admit their symptoms, rather than symptoms among females increasing from childhood to adulthood in comparison to males.

Consistent with these findings regarding ADHD symptoms, and consistent with the observations from longitudinal studies reported earlier, existing cross-sectional data suggest strongly that at a global level, the overall levels of impairment of girls and boys with ADHD are equivalent (Bauermeister et al., 2007; Biederman et al., 2005; Graetz et al., 2005; Rucklidge & Tannock 2001). Indeed, and consonant with findings on adults' symptom levels, there have been at least two reports of greater impairment among adult women with ADHD (Arcia & Conners, 1998; Fedele et al., 2012; Gjervan, Torgersen, Nordahl, & Rasmussen, 2012), although Biederman and colleagues (2004) report equivalent levels of overall impairment in men and women. We consider specific types of impairment (e.g., academic and social) below. All told, although during childhood boys with ADHD do outnumber girls, the gender ratio is much smaller than previously believed. Among adults, it may be close to equal. Similarly, previous misconceptions about symptom levels being higher among males are generally unfounded. And it is quite clear that at all ages, males and females with ADHD have equivalent impairment.

Comorbidities

As suggested in three previous reviews (Arnold, 1996; Gaub & Carlson, 1997; Gershon, 2002), some document that girls with ADHD appear to have fewer externalizing comorbidities than boys with ADHD (Bauermeister et al., 2007; Biederman et al., 2002). Some have reported the same gender discrepancy among adults (Biederman et al., 1994; Rasmussen & Levander, 2009). However, a number of studies suggest instead that rates of externalizing comorbidities and co-occurring behavior problems are equivalent between (1) boys and girls and (2) men and women with ADHD (Biederman et al., 1994, 2005; Brown et al., 1991; Gabel, Schmitz, & Fulker, 1996; Levy, Hay, Bennett, & McStephen, 2004). Although no direct comparison with boys was made, Hinshaw (2002) found high rates of ODD comorbidity (71% for girls with ADHD-C and 47% for girls with ADHD-PI) among young girls with ADHD. Rates during adolescence (51% of all girls with ADHD-C or ADHD-PI; Hinshaw et al., 2006) and young adulthood (49% of girls with childhood ADHD-PI and 41% of girls with ADHD-C had comorbid CD or ODD;

Hinshaw et al., 2012) were slightly lower but still quite high, as is typically found in samples of boys. Noteworthy, however, is that these reports of externalizing comorbidity pertained exclusively to parent-reported symptomatology. Rated by objective observational data during summer enrichment programs (Hinshaw, 2002), girls' levels of antisocial and aggressive behavior were far lower than those of comparable samples of boys attending parallel programs. It may be that parental report of externalizing symptoms in girls reflects particular parental stressors or "views" of the incorrigibility of their daughters' behavior. In short, the jury is still out regarding gender differences in rates of comorbid externalizing problems.

Most investigations find no reliable differences in rates of comorbid internalizing problems, both anxiety and depression, in boys versus girls or men versus women with ADHD (Bauermeister et al., 2007; Biederman et al., 1994, 2004, 2005; Brown et al., 1991; Friedrichs, Igl, Larsson, & Larsson, 2012; Gabel et al., 1996; Graetz et al., 2005; Greene et al., 2001; Hartung et al., 2002; Rucklidge, Brown, Crawford, & Kaplan, 2007), although there is at least one report of increased rates for boys (Biederman et al., 2002). This rate equivalence between genders is somewhat different than the heightened longitudinal risk for an internalizing outcome that girls generally experience (noted earlier). It seems that the higher risk of later internalizing problems for girls with ADHD, as for all girls, does not obtain until adolescence. Relatedly, Rasmussen and Levander (2009) and Katz, Goldstein, and Geckle (1998) reported more symptoms of anxiety and depression in women than in men with ADHD. Also of note, gender differences in comorbidities may be moderated by subtype. Bauermesiter and colleagues (2007) found that boys with ADHD-C were more likely to have a comorbid mood disorder than girls, and that girls with ADHD-PI were more likely to have comorbid anxiety than boys. Researchers should be more cognizant of subtype (or "current presentation") issues as more data become available in which children are classified by subtype.

As noted earlier, longitudinal investigations suggest that risk for substance use or abuse is equivalent for girls and boys. However, in cross-sectional work, Biederman and colleagues (1999, 2002) found that girls with ADHD, ages 6–17, have a much greater risk for a substance use disorder compared to boys with ADHD. This is noteworthy because the girls were also much less likely to have comorbid CD, which is often identified as a moderator of substance use among children with ADHD. Thus, despite longitudinal evidence that is equivocal regarding gender effects, there is reason to be cognizant of possibly increased risk for substance use problems specific to girls with ADHD.

School Functioning

On balance, evidence suggests that boys with ADHD may have more trouble at school than girls, especially with behavior (as opposed to objectively measured academic achievement), although the evidence is not uniform. Some investigations report more school problems, both academic and behavioral, for boys. Biederman and colleagues (2002) found that boys with ADHD have higher rates of learning disabilities and lower levels of reading achievement. Bauermeister and colleagues (2007) reported more grade failure and suspensions, and DuPaul and colleagues (2006) reported less academic motivation among boys than among girls with ADHD. Based on classroom observations, Abikoff and colleagues (2002) found that boys with ADHD demonstrate much more aggression, rule breaking, interference, and out-of-seat behavior than do girls with ADHD, even those with ADHD-C. However, there were no significant gender differences in off-task behavior, and DuPaul and colleagues reported no gender differences in observed classroom behavior. Furthermore, Brown and colleagues (1991), DuPaul and colleagues (2006), and Rucklidge and Tannock (2001) reported no significant gender-based differences in achievement among children with ADHD. These findings are consistent with a meta-analyses regarding ADHD and achievement (Frazier, Youngstrom, Glutting, & Watkins, 2007).

On the other hand, DuPaul and colleagues (2006) showed that the school-related differences between girls with and without ADHD are larger than for boys with and without ADHD. Despite the equivalence of boys and girls on most of their measures of school functioning, they suggested that school problems among girls might be considered more severe because they reflect functioning further from what is considered normative. Of note, Graetz and colleagues (2005) found that boys with ADHD-C had more trouble with schoolwork than girls with ADHD-C, a difference that was not found among those with ADHD-PI. Again, consideration of subtype differences might clarify the varied findings on school functioning and gender.

Social Functioning

Although it was initially suggested that girls with ADHD have lower levels of peer rejection than do boys with ADHD (Arnold, 1996), most subsequent studies and reviews suggest that the compromised social functioning of boys and girls with ADHD is equivalent (Gaub & Carlson, 1997; Greene et al., 2001; Hartung et al., 2002; Hoza et al., 2005, Mikami et al., 2013). The longitudinal observations reported earlier also support this conclusion. However, some have suggested poorer social functioning among girls (Ek, Westerlund, Holmberg, & Fernell, 2008) and women with ADHD (Fedele et al., 2012). ADHD subtype may be relevant. In community samples, Graetz and colleagues (2005) found that boys with ADHD-C had more social problems than girls with ADHD-C, whereas Elkins and colleagues (2007) found that girls, especially those with ADHD-PI, were more bullied and less popular. At a global level, girls and boys with ADHD may be equally impaired in the social domain. As more data become available regarding gender × subtype interactions, gender differences in the social and peer domains may be clarified.

Neuropsychological Functioning

Neuropsychological differences between girls and boys with ADHD have been reported, but they are few and far between and inconsistent across studies. The vast majority of studies indicate that poor performance of girls and boys with ADHD is equivalent across neuropsychological measures, mostly reflecting executive functioning (Arcia & Conners, 1998; Balint, Czobor, Meszaros, Simon, & Bitter, 2009; Hinshaw, Carte, Fan, Jassy, & Owens, 2007; Houghton et al., 1999; Rucklidge, 2006; Rucklidge & Tannock, 2002; Seidman et al., 2005; Uebel et al., 2010; Wodka et al., 2008; Yang et al., 2004). In terms of overall intellectual performance, the Gaub and Carlson (1997) and Gershon (2002) reviews concluded that girls with ADHD might have somewhat lower IQs than boys with ADHD. Yet additional investigations have revealed no significant overall IQ differences (Arcia & Conners, 1998; Biederman et al., 1999, 2005; Gross-Tur et al., 2006; Hartung et al., 2002; Rucklidge & Tannock, 2001; Seidman et al., 1997; Yang et al., 2004). An exception was Biederman and colleagues (2002), who found that girls with ADHD have lower IQs than boys with ADHD.

Some of the isolated differences on neuropsychological tests include the following: higher scores on a design replication test for boys versus girls with ADHD (Brown et al., 1991; Yang et al. 2004); more impaired inhibition among boys (Rucklidge, 2006); the seemingly contradictory findings of poorer response inhibition and better planning by boys compared to girls (O'Brien et al., 2010); and slower processing speed for boys (Rucklidge & Tannock, 2001), although this finding was not replicated in Rucklidge (2006). Rucklidge (2010) suggests that type I error (i.e., incorrectly identifying associations that are in fact due to chance) accounts for these varied and isolated findings. It is also possible that the substantial variability in neuropsychological functioning found in children with ADHD is implicated. Alternatively, or additionally, it may be that gender interacts with subtype, so that analyses in which children with ADHD-C and ADHD-PI are grouped together may produce conflicting results. In support of this idea, Wodka and colleagues (2008) found a gender × subtype interaction on a measure of verbal fluency: Boys with ADHD-C outperformed girls with ADHD-C, whereas girls with ADHD-PI outperformed boys with ADHD-PI (although performance of girls and boys with ADHD was equivalent on measures of executive functioning at an overall level).

Gender comparisons in adults with ADHD are sparse, with the bulk of empirical evidence concerning neuropsychological differences between adults with ADHD, in general, and comparisons. However, in a meta-analysis of 25 studies that used 12 different neuropsychological measures, Balint and colleagues (2009) did find one possible gender difference among adults with ADHD. In studies with larger percentages of male participants, the ADHD–comparison difference on the Stroop interference task was larger than it was in studies with smaller percentages of male participants. In summary, although isolated neuropsychological differences between girls and boys, and men and women with ADHD are reported, no consistent intellectual or executive functioning difference has been found across samples.

Treatment History and Response

It is commonly assumed that the increased ratio of boys to girls in clinically referred samples is because boys may be more likely to have disruptive behavior

problems that motivate their parents and teachers to seek help. If, in fact, at least some of the gender differences in ADHD prevalence across clinical and community samples are due to such referral bias, one might expect to find differences in treatment history across community samples of girls and boys with ADHD. Bauermeister and colleagues (2007) showed this to be the case, with boys more likely to be referred for all types of treatment than girls. The likelihood of treatment with medication was especially large for boys compared to girls (odds ratio = 10.6). Bussing and colleagues (2005) found boys with ADHD to be more than twice as likely as girls to receive medication. Derks, Hudziak, and Boomsma (2007) also found far higher rates of treatment referral in boys than in girls with ADHD, which they attributed to teacher complaints about the children's behavior, rather than to parent perceptions of ADHD and behavior problem severity. However, not all investigations have shown gender differences in treatment history among nonreferred samples (Biederman et al., 2005; Visser et al., 2013), and some studies of adults also report no gender differences in treatment history (Biederman et al., 2004).

Not surprisingly, among girls and boys with ADHD referred for treatment, differences in treatment history are not found. Even though the ratio of boys to girls who present for treatment in psychiatric clinics is large, those girls and boys who do come in have similar treatment histories (Biederman et al., 2002; Rucklidge & Tannock, 2001). Furthermore, when girls and boys are treated for ADHD, they respond similarly. In the MTA, gender did not moderate response to treatment (MTA Cooperative Group, 1999; Owens et al., 2003). Girls and boys did not differ significantly in terms of response to behavioral treatment, to treatment with medication, and to combined treatment. Mikami and colleagues (2009) and Sharp and colleagues (1999) also found no gender differences in response to stimulant medications. We know of only two reports of gender-based differential response to treatment. First, Sonuga-Barke and colleagues (2007) found that within a single day, girls have a better response to stimulants earlier and a worse response later. If this finding were replicated there would be implications for adjusting dosing schedules based on gender. Second, Mikami and colleagues (2013) found that the beneficial effects of a novel classroom intervention encouraging peer inclusion were obtained predominantly for boys relative to girls.

SUMMARY AND FUTURE DIRECTIONS

Decades of longitudinal research have clearly documented the persistence of ADHD for many, if not most, children across developmental periods, even into middle adulthood. Serious consequences across many domains of symptomatology and impairment, for both boys and girls, are also clear. At the same time, as clinicians and researchers finally have begun to recognize the reality of ADHD in girls and women, longitudinal and cross-sectional evidence for gender differences in manifestations of ADHD is not compelling. However, rather than arguing that ADHD in girls is important *even though* it generally manifests as it does in boys, the take-home message regarding gender differences is that ADHD in girls is important *because* it manifests as it does in boys. Although gender differences might be interesting, the fact that girls generally do not show unique correlates or outcomes is important news because it means the condition is serious and debilitating for both genders. At the same time, the condition, over the long term, is not debilitating for every individual with ADHD. Despite ADHD persistence and increased risk for poor outcome in many domains, some children do not suffer over the long term. Future work needs to focus on explaining who will continue to be affected by ADHD and who will experience positive adjustment despite the initial challenge of ADHD. Identification of moderators on which ADHD persistence and outcome depend, and of mediators by which they are explained, is especially important for clinicians, educators, and parents trying to meet the needs of children with ADHD as they grow.

KEY CLINICAL POINTS

- ✓ Despite the earlier belief that ADHD is outgrown by adolescence, modern follow-up studies demonstrate a relatively high rate of persistence of ADHD from childhood to adolescence (50–80%) and into adulthood (35–65%).
- ✓ Differences in rates of persistence are substantially (four to nine times) lower if self-report is used rather than parent or other reports.
- ✓ Symptoms of hyperactivity (and perhaps impulsivity) decline more steeply with age than do symptoms of inattention.

- ✓ Childhood ADHD increases the risk for concurrent and later ODD and CD. But this risk may be partially or entirely due to childhood-associated externalizing disorders rather than to ADHD specifically. These disorders in turn increase the risk for adolescent and adult substance use and abuse, as well as antisocial activities and antisocial personality disorder.
- ✓ It is less clear that childhood ADHD predisposes children to later internalizing disorders. Comorbidity with other disorders may be more of a determining factor in this risk.
- ✓ Children with ADHD are more likely to experience academic impairment, driving problems, social impairment, and overall impairment by adolescence and adulthood than are typically developing children. They are also more likely to experience problems and adverse outcomes associated with driving and risky sexual behavior.
- ✓ Childhood ADHD is related to an increased risk of nicotine use by adolescence. Far less clear is whether ADHD in childhood predisposes individuals to adolescent alcohol use or abuse. Should comorbid ODD or CD be present, this increases the risk for adolescent and adult use and abuse of other substances.
- ✓ Children with ADHD are more likely to have problems with occupational functioning by adulthood than are typically developing children.
- ✓ Girls with ADHD are at risk for all of the aforementioned comorbid disorders and impairments, and often to the same extent as are boys with ADHD, but may be at higher risk for later internalizing disorders than boys, who may show a higher risk for externalizing disorders, substance use and abuse, and other risk-taking behavior. Yet these gender differences are those seen between girls and boys in population samples and may therefore not be specific to ADHD. Nor are they consistently evident across studies.
- ✓ Girls with ADHD as children may by adolescence be more prone than boys with ADHD and control girls to eating pathology, binge eating, and bulimia, but such differences attenuate by young adulthood.
- ✓ Although in childhood ADHD is somewhat more prevalent in boys than in girls, perhaps by a ratio of 3:1, this difference attenuates such that by adulthood, prevalence is nearly the same.
- ✓ Few if any gender differences in people with ADHD are evident in neuropsychological deficits or treatment response.

NOTES

1. A variable measured at baseline that is associated with outcome for all children (in this case, both those with ADHD and a comparison group) is called a "predictor," whereas a baseline variable differentially associated with outcome across groups is called a "moderator." Also, as subsequently described in the text, factors or variables that exist during initial evaluations and that differentially influence outcomes are called "moderators," whereas factors or variables that transpire during intermediate periods of development (between baseline and follow-up) and help to explain the genesis of relevant outcomes are termed "mediators" (see Kraemer, Wilson, Fairburn, & Agras, 2002).
2. There is debate about whether differences between males and females should be termed "sex differences" (in which "sex" refers to biological males and biological females) or "gender differences" (in which "gender" refers to a broader pattern of gendered expectations and roles, over and above biological sex). We choose the broader term "gender differences," realizing that there is inconsistent use of terms across the vast literature on this topic.
3. Note that in DSM-5 (American Psychiatric Association, 2013), the designation of types of ADHD has been softened to "current presentations," given extensive evidence that the supposed subtypes of ADHD-PI, ADHD-C, and ADHD-HI are not particularly stable over time.

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CHAPTER 10

Executive Function Deficits in Adults with ADHD

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Adults with attention-deficit/hyperactivity disorder (ADHD) have difficulty in self-management, including organization, planning, initiating and completing tasks on a timely basis, tracking and shifting tasks, self-monitoring, and self-inhibition. In the aggregate, these are termed "executive functions" (EFs) which, according to a recent definition, can be viewed as "those self-directed actions needed to choose goals and to create, enact, and sustain actions toward those goals (Barkley, 2012b, p. 60). This difficulty in self-regulation typically results in reduced productivity, inefficiency, missed deadlines, poor planning, "careless" errors, and losing and forgetting things as a result of disorganization. In some (particularly those with the combined form of ADHD), reduced inhibitory control may also lead to emotional dysregulation and inappropriate verbal and/or physical behavior in interpersonal interactions. Over the lifetime of the individual, these difficulties contribute to failure to achieve goals—personally, academically, and occupationally. These failures in turn likely contribute to the high rates of anxiety and depression in adults with ADHD (Kessler et al., 2006; also see Chapter 13).

Although not listed as a symptom of ADHD in the DSM-5 (American Psychiatric Association, 2013) or any previous edition of the DSM, executive dysfunction has in recent years come to be regarded by many in the field as a defining characteristic of ADHD in both children and adults (Barkley & Murphy, 2011; Barkley, Murphy, & Fischer, 2008; Brown, 2013).

MEASUREMENT OF EF ON NEUROPSYCHOLOGICAL TESTS

Executive dysfunction has traditionally been measured by neuropsychological tests. However, research over the last decade has called into question whether these tests capture the scope of executive dysfunction in ADHD and other disorders. Most notably, the effect sizes (i.e., difference between groups in mean scores, expressed in standard deviation [*SD*] units) reported in meta-analytic studies of the neuropsychological tests commonly used to assess EF have largely been between 0.5 and 0.65 (i.e., moderate) for differences between groups with ADHD and normal adult groups (Boonstra, Oosterlaan, Sergeant, & Buitelaar, 2005; Hervey, Epstein, & Curry, 2004; Schoechlin & Engel, 2005). In addition, effect sizes for non-EFs such as attention, verbal memory, and processing speed have generally been equivalent to those reported for EFs (Boonstra et al., 2005; Hervey et al., 2004). Similar results, showing only moderate effect sizes and lack of universality, were reported in a pivotal meta-analysis of studies of chil-

dren with ADHD (Willcutt, Doyle, Nigg, Faraone, & Pennington, 2005).

The diagnostic efficiency of neuropsychological tests with respect to ADHD appears to be quite limited both in adults and children. Biederman and colleagues (2008) reported that neuropsychological tests identified only 14% of a sample of 194 adults with ADHD. Similar results were found in the study by Barkley and Murphy (2011) and the follow-up study by Barkley and Fischer (2011). In contrast, more than twice that number (34.4%) were identified as impaired in the Biederman and colleagues study on the basis of self- ratings of EF on the Current Behavior Scale, a precursor of the Barkley Deficits in Executive Function Scale (BDEFS), described further below. In another study, 89–98% of adults with ADHD were in the clinically impaired range (i.e., more than 1.5 *SD* from the normal mean or 7th percentile) on a prototype of the BDEFS (Barkley & Fischer, 2011; Barkley & Murphy, 2011).

In children, Doyle, Biederman, Seidman, Weber, and Faraone (2000) reported that scores in the impaired range on tests of EF had good positive predictive validity with respect to identifying boys with ADHD (i.e., few false positives) but only moderate negative predictive validity (i.e., a higher rate of false negatives). Biederman and colleagues (2004) reported that, based on a neuropsychological test battery, only 33% of a sample of 259 children and adolescents with ADHD had EF deficits. Moreover, numerous studies find little or no significant correlations between tests of EF and rating scales of this same construct (Toplak, West & Stanovich, 2013). In addition, rating scales appear to be more predictive of impairments in major domains of life activities than are tests of EF (Barkley & Fischer, 2011; Barkley & Murphy, 2010). Neuropsychological testing is therefore not appropriate to rule in or rule out a diagnosis of ADHD. Such testing may be useful, however, in assessing an individual's profile of cognitive strengths and weaknesses, including possible reading or other learning deficits, for educational or occupational purposes, and may be required to document the need for academic accommodations.

COMPONENT EXECUTIVE FUNCTIONS

Although not useful for clinical diagnostic purposes, the measurement of specific EFs via testing has nonetheless yielded insights into core components of executive dysfunction that affect at least a subgroup of those with ADHD. In the following, I describe the EFs that most consistently differentiate adults with and without ADHD in the meta-analytic studies: response inhibition, working memory, and set shifting. It should be noted that since the three meta-analyses with adults were published close together in time, there is considerable overlap in the studies they include.

Response Inhibition

"Response inhibition" refers to the ability to withhold a cognitive or behavioral impulse that may be inaccurate or maladaptive. Impulsivity–hyperactivity is one of two core symptom dimensions of ADHD in the DSM-5 (American Psychiatric Association, 2013). In ADHD, poor response inhibition may occur cognitively, behaviorally, or emotionally (see Chapter 3), as reflected in (1) responding before a question is completed or before instructions are reviewed; (2) responding without first considering all response options; (c) failing to withhold a behavioral or cognitive response to an irrelevant or inappropriate stimulus; (4) acting before considering the consequences of a socially offensive or aggressive behavior; and (5) being easily excited, overly aroused, and impatient or easily frustrated.

The continuous-performance test (CPT), of which there are numerous variants, has been widely used for clinical and research purposes as a measure of both response inhibition and attention/vigilance. On the visual CPT, the examinee views a series of stimuli (e.g., letters, numbers, or shapes) presented sequentially on a computer screen, with an interstimulus interval on the order of 1–4 seconds, and is instructed to respond whenever a predesignated target stimulus (e.g., a given letter or sequence of two letters) appears. CPTs with infrequent targets primarily tax attention processes as indexed by errors of omission, as, for example, on the integrated visual and auditory CPT (IVA; Sandford & Turner, 1995), whereas CPTs on which target stimuli predominate create a "set" to respond, thereby primarily taxing inhibitory control, as indexed by errors of commission on the Conners CPT-II (Conners, 2000). The frequency of CPT commission errors discriminated between groups with a moderately large effect size of 0.65 in five studies reported in the meta-analyses (Boonstra et al., 2005; Hervey et al., 2004; Schoechlin & Engel, 2005) and is one of the larger and most consistently demonstrated differences across studies in both children (Willcutt et al., 2005) and adults with ADHD.

The stop-signal task, a measure of response inhibition, is widely used in research on ADHD and other conditions. On this task, the individual performs a visual choice reaction time task (the primary task). On a proportion of trials, randomly selected, a tone (the "stop-signal") is presented immediately *after* the primary task stimulus ("go-signal") and is the cue to *inhibit* the response to the go-signal on that trial. The reaction time (RT) to the stop-signal ("stop-signal RT") is the critical index of inhibitory control and has been used in numerous studies with adults (Hervey et al., 2004; Schecklmann et al., 2013) and children (Alderson, Rapport, & Kofler, 2007; Willcutt et al., 2005) to differentiate groups with and without ADHD.

Working Memory

"Working memory" is a system that actively holds multiple bits of transitory verbal or nonverbal information in mind while they are manipulated (Cowan, 2008). An example of a cognitive activity that depends on working memory is mental arithmetic, in which numbers are held in memory while some operation (e.g., addition, multiplication) is performed on them and the result also is sustained in memory. Difficulties in working memory may underlie diverse difficulties in ADHD, such as keeping track of time; tracking conversations in order to be able to generate appropriate responses; keeping track of the location of one's belongings; and expressive writing, in which there is a need simultaneously to maintain both one's own and the reader's perspective in order to maximize cohesiveness.

Baddeley and Hitch (1994) delineated three main components of working memory: (1) the visual–spatial sketchpad and (2) the phonological loop (for temporary storage of nonverbal and verbally based information, respectively), and (3) the central executive, which is the controlling component responsible for the allocation of attention between these two "slave" components. Alderson, Kasper, Hudec, and Patros (2013) conducted a meta-analysis of 38 studies comparing working memory in adults with and without ADHD that yielded an effect size of 0.49 for the visual–spatial sketchpad and 0.55 for the phonological loop. A separate study conducted by the authors (Alderson, Hudec, Patros, & Kasper, 2013) also documented involvement of the central executive, but with smaller group differences than in previous studies of children with ADHD, which the authors interpreted to suggest maturation in this function over time.

Among the frequently used *verbal* tests of working memory in the studies included in the meta-analysis is the Paced Auditory Serial Attention Task (PASAT), on which the examinee hears a new number every 3 seconds and is asked to add that number to the one heard just before it. The Digit Span Backward subtest of the Wechsler Adult Intelligence Scale (WAIS) also differentiated groups. This subtest makes more demands on working memory than Digit Span Forward subtest, in that it requires a manipulation (reversal of order) to be performed on the number string held in memory. Rohlf and colleagues (2012) recently demonstrated that the *difference* between the highest number of digits repeated in the forward direction and the backward direction significantly differentiated groups, with an effect size of 0.51.

Among the visual–spatial tests that figured in the meta-analysis were the Spatial Working Memory subtest of the Cambridge Neuropsychological Test Automated Battery (CANTAB) and the Rey-Osterrieth Complex Figure Test (in which the examinee is asked to copy a complex figure, draw it from memory after a brief delay, then draw it again after a longer delay). Interestingly, the Wisconsin Card Sorting Test (WCST), widely used to index working memory, set formation, and set shifting, did not discriminate between groups in virtually any study with adults (Hervey et al., 2004), although it has been shown to do so in children with ADHD (Romine et al., 2004).

Set Shifting

"Set-shifting" has been defined as the ability to move back and forth between multiple tasks, operations, and mental sets (Miyake et al., 2000). Examples relevant to ADHD include shifting between tasks that have different cognitive demands (e.g., making business calls vs. writing a memo; cooking dinner while supervising children; watching videos vs. studying for a test). The last example may also make heavier demands on response inhibition given that one must first inhibit an activity that presumably provides much more immediate gratification.

The Stroop Color–Word Test is variably considered a measure of cognitive inhibitory control, resistance to interference, and set shifting. On the traditional version of this task, the individual is asked to read, as quickly as possible, a list of color words printed in black ink, then to name the color in which each of a list of XXXX's are printed; and finally to name the *color* in

which color names are printed while ignoring the incongruent word itself (e.g., the word "blue" printed in red ink). The "interference" score, which is calculated from the word reading, color naming, and color–word naming scores, reflects the individual's ability to inhibit the interference from reading the word while naming the colors.

Despite its intuitive appeal as a measure of set shifting, the meta-analyses indicated that the Stroop interference score does *not* differentiate between groups (Boonstra, Kooij, Oosterlaan, Sergeant, & Buitelaar, 2010; Hervey et al., 2004). This finding emerged as well in a more recent study of a similar task from the Delis–Kaplan Executive Function System's Color–Word Interference Task (CWIT), in which the difference between groups in interference score was reduced to nonsignificance after controlling for IQ and working memory (Halleland, Haavik, & Lundervold, 2012). In this study, however, a significant difference between groups did emerge on the final condition within the CWIT (not present on the Stroop), which requires the examinee to alternate between inhibiting and reading the color word if it is framed, This condition may be considered to be a more rigorous test of set shifting that is not confounded by speed of word or color naming, or by response inhibition. Thus, while interference control or response inhibition, as measured by the Stroop, does not appear to differentiate groups, set shifting, particularly on more complex tasks, may still be a discriminating variable.

The Trail-Making Test (TMT) is divided into two parts. On Part A the examinee is given a sheet of paper with the numbers 1 to 25 randomly scattered on the page, and is asked to draw lines connecting the numbers in order, as quickly as possible. On Part B, the subject is given a similar sheet of paper with the numbers 1 to 12 and the letters A to L randomly placed on the page, and is required to draw the connecting lines as quickly as possible, alternating between numbers and letters, in order (A, 1, B, 2, C, 3, etc.). Both parts of the TMT measure visual scanning and psychomotor speed. The TMT-B measures, in addition, set shifting (between numbers and letters). The meta-analyses each found moderate effect sizes. In the study by Boonstra and colleagues (2005), the effect sizes, based on six studies, were 0.46 for TMT-A and 0.65 for TMT-B. However, in order to isolate an effect of set shifting, the score on TMT-B must be adjusted for the score on TMT-A, which was not done in the meta-analyses. This was accomplished in a recent study by Rohlf and colleagues (2012), in which the total time to complete TMT-B was divided by total time to complete TMT-A. This "shifting score" did significantly discriminate between adults with (n = 37) and without (n = 32) ADHD, again providing support for differences in set-shifting adults with and without ADHD.

PLANNING AND EXECUTING FOR THE FUTURE: THE ROLE OF TEMPORAL DISCOUNTING

Adults with ADHD have difficulty planning for the future and successfully executing those plans. Notably, they are less likely than others to engage in positive behaviors in the present that will yield greater benefits in the future. Examples in the daily life of individuals with ADHD abound: going to bed at an hour that will allow sufficient sleep time; beginning and continuing work on academic and occupational assignments and projects within a time frame that allows for successful completion before deadlines; putting things away immediately after use to prevent inefficient searching; practicing healthful eating and exercise habits; and saving money for future needs. All of these activities require that the individual forgo some measure of current pleasurable activities in order to achieve larger rewards in the near-term or more distant future. Reluctance to forgo immediate rewards makes it less likely that adults with ADHD will sustain effort to pursue and achieve the larger, more significant goals of life, such as higher academic degrees, job advancement, and accumulation of savings for significant purchases.

Research suggests that this behavioral characteristic reflects the phenomenon known as "temporal discounting," wherein future rewards are discounted in value relative to immediately available rewards (Scheres, De Water, & Mies, 2013). It is hypothesized that the value of delayed rewards decreases *more* steeply as a function of the delay interval in individuals with ADHD than in typical individuals. Support for this hypothesis has been generated in multiple studies using experimental tasks in children with ADHD (Antrop et al., 2006; Kuntsi, Oosterlaan, & Stevenson, 2001; Scheres, Tontsch, Thoeny, & Kaczkurkin, 2010). Temporal discounting was also increased in adolescents with ADHD (plus oppositional defiant disorder [ODD]), relative to those without (Barkley, Edwards, Laneri, Fletcher, & Metevia, 2001), and greater temporal discounting was associated with reduced academic achievement in typical college students (Lee et al., 2012).

In adults, temporal discounting has been shown to occur more steeply in groups with less impulse control, including substance abusers and those with neuropsychiatric disorders (Wittmann & Paulus, 2008). Research with adults with ADHD is limited thus far to two studies: preference for smaller, sooner rewards in adults with ADHD was reported as a trend in a small imaging study (Plichta et al., 2009) and was a robust finding in a larger sample studied by Hoogman and colleagues (2011). Research in this area is actively continuing. A recently developed cognitive-behavioral intervention for adults with ADHD includes a component designed to offset temporal discounting by enhancing the salience—and therefore the motivating power—of distant rewards (Solanto, 2011, pp. 149–151).

NON-EFs

As mentioned, effect sizes in the meta-analyses were similar for EF and non-EF functions, suggesting that ADHD involves deficits in the latter as much as the former abilities (Boonstra et al., 2005; Hervey et al., 2004).

Attention

Given that an attention deficit has long been viewed as the defining feature of the disorder, it is not surprising that tests of attention have been widely used to assess patients with ADHD. Prominent among these, for both clinical and research purposes, is the visual CPT, on which the errors of omission (failures to respond to the target stimulus) are considered to index selective attention and sustained attention over time. Frequency of omission errors did discriminate between adult groups with a moderately large effect size in all three meta-analyses (Boonstra et al., 2005; Hervey et al., 2004; Schoechlin & Engel, 2005), as was also true for children (Willcutt et al., 2005). Simple RT to the target stimuli on the CPT and on other tasks (e.g., the go RT on the stop-signal task) did *not* distinguish the groups. However, the *variability* (standard error) in the CPT RT score did consistently differentiate groups in adults (Boonstra et al., 2005; Hervey et al., 2004), as well as children (Castellanos & Tannock, 2002), suggesting that variability of attention to task may be a critical feature of ADHD (Gonzalez-Gadea et al., 2013).

Memory

There is much interest in memory functions in adults with ADHD, who frequently report that they have trouble retaining material they have just learned or to which they have been exposed. Two prior meta-analyses have shown moderate deficits on verbal memory tasks, whereas visual memory was not affected (Hervey et al., 2004; Schoechlin & Engel, 2005). An important question, therefore, is whether adults with ADHD have fundamental problems with learning and/or memory. Skodzik, Holling, and Pedersen (in press) recently sought to address this problem by determining the extent to which problems of memory are explainable by difficulties in acquisition, and whether adults with ADHD have an additional retrieval deficit. Their meta-analysis of studies of verbal memory (including the California Verbal Learning Test, the Logical Memory subtest of the Wechsler Memory Scale (WMS), and the Rey Auditory Verbal Learning Test), revealed that adults with ADHD showed deficits in both delayed (e.g., 20 minutes) Free Recall (effect size = 0.49) and Acquisition (effect size = 0.58). Nearly all the variance (95%) in memory, however, was explained by differences in memory acquisition, suggesting that memory deficits in ADHD reflect a learning difference at the stage of encoding. The latter may result from problems of attention or learning strategies, including those affected by working memory and other EFs. There were no significant differences between groups in visual memory (tested via the Rey-Osterrieth Complex Figure Test, the Visual Reproduction subtest of the WMS, and a modified version of the Visual Learning and Memory Test for Neuropsychological Assessment). The lack of differences in visual memory is interesting and consistent with the results of previous meta-analyses, which found that tests of verbal fluency, but not spatial–figural fluency, distinguished adults with and without ADHD (Hervey et al., 2004). Skodzik and colleagues also reported that there was no additional deficit in retrieval processes in adults with ADHD. This suggests that patients with ADHD should be encouraged to use visual–spatial prompts and aids when attempting to learn new material.

NORMED RATING SCALES OF EFs

There are three widely available self-ratings of EFs in adults, one of which was developed specifically for use

in the assessment of ADHD: the Brown Attention-Deficit Disorder Scales (BADDS; Brown, 1996). The other two rating scales, the Behavior Rating Inventory of Executive Function—Adult Version (BRIEF-A; Roth, Isquith, & Gioia, 2005), and more recently, the BDEFS (Barkley, 2011), were developed to assess EFs across various conditions and disorders.

The BADDS scale was developed on the basis of clinical studies and observations and comprises 40 items, divided into five subscales: Activation (initiation of tasks, time estimation, and prioritization), Attention (focused, sustained, and shifting), Memory (working and short-term), Effort (regulation of alertness, sustaining effort), and Affect (managing and modulating emotions). The scale was normed on 142 adults diagnosed in clinical interviews on the basis of DSM criteria, and 143 controls matched for age and socioeconomic status (SES). The scale demonstrated significant differences between the ADHD and control groups, and includes a helpful index of likelihood that the individual meets diagnostic criteria for ADHD. Problematic with the scale is the very limited sample of normal or typical adults that is not representative of the U.S. population and the construction of the subscales based on the developer's view of each EF domain rather than empirical or statistical analyses, such as factor analysis, that are typically used to guide the constructions of dimensions in rating scale development.

The BDEFS was developed in three stages (Barkley, 2011). First, items were constructed for the scale based on existing theories of EF and its components, as well as on chart reviews of several hundred cases of adults with ADHD. Second, this 91-item prototype scale (P-DEFS) was then given to three groups of adults (Barkley et al., 2008): 146 adults with ADHD participating in the author's University of Massachusetts (UMASS) Study of Adult ADHD; 97 community controls presenting with possible ADHD but ultimately diagnosed with other conditions; and 109 volunteers from the community. Factor analysis that included all participants yielded five scales (Barkley & Murphy, 2011): Self-Management to Time (prepared on time for work or assigned tasks, time estimation, prioritization, punctuality, planning ahead); Self-Organization/Problem Solving (organization of thinking, action, and writing; overcoming obstacles to goals); Self-Restraint (considering consequences before commenting, deciding, or acting); Self-Motivation (sustaining effort, quality, and output in work; choosing larger more delayed rewards over immediate rewards); and Self-Activation/Concentration (distractibility, sustaining alertness and concentration while working). This same scale was also given to a group of children with ADHD followed for 20 years to young adulthood and a control group in the Milwaukee longitudinal study (Barkley et al., 2008). Factor analysis of the scale for these participants revealed the same five-factor structure (Barkley & Fischer, 2011). Finally, the scale was administered to 1,249 adults drawn from the general U.S. population (Barkley, 2011). It was factor-analyzed again with this sample. The results did not perfectly replicate the earlier analyses because the factor of Self-Activation/Concentration did not emerge and was therefore removed from the final scale. Also, since the original scale lacked content for an important domain of EF, the self-regulation of emotions, a new subscale was created for that domain. The final scale contains 89-items including this new scale, Self-Regulation of Emotions (Barkley, 2011). The scale demonstrates considerable differentiation of adults with ADHD from those who have sluggish cognitive tempo and from adults in a general population sample who have neither condition (Barkley, 2012a).

There is some overlap in the content of the BADDS scales and those from the final BDEFS. For instance, the Effort scale (BAADS) has some item overlap with the Self-Motivation scale (BDEFS); likewise, there is overlap on the Affect scale (BAADS) and Self-Restraint (BDEFS) and Self-Regulation of Emotions scales (BDEFS). Items that reflect Activation on the BADDS scale are included in Self-Management to Time on the BDEFS. Attention and Memory on the BADDS scale are variously represented on the BDEFS Self-Management to Time and Self-Organization. Items found on the BDEFS Organization/Problem Solving (BDEFS) do not appear to be represented on the BAADS.

The BRIEF-A was developed to assess EFs across a wide array of neurological and cognitive disorders, and was normed in a sample of 1,136 adults drawn from the general population after exclusion of adults with various disorders and medical conditions. It was then further evaluated on 1,050 adults with a range of impairments (Gioia, Isquith Guy, & Kenworthy., 2000). The BRIEF-A comprises nine conceptually developed subscales that place on two dimensions (indices) when factor-analyzed. The Behavioral Regulation Index (BRI) includes the Inhibit, Shift, Emotional Control, and Self-Monitor scales. The Metacognitive Index

(MCI) includes Initiate, Working Memory, Plan/Organize, Task Monitor, and Organization of Materials. A Global Executive Composite is generated from the BRI and MCI. Although the BRIEF-A has been used as an outcome measure in several studies of executive dysfunction in adult ADHD, there is as yet no research to indicate how well it differentiates or classifies individuals with and without ADHD, or the extent to which scores on this measure are correlated with functional impairment in ADHD. Nevertheless, given the overlap of its item content with the BDEFS and BADDS, one would expect it to perform similarly to those scales in differentiating groups of ADHD and control adults.

COMPARISON BETWEEN EF TESTS AND EF RATINGS

In two studies of adults with and without ADHD, Barkley and colleagues compared self-ratings of EF on the P-DEFS with a battery of EF tests with respect to their predictive utility vis-à-vis overall functional impairment. They reported that whereas the ratings accounted for more than half of the variation in impairment, the best combination of EF tests explained less than 10% of the variance (Barkley & Fischer, 2011; Barkley & Murphy, 2010). In addition, the P-DEFS was significantly more predictive of occupational functioning than was the neuropsychological test battery in both studies (Barkley & Fischer, 2011; Barkley & Murphy, 2010).

EF tests and EF ratings also appear to identify different subgroups of individuals with ADHD having different correlates of impairment. In a study of 194 adults with ADHD, those who performed poorly on the psychometric tests (n = 28, 14%) had lower IQ and academic achievement, whereas those identified as impaired in EF ratings (n = 67, 34.4%) had more severe ADHD symptoms, worse social adjustment, more comorbid conditions, and lower overall functioning. Twenty-eight adults (14%) in the sample were impaired on both, whereas 71 (37%) were impaired on neither. Parallel differences with respect to academic functioning have been shown for children with ADHD with and without tested executive dysfunction (Biederman et al., 2004).

Disparity between performance on EF tests and EF ratings is not limited to ADHD. A recent meta-analysis examined correlations between performance-based measures and ratings of EF in a total of 20 studies (13 child and 7 adult) across a variety of clinical disorders. The results revealed an overall median correlation of only .19 (Toplak et al., 2013). The authors concluded that these two approaches to the measurement of EF are not assessing the same construct.

This challenge to the role of EF tests in assessing EF in ADHD and various other disorders has been based on arguments that psychological tests in current use are not sufficient or comprehensive measures of EF (Barkley, 2012b; see also Chapter 16). This may be the case in part because they are highly structured, focus on "cool" purely cognitive (as opposed to "hot" affective) aspects of EF, and thus overlook emotional and social features of EF, and involve a time frame on the order of minutes, whereas real-world executive control requires self-regulation in the absence of external structure, and involves social and emotional contexts over much longer periods, extending to days, weeks, months, and years.

HETEROGENEITY IN EXECUTIVE DYSFUNCTION

Variability in the expression of EFs suggests the possibility of subtypes within the pool of adults who are diagnosed as having ADHD. This variability may in part correspond to differences between the combined and inattentive presentations/subtypes of ADHD (American Psychiatric Association, 2013) or between individuals with and without sluggish cognitive tempo (Barkley, 2012a; see Chapter 17), but these factors have been largely unexamined in studies to date.

Barkley has propounded a developmental hierarchy of EFs, in which elemental component functions (e.g., working memory, inhibitory control) are organized into progressively higher and more complex levels of functioning over the course of development (Barkley, 2012b; see Chapter 16). The model delineates six levels: At the lowest level are *preexecutive* functions, such as arousal, attention, and memory, essentially reflecting largely automatic brain functions shared with other primates. This is followed by the *instrumental* level of self-directed, moment-to-moment, largely mental actions used for self-control (e.g., inhibition, verbal and nonverbal working memory, emotional self-regulation, timing, planning). The third level, the *adaptive* level, uses the instrumental EFs for meeting daily needs of survival and self-care. The fourth level is the *tactical* level, which uses lower EF levels for self-regulation across daily activities occurring in reciprocal social relationships with others and is manifested in self-management

to time, self-organization, self-motivation, self-restraint, and self-regulation of emotions. The fifth level, the *strategic* level, uses the tactical and lower EF levels to achieve midterm to longer-term goals involving cooperation with others and as reflected in educational, occupational, economic, and social pursuits. Likely contained within that level is one that Barkley calls *principled,* which uses all earlier EF levels to achieve long-term, highly abstract goals related to one's longer-term welfare and happiness, as well as one's contribution to society. An implication of this model is that individuals with deficits at the lowest level of EF may be expected to have difficulties at all subsequent levels, whereas some individuals may experience deficits only at higher levels that do not radiate downward to impact lower levels adversely. This may account in part for the heterogeneity in expression of executive dysfunction in adults with ADHD, in that only the more severely affected are likely to show deficits at the instrumental level, such as those assessed on EF tests in current use. Others may experience difficulty only later in development when encountering demands for higher and more complex levels of EF at work or in higher education.

TREATMENT OF EXECUTIVE DYSFUNCTION

Recent reviews and integration of the literature on the effects of stimulants on cognitive tests, largely in children, indicate that improvement is more likely seen on non-EF tasks (e.g., delayed match-to-sample complex RT, spatial recognition RT) rather than on tasks with an EF component (e.g., inhibitory control, organization, set-shifting, planning; Swanson, Baler, & Volkow, 2011). In adults, whereas stimulant and nonstimulant medications reduce or ameliorate core symptoms of ADHD, there is little evidence from clinical observation that they substantially improve EF as it is employed in daily life. In a randomized controlled study, Biederman and colleagues (2011) found no effects of oral osmotic release drug delivery system (OROS)-methylphenidate on EF, whether ascertained on the basis of neuropsychological tests or the BRIEF-A. However, positive changes in EF, as measured on EF rating scales, have been shown following treatment with Vyvanse (Adler et al., 2013; Brown, Brams, Gao, Gasior, & Childress, 2010), mixed amphetamine salts (Brown & Landgraf, 2010) and atomoxetine (Durell et al., 2013). Continued research is needed to resolve discrepancies among the stimulant studies, and to ascertain the clinical significance of rated changes in EF over time across medications.

Cognitive-behavioral interventions designed to improve EFs as used in daily life activities in adults with ADHD have been shown to be effective in two randomized controlled double-blind trials (Safren et al., 2010; Solanto et al., 2010; see also Chapter 31). Further research is needed to delineate the relative benefits and mechanisms of action of cognitive-behavioral therapy (CBT) and pharmacotherapy for improving EF, and to identify clinical predictors of differential response to treatments.

SUMMARY

Adults with ADHD are impaired in numerous aspects of EF, including response inhibition, verbal and nonverbal working memory, set shifting, and possibly temporal discounting, as reflected in cognitive tests of these EFs. On rating scales, their deficits are manifested in poor time management, self-organization and problem solving, self-restraint (inhibition), self-motivation, and self-regulation of emotions.

Neuropsychological tests in common use do not fully capture the wide range of EF deficits and are associated with a high rate of false negatives when used to diagnose ADHD. Standardized self-report rating scales of EF have greater predictive utility with respect to identifying adults with ADHD and are far more highly correlated with functional impairment in general, and occupational impairment in particular. A recently proposed developmental hierarchy of EFs may help to account for heterogeneity of EF deficits in ADHD. This model suggests that the elemental EFs (e.g., working memory, inhibitory control), which operate on a time scale of minutes to hours, are organized into progressively higher and more complex levels of functioning that operate over broader time spans and ultimately involve longer term educational, occupational, economic, and social goals. Whereas some more severely affected individuals may be impaired at the instrumental EF level, such as that typically assessed on EF tests, others may experience difficulty only when they encounter demands for higher and more complex levels of EF at work or at advanced levels of education. The development of treatments to enhance EFs in adult ADHD is at an early stage, but it holds promise, particularly with respect to the utility of cognitive-behavioral interventions (see Chapter 32).

KEY CLINICAL POINTS

- ✓ Adults with ADHD are impaired in numerous aspects of EF, including response inhibition, verbal and nonverbal working memory, and set shifting, as reflected in cognitive tests of these EFs.
- ✓ They are also deficient in time management, planning and problem solving, self-organization, self-motivation, self-restraint, and emotional self-regulation, as reflected in rating scales of EF in everyday life.
- ✓ Neuropsychological tests in common use do not fully capture the wide range of EF deficits and are associated with a high rate of false negatives when used to diagnose ADHD.
- ✓ Standardized self-report rating scales of EF have greater predictive utility with respect to identifying adults with ADHD and are far more highly correlated with functional impairment in general, and occupational impairment in particular.
- ✓ A recently proposed developmental hierarchy of EFs (Chapter 16) may help to account for heterogeneity of EF deficits in ADHD. This model suggests that the elemental EFs (e.g., working memory, inhibitory control), which operate on a time scale of minutes to hours, are organized into progressively higher and more complex levels of functioning that operate over broader time spans and ultimately involve longer-term educational, occupational, economic, and social goals. Whereas some more severely affected individuals may be impaired at the instrumental EF level, such as that typically assessed on EF tests, others may experience difficulty only when they encounter demands for higher and more complex levels of EF at work or at advanced levels of education.
- ✓ The development of treatments to enhance EFs in adult ADHD is at an early stage, but it holds promise, particularly with respect to the utility of cognitive-behavioral interventions (see Chapter 32).

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CHAPTER 11

Health Problems and Related Impairments in Children and Adults with ADHD

Russell A. Barkley

As the authors of the previous chapters have attested, attention-deficit/hyperactivity disorder (ADHD) is associated with a variety of difficulties other than the central problems with inattention, impulsivity, and overactivity. Such children and adults have a higher likelihood of having other cognitive, developmental, academic, and social difficulties. This chapter focuses instead on the health problems and related domains of impairment not addressed in prior chapters. Not all children and adults with ADHD display all these risk factors. But as a group, they are more likely to do so and to display them to a degree that is greater than expected in typical people. Because the health-related problems are not considered to be the core or essence of the disorder, they are not diagnostic of the disorder when present, nor do they rule out the diagnosis when absent.

GENERAL HEALTH STATUS AND CHRONIC ILLNESS

ADHD appears to be associated with a number of health risks as well as increased use of medical services and resultant expenses (Cuffe, Moore, & McKeown, 2009; Nigg, 2013). Research indicates that children with externalizing disorders, including ADHD, appear to be at risk for a several chronic physical conditions, including obesity, atopic eczema, epilepsy, and asthma (Goodwin et al., 2009). But what of ADHD specifically? Several early studies found that children with ADHD have more problems with general health than do normal children. Nearly 30 years ago, Hartsough and Lambert (1985) found that 50.9% of hyperactive children were described as having been in *poor health during infancy,* whereas Stewart, Pitts, Craig, and Dieruf (1966) found that this prevalence described 24% of their sample. Both figures are greater than those for control children (29.2 and 2.7%, respectively).

A small body of evidence also suggests that both children with ADHD (Giacobo, Jane, Bonillo, Arrufat, & Araujo, in press) and children with ADHD followed to adulthood (Barkley, Murphy, & Fischer, 2008) voice more complaints about functional somatic symptoms (e.g., headaches, stomachaches, and vague bodily concerns) that may have little if any medical origins. The number of such complaints was related to the extent to which ADHD had persisted to the adult follow-up (Barkley et al., 2008). The degree of such complaints seems to be linked to the degree of anxiety in both children and adults (Barkley et al., 2008; Giacobo et al., in press), as well as the degree of parental over-

protection of children (Giacobo et al., in press). Longitudinal studies (Barkley et al., 2008; Brook, Brook, Zhang, Seltzer, & Finch, in press) likewise reveal an elevated risk for impaired general health in teens with ADHD followed to adulthood (ages 27–36).

Health concerns across a variety of domains also appear to afflict adults diagnosed with ADHD at adulthood. In our study of large samples of adults with ADHD compared to a clinical and a community control group (Barkley et al., 2008), we used the Skinner Health and Lifestyle Interview (Skinner, 1994), a self-administered computerized survey of health-related behavior that expresses results in a figural form, with each domain coded as a strength, concern, or risk. The results for this lifestyle assessment appear in Figure 11.1 for those domains in which significant group differences were evident. The groups did not differ in the percentage scored as having a concern or risk in the lifestyle domains of nutrition, eating habits, caffeine use, physical activity, or body weight. However, the ADHD group had a higher percentage of individuals reporting problems in nine out of 16 areas assessed than did the community control group. These areas included sleep, social relationships, family interactions, tobacco use, nonmedical drug use, medical/dental care, motor vehicle safety, work and leisure, and emotional health. The ADHD group had more members with concerns/risks in nonmedical drug use, motor vehicle safety, and emotional health than did the clinical control group. Obviously, lifestyles of adults with ADHD pose greater concerns/risks for more of them across many more lifestyle domains than do the lifestyles of community adults. But illicit drug use, driving, and emotional health are areas in which adults with ADHD differ specifically from clinic-referred adults who do not have ADHD.

The presence of chronic health problems, such as recurring *upper respiratory infections and allergies*, were also noted more often in hyperactive children than in normal children (39–44% vs. 8–25%) (Hartsough & Lambert, 1985; Mitchell, Aman, Turbott, & Manku, 1987; Szatmari, Offord, & Boyle, 1989). Likewise, Trites, Tryphonas, and Ferguson (1980) noted more

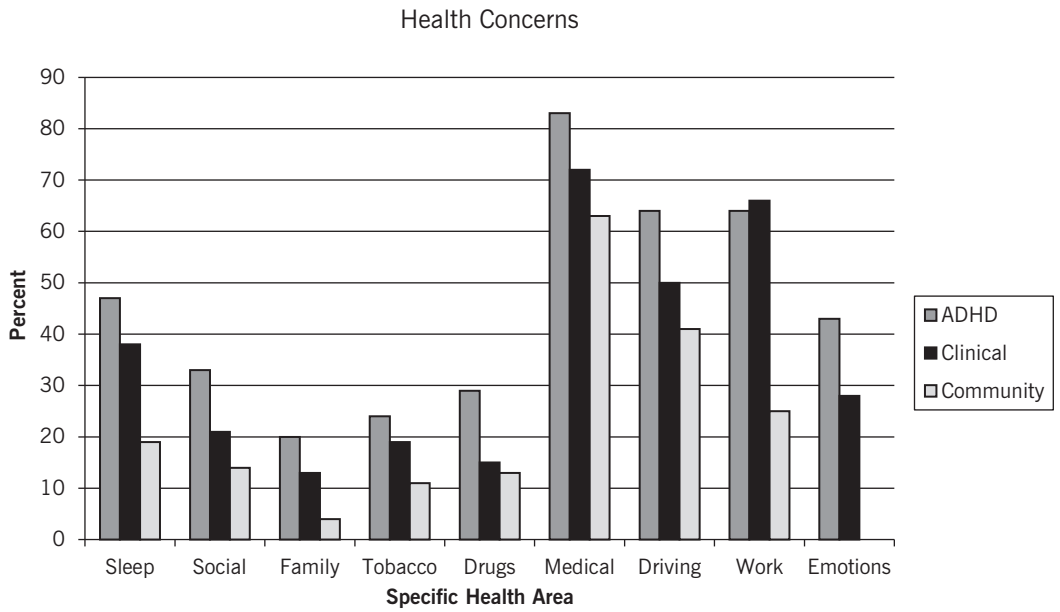

FIGURE 11.1. Percent of each group scoring in the risk or concern range on the Skinner Health and Lifestyle Interview from the UMASS Study. The figure depicts those health and lifestyle areas on which the ADHD group differed from the community control group. From Barkley, Murphy, and Fischer (2008). Copyright 2008 by The Guilford Press. Reprinted by permission.

allergies in hyperactive children than in normal children, and others have noted the inverse, that is, more ADHD symptoms in children with atopic (allergic) disorders (Roth, Beyreiss, Schlenzka, & Beyer, 1991). A recent prospective, 10-year follow-up of a large sample of preschool children with atopic eczema found that degree of early eczema was significantly associated with risk for early- but not late-onset ADHD (Genuneit et al., 2014). In one study, only children with hyperactivity not associated with conduct problems were more likely to have allergies (Blank & Remschmidt, 1993). But others have not found an association between ADHD and allergies (McGee, Stanton, & Sears, 1993; Mitchell et al., 1987); the association between the specific allergy of atopic rhinitis (hay fever) and ADHD has shown mixed results (Hart, Lahey, Hynd, Loeber, & McBurnett, 1995; Genuneit et al., 2014; Yang et al., in press). Yang and colleagues (in press) also reported that children with allergic rhinitis were more likely to have elevated symptoms of ADHD. Thus, the nature of any association between ADHD and allergies remains unclear as of this writing.

One large-scale study suggests that individuals suffering from *acne* may be 2.5 times more likely to have ADHD than do those without this skin condition (Gupta, Gupta, & Vujcic, 2014). Although this was a very large population-based study, further research in this area is warranted.

Otitis media, or middle ear infection, has been noted to be more common and more recurrent in children with ADHD (Adesman, Altschuler, Lipkin, & Walco, 1990). Also, early-onset and recurrent otitis media has been linked to greater severity of ADHD symptoms (Padolsky, 2008). Even so, two studies are inadequate for rendering any definitive conclusions about this association, though current evidence is suggestive of one.

Several early studies examined whether children with ADHD were more likely to suffer from *asthma*. An initial report by Hartsough and Lambert (1985) suggested such an increased risk for asthma among children considered to be hyperactive. Yet several subsequent studies using larger samples (n = 140) did not find this to be the case when clinical diagnostic criteria for ADHD were used to identify the children (Biederman, Milberger, Faraone, Guite, & Warburton, 1994; Biederman, Milberger, et al., 1995). Nevertheless, several more recent population-based studies have resurrected this small but significant risk. For instance, in one study involving a birth cohort of 4,119 children, those who met criteria for ADHD (about 7.5%) had a significantly increased risk of asthma (22 vs. 13%) (Leibson, Katusic, Barbaresi, Ransom, & O'Brien, 2001). This assertion that asthma is twice as common in children with ADHD was evident in a more recent U.S. population-based study (Cuffe et al., 2009). The reverse relationship is also evident: Children with asthma are slightly but significantly more likely to have ADHD (odds ratio = 1.31) than are children without asthma (7 vs. 4.6%, respectively) (Chen et al., 2013). Therefore, a small relationship of risk is shared between ADHD and asthma.

In contrast to the findings for asthma, there appears to be no association between ADHD and a higher risk for *diabetes* or *hypertension* (Nigg, 2013) despite the association between ADHD and obesity, discussed below. However, a small emerging body of evidence suggests an association between ADHD and risk for *coronary heart* and *lung disease,* which, if not a consequence of obesity, may well be related to other risky lifestyle factors, such as a greater use of alcohol, tobacco, and other drugs (Barkley et al., 2008). In the Milwaukee follow-up study of children with ADHD, Barkley and colleagues (2008) conducted complete physical and laboratory examinations of their children with ADHD and control children years later at a mean age of 27 years. By that time, the hyperactive children were beginning to show some significant signs of risk for coronary heart disease, as assessed through several risk rating methods and their lipid profiles. Likewise, a recent population-based study of older adults in The Netherlands found that elevated ADHD symptoms were positively associated with the presence of nonspecific chronic lung diseases and cardiovascular diseases, and the number of chronic diseases, while being negatively associated with self-perceived health status (Semeijn et al., 2013). Those relationships were not found to be mediated by lifestyle variables, implying a more direct linkage between ADHD and these health risks. Such findings are consistent with observations in another study of 1,122 men followed for 12 years, in which degree of self-regulation was negatively predictive of risk for various types of coronary heart disease (CHD), even after researchers controlled for levels of anxiety, depression, and anger (Kubzansky, Park, Peterson, Vokonas, & Sparrow, 2011). Conscientiousness, the personality trait, is clearly related to self-regulation and is also repeatedly associated with adult health and well-being (Hampson, 2008). Given that children and adults with ADHD place toward the lowest end of the spectrum of self-regulation or Conscientiousness in the population, one should not be surprised to see an el-

evated risk of CHD associated with the disorder, as well as lower levels of adult health and well-being more generally. Obviously there is a need here for more research into the link between ADHD and CHD given that the latter is one of the major causes of mortality in the U.S. adult population.

The little evidence that is available on the subject suggests that ADHD is not associated with difficulties with vision, such as refractive errors (Fabian et al., 2013; Hartsough & Lambert, 1985).

DENTAL HEALTH

Only a few studies have examined the relationship of ADHD to dental health, but their findings consistently indicate that children with ADHD are at increased risk for various dental problems. The earliest study I was able to locate on this topic was conducted in New Zealand by Broadbent, Ayers, and Thomson (2004), who found that children with ADHD were 12 times more likely to have diseased, missing, or filed teeth (DMFT) even after they controlled for fluoride history, medical problems, diet, and oral hygiene habits. A subsequent study likewise documented a greater number of caries in the primary and secondary dentition of children with ADHD (Grooms, Keels, Roberts, & McIver, 2005). Several studies were conducted by Blomqvist and colleagues in Sweden. In the initial report, they found that children with ADHD had significantly more DMFT, as well as more behavior management problems during the office examination, than did control children (Blomqvist, Holmberg, Fernell, Ek, & Dahllof, 2006). The results of these earlier studies were subsequently replicated by Chandra, Anandakrishna, and Rey (2009), who found that children with ADHD not only had more dental caries, but they also were not likely to engage in proper dental hygiene as often as control children, and they ate more sugary substances—both of which likely contributed to their higher rate of caries. However, as Broadbent and colleagues (2004) noted, even after researchers control for factors such as diet and hygiene, a significant risk for DMFT problems remains. A separate study also revealed that adolescents with ADHD had lower rates of caries-free teeth and higher rates of decay or filed surfaces, as well as higher rates of gingivitis that caused their gums to bleed when probed, than did control teens (Blomqvist, Ahadi, Fernell, Ek, & Dahllof, 2011). Besides DMFT problems, some research indicates that children with ADHD have more problems with toothache, bruxism, bleeding gums, poorer hygiene, and oral trauma histories than do typical children (Atmetlla, Burgos, Carillo, & Chaska, 2006; Bimstein, Wilson, Guelmann, & Primosch, 2008; Katz-Sagi, Redlich, Brinsky-Rapoport, Matot, & Ram, 2010; Pessah, Montluc, Bailleul-Forestier, & Decosse, 2009), with a five times greater risk for oral trauma than control children (Katz-Sagi et al., 2010). The reverse has also been found in a study in which children presenting with dental trauma were more hyperactive and impulsive than control children (Thikkurissy, McTigue, & Coury, 2012). One study in Israel did not find higher rates of DMFT or any differences in diet or oral hygiene between children and adults with ADHD and control cases, but sample sizes were small (n = 31 and 30, respectively) limiting its statistical power to detect such differences (Hidas et al., 2011). It did find that rates of unstimulated saliva flow were reduced in children with ADHD, whether medicated or not, and that children with ADHD had more plaque. Other research has not found evidence of reduced salivary flow, however (e.g., Grooms et al., 2005). Later research in Germany, using far larger samples, found that hyperactivity and inattention were significantly associated with the extent of lesions of noncavitated caries and molar–incisor hypermineralization (Kohlboeck et al., 2013).

Not surprisingly, office behavior problems are more common in children with ADHD, as noted earlier in that they may show not only more behavior problems, as noted here and in other research (Atmetlla et al., 2006; Pessah et al., 2009). Children with ADHD may also demonstrate lower stress reactivity to the dental examination, as manifested in lower salivary cortisol levels after the examination than levels in comparison children (Blomqvist et al., 2007). They may also demonstrate poorer quality communication with the dentist at the time of examination (Blomqvist et al., 2005).

No studies have yet examined the role of parental ADHD, which is likely to be present in a sizable minority of parents of children with ADHD, in the problems with diet and oral hygiene noted earlier. And while the greater risk for oral trauma is consistent with the larger body of evidence on risk for accidental injuries linked to ADHD (see below), I found no studies that have investigated this issue of shared liability across types of dental and nondental accidents.

Far less research has been conducted on the oral health of adults with ADHD. A recent study indicated that adult ADHD is more common (16%) in adults referred for treatment of dental anxiety than would be

expected from population prevalence estimates (Carlsson, Hakeberg, Blomkvist, & Boman, 2013). Such adults with ADHD had higher levels of dental anxiety and self-reported poorer oral health than did dentally anxious adults without ADHD, but clinical evidence of poorer oral health did not differ significantly between these groups. Far more research on the dental health and problems that may be associated with ADHD in adults is needed given the more abundant literature demonstrating the many dental risks evident in children with ADHD.

It is now reasonably well established that ADHD in children and teens is associated with a substantially greater risk of dental caries specifically and DMFT more generally, as well as an increased risk for oral trauma. There is also some evidence of poorer dental hygiene and possibly even poorer diet associated with the disorder, but these factors have been less well studied and do not seem to account for the heightened risk for caries. As one might expect, children with ADHD may be more difficult to manage during the office dental examination. Far less is known about the dental problems associated with ADHD in adults. Clearly, there is a need not only to make primary care pediatric dentists aware of these heightened risks but also to develop interventions that promote better hygiene and diet in families of youth with ADHD, as well as greater preventive utilization of dental care. A number of common-sense recommendations for dental care can be found in the review by Murray, Naysmith, Liu, and Drummond (2012).

SUBSTANCE USE

As discussed in the chapters on comorbidity (Chapters 5 and 13), teens and adults with ADHD are more likely to qualify for a diagnosis of substance use disorder. This is especially the case for those who already have comorbid conduct disorder (CD). But apart from being more likely to have a diagnosis of a substance dependence or abuse disorder, teens and adults with ADHD use both legal (alcohol, tobacco) and illegal (marijuana) substances more often than do controls, even if they do not have CD (Barkley et al., 2008).

Much of the research on drug use in adults with ADHD comes from longitudinal studies of children with ADHD followed to adulthood (Weiss & Hechtman, 1993). That youth with ADHD are at higher risk for increased tobacco use as adolescents and young adults has been demonstrated in several studies (see Tercyak, Peshkin, Walker, & Stein, 2002, for a review; Barkley, Fischer, Edelbrock, & Smallish, 1990; Barkley, Fischer, Smallish, & Fletcher, 2004; Borland & Heckman, 1976). For instance, Milberger, Biederman, Faraone, Chen, and Jones (1996) followed 6- 17-year-olds with and without ADHD for 4 years and found that ADHD was specifically associated with a higher risk for initiating cigarette smoking, even after they controlled for social class, psychiatric comorbidity, and intelligence. Stable smoking by adolescence has also been found to be linked to childhood ADHD (Wilens et al., 2011). Molina, Smith, and Pelham (1999), in a study of 202 adolescents, reported that ADHD is associated with increased use of all substances, including nicotine, but only when it is associated with comorbid CD. Yet they also found that it was the hyperactive–impulsive (HI) dimension of ADHD within this comorbid group that was most closely associated with this elevated risk of substance use. In partial agreement with these results, Burke, Loeber, and Lahey (2001) followed 177 clinic-referred boys with ADHD to age 15 years and found that 51% of these teens reported tobacco use, but that this risk was only elevated in the group with comorbid ADHD and CD. Unlike Molina and colleagues, these authors found that it was the inattention dimension that was specifically associated with a 2.2 times greater risk for tobacco use by adolescence, even after they controlled for other factors known to be associated with such use (CD, poor parental communication, ethnicity, etc.). Tercyak, Lerman, and Audrain (2002) also confirmed this link between not just ADHD but specifically its inattention symptoms and the risk for cigarette use by adolescence. Even mild levels of ADHD symptoms appear to elevate this risk for smoking (Whalen, Jamner, Henker, Delfino, & Lozano, 2002). The risk of smoking by adolescence may be accentuated in youth with ADHD whose school adjustment is poor (Flory, Malone, & Lamis, 2011). Later research also indicated that adults with ADHD are more likely to have smoked tobacco in their lives and are more likely to be current smokers than individuals in a community control group, but not more than those in a clinical control group (Barkley et al., 2008).

A study (Kollins, McClernon, & Fuemmeler, 2005) further cements the relationship between ADHD symptoms and increased risk of nicotine use. Kollins and colleagues (2005) used the National Longitudinal Study of Adolescent Health, a nationally representative sample, to examine whether ADHD symptoms were associated with increased smoking risk. They followed 15,197 ado-

lescents into young adulthood. Analyses showed a linear relationship between inattentive and HI ADHD symptoms and lifetime likelihood of being a regular smoker (having smoked at least 1 cigarette per day for at least 30 days). Even when they controlled for CD symptoms, each additional ADHD symptom significantly increased the risk for regular smoking. For those who did smoke, more symptoms were associated with an earlier age of smoking onset. ADHD symptoms are therefore a useful predictor of risk for smoking, and for an earlier onset of smoking, even outside of a clinical setting. Given the stimulant-like action of nicotine on the dopamine transporter in the striatum of the brain and its similarity to the effects of methylphenidate on that site (Krause et al., 2002), these findings suggest that greater nicotine use in those with ADHD could be a form of self-medication in an effort to treat their own symptoms. However, other mechanisms that possibly account for this link between ADHD and nicotine use (social, cognitive, psychological) need to be explored more fully (Glass & Flory, 2010), such as recent evidence that behavioral disinhibition, novelty seeking, and risk taking may contribute to the risk for smoking as much as self-medication (Sousa et al., 2011). Some studies also have indicated that certain risk genes for ADHD may also increase nicotine use, and that severity of ADHD interacts with these risk genes to further promote risk for nicotine use (Bidwell et al., 2012; McClernon, Fuemmeler, Kollins, Kail, & Ashley-Koch, 2008; Thakur, Sengupta, Grizenko, Choudhry, & Joober, 2013).

Concerning alcohol use, the picture from research studies had been initially mixed. Blouin, Bornstein, and Trites (1978), in a retrospective study, were among the first to report that children with hyperactivity may be more at risk than control children for adolescent alcohol use (57% of hyperactive children vs. 20% of the controls). Larger, more recent epidemiological studies have also suggested that ADHD increases the likelihood of both nicotine *and* alcohol use by adolescence (Gudjonsson, Sigurdsson, Sigfusdottir, & Young, 2012). Weiss and Hechtman (1993) found that as teenagers, somewhat more of their hyperactive participants used alcohol than did their control group, but this was not found at the adult follow-up. Hartsough and Lambert (1985) also did not find increased alcohol use at their adolescent follow-up. More recent longitudinal studies may help to clarify these inconsistent results. Some large studies indicate that ADHD may increase the risk for later teen alcohol use, but only in those children who engages in deviant peer relationships and whose parental monitoring is low (Molina et al., 2012). Some research (Knop et al., 2009) and two meta-analyses found that childhood ADHD does not necessarily increase the risk for alcohol and drug use disorders in adolescence, but it does so eventually by adulthood (Charach, Yeung, Climans, & Lillie, 2011; Lee, Humphreys, Flory, Liu, & Glass, 2011). By contrast, childhood ADHD is more strongly associated at adolescence with nicotine use (Charach et al., 2011; Lee et al., 2011). Moreover, family association studies suggest that the liability for alcohol use disorders in families of children with ADHD may be specifically transmitted, separate from any liability conferred by ADHD, rather than being a general risk associated with ADHD liability in the family, as are other drug use disorders (Biederman, Petty, et al., 2008).

These and other studies have also documented greater frequency of use of other substances, especially marijuana, in adolescents with ADHD (Barkley et al., 2008; Chilcoat & Breslau, 1999). Given the well-known association between CD and risk of drug use, Barkley and colleagues (2004) subdivided their hyperactive group into those who did and did not have lifetime CD by young adulthood (self-reported) and compared their frequency of use of various drugs to that of the control group. Results indicated significant group differences for nine of the 11 drug use activities surveyed. In all cases, it was the hyperactive group with CD that accounted for these differences, with no significant differences between the hyperactive-alone and control groups in any form of drug use.

Most studies concur with the Milwaukee study in finding that the elevated risk for substance use and abuse in adolescence is mostly, but not entirely, accounted for by comorbidity with conduct problems in childhood or a frank diagnosis of CD (August, Stewart, & Holmes, 1983; Barkley, Fischer, et al., 1990; Biederman et al., 1997; Chilcoat & Breslau, 1999; Claude & Firestone, 1995; Flory, Lynam, Milich, Leukefeld, & Clayton, 2001; Gittelman, Mannuzza, Shenker, & Bonagura, 1985; Kuperman et al., 2001; Lynskey & Fergusson, 1995; Mannuzza, Gittelman-Klein, Bessler, Malloy, & LaPadula, 1993; Molina et al., 1999; Realmuto et al., 2009; Wilson & Marcotte, 1996). Likewise, youth diagnosed with alcohol dependence have a markedly higher incidence of ADHD and CD, with a developmental sequence that is a progression from initial alcohol or tobacco use to marijuana, and finally to other street drugs (Kuperman et al., 2001). Such findings are quite consistent with studies of community samples in show-

ing that primarily CD, more than ADHD, is associated with greater risk for substance use, dependence, and abuse (Armstrong & Costello, 2002). But ADHD and its symptoms continue to contribute to a child's future risk for illicit substance use even after researchers control for conduct problems (Knop et al., 2009; Malone, Van Eck, Flory, & Lamis, 2010; Sihvola et al., 2011). Once again, it may be the attention deficit symptoms in ADHD that are most predictive of later tobacco and other substance use problems (Tapert, Baratta, Abrantes, & Brown, 2002). This greater use of drugs among youth with comorbid ADHD and CD may contribute to further problems with learning, memory retention, and attention (Tapert, Granholm, Leedy, & Brown, 2002). What recent studies do suggest is that there is a shared familial–genetic liability between ADHD and substance use disorders that may arise from a shared genetic network (Arcos-Burgos, Velez, Solomon, & Muenke, 2012; Biederman, Petty, et al., 2008). For a fine discussion of the possible mechanisms by which ADHD contributes to an increased risk for substance use disorders over the course of development, such as CD, school failure, deviant peer relationships, and risk-taking behavior, see the review by Eme (in press).

In one early study authors asserted that childhood treatment with stimulant medication increased the risk for substance use in adolescence, particularly for other stimulants (Lambert & Hartsough, 1998). More than 17 studies have since examined this issue, and all have failed to find that such childhood treatment with stimulant medication increased the later risk for excessive use of abuse of other substances, such as illegal use of stimulants (Barkley, Fischer, Smallish, & Fletcher, 2003; Barkley et al., 2008; Chang et al., in press; Wilens, Faraone, Biederman, & Gunawardene, 2003) and a few even found a small potentially protective effect if medication use was sustained into adolescence (Chang et al., in press; Wilens et al., 2003).

Studies of the drug use patterns of clinic-referred adults with ADHD have been far fewer in number and less consistent in their findings. Generally they indicate a significant two-way risk relationship between ADHD and substance abuse (Kalbag & Levin, 2005). Adults with ADHD are more prone to be diagnosed as having drug dependence or abuse disorders, particularly for alcohol and marijuana (Barkley et al., 2008). ADHD in adults in that study was specifically associated with a greater number of cocaine and LSD (lysergic acid diethylamide) users, as well as users of illegal prescription drugs, compared to both of our control groups. While the presence of a history of CD accounted for the elevated use of cocaine, it did not explain the greater likelihood of adults with ADHD using other substances. Other studies using self-reported information also found that adults with ADHD rate themselves as more likely to use or abuse drugs (De Quiros & Kinsbourne, 2001; Murphy & Barkley, 1996), primarily marijuana, cocaine, and psychedelics (Murphy & Barkley, 1996) as well as illegal prescription drugs (Barkley et al., 2008). Not only are substance use disorders more common in adults with ADHD, but adult ADHD symptoms are more common in drug users, and ADHD cast as a disorder may be three to seven times more likely (11–35%) to be present in adults with various substance use disorders (Carpentier, van Gogh, Knapen, Buitelaar, & De Jong, 2011; de los Cobos et al., 2011; Fergusson & Boden, 2008; Kalbag & Levin, 2005; Parrott et al., 2012; Romo, Kern, Mille, & Dubertret, 2012). The presence of ADHD in these cases appears to be associated with worse psychopathology and drug use (Arias et al., 2009; Carpentier et al., 2011). Such drug use may further worsen the attentional problems and ADHD symptoms over time (Fergusson & Boden, 2008; Tapert, Granholm, et al., 2002; Wilens et al., 2011).

The specific risk of excessive alcohol use or abuse by adults with ADHD has previously presented a mixed picture. In two small studies, Barkley, Murphy, and Kwasnik (1996) and De Quiros and Kinsbourne (2001) did not find a greater frequency of alcohol use or bouts of intoxication in their ADHD groups than in their control groups. But in later, larger studies, the ADHD group reported consuming significantly more alcoholic drinks per week, and getting drunk and using illegal drugs significantly more often in the previous 3 months than had the members of our control group (Barkley, Murphy, DuPaul, & Bush, 2002; Barkley et al., 2008). One study of Norwegian adults with ADHD also found higher rates of alcohol use disorders, as well as use of cannabis, amphetamines, and opiates (Torgersen, Gjervan, & Rasmussen, 2006). Therefore, while the relationship of adult ADHD to excessive alcohol use was not consistently corroborated in past research, more recent, larger studies confirm such a relationship.

GROWTH, HEIGHT, WEIGHT, AND EATING PATHOLOGY

For years, ADHD was not thought to be associated with problems with height, weight, or physical growth.

Indeed, if there were an association with weight, it was speculated to be a negative one due to the increased activity levels of children with ADHD, which one would expect to result in a lower body mass index (BMI). One earlier study examined a sample of 124 children and adolescents with ADHD for the presence of growth deficits in height and weight (Spencer et al., 1996). No evidence of weight deficits was found in the children with ADHD, even though 89% of the sample had been treated with stimulant medications, drugs thought to result in reductions in weight. There were small but significant deficits in height in the children with ADHD compared to their control group, but not between the adolescents with ADHD and their control group, implying that any growth delay may be time limited to childhood. These height deficits were not related to treatment with stimulant medications. The authors concluded that ADHD-associated temporary deficits in growth in height in childhood through midadolescence may no longer be evident by late adolescence. In contrast, a subsequent study of more than 7,000 children in France suggested that medication-naive children with ADHD were somewhat taller and heavier than were typical children, but no such differences were evident in adolescents (Faraone, Lecendreux, & Konofal, 2012). The authors speculated that this might reflect a problem with growth regulation in children with the disorder that is no longer evident by adolescents.

However, a study in the United States revealed a link between ADHD and greater body mass or frank obesity in the current generation of children with ADHD (Holtkamp et al., 2004). The authors found that nearly 20% of children with ADHD had a BMI at or greater than the 90th percentile, and more than 7% were at or above the 97th percentile. This difference between more recent and older research findings may reflect an interaction of personality traits (ADHD symptoms, especially impulsivity), with greater ecological availability of "junk" food for current as opposed to prior generations of children. Subsequently, Waring and Lapane (2008) surveyed more than 62,800 U.S. children and found that those with a prior history of a professional diagnosis of ADHD who had not received stimulant treatment for their disorder were 1.5 times more likely to be overweight. In contrast, those with ADHD who were being treated were 1.6 times more likely than typically developing children to be underweight. Recent research has also shown that boys diagnosed as hyperactive (ADHD) in childhood were twice as likely to be obese in midlife at a 33-year follow-up as boys in a control group followed for the same period of time (41 vs. 21%; Cortese et al., in press). Additional analyses showed that it did not matter whether the individual's ADHD had persisted at follow-up or remitted because these groups' rates of obesity did not differ from each other, but both had higher obesity rates than the control group. In my own follow-up study with Mariellen Fischer (analyzed for this chapter), in which we followed hyperactive boys to a mean age of 27 years, we found precisely the same results, with boys whose ADHD did or did not persist to that age having twice the rate of obesity (BMI ≥ 30) as boys in the control group (40% in each ADHD group vs. 20% for controls; see Barkley et al., 2008, for study details). Note that the rates of obesity are nearly identical in these two large-scale follow-up studies. In a study that documented the reverse association of ADHD and obesity, more than 57% of children hospitalized for obesity (BMI ≥ 85th percentile) were subsequently diagnosed with ADHD (Agranat-Meged et al., 2005).

Several recent studies have explored which symptoms of ADHD and other variables may account for children's risk of being overweight or obese. In a recent study of more than 11,600 German children, results indicated that the HI symptoms in particular showed a negative correlation with nutritional quality, and positive associations with greater high-energy food intake and greater exposure to television (van Egmond-Fröhlich, Weghuber, & de Zwaan, 2012). These findings suggest that these may be the variables that increase the risk for being overweight in children, especially girls, with ADHD. In a subsequent study, van Egmond-Frölich, Widhalm, and de Zwaan (2012) evaluated more than 17,600 German children and once again noted a relationship between HI symptoms and overweight and obesity in both sexes of children. But after controlling for possible confounding variables, such as socioeconomic status, parental BMI, and parental smoking, only the relationship between HI symptoms and being overweight for girls remained significant. Still to be disentangled, however, is the extent to which the obesity is related to comorbid disorders, such as anxiety and depression, that often coexists in a significant minority of patients with ADHD (Nigg, 2013). For instance, the recent report from the Ontario Child Health Survey follow-up study noted that although ADHD symptoms in childhood were significantly associated with greater BMI and obesity in adulthood, this risk was explained entirely by comorbidity with CD (Korczak, Lipman, Morrison, Duku, & Szatmari, 2014).

Though less studied, the relationship between ADHD and risk for being overweight or obese appears to hold true for adults. For instance, Barkley and colleagues (2008) documented significantly greater BMI in children whose ADHD had persisted to the young adult follow-up (mean age 27 years) in comparison to those whose ADHD had not persisted and the control group. Apart from such clinical samples, population-based studies in adults have likewise demonstrated what was found for children (discussed earlier): ADHD symptoms are significantly associated with risk for being overweight. Pagoto and colleagues (2009) assessed 6,735 U. S. adults ages 18–44 years. Those who had sufficient symptoms to warrant a diagnosis of adult ADHD were significantly more likely to be overweight (odds ratio = 1.58) or obese (OR = 1.81) compared to those with no history of ADHD symptoms. These relationships remained significant despite controls for demographic variables and degree of depression. Further analyses suggested that it may have been the link between ADHD and binge-eating disorder (BED) that mediated this risk for excess weight. As with children, the inverse relationship seems to hold true as well, with ADHD being overrepresented (28–32%) in patients being seeking treatment for obesity (Cortese, Comencini, Vincenzi, Speranza, & Angriman, 2013; Levy, Fleming, & Klar, 2009; Nazar et al., 2012), a prevalence far higher than that seen in the typical adult population (approximately 4–5%). Both the ADHD symptoms directly and the more general deficits in executive functioning may contribute to eating pathology and obesity and also foster unsuccessful efforts at weight loss (Cortese et al., 2013).

The link between ADHD and obesity may arise from several possible pathways as discussed by Nigg (2013) and earlier by Cortese and Penalver (2010; Cortese & Vincenzi, 2012). It is possible that the pathway is from obesity to ADHD or at least its attention problems as a function of sleep deprivation that can arise from disordered breathing or frank airway obstruction during sleep that may be more common in obese children. Nigg (2013) discounts this explanation due to the fact that ADHD often arises earlier than does obesity. Moreover, ADHD is not merely daytime inattention; thus, it is unclear whether the clinically complex disorder of ADHD is more likely to occur in children with such disrupted sleep. It is also possible that there exists some underlying shared genetic mechanism between ADHD and obesity, such as microdeletions in chromosome 11p14.1 that occur in both disorders (Shinawi et al., 2011). Another possibility may be shared intergenerational transmission of risk for both disorders perhaps through fetal programming effects. Most likely, as Nigg (2013) and others (Cortese et al., 2013) have noted, is that ADHD symptoms and related executive function (EF) deficits increase the likelihood of poor eating, more limited exercise, and more engagement with visual media, all of which may limit physical activity (discussed earlier), therefore leading to excessive weight. So people with ADHD should be evaluated for obesity and whether treatment of ADHD, as with medications, may reduce this risk, which seems to be the case (Waring & Lapane, 2008). This seems especially likely in view of the well-known anorectic effects of these medications, especially the stimulants (see also Chapter 27). Moreover, children and adults who are obese should be screened for ADHD because it is possible that treatment of their ADHD (e.g., with medication), may reduce their weight and future obesity risk, as has been the case with adult obese patients (Cortese et al., 2013; Levy et al., 2009). Moreover, stimulants such as methylphenidate have been found to reduce fat and carbohydrate intake in obese adolescents and may well do the same for obese teens with ADHD (Danilovich, Mastrandrea, & Quattrin, 2014).

As suggested earlier, ADHD seems to be linked not only to risk for being overweight or obese but also, at least in females, to a growing risk for eating pathology, particularly binge eating (BE), to more severe BE, and to its more extreme form, bulimia nervosa (BN; Nazar et al., 2012). Studies of children and adolescents, as well as adults, who have such eating pathologies likewise have long documented that impulsivity, as well as comorbid anxiety and depression, and parental obesity and eating pathology, are overrepresented and are likely risk factors that predispose individuals toward BE and BN (Nazar et al., 2012, 2014; von Ranson & Wallace, in press). In fact, some research indicates that 21% of those patients treated for BN had ADHD in childhood and were four times more likely to have a current diagnosis of adult ADHD (Seitz et al., 2013). The presence of ADHD was associated with more severe eating pathology and more symptoms of general psychopathology (Nazar et al., 2014; Seitz et al., 2013). Yates, Lund, Johnson, Mitchell, and McKee (2009), however, did not find such an overrepresentation, although they did report elevated symptoms of ADHD in at least 21% of women seeking treatment for an eating disorder. Problematic in such studies was the use of outdated DSM-IV criteria designed for children, which may have set too

high a symptom threshold to detect cases of ADHD in adults (Barkley et al., 2008; McGough & Barkley, 2004), leading to the lower threshold for diagnosing adult ADHD in DSM-5. But consistent with earlier studies that found such a link, in a large study of 12,366 twin pairs in Sweden, Råstam and colleagues (2013) found that children who rated high on eating problems scales had a higher prevalence of ADHD and/or autistic spectrum disorders (ASDs; 40%) in comparison to children without such eating problems. In girls, social interaction problems predicted risk for eating problems while in boys, impulsivity and activity level did so.

Some authors believe that it is not just the contribution of impulsivity to BE and BN that occurs in cases with ADHD. Such patients with comorbid ADHD symptoms may also demonstrate an increased reward sensitivity that may interact with impulsivity and inattention to heighten the likelihood of BE and BN eating pathology (Appelhans et al., 2011; Pagoto, Curtin, Appelhans, & Alonso, 2012; Seitz et al., 2013). Besides being linked to eating pathology, the presence of ADHD symptoms in people seeking treatment for obesity is also associated with more prior attempts to lose weight and less weight loss in those attempts (Pagoto et al., 2010). For these reasons, clinical researchers believe that ADHD may serve both as a marker for more severe eating pathology and comorbidity, and an obstacle to clinical management of eating pathology in patients who need treatment to improve their responsiveness (Levy et al., 2009; Nazar et al., 2014; Pagoto et al., 2012).

Given the totality of the evidence discussed earlier, it seems reasonable that the inverse would also be the case: People with ADHD, particularly those with elevated HI symptoms, are at risk for such eating pathologies. Only a limited amount of research has addressed this issue, but such risks became evident in large studies of girls with ADHD followed up in adolescence. Biederman and colleagues (2007) followed 140 girls with ADHD (ages 6–18) in Massachusetts for a period of 5 years and contrasted them with a control group of girls on measures of eating pathology. At follow-up, 16% of the ADHD girls met criteria for a current or past eating disorder (30% for anorexia, 50% for bulimia, and 20% for both AN and BN). The girls with ADHD were 3.6 times more likely to be diagnosed with any eating disorder and 5.6 times more likely to have BN specifically. Those with such eating disorders also had higher levels of depression, anxiety, and other disruptive behavior disorders, as well as earlier onset of menarche than girls with ADHD without eating disorders, consistent with previously discussed studies of girls with eating disorders. Similarly, in California, Mikami, Hinshaw, Patterson, and Lee (2008) followed 140 girls with ADHD and a control group of 88 girls over a 5-year period, evaluating them for body image dissatisfaction, eating pathology generally, and bulimic symptoms specifically. Girls with ADHD had larger BMIs than comparison girls; girls with the combined type of ADHD had significantly more eating pathology than did control girls, whereas girls with mainly the inattentive form of ADHD placed between these two groups. Consistent with earlier research, degree of impulsivity was a major predictor of risk for eating pathology by follow-up, as was degree of peer rejection and parent–child relationship problems (particularly punitive parenting). Sobanski and colleagues (2008) reported that 10–21% of their adults with ADHD, depending on subtype, had an eating disorder, compared to 0% of their control group. A recent review of the literature found eight studies of this issue of ADHD and eating pathology, five of which found a clear association (Curtin, Pagoto, & Mick, 2013). The authors concluded that youth with ADHD had a three to six times greater risk of developing an eating disorder and were significantly more likely than control groups to have eating pathology. Thus, the limited evidence to date suggests that ADHD in adolescent and adults, especially in females, may predispose to eating pathology generally and to binge-eating disorders specifically. Likewise, adults with BED or BN are more likely to have ADHD or significant problems with impulse control. Many of the researchers in these initial studies have recommended that clinicians specializing in the evaluation and treatment of either ADHD or obesity/eating pathology be aware of this comorbidity and screen for the overlap during their evaluations.

ELIMINATION DISORDERS

Enuresis, particularly nighttime bedwetting, was noted in early studies of hyperactive children to occur in as many as 43% of children, compared to 28% of normal children (Stewart et al., 1966). Two subsequent studies, however, did not find this to be the case (Barkley, DuPaul, & McMurray, 1990; Kaplan, McNichol, Conte, & Moghadam, 1987). But more recent studies do seem to suggest an overrepresentation of enuresis, at least in a minority of children with ADHD (11–30%) (Baeyens et al., 2006; Biederman, Santangelo, et al., 1995; Duric

& Elgin, 2011; Ghanizadeh, Mohammadi, & Moini, 2008). Enuresis seems to be 3.4 times more likely to occur in children diagnoses with ADHD (Baeyens et al., 2006). A study of Norwegian children seen in an outpatient clinic found that enuresis or encopresis was present in 15% of children with ADHD versus 3% of comparison children (Duric & Elgin, 2011). Among those children with both ADHD and enuresis, rates of oppositional defiant disorder (ODD) are significantly higher than in children with ADHD without enuresis (Baeyens et al., 2006; Ghanizadeh, 2010).

Hartsough and Lambert (1985) reported that children with ADHD are more likely to have difficulties with *bowel training* compared to normal children (10.1 vs. 4.5%), whereas Munir, Biederman, and Knee (1987) found that 18% had functional *encopresis*. As noted earlier, Duric and Elgin (2011) found that children with ADHD had a higher risk for encopresis–enuresis (15%) than did comparison children, but they did not report the rate for encopresis specifically. My colleagues and I were unable to replicate either of these findings, but our sample size was relatively small and therefore had limited power to detect such a small association (Barkley, DuPaul, et al., 1990). A recent population-based study using more than 735,000 children did find a small but significant relationship between children with ADHD and risk for constipation (4.1%) compared to children without ADHD (1.5%), as well as a more modest relationship of ADHD and fecal incontinence (0.9 vs. 0.15%) (McKeown, Hisle-Gorman, Eide, Gorman, & Nylund, 2013). Thus, children with ADHD are only slightly but significantly (statistically) more likely to have problems with constipation and encopresis, and this risk applies to a very small minority of cases.

SEIZURES, EPILEPSY, BRAIN ELECTRICAL ABNORMALITIES, AND NEUROFEEDBACK

A population-based study in Iceland employing a case–control design demonstrated a significant association between child ADHD and risk for *epilepsy and unprovoked seizures* (Hesdorffer et al., 2004). In this study, children with ADHD were 2.5 times more likely to have epilepsy or unprovoked seizures, particularly if they had the predominantly inattentive type of the disorder. The inverse relationship also held true; a history of ADHD (predominantly inattentive type) was found to be 2.5 times more common among children with epilepsy or unprovoked seizures. The finding of a link between epilepsy and ADHD was also replicated in Norway in a large study of children with ADHD (n = 607). Socanski, Aurlien, Herigstad, Thomsen, and Larsen (2013) noted a risk of 2.3% for epilepsy in this sample, which is more than four times the population prevalence for children of this age (0.5%). No research on adults with ADHD could be located on this issue, so it is unclear whether this same relationship holds for that age group.

Apart from this small but elevated risk for frank seizure disorders or epilepsy in children with ADHD, there is abundant evidence of abnormal electroencephalographic (EEG) patterns in children with this disorder (Hastings & Barkley, 1978; Loo & Makieg, 2012). While such findings are pertinent to the neurobiological etiology of ADHD, they also speak to the poorer neurological health and developmental status of children with the disorder. The most common finding in children and adults with the disorder is increased frontal–central theta band activity, believed to be associated with reduced arousal (Loo & Makieg, 2012). A meta-analysis of EEG studies (Snyder & Hall, 2006) found an effect size difference between ADHD and control groups of 1.31, which is substantial, with an average of 32% excess theta band power in the ADHD group. Not surprisingly, then, because the ratio of theta-to-beta band power is also substantially higher in ADHD cases than in controls, with an effect size greater than 3.0 (Loo & Makieg, 2012; Snyder & Hall, 2006), these two EEG measures are of great interest to clinicians and researchers seeking a laboratory measure that might prove useful in the diagnosis of ADHD (Snyder et al., 2008). Results of research examining the predictive accuracy of this ratio for the diagnosis of ADHD generally find an 84–95% accuracy rate, but with 16–18% of ADHD cases being misclassified as normal due to normal ratios (Loo & Makieg, 2012; Loo & Barkley, 2005; Snyder et al., 2008). Other researchers have found an accuracy rate considerably lower (58%) (Magee, Clarke, Barry, McCarthy, & Selikowitz, 2005), and in a recent meta-analysis, Arns, Conners, and Kraemer (2013) found much lower effect sizes for group differences on these measures and significant heterogeneity across studies, which argues against the use of these measures diagnostically. Such a mixed pattern of results and an elevated rate of false negatives in even the most supportive studies should reduce one's enthusiasm for using EEG alone as a diagnostic tool. Moreover, Loo and Makieg (2012) and others (e.g., Snyder et al., 2008) have cautioned that increased theta band

activity has also been associated with other disorders, such as bipolar and substance use disorders; it is therefore not specific just to ADHD. Moreover, theta–beta ratios cannot identify comorbidities in people with ADHD, which makes them poor stand-alone measures for diagnosis.

The findings of increased theta band activity and a higher theta-to-beta ratio on EEG led earlier clinical investigators, such as Joel Lubar (e.g., Lubar & Shouse, 1976) to speculate that operant conditioning of brain electrical activity might be a means of improving the symptoms of ADHD. A number of early studies indeed reported some promise for this intervention, leading to claims among practitioners that such neurofeedback treatment (NF) was as effective as medication, had no side effects, and produced permanent improvements in ADHD symptoms that lasted into adulthood in up to 80% of treated children. But these studies were not scientifically rigorous, often amounting to simply pre- versus posttreatment comparisons of treated children using various behavior rating scales and psychological tests, such as IQ tests or continuous performance tests. None of these early studies used sham, placebo, or alternative treatment comparisons groups, or employed blinded assessments of the children. More recently, better controlled studies have led to rather mixed results; some have found benefits in comparison to alternative or sham treatments, while others have not. Loo and Makieg (2012), among others, recently reviewed the literature on NF treatment for ADHD.

The reviewers found four studies that employed sham or placebo control treatments for comparison to children who received the NF protocol. These were the most rigorous studies included in their review. All kept parents blinded to the treatment conditions their children were receiving and three of the four studies kept therapists blinded as well. Results for all four studies found that both groups improved as a result of treatment, with no differences in improvement between them, which means that there was no specific treatment effect for NF. Of import in these studies is that there were no significant changes in the EEG and no differences in neuropsychological test performance, as well as no differences in parent or teacher ratings. This indicates that the very mechanism by which NF is argued to work—improving EEG activity in critical frequency bands—did not change in the NF and sham feedback groups. The fact that earlier studies using just waiting-list control groups and unblinded evaluations of NF effectiveness found apparent improvements as a result of NF, while placebo–sham-controlled, blinded and hence more rigorous studies do not, strongly suggests that much, if not all, of the improvement attributed to NF is actually nonspecific (therapist contact time, parental expectancy effects, participant motivation, etc.) (Loo & Makieg, 2012).

The reviewers located five additional studies in which NF was compared to another active treatment condition, such as another form of cognitive training software. Although some of these studies found that ADHD inattention significantly improved with each treatment, as rated by parents, the statistical analyses were questionable because they were biased toward finding a treatment effect (use of one-tailed tests), but none directly compared posttreatment scores between the groups. None of the studies found any improvements in teacher-rated ADHD symptoms in the school setting, and three of the four studies that used neuropsychological testing found no improvement in EF (Loo & Makieg, 2012). These results, coupled with those from the earlier placebo–sham-controlled studies are not especially supportive of a specific NF effect on ADHD symptoms, leading the reviewers to conclude that "studies reviewed herein do not support NF training as a first-line, stand alone treatment for ADHD" (p. 583). Moriyama and colleagues (2012) also reviewed this same literature, reaching similar though more optimistic conclusions and acknowledging that the more rigorous studies found little if any improvements in ADHD symptoms, yet continuing to view NF as a valid treatment option based largely on the more poorly conducted studies. In contrast, yet another review of this literature found that the promising results seen in randomized trials in which assessments were not blinded did not carry forward into subsequent studies using blinded evaluations and sham–placebo control groups (Lofthouse, Arnold, & Hurt, 2012). More recently, Sonuga-Barke and colleagues (2013) conducted a meta-analysis of various nonmedical interventions for ADHD, including NF. They only examined studies in which participants had been randomized to NF-treated and comparison groups (placebo, sham, active treatments). Their results indicated that studies using nonblinded ratings from people closest to the training setting (e.g., parents or therapists) produced significant results, but these comparisons became nonsignificant in better studies using blinded evaluations and examining treatment effects further from the treatment setting (e.g., school). Since these reviews were published, an additional study of NF involving a sham–placebo treat-

ment control group with blinded evaluations has been reported. It found that both groups improved significantly with their being no differences between them (van Dongen-Boomsma, Vollebregt, Slaats-Willemse, & Buitelaar, 2013). Despite promising results from uncontrolled or nonblinded studies of NF, more rigorous studies to date have not demonstrated that NF offers specific treatment effects beyond what can be attributable to various nonspecific factors. Thus, as Loo and Makieg (2012) concluded, NF is not established as a valid front-line, stand-alone intervention for ADHD at this time.

SLEEP PROBLEMS

Several early studies found that children with ADHD are more likely to have *sleeping problems* than are normal children. Difficulties with time taken to fall asleep may be seen in as many as 56% of children with ADHD compared to 23% of normal children, and up to 39% of children with ADHD may have problems with frequent night waking (see Corkum, Tannock, & Moldofsky, 1998, for a review; Greenhill, Anich, Goetz, Hanton, & Davies, 1983; Kaplan et al., 1987; Stein, 1999; Stewart et al., 1966; Trommer, Hoeppner, Rosenberg, Armstrong, & Rothstein, 1988). For example, Ball, Tiernan, Janusz, and Furr (1997) found that 53–64% of their ADHD group had sleep problems, as reported by parents, and that whether or not the children were taking stimulant medication did not seem to influence these results. Later studies have continued to support an association between ADHD and parent-reported sleeping disturbances (Hvolby, Jorgensen, & Bilenberg, 2009). This higher incidence of sleep difficulties may appear as early as infancy in children with ADHD (Stewart et al., 1966; Trommer et al., 1988), with as many as 52% described as such in infancy, compared to 21% of normally developing children. Resistance to going to bed, frequent night wakenings, and fewer total sleep hours may be the most obvious sleep difficulties that children with ADHD experience, as reported by parents (Hvolby et al., 2009; Stein, 1999; Wilens, Biederman, & Spencer, 1994). But difficulties with sleep onset and night waking are believed to characterize an unstable sleep pattern that has been shown to have a significant association with ADHD (Gruber, Sadeh, & Raviv, 2000). More than 55% of children with ADHD also have been described by parents as being tired on awakening, compared to 27% of normal children (Trommer et al., 1988). And children with ADHD manifest more frequent episodes of sleepiness during the day (Lecendreux, Konofal, Bouvard, Falissard, & Mouren-Simeoni, 2000).

Studies using objective measures of sleep, such as polysomnograms during overnight sleep, have not documented any difficulties in the physiological nature of sleep itself associated with this disorder (Ball & Koloian, 1995; Corkum et al., 1998; Cortese, Faraone, Konofal, & Lecendreaux, 2009; Lecendreaux et al., 2000). Sleep quality (objectively or electrically measured) does not seem to account for these parental reports of daytime tiredness and sleepiness, delayed sleep onset at nighttime, and disrupted sleep. Two meta-analyses of objective sleep measures (Cortese, Konofal, Yateman, Mouren, & Lecendreaux, 2006; Sadeh, Pergamin, & Bar-Haim, 2006) did find objective evidence that children with ADHD are more active during sleep, and have more daytime sleepiness and a greater Apnea–Hypopnea Index (AHI), despite their inability to find any evidence of electrical abnormalities on overnight polysomnograms. In a subsequent meta-analysis of 16 studies that also included subjective measures of sleep difficulties along with additional studies using objective measures, Cortese, Faraone, and colleagues (2009) found substantial evidence of ADHD-associated sleeping difficulties in parent reports of greater bedtime resistance, delayed sleep onset, more night awakenings, difficulties with morning awakening, and sleep-disordered breathing. Compared with controls, no differences were found for parasomnias, restless sleep, or sleep duration in children with ADHD. Objective measures also revealed greater sleep-onset latency, more frequent shifts in sleep stage per hour of sleep, and a higher AHI (disordered breathing) associated with ADHD. They also had lower sleep efficiency and less true sleep time, as assessed by various laboratory measures. But in the percentage of time spent in various sleep stages and other measures, such as night awakenings (assessed by actigraphy), children with ADHD did not differ from controls. Some of these results partially replicate the reports of parents concerning some of these sleep parameters while others do not. More recently, in a meta-analysis of 18 studies in which sleep-disordered breathing (SDB) was examined, Sedky, Bennett, and Carvalho (in press) found a significant association between ADHD and SDB. The total portrait here is one of considerable difficulties, with bedtime resistance, sleep onset, sleep efficiency, SDB/AHI, and daytime tiredness being linked to ADHD.

It seems likely, as noted by Cortese, Faraone, and colleagues (2009), that four other factors may operate here to contribute to the larger number of parent reports. Some of the parent-reported difficulties with bedtime resistance may be part and parcel of the difficult behavior and oppositionality often observed in children with ADHD rather than a reflection of true sleeping difficulties or disorders. Moreover, some research suggests that parents of children with ADHD may be more likely to have inappropriate sleep habits having to do with environmental management around bedtime, scheduling of bedtimes, and other factors that could impact bedtime difficulties (Owens, 2008). Parents may also overreport their children's bedtime problems due to the high level of behavior problems such children may manifest throughout the day; parents' impressions of these may carry over to late day/bedtime difficulties. However, some of the significant group differences on the objective measures noted earlier would suggest that this explanation cannot account entirely for the more numerous parental reports of sleep-related problems in children with ADHD because such children do have some sleeping abnormalities (onset, sleep stage changes, AHI, daytime sleepiness, etc.). Nor can the use of stimulant medication to treat ADHD and well known for producing insomnia account for these difficulties because children on medication were excluded from the Cortese, Faraone, and colleagues meta-analysis.

But it appears that some of these parent-reported behavioral difficulties surrounding children's bedtime are more a function of disorders that often are comorbid with ADHD (ODD, anxiety disorders) than of ADHD (Corkum, Beig, Tannock, & Moldofsky, 1997; Corkum, Moldofsky, Hogg-Johnson, Humphries, & Tannock, 1999). Or they may be nonspecific to ADHD, in that they characterize other behavior problems or learning disorders as well (Gregory & O'Connor, 2002; Marcotte et al., 1998). One such candidate is ODD, which is evident in a majority of children with ADHD. Another is anxiety/depression, which also exists in a substantial subset of children with ADHD. Mayes and colleagues (2009) studied the extent to which comorbidity accounts for sleep problems and found that only children with the combined type of ADHD had more parental reports of sleeping difficulties relative to controls and those with the inattentive type of ADHD. Interestingly, the inattentive type of ADHD was associated with sleeping more, not less, than controls. The presence of comorbid anxiety/depression increased the frequency of sleep problems, whereas ODD did not. Likewise, Hvolby and colleagues (2009) did not find ODD to be associated with parent-reported sleeping difficulties beyond those attributable to ADHD alone. Consistent with these results are other studies showing that anxiety in children can be associated with sleeping difficulties, especially night waking, whether or not it is associated with ADHD (Hansen, Skirbekk, Oerbeck, Richter, & Kristensen, 2011). A recent study also found that children with ASDs had elevated levels of sleeping difficulties that did not differ from the elevated rates in children with ADHD, although both groups had more sleep problems than did children with epilepsy or typical controls (Tsai et al., 2012). So comorbid conditions, such as anxiety/depression and ASD more than ODD, may well make some contribution to bedtime behavioral and sleeping difficulties apart from those attributable specifically to ADHD (Corkum et al., 1999; Gregory & O'Connor, 2002; Marcotte et al., 1998). This in fact also proved to be the case in a study by Willoughby, Angold, and Egger (2008), in which preschool children with ADHD, without regard to comorbidity, showed elevated levels of sleep assistance, parasomnias, and dyssomnias related to their level of HI symptoms. But once researchers controlled for comorbid disorders, these relationships became nonsignificant. Only the inattention symptoms of ADHD remained significantly associated with daytime sleepiness in these children, implying that need for sleep assistance, parasomnias, and dyssomnias seen in conjunction with ADHD are related to comorbidity.

A possible mechanism for some of the sleep-related difficulties evident in ADHD is the recent finding that children with ADHD have a more elevated level of certain polymorphisms of *CLOCK* genes than that seen in the typical population (Cao, Cui, Tang, & Chang, 2012; Dueck, Thome, & Haessler, 2012). Chinese children with ADHD were noted to have a more elevated incidence of the *T3111C* polymorphism of this *CLOCK* gene that was even more prevalent in children whose parent reported sleep disturbances than in those whose parent did not (Cao et al., 2012). The normal circadian rhythm is known to be affected by these *CLOCK* genes that also affect the sleep–wake cycle and release of hormones such as melatonin and cortisol (Dueck et al., 2012). Thus, there seems to be some promise in exploring in future research this relationship of genetic predispositions to sleep problems in ADHD.

One finding that may be characteristic of the sleep of ADHD children is greater movement during sleep (Corkum et al., 1998, 1999; Cortese, Faraone, Kono-

fal, & Lecendreux, 2009; Cortese et al., 2006; Porrino et al., 1983). While not evident in their meta-analysis, Cortese, Faraone, and colleagues (2009) note that the sample of such studies was limited, as was the reliability of objective measurement of movement, thus limiting their conclusions on this issue. Such findings of greater periodic limb movements during sleep in some studies using both parent report and objective measurement (Sadeh et al., 2006) have suggested a possible connection between ADHD and restless legs syndrome (RLS; Pichietti et al., 1999). That connection needs to be explored more fully, though one recent study also noted support for this relationship (Jesus et al., 2013). The authors found that such abnormal sleep movements, as well as difficulty transitioning to sleep, were related to low serum ferritin levels (iron) in children with ADHD (Cortese, Konofal, Bernardina, Mouren, & Lecendreux, 2009). However, other research indicates that low ferritin levels are more likely to be related to comorbid disorders than to ADHD generally (Oner & Oner, 2008), suggesting that it may be comorbid behavioral problems that mediate the findings of the Cortese, Konofal, and colleagues (2009) study.

Miano, Parisi, and Villa (2012) have hypothesized that the sleeping difficulties associated with ADHD may be subdivided into five phenotypes: "(i) a sleep phenotype characterized mainly by a hypo-arousal state, resembling narcolepsy, which may be considered a "primary" form of ADHD (i.e., without the interference of other sleep disorders); (ii) a phenotype associated with delayed sleep onset latency and with a higher risk of bipolar disorder; (iii) a phenotype associated with sleep disordered breathing (SDB); (iv) another phenotype related to restless legs syndrome (RLS) and/or periodic limb movements; (v) lastly, a phenotype related to epilepsy/or EEG interictal discharges" (p. 147). The management of the sleeping difficulties in these cases would be different and target the possible origin of the sleep problem, such as bipolar disorder, SDB, RLS, epilepsy, and so forth, rather than treating all ADHD-related sleeping difficulties the same. While both sensible and intriguing, to my knowledge, no research has examined either empirical evidence for such phenotyping (e.g., cluster analysis) or the benefits of these management ideas in any controlled fashion.

The quantity of sleep a child receives, as well as elevated symptoms of disordered breathing during sleep, are associated with teacher ratings of externalizing behavioral problems, particularly inattention (Aronen, Paavonen, Fjallberg, Soininen, & Torronen, 2000; Cortese, Konofal, et al., 2009). One study examined the relationship between different dimensions of children's psychopathology and different dimensions of sleeping problems (Stein, Mendelsohn, Obermeyer, Amromin, & Benca, 2001). Insomnia was the only sleep problem related to ratings of inattention, whereas noisy sleep was related to ratings of aggression, and parasomnias (sleep walking, nightmares, night terrors, head banging) were related to anxiety/depression, thought problems, and social problems. The latter finding may explain why past meta-analyses have not found consistent evidence that links parasomnias to ADHD; it is more likely a function of comorbidity. Some authors have argued that this means that sleep problems may be contributing to psychopathology in children (Aronen et al., 2000). But the direction of effect in these studies is unclear given the correlational nature of these findings. Is limited sleep (insomnia) a direct contributor to school behavioral problems and inattention, or is it that children who are more likely to misbehave and be inattentive are also more likely to have difficulties getting to sleep at night and with other facets of sleep? These correlation-based results simply do not provide an answer.

One study, however, did manipulate sleep quantity, while examining its impact on daytime behavioral problems in normal healthy children (Fallone, Acebo, Arnedt, Seifer, & Carskadon, 2001). Children whose sleep had been restricted to 4 hours on one occasion were found the next day to have increased inattention but not increased hyperactive or impulsive behavior. Nor did they have impaired performance on a laboratory measure of inattention and impulsiveness. The study suggests that limited sleep may well increase inattentiveness in children, but the short duration of the sleep manipulation may have limited the researchers' ability to test this relationship between sleep and other behavioral indicators. Sadeh, Gruber, and Raviv (2003) restricted the sleep of normal children by 1 hour over three consecutive nights and did find an effect on laboratory measures of neurobehavioral functioning (attention, inhibition, etc.). Also of interest is the recent finding that whereas reduced sleep may well be associated with inattentiveness in healthy normally developing children, it is not related to the behavioral symptoms in children with ADHD (Gruber & Sadeh, 2004). These results suggest that the likely causal connection between disrupted sleep and inattention that may be evident in normal children does not arise from the same mechanism(s) as what may exist between

ADHD and its associated sleep problems. Indeed, some research suggests that the pattern of objectively assessed sleeping difficulties that is evident in ADHD is not the same as that in children with sleeping problems, and that when ADHD and sleep problems are comorbid, the pattern of findings is also distinct from that for either condition alone (Sawyer et al., 2009). The authors concluded that the neurological difficulties that may be contributing to sleep difficulties are not the same as those associated with ADHD, or with ADHD plus sleeping disturbances. This should give some pause to clinicians considering the widespread use of tonsillectomies or other interventions for improving disordered breathing during sleep as a means of addressing the daytime inattention of children with clinically diagnosed ADHD. Both clinical data and a recent meta-analysis of adenotonsillectomy for SDB (Sedky et al., in press) suggest that improvement in ADHD may be seen within a year after such surgery (Chervin et al., 2006). Sedky and colleagues (in press) recommended screening all children with ADHD for possible SDB given its greater frequency in children with ADHD. If SDB is present, they suggest that surgical intervention be considered. More rigorous randomized trials of this treatment have not been done, yet clearly they are necessary before considering surgical intervention in children with ADHD and SDB.

One intervention for delayed sleep onset in ADHD that has been studied is the use of sublingual melatonin (Van der Heijden, Smits, Van Someren, Ridderinkhof, & Gunning, 2007) prior to bedtime. Improvement in sleep onset was reported (averaging nearly half an hour earlier) in children with ADHD receiving the melatonin compared to placebo, in which the sleep onset actually lengthened during the study period. However, no improvement in bedtime behavior, cognition, or quality of life was associated with the intervention condition. A recent review of the literature on medication interventions for sleep problems in children with ADHD concluded that clonidine and L-theanine also improved sleep, as did melatonin, while zolpidem was not effective and had substantial side effects associated with its use (Barrett, Tracy, & Giaroli, 2013).

In a recent study, Jesus and colleagues (2013) found results that suggest ADHD medications may result in reduced sleep problems in children with ADHD. But the study was purely a naturalistic one and did not involve randomization to treated and untreated groups, so the conclusion that treating ADHD with medication improves sleep problems needs to be studied fully.

Another type of intervention for bedtime problems was evaluated in a study by Sciberras and colleagues (Sciberras, Fulton, Efron, Oberklaid, & Hiscock, 2011) who invented a behavioral sleep training program to help parents reduce the sleeping difficulties of children with ADHD. In a pilot study using a randomized trial of small samples, they compared two groups of families whose children with ADHD had sleep problems. Families received either one session of counseling on behavioral strategies to employ at bedtime and overnight (n = 13) or two to three sessions (n = 14). At posttreatment most parents, regardless of treatment group, reported the strategies to be useful, and at 5 months postrandomization, 67% of parents in both groups stated that their children's sleeping problems had resolved. There were also improvements in children's quality of life and daily functioning, and parental anxiety, but there was little change in children's ADHD symptoms.

To date, very few studies have specifically examined sleeping difficulties in adolescents with ADHD; they are typically lumped in with younger children in prior research. But one study that did so found no greater frequencies of various sleep difficulties in a teen group with ADHD than in a control group (Stein et al., 2002). Only stimulant medication status was associated with elevated sleep difficulties in the ADHD teen group. However, depression was significantly associated with sleeping difficulties in these adolescents, a finding that is consistent with other results suggesting that, with age, it is depression, not ADHD, that is more predictive of sleeping difficulties from adolescence to adulthood (Gregory & O'Connor, 2002).

Likewise, only a few studies have evaluated the sleeping difficulties associated with ADHD in adults. Surman and colleagues (2009) compared large samples of both adults with ADHD and adults in a control group. They found that adults with ADHD went to bed later; had a wider range of bedtimes; were more likely to take over an hour to fall asleep; and reported more difficulties going to sleep, sleeping restfully, and waking in the morning. They also reported more daytime sleepiness than did control adults. These differences remained despite researchers'controls for use of ADHD medications (which can induce insomnia), age, and comorbidity with other psychiatric disorders. Similar results were reported in a population-based survey of a large sample of adults in Romania, in which severity of ADHD symptoms was associated with greater insomnia, longer latencies to sleep onset, shorter sleep duration, and more frequent (unwanted) awakenings during sleep, as well

as an evening orientation to their circadian rhythm (Voinescu, Szentagotai, & David, 2012). These difficulties are similar to those seen in the studies of children with ADHD discussed earlier. Two studies suggest that the sleep quality in adults with ADHD is associated with the HI symptom dimension (especially impulsivity) and not with the inattentive dimension (Mahajan, Hong, Wigal, & Gehricke, 2010; Voinescu et al., 2012). It is possible that the longer latencies to sleep and greater insomnia associated with adult ADHD is linked to delayed circadian rhythm, in which these adults show a strong evening over morning preference (41 vs. 18%; Rybak, McNeely, Mackenzie, Jain, & Levitan, 2007).

INTERNET USE AND ADDICTION

The increasing availability of computers, computer games, and the Internet and Web-based gaming networks has opened up the possibility for a new domain of health- (and mental health)-related difficulties for people, such that, on average, 9% of youth may qualify as Internet or video game addicts or as having problematic Internet usage (Gentile et al., 2011). Those with ADHD may be especially prone to such addictions—a risk that was not available to prior generations of children with ADHD. It can be easily reasoned that individuals who have difficulties with impulse control and self-regulation, such as those with ADHD, may find that the opportunities the Internet affords for stimulation seeking and risk taking can lead to a new source of "addictive behavior" (Gentile et al., 2011; Weiss, Baer, Allan, Saran, & Schibuk, 2011). Moreover, people who have difficulties with face-to-face interpersonal behavior, such as occurs in ADHD, may find the greater anonymity of the Internet and the opportunity to create an alternate persona, to provide an alternative means of socializing. In the past 5 years, more than 58 studies have been done on internet addiction (IA) in general (Kuss & Griffiths, 2012) with more than 21 studies examining the role of ADHD, among other psychiatric symptoms, in the risk for addiction (Carli et al., 2013; Ko, Yen, Yen, Chen, & Chen, 2012).

Among the first articles to appear on this topic relative to ADHD was a U.S. study in which 72 teens and their parents were interviewed about Internet and computer video game use in relation to symptoms of ADHD. The study found significant associations between gaming more than 1 hour per day and elevated symptoms of ADHD, particularly inattention (Chan & Rabinowitz, 2006). Later, in a study in France, Bioulac, Arfi, and Bouvard (2008) rated a small sample of children with ADHD and control children, ages 6–16, on the Problem Video Game Playing Questionnaire (PVP), which can be used to define addictive video game playing. The symptoms of such addiction were adapted from DSM-IV criteria for substance dependence and for gambling addiction. The findings did not show any differences in the frequency or duration of play between the two samples. Control children often played less than once a week, whereas one-third of the children with ADHD played between one and three times a week, but the groups did not differ statistically in that respect. The time spent per session also did not differ, with most children spending between 1 and 2 hours (65% for ADHD, 50% for controls). The authors also reported that the two groups did not differ with regard to the type of video game, since both groups played action and reflection games (adventure games, role playing games, logic games). However, they did find that, according to the parents, the children with ADHD were less likely than controls to stop playing of their own accord (59% of the ADHD group vs. 90% of the control group). About three times as many children with ADHD reacted to such parental restrictions on game use with refusal, anger, violence, or tears (59 vs. 19%). On the PVP, 34% of children with ADHD had scores of 5 or more problematic symptoms, which is sufficient to be considered addicted to video games, compared to none of the control children. Children with ADHD meeting such criteria differed from those who did not in that they had more severe hyperactivity/ADHD symptoms and higher parent ratings of aggression and conduct problems. In a subsequent study, Bioulac and colleagues (in press) examined the nature of video game performance between small samples of children with ADHD and control children, and reported no evident differences across three types of games despite the groups' differing performances on a continuous-performance test. The small sample sizes, however, greatly limited the power of this study to detect less than large effect sizes. Thus, problematic video game playing or addiction may be significant problems for children with ADHD, even if the quality of their play does not differ from that of control children.

But what of Internet use and the forms of gaming it permits? Studies on this topic have mostly been done in Asia, and they repeatedly support an association between ADHD and some of its comorbid conditions and IA (Carli et al., 2013; Ko et al., 2012). For instance,

Yen, Ko, Yen, Wu, and Yang (2007), in a sample of 2,114 students, found that in males and females, both ADHD symptoms and depression were associated with hostility, making an additional contribution to risk in males. In a subsequent large study surveying 2,293 students across 10 different junior high schools in Taiwan, using the Chen Internet Addiction Scale, Ko, Yen, Chen, Yeh, and Yen (2009) evaluated their sample across four time periods (upon entry, 6 months, 12 months, and 24 months later). IA symptoms, such as preoccupation with the Internet, uncontrolled impulses to use it, usage more than intended, tolerance, withdrawal, impairment of control, excessive time and effort spent, and impairment in decision-making ability were captured by the Chen scale using 26 items rated on a 4-point Likert scale. Scores of 64 or higher were defined as representing addiction. The authors found that ADHD symptoms and hostility were the most significant predictors of IA in both males and females. Although ADHD was the most important predictor among females with IA, depression and social phobia were lesser but also significant predictors. By the 2-year follow-up, ADHD, depression, social phobia, and hostility were predictive of IA. Similar results were more recently observed in Turkish university students where degree of ADHD symptoms, especially HI, was significantly associated with degree of IA even after researchers controlled for anxiety, depression, and personality traits (although these factors were also linked to IA after they controlled for ADHD as well) (Dalbudak & Evren, 2014).

Ko and colleagues (2009) reasoned that ADHD may be an important determinant of IA for various reasons, including subjects' poor impulse control; impaired interpersonal socializing; penchant for greater hostility, which can be expressed through Internet war gaming; and the fact that for those with social phobia, the Internet may be a less threatening form of socializing than are face-to-face encounters. Also, Internet gaming may increase the release of dopamine into the brain, which may temporarily compensate for the dopamine deficiency believed to be involved in some cases of ADHD (Koepp et al., 1998) and may therefore be a form of self-medication. The authors further speculated that the screening and treatment of ADHD and these related predictors may be needed as part of any effective intervention for IA, and that those studying patients with ADHD and these other conditions must be aware of their increased risk for such addiction.

Computer game addiction appears to share some similar risk factors with other forms of addiction, such as substance use and gambling, in a recent study of 16- to 25-year-old German youth (Walther, Morgenstern, & Hanewinkel, 2012). In particular, problem gaming appears to link up with cannabis use rather than tobacco or alcohol use. The most common personality trait linked to five forms of addictive behavior, including gaming, was high impulsivity, perhaps explaining why those with ADHD may be more prone than others to various forms of addiction, including gaming. But computer game addiction was also associated with irritability/aggression, social anxiety, and low self-esteem, in agreement with the study by Ko and colleagues (2009) on the importance of these factors in predicting Internet and video game addiction. In a small study of adults diagnosed with IA, Bernardi and Pallanti (2009) found a higher than expected prevalence of ADHD, along with other disorders, including anxiety, obsessive–compulsive disorder (OCD), dysthymia, and personality disorders. These findings were largely replicated in a sizable study of IA in Taiwanese college students (n = 2,793), in which ADHD was the most significant predictor of IA, followed by general impulsivity (Yen, Yen, Chen, Tang, & Ko, 2009). A recent review of 20 studies on the topic of problematic Internet use (PIU) likewise identified ADHD as the most consistent predictor across these studies (100% of studies), followed by depression (75% of studies), hostility/aggression (66%), obsessive–compulsive symptoms (60%), and anxiety (57%), with males being more prone to this form of problematic behavior than females (Carli et al., 2013). The review concludes that symptoms of depression and ADHD have the most significant and consistent correlations with PIU. A similar conclusion was reached in the review of much of this same literature by Ko and colleagues (2012). Several longitudinal studies suggest that while greater amounts of gaming, high impulsivity, and low social competence may be precedents to and risk factors for pathological gaming, depression, suicidality, anxiety, social phobia, and low school achievement might well be the consequences of such gaming (Gentile et al., 2011; Messias, Castro, Saini, Usman, & Peeples, 2011). One study suggests that methylphenidate treatment of children with ADHD who play video games may decrease their symptoms and game usage (Han et al., 2009). And while not specifically targeting IA or gaming addicts with ADHD, literature reviews suggest that cognitive-behavioral therapy combined with family therapy, as well as psychopharmacology, might be the most efficacious intervention for youth who are IA or video game addicts (King, Delfabbro, & Griffiths, 2012; Liu, Liao, & Smith, 2012; Winkler,

Dorsing, Rief, Shen, & Glombiewski, 2013), approaching the disorder as one would other, more established forms of addiction.

Given that children with ADHD are more likely to be aggressive and hostile than other children, and that these factors increase the use of video gaming, including violent games, one might ask whether playing violent video games feeds back to increase such aggressive behavior. In a study of 377 children with elevated ADHD and depression symptom scores, however, those who play such games were not more likely to engage in delinquent behavior or bullying than those who do not play these violent games (Ferguson & Olson, 2014). This lack of association also seems to be the case for typical youth, in whom longitudinal data do not support an adverse effect of video game violence on either violent behavior or academic abilities (Ferguson, 2011; Ferguson, Garza, Jerabeck, Ramos, & Galindo, 2013). Instead, depression and antisocial behavior in youth are better prospective predictors of later violence than exposure to violent video games or violent media (Ferguson, 2011). In contrast to this, in a study in Pakistan that surveyed the opinions of teachers on the role of exposure to violent media and behavior problems in children with ADHD, the vast majority of teachers believed that media violence has an adverse affect on these children (Kazimi, Sadruddin, & Zehra, 2013). Of course, this study provides no evidence that teachers are correct in holding such opinions, only that they believe this relationship holds true. Thus, the results of the limited research to date do not support the belief that exposure to violent gaming specifically and violent media more generally has an adverse impact on children with ADHD, although teachers (in Pakistan) seem to hold such an opinion.

In conclusion, Internet and video game use and addiction are problematic for a substantial proportion of both children and adults with ADHD, and those who engage in the excess use of such media are more likely to have elevated symptoms of ADHD, among other disorders. More research is needed on treatments for these difficulties, but at least one study suggests that ADHD medications may help to reduce the time people spend in these media.

ACCIDENTAL INJURIES

Children with ADHD are considerably more likely to experience a variety of physical injuries due to accidents than are normal children (for reviews, see Barkley, 2001; Nigg, 2013). Early researchers found that up to 57% of children with ADHD are described as accident prone, and 15% have had at least four or more serious accidental injuries, such as broken bones, lacerations, head injuries, severe bruises, lost teeth, or accidental poisonings (Hartsough & Lambert, 1985; Mitchell et al., 1987; Reebye, 1997; Stewart et al., 1966). Results for the comparison or normal groups of children in these early studies were 11 and 4.8%, respectively. Stewart, Thach, and Friedin (1970) found that 21% of hyperactive children had experienced at least one accidental poisoning compared to 7.7% for typically developing children. In a much larger study of more than 2,600 children, Szatmari and colleagues (1989) found that 7.3% of children with ADHD children had experienced an accidental poisoning and 23.2% had suffered bone fractures compared to 2.6 and 15.1%, respectively, in the control group. Leibson and colleagues (2001) reported an elevated risk for major injuries among children with ADHD (59 vs. 49%) using a large birth cohort. Consistent with this finding, Swensen and colleagues (2004) also found a higher incidence of accident claims among children (28 vs. 18%) and adolescents (32 vs. 23%) with ADHD when they examined medical claims for a large population of employees of national manufacturers.

The injuries sustained by children with ADHD may also be more severe and occur more frequently. For instance, Mangus, Bergman, Zieger, and Coleman (2004) examined children admitted over a 7-year period to a regional pediatric burn unit and found that those with ADHD had a greater likelihood of a thermal rather than a flame burn, more extensive burn injuries, and a greater length of stay in the unit. Hoare and Beattie (2003) compared children with ADHD and control children who had attended an accident and emergency department in Edinburgh, Scotland, and noted that children with ADHD were more likely to attend because of injury, and that they had a greater frequency of injury, as well as differences in type of injury (head, wound–laceration, poisoning). It seems clear, then, that children with ADHD have an elevated risk of physical injury, of more frequent injuries, and of more severe injuries than do normal children. More recent studies continue to bear out the significant association between childhood ADHD and physical injuries (Badger, Anderson, & Kagan, 2008; Marcus, Wan, Zhang, & Olfson, 2008; Merrill, Lyon, Baker, & Gren, 2009; Pastor & Reuben, 2006). Population-based surveys of parents indicate that children with ADHD are 1.8 times more likely to experience a physical injury each

year than are control children (20.4 vs. 11.5% per year; Pastor & Reuben, 2006).

Some early retrospective and prospective studies generally found a relationship between degree of aggressiveness, not the degree of overactivity, and the likelihood of accidental injury in preschoolers (Davidson, Hughes, & O'Connor, 1988; Langley, McGee, Silva, & Williams, 1983). Because children with ADHD are more likely to be aggressive or oppositional, it may be that this characteristic increases their accident proneness rather than their higher rates of activity level or impulsivity (Langley et al., 1983). Yet a large population study of 10,394 British children found that both overactivity and aggression contributed independently to the prediction of accidents (Bijur, Golding, Haslum, & Kurzon, 1988). The linkage between childhood aggression and injury risk also has been demonstrated in more recent large population studies (Carlson et al., 2009). In a different study, Lalloo, Sheiham, and Nazroo (2003) examined 6,000 children in England and found that only hyperactivity was predictive of an increase in accidental injury, once they controlled for demographic and socioeconomic factors. Since the latter factors are more likely to be related to childhood aggressiveness, controlling for them may explain why aggressiveness itself was no longer predictive of accident risk in this study. Rowe, Maughan, and Goodman (2004), in a study of injuries among more than 10,000 children in Britain, found that ADHD was most likely to be related to fractures, while ODD was more related to burns and poisonings. Recent research shows that the risk of injury may be five times higher in children diagnosed with ADHD than in those without the disorder (van den Ban et al., 2014). Thus, both ADHD and aggression or ODD are linked to accidental injuries but of different forms in most studies. Yet the relationship to child psychopathology generally and ADHD specifically is not always evident (Dudani, Macpherson, & Tamin, 2010). In summary, the totality of evidence indicates that there is likely to be a direct association of ADHD specifically and externalizing behavior more generally with increased physical injuries that is independent of comorbidity for other disorders. Perhaps this is why children placing in the upper quartile of externalizing behavior problems are more than twice as likely to die by age 46 as children in the lowest quartile (Jokela, Ferrie, & Kivimaki, 2008).

Adults with ADHD also demonstrate an increased risk of physical injuries apart from those that may be associated with driving accidents (discussed below; Merrill, Lyon, Baker, & Gren, 2009; Swensen, Allen, Kruesi, Buesching, & Goldberg, 2004). The few studies of this issue indicate that such adults are more likely to file claims for accidental injury and poisoning, and to exhibit a greater rate of injury.

And what of the inverse relationship? Do children who experience more accidental injuries show an elevated level of ADHD? More than 40 years ago, research suggested that children experiencing accidents are more likely to be overactive, impulsive, and defiant (Cataldo et al., 1992; Rosen & Peterson, 1990; Stewart et al., 1970). Pless, Taylor, and Arsenault (1995) found that children injured as pedestrians or bicycle riders in traffic accidents performed more poorly on tests of vigilance and impulse control, and received higher parent and teacher ratings of hyperactive–aggressive behavior. More recent research has continued to show that a substantial minority (28.6%) of children admitted to hospitals as a consequence of physical injury related to falls, auto–bicycle, auto–pedestrian, or bicycle accidents are likely to have ADHD (Maxson, Lawson, Pop, Yuma-Guerrero, & Johnson, 2009). These studies suggest that a high percentage of those experiencing such serious accidents may have ADHD or greater-than-normal ADHD symptoms. In short, children admitted to hospitals for injuries are three times more likely to have ADHD than those admitted for other reasons, such as appendicitis (Nigg, 2013). And among children with ADHD who may suffer a traumatic brain injury (TBI) specifically, they are more likely to be disabled and more severely disabled following the injury than are children without ADHD who experience TBI, even after researchers control for other possible mediating variables (Bonfield, Lam, Lin, & Greene, 2013).

Why do those with ADHD apparently have a greater risk for accidents, accidental injuries (non-head), and accidental poisonings than those without the disorder? Obviously, the symptoms of the disorder contribute to such risk and may explain the greater risk of injury to males than females given the greater preponderance of ADHD symptoms in male children (Karazsia, Guilfoyle, & Wildman, 2011). Parents report that their children with ADHD are inattentive while engaging in risky activities and are more heedless or thoughtless of the consequences of their actions (impulsive), thus placing themselves in situations or engaging in activities that are more likely than usual to result in physical harm. In population-based studies, hyperactivity (Junger, Japel, Cote, Xu, Bolvin, & Tremblay, 2012) and impulsivity are specifically associated with injury risk

(Rowe, Simonoff, & Silberg, 2007). But there may be other reasons as well that deserve consideration, such as the following:

- *Motor incoordination.* As discussed in other chapters, children with ADHD demonstrate greater motor clumsiness, awkwardness, and more rapid and ill-timed motor movements than other children. They also demonstrate slower reaction times than typically developing children. It is not hard to see how such clumsiness or even developmental coordination disorder (DCD) might contribute to accident risk, particularly in an already impulsive group of children (Rowe et al., 2007).

- *Comorbid ODD and CD.* Another, more important contributor may be comorbidity for ODD and CD. As I have repeatedly noted, children experiencing accidents are frequently more aggressive, defiant, and oppositional than other children, or at least they pose more discipline problems for parents. And children with externalizing symptoms such as aggression and antisocial behavior are more likely to experience injuries across their lifetimes into adult midlife (Jokela, Power, & Kivimaki, 2009). ODD and CD, as stated earlier, are far more common in children with ADHD and may contribute to even greater risk for accidents and injuries than would be the case in children with ADHD alone. Indeed, some have argued that this pattern of defiant and aggressive behavior is far more contributory to accident risk than is hyperactivity, although other studies indicate that each makes an independent contribution to injury risk, as noted earlier.

- *Comorbid anxiety and depression.* Cross-sectional and longitudinal research have repeatedly indicated a link between internalizing symptoms (anxiety and depression) and increased risk for unintentional injury (Carlson et al., 2009; Marcus et al., 2008; Rowe et al., 2007), and that the internalizing symptoms are not the consequence of such injuries (Rowe et al., 2007). Since ADHD is associated with a higher likelihood of anxiety and depression (see Chapters 5 and 13), it is possible that ADHD remains the mediator in this relationship. However, researchers who control for comorbidity continue to find anxiety and depression to be factors in injury risk (Carlson et al., 2009; Rowe et al., 2007). It is unclear why higher levels of anxiety or depression would increase injury risk, but one proposed mechanism is that, like ADHD, psychopathology more generally is associated with greater motor clumsiness and, as noted earlier, this may increase the risk for injury (Rowe et al., 2007). Anxious or depressed children may also be more inattentive and mentally preoccupied, which might create higher risk for injury, though this remains conjectural. However, other longitudinal research has indicated a reduced risk of accidental injuries, particularly in sports, traffic, and home settings, in children with higher internalizing symptoms (Jokela et al., 2009), so further research on the nature and direction of this relationship seems to be in order.

- *Diminished EF.* Children and adults with ADHD demonstrate substantial and varied deficits in the various components of EF, as discussed in other chapters in this volume. Indeed, on rating scales of EF in daily life, the vast majority of children and adults with ADHD demonstrate substantial deficits in the components of EF (Barkley, 2012; Barkley & Murphy, 2011). Research using simulated pedestrian–automobile situations indicates that children with low behavioral control more generally (Barton & Schwebel, 2007) and ADHD specifically are more likely to choose riskier environments (smaller traffic gaps) in which to cross streets and are therefore more susceptible to pedestrian–auto accidents than other children, despite demonstrating normal curbside behavior (waiting, looking both ways before crossing, etc.; Stavrinos et al., 2011). The latter study also found that deficits in EF (decision making) were associated with this risk, in that children with ADHD did not evaluate the information in the immediate crossing situations as posing as much risk to them as did control children, perhaps due to greater distractibility or because they were delay averse or less patient. Clarifying the nature of this relationship of child EF factors and injury risk, research indicates that both risk taking and errors in judgment, perhaps due to inattention, are both contributors to injury risk and mediate the link between conduct problems and hyperactivity with injury risk (Rowe & Maughan, 2009).

- *Parental characteristics.* A few studies of children's accidents, particularly those taking place out of doors, suggest that parents of these children may supervise their children's play activities less than other parents. Accident proneness is therefore moderated by certain parental characteristics, such as degree of monitoring of child behavior, low maternal conscientiousness, maternal neuroticism, maternal antisocial behavior, and low paternal self-control (van Aken, Junger, Verhoeven, van Aken, & Dekovic, 2007; Davidson et al., 1988; Davidson, Taylor, Sandberg, & Thorley, 1992; Junger et al., 2012). Other researchers using a controlled "hazard

room" situation observed that mothers of children with behavior disorders monitored their children less often in such hazard-prone environments than did mothers of control children (Schwebel, Hodgens, & Sterling, 2006). Parents of hyperactive or injury-prone children play less often with them, allow their children out of their homes for longer periods of time, and let their children go to school alone more often than do parents of control children. For instance, Schwebel, Brezausek, Ramey, and Ramey (2004) found that among children at high risk for injury (males, hyperactive children, families in poverty), positive parenting and greater availability of time to be with children were protective of risk for injuries. Though far more research remains to be done on the issue of parental supervision and its quality in relation to accident risk, current evidence suggests that parental characteristics, particularly low monitoring, may be risk or protective factors, and that such monitoring may be less adequate in parents of children with ADHD, probably because more such parents have ADHD and other psychopathology themselves (see Chapter 7).

Worth considering here is that current research also suggests that predictors of single unintentional injuries in children may differ from those for repeated unintentional injuries (Junger et al., 2012), such that mother's antisocial behavior and older age at first birth, as well as child sex (male) and being hyperactive, are associated with single unintentional injuries. In contrast, maternal smoking during pregnancy, nonmedical prescription drug use, single motherhood, and possibly self-efficacy, as well as an interaction between child sex and temperament (male with negative temperament) predicted repeated unintentional injury. Other research shows that living with a single parent or no parent likewise increases injury risk over time (Dudani et al., 2010).

These and other factors are worth the consideration of clinicians in efforts to reduce the injury risk to children with ADHD. Available evidence is quite limited, but it implies that ADHD medications do not completely reduce such injury risks even though nonsignificant reductions are evident (van den Ban et al., 2014; Marcus et al., 2008; Merrill et al., 2009). The previously discussed factors that accentuate accident risks may explain why ADHD medications are unlikely to completely reduce that risk given that those medications do not influence factors such as comorbidities for internalizing disorders or parental characteristics that may contribute to injury risk beyond child ADHD. Thus, other interventions may be needed, beyond simply medicating patients with ADHD so as to reduce symptoms, then assuming, wrongly, that such symptom reduction translates into a normalization of injury risk.

CHILD ABUSE

Only minimal research has examined the extent to which children with ADHD are abused. That they are abused should not be surprising given that increased aggression, externalizing disorders, impulsivity, and other common characteristics associated with ADHD are linked to risk for abuse (Margolin & Gordis, 2000). Moreover, the parents of children with ADHD are more likely to have ADHD themselves and problems with parenting, to report more parenting stress and less parenting competence, and to experience marital distress or divorce, and more depression than other adults (see Chapter 7), all of which can heighten the risk of such parents abusing a child. In an early study of this issue, Wozniak and colleagues (1999) did not report a link among ADHD, child abuse, and posttraumatic stress disorder (PTSD), but examination of the results indicates that their ADHD group had suffered more exposure to traumatic events, including sexual abuse, physical abuse, and witnessing domestic violence, than did the control group (7.0 vs. 0.1%, respectively). The lack of any significant difference here may be due to the fact that the authors did not disaggregate the types of possible abuse to which the children may have been exposed (Briscoe-Smith & Hinshaw, 2006).

Ford and associates (1999) examined a large sample (n = 165) of children seen in an outpatient psychiatric clinic for trauma exposure (both victimized and nonvictimized events). While they found an initial association of both ADHD and ODD to having been exposed to victimization trauma, they found no relationship between ADHD and trauma exposure after appropriately controlling for various child and family factors that could potentially confound this relationship. However, ODD remained significantly associated with likelihood of victimization trauma events, regardless of whether it coexisted with ADHD. This makes sense given the frequent association of ODD with disrupted parenting, family social adversities, and parent psychopathology (see earlier discussion of ODD/CD). Indeed, Ford and colleagues found that family psychopathology was a significant predictor of victim-

ization in the study samples. Traumatic victimization was found to contribute uniquely to the child's risk for ODD, but not for ADHD. The percentage of children exposed to any traumatic events was 63% for ADHD, 62% for ODD, 91% for ADHD + ODD, and 48% for the adjustment disorder group, with only the comorbid ADHD + ODD group differing significantly from the control group. This difference was entirely accounted for by experiences of victimization trauma rather than nonvictimization events, such as accidents, injuries, illness exposure. In further analyses of these samples, the authors found that 6% of children with ADHD were likely to have PTSD, whereas 24% of children with ODD and 22% of children with ADHD + ODD qualified for the PTSD diagnosis (Ford et al., 2000). Only the two groups with ODD differed significantly from the control group of children diagnosed with adjustment disorders (0% occurrence of PTSD) and showed significantly elevated PTSD-specific symptoms (hyperarousal, sleep disturbance, generalized arousal, and startle response). Since ODD may have a substantial overlap in clinical presentation with borderline personality disorder (BPD), it is not clear from this study whether it is actually childhood BPD within the ODD group that accounts for this relationship. Even so, ODD is the more common disorder among outpatient referrals and, for now at least, should be considered to be a significant risk factor for victimization trauma.

In a study of girls with ADHD, Briscoe-Smith and Hinshaw (2006) found that they were three times more likely to have been abused than comparison girls without ADHD (14.3 vs. 4.5%). The subgroup of girls with ADHD who were abused manifested higher rates of externalizing behaviors, such as aggression, and more peer rejection (owing in large part to their aggressive behavior).

While no definitive conclusions can be drawn from such a limited a number of studies on this topic, it does appear that only a minority of children with ADHD may be exposed to any form of child abuse, and that this risk is greatest among those who may have ODD specifically or other externalizing symptoms more generally.

DRIVING RISKS

Until 20 years ago, the one domain of major life activity for teens and adults that had not been well explored in research on ADHD was driving, or the independent operation of a motor vehicle. Driving is often an underappreciated domain of self-sufficiency and major life activity for teens and adults because it is such a commonplace and therefore ordinary life activity. Yet it is a domain that facilitates most other adaptive domains (including employment, family care, responsibilities, overall functioning, education, social engagements, shopping, and entertainment, among others), all of which would suffer extreme curtailment if an adult were to be deprived of this privilege, especially in the United States. In all of these other domains of major life activity, driving permits greater independence from others, exposure to more numerous opportunities, and greater efficiency in accomplishing various goals. It also, however, opens up greater exposure to harm to oneself, to others, and to property, by providing access to a 1–2 ton projectile that is often used at speeds in excess of 50–60 miles per hour. Like the domain of unintentional injuries discussed earlier, driving is a domain that can markedly increase morbidity and mortality. Thus, any disorder that may adversely impact driving also would be expected to have a pervasive, albeit secondary, impact on many other domains of daily adaptive functioning in other major life activities, while simultaneously exposing the individual to greater liability for the various harms noted earlier. ADHD is just such a disorder that should have some impact on operation of a motor vehicle.

Probably the first to document driving risk in ADHD was an early longitudinal study of hyperactive children followed to adulthood. Weiss, Hechtman, Perlman, Hopkins, and Wener (1979) found that as adolescents and as young adults, individuals with ADHD, as drivers, were more likely to be involved in traffic accidents than their normal peers. They were also more likely to incur greater damage to their vehicles relative to normal controls (Hechtman, Weiss, Perlman, & Tuck, 1981). As interesting as the results were concerning a likely relationship of ADHD and poor driving, these risks were largely determined through self-reports and not corroborated through the official driving records of the participants. Nor was the basis for these driving-related adverse outcomes evident in this early study. Was it the attention deficits associated with ADHD that led to such risks, the impulsiveness, both, or some third set of attributes linked to ADHD? Or were these risks the result of comorbid disorders, especially CD, and therefore one more manifestation of antisocial conduct?

These various lines of reasoning led my colleagues and myself to undertake a series of studies on the driving

problems associated with ADHD. Others also followed suit, generating what is today a substantial literature on this topic. The essence of these results is summarized here (for more detailed reviews, see Barkley, 2004; Barkley & Cox, 2007; Jerome, Habinski, & Segal, 2006; Vaa, 2014). In comparison to control groups of typical people, the following is true of clinic-referred teens and adults with a diagnosis of ADHD, or those derived from population samples with elevated symptoms of ADHD:

- They are more likely to have driven an automobile illegally prior to the time they became eligible as licensed drivers (Barkley, Guevremont, Anastopoulos, DuPaul, & Shelton, 1993; Barkley et al., 2002) or to have driven at other times without a valid license (Barkley et al., 2008).
- They are more impaired on predriving basic cognitive tests and on driving simulator measures of driving performance (e.g., visual field deficits, motor incoordination, erratic steering); they have reduced or more erratic reaction times to critical events, greater distractibility, and more impulsiveness; and they are less rule-governed, more prone to errors, and have more speeding and accidents (scrapes and crashes) (Barkley et al., 1996; Classen, Monahan, & Brown, 2014; Fischer, Barkley, Smallish, & Fletcher, 2007; Narad et al., 2013), but not in all studies (Barkley et al., 2002). They may be more prone to distractions while driving in simulators than controls in the context of engagement in low-stimulation or boring secondary tasks than when doing higher demand tasks (Reimer, Mehler, D'Ambrosio, & Fried, 2009).
- They are more impulsive, take more risks, and are more distracted in behind-the-wheel driving observations in natural settings either by observers (Fischer et al., 2007) or in-vehicle cameras (Merkel et al., in press), and they experience more crashes, minor adverse events, and rapid acceleration or deceleration events during actual driving, as monitored by in-vehicle cameras (Merkel et al., in press).
- They are less likely to employ sound driving habits in their current driving performance, as reported by themselves and others (Barkley et al., 1993, 1996; Fischer et al., 2007; Fried et al., 2006; Garner et al., 2012; Reimer et al., 2005; Richards, Deffenbacher, Rosén, Barkley, & Rodricks, 2006; Rosenbloom & Wultz, 2011). Such ratings are predictive of actual adverse events in the driving histories of participants in these studies (Barkley et al., 1993).
- They display greater levels of anger, hostility, and aggression while driving (road rage; Richards, Deffenbacher, & Rosén, 2002; Richards et al., 2006).
- They are more likely to have had their licenses suspended or revoked (Barkley et al., 1993, 2002, 2008; Fischer et al., 2007).
- They are more likely to have received traffic citations, and have received more such citations, often repeatedly for the same infractions, most notably for speeding and reckless driving (Barkley et al., 1993, 1996, 2002, 2008; DeQuiros & Kinsbourne, 2001; Fischer et al., 2007; Fried et al., 2006; Garner et al., 2012; Lambert, 1995; Murphy & Barkley, 1996; Nada-Raja et al., 1997; Narad et al., 2013; Thompson, Molina, Pelham, & Gnagy, 2007; Woodward, Fergusson, & Horwood, 2000).
- They are more likely to have had a crash or to have had more such crashes while driving a vehicle (Barkley et al., 1993, 1996, 2008; Fischer et al., 2007; Garner et al., 2012; Murphy & Barkley, 1996; Nada-Raja et al., 1997; Thompson et al., 2007; Woodward et al., 2000). This link between ADHD and accident risk also includes motorcycle delivery drivers (Kieling et al., 2011).
- They are more likely to be held as being at fault in such crashes (Barkley et al., 2002, 2008).
- They are more likely to have had crashes involving injuries (Barkley et al., 1996; Woodward et al., 2000).
- They have more severe crashes, as reflected in dollar damage (e.g., $4221 vs. $1665) (Barkley et al., 2002; Fischer et al., 2007).
- They are more likely to have their driving performance adversely affected by alcohol (Barkley, Murphy, O'Connell, Anderson, & Connor, 2006).
- They are unlikely to differ in some of their basic cognitive abilities essential for driving (e.g., capacity for visual discrimination; Barkley et al., 2002), yet they are inferior in others (e.g.,more difficulties with tasks requiring visual scanning and rule-governed behavior; Barkley et al., 2002).
- They are not likely to differ in their driving knowledge in terms of perceptual skills, traffic risk situations, and driving procedures (Barkley et al., 1996, 2002), but they may be less knowledgeable about driving laws and rules of the road, at least during rapid decision making in high-risk situations (Barkley et al., 2002).
- They are not likely to view their driving performance as being that different from other, typical drivers even though it is, as noted earlier, signifi-

cantly worse (Knouse, Bagwell, Barkley, & Murphy, 2005).

Many of the adverse outcomes noted earlier (citations, crashes, suspensions, etc.) are also corroborated in official driving records (Barkley et al., 1996, 2002, 2008), and they appear to exist in not only clinic-referred adults with ADHD but also children with ADHD followed into adulthood (Barkley et al., 2008). It is likely that comorbid ODD and CD exacerbate the risk for these adverse outcomes and may even account for some of the differences between ADHD groups and control groups. For instance, in the recent meta-analysis by Vaa (2014), the relative risk (odds ratio) for accidents was 1.86 for drivers with ADHD, most of whom had these comorbidities, and 1.31 for those without them. The problem with making such comparisons is that ODD and CD are directly correlated with severity of ADHD and share much of the underlying genetic predispositions that influence all three disorders (see Chapter 14; also Tuvblad, Zheng, Raine, & Baker, 2009). Thus, ODD and CD are not confounding factors in such studies but are directly linked to the severity of ADHD. Differences found across groups of participants with ADHD who do and do not have these comorbidities are largely just comparisons of groups whose severity of ADHD varies or the shared liability for externalizing disorders more generally. Hence, efforts to remove them via statistical covariation methods or participant recruitment are actually removing a large part of the variance in the driving measures that is due to ADHD itself. Thus, it makes little sense to argue, as Vaa does, that it is largely these comorbid disorders that account for the elevation in crash risk, and that the crash risk associated with ADHD alone is only modestly elevated. Moreover, the number of studies of pure ADHD, uncomplicated by these comorbidities, is very small and therefore not as robust an indicator of crash risk in noncomorbid cases, as this study implies.

While the severity of current ADHD symptoms was significantly associated with driving risks, some risks were further associated with the severity of oppositional and conduct problems (Barkley et al., 1993, 2008; Thompson et al., 2007) or, at least in girls, were mediated by deviant peer affiliations in adolescence (Cardoos, Loya, & Hinshaw, 2013). The association between attention difficulties and risk for accidents involving injury, driving without a license, and traffic violations, however, holds up in general population samples not selected for ADHD (Nada-Raja et al., 1997; Woodward et al., 2000). The associations also withstand controls for subtype of ADHD, conduct problems, driving experience, and sex (Barkley et al., 2002, 2008; Woodward et al., 2000). Males with ADHD, however, are more likely than females with the disorder to experience license suspensions, to drive without a valid license, and to receive speeding citations (Barkley et al., 2008). When considering the fact that driver inattention is among the most common reasons given for vehicular crashes (Barkley et al., 2002), these adverse outcomes should not be surprising. It is now abundantly evident that ADHD is associated with various driving performance problems and associated adverse outcomes. Noteworthy as well is that in the general population, texting on cell phones while driving has become a major source of inattention (distraction), and that such texting adversely affects the driving of both teens with ADHD and controls (Narad et al., 2013). This would be expected to place teens with ADHD in even greater harm than control teens given that their driving is already significantly impaired, even when they are not texting.

While driving has been the most studied domain of vehicular use in teens and adults with ADHD, one recent study suggests that they are also more likely to engage in other motor sports. These sports pose high risks for injury, particularly if the individual has comorbid CD or antisocial personality disorder and engages in heavy drinking (Wymbs et al., 2013).

Various factors seem to improve the driving performance of teens and adults with ADHD. Among them may be the use of a standard rather than an automatic transmission (Cox et al., 2006). The reason for this may be that the manual transmission requires more mental and motor engagement in the task of driving than the automatic transmission, which may help drivers with ADHD sustain their attention better on driving. Cumulative evidence certainly indicates that ADHD medications can significantly improve the driving performance of teens and adults with ADHD (Barkley, Anderson, & Kruesi, 2007; Barkley & Cox, 2007; Biederman et al., 2012; Kay, Michaels, & Pakull, 2009; Sobanski et al., 2012), which, one hopes, would eventually translate into a reduction in the adverse driving outcomes noted earlier (crashes, injuries, citations, etc.).

RISKY SEXUAL BEHAVIOR

The sexual activities of teens and adults with ADHD have received some research attention since the previous edition of this book. Barkley and Fischer were the

first to report a pattern of early initiation (1 year earlier on average) and riskier sexual activity (more partners, less use of contraception) in their hyperactive (ADHD) group by the follow-up in young adulthood (age 21) of their Milwaukee longitudinal study (Barkley, 1998; Barkley, Fischer, Smallish, & Fletcher, 2006). This riskier pattern of conduct led to a markedly greater risk for teen pregnancy (38 vs. 4%) and sexually transmitted diseases (STDs; 17 vs. 4%) in the hyperactive group than in the control group. Others have demonstrated a similar pattern of sexual conduct in young male adults with a history of childhood ADHD (e.g., Flory, Molina, Pelham, Gnagy, & Smith, 2006), whose childhood ADHD was associated with earlier initiation of sexual activity and intercourse, more sexual partners, more casual sex, and more partner pregnancies. Both longitudinal studies found that these risks were further elevated by higher levels of conduct problems, but such problems did not account for the separate contribution made by ADHD. At the age 27 follow-up of the Milwaukee study, the hyperactive group had already engaged in far higher rates of risky sexual behavior and had more members who experienced teen parenthood and sexually transmitted disease (Barkley et al., 2008). The groups did not differ in the percentages experiencing any of the sexual problems reviewed with the participants using an interview format, such as premature ejaculation or impotence in males, or in inability to climax, exhibitionism, cross dressing, or voyeurism. But the children whose ADHD had persisted to the adult follow-up (persistent group) were twice as likely to experience low levels of sexual interest sometimes or more often than experienced in the nonpersistent or control groups (49 vs. 25 and 24%, respectively. The study also found that the two ADHD groups (persistent and nonpersistent) reported a higher the number of lifetime sex partners (means of 17 and 13, respectively) than the control group (mean = 8). As in the age 21 follow-up, members of both hyperactive groups were more than three times as likely either to have become pregnant, in the case of females, or to have gotten someone else pregnant, in the case of males, than was the case for the control group. Not surprisingly, this led to more of the hyperactive groups having biological offspring than was the case for the control group. While neither of these studies reported any link of ADHD to any type of sexual dysfunction, in a more recent article, Soydan and colleagues (2012) observed the reverse relationship, in that 42% of males experiencing premature ejaculation had clinically elevated symptoms of ADHD versus just 3.7% of males in a control group.

Both the Milwaukee study and the report by Flory and colleagues involved primarily (Barkley, Fischer, et al., 2006) if not exclusively males (Flory et al., 2006). Two later studies examined the relationship of ADHD in females to risky sexual behavior. One found that in a general community sample, level of ADHD symptoms was correlated with some risky sexual behaviors (Hosain, Berenson, Tennen, Bauer, & Wu, 2012). In the other study, in a small sample of college women, those with higher levels of ADHD reported having had more unprotected sex than did women with low ADHD symptoms or college men (Huggins, Rooney, & Chronis-Tuscano, 2012). No studies have examined women with a clinical diagnosis of ADHD. Interestingly, a recent study of a small sample (n = 50) of college men likewise found that elevated symptoms of ADHD along with low birthweight were linked to a more favorable attitude toward casual sex, and that such an attitude predicted a greater number of sex partners (Frederick, 2012). That author believes such findings may indicate an evolutionary adjustment, such that men experiencing reduced fitness in reproductive terms may offset this disadvantage by being more willing to engage in casual sex.

In the past 5 years, several much larger population-based studies have examined whether ADHD symptoms are linked to risky sexual activity. Galera and colleagues (2010) evaluated 1,148 adolescents in a follow-up study in France and noted that symptoms of CD were linked to an earlier age of first intercourse when associated with elevated HI symptoms. It was the combination of symptoms of both disorders that significantly elevated the risk for early sexual activity. Likewise, in a large study of 881 females in Quebec, Canada, mean age of 21 years, Fontaine and colleagues (2008) found that females with a joint risk factor (odds ratio of 2.31) of combined high levels of hyperactivity and physical aggression were more likely to experience early pregnancy than other females. In a study of 420 children followed for 14 years to age 18 in Rotterdam, The Netherlands, Timmermans, van Lier, and Koot (2008) similarly found that early physical aggression (as measured by the Child Behavior Checklist) was linked to risky sexual behavior (early age of first intercourse, more partners, lack of use of contraceptives, sexually transmitted disease, early pregnancy, possible HIV infection, etc.) by age 18 years. Surprisingly the authors did not evaluate the role of ADHD symptoms from this scale, but their results are consistent with those presented earlier, showing the link between early aggression and an elevated likelihood of risky sexual behavior.

A later and larger population-based study in Sweden that involved 2,388 twin pairs evaluated at ages 9, 12, and 15 years, examined the relationship of ADHD, ODD, and CD to likelihood of having had intercourse by age 15 years and the number of sex partners by that age (Donahue et al., 2013). The initial results showed that all three disorders were associated with an increased likelihood of intercourse and that furthermore, ADHD and ODD were associated significantly with an increased number of sex partners. Both findings confirm the results of the studies of clinically diagnosed cases of ADHD reported earlier. The authors further noted that these associations were slightly attenuated after they controlled for parental criminal history and limited education, but most remained significant. However, after the authors controlled for unmeasured family variables (shared environment and genetic effects), the associations were no longer significant. The latter analysis, however, would have removed the genetic contribution to these disorders in the probands, which is known to be substantial, especially for ADHD. The results suggest that there exists a shared familial–genetic risk for risky sexual behavior in teenagers with ADHD, ODD, and/or CD.

The evidence to date therefore clearly indicates an association between ADHD symptoms or disorder and risky sexual behavior, but this risk is often compounded by comorbidity with conduct problems and physical aggression, or frank ODD and CD. Moreover, there may be shared familial environmental and genetic predispositions to such an increase in the likelihood of risky sexual activities. Given the consistency of these results, further work in this area seems promising and warrants development of an early intervention program to reduce these risks. As for any relationship between ADHD and sexual dysfunctions, evidence to date is scant, with just one small study in which males experiencing premature ejaculation were 10 times more likely to have high ADHD symptoms than were males in a control sample.

SUICIDAL THINKING AND ACTIONS

There has been only limited research on the association between ADHD and suicidality but what evidence exists supports an increase in risk for both suicidal thoughts and especially suicide attempts (a combination known as "suicidality") in those with the disorder. Three reasons would lead one to expect suicidality to be higher in this those with ADHD than in a typical community sample. First, there is higher than expected comorbidity between ADHD and mood disorders, especially dysthymia and probably major depression, in clinic-referred children and particularly adults with ADHD (see Chapter 5; Barkley et al., 2008). This would automatically lead one to hypothesize a higher frequency of suicidality in those with ADHD who experience these additional comorbidities than in a normal population. Another is that suicide attempts and completions in particular have been shown to be linked in part to impulsivity (Kim et al., 2003), as discussed further below.

The third reason is that two follow-up studies of hyperactive (ADHD combined type) children into adulthood have found that their rate of suicidality is higher than expected compared to control groups followed contemporaneously.

Children with ADHD as Adults

In their excellent textbook *Hyperactive Children Grown Up*, Weiss and Hechtman (1993) briefly dealt with this issue in describing the results of their own longitudinal study of hyperactive children followed to adulthood. They indicated that the great majority of their hyperactive participants who made suicide attempts requiring psychiatric hospitalization were part of the approximately 10% who had significant psychiatric or antisocial disturbance at their 15-year follow-up (mean age of 25). However, they did not present an actual incidence rate. In reviewing the published reports of the New York (Mannuzza et al., 1993; Mannuzza, Klein, Bessler, Malloy, & LaPadula, 1998) and Swedish (Rasmussen & Gillberg, 2001) longitudinal studies, no reference to such outcomes could be found.

Barkley and Fischer (2005) reported more detailed results about suicidality in their Milwaukee follow-up study of hyperactive children at their age 21 follow-up. At the young adulthood follow-up, they interviewed participants about 12 questions dealing with the topic of suicidality. During high school, members of the hyperactive group were somewhat more likely to have considered suicide than members of the control group (36 vs. 22%, respectively), but they were five times more likely to have attempted suicide if they had considered it (16 vs. 3%). And they were more likely to have been hospitalized for such an attempt (7 vs. 0%). After high school, the rate of suicidal thinking declined somewhat for both groups but remained significantly elevated in the hyperactive group (25 vs. 12%), while the likelihood of a suicide attempt declined substantially and was no

longer significantly different from that in the control group (6 vs. 3%). Nor was the rate of hospitalization following the attempt any greater for the hyperactive group (5 vs. 1%). It is in high school, therefore, that the greatest risk for suicidal ideation, attempts, and hospitalizations after such attempts appears likely to arise in the hyperactive group. Even so, an elevated risk of suicidal ideation remains in the hyperactive group after leaving high school up to age 21. Barkley and Fischer reported that the elevated risks for suicidal thinking and attempts were chiefly mediated by the presence of major depression, though the presence of CD and, to a lesser extent, the severity of ADHD in childhood, made significant additional contributions to risk for suicidality. By the age 27 follow-up of these same participants (Barkley et al., 2008), the authors asked two questions dealing with suicidality. Had the participant considered suicide or attempted suicide in the interim 6 years (on average) since they had last been evaluated? (*Note:* Such questions do not reflect lifetime risk, which we studied at the age 21 follow-up, but only interim risk or risk going forward from age 21 to 27.) They found that a significantly greater percentage of both hyperactive groups, once again, had considered suicide (persistent ADHD to age 27: 38%; nonpersistent ADHD: 24%) in comparison to the control group (8%). There were no group differences in the risk of suicide attempts in the interim (13, 9, and 3%, respectively). Such findings are consistent with the age 21 follow-up in showing that after high school, only the risk of ideation is greater in hyperactive children growing up, and it is not a function of whether ADHD has persisted to age 27. Risk for suicidal ideation since the age 21 follow-up was largely predicted by current mood disorder and, to a lesser extent, current anxiety disorder. Also, having any past mood disorder and severity of ADHD were associated with this risk.

Further corroboration of a link between childhood ADHD and risk for later suicide attempts was reported in a follow-up study of children diagnosed with ADHD at ages 4–6 years compared to control children by 9–18 years of age (Chronis-Tuscano et al., 2011). Young children with ADHD were 3.6 times more likely to attempt suicide during the follow-up period and 4.3 times more likely to experience a mood disorder during follow-up. Girls showed a greater risk for suicide attempts than boys. The authors found that maternal depression and, as in the Milwaukee study (discussed earlier), child comorbidity of emotional and behavior problems at study entry were predictive of later risk for suicidal behavior. In their larger follow-up study of girls with ADHD into early adulthood, Hinshaw and colleagues (2012) likewise found these women to have significantly higher rates of suicide attempts and self-injury than did control girls followed over the same time span, even after controlling for childhood demographic factors and comorbid disorders.

The converse relationship has also been identified, in which children and adolescents hospitalized for injuries are found to have a greater likelihood of having ADHD, as discussed earlier, but especially injury from assault or from suicide and self-harm (Lam, 2005). And those individuals who did have ADHD were likely to remain in the hospital longer than injured children without ADHD, suggesting that the injuries were more severe.

Adults Diagnosed with ADHD

In a study of clinic-referred adults with ADHD, Barkley and colleagues (2008) found that their ADHD group did not differ from either a clinical or community control group in either suicidal ideation or suicide attempts prior to 18 years of age, in contrast to the findings of the Milwaukee study. They reported that 15–16% of both control groups had considered suicide and 2–4% had made an attempt at suicide before age 18 years, in comparison to 25% of the ADHD group that had considered it and 6% that had attempted it. After age 18 years, however, they found that more of both the ADHD and clinical control groups had considered suicide (29 and 27%, respectively) than was the case for the community control group (6%). But these two groups did not differ from each other, implying that suicidal ideation at least was associated with outpatient psychopathology rather than specifically with ADHD in these adults. A significantly greater proportion of the ADHD group (8%), however, had also made a suicide attempt (1%) than that in the community control group since age 18. The clinical control group fell between both of these groups in this case and did not differ significantly from either of them. These results were corroborated in a study of ADHD in adults in Korea, which found a significant association between ADHD and adults with suicidality, among other comorbid disorders (Park et al., 2008). Likewise, in a population-based study, Agosti, Chen, and Levin (2011) reported a suicide attempt rate of 16% of adults with ADHD identified in their sample, whereas in a study of Turkish outpatient adults with ADHD, Duran, Fisticki, Keyvan, Bilici, and Caliskan (2013) reported that 38.5% had attempted suicide.

The risk for suicidality in the UMASS Study of Adults with ADHD was largely related to comorbidity for both major depressive disorder (MDD) and dysthymia (Barkley et al., 2008). Three times as many people who had thought of suicide before age 18 years had MDD compared to those who had not considered suicide. The same was true for those who had attempted suicide (73 vs. 25%). Dysthymia was somewhat less prevalent than MDD among those who had or had not considered or attempted suicide before age 18 years, but the pattern of risk associated with dysthymia was still similar, such that more than 2.5 times as many people who considered or attempted suicide had dysthymia compared to those who had not considered or attempted suicide. The results are even more striking for those who considered or attempted suicide after 18 years of age. Here more than four times as many people who considered or attempted suicide had MDD, and more than three times as many had dysthymia. CD was not related to suicidal thinking at this age, but it was related to attempts. More than twice as many attempters as nonattempters had CD. In a regression analysis, MDD, dysthymia, and ADHD severity (number of symptoms from interview) all significantly contributed to the prediction of suicidal thinking. Only MDD significantly predicted a suicide attempt after age 18 years.

These results corroborate the earlier findings of the Barkley and Fischer (2005) follow-up study, showing that the risk for suicidality among individuals with ADHD was mostly a function of their higher rate of comorbidity with MDD and dysthymia, and to a far lesser extent their severity of ADHD. CD was not predictive of these outcomes after researchers statistically controlled for these other disorders. Biederman and colleagues also found much the same mediational pathway of ADHD in females to their risk for suicidality via comorbidity with major depression (Biederman, Ball, et al., 2008). And the later study by Agosti and colleagues (2011) likewise found that although adult ADHD was a risk factor for suicide attempts, it was only weakly associated with that risk, in that comorbid disorders were a much stronger contributor.

ADHD as a Risk Factor in Other Clinical Populations

The inverse relationship of higher rates of ADHD in adolescent suicide attempters has also been suggested. In one study of admissions to an emergency room for suicide attempts (Manor et al., 2010), results indicated that 65% of these attempters qualified for a diagnosis of ADHD, although only five had received a prior diagnosis of the disorder. Approximately 43% qualified for a diagnosis of depression and 39% had a Cluster B personality disorder. Similarly, Kelly, Cornelius, and Clark (2004) studied adolescents with substance use disorders and their risk for suicide attempts, and found that the risk was a function of severity of ADHD and mood disorders, as did Arias and colleagues (2009). Kelly and colleagues found that ADHD was a risk factor mainly in males; in females, primarily mood disorders and alcohol use disorders were linked to risk of suicide. The same pattern seems to hold among incarcerated youth. Putnins (2005) found that risk of suicide attempt was a function of ADHD as well as mood disorders, substance use, and having a bad temper. Goodman, Gerstadt, Pfeffer, Stroh, and Valdez (2008) studied 43 psychiatrically hospitalized children for their level of assaultive and suicidal behavior, and found that children with a pattern of combined assaultive and suicidal behavior were more likely to be diagnosed with ADHD than those who were assaultive only. Aggression seemed to mediate the link between ADHD and suicidal behavior.

The Mediating Role of Comorbidity

The linkage of ADHD to suicide and the possible mediating role of depression and CD is also documented in both early (James, Lai, & Dahl, 2004) and later reviews (Impey & Heun, 2012) of this literature. It is evident not just in clinical samples, described earlier, but also in population-based studies, such as that by Cho and colleagues (2007) in Korea, who evaluated 788 high school girls. They noted that ADHD symptoms correlated with risk for suicidal ideation and that symptoms of depression partially mediated that link. Similarly, Galera, Bouvard, Encrenaz, Messiah, and Fombonne (2008) studied 917 French children and teens, ages 7–18 from the general population. They found that the HI symptom dimension of ADHD increased the lifetime and current risks for suicidal thoughts and attempts in males but not in females. They did not assess depression in this study, which may have been the link to suicidality in females, as suggested earlier.

Studies besides that of Galera and colleagues (2008) also suggest that psychomotor restlessness is a risk factor for suicidal behavior, beyond the risks posed by a mood disorder in youth with adjustment disorders (Pelkonnen, Marttunen, Henriksson, & Lonnqvist,

2005). It may be through its heightened HI that ADHD elevates this risk for suicidality in youth with comorbid depression. Research shows that in individuals with depression, degree of impulsivity specifically and ADHD symptoms more generally are significantly correlated with the likelihood of suicidal behavior, as well as the lethality of the attempt (Keilp, Gorlyn, Oquendo, Burke, & Mann, 2008; Patros et al., 2013). This may explain the earlier Milwaukee study finding that ADHD is linked more to risk for attempts than to suicidal ideation, which is more a function of comorbid depression. And once suicidal thinking or behavior has occurred, it serves as a predictor of increased risk for later suicidal ideation and attempts beyond the contribution made by major depression or substance use disorders (Fergusson, Horwood, Ridder, & Beautrais, 2005; Putnins, 2005).

Even further corroboration of this link between ADHD and suicidality and the meditational role of mood disorders comes from a recent, large-scale (n = 418) investigation of clinic-referred children and teens in Hungary (Balazs, Miklosi, Kereszteny, Dallos, & Gadoros, 2014). Of these, 211 met DSM criteria for ADHD and another 105 had sufficiently elevated symptoms to be considered subthreshold cases. All cases were treatment naive, so treatment, particularly with ADHD medications, is neither a beneficial nor a detrimental factor in these results. This issue is important given that some have claimed that these medications may increase the risk of suicide, although a recent meta-analysis has found this not to be the case (Bushe & Savill, 2013). In the study, 12.9% of children (age 12 and under) and 38.9% of teens reported either thinking about or attempting to end their own lives within the past month (current suicidality). Older age was associated with an increase risk for suicidality, as earlier research has shown. Gender was not. Mediational analyses clearly showed that ADHD does increase the risk for current suicidality, but that this relationship was entirely mediated by comorbidity and age. In children age 12 years and under, more severe ADHD symptoms were associated with greater comorbid anxiety symptoms, and the latter served to mediate the link between ADHD and current suicidality. Although more severe ADHD symptoms were also significantly associated with more symptoms of other, comorbid disorders (depression, ODD, CD, tics, OCD, etc.), those comorbidities did not mediate the link to suicidality. In contrast, in adolescents, the link between ADHD and current suicidality was a function of comorbidity with symptoms of major depression and dysthymia, as well as those of substance abuse/dependence. Once again, more severe ADHD symptoms increased the risk for symptoms of these mediating disorders, which then accounted for the link to current suicidality. Yet again, more severe ADHD symptoms increased the likelihood of other, comorbid symptoms but those comorbidities did not mediate this link to suicidality.

In summary, and hardly surprising, it is largely the existence of anxiety in children, and especially mood disorders in adolescents and adults, that elevates the suicidality risks in ADHD. Current ADHD severity (probably impulsivity) does seem to make a further though smaller contribution to risk, especially for suicide attempts, and probably more in males than in females.

HEALTH CARE COSTS

Early, small studies did not find that children with ADHD had more hospitalizations, length of hospital stays, or surgeries than normal children (Barkley, DuPaul, et al., 1990; Hartsough & Lambert, 1985; Stewart et al., 1966). But in view of their clearly elevated risks for various injuries, children with ADHD probably should use more medical care and generate greater medical costs. This has been observed in more recent studies over the past decade using larger samples and even populationwide databases. Children with ADHD have a significantly greater use of outpatient medical services, more hospitalizations, and are especially more likely to utilize emergency department services (Chan, Zhan, & Homer, 2002; Cuffe et al., 2009; Guevara, Lozano, Wickizer, Mell, & Gephart, 2001; Leibson & Long, 2003). This resulted in a doubling of the medical care cost associated with ADHD relative to control cases (Guevara et al., 2001; Leibson et al., 2001; Leibson & Long, 2003). The same findings of increased costs linked to ADHD have been recently reported in a longitudinal study in Australia, which found substantially higher costs linked to ADHD in children ages 4–9, with the excess costs amounting to $25–30 million AUS (Sciberras, Lucas, Efron, Gold, Hiscock, & Nicholson, in press). Swensen and colleagues (2004) studied a large population sample (more than 100,000) and also found that annual medical care costs for children with ADHD were three times greater than in control cases ($1,574 vs. $571). But they and others in The Netherlands (e.g., Le et al., in press) also found that

medical care cost claims were also greater among immediate family members of the children with ADHD as well ($2,728 vs. $1,440 in the study by Swensen et al., 2004), perhaps owing to the greater risk of psychopathology, substance dependence and abuse, stress, and depression among these family members. More recent studies bear out this substantial increase in health care costs (billions of dollars) associated with children with ADHD and their immediate family members (Birnbaum et al., 2006; Le et al., in press; Pelham, Foster, & Robb, 2007; van Roijen et al., 2007). Medical care costs are also found to be markedly higher in adults with ADHD (Secnik, Swensen, & Lage, 2005).

LIFE EXPECTANCY

The relationships between ADHD and increased (1) accident proneness in childhood, (2) speeding and auto accidents in adolescence and young adulthood, (3) crime (Satterfield, Hoppe, & Schell, 1982), (4) suicide attempts (Weiss & Hechtman, 1993), (5) substance use and abuse (alcohol and tobacco primarily) in adolescence and adulthood (Barkley et al., 2008), and (6) a general pattern of risk-taking behavior all intimate that ADHD might be expected to be associated with a *reduced life expectancy*. The diminished regard for the future consequences of one's behavior that characterizes many adolescents and adults with ADHD would also predict a reduced concern for health-conscious behavior, such as exercise, proper diet, and moderation in using legal substances (caffeine, tobacco, and alcohol) throughout life (Barkley et al., 2008; Milberger et al., 1996).

Further cause for concern arises from the follow-up study of Terman's original sample of highly intelligent children. Most of those subjects were in their 70s or older at follow-up, and half of them are deceased (Friedman et al., 1995). The follow-up study of that group indicated that the most significant childhood personality characteristic predictive of reduced life expectancy by all causes was related to impulsive, undercontrolled personality characteristics. Individuals classified as having this set of characteristics lived an average of 8 years less than those who did not (73 vs. 81 years). Subjects in this study were defined as impulsive by virtue of falling within the lowest 25% of the sample in impulse control. Given that subjects defined as having ADHD typically fall well below this threshold, in the lowest 5–7%, the risk for reduced longevity in those with ADHD would seem to be even greater than was found among Terman's subjects. That conclusion would seem to be further supported by the fact that Terman's subjects were intellectually gifted and came from families of above-average or higher economic backgrounds. Both of these factors probably would have conveyed a greater advantage toward longer life expectancy than would be the case for intellectually normal children with ADHD who tend to come from middle or lower economic backgrounds.

No follow-up studies of children with hyperactivity or ADHD have lasted long enough to document unequivocally such a reduction in life expectancy; the oldest subjects now appear to be entering their 40s (Klein et al., 2012; Weiss & Hechtman, 1993). Yet concern over life expectancy in ADHD is not unfounded. Swensen, Allen, and colleagues (2004) found that individuals with ADHD are more than twice as likely as controls to die prematurely from their misadventures. Results of the British National Child Development Study also indicate that children in the upper quartile of externalizing behavior were twice as likely to die by age 46 years as children in the lowest quartile (3.2% vs. 1.4%, respectively), with mortality increasing across these quartiles from lowest to highest (Jokela et al., 2008). And more recently Klein and colleagues (2012) reported that children with ADHD were more likely than children without ADHD to have died by the age 41 follow-up. But the sample sizes in that study were small and therefore warrant replication with larger samples. Yet in view of the numerous adverse effects of ADHD on health discussed in this chapter, one can reasonably foresee that people with ADHD are more likely to experience significantly life-shortening medical problems, such as accidental injuries, coronary heart disease or cancers, to a greater extent than typically developing people as they enter middle age or late life stages.

CONCLUSION

This chapter has reviewed the myriad health problems and risks associated with ADHD in children, teens, and adults. They are clearly substantial and serious, indicating unequivocally that ADHD is more than just a serious mental health problem—it is a serious *public health* problem. At the very least, such findings ought to give considerable pause to anyone who contends that ADHD is a phantom disorder, that it is simply a label used to provide a psychiatric diagnosis for oth-

erwise normally exuberant children who do not want to take responsibility for their own behavior, that it merely reflects parental or teacher intolerance for such childhood exuberance, or that it is an otherwise benign condition with little or no health consequences. Henceforth, such claims ought to be dismissed as the scientifically illiterate statements they represent rather than be considered to reflect a true scientific debate over the validity and worth of the diagnosis of ADHD. That validity and utility have been well established by more than two centuries of research and thousands of published studies on the distinguishing symptoms, associated impairments, and developmental risks that befall those children and adults unfortunate enough to receive a clinical diagnosis of this condition. Even more than the evidence presented in previous chapters, the evidence reviewed here overwhelmingly demonstrates that ADHD comprises a harmful dysfunction (Wakefield, 1992, 1997) and is therefore deserving of the status of a true mental disorder as much or more than any other child or adult psychiatric disorder currently known.

KEY CLINICAL POINTS

- ✓ ADHD in both children and adults is associated with poorer health, more general bodily concerns, a slightly greater risk for otitis media, twice the population risk for asthma, and a greater risk for coronary heart disease by adulthood than controls. The disorder does not appear to be associated with any greater risks for diabetes or hypertension, despite a greater risk for obesity, and is not associated with significant visual difficulties.
- ✓ Compared to typically developing children, children with ADHD on average have poorer dental health and, specifically, a greater risk for caries, missing teeth, filed surfaces, gingivitis, bruxism, bleeding gums, plaque buildup, and a five times greater likelihood of oral trauma, as well as poorer oral hygiene, greater dental office behavioral difficulties, and less effective communication styles toward dental professionals. Dental health in adults with ADHD remains to be studied in any detail.
- ✓ Teens and adults with ADHD are more likely to smoke tobacco, to smoke more often, and to be more stable or consistent smokers than are comparison individuals. While the predilection to smoke may be a form of self-medication given the positive impact of nicotine on ADHD symptom reduction, it may also be a consequence of poorer inhibition, novelty seeking, and risk taking.
- ✓ Teens and adults with ADHD appear to be more likely to use marijuana, but this risk may be a function of comorbid CD.
- ✓ Teens with ADHD may not be more likely to use alcohol than other teens unless they have comorbid CD or deviant peer relationships accompanied by reduced parental monitoring. But by adulthood, there is greater evidence of an association between ADHD and increased alcohol use and abuse, regardless of any relationship between ADHD with CD.
- ✓ Teens and adults with ADHD may be at greater risk of abusing other substances, but this risk is primarily, although not completely, a function of comorbid CD or antisocial personality disorder. The abuse of substances may feed forward to worsen the symptoms of ADHD and associated EF deficits, such as working memory.
- ✓ Treatment of ADHD in childhood with stimulant medications is not linked to any increased risk of substance use in adolescence or adulthood. Continued treatment with medication into adolescence may reduce the likelihood of substance use and abuse, although the findings for such a protective effect are mixed and rather weak.
- ✓ Adults with substance use disorders are three to seven times more likely to have ADHD, which makes it essential that clinicians who work with drug-abusing clients routinely screen them for the possibility of ADHD. The presence of ADHD in substance-abusing adults is associated with worse drug use and other forms of psychopathology, and may interfere with response to treatment of the substance abuse.
- ✓ The recent generation of children with ADHD is 1.5 times more likely to be overweight or obese, whereas adults growing up with ADHD are 1.5 to 2.0 times more likely to be overweight or obese. ADHD is also more common in children and adults who are being treated for obesity, again necessitating in such cases the screening and appropriate management of ADHD. It is primarily the HI symptom dimension of ADHD that increases these risks.
- ✓ Females with ADHD demonstrate an increased risk (three to six times) for eating disorder pathology generally and BED or frank bulimia by adolescence. Con-

versely, women in treatment for BED or bulimia are four times more likely to have adult ADHD. Persons with eating disorder pathology and ADHD are likely to have more severe eating problems and to be less responsive to efforts at self-change or interventions for weight control than are those who do not have ADHD. Again, these risks are a function more of the HI symptom dimension of ADHD than of its inattentive dimension, but they are also related to the degree of peer and parent–child relationship problems.

- ✓ Children with ADHD may be three to five times more likely to manifest enuresis or encopresis than are typically developing children. This risk appears to be greater in children who also have comorbid ODD. Yet these difficulties affect only a minority of children with ADHD.
- ✓ There is a two-way relationship between ADHD and seizure disorders or epilepsy, in which the presence of one condition increases the risk for the other by 2.5 times over the population risk for either condition. More children with ADHD manifest increased frontal–temporal theta band activity on EEG, which is thought to indicate reduced brain arousal or responsiveness to stimulation.
- ✓ Both children and adults with ADHD are more likely to have sleep difficulties, including greater time to fall asleep, frequent night waking, restlessness during sleep, and sleep-disordered breathing; more than half of these individuals are described as having daytime tiredness and more frequent episodes of daytime sleepiness than typically developing children or adults. The presence of anxiety or depression or ASDs with ADHD may further increase the risk for these sleeping difficulties. These sleeping problems may be more associated with the severity of the HI symptom dimension of ADHD than the inattentive dimension, but reduced sleep and daytime tiredness may exacerbate daytime inattention.
- ✓ Children, adolescents, and young adults with ADHD manifest a significantly increased risk for PIU or computer gaming usage or frank Internet/gaming addiction. Moreover, individuals with PIU or Internet/gaming addiction are more likely to have elevated ADHD symptoms. The risk for PIU/addiction is linked not only to severity of ADHD symptoms, especially impulsivity, but also depression, anxiety, and hostility/aggression. Yet it is possible that the association with depression (and possibly suicidality) and anxiety may be as much consequences of rather than predispositions to PIU/addiction.
- ✓ ADHD in children, and more recently in adults, has been repeatedly linked to an increased risk (two to five times) for accidental injuries of all types (trauma, burns, poisonings, etc.), more severe injuries, as well as repeated injuries. The comorbidity of ODD/aggression and ADHD in children further exacerbates these risks. Likewise, children admitted to hospitals due to accidental injuries are three times more likely to have ADHD (approximately 30%) than children admitted for other reasons. Factors contributing to these elevated risks are inattention, impulsivity and risk taking, motor incoordination, comorbidity with ODD/CD, anxiety, and depression, and parental characteristics such as reduced parental monitoring of the child's activities.
- ✓ The evidence base is relatively thin, but it suggests that children with ADHD may be three times more likely to be abused than typically developing children, with this risk greatly increased by the presence of comorbid ODD.
- ✓ ADHD in teens and adults is consistently associated with less safe driving habits, deficient driving performance, and greater inattention and impulsivity while driving. The disorder is also linked to more adverse driving outcomes, such as a greater risk for traffic citations (especially for speeding) and more such citations; license suspensions/revocations; and a greater risk for crashes, more crashes, more severe crashes; and being considered to be at fault in such crashes. Some of these adverse outcomes are also linked to and exacerbated by comorbidity with ODD/CD or antisocial personality disorder. ADHD medications may improve these driving performance problems. Adults with ADHD are also more likely to manifest aggression while driving, or road rage, and such hostility and emotional dysregulation is also a factor in their crash risks. Texting on cell phones while driving markedly worsens the driving performance of both teens with ADHD and typical teens.
- ✓ With regard to sexual behavior and adjustment, teens with ADHD engage in more risky sexual behavior in terms of starting sexual activity earlier; having more sex partners; and being less likely to use contraception and therefore more likely to be involved in a pregnancy or to contract a sexually transmitted disease. Risky sexual behavior is also related to ODD and CD, with the combination of both disorders associated with the

highest risks for early sexual activity and pregnancy involvement. In adults, ADHD in males may be associated with an increased risk for premature ejaculation.

- ✓ ADHD in teens and young adults may be linked to a mild increase in the likelihood of suicidal thinking or suicide attempts, but these risks are more a function of comorbidity with depression and, to a lesser extent, CD. The role of ADHD in suicidality appears to increase the likelihood of making an impulsive suicide attempt. These risks appear to be at their highest during high school, then decline throughout the 20s in longitudinal studies of ADHD and typical youth. Adolescent suicide attempters are also more likely to have ADHD, most likely owing to the role of impulsivity in distinguishing those who attempt suicide from those who are only depressed.
- ✓ In view of all of these factors, it is not surprising that ADHD in children and adults is associated with two to three times greater health care costs.
- ✓ A small but growing literature intimates that ADHD may be associated with a reduced life expectancy given that self-regulation/conscientiousness remains one of the most robust predictors of longevity, and that those with ADHD occupy the extreme lower end of the population distribution of these traits.

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CHAPTER 12

Educational, Occupational, Dating and Marital, and Financial Impairments in Adults with ADHD

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Both trade books (Adler, 2006; Barkley, 2010b; Hallowell & Ratey, 1994; Wender, 1995) and clinical textbooks (Goldstein & Ellison, 2002; Gordon & McClure, 1996; Triolo, 1999; Weiss, Hechtman, & Weiss, 2001) have noted the serious and pervasive adverse effects associated with attention-deficit/hyperactivity disorder (ADHD) in the domains of educational, occupational, social, and financial functioning. However, despite such trenchant clinical observations, the research literature on each of the domains of major life activities is relatively small. Here I briefly review what is known from research about each of these domains of impairment associated with adults with ADHD. Some of this material is adapted from that published in my earlier textbook on the topic of adults with ADHD (Barkley, Murphy, & Fischer, 2008).

EDUCATIONAL FUNCTIONING

Children with ADHD Growing Up

Chapter 6 on educational impairments indicates that the vast majority of clinic-referred children with ADHD are doing poorly at school, typically underperforming relative to their known levels of ability, as determined by intelligence and academic achievement tests. These classroom difficulties are believed to be the result of the inattentive, impulsive, and restless behavior associated with ADHD, the typically lower than average intelligence associated with the disorder (see Chapter 6; Rapport, Scanlan, & Denney, 1999), as well as the higher comorbidity of ADHD and learning disabilities (DuPaul, Gormley, & Laracy, 2012; Tannock & Brown, 2000). Given these deficits in academic skills and behavior, it is not surprising to find that as many as 56% of children with ADHD may require academic tutoring, approximately 30% may repeat a grade in school, and 30–40% may be placed in one or more special education programs. As many as 46% may be suspended from school and 10–35% may drop out entirely and fail to complete high school (Barkley, DuPaul, & McMurray, 1990; Barkley, Fischer, Edelbrock, & Smallish, 1990; Barkley, Fischer, Smallish, & Fletcher, 2006; Faraone et al., 1993; Szatmari, Offord, & Boyle, 1989; Weiss & Hechtman, 1993).

By adolescence, these chronic and cumulative experiences with school failure, learning disorders, school misbehavior, and sometimes lower intelligence begin to generate other adverse educational outcomes. For instance, the academic outcome of the hyperactive (ADHD) adolescents was considerably poorer in Barkley and Fischer's Milwaukee follow-up study at the

teen follow-up than that of the typically developing adolescents followed concurrently. At least three times as many hyperactive (ADHD) children had failed a grade (29.3 vs. 10.0%) or been suspended (46.3 vs. 15.2%) or expelled (10.6% vs. 1.5%) (Fischer, Barkley, Edelbrock, & Smallish, 1990). Others have also identified such high educational risks in longitudinal studies dating back as much as 40 years (Ackerman, Dykman, & Peters, 1977; Mendelson, Johnson, & Stewart, 1971; Stewart, Mendelson, & Johnson, 1973; Weiss, Minde, Werry, Douglas, & Nemeth, 1971; Wilson & Marcotte, 1996). In another sample of clinic-referred teenagers with ADHD, a similar risk for school retention and suspension was documented (Barkley, Anastopoulos, Guevremont, & Fletcher, 1991). Almost 10% of the hyperactive sample followed into adolescence had quit school at this follow-up point in the Milwaukee Study, compared to none of the normal sample (Barkley, Fischer, et al., 1990). Fischer and colleagues (1990) also found that the levels of academic achievement on standard tests were significantly below normal on tests of math, reading, and spelling, falling toward the lower end of the normal range (standard scores between 90 and 95).

Fischer and colleagues (1990) examined whether the presence of conduct disorder (CD) within the hyperactive group at follow-up accounted for these greater than normal rates of academic failure. The results indicated that although hyperactivity alone increased the risk of suspension (30.6% of pure hyperactives vs. 15.2% of controls) and dropping out of school (4.8% of pure hyperactives vs. 0% of controls), the additional diagnosis of CD greatly increased these risks (67.4% were suspended and 13% dropped out). Moreover, the presence of CD accounted almost entirely for the increased risk of expulsion within the hyperactive group, in that the pure hyperactive group did not differ from typically developing children in expulsion rate (1.6 vs. 1.5%), whereas 21.7% of the mixed hyperactive/CD group had been expelled from school. In contrast, the increased risk of grade retention in members of the hyperactive group was entirely accounted for by their hyperactivity, with no further risk occurring in the mixed hyperactive/CD group.

In general, it appears that academic performance difficulties in adolescence are associated with having had persistent ADHD since childhood, whereas school disciplinary actions such as suspensions and expulsions are more closely linked to comorbid conduct problems or CD than to ADHD alone (Barkley, Fischer, et al., 1990; Fischer et al., 1990; Wilson & Marcotte, 1996). Children with ADHD who have the lowest levels of adaptive functioning in childhood are also the most likely to have comorbid psychiatric disorders and academic impairments in adolescence (Barkley, Shelton, et al., 2002; Greene, Biederman, Faraone, Sienna, & Garcia-Jetton, 1997; Wilson & Marcotte, 1996). Here *adaptive functioning* refers to the development of self-sufficiency, as measured by instruments such as the Vineland Adaptive Behavior Scales.

These evident trends toward lower academic performance and more grade retentions, suspensions, and expulsions in the adolescent years increase, such that by adulthood, the percentage of those with ADHD dating to childhood have even greater difficulties in these areas than the percentages noted in adolescence and, of course, greater than those in control groups. Hyperactive children in follow-up studies into adulthood had less education, achieved lower academic grades, failed more of their courses, were more often retained in grade, failed to graduate high school, and did not attend college than were children in control groups (Klein et al., 2012; Lambert & Hartsough, 1998; Mannuzza, Gittelman-Klein, Bessler, Malloy, & LaPadula, 1993; Mannuza, Klein, Bessler, Malloy, & LaPadula, 1998; Weiss & Hechtman, 1993).

Children Diagnosed with ADHD Followed to Adulthood

The Milwaukee Study (Barkley, Fischer, Smallish, & Fletcher, 2006) found much the same results at the age 21 follow-up: More than three times as many hyperactive than control group members had been retained in grade at least once (42 vs. 13%) during their schooling or had been suspended from high school at least once (60 vs. 18%). The hyperactive group members had completed fewer years of education and had a lower grade point average (1.69 vs. 2.56 out of a possible 4.0) and class ranking in their last year of schooling (69th percentile vs. 49th percentile) than those in the control group. More of the hyperactive group than the control group had also received special educational services while in high school, and had been suspended or expelled from school or been truant relative to the control group. Of significant social and economic impact, however, was the finding that 32% of the hyperactive group had failed to complete high school compared to 0% of the control group. Substantially fewer hyperactive than control children had ever enrolled in college

(21 vs. 78%) or were currently attending at this follow-up point (15 vs. 66%). These findings were reaffirmed 6 years later at the age 27 follow-up (Barkley et al., 2008). In the Canadian follow-up study, approximately 20% attempted a college program, yet only 5% completed a university degree program, compared to over 41% of control children (Weiss & Hechtman, 1993). The longest running (30-year) follow-up study of hyperactive children into midlife likewise indicates that less education is an outcome of childhood ADHD, with 30% either not completing high school or getting a general equivalency diploma (GED), compared to just 4% of the control group (Klein et al., 2012). These findings demonstrate that the educational domain is major in terms of impaired functioning and reduced attainment for children growing up with ADHD.

As noted previously, children with ADHD followed into adulthood in the previous studies and clinic-referred adults diagnosed with ADHD do not have identical impairments. Studies of children with ADHD often indicate that estimates of their intellect are significantly lower than estimates for children in control groups, averaging about 7–10 IQ points difference (see earlier discussion; Barkley, 2006). This does not seem to be the case for clinic-referred adults with ADHD in prior studies. For them, intelligence estimates seem to fall within the normal range and are comparable to estimates for control groups of clinic-referred adults (Barkley, Murphy, & Kwasnik, 1996; Murphy & Barkley, 1996; Murphy, Barkley, & Bush, 2002). Although Biederman and colleagues (1993) found that their sample of adults diagnosed with ADHD had IQ scores significantly below scores of their control groups, the IQ scores for the adults with ADHD were 107–110, nearly identical to the results of our own studies of adults with ADHD. The adults with ADHD in the Biederman and colleagues study therefore seem to differ significantly from the control groups only by virtue of the fact that the control group had above-average IQs (110–113).

Academic Histories of Adults Diagnosed with ADHD

Even so, individuals diagnosed with ADHD in adulthood do seem to have a higher risk for adverse educational outcomes and lower academic functioning at some time during their schooling than do control adults, just as was found in children with ADHD followed over development (Able, Johnston, Adler, & Swindle, 2007; Young, Toone, & Tyson, 2003). My colleagues and I (Barkley et al., 2008) studied this domain extensively in a large sample of clinic-referred adults diagnosed with ADHD relative to two different control groups (clinical, community). The adults reported that they were significantly more likely to be currently impaired in any educational activities (89%) and to have been so impaired during childhood (91%), and they were more often rated by others as such in their current and childhood educational adjustment (63 and 66%, respectively) than were those in a community control group (1, 6, 3, and 2%, respectively; Barkley et al., 2008). These adults with ADHD were also rated by others (mostly parents) who knew them well as more likely than control adults to have been impaired as children in various school situations, such as classwork (64 vs. 2%), homework (66 vs. 3%), classroom behavior (37 vs. 0%), recess (27 vs. 0%), and interactions with other children (35 vs. 0%), among other settings, and in overall time management at school (65 vs. 2%) compared to the community group (Barkley et al., 2008). In most settings, adults with ADHD were at least two to three times more likely than the clinical control group to have been impaired, and vastly more so than the community control adults. These results show that adult ADHD has a substantial adverse impact on school activities in the childhood histories of these adults.

Between 16 and 40% of clinic-referred adults have repeated a grade, in keeping with the figures reported for children with ADHD discussed earlier in this chapter (Barkley et al., 2008; Biederman et al., 1993; Murphy & Barkley, 1996; Roy-Byrne et al., 1997). Up to 43% have also received some form of extra tutoring services in their academic histories to assist them with their schooling (Biederman et al., 1993). My colleagues and I found that 16–35% of young adult samples with ADHD had received special educational services in our prior studies (Barkley et al., 2008; Murphy et al., 2002; Roy-Byrne et al., 1997), a figure about half that found in hyperactive children followed to young adulthood but still higher than normal. A history of behavioral problems (41–44%) in school occurs more often in adults with ADHD than in control adults (1–3%), and they are likely to have been suspended from school four times more often and to have been truant from school three times more often than controls (Barkley et al., 2008; Murphy & Barkley, 1996). Yet adults with ADHD seen in clinics are far more likely to have graduated high school (78–92%) and attended college (68%)

than are clinic-referred children with ADHD followed to adulthood (discussed earlier), for whom the high school graduation rate is only about 64% (Barkley et al., 2008). However, fewer of them graduate from college (30%) compared to control groups (54–62%; Barkley et al., 2008).

A number of studies indicate that clinic-referred adults with ADHD may have less education than adults without ADHD seen at the same clinic or community control groups (Able et al., 2007; Barkley et al., 2008; Roy-Byrne et al., 1997; Sobanski et al., 2008; Young et al., 2003), a finding that is consistent with adult follow-up studies of ADHD children (Mannuzza et al., 1993). Yet this is not always the case because a few studies did not find such differences (Murphy & Barkley, 1996; Murphy et al., 2002). Torgersen, Gjervan, and Rasmussen (2006), using a Norwegian sample (n = 45), found that only 20% of their adults with ADHD had 12 or more years of education—a figure well below those of other studies of ADHD in adults. This Norwegian sample appears to have far greater severity of ADHD than is typical of North American samples.

In summary, while adults with ADHD diagnosed in adulthood may be less likely to experience adverse educational outcomes than children followed to adulthood, the former still do so at far higher rates than control groups. And while a higher proportion of them graduate from high school than children with ADHD followed to adulthood, the adult-diagnosed groups are less likely than control groups to graduate from college, and they have less education. Certainly a majority of these individuals diagnosed with ADHD as adults report current impairment in educational activities and as having been so as children compared to the reports of control groups. As in general population samples, generally males were also more likely than females to experience these adversities; these differences were further exacerbated by the presence of ADHD. But both sexes with adult-diagnosed ADHD are more impaired educationally than are controls of the same sex (Barkley et al., 2008).

Archival Records of Academic Performance

Our large study of adults with ADHD (Barkley et al., 2008) obtained high school transcripts of many of the participants. From those transcripts, we coded the percentage of grades that were D's, F's, or unsatisfactory (U's). For elementary school and high school, we also coded the average number of days per year they were reported as having been absent from school on the transcript. From high school transcript we recorded participants' class ranking in their senior year or last year of high school attended. For high school and college, we computed their grade point averages using a 0- to 4-point scale (grades of F to A) or, if reported in numerical scores (e.g., 90–100, 80–89, 70–79), we recoded them as 0 (50–59), 1 (60–69), and so on. If any verbal or quantitative test scores were available on the high school transcript, we recorded that information as well. We did likewise for the verbal and quantitative Scholastic Aptitude Test (SAT) scores that may have been recorded on college transcripts. All of this information is reported in Table 12.1 for each group. On their elementary school transcripts, the ADHD group had a greater percentage of poor or failing grades than either of the control groups, which did not differ from each other. The clinical and community control group participants did not differ in their average number of days absent in elementary school.

In high school, once again, the adults with ADHD had a significantly greater percentage of D's and F's on their transcripts and were ranked lower in class standing than were adults in either control group. Not surprisingly, then, we also documented a lower grade point average for the ADHD group than for the two control groups. Unlike their elementary school period, we did find that the ADHD group had significantly more days absent from high school than did adults in either of our control groups. Of note is that the clinical and community control groups did not differ in any of these respects.

Among those who attended college and consented to allow us to obtain their transcripts, we found results consistent with those noted earlier for high school. Once again, the percentage of unsatisfactory grades (D's and F's) was significantly higher for the ADHD group than for either control group. The adults with ADHD had also withdrawn from more college classes for which they had initially registered than had the clinical control adults, a finding subsequently noted by Advokat, Lane, and Luo (2011) in their study of college students with ADHD. Again, we found a significantly lower college grade point average in the adults with ADHD than was the case in either control group. This was also the case in other studies of college students with ADHD (Advokat et al., 2011; Heiligenstein, Guenther, Levy, Savino, & Fulwiler, 1999). The results for SAT scores did not differ across the groups, however. But Advokat and colleagues (2011) did find such ADHD students to

TABLE 12.1. Educational Functioning by Group for the UMASS Study

Measure	(1) ADHD Mean	(1) ADHD *SD*	(2) Clinical Mean	(2) Clinical *SD*	(3) Community Mean	(3) Community *SD*	*F*	*p*	Pairwise contrasts
From interview									
Education (years)[G×S]	14.2	2.2	16.3	2.8	15.4	2.7	15.0	<.001	1 < 3 < 2
No. of times suspended[S, G×S]	1.6	3.1	0.4	0.8	0.4	0.9	8.8	<.001	1 > 2, 3
No. of times truant	18.8	33.9	8.5	15.2	5.1	10.4	8.0	<.001	1 > 2, 3
Elementary school transcript									
Percent D, F, and U grades	8.1	13.9	2.7	6.7	0.8	2.1	4.7	.01	1 > 2, 3
Days absent[A]	10.4	7.8	10.0	7.9	7.5	4.2	2.4	NS	
High school transcript									
Percent D's	17.1	15.8	8.5	11.9	7.1	9.8	12.4	<.001	1 > 2, 3
Percent F's	6.6	10.3	1.7	3.6	1.5	4.0	11.2	<.001	1 > 2, 3
Class ranking	36.2	24.6	54.4	29.0	65.0	25.3	13.0	<.001	1 < 2, 3
Mean days absent/sem.	8.4	7.0	5.7	4.0	5.0	5.0	3.1	<.049	1 > 2, 3
Verbal test percentile	63.1	26.9	72.2	25.4	69.0	24.8	1.29	NS	
Quantitative test percentile	62.5	31.6	69.5	28.9	66.5	26.9	0.6	NS	
Grade point average[S]	2.2	0.7	2.7	0.7	2.9	0.7	19.2	<.001	1 < 2, 3
College transcript									
Percent D's[G×S]	8.6	10.6	5.0	6.3	4.6	8.3	5.5	.005	1 > 2, 3
Percent F's	6.7	10.7	3.8	9.0	1.0	2.6	7.3	<.001	1 > 2 > 3
Percent withdrawals	7.5	11.2	2.5	4.3	5.1	11.6	3.9	.021	1 > 2
Grade point average[S]	2.5	0.7	2.7	0.7	3.0	0.6	9.4	<.001	1 < 2 < 3
SAT verbal score	492.2	124.5	540.2	111.1	510.7	121.1	1.29	NS	
SAT quantitative score	491.8	127.5	547.0	119.1	532.3	121.2	2.1	NS	

Note. Sample sizes for interview reports are ADHD = 145, clinical control = 94, and community control = 108. For elementary school, they were 70, 44, and 33, respectively. For high school percent D's and F's, they were 116, 80, and 86. For class ranking, they were 52, 37, and 53. For days absent, they were 44, 30, and 35. For verbal and quantitative test percentiles, they were 51, 31, and 41. For grade point average, they were 115, 78, and 86. For college, percent D's and F's. they were 75, 75, and 69. For percent withdrawals, they were 74, 71, and 69. For grade point average, they were 74, 73, and 68. For SAT scores, they were 42, 39, and 34.

SD, standard deviation; *F, F*-test results of the analysis of variance (or covariance); *p,* probability value for the *F*-test; NS, not significant; [S], significant main effect for sex (see text for details); [G×S], significant group × sex interaction (see text for details); [A], age used as a covariate in this analysis; percent D, F, and U grades, percentage of grades recorded on transcript that were D's, F's, or unsatisfactory; mean days absent/sem., average number of days absent per semester.

Statistical analyses: Groups were initially compared using two-way (groups × sex) analysis of variance (or covariance as necessary). Where this analysis was significant ($p < .05$) for the main effect for group, pairwise comparisons of the groups were conducted, the results of which are shown in the last column.

have lower ACT scores than control students. These educational problems in college students with ADHD may be linked to not only their inattentive symptoms but also decreased social and academic adjustment, lower decision-making self-efficacy, and poorer study skills, which are known to be linked to that inattention (Norvilitis, Sun, & Zhang, 2009).

The large study by Barkley and colleagues (2008) used official archival records to document objectively the adverse educational impact of ADHD in clinic-referred adults in comparison to both clinical and community control groups. Our extensive and detailed examination of school records corroborated the self-reported information of these adults and the reports obtained from others (see Table 12.1) with regard to the lower educational functioning of adults with ADHD. They did so across elementary, high school, and college educational histories. Despite having verbal and quantitative test scores that were comparable to those of the control groups in both high school and college, adults with ADHD received more unsatisfactory grades, had a lower overall grade point average, received a poorer class ranking, and, in high school, had more days absent from school than adults in our other two groups. That ADHD in adults is associated with educational impairment, and is more likely to be so than other outpatient clinical disorders, is readily apparent in the totality of these findings. Moreover, the most salient of these adverse educational outcomes (years of education, high school graduation, grade point average, etc.) both in children with ADHD followed to adulthood and adult-diagnosed cases, are mainly linked to severity of ADHD, and to a lesser extent to academic achievement skills and antisocial behavior, and not to other confounding factors, such as IQ (Barkley et al., 2008; Biederman et al., 2008). ADHD may be seen to exact a considerable toll in the educational sphere of major life activities that can be specifically attributed to it, and not to just outpatient psychiatric status, as represented in our clinical control group.

Academic Achievement Skills

Concerning actual tested academic achievement skills, children and adolescents with ADHD often manifest significant deficits on tests of academic achievement relative to control groups (Frazier, Youngstrom, Glutting, & Watkins, 2007). Early studies of adults diagnosed with ADHD indicate that they perform significantly more poorly on tests of math than those in control groups (Biederman et al., 1993). Only those adults with ADHD who were relatives of children with ADHD were found to score significantly lower on tests of reading in that study. As a consequence, more of the adults with ADHD qualified as reading disabled (6%) than did control adults (0%). Other studies have also found that clinic-referred adults with ADHD perform more poorly on reading achievement tests than do members of control groups from the same clinic (Roy-Byrne et al., 1997). Yet the mean scores on both achievement tests in these studies were still within the normal range for these adults with ADHD. And in a more recent study, the small differences in achievement did not survive statistical control for IQ (Laasonen, Lehtinen, Leppamaki, Tani, & Hokkanen, 2010). Instead, deficient academic achievement skills were most notable in adults with dyslexia, a condition that can coexist in a small but significant percentage of children and adults with ADHD (Barkley et al., 2008). The problem in some of these studies is the use of supernormal control groups and not subnormal functioning in the clinic-referred adults with ADHD. Studies of children with ADHD almost routinely indicated that they scored below normal in their academic achievement skills both in childhood (Barkley, 2006; Frazier et al., 2007; Rapport et al., 1999) and when followed into young adulthood (see Barkley et al., 2008).

Our large study of adult-diagnosed ADHD also found that participants were deficient in math, spelling, reading rate, and reading comprehension, even if their single-word reading was not different from that of our two control groups (Barkley et al., 2008). There is evidence from that study as well that in growing into adulthood, childhood ADHD has a more adverse impact on these skills than is seen in clinic-referred adults, but both groups of people with ADHD are more deficient in most domains of achievement than control groups. Impairment of reading comprehension, in contrast, seemed to occur more in the clinic-referred adults with ADHD than in the children growing up with ADHD, for some unknown reason. In general, large studies and meta-analyses of the literature on this issue seem to confirm that adults with ADHD (including college students), like children with the disorder, are more deficient than control groups on academic achievement tests (Frazier et al., 2007) even if the differences are not as substantial as they are in children and teens diagnosed with ADHD.

Learning Disabilities

The prevalence of actual learning disabilities (LD) in adults diagnosed with ADHD (0–22%) (Barkley et al., 2008; Biederman et al., 1993; Matochik, Rumsey, Zametkin, Hamburger, & Cohen, 1996; Torgersen et al., 2006) is well below that found in children with ADHD (19–50%) (Barkley et al., 2008; DuPaul et al., 2012; Lambert & Sandoval, 1980l Semrud-Clikeman et al., 1992). However, in our large study of adult-diagnosed individuals with ADHD, we found that 21% could be considered to have an LD in reading comprehension and 41% in listening comprehension (both defined as placing at or below the 14th percentile on achievement tests) compared to our community control group (10 and 15%, respectively; Barkley et al., 2008).

Differences between Adults with Child-Diagnosed ADHD and Adult-Diagnosed Cases

All this suggests the following: Clinically diagnosed adults with ADHD share some of the same types of academic difficulties in their histories as do children who were hyperactive or diagnosed with ADHD, then followed over development; however, their intellectual levels and high school graduation rates are higher, more are likely to have attended college, and their likelihood of having achievement difficulties or LD is considerably less in most respects than that seen in children with ADHD followed to adulthood. Yet adults diagnosed with ADHD in adulthood are still more impaired in most of these educational areas than adults in the control groups.

This higher level of intellectual and academic functioning in clinic-referred adults with ADHD relative to children with ADHD followed to adulthood makes sense given that they are self-referred to clinics in comparison to children with ADHD. This fact makes it much more likely that these adults have the sufficient educational level to be employed and to have health insurance. They may also be expected to have a sufficient level of intellect and self-awareness to perceive themselves as needing assistance with their psychiatric problems and difficulties in adaptive functioning. Children with ADHD who are brought to clinics by their parents are less likely to have these attributes by the time they reach adulthood. They are not as educated, they have considerable problems sustaining employment, they are more likely to have had a history of aggression and antisocial activities, and they are not as self-aware of their symptoms as adults with ADHD who are self-referred to clinics (Barkley et al., 2008). Only 3–5% of hyperactive children followed to adulthood in the Milwaukee Study endorsed sufficient symptoms to receive a clinical diagnosis of ADHD at age 21. That figure was 48% if their parents' reports were used and 66% if a developmental reference (98th percentile) was used instead of the DSM criteria (Barkley, Fischer, Smallish, & Fletcher, 2002), as noted in earlier chapters. This suggests that children with ADHD brought to clinics as children may have a more severe form of ADHD with earlier onset, or one that at least predisposes them to more severe impairments in school, than do adults self-referred to clinics and then diagnosed with ADHD.

OCCUPATIONAL FUNCTIONING

Children with ADHD as Adults

As I discussed in our earlier text on adult ADHD (Barkley et al., 2008), the results from past studies suggest that, as adolescents, individuals with ADHD function no differently in their jobs than do normal adolescents (Weiss & Hechtman, 1993). However, these findings need to be qualified by the fact that most jobs taken by adolescents are unskilled or only semiskilled, usually part-time, and typically of limited duration (summer months). As children with ADHD enter adulthood and take on full-time jobs that require skilled labor, independence of supervision, acceptance of responsibility, and periodic training in new knowledge or skills, their deficits in attention, impulse control, and regulation of activity level, as well as their poor organizational and self-control skills may begin to handicap them on the job. The findings from the few outcome studies that have examined job functioning suggest this may be the case. Two studies (in New York and Montreal) examined occupational status by adulthood and reported that their hyperactive groups ranked significantly lower than control groups (Mannuzza et al. [1993] and Weiss & Hechtman [1993], respectively). This remained the case at the 33-year follow-up of the New York study (Klein et al., 2012). Employer ratings captured in the Montreal study revealed significantly worse job performance in the hyperactive group than in the control group (Weiss & Hechtman, 1993). More of the hyperactive group had also reported having been fired or laid off than had members of the control group.

The Milwaukee follow-up study (Barkley et al., 2006) obtained employer ratings of work performance at the age 21 young adult assessment and found that hyperactive participants were rated as performing significantly worse at work than were controls.

Children who grow up with ADHD are likely to have lower socioeconomic status than their peers or control subjects in these studies (Klein et al., 2012; Weiss & Hechtman, 1993) and not only to move and change jobs more often but also to have more part-time jobs outside their full-time employment. They are also more likely to have had an adverse impact on their parents' own occupational involvement, absenteeism, lost work income, and need for disability assistance (Birnbaum et al., 2005; Kvist, Nielsen, & Simonsen, 2013; van Roijen et al., 2007; Swensen et al., 2003, 2004). Employers have been found to rate adults who as children grew up with ADHD as less adequate in fulfilling work demands, less likely to be working independently and to complete tasks, and less likely to be getting along well with supervisors. They also do more poorly at job interviews than do typically developing individuals (Weiss & Hechtman, 1993). And these adults report that they find certain tasks at work too difficult for them. Finally, children with ADHD followed to adulthood are more likely to have been fired from jobs, as well as laid off from work, relative to control participants. In general, adults who grow up with hyperactivity or ADHD appear to have a poorer work record and lower job status than typically developing adults (Weiss & Hechtman, 1993). These findings were corroborated in my own Milwaukee follow-up study with Mariellen Fischer at the age 21 follow-up (Barkley et al., 2006).

My colleagues and I studied these occupational outcomes again at the age 27 follow-up (Barkley et al., 2008). We found that significantly fewer members of the group whose ADHD had persisted to adulthood (H + ADHD group) were currently employed compared to both those whose ADHD had not persisted (H – ADHD) and the community control adults. Members of the H + ADHD group also reported that they were more likely to have problems with others at work, as well as difficulties with their behavior and workplace performance more generally compared to these other two groups. Table 12.2 shows the results of our dimensional measures of occupational functioning in this study. It is mainly the hyperactive group with current ADHD that is most impaired in these various indices of occupational success relative to the control groups. While both hyperactive groups had lower status jobs relative to our control group at this follow-up, the H + ADHD group rated themselves as having lower workplace performance quality than the other two groups. The groups did not differ in members' current annual salary or in the length of time they had held their current positions. But the two hyperactive groups reported working fewer hours per week than did the community control group. While we have yet to document differences in earnings in adults who were children with ADHD, our participants were still relatively young (mean age 27). In contrast, the 30-year follow-up of the New York samples did show a lower annual salary in their hyperactive group by midlife (mean age 41; Klein et al., 2012).

The H + ADHD group also had held more jobs since leaving high school. Given such a higher job turnover rate, we again adjusted for this difference across groups in the questions dealing with workplace adjustment by computing the percentage of jobs held in which these problems had been reported to occur. The H + ADHD group experienced a greater percentage of jobs in which members had trouble getting along with others, behavior problems, got fired or dismissed from the job, or had been disciplined formally by their supervisors than both the H – ADHD and control groups. Members of the currently employed ADHD group also reported quitting more jobs due to hostility with others than the community group, with the H – ADHD group placing between these two extremes but not differing significantly from either of the other groups. In summary, this follow-up study provides even more detailed findings on the adversities in adult occupational outcomes experienced by adults who as children were diagnosed with ADHD.

More recent follow-up studies of children with ADHD continue to document poorer occupational adjustment in these cases by adulthood. Using a large population-based sample followed from adolescence to age 37, Brook, Brook, Zhang, Seltzer, and Finch (in press) documented that teens diagnosed with ADHD as adults had 2.46 greater odds of having impaired work performance than those without ADHD (47 vs. 21%, respectively). Impaired work performance in that study was a composite variable of eight questions dealing with having been unemployed or laid off, truant from work, and receiving negative evaluations of work quality from a workplace supervisor. Age, annual income, and current marijuana use were also significant, though lesser, predictors of adult impaired work performance, in addition to adolescent ADHD.

TABLE 12.2. Occupational Functioning for Each Group on Dimensional Measures in the Milwaukee Study

	(1) H +ADHD		(2) H – ADHD		(3) Community				Pairwise
Measure	Mean	*SD*	Mean	*SD*	Mean	*SD*	*F*	*p*	contrasts
Hollingshead Job Index[IQ]	35.2	19.8	42.4	20.6	51.7	27.3	7.05	.001	1, 2 < 3
Time at current job (mos.)	22.7	22.8	27.0	29.7	30.8	26.5	1.20	NS	
No. of Jobs since high school	4.9	5.4	3.5	2.0	2.5	1.4	8.92	<.001	1 > 2, 3
Hours worked per week	43.3	14.0	44.4	9.6	49.2	12.8	4.23	.016	1, 2 < 3
Annual salary (K)[IQ]	26.3	14.4	30.7	19.4	35.1	18.1	2.83	NS	
Self-rated work quality	2.0	0.7	1.6	0.6	1.5	0.6	7.17	.001	1 > 2, 3
% jobs trouble with others	25.7	30.8	6.9	16.2	6.2	21.0	13.58	<.001	1 > 2, 3
% jobs behavior problems	26.5	36.6	6.0	15.4	2.1	9.2	20.94	<.001	1 > 2, 3
% jobs fired (dismissed)	43.2	39.1	30.0	34.4	14.0	30.3	11.45	<.001	1 > 2 > 3
% jobs quit for hostility	31.1	34.6	21.3	35.7	14.8	29.9	3.61	.029	1 > 3
% jobs quit for boredom	30.5	40.1	25.1	38.8	25.8	37.6	0.33	NS	
% jobs disciplined	28.1	34.0	8.1	22.2	3.1	15.6	17.77	<.001	1 > 2, 3

Note. SD, standard deviation; *F, F*-test results of the analysis of variance (or covariance); *p* = probability value for the *F*-test; NS, not significant; K, thousands of dollars; work quality rated 1–5 (1 = *excellent,* 5 = *poor*); [IQ], Wechsler Adult Intelligence Scale WAIS–III Vocabulary and Block Design scores were used as covariates on these measures. Where covariates were used, means are marginal means. Work quality was rated from 1 (*excellent*) to 5 (*poor*).

Statistical analyses: Groups were initially compared using one-way (groups) analysis of variance (or covariance as necessary). Where this analysis was significant ($p < .05$) for the main effect for group, pairwise comparisons of the groups were conducted, the results of which are shown in the last column.

Mordre, Groholt, Sandstad, and Myhre (2012) found that among children admitted to psychiatric hospitals in Norway and followed for 28 years, 19% were currently receiving a disability pension (comparable to Supplemental Security Income [SSI] in the United States) and that among those with current ADHD, 30% were on disability pension. This greater likelihood of disability proved not to be a consequence of comorbid internalizing or externalizing disorders but specific to ADHD. Another study, this time in the United States, found that, by adulthood, a childhood diagnosis of ADHD was associated with a reduction in employment by 10–14 percentage points, a 33% reduction in earnings, and a 15 percentage point greater likelihood of being on social assistance (Fletcher, 2014). While some of these results were also a function of reduced educational attainment and comorbid health and mental health conditions, ADHD remained a significant contributor to these outcomes.

Adult-Diagnosed Individuals with of ADHD

The previous findings pertain to children with hyperactivity or ADHD followed into adulthood, some of whom no longer have the disorder. In contrast, all clinic-referred adults diagnosed with ADHD, by definition, have the disorder. As noted earlier, for these and other reasons, the results of children with ADHD followed to adulthood may not necessarily be representative of clinic-referred adults diagnosed with the disorder. Though opinions abound on the topic in trade books on ADHD in adults, there was very little research on the occupational functioning of clinic-referred adults with ADHD until the late 1990s. In one such study of 172 adults with ADHD, we (Murphy & Barkley, 1996) reported that such adults were more likely to have been fired from employment (53 vs. 31%), to have impulsively quit a job (48 vs. 16%), and were more likely to report chronic employment difficulties (77 vs. 57%). The ADHD group also had changed

jobs significantly more often than those in the control group (6.9 vs. 4.6). Similar findings were later reported by De Quiros and Kinsbourne (2001). Their adults with ADHD reported more frequent job changes and poorer job performance than did control adults. More recently, in a large survey of 10 national samples using an adult ADHD symptom screener, de Graaf and colleagues (2008) found that 3.5% of the workers surveyed screened positive for likely ADHD, and those cases had a greater number of days of lost work performance than those without such high ADHD symptoms. Based on their level of intelligence, research shows that adults with ADHD are significantly underemployed in their occupational level of attainment (Biederman et al., 2008; Faraone & Biederman, 2005). They may also have higher rates of unemployment, particularly if they have comorbid psychiatric disorders, and be more impaired in terms of their occupational training (Sobanski et al., 2007, 2008).

A notably poor picture for employment comes from two Scandinavian countries. Torgersen and colleagues (2006) used a Norwegian sample in which just 16% of individuals with ADHD were employed at the time of referral—a figure well below that seen in studies using U.S. samples. The selection criteria used in that study suggest that members of this sample had severe ADHD and antisociality, so the symptoms likely do not correspond to the severity of ADHD seen in adult outpatient clinics. Yet the general pattern of employment difficulties is in keeping with the outcomes of follow-up studies of hyperactive children. A similar result was evident in another Norwegian study in which just 22% of adults diagnosed with ADHD were currently working, compared to 72% of the general population (Gjervan, Torgersen, Nordahl, & Rasmussen, 2012). In a study of a large Swedish sample of adults with ADHD (n = 414), Halmoy, Fasmer, Gillberg, and Haavik (2009) found that only 24% were currently employed (vs. 79% of controls). ADHD was found to predict being unemployed. Other predictors were anxiety, depression, and substance abuse. But being in treatment for ADHD was associated with a higher likelihood of being employed, as it was in the previously discussed Gjerven, Torgersen, and colleagues (2012) study, with stimulant treatment in childhood increasing the likelihood of being employed in adulthood by more than three times (odds ratio = 3.2; Halmoy et al., 2009). The inverse is also the case, in that ADHD is more prevalent among adults who are unemployed or disabled (Kessler, Adler, et al., 2006).

One study of workplace beliefs and attitudes revealed that degree of ADHD symptoms among employees was associated with more dysfunctional career beliefs, more decision-making confusion, more commitment anxiety, and more external conflict (Painter, Prevatt, & Welles, 2008). In another study college students working in teams have more difficulties performing necessary but uninteresting tasks (Coetzer & Trimble, 2010). College students have reported more difficulties and more severe difficulties in their workplace functioning in the jobs they may be doing while in college (Shifrin, Proctor, & Prevatt, 2010).

In 2008, we reported the results of our large study of adults diagnosed with ADHD in adulthood (Barkley et al., 2008). We interviewed participants about their occupational history, adversities, and current functioning, and we also obtained ratings from employers, with permission, for a subset of adults in our three groups (ADHD, clinical control, and community control). Employers were kept blind to the diagnostic status of the participants and were told only that we were conducting a survey of job satisfaction and performance, and requested their cooperation in completing a short questionnaire, for which they received $20. The interviewer also completed a global estimate of the social and occupational functioning adapted from the DSM-IV to provide a clinician summary rating of the current occupational, social, and academic functioning of the participant.

We reported that, contrary to some studies cited earlier, the groups did not differ in the proportion of members currently employed: ADHD = 73%, clinical = 71%, and community = 77%. But significantly more members of both the ADHD and clinical groups claimed that they had problems getting along with others at work (30, 18, and 7%, respectively) and had difficulties with their behavior or work performance on the job (53, 50, and 5%, respectively). The results for the dimensional measures obtained in this study are shown in Table 12.3; those measures are similar to the ones obtained in the Milwaukee age 27 follow-up shown in Table 12.2. The groups did not differ in the length of time members' had held their current work positions, averaging between 4 and 5 years, nor did they differ in the average number of hours per week they reported working (38–43 hours). Interestingly, adults in the clinical control group had significantly higher status employment, as determined on the Hollingshead Job Index, than did participants in either the ADHD or community group, who did not differ from each other.

TABLE 12.3. Occupational Functioning for Each Group on Dimensional Measures in the UMASS Study

Measure	(1) ADHD Mean	(1) ADHD *SD*	(2) Clinical Mean	(2) Clinical *SD*	(3) Community Mean	(3) Community *SD*	*F*	*p*	Pairwise contrasts
From interview									
Clinician SOFAS rating[A, G×S]	60.7	7.0	68.6	10.3	87.6	6.9	315.4	<.001	1 < 2 < 3
Hollingshead Job Index[A]	38.1	26.8	54.1	31.3	42.3	26.7	64.9	.004	2 > 1, 3
Time at current job (mos.)[S]	49.4	60.8	65.5	88.3	69.4	88.9	1.5	NS	
No. of jobs since high school[A, G×S]	7.6	7.0	8.6	7.5	5.0	3.8	10.3	<.001	1, 2 > 3
Longest time held job (in mos.)[A,S]	65.9	63.2	84.1	88.3	97.0	91.6	3.9	.022	1, 2 < 3
Hours worked per week[A,S]	42.4	15.6	43.1	12.1	38.4	13.7	1.8	NS	
Annual salary (K)[A,S,G×S]	32.6	25.8	48.1	38.0	25.8	15.5	9.5	<.001	2 > 1 > 3
Self-rated work quality	2.0	0.8	1.8	0.7	1.5	0.7	8.2	<.001	1, 2 > 3
% jobs trouble with others	32.8	37.7	19.7	28.3	12.4	23.3	13.8	<.001	1 > 2, 3
% jobs behavior problems	44.6	41.2	32.4	36.6	2.4	7.1	42.7	<.001	1 > 2 > 3
% jobs fired (dismissed)[S]	17.4	21.9	9.3	14.6	3.7	9.6	13.1	<.001	1 > 2 > 3
% jobs quit for hostility[A]	17.3	26.5	11.5	23.4	4.9	11.0	7.94	<.001	1 > 3
% jobs quit for boredom[A]	32.6	37.8	17.9	28.7	15.5	28.4	5.65	.004	1 > 2, 3
% jobs disciplined	11.1	23.4	2.4	5.3	0.6	2.3	16.3	<.001	1 > 2, 3
From employer ratings									
Inattention score[S, G×S]	9.0	7.2	5.8	5.9	1.9	2.7	21.8	<.001	1 > 2 > 3
Hyperactive–imp. score[A]	7.1	5.3	7.4	6.0	3.1	4.2	12.8	<.001	1, 2 > 3
Impair coworker relations[S]	0.7	0.7	0.7	0.8	0.4	0.7	3.4	.036	NS
Impair assigned work	1.1	1.0	0.7	0.8	0.3	0.6	13.0	<.001	1 > 2 > 3
Impair supervisor relations	0.7	0.8	0.6	0.6	0.3	0.6	2.39	NS	
Impair client relations[G×S]	0.7	0.9	0.5	0.8	0.3	0.7	3.0	NS	
Impair education at work	0.6	0.8	0.5	0.7	0.1	0.3	8.0	.001	1, 2 > 3
Impair punctuality	0.9	1.1	0.6	1.0	0.2	0.5	7.7	.001	1 > 3
Impair time management	1.2	1.0	0.7	0.8	0.4	0.7	9.36	<.001	1 > 2, 3
Impair equipment use	0.5	0.8	0.3	0.7	0.1	0.3	2.8	NS	
Impair vehicle use	0.3	0.7	0.1	0.4	0.0	0.2	3.32	.041	NS
Impair daily responses[S, G]×S	1.0	0.9	0.6	0.8	0.3	0.6	9.3	<.001	1 > 2, 3
Overall work performance	2.5	1.0	2.0	1.0	1.7	0.8	8.7	<.001	1 > 2, 3

Note. Sample sizes: For SOFAS, ADHD = 145, clinical control = 94, and community control = 108. Job index: 137, 87, 103, respectively. Time on job: 104, 66, 83. No. of jobs since high school: 139, 91, 97. Longest time held job: 142, 90, 105. Hours worked: 105, 66, 81. Salary: 105, 65, 79. Self-rated work quality: 105, 66, 84. % jobs trouble with others: 141, 90, 104. % jobs behavior problems and % jobs quit for boredom: 138, 90, 96. % jobs fired: 139, 89, 97. % jobs quit for hostility, % Jobs disciplined: 139, 90, 97. Employer ratings: 39, 25, 50.

SD, standard deviation; *F, F*-test results of the analysis of variance (or covariance); *p,* probability value for the *F*-test; NS = not significant; [S], significant main effect for sex (see text for details); [G]×S, significant group × sex interaction (see text for details); [A], age used as a covariate in this analysis; K, thousands of dollars; work quality rated 1–5 (1 = *excellent,* 5 = *poor*); impair scores are rated 0–3 (0 = *never or rarely,* 3 = *very often*); hyperactive–imp., hyperactive–impulsive symptom score; Respons., responsibilities; overall work performance rated 1–5 (1 = *excellent,* 5 = *poor*).

Statistical analyses: Groups were initially compared using two-way (groups × sex) analysis of variance (or covariance as necessary). Where this analysis was significant ($p < .05$) for the main effect for group, pairwise comparisons of the groups were conducted, the results of which are shown in the last column.

This was also true with regard to current annual salary. But on numerous other measures, the adults with ADHD showed greater occupational impairment and more adverse outcomes related to their employment than did community control adults and, to a lesser extent, than those in the clinical control group.

The adults with ADHD were rated by the clinician as functioning at a lower level than those in the clinical and community groups. While the clinical group members were also rated as more impaired, they were less so than the ADHD group. Both the ADHD and clinical groups had held more jobs since leaving high school than had the community group, even after we controlled for the age differences between these groups. And the longest time they had ever held a specific job was significantly lower for members of these groups than for the community group. The ADHD and clinical group members rated themselves as demonstrating significantly lower work quality than did participants in the community group, but they did not differ from each other in this regard. Participants reported the number of jobs on which they had experienced various difficulties, and we converted these to a percentage of the total jobs they had held since leaving high school. The adults with ADHD reported having trouble with others, behavior problems at work, being fired or dismissed from a job, quitting a job out of boredom, and being disciplined by their supervisor on the job in a higher percentage of the jobs they had held than did participants in both the clinical and the community control groups. The adults in the ADHD group also reported quitting more jobs due to their own hostility in the workplace than did adults in the community group. Based on this self-reported information, our results clearly show that adults with ADHD have greater problems with occupational functioning than those seen in the same clinic who are not diagnosed with ADHD, or those from our general community control group. Just as ADHD was found earlier to exact a significant toll on educational functioning, so also does it adversely impact the occupational functioning of adults diagnosed in adulthood.

The results of the employer ratings are also shown in Table 12.3. Although employers were blind to the diagnoses of our participants, they rated the adults with ADHD as having significantly greater problems with inattention in the workplace than was the case for either control group. Even though the clinical control adults had higher symptom ratings in this domain than those in the community group, they still fell well below the adults with ADHD. Interestingly, employers rated both the ADHD and clinical control adults as having more symptoms of hyperactive and impulsive behavior than adults in the community group. Compared to community control adults, the adults with ADHD were rated as being more impaired by their symptoms in performing assigned work, pursuing educational activities at work, being punctual, using good time management, and managing their daily responsibilities. When compared to the clinical control group, the ADHD group was again rated as more impaired in performing assigned work, time management, and daily responsibilities. As a consequence, the adults with ADHD were rated as having a poorer overall work performance level than were adults in either of the control groups. Such findings are important, in that they corroborate the reports of the adults themselves that indicate ADHD has a detrimental effect on occupational functioning, and that this effect is greater than that seen in clinically referred adults who are not diagnosed with ADHD.

The foregoing results for employer ratings must be qualified by the exceptionally small samples within each group for whom permission was granted to obtain employer ratings, and those ratings were returned to the project staff. Specifically, we had employer ratings for just 39 ADHD group members (27%), 25 clinical control members (26%), and 50 of the community control members (46%). However, additional analyses indicated that those on whom we had employer ratings in each group could be viewed as being representative of their entire group because the subsets did not differ from the entire groups on key demographic and ADHD severity measures, as well as clinician social and occupational functioning ratings.

The totality of previous results shows consistentcy between the literature on children with ADHD followed to adulthood and the smaller literature on clinic-referred adults with ADHD in finding numerous adverse events and greater impairment in the occupational functioning of adults with ADHD. These problems typically exceed the levels of impairment noted in adults having other, non-ADHD outpatient disorders, as well as community control adults, once more attesting to the fact that ADHD in adults is a more impairing disorder than most seen in outpatient settings. Children growing up with persistent ADHD, as discussed earlier in our age 27 follow-up (Barkley et al., 2008), may experience even more workplace adjustment problems than do clinic-referred adults with ADHD. While both groups have comparable rates of difficulties getting along with others and behavior problems at work,

children whose ADHD continued to adulthood experience far more firings and disciplinary actions at work as a percentage of jobs held than do clinic-referred adults with this same disorder. All of this suggests that childhood ADHD may predispose individuals toward lower occupational status regardless of the persistence of ADHD to age 27, most likely due to its adverse effects (noted earlier) on educational success and eventual years attained. But ADHD that persists to adulthood appears to have a far more adverse impact on current job functioning than does simply having ADHD as a child.

In our large study of ADHD cases diagnosed in adulthood, we (Barkley et al., 2008) were able to examine predictors of several of these outcomes (employer ratings of workplace performance and the percentage of jobs from which individuals had been dismissed or fired). We found that lower workplace performance ratings were significantly predicted by severity of self-reported ADHD symptoms and by employer ratings of severity of ADHD in the workplace. The percentage of jobs from which participants had been fired was also predicted by severity of self-reported current ADHD, severity of childhood CD, and higher antisocial activity. In a separate study, we also found that emotional impulsiveness was also a contributor to the likelihood of being fired from a job in this study (Barkley, 2010a; Barkley & Murphy, 2010). Others have subsequently found that emotional dysregulation in the workplace largely mediated the link between ADHD and occupational impairment (Gjervan, Hjerndal, & Nordahl, 2012).

The same analyses were done for the children in the Milwaukee Study samples followed to age 27 (discussed earlier). However, we did not have employer-rated workplace performance at this follow-up, so instead we substituted self-rated work performance quality, which was also significantly impaired in the H + ADHD group (see earlier discussion). Work performance was again predicted by the number of current ADHD symptoms (self-reported). But nonverbal IQ was important here as well, to our surprise. The percentage of jobs from which participants had been fired was predicted by years of education and by the number of self-rated current oppositional defiant disorder (ODD) symptoms. Better educated and less emotionally dysregulated (oppositional) participants were less likely to be dismissed from their jobs. Both predictors make perfect sense. While ADHD symptoms, criminal activities, and childhood CD symptoms had been associated with job dismissals in the UMASS Study adults, that did not occur here.

Other research and later reviews of this literature find that ADHD in adults is associated with a variety of adversities in workplace functioning and occupational health (Kupper et al., 2012), as well as work-related economic costs (Secnik, Swensen, & Lage, 2005). Adults with ADHD who are drawn from general population samples manifest higher levels of unemployment (Kupper et al., 2012) even if this is not always evident in clinic-referred samples, such as those used in my own studies (discussed earlier). They also have reduced workplace productivity; more behavioral and emotional difficulties in the workplace; and a greater risk for accidents, trauma and workplace injuries, and traffic accidents (Chapters 11 and 29). As a consequence, adult ADHD has substantial economic impact through greater absenteeism and lost productivity, among other problems it contributes to workplace functioning (Kupper et al., 2012; Secnik et al., 2005).

DATING, MARRIAGE, AND COHABITATION

Studies of adults with ADHD routinely document impairment in globally rated measures of psychosocial impairment (Barkley et al., 2008; Rucklidge, Brown, Crawford, & Kaplan, 2007; Sobanski et al., 2007, 2008; Young et al., 2003), and a small body of research has demonstrated a pattern of risky sexual behavior in teens and young adults with ADHD (see Chapter 11). All this might imply that adults with ADHD would have more difficulties in their dating and marital/cohabiting relationships. However, very few studies have focused on relationships that adults with ADHD have with others.

Children with ADHD as Adults

Most longitudinal studies of children with ADHD followed to young adulthood have not typically studied or reported problems in dating and marriage or rates of marriage, separation, or divorce (Weiss & Hechtman, 1993), most likely owing to the relatively young age of the children at adult follow-up (typically 20–30 years of age). In the Weiss and Hechtman (1993) study, just 29% of their participants were married at the young adult follow-up (mean age = 25 years). My follow-up study with Mariellen Fischer, discussed earlier, also did not find any group differences in the percentages of our groups that were currently married or separated/divorced (Barkley et al., 2008). The majority of our

groups were still single at the age 27 follow-up (55–67%). But that some marital difficulties are in the offing for the hyperactive group with persistent ADHD (H + ADHD) was evident in the self-reported quality of current marital or cohabiting relationships among those living with an intimate partner or who were formally married. Ratings of marital dissatisfaction and a greater likelihood of extramarital affairs were more apparent in this group than in the H – ADHD (nonpersistent) and community groups (Barkley et al., 2008). We found no differences in the percentages of our groups that were currently dating someone, if they were currently unmarried. Nor did we find any differences in the average number of people they had dated in the past 5 years (two people), in the average length of that dating relationship (3 years), or in the longest time they had dated someone continuously (average = 3.5 years). But ADHD groups had a higher percentage reporting fair-to poor-quality dating relationships, with a percentage four to five times that in the community group. Supporting the idea that the lack of differences in divorce rates in these follow-up studies may have to do with the relatively young age at follow-up of the samples, the 30-year follow-up study in New York recently reported higher divorce rates in their hyperactive children than in their control group by midlife (31 vs. 12%; Klein et al., 2012).

Adult-Diagnosed ADHD

That ADHD in clinic-referred adults might eventually be associated with higher percentages of separation and divorce has been suggested not only in the follow-up studies but also in a few earlier studies of adults with ADHD. Using an older sample of adults, Biederman and associates (1993) were among the first to report a higher incidence of separation and divorce among adults with ADHD, whether clinic-referred and diagnosed (28%) or as non-referred adult relatives of children with ADHD who subsequently met criteria for the disorder in a research study (36%). Murphy and Barkley (1996), also using an older sample than that in the Milwaukee Study, replicated these marital risks in a large study of clinic-referred adults in comparison to a clinical control group of adults without ADHD seen at the same clinic. They also found a marginally significant reduction in self-reported marital satisfaction on the Locke–Wallace Marital Adjustment Test (Locke & Wallace, 1959; $p < .08$) and lower but nonsignificant spouse reports on this same instrument. However, in another study, we did not find a higher divorce rate in clinic-referred adults with ADHD (Murphy et al., 2002), so the pattern here is rather mixed. But the weight of the evidence suggests that there is some relationship between ADHD and adults' risk for divorce.

This relationship between ADHD and separation/divorce or even never having been married may increase with age, as suggested in the previously discussed studies of children with ADHD followed to adulthood. Indicative of such a relationship is the recent finding that older adults diagnosed with ADHD (ages 60–94) are more likely to be divorced or never married, to have fewer family members in their social network, and to experience emotional loneliness (Michielsen et al., in press). Level of ADHD symptoms in that study was positively correlated with degree of emotional support given, emotional and social loneliness, greater recreational social participation, and lower income.

In a Canadian study with small samples of adults with ADHD ($n = 33$) and control adults ($n = 26$), Minde and colleagues (2003) found that marital and family functioning were more impaired in the ADHD group than in the control group, regardless of the sex of the parent with ADHD. As in the study by Murphy and Barkley (1996), self-reported marital adjustment was lower in adults with ADHD than in control adults, with 58% falling in the maladjusted range of their measure (vs. 25% for the control group). Yet their spouse reports on this same measure did not differ from spouse reports of the control group members. These authors, however, did not find higher rates of separation or divorce in the ADHD group, despite its comparable divorce/separation rate (27%) to that found in the Biederman and colleagues (1993) study (discussed earlier). This may have been a result of the small samples and low statistical power in that Canadian study.

The greater marital dissatisfaction and functioning in adults with ADHD are not surprising given that adults with ADHD rate themselves as being more impatient, easily frustrated and angered; as having frequent temper outbursts and more unstable personal relationships; and as breaking off those relationships over trivial matters and have difficulty maintaining friendships (Barkley & Murphy, 2010; De Quiros & Kinsbourne, 2001; Murphy & Barkley, 1996). Such dissatisfaction also arises from the emotional dysregulation associated with the disorder (Barkley & Murphy, 2010). Relationship and marital problems would also be expected given all of the difficulties with impulsiveness, attention, self-regulation, and executive functioning evident in adults

with ADHD (see earlier chapters). The greater likelihood and diversity of financial difficulties demonstrated below in the ADHD groups also would be expected to weigh heavily on marital or cohabiting relations.

A small Canadian study by Eakin and colleagues (2004) found evidence of poorer self-rated marital adjustment and family dysfunction in adults with ADHD (n = 33) than in control adults (n = 26). But the partners of these adults did not rate their marriages as more poorly adjusted or dysfunctional. Once more, the problem in such small studies is low statistical power to detect group differences. In our large study of adults with ADHD compared to clinical and community control groups (discussed earlier), we collected some measures of marital status and satisfaction (Barkley et al., 2008). Our results showed that the two clinic-referred groups (ADHD, clinical control) were less likely ever to have been married than members of the community control group. The ADHD group specifically was significantly less likely to be currently married (vs. being currently single) than the community group, but it did not differ significantly from the clinical group. Among those who were currently married, the ADHD group also had a higher percentage of members who rated the quality of their marriage as poor than the community group. The clinical group, once again, did not differ from either of these other groups. There was no difference in the incidence of divorce among our groups. In that sense, our results disagree with the earlier reports of higher divorce rates by Biederman and colleagues (1993) and our own earlier study (Murphy & Barkley, 1996), but they agree with the report of Minde and colleagues (2003) and another of our large studies (Murphy et al., 2002). The disparity in findings across these studies is not readily explained at this time and leaves open to doubt whether ADHD in adults is associated with a greater likelihood of divorce. Less in doubt is the consistently greater proportion of ADHD groups reporting poorer quality of their marital relationships.

When we examined sex differences in marital status in our studies, a few findings were noteworthy. Comparisons of males and females with ADHD within each group showed no differences for ever being married or being currently married, but females were more likely to have been divorced (21 vs. 7%, p = .013). As for the duration of marriage, the average length of time participants had been in their current marriages did not differ across groups when we controlled for age. (Marginal means were ADHD = 14.2, clinical = 12.9, community = 14.7 years.) And there were no sex differences in the percentage of participants who rated the quality of their marriage as poor.

We administered the Locke–Wallace Marital Adjustment Test (Locke & Wallace, 1959) to our participants and to a smaller sample of current spouses of our participants. We found that both the ADHD and clinical control groups reported significantly lower marital satisfaction than did the community control group (Barkley et al., 2008). In fact, the average score for both the ADHD and clinical groups fell within the range believed to reflect marital dysfunction (less than 100). The same results were observed for spousal reports on this same measure. Our results agree with our earlier report (Murphy & Barkley, 1996) and those of Minde and colleagues (2003) and Eakin and colleagues (2004) concerning greater marital dissatisfaction in the adults with ADHD compared to adults from a community sample. But unlike those studies, we also found greater dissatisfaction in the reports of their spouses. Nevertheless, our large study shows that such marital dissatisfaction, whether in self- or spousal reports, is not specific to just the ADHD group but also can be found in clinic-referred adults who are not diagnosed with ADHD. This is hardly surprising given that adults in the clinical group were also experiencing significant psychiatric disorders and psychological maladjustment, as well as higher than normal levels of ADHD symptoms, even if not formally diagnosed with ADHD, that would be expected to have some impact on marital relationships.

More detailed ratings of marital problems were collected in a pilot study involving 80 couples in which one partner had ADHD (Robin & Payson, 2002). The partners with ADHD had significantly more marital issues, felt more unloved, and reported a more negative impact of ADHD on the marriage than did the partner in the dyad without ADHD. Interestingly, the male partners without ADHD reported significantly greater marital problems on these three scores than did the female partners who did not have ADHD. This may imply perhaps that female partners who do not have ADHD are either more tolerant of their male partner's ADHD in a marriage or are less willing to express it than are males reporting about their female partner with ADHD. In research on a related topic, two studies with college students, one in the United States and the other in China, found that increased levels of ADHD symptoms were associated with increased fear of intimacy in intimate relationships, lower expectations for intimacy in the relationships, and lower relationship

self-confidence but no greater levels of sexual anxiety (Marsh, Norvilitis, Ingersoll, & Li, in press).

A recent study that observed the patterns of communication in intimate romantic couples directly corroborates results of these earlier studies based on rating scales. That study compared couples in which one partner had ADHD to a control group of couples without ADHD (Canu, Tabor, Michael, Bazzini, & Elmore, in press). Results indicated that couples in which one partner had ADHD combined type reported less relationship satisfaction and showed more negativity and less positivity in their behavior during a conflict resolution task compared to couples in which the partner had either the inattentive type of ADHD or no ADHD.

Such conflicted communication patterns seem to be consistent with another recent finding from two studies on the risk of violence in intimate partner relationships in adults with ADHD. Wymbs and colleagues (2012) studied adult males with a history of ADHD and found them to be more verbally aggressive and violent with their romantic partners than males without such a history. The presence of conduct problems in the males with early ADHD also was found to contribute further to the risk for such behavior. More recently, a study in England employed a representative sampling involving 7,369 households from which ratings of adult ADHD symptoms and reports of violence were collected (González, Kallis, & Coid, 2013). After adjusting for demographic factors and known clinical predictors of violence (antisocial personality, substance misuse, anxiety disorders), results indicated that adult ADHD was moderately associated with violence (odds ratio = 1.75) and that principally the hyperactive–impulsive dimension of the disorder, and not inattention, was linked to violence. Such violence was primarily with intimate partners. Mild to moderate levels of ADHD were linked to such intimate partner violence, whereas severe ADHD levels were linked to violence primarily through comorbidity.

To summarize, longitudinal studies of ADHD in children followed into young adulthood have not consistently documented differences in marriage or divorce probabilities, but the one study that went on the longest (33 years), extending into midlife, did so. Even so, the few studies examining ratings of marital or cohabitation satisfaction have suggested emerging problems in those relationships by young adulthood. Studies of clinic-referred adults or those ascertained by other means (parents of ADHD children) present a more mixed picture, with half finding higher divorce rates and others not doing so. But all studies have found that clinic-referred adults with ADHD, like children growing up with persistent ADHD to age 27, rate the quality of their marital or intimate relationships as less satisfactory. Ratings of marital communication, as well as direct observations of communication patterns, suggest lower positivity and greater negativity in conflict resolution tasks. And populationwide studies suggest a link between moderate levels of ADHD symptoms and likelihood of intimate partner violence. Adults with ADHD may be more likely to have children with ADHD, and child ADHD has been shown to increase the likelihood of parental marital problems and divorce even after researchers account for genetic relationships between parents and children (Schermerhorn et al., 2012). Perhaps this explains the mixed results in studies of adults with ADHD concerning rates of divorce; it is primarily couples with one partner who has ADHD *and* a child with ADHD that are likely to have the highest risk of marital problems and divorce. It is a hypothesis worthy of future research.

The problems noted earlier in intimate (dating, cohabiting, and marriage) relationships mean that clinicians working with adults with ADHD will have to identify regional resources to help their clients address these social difficulties. Intervention in relationships will be required for many adults with ADHD and their partners, beyond just the clinical management of ADHD symptoms via traditional treatments. Clinicians may also find it useful to share the trade book by Pera (2008) with adult clients with ADHD who have difficulties in their intimate relationships (see also Chapter 34).

MONEY MANAGEMENT

Until 2006, there seemed to be no research on the specific money management problems that may be associated with ADHD in clinic-referred adults other than off-handed references to a few problems. De Quiros and Kinsbourne (2001) reported that two items from their Adult Problem Questionnaire that pertained to money matters were rated as occurring more often in their ADHD group than in their control group. These concerned frequent shopping sprees and having trouble sticking to a budget. But given the poor impulse control and self-regulation associated with the disorder, it is reasonable to anticipate problems with handling money in adults with ADHD.

Children with ADHD as Adults

Subsequently, one study of children with ADHD (hyperactivity) followed to adulthood reported results for financial management at the age 21 follow-up. In the Milwaukee Study, Mariellen Fischer and I found that significantly more members of the community control group than members of the hyperactive (ADHD) group had ever had a credit card; otherwise, the groups did not differ in the proportion ever having had a car loan, other bank loans, or currently owing money to others (Barkley et al., 2006). Significantly fewer members of the ADHD group had a savings account, and more of the ADHD group reported having trouble saving money to pay their monthly bills. The groups' current annual salary amounts did not differ. Although the amount of average savings for the ADHD group was lower than that of the community control group, this difference was not significant. The groups did not differ in terms of debt amount of members who had owned credit cards, taken out current car loans, or had other outstanding bank loans. But members of the ADHD group owed significantly more money to other private individuals than did members of the community control group. Given that the participants in this study had an average age of approximately 21 years, there was not enough time since leaving school for differences in financial status and management issues to have become apparent. Even so, this study intimates that there might be some impact of ADHD on financial management later in adulthood if these current trends were to continue.

To explore that possibility, we again collected data on financial management from these same participants 6 years later at the age 27 follow-up (discussed in an earlier textbook; Barkley et al., 2008). The results for the categorical measures can be observed in Table 12.4. In all but one of the 13 money issues, a significantly larger percentage of the group with persistent ADHD to age 27 (H + ADHD) had problems than did the community control group. The exception was for writing checks with insufficient funds, in which no group differences were found. In seven of these problem areas, the H + ADHD group also had a higher risk than the nonpersistent ADHD (H – ADHD) group, including trouble managing their money, buying on impulse, missing rent and credit card payments, exceeding credit card limits, not having a savings account, and having a poor credit rating (self-reported). In some areas, the two hyperactive groups had more participants with problems than the community group but did not differ from each other, suggesting that having been a child with hyperactivity and ADHD carried some risk for financial problems even if the ADHD had not persisted to this follow-up. These areas included difficulty saving money, having utilities turned off for nonpayment, having a vehicle repossessed, declaring bankruptcy, and not saving for retirement. This was also evident in other problem areas in which the H – ADHD group fell below the H + ADHD group's level of risk yet remained at higher risk than the community controls, such as in managing money, buying on impulse, missing rent payments, and having a poor credit rating. In summary, both hyperactive groups had higher percentages of many of these financial problems than did the control group, which suggests that growing up with ADHD from childhood is a risk factor for financial difficulties even if that ADHD does not fully persist to age 27. But when ADHD does persist, it increases the risks of financial difficulties even more. Those problem areas in which the H + ADHD group differed from both the H – ADHD and community groups are visually depicted in Figure 12.1.

On the dimensional measures of finances, we found that members of both hyperactive groups were earning less money per month than members of the community control group, yet the hyperactive groups did not differ from each other in terms of income. The three groups did not differ statistically in the amount they had saved. A better index here of propensity to save is probably the ratio between money currently saved and total annual income, which in part controls for the greater income received by the control group, whose members therefore have the potential to save more. This measure clearly showed that members of the two hyperactive groups were saving proportionately about three times less as a function their annual income than were the control participants (3 vs. 4 vs. 11%, respectively).

The frequency with which various money problems had occurred in these groups did not differ across most other measures, except for exceeding the credit limit on credit cards, which the H + ADHD group did more often than the other two groups. The H + ADHD group members also reported having significantly poorer credit ratings than the other groups. Yet the H – ADHD group also reported a poorer rating than the community control group, which indicates that poor credit can be associated with children with ADHD growing up, even if their ADHD does not persist fully

TABLE 12.4. Money Management Problems by Group for the Milwaukee Study

Measure	(1) H + ADHD		(2) H – ADHD		(3) Community		χ^2	*p*	Pairwise contrasts
	N	%	*N*	%	*N*	%			
Trouble managing money	39	71	33	41	15	20	33.89	<.001	1 > 2 > 3
Difficulty saving money	35	65	43	54	14	19	32.11	<.001	1, 2 > 3
Problems buying on impulse	42	78	36	45	18	24	36.61	<.001	1 > 2 > 3
Ever missed paying rent	17	31	13	16	4	5	15.70	<.001	1 > 2 > 3
Had utilities turned off for nonpayment of bills	23	43	26	33	8	11	18.03	<.001	1, 2 > 3
Missed credit card payment	28	58	26	37	29	39	6.22	.045	1 > 2, 3
Exceeded credit limits on cards	28	64	27	39	33	45	6.98	.031	1 > 2, 3
Wrote check with insufficient funds to cover the amount	37	70	50	63	38	51	5.21	NS	
Had a vehicle repossessed	6	15	8	13	1	1	7.96	.019	1, 2 > 3
Declared bankruptcy	5	9	15	19	3	4	8.90	.012	1, 2 > 3
Do not have a savings account	33	61	31	39	20	27	15.61	<.001	1 > 2, 3
Not saving for retirement	42	76	53	66	34	45	14.16	.001	1, 2 > 3
Have a poor credit rating	29	54	26	32	6	8	32.42	<.001	1 > 2 > 3

Note. Sample sizes for the comparisons on interview information were H + ADHD = 55 , H – ADHD = 80, and community = 75 for all categories except car repossession, where N's = 40, 61, 69, respectively; exceeding credit card limits, where *N*'s = 44, 70, and 73, respectively; and missing a credit card payment, where *N*'s = 48, 71, and 74, respectively. *N,* sample size endorsing this item; %, percent of group endorsing this item; χ^2, results of the omnibus chi-square test; *p* = probability value for the chi-square test; pairwise contrasts, results of the chi-square tests involving pairwise comparisons of the three groups. H + ADHD, hyperactive group that currently has a diagnosis of ADHD at follow-up; H – ADHD, hyperactive group that does not have a diagnosis of ADHD at follow-up. A poor credit rating was categorized as a self-report of a credit rating of 4 or 5 (*poor* or *very poor*).

Statistical analyses: Pearson chi-square.

to age 27. We created a sum of the number of different money problems experienced by the participants. Here again the group with persistent ADHD (H + ADHD) had significantly more such problems than the other two groups, but the H – ADHD group also had more problems than the control group. Such findings suggest that as clinic-referred children with ADHD develop into adulthood, they have significantly more financial problems than do community control children, but the greatest money problems are found in children whose ADHD persists to age 27.

The Milwaukee Study also delved in detail into the extent to which participants engaged in various gambling activities and the size of their wagers. We did so because we believed that the impulse control problems experienced by the hyperactive groups might make them more susceptible to the ubiquitous opportunities for gambling now extant in the United States. We found little evidence for this hypothesis. The groups did not differ in the percentage that had ever bet money (73–80%), and specifically bet at state lotteries (73–78%), racetracks (25–37%), sports (47–56%), card games (48–67%), and slot machines (71–78%). There was also no difference in how often they played the state lottery; the average was less than four times per year across the groups and $3–5 per bet. There also were no differences in the frequency with which the groups had engaged in betting at the racetrack, on sports, and at slot machines, or differences in how much they spent each time, lost in a day, or the largest amount ever lost for those activities. The groups also did not differ in the number of different types of betting activities in which members had ever engaged. So we concluded that ADHD in children is not associ-

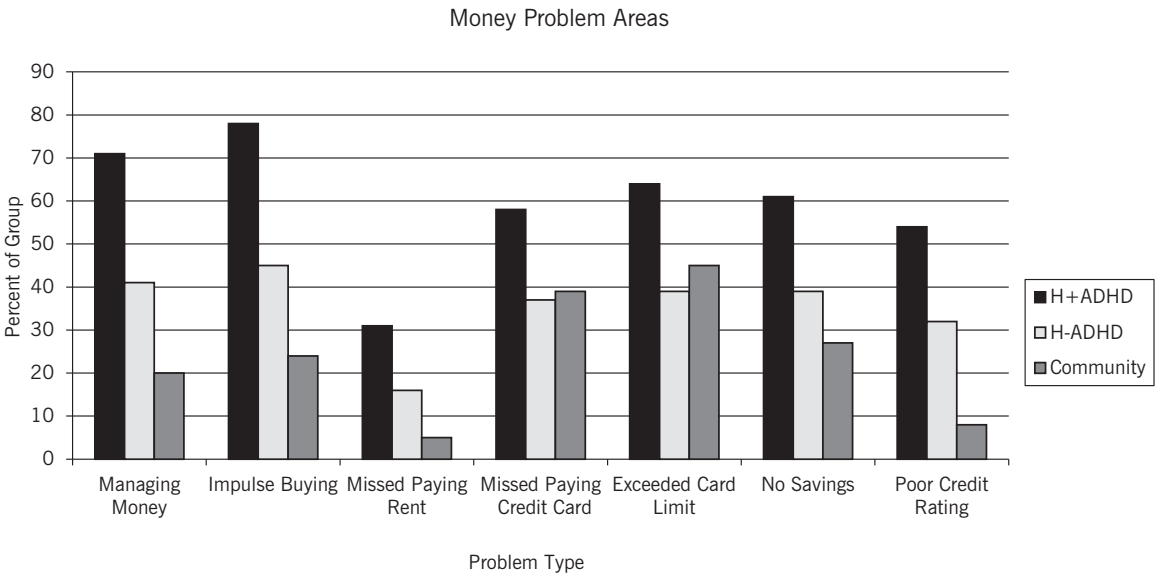

FIGURE 12.1. Percent of each group having various money management problems in the Milwaukee Study. These are the measures on which the H + ADHD group differed significantly from the H – ADHD and the community control groups. H + ADHD, hyperactive group that currently has a diagnosis of ADHD at follow-up; H – ADHD, hyperactive group that does not have a diagnosis of ADHD at follow-up. From Barkley, Murphy, and Fischer (2008). Copyright 2008 by The Guilford Press. Reprinted by permission.

ated with elevated levels of gambling by young adulthood except perhaps for card playing as they grow up. We should not be surprised by this finding for several reasons. First, in the New York follow-up study (Mannuzza et al., 1993, 1998), the hyperactive group was no more likely than the control group to be diagnosed with pathological or addictive gambling-related disorders. Second, other research indicates that excessive gambling is related principally to antisocial personality disorder (ASPD) and not to ADHD (Raylu & Oei, 2002). Our subsequent analyses of our young adults who did and did not have ASPD largely confirmed this conclusion. Our young adults with ADHD were not more prone to gambling than the general population but those who likely had ASPD were more likely to bet on sporting events, play slot machines more often, and bet and lose significantly more money at most of these activities (Barkley et al., 2008). One recent study (Dai, Harrow, Song, Rucklidge, & Grace, in press) did find an association between ADHD and problem gambling, as well as performance on a simulated gambling task in small samples of adults with ADHD and controls. The findings were partially mediated by degree of impulsivity in the adults. But the study neither examined nor controlled for the overlap of adult ADHD with CD or ASPD; thus, it is not clear whether ASPD accounted for this association, which was not evident in prior studies. Future research is needed to sort out factors that may contribute to problem gambling in adults with ADHD.

More recent research has confirmed this association between ADHD in childhood and financial problems in adulthood. A large population-based study revealed that the presence of ADHD at ages 14–16 years was associated with 3.33 times the likelihood of high financial stress (worries about finances) by age 37 years (Brook et al., in press). Nearly 30% of those with ADHD were classified as having such a degree of financial distress, compared to just 11% of individuals without ADHD. Apart from ADHD, current smoking was also a significant, though far less substantial, predictor of high financial stress in adults.

Adults Diagnosed with ADHD

The only other study to look at financial problems in adults with ADHD to date was our UMASS Study, in which we compared adults clinically diagnosed with ADHD to clinical and community control groups (Barkley et al., 2008). The following discussion is taken largely from that textbook. We used the same financial interview with these participants as we did in the previously discussed Milwaukee age 27 follow-up, permitting a direct comparison of the two studies. The results concerning the percentage of each group experiencing 12 different money management problems appear in Table 12.5. More adults with ADHD reported problems in eight of the 12 areas of money management than did adults in the community control group. The ADHD group had a higher proportion of its members reporting problems with managing money, saving money, buying on impulse, nonpayment of utilities resulting in their termination, missing loan payments, exceeding credit card limits, having a poor credit rating, and not saving for retirement. Relative to the community control group, the adults with ADHD appeared to have pervasive problems with the management of their finances. The findings are consistent with the far less comprehensive report of De Quiros and Kinsbourne (2001) noted earlier, indicating a greater likelihood of shopping sprees and poor adherence to a budget in their adults with ADHD.

There were also problems that affected even a larger percentage of the ADHD group than the clinical control group. Those comparisons are depicted in Figure 12.2 and give a better picture of the risks associated specifically with ADHD and not just outpatient referral status. The ADHD group members were more likely to have trouble saving money, to buy on impulse, to avoid paying their utilities, and not to save for retirement. While the clinical control group also had difficulties in five of these areas compared to the community control group, they were less likely than the ADHD group to have such difficulties, particularly in saving money and buying on impulse. Those four areas of money management in which the ADHD group differed from both the clinical and community control groups involve rather specific financial problems having to do with deferred gratification (saving and putting money away for retirement), impulse buying, and probably organization and

TABLE 12.5. Money Management Problems by Group in the UMASS Study

	(1) ADHD		(2) Clinical		(3) Community				
Measure	*N*	%	*N*	%	*N*	%	χ^2	*p*	Pairwise contrasts
Trouble managing money	97	67	53	57	16	15	72.3	<.001	1, 2 > 3
Difficulty saving money	94	65	47	50	19	18	57.3	<.001	1 > 2 > 3
Problems buying on impulse	90	62	44	47	13	12	65.4	<.001	1 > 2 > 3
Ever missed paying rent	34	23	18	19	16	15	2.9	NS	
Had utilities turned off for nonpayment of bills	46	32	16	17	14	13	14.5	.001	1 > 2, 3
Missed loan repayment	83	57	50	53	29	27	25.2	<.001	1, 2 > 3
Exceeded credit limits on cards	68	47	38	40	31	29	8.6	.013	1 > 3
Wrote check with insufficient funds to cover the amount	92	63	60	64	60	56	2.0	NS	
Had a vehicle repossessed	10	7	3	3	4	4	2.2	NS	
Declared bankruptcy	8	6	5	5	9	8	2.4	NS	
Have a poor credit rating	34	26	17	19	7	7	14.3	.001	1, 2 > 3
Not saving for retirement	101	71	48	52	45	42	22.0	<.001	1 > 2, 3

Note. Sample sizes for the group comparisons on interview information were ADHD = 144, clinical = 93, and community = 108. *N*, sample size endorsing this item; %, percent of group endorsing this item; χ^2, results of the omnibus chi-square test; *p* = probability value for the chi-square test; pairwise contrasts, results of the chi-square tests involving pairwise comparisons of the three groups.

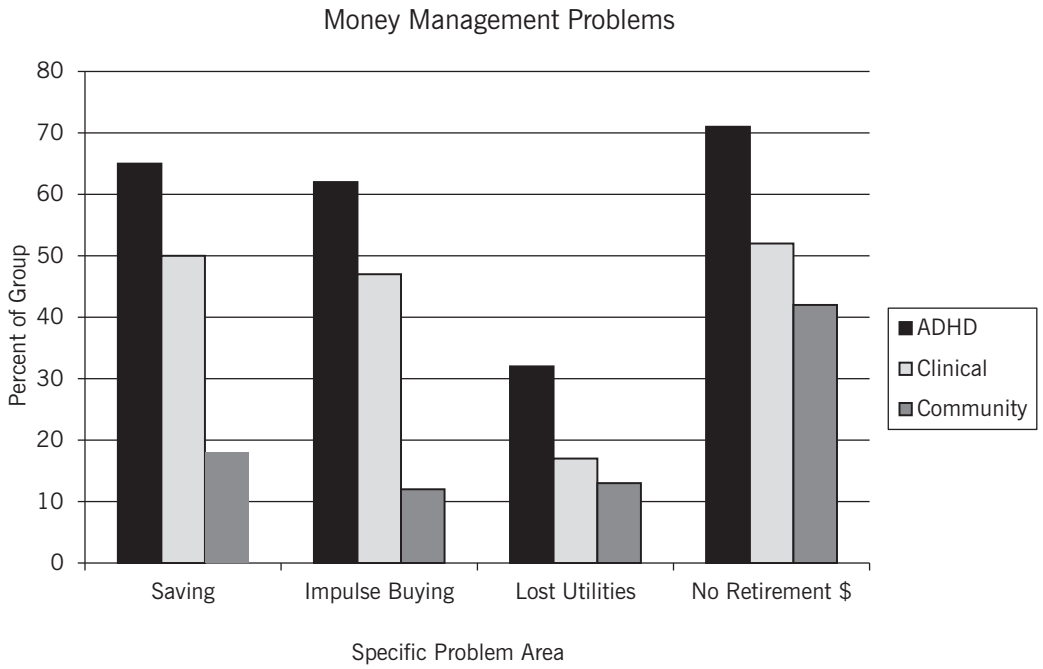

FIGURE 12.2. Percent of each group having various money management problems in the UMASS Study. These are the measures on which the ADHD group differed significantly from the clinical control and community control groups. $, savings. From Barkley, Murphy, and Fischer (2008). Copyright 2008 by The Guilford Press. Reprinted by permission.

meeting deadlines (nonpayment of utilities resulting in their termination).

We also had participants answer five financial questions having to do with the frequency with which they may have experienced a money problem. In all five measures, adults with ADHD reported these difficulties more often than did adults in our community control group. Money difficulties were also more common in the ADHD than in the clinical control group in at least four of these six areas, including missed rent, utility, and loan payments and having more total money problems. Missed loan payments was the most common problem reported across groups, though it was significantly more common in ADHD group members than in the two control groups. We also computed a money problem diversity score by summing the number of different problem areas in which a participant reported a problem across the 12 areas reported in Table 12.5. This index revealed that adults with ADHD have a significantly greater number of financial problems than either adults without full ADHD referred to clinics or community adults.

In comparison to the persistent ADHD group in our Milwaukee follow-up study discussed earlier, the percentages here for the clinic-referred adults with ADHD in the UMASS Study are nearly identical, with one exception: the larger percentage of the former group that had not yet begun saving for retirement—a difference that likely arises from the younger age of the sample used in the Milwaukee Study than that used in the UMASS Study. Overall, this is a striking replication of findings across two different methods of ascertaining ADHD in adults. It suggests that the disorder is strongly associated with financial management problems. What the Milwaukee Study adds to this conclusion is that risk for financial problems is also higher in individuals who had ADHD in childhood but were diagnosable as such at age 27 years.

One should not be surprised at these difficulties with money management given the problems with in-

hibition, executive functioning, and self-regulation associated with ADHD, as discussed in earlier chapters. These seem to be the only studies of adults with ADHD or children who grew up with ADHD that actually examine the impact of ADHD on specific problem areas related to financial management. From these studies we can conclude that the disorder does seem to have an adverse impact on many aspects of financial management, such as saving money, buying on impulse, and repaying debts. With advancing age, there will likely arise greater opportunities for problems with finances to become even more apparent in adults with ADHD at midlife.

Predicting Financial Problems

In the prior studies, my colleagues and I examined some possible predictors of these financial problems (Barkley et al., 2008). The financial outcome (criterion) we selected for prediction was the number of different money problems experienced by participants, described earlier as the money problem diversity score. Our analyses of the adults in the UMASS Study (clinic-referred adults) indicated that the severity of ADHD symptoms made a significant contribution to this money problem index, but so did the number of childhood CD symptoms retrospectively recalled, though of a much smaller magnitude than the degree of variance explained by ADHD severity. Education, IQ, criminal diversity, and the self-ratings of depression, anxiety, and hostility made no significant contribution to this index. Thus, ADHD largely accounts for the degree of financial difficulties of these participants, explaining nearly 21% of variance in the diversity of such problems.

We conducted the same type of analysis in the Milwaukee Study, which indicated that five predictors were significant and accounted for nearly 24% of the variance. These were severity of childhood hyperactivity (ADHD), pervasiveness of childhood ADHD and behavior problems, the number of CD symptoms at adolescent follow-up, and the number of ADHD symptoms (self-reported) and years of education at the prior age 21 follow-up. This study shows that not only adulthood (age 21) but also childhood ADHD symptoms and their pervasiveness predict current money problems. Beyond that, the severity of teen CD symptoms made a smaller additional contribution. In other words, persistence of ADHD to age 21 is a further predictor of financial problems; in addition, initial childhood ADHD severity and the development of CD symptoms by age 15 may further accentuate these financial adversities. Years of education was not an unexpected predictor given its link to occupational status and hence to income. These two studies are consistent in showing that ADHD and, to a lesser extent, CD are related to extent of current financial problems in adulthood. In subsequent papers, we reported that emotional impulsiveness is also associated with some of the financial problems (impulse buying, exceeding credit card limits, etc.) in both the Milwaukee follow-up study (Barkley & Fischer, 2010) and the study of adults diagnosed with ADHD (Barkley & Murphy, 2010).

In conclusion, it makes sense that adult ADHD would be associated not only with financial problems, in view of the symptoms and poor self-regulation associated with the disorder that would hamper sound judgments about managing money and credit, but also educational and occupational problems (discussed earlier) that would place more constraints on income of people with ADHD relative to those without the disorder. These findings also imply that clinicians who work with adults with ADHD need to be aware of resources in their region that may be able to assist these adults with managing their finances and associated legal problems, beyond just the traditional mental health treatments used to manage the disorder. Resources that may prove useful include the trade book for adults with ADHD on managing finances (Klein & Sarkis, 2009) and my own book for adults with ADHD (Barkley, 2010a).

ECONOMIC IMPACT OF ADHD-RELATED IMPAIRMENTS

Only a few studies have examined the economic costs associated with some of the previously discussed impairments, but their findings are both sobering and rather staggering in terms of the likely adverse economic impact associated with ADHD. The previous chapter covered the substantial health care-related costs of ADHD for both the patient and his or her immediate family. Recall the $1.8 billion in annual costs for ADHD-related treatments and $12.1 billion in other health care costs, not to mention $14.1 billion in health care costs for immediate family members (Birnbaum et al., 2005). But these other domains of impairments in major life activities discussed here for adults with ADHD also have their economic costs. For instance, in the aforementioned study, Birnbaum and colleagues also calculated that the cost of work loss associated with ADHD

was $3.7 billion annually. In a later study, Kessler, Lane, Stang, and van Brunt (2009) found that adult ADHD is linked to 4–5% poorer work performance and estimated the cost in the workplace to be $4,336 annually per worker with ADHD. This did not include the cost of workplace accidents that are believed to be more common in adults with ADHD. Fletcher (2014) recently estimated that by adulthood, childhood ADHD is associated with a 15% increase in the likelihood of receiving social (financial) assistance, a 10–14% reduction in the likelihood of being employed, and a 33% lower annual income. Furthermore, the cost in lost wages (hence, associated taxes paid and value to society) for those who do not complete high school compared to those who do is $630,000 over the course of one's working life (Chapman, Laird, Ifill, & KewalRamani, 2011). Considering that teens and young adults with ADHD are two to three times less likely to complete high school than the general population greatly adds to the societal economic costs of this disorder. Consider also the cost of criminal activities by teens and young adults with ADHD, which has been estimated to be $25,000 per youth and perhaps as high as $65,000 (Jones, Foster, and the Conduct Problems Prevention Research Group, 2009) at an estimated annual cost of $2–4 billion. Add to all of this the excessive costs of auto crashes associated with ADHD, estimated to be $2,600 per crash, with three times more crashes per patient with ADHD, and the uncalculated costs of greater bodily injury from such crashes (Barkley & Cox, 2007), and one can see the remarkable economic toll of ADHD that extends into adulthood. It should be evident that the total economic burden of ADHD far exceeds the intervention costs to treat this disorder in adults.

KEY CLINICAL POINTS

- ✓ The small literature on the educational histories of adults with ADHD has suggested numerous adverse effects of the disorder in the domain of major life activities, consistent with the follow-up studies of children with ADHD, but often suggesting somewhat less impairment in clinic-referred adults.
- ✓ Compared to children with hyperactivity or ADHD followed over development, the intellectual levels of adults with ADHD are higher, their high school graduation rates are higher, more are likely to have attended college, and their likelihood of having achievement difficulties is considerably less in most respects than that seen in children with ADHD followed to adulthood.
- ✓ Nevertheless, adults with ADHD rate themselves as being more impaired in educational settings than do adults in control groups. This is corroborated by the ratings provided by others, which indicate that ADHD is associated with impaired functioning in all of the specific educational situations we examined, including classwork, homework, class behavior, and behavior at recess and in the lunchroom, as well as overall time management.
- ✓ Although as many adults with ADHD graduate high school as those in control groups, fewer graduate from college, resulting in the ADHD group having less years of education.
- ✓ More of the adults with ADHD reported having been retained in grade, receiving special education, and being diagnosed with LD or behavior disorders while in compulsory schooling than did adults in either of the two control groups.
- ✓ Studies that obtained official school transcripts revealed a similar pattern. The ADHD group has a significantly greater percentage of poor (D) or failing grades (F) both on their elementary school and high school transcripts. They also had a lower grade point average and more days absent from school during high school than did the adults in control groups.
- ✓ Similarly, among college attendees, those with ADHD had more unsatisfactory grades, withdraw from more classes, and may have lower college entrance testing scores than did college students in control groups.
- ✓ Evidence of lower academic ability was also found on tests of educational achievement. The adults with ADHD had poorer scores in their arithmetic, spelling, and reading and listening comprehension skills than did adults in control groups.
- ✓ In contrast, follow-up studies indicate that having ADHD as a child is a major risk factor for most types of educational problems, whether or not the ADHD persists to age 27. Both hyperactive (child ADHD) groups were less educated, less likely to graduate high school, less likely to attend college, and more likely to have received various forms of educational assistance in school than the control groups. Compared to clinic-referred adults, it is clear that children growing up with ADHD are even more adversely affected in their edu-

cational careers, as implied by the earlier research literature.

- ✓ Concerning specific LD, clinic-referred adults with ADHD were more likely to have spelling and comprehension disorders (reading and listening) than community control groups, whereas only their listening comprehension disorders distinguished them from clinical control groups. Noteworthy is that the most common area of deficiency or specific LD had to do with reading and listening comprehension—abilities established previously to be deficiencies in children with ADHD related to their working memory deficits. Childhood ADHD, regardless of its persistence to adulthood, is more likely to be associated with specific LD than is adult ADHD.
- ✓ In their occupational functioning, clinic-referred adults with ADHD are rated by clinicians as functioning at a lower level overall than adults in control groups. They have experienced a number of problems in a higher percentage of their previous jobs than adults in control groups: getting along with others, demonstrating behavior problems, being fired, quitting out of boredom, and being disciplined by supervisors.
- ✓ Growing up as a child with ADHD is associated with lower job status and fewer current working hours per week regardless of whether the ADHD persists into adulthood. Even so, those with persistent ADHD experience even more difficulties in current workplace functioning than do either nonpersistent or control groups. Noteworthy is that children with ADHD that persists to adulthood have a far greater percentage of jobs in which they are fired or experience disciplinary actions than do clinic-referred adults with the disorder.
- ✓ Employer ratings corroborate these self-reports. Adults with ADHD are rated as having significantly more symptoms of inattention in the workplace, and as being more impaired in performing assigned work, pursuing educational activities, being punctual, using good time management, and managing daily responsibilities. These problems have also been found in earlier studies of children with ADHD followed to adulthood. Both types of studies provide direct evidence via not only self-reports but also blinded employer rating that ADHD has an adverse impact on workplace functioning.
- ✓ The results presented here clearly demonstrated that ADHD in adults is associated with a number of adverse outcomes and more impaired functioning in their educational and occupational histories than is the case for adults without ADHD or those diagnosed with other clinical disorders. Being diagnosed as ADHD in childhood has an even more adverse effect on one's educational career, eventual job status, and workplace adjustment problems (firings and disciplinary actions) than when ADHD is diagnosed in self-referred adults.
- ✓ Treating children with ADHD medications was found in several studies to result in a greater likelihood of being employed by adulthood, suggesting that longer term treatment of children and teens with medication may have downstream benefits on employability.
- ✓ Clinicians are likely to be asked to involve themselves in the educational impairments of those adults with ADHD still pursuing further education at the time of clinical evaluation. They may be asked to make recommendations concerning the need for and types of accommodations these adults are likely to require in those settings. In so doing, clinicians need to familiarize themselves with the standards of evidence required under the Americans with Disabilities Act for obtaining such accommodations (see Chapter 33).
- ✓ Clinicians may also be asked to evaluate workplace impairments and the types of accommodations that may be needed to deal with these impairments. When clinicians are untrained or uncomfortable in doing so, they should refer their patients to other professionals who specialize in vocational assessment, accommodations, and rehabilitation for the expertise that may be required to address the workplace difficulties of adults with ADHD. Here, again, familiarity with the appropriate aspects of the Americans with Disabilities Act will be required to obtain such accommodations (see Chapter 33).
- ✓ The pervasive adverse impact of ADHD in the workplace also indicates that long-acting ADHD medications will likely help adults with ADHD, much as they have done for the educational functioning of children with ADHD, and they may be even more useful given the longer hours adults spend in their jobs than they likely spent in school settings as children. In fact, long-acting medications may even need to be supplemented further with immediate-release medications to provide the additional hours of coverage these adults are likely to require beyond what was necessary to cover a child's school day. Less likely to be feasible

or adopted in employment settings are the behavioral interventions that have proven so useful in educational settings with children with ADHD; medication is therefore a more convenient and effective intervention component for adults with the disorder. Workplace accommodations may offer some additional benefits beyond medication for adults with ADHD, but no research is available to demonstrate their efficacy.

- ✓ Both children with ADHD as adults and clinically diagnosed adults with ADHD have more negative communication styles and display less positivity during conflict resolution discussions. They also report less satisfaction in their intimate, cohabiting, or marital relationships than do those without ADHD. The partners of these adults are also more likely to report that the relationship is less satisfying, particularly if the unaffected partner is male. Some recent evidence suggests that ADHD in adults may be associated with an increased risk of verbal aggression and violence in intimate domestic relationships apart from any risks posed by comorbid ASPD and mood or anxiety disorders.
- ✓ Evidence is mixed as to whether adult ADHD is associated with a higher frequency of divorce, but this may be partly due to the age of the sample. In contrast, children with ADHD followed to midlife do experience higher divorce rates that were not evident at earlier follow-ups.
- ✓ Clinicians need to be prepared to identify regional resources that assist adults with ADHD and their partners in addressing these intimate relationship problems apart from applying traditional ADHD interventions.
- ✓ Detailed research now documents that both children growing up with ADHD followed to ages 27–36 years and adults with ADHD diagnosed in adulthood experience a diversity of financial management problems, impulsive buying, excessive use of credit, and lower credit ratings and higher financial stress than comparison cases. These financial problems are largely due to the severity of ADHD itself, with symptoms of CD by adolescence making a smaller contribution to these adult financial problems.
- ✓ Excessive or pathological gambling may not to be a problem for adults with ADHD or children grown up with the disorder, although research on the issue is scant and results are mixed. That risk seems mostly to be the result of any comorbidity with ASPD or CD but possibly may be linked to impulsivity.
- ✓ Again, clinicians need to be cognizant of regional resources that can be brought to bear to assist adults with ADHD who have financial (and related legal) difficulties, beyond just applying traditional ADHD interventions for symptom reduction and management.
- ✓ The economic costs of ADHD calculated to date for lost workplace productivity, annual income, social assistance, educational failure, crime, and driving risks, not to mention the health care costs linked to the disorder apart from costs of treatment, are substantial. They far outweigh the costs that would be associated with intervention to treat this disorder effectively.

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CHAPTER 13

Comorbid Psychiatric Disorders and Psychological Maladjustment in Adults with ADHD

Russell A. Barkley

As discussed in earlier chapters, children and adolescents diagnosed with attention-deficit/hyperactivity disorder (ADHD), especially if they are clinic-referred, have a high likelihood of having a second or even third psychiatric disorder besides ADHD. So do adults given a clinical diagnosis of ADHD (Marks, Newcorn, & Halperin, 2001). This chapter is an updated version of my earlier review of this topic in our book on ADHD in adults (Barkley, Murphy, & Fischer, 2008). Although the pattern of comorbidity in adults with ADHD is generally consistent with that seen in children followed to adulthood, there are some important differences that will be noted here. In general, 84% of children with ADHD that persists to adulthood have at least one other disorder; 61% have two disorders, and 45% have three comorbid conditions (Barkley et al., 2008). For clinic-referred adults with ADHD, these figures are 80, 66, and 20%, respectively. Adults with ADHD were also more likely to have at least three or more disorders (39%) compared to the clinical (20%) and community (4%) control groups (Barkley et al., 2008). Sobanski and colleagues (2008) likewise found that 76–87% of adults with ADHD (depending on subtype) had a lifetime occurrence of another psychiatric disorder. Similarly high rates of comorbidity were found by McGough and associates (2005) among parents of children with ADHD. They assessed psychiatric comorbidity in the parents of children with ADHD who were participating in a study of the genetics of the disorder. McGough and colleagues evaluated 435 parents who completed rating scales and structured diagnostic interviews. Parents who also had ADHD were more likely to have experienced psychopathology over their lifetime; 87% (vs. 64% of parents without ADHD) had at least one other disorder, and 56% (vs. 27% of parents without ADHD) had at least two other disorders. Specifically, ADHD was associated with higher rates of disruptive behavior disorders, substance use, and mood and anxiety disorders. Males were more likely to have exhibited disruptive behavior disorders, while female sex and oppositional defiant disorder (ODD) increased the risk of anxiety and depression.

EXTERNALIZING DISORDERS

ODD and Conduct Disorder

As in childhood ADHD, children with ADHD followed to adulthood have a considerable risk for comorbid ODD and conduct disorder (CD). For instance, in

the Milwaukee Study, Barkley and colleagues (2008) found that by age 27, approximately 47% of children whose ADHD persisted to that age had ODD compared to 16% of those with nonpersistent ADHD and 4% of control children followed to adulthood.

This is also true for clinic-referred adults with ADHD compared to both clinical control groups without a diagnosis of ADHD and normally developing, non-referred adults. For example, Miller, Nigg, and Faraone (2007) found that 27–31% of adults with ADHD had one externalizing disorder, and 27–41% had two or more compared to 28 and 14%, respectively, of control adults. Approximately 24–50% of adults with ADHD had current ODD, and 7–36% either had CD currently or at some time over the course of their earlier development (Barkley et al., 2008; Barkley, Murphy, & Kwasnik, 1996; Biederman et al., 1993; Cumyn, French, & Hechtman, 2009; Harpold et al., 2007; Murphy & Barkley, 1996; Murphy, Barkley, & Bush, 2002, Spencer, 2004; Wilens et al., 2009). Although these figures are below those reported in studies of children with ADHD, the rates for adults with ADHD may still be double or triple the rates reported for control adults (Barkley, Fischer, Edelbrock, & Smallish, 1990; Fischer, Barkley, Smallish, & Fletcher, 2002; Weiss & Hechtman, 1993).

Epidemiological studies also indicate an association between ADHD and these other disruptive disorders (Kessler et al., 2006; Secnik, Swensen, & Lage, 2005) as did the study of nonreferred adult relatives with children with ADHD reported by McGough and colleagues (2005). Among parents of children with ADHD who also meet criteria for ADHD, disruptive behavior disorders also occur significantly more often (Minde et al., 2003). For instance, Biederman and colleagues (1993) found that 53% of these parents have had ODD and 33% have had CD sometime in their lives, figures that are closer to those seen in follow-up studies of children with hyperactivity or ADHD.

Adults with ADHD who had ODD sometime in their childhood were at significantly greater risk of having bipolar disorder, multiple anxiety disorders, and substance use disorders than adults with ADHD without such a history of childhood ODD (Harpold et al., 2007). In a community sample, they were also at greater risk for more severe psychiatric symptoms, except for those involving dysthymia, generalized anxiety disorder, social phobia, and somatization, than were adults with ADHD alone or ODD alone. However, in a clinical sample, the comorbid group had more severe symptoms of antisocial personality, borderline personality, CD, mania, and schizophrenia (Gadow et al., 2007), with both disorders acting synergistically on the latter disorders when comorbid. The comorbid group was also likely to be younger, less likely to be employed, and less likely to be married. Likewise, adults with ADHD and comorbid ODD also manifest a distinct set of personality traits compared to those with ADHD alone; they are more likely to show histrionic, narcissistic, aggressive–sadistic and negativistic personality traits (May & Bos, 2000). All of this suggests that comorbid ADHD and ODD in adults, like its childhood counterpart, comprises a far more severe impairment constellation than is seen in either disorder alone.

Personality Disorders

Antisocial personality disorder (ASPD) is often an associated adult outcome in a significant minority of those children or adolescents who have both ADHD and CD. For instance, in the Milwaukee follow-up study (Barkley et al., 2008), 39% of children in whom ADHD persisted to adulthood had ASPD compared to 16% with nonpersistent ADHD and 4% of control children. It should therefore not be surprising to find that 7–44% of clinic-referred adults diagnosed with ADHD also qualify for a diagnosis of this personality disorder, typically between 25 and 30% (Biederman et al., 1993; Shekim, Asarnow, Hess, Zaucha, & Wheeler, 1990; Torgersen, Gjervab, & Rasmussen, 2006). Even among those who do not qualify for this diagnosis, many receive higher than normal ratings on personality traits associated with this personality disorder (Tzelepis, Schubiner, & Warbasse, 1995). A study of a large general population sample likewise found an association between ADHD in adults and ASPD (Kessler et al., 2006). Not surprisingly, then, a recent review of the literature involving 18 prospective studies documented a link between ADHD (with or without CD) and greater risk for later ASPD, a link also documented in 13 cross-sectional or retrospective studies (Storebø & Simonsen, in press). When the two disorders are comorbid, there is a greater incidence of violence and repetition of violence that may also be further exacerbated by comorbidity of ASPD and substance use disorders (González, Kallis, & Coid, 2013).

Although the issue has not been as extensively studied as ASPD, other personality disorders (PDs) are

also more common in children with ADHD that persists to adulthood (66%) in comparison to those with nonpersistent ADHD (28%) or control children (12%) (Barkley et al., 2008). For instance, in that study, a sizable minority of children with persistent ADHD had passive–aggressive (33%) or borderline PD (30%) compared to the nonpersistent group (19 and 13%) and the control group (3 and 0%).

There has been much less research on the occurrence of PDs other than ASPD in clinic-referred adults with ADHD. Miller and colleagues (2007) did find that ADHD in adults is a significant predictor of risk for having either a Cluster B or Cluster C PD. Whereas just 1–9% of adults with ADHD had a Cluster A disorder (compared to 3% of controls), 22–47% had a Cluster B disorder (vs. 9% of controls), and 16–23% had a Cluster C disorder (vs. 4% of controls). Consistent with these findings, Cumyn and colleagues (2009) found high rates of PDs (51%) compared to a clinical control group of adults (38%), as did Williams and colleagues (2010), who found that 45% of adults with ADHD had a PD (9% Cluster A, 17% Cluster B, and 28% Cluster C).

With regard to the comorbidity of adult ADHD and borderline PD, some results suggest that it is the overlap of ADHD and borderline PD that creates the impulsivity associated with this PD, along with a higher risk for substance use disorders and ASPD (Ferrer et al., 2010). In contrast, those adults who have borderline PD without ADHD seem to be at a greater risk for mood and anxiety disorders (Ferrer et al., 2010).

Substance Use Disorders

As noted in Chapters 5 and 11, substance dependence and abuse are known to occur to a more frequent degree among children with hyperactivity or ADHD followed to adolescence or adulthood, especially among those who had ODD in childhood and developed CD by adolescence or ASPD by adulthood (Barkley, 2006; Barkley et al., 2008; Harpold et al., 2007; Tercyak, Peshkin, Walker, & Stein, 2002). For instance, the Milwaukee Study (Barkley et al., 2008) revealed that children with ADHD that persisted to adulthood were significantly more likely to have a current substance use disorder (SUD) but not necessarily a past SUD (25% currently, 54% past) than children whose ADHD did not persist (10% currently, 53% past) or the control group (13% currently, 40% past).

Studies have found lifetime rates of alcohol dependence or abuse disorders ranging between 13 and 53% in adults diagnosed with ADHD, whereas 8–59% may manifest some other form of substance dependence or abuse disorder (Barkley et al., 1996, 2008; Biederman, 2004; Biederman et al., 1993, 1995; Duran, Fistikci, Keyvan, Bilici, & Caliskan, 2013; Minde et al., 2003; Murphy & Barkley, 1996; Murphy et al., 2002; Roy-Byrne et al., 1997; Shekim et al., 1990; Sobanski et al., 2008; Wilens, 2004; Wilens et al., 2009). Two large epidemiological studies (Kessler et al., 2006; Secnik et al., 2005) likewise found a link between alcohol use disorders and ADHD.

Tzelepsis and colleagues (1995) reported that of their 114 adults with ADHD, 36% had experienced alcohol dependence or abuse, 21% for cannabis, 11% for cocaine or other stimulants, and 5% for polydrug dependence. Moreover, at the point of their initial evaluation, 13% met criteria for alcohol dependence or abuse within the past month. Likewise, Torgersen and colleagues (2006) found that 45% of their sample of 45 adults with ADHD in Norway had lifetime alcohol abuse (33% currently), 51% for cannabis (36% currently), 49% for amphetamines (33% currently), and 16% for opiates (4% currently). Parents of children with ADHD who themselves have ADHD have also been found to have elevated risks for SUDS, primarily involving alcohol (McGough et al., 2005; Minde et al., 2003). Even so, the risk of SUDS in adults with ADHD, as in children with ADHD, may be mediated mainly by earlier or comorbid ODD, CD, or ASPD (Barkley et al., 2008; McGough et al., 2005; Wilens, 2004). The interaction of ADHD with ASPD and SUDS, as noted earlier, comprises a set of major predictors for violence and repetition of violence (González et al., 2013).

In summary, there is a clear clustering of risks for disruptive, antisocial, and drug use disorders with ADHD in adults, as was evident in Chapter 5 for children and adolescents with ADHD, especially as those youth enter young adulthood. While the elevated risks exist in the minority of cases, and is especially evident in those who had conduct problems as children or among their family members, the proportion is not inconsiderable. It poses a substantial economic cost to society, averaging $40,000 per youth with ADHD and CD by adolescence (Jones, Foster, & Conduct Problems Prevention Research Group, 2009) and by adulthood the cost for antisocial activities alone ranges between $2 and $4 billion U.S. dollars (Fletcher & Wolfe, 2009).

INTERNALIZING DISORDERS

In general, it appears that 32–41% of adults with ADHD may have one internalizing disorder, and 25–29% may have at least two or more (Miller et al., 2007). Below I discuss the risk for specific internalizing disorders.

Anxiety Disorders

As noted in Chapter 5, perhaps 25% of children with ADHD have an anxiety disorder (see Tannock, 2000). This was also the case in some adulthood follow-up studies of children whose ADHD had persisted to adulthood. The group with persistent ADHD had a greater risk for generalized anxiety disorder than did the nonpersistent ADHD group (16 vs. 1%) or controls (3%). Overall, the persistent ADHD group had twice the rate of risk for any anxiety disorders (46%) than that for children whose ADHD had not persisted (23%) or the community control group (9%) (Barkley et al., 2008). Posttraumatic stress disorder (PTSD) was also more common in the persistent than in the nonpersistent ADHD group (18 vs. 6%) or the control group (1%) in that same follow-up study. These results, however, do not agree with prior follow-up studies of children with ADHD into adulthood, where no such elevated risk of anxiety disorders or PTSD was evident (Mannuzza, Gittelman-Klein, Bessler, Malloy, & LaPadula, 1993; Mannuzza, Klein, Bessler, Malloy, & LaPadula, 1998; Rasmussen & Gilberg, 2001; Weiss & Hechtman, 1993). Why this should be so is not immediately obvious. But some research on adults with PTSD does imply that ADHD may be a risk factor for developing PTSD in male veterans (Adler, Kunz, Chua, Rotrosen, & Resnick, 2004).

Many studies of clinic-referred adults diagnosed with ADHD find an overrepresentation of anxiety disorders. The corresponding figure among adults is 16–43% for generalized anxiety disorder and 52% for a history of overanxious disorder (Barkley et al., 1996, 2008; Biederman et al., 1993; Duran et al., 2013; Minde et al., 2003; Shekim et al., 1990) with 37–43% having had any anxiety disorder compared to 11–26% of controls (Michielsen et al., 2013; Sobanski et al., 2008; Wilens et al., 2009). Torgersen and colleagues (2006) found that 13% of their adults with ADHD had lifetime panic disorder, and 18% had lifetime social phobia. Moreover, some recent research suggests that the risk for comorbid anxiety symptoms increases with age in adults with ADHD (Michielsen et al., 2013) and may be a function, in part, of comorbidity with depression (Fischer et al., 2007) and a childhood history of ODD (Harpold et al., 2007).

But not all studies of ADHD in adults have found it to be associated with anxiety disorders. Several of our own prior studies (Murphy & Barkley, 1996; Murphy et al., 2002) did not find that anxiety is overrepresented in those clinical samples of adults with ADHD compared to either community or clinical control groups (Barkley et al., 2008). Neither did Roy-Byrne and colleagues (1997) in comparison to a clinical control group. And although Cumyn and colleagues (2009) found a significantly higher rate of anxiety disorders in their adults with ADHD versus clinical control adults, they nevertheless reported relatively low rates in both groups (6–11% of adults with ADHD and 1–4% of controls).

Despite conflicting results for clinic-referred adults, the weight of the evidence leans toward such an association of risk. Moreover, the link between ADHD and anxiety disorders has been reported in two large epidemiological studies of adults (Kessler et al., 2006; Secnik et al., 2005). The prevalence of anxiety disorders among adults with ADHD who are relatives of clinically diagnosed children with ADHD is 20%, again suggesting some comorbidity with ADHD (Biederman et al., 1993). Parents of children with ADHD who themselves have ADHD likewise have significantly more anxiety disorders than do those parents in a control group (McGough et al., 2005; Minde et al., 2003). And college students with ADHD reported higher ratings of anxiety symptoms than do control students (Prevatt, Dehili, Taylor, & Marshall, in press). In conclusion, although there is some inconsistency concerning the comorbidity of ADHD in adults with anxiety disorders, the weight of the evidence favors some association, as it does in childhood ADHD (Angold, Costello, & Erkanli, 1999).

Research primarily with children with both ADHD and anxiety, as discussed in the Chapter 5, suggests that the comorbid conditions may result in reduced impulsivity and possibly more impaired working memory, and may manifest different symptoms of anxiety than anxiety disorders or phobias seen alone (see also Schatz & Rostain, 2006). No research could be located on these issues in adults with ADHD.

Mood Disorders

Major depression does seem to have some inherent affinity with ADHD in children, especially those hav-

ing CD (Angold et al., 1999; Spencer et al., 2000). Yet some follow-up studies have not been able to document an increased risk for depression or dysthymia among hyperactive children followed to adulthood (Klein et al., 2012; Mannuzza et al., 1998; Weiss & Hechtman, 1993). My own Milwaukee follow-up study of a large sample of hyperactive children found a prevalence of 28% for major depression by young adulthood (age 21)—a finding quite consistent with the studies on clinic-referred adults diagnosed with ADHD. But this excess comorbidity for depression or dysthymia did not persist to the age 27 follow-up (Barkley et al., 2008). However, at that follow-up, children whose ADHD had persisted to that age were more likely to have at least one mood disorder (13%) or depressive personality disorder (15%) than those whose ADHD had not persisted (3 and 5%) or the community control group (4 and 0%).

In contrast, the relationship seems to be stronger or more evident in clinic-referred adults diagnosed with ADHD. Approximately 13–45% of adults meeting ADHD diagnostic criteria also have concurrent major depressive disorder (Barkley et al., 1996, 2008; Biederman et al., 1993; Cumyn et al., 2009; Duran et al., 2013; Fischer et al., 2007; Michielsen et al., 2013; Roy-Byrne et al., 1997; Tzelepis et al., 1995), and 36–71% may experience it over their lifetime (Barkley et al., 2008; Sobanski et al., 2008; Spencer et al., 2000). And the risk for depressive symptoms appears to increase with age in adults with ADHD (Michielsen et al., 2013). However, in studies that used a clinical control group, this rate of depression did not differ even if the rate was higher than that in a community sample (Barkley et al., 2008; Murphy & Barkley, 1996; Roy-Byrne et al., 1997). One study of Norwegian adults with ADHD reported a lifetime prevalence of 53% and current prevalence of 9% for major depression (Torgersen et al., 2006).

Dysthymia, a milder form of depression, has been reported to occur in 19–37% of clinic-referred adults diagnosed with ADHD (Barkley et al., 2008; Murphy et al., 2002; Roy-Byrne et al., 1997; Shekim et al., 1990; Tzelepis et al., 1995). This rate is greater than that seen in a clinical control group, at least for current depression (27 vs. 16%, respectively) if not for lifetime occurrence (Barkley et al., 2008). Rucklidge and Kaplan (1997), in one of the few studies of women with ADHD, found that they reported more symptoms of depression, anxiety, stress, and low self-esteem, and a more external locus of control than did women in the control group. But psychiatric diagnoses were not reported in this study, which makes it difficult to compare this to earlier research using such diagnoses. In a study of parents of children with ADHD who also have ADHD, Minde and colleagues (2003) did not find a greater prevalence of major depression relative to a control group of parents (15 vs. 8%). The study, however, used small samples, limiting its representation of parents with ADHD and its statistical power to detect group differences. It also did not find elevated rates of ASPD, which, as discussed earlier, might be a potential moderator between ADHD and depression. In contrast, in the much larger study of a group of parents with ADHD who also had children with ADHD, McGough and colleagues (2005) did find more mood disorders than in their comparison group.

In general, the weight of the evidence suggests a low but significant relationship between ADHD in adults and risk for depression, or at the very least dysthymia. The fact that relatives of children with ADHD who themselves have ADHD also have elevated rates of depression does suggest some familial/genetic association between the disorders, as does the literature on comorbidity between the disorders in epidemiological samples of children (Angold et al., 1999) and adults (Kessler et al., 2006).

Some recent research has examined possible mediators of the connection between ADHD symptoms and those of depression. For example, Meinzer, Petit, Leventhal, and Hill (2012) found that the degree of hedonic responsiveness is a direct mediator between severity of ADHD inattentive (but not hyperactive–impulsive [HI]) symptoms and those of depression. "Hedonic responsivity" refers to the degree of reactivity to pleasurable or rewarding stimuli and is a heritable difference among individuals. The failure to respond to rewarding stimuli has previously been linked to the degree of anhedonic symptoms in depression and, in this study, to the degree of inattention in ADHD adults. Seasonality may be another risk factor, in that adults with ADHD have significantly higher rates of seasonal affective disorder (Rybak, McNeely, Mackenzie, Jain, & Levitan, 2007), which, according to one study, occurs in as many as 27% of adults with ADHD and in significantly more women than men (Amons, Kooij, Haffmans, Hoffman, & Hoencamp, 2013). A third mediator between adult ADHD and depression may be the degree of deficits in executive functioning (EF), particularly as assessed by ratings of EF in daily life rather than neuropsycho-

logical EF tests (Knouse, Barkley , & Murphy, 2012). Poor time management, poor self-organization, and poor problem solving in particular showed the most robust relationships between adult ADHD and depression. These various mediators, along with the degree of stress and occupational, educational, social, financial, and other impairments adults with ADHD are likely to experience, may act to increase the risk for depression, along with the increased family/genetic loading for depression among those clinically diagnosed with ADHD (Faraone & Biederman, 1997).

Noteworthy to recall, as discussed in Chapter 11, is that depression and ADHD may be a potent combination for increasing the risk of suicide attempts in not only children but also adults.

The relationship between ADHD in adults and *bipolar disorder* (BPD) is less well established. Follow-up studies of children with ADHD into adulthood typically do not report elevated rates of this disorder by adult outcome (Barkley et al., 2008; Barkley, Fischer, Smallish, & Fletcher, 2002; Mannuzza et al., 1993, 1998; Weiss & Hechtman, 1993). This was also the case in the large epidemiological study by Kessler and colleagues (2006) and in the Norwegian sample studied by Torgersen and colleagues (2006), in which the prevalence was 7% for lifetime disorder and 2% currently. These figures are comparable to what is often found in follow-up studies of children with hyperactivity and ADHD into adulthood (Barkley et al., 2008; Fischer et al., 2002) and are close to the base rate for the general population. Conflicting with these results are those studies by Biederman (2004), who reported an elevated risk for this disorder in clinic-referred adults (11–14%). Secnik and colleagues (2005) also reported a small but elevated risk for comorbidity of ADHD and BPD (4.5 vs. 0.6%). Even if adults with ADHD do not qualify for a diagnosis of BPD, they may show elevated levels of symptoms of bipolar spectrum disorder (51%; Halmoy et al., 2010). The relationship of ADHD to BPD in adults is therefore open to some doubt; most studies do not find this association. Clearly the issue is in need of more research before we can be confident of any directionality of this pattern of comorbidity between ADHD and BPD.

However, the reverse direction is far less in doubt. There is a very high occurrence of ADHD in samples diagnosed with BPD that is related to age of onset; it can run as high as 38–98% of child and adolescent BPD cases and 9–35% of adult cases (Klassen, Katzman, & Chokka, 2010). The coexistence of these two disorders in adulthood is associated with a more severe course of disorder, more severe mood symptoms, and lower psychosocial functioning (Klassen et al., 2010). Childhood or current ADHD may also be associated with an earlier onset of first affective episode, more frequent occurrence of such episodes, and more interpersonal violence than is seen in adults with BPD without ADHD (Ryden al., 2009).

Obsessive–Compulsive Disorders

Obsessive–compulsive disorder (OCD) has not been shown to be overrepresented in children with ADHD followed to adulthood (Barkley et al., 2008; Mannuzza et al., 1993, 1998; Weiss & Hechtman, 1993). One study initially reported the occurrence of OCD in 14% of clinically diagnosed adults with ADHD (Shekim et al., 1990). In a later study in Turkey, Duran and colleagues (2013) also found a higher than expected prevalence of OCD in adult outpatients with ADHD (17%). However, Tzelepis and colleagues (1995) were unable to replicate this finding and reported that only 4% of their adults met diagnostic criteria for OCD. Roy-Byrne and colleagues (1997) likewise reported a 4.3–6.5% prevalence rate, which was not significantly different from that of their clinical control group. In a large sample of clinic-referred adults, we found no higher incidence of OCD relative to a community group, but did find it to a small degree in a clinical control group (Barkley et al., 2008). Spencer (1997) found that OCD was more common (12%) only among those adults with a comorbid tic disorder, whereas the figure for adults with ADHD without tics was approximately 2%. Thus, OCD does not appear to be significantly associated with ADHD in clinic-referred adults unless tic disorders or Tourette syndrome are also present.

To summarize, past research suggests a higher than expected association between ADHD in adults and comorbid ODD, CD, ASPD, SUDs, and probably depressive disorders (major depression and dysthymia). The link between ADHD and SUDs is likely mediated by the association of ADHD with CD or ASPD. So may be the link between ADHD and major depression. The relationship between adult ADHD and adult anxiety disorders is inconsistent in past research. The link between ADHD and BPD is even less well established, especially for adults with ADHD. There seems to be no elevated risk for OCD among adults with ADHD.

PSYCHOLOGICAL MALADJUSTMENT

The previously discussed research has approached the subject of comorbidity with ADHD from the psychiatric, categorical view of disorders. Another approach is the psychological, dimensional view that examines differences between groups on more continuously scaled measures of these same domains of psychological maladjustment. At least five prior studies have taken this approach to evaluating their clinic-referred adults with ADHD using variations of the Symptom Checklist–90—Revised (SCL-90-R; Derogatis, 1986). The study by Shekim and colleagues (1990) also reported results for this instrument, but not in comparison to any clinical or community control group. They did find that patients with ADHD and panic disorder have significantly higher scores on many of the scales of this instrument than did those without panic disorder. My colleagues and I conducted four of these five studies (Barkley et al., 1996, 2008; Murphy et al., 1996, 2002) and found that clinic-referred adults with ADHD had significantly greater elevations on most if not all scales of the SCL-90-R relative to either clinical control or community control groups. The exception is the study in which Roy-Byrne and colleagues (1997) compared individuals with probable ADHD and those with possible ADHD and no ADHD, all of whom had been seen at a psychiatric clinic. No differences were found among these groups on any of the SCL-90-R scales. Likewise, we have found children whose ADHD persisted to adulthood to be significantly more maladjusted on all of these scales than either children whose ADHD did not persist or a control group (Barkley et al., 2008). Thus, the previous study by Roy-Byrne and colleagues is truly the exception to the rule: Adults with ADHD do manifest significantly more psychological maladjustment on most dimensions of such maladjustment than do either community control adults or clinic-referred adults without ADHD seen at the same clinic.

Using a different set of instruments, Ramirez and colleagues (1997) found that adults with high levels of ADHD symptom express more anger, and in more dysfunctional ways, and are more labile in anxious/depressed moods than those with lower symptoms levels. Higher elevations on all scales of psychopathology were also evident on the Young Adult Behavior Checklist (Achenbach, 2001; for both self- and other-report forms) in our study of clinic-referred adults with ADHD (Barkley et al., 2008). In these findings, displayed in Table 13.1, one can also see that adults referred to the same clinic who did not meet DSM-IV criteria for ADHD, although they had significantly elevated symptoms of the disorder, were significantly more maladjusted than control adults on all scales. Similar to the findings of Ramirez and colleagues, noted earlier, the scales on which members of the ADHD group displayed even more maladjustment than the clinical control group included Anxiety–Depression, Attention Problems, Intrusive, Aggressive, and Delinquent on the self-report version and just the Aggressive scale on the other-report form.

Taken together, these results indicate that adults with ADHD are more psychologically maladjusted than adults without diagnosable ADHD seen at the same clinic or community adults. They are most likely to differ from adults with other psychiatric disorders on scales evaluating externalizing psychopathology, such as Aggression, Antisocial Behavior, and Disruptive or Intrusive Behavior, and to a lesser extent on Anxiety–Depression. Given the variety of comorbid disorders associated with ADHD in adults documented earlier, this pattern is not unexpected.

INTELLECTUAL DISABILITY

There is very little research on comorbidity of intellectual disability (ID) and ADHD. Perhaps this is because most researchers who study ADHD in adults select samples with at least low-average or higher IQ. As indicated in Chapter 4, children with ADHD typically average 7-10 points less in their intelligence quotients (IQs) than do control groups of typically developing children (Frazier, Demareen, & Youngstrom, 2004). Although the correlations between ADHD severity and IQ in these studies and those of general population samples are rather small ($r < .30$), most are significant, implying some relationship between these two variables that share 5–9% of their variance (see Barkley, 1997, for a discussion; also Simonoff, Pickles, Wood, Gringras, & Chadwick, 2007). The detrimental relationship of ADHD to IQ can emerge as early as the preschool years (Friedman-Weieneth, Harvey, Youngwirth, & Goldstein, 2007; Loe et al., 2008). All this seems to imply that children with the disorder would have a higher percentage of cases qualifying as ID than would control cases, since the ADHD distribution of IQ would be shifted about one-half a standard devia-

TABLE 13.1. Young Adult Self-Report and Other-Report Form Scales for the Adult Behavior Checklist by Group (*T*-Scores) in the UMASS Study

	(1) ADHD		(2) Clinical		(3) Community				Pairwise
Measure	Mean	*SD*	Mean	*SD*	Mean	*SD*	*F*	*p*	contrasts
Self-report scales									
Anxiety–Depression[S]	64.8	10.5	61.8	12.3	51.2	3.0	58.3	<.001	1 > 2 > 3
Withdrawn	60.2	8.4	58.8	8.7	52.3	5.2	27.0	<.001	1, 2 > 3
Somatic Complaints	59.8	8.3	57.9	8.5	51.6	4.1	29.4	<.001	1, 2 > 3
Thought Problems[A]	58.4	9.4	56.3	8.3	50.6	2.4	22.0	<.001	1, 2 > 3
Attention Problems[S, G×S]	69.1	8.8	65.0	9.4	50.7	2.3	152.5	<.001	1 > 2 > 3
Intrusive[A]	58.4	8.2	56.2	7.3	50.7	2.1	28.9	<.001	1 > 2 > 3
Aggressive[A]	61.3	8.9	56.5	8.5	50.9	2.5	45.9	<.001	1 > 2 > 3
Delinquent[A,S]	59.6	8.5	57.3	7.8	51.5	3.3	30.5	<.001	1 > 2 > 3
Other-report scales									
Anxiety-Depression[S]	66.6	10.5	65.7	9.8	51.2	2.7	45.3	<.001	1, 2 > 3
Withdrawn[A]	58.8	8.3	61.6	10.3	51.1	2.9	18.0	<.001	1, 2 > 3
Somatic Complaints	59.8	8.1	58.1	6.7	52.0	4.5	18.2	<.001	1, 2 > 3
Thought Problems[A]	59.4	8.6	58.1	8.4	50.7	2.8	21.6	<.001	1, 2 > 3
Attention Problems[A]	67.4	9.4	65.9	9.1	50.9	2.6	54.0	<.001	1, 2 > 3
Intrusive[A]	59.2	8.2	56.3	10.0	50.8	2.4	13.2	<.001	1, 2 > 3
Aggressive[A, G×S]	62.4	7.6	58.7	6.3	50.6	1.7	48.7	<.001	1 > 2 > 3
Delinquent[A]	59.5	7.6	57.9	7.8	51.5	3.5	14.1	<.001	1, 2 > 3

Note. Sample sizes for the self-report scales are ADHD = 120, clinical control = 75, and community control = 83. For the other-report scales, they are ADHD = 76, clinical control = 38, and community control = 45.

SD, standard deviation; *F, F*-test results of the analysis of variance (or covariance); *p,* probability value for the *F*-test; NS, not significant, [S], significant main effect for sex (see text for details), [G×S], significant group × sex interaction (see text for details); [A], age used as a covariate in this analysis.

Statistical analyses: Groups were initially compared using two-way (groups × sex) analysis of variance (or covariance as necessary). Where this analysis was significant ($p < .05$) for the main effect for group, pairwise comparisons of the groups were conducted, the results of which are shown in the last column.

tion to the left of that for typical children. Children with ADHD followed to adulthood likewise are typically found to have slightly but significantly lower IQ scores than controls (Barkley et al., 2008) and could therefore be expected to have a higher than usual percentage of individuals with ID, though few researchers have actually computed the figure from their results.

Study results are mixed on whether ADHD in clinic-referred adults is associated with lower IQ, however. Intelligence estimates for such adults fell in the normal range and were comparable to those of control groups of clinic-referred adults in several of my own prior studies (Barkley et al., 1996, 2008; Murphy & Barkley, 1996; Murphy et al., 2002). In contrast, Biederman and colleagues (1993) found that their adults diagnosed with ADHD had IQ scores significantly below those of their control groups. Yet the IQ scores for the adults with ADHD were 107–110, nearly identical to the results of our own studies of adults with ADHD. The adults with ADHD in the Biederman and colleagues study therefore seem to differ significantly from the control groups only by virtue of the fact that the control group had above-average IQs (110–113).

Among adults with borderline to mild ID, one study indicated that IQ was negatively affected by ADHD severity to a low but significant degree (Xenitidis, Paliokosta, Rose, Maltezos, & Bramham, 2010). Similarly, two separate meta-analyses examining IQ in adults

with ADHD indicated mild reductions in IQ associated with the disorder (effect size of about 0.25) (Bridgett & Walker, 2006; Hervey, Epstein, & Curry, 2004), with one review concluding that the differences were not of much clinical significance and likely arose due to comorbidity rather than to ADHD itself (Bridgett & Walker, 2006). In the other review, Hervey and colleagues (2004) implied that such mild deficits may be linked to the few subtests of IQ batteries that evaluate working memory, as well as perceptual–motor speed, both of which are often moderately or more impaired in adults having the disorder. Excepting these subtests, adult ADHD may not be associated with lower intelligence, as indexed by other subtests. In general, then, it seems reasonable to conclude that at least clinic-referred adults with ADHD are of average IQ, probably do not differ from typical adults to an appreciable extent, and therefore likely do not have a higher percentage of ID than the adult population. This is not the case for children growing into adulthood with ADHD, in whom deficits in IQ are greater and so the likelihood of a higher percentage placing in the ID range of IQ would be expected.

What of the inverse relationship of ADHD among individuals with ID? Studies of children and teens routinely reveal that those with ID are 2.5–4.0 times more likely to have ADHD than controls without ID, or about 14–40%, and that ADHD is the most common comorbidity found in cases of ID (Baker, Neece, Fenning, Crnic, & Blacher, 2010; Neece, Baker, Crnic, & Blacher, 2013). Studies of adults with ID suggest that approximately 20% may qualify for a diagnosis of ADHD based on high levels of symptoms on an ADHD screening scale (La Malfa, Lassi, Bertelli, Pallanti, & Albertini, 2008); this is likewise about four or five times greater than the prevalence in general population samples. Among individuals with comorbid ID, ADHD symptoms are often more severe and appear to cohere with each other in ways not seen in individuals with ADHD only, they are less likely to remit with age (Xenitidis et al., 2010), and they may be linked to higher rates of aggressive behavior (Cooper et al., 2008). Objective measures of attention and inhibition likewise show adults with comorbid ADHD + ID to have more severe deficits, even after researchers control for general IQ, than do adults with ADHD only (Rose, Bramham, Young, Paliokosta, & Xenitidis, 2009).

Thus, the comorbidity of ADHD and ID is largely a one-way risk. Whereas having adult ADHD does not appear to increase the risk for ID, though a very minor detrimental effect on IQ may be evident in large samples, having ID results in a substantially elevated risk of having ADHD. The coexistence of both disorders is a harbinger for more severe and persistent ADHD and increased aggression.

KEY CLINICAL POINTS

- ✓ In general, there appears to be convincing evidence that ADHD increases the liability for certain other psychiatric disorders. More than 80% of members of ADHD groups had at least one other disorder, more than 50% had two other disorders, and more than 33% had at least three other disorders—all of which occurred more frequently than in control groups.
- ✓ There is a markedly elevated risk for ODD, and to a lesser extent for CD, in clinic-referred adults with ADHD and in adults who as children had ADHD.
- ✓ Adults with ADHD show a greater risk for alcohol use disorders than do clinic-referred or control adults and also a greater risk for cannabis use disorders compared to community controls. Results suggest that alcohol use disorders and risk for any drug use disorder may be specifically linked to ADHD, though the level and type of drug use disorders probably have more to do with comorbid ODD, CD, and ASPD, as well as local access to specific drugs, than to ADHD per se.
- ✓ The internalizing disorders of major depressive disorder, dysthymia, and anxiety disorders are more likely to occur in individuals with ADHD referred to clinics than in a community control group. But major depressive disorder and anxiety disorders are also significantly elevated in clinical controls without ADHD seen at the same ADHD clinic and therefore may not be linked as specifically to ADHD as to general outpatient psychopathology. Even so, epidemiological studies in both children (Angold et al., 1999) and adults (Kessler et al., 2006) find some association between ADHD and depression, which makes it unlikely that findings of a limited association are purely due to referral bias.
- ✓ It seems to be dysthymia or depressive personality disorder that is most convincingly elevated in individuals with ADHD beyond that risk seen in clinical control groups.
- ✓ BPD and OCD symptoms are not significantly elevated in the ADHD groups and are therefore not generally comorbid with ADHD.

- ✓ In a dimensional examination of comorbidity using the SCL-90-R, adults with ADHD (whether clinic-referred adults or children who have grown up) showed elevations on all scales of psychological maladjustment relative to community controls and on most of the scales relative to clinical control groups. There is clearly greater maladjustment of all types associated with ADHD than in clinical or community comparison groups. Such findings imply that ADHD is a more severe psychological disorder than many outpatient disorders seen in the same clinics.
- ✓ IQ may be somewhat lower, if at all, in adults with ADHD, and this difference may be due in part to subtests of IQ batteries that evaluate working memory and perceptual–motor speed, both of which are typically deficient in individuals with ADHD. It is therefore unclear whether ADHD is associated with mild intellectual delay, independent of these subtests.
- ✓ Clinicians need to be aware and to assess specifically for the high comorbidity of ADHD and other psychiatric disorders, particularly dysthymia, depression, ODD, CD, alcohol use disorders, and drug use disorders more generally.
- ✓ Such comorbid disorders and psychological problems are highly likely to require separate treatment approaches than those likely to be aimed at the management of ADHD symptoms and their related impairments.
- ✓ ADHD in adults, particularly when seen in clinic-referred adults, is therefore likely to require polypharmacy more than is the case for childhood ADHD given the higher risk for comorbid mood and anxiety disorders than is seen in children. While ADHD drugs, such as stimulants and nonstimulant norepinephrine reuptake inhibitors, are clearly indicated for such cases, they are unlikely to address the risk for mood disorders evident here, and likely require separate medical (i.e., antidepressant, mood stabilizing) and psychological (i.e., cognitive-behavioral) treatments in their own right.
- ✓ The elevated risk for anxiety disorders in both clinic-referred adults with ADHD and children with ADHD that persists to adulthood also suggests (1) that the nonstimulant, atomoxetine, may be of some benefit for these comorbid cases given that it does not exacerbate anxiety and may reduce it to some extent, and (2) that cognitive-behavioral interventions having utility in management of anxiety disorders generally may be of some benefit for this comorbid population.
- ✓ Drug detoxification and rehabilitation programs will also be required for that subset of individuals with comorbid ADHD and drug use disorders, many of whom are also likely to have ASPD or a history of CD. Early and aggressive treatment of the ADHD seen in these comorbid cases at initial entry into detox or rehabilitation programs offers the best chance of assisting these individuals with their rehabilitation efforts. Ignoring it is highly likely to result in recurrent treatment failures due to the significant self-regulation and executive deficits identified with this disorder.

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CHAPTER 14

Etiologies of ADHD

Russell A. Barkley

Since the previous edition of this volume, great strides have been made in understanding the etiologies of ADHD; indeed, this may be the area that has advanced most in research in the interim, more than any other topic on ADHD. Certainly it is among the most important topics because an understanding of etiologies may lead to improvement in treatments of the disorder and possibly someday even its prevention in some cases. Despite some inconsistencies across studies, laboratories, samples, and measures that will always be part of scientific research into mental disorders, important conclusions can be drawn about the causes of ADHD from the extant and voluminous research. There is no doubt now among investigators that ADHD is a "neurodevelopmental" disorder and has even been classified as such in the most recent edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-5; American Psychiatric Association, 2013; also see Chapter 2). Although, clearly, there are multiple etiologies that contribute to attention-deficit/hyperactivity disorder (ADHD), evidence points to primarily genetic and neurological factors and their interactions with the environment, particularly biologically influential features of it (e.g., toxins and infections), as the greatest contributors to the occurrence of the disorder.

Our knowledge of the final common neurological pathways through which these factors produce their effects on cognition and behavior has advanced remarkably in the past decade. Converging lines of evidence come from multiple studies using functional and structural neuroimaging research, such as positron emission tomography (PET), magnetic resonance imaging (MRI), and functional MRI (fMRI), along with more recent means of analyzing their results to obtain incredibly detailed portraits of neuronal networks and brain microstructure. So numerous are these studies that meta-analyses of their findings have become routinely published reviews in this literature every few years. While abnormalities at the neurochemical or cellular level that may underlie this disorder have still proven difficult to document, even this area of research suggests that more is involved in this disorder than just disturbances in one or two neurotransmitter activities, such as dopamine or norepinephrine, and that developmental processes involved in cell migration, termination, and support may also be implicated. Remarkably, studies have begun to combine the findings from molecular genetics with those from neuroimaging and neurophysiology to allow us to better understand the impact of variations in gene structure (polymorphisms and copy number variants, both common and rare) on the development, structure, and functioning of various brain networks that are implicated in the disorder. Yet further evidence also indicates that some cases of

ADHD may be acquired as a consequence of exposure to various biohazards, especially during fetal brain development and, to a lesser but still significant extent, postnatal development. Still others may arise from interactions between genetically at-risk children and environmental toxins and adversities. In a few cases, ADHD might arise from new genetic mutations that occur in the sperm or fertilized egg in becoming a fetus; thus, a new genetic case of ADHD may arise that was not inherited from parents.

Just as importantly, there is no credible evidence to indicate that the social environment alone can lead to a child developing ADHD. No convincing evidence or even a scientific hypothesis concerning the social causation of ADHD has been advanced during the past decade, and those from earlier decades, such as poor parenting, have not proven to be consistent with the known scientific findings on the disorder. Nor do such simplistic ideas have any explanatory or predictive value in understanding the disorder or driving further scientific research to test it (falsifiability). What was stated in the previous edition and has now become even more abundantly clear, given what is now known, there cannot be such explanations. That is, because numerous studies of twins and families have made it abundantly clear that the majority of variation in behavioral traits that comprise ADHD is the result of genetic and related factors. What little variation remains is best explained by the child's exposure to unique events that affect him or her and not the siblings. These environmental events are likely to be biological hazards such as prematurity, prenatal toxins, prenatal infections, and postnatal events (lead poisoning, traumatic brain injuries, etc.) that disrupt further brain development and even interact with genetic susceptibilities (candidate gene polymorphisms) to the disorder. Some unique psychosocial adversities may likewise interact with genetic susceptibilities toward disorder. Yet we are now able to conclude unequivocally that ADHD cannot and does not arise from purely main effects of social factors, such as childrearing, family conflict, marital difficulties, insecure infant attachment, television or video games, the pace of modern life, or interactions with peers, among other popular notions from earlier epochs. This is not to say that such social factors may not have some influence on current and future functioning in cases of ADHD, or that they may not interact with neurological and genetic susceptibilities to the disorder. Social environments are clearly influential and supported in research based on their impact on risks for subsequent impairments in major life activities; risks for comorbid disorders; and access to diagnostic, treatment, and educational resources, all of which impact current and eventual adult outcomes of the disorder. But the prevailing evidence makes clear that these social factors alone do not create ADHD de novo in an otherwise normal or typical child. Continuing claims to the contrary by lay critics bespeak a stunning ignorance of the prolific literature on the neurological and genetic contributions to the disorder and even the growing evidence of gene × bioenvironment interactions.

So abundant is the literature now on the etiologies of ADHD that only broad highlights can be presented here given the space limitations. But the advances have been most impressive even if the detailed results of individual studies cannot be thoroughly presented here.

GENETIC FACTORS

It is now clear that ADHD is among the most genetically influenced of all psychiatric disorders, rivaled perhaps by bipolar disorder and autistic spectrum disorders, but exceeding the genetic contribution to anxiety and depression, among most other disorders. For the most part, ADHD is not the result of abnormal chromosomal structures, as in Down syndrome; chromosomal fragility (as in fragile X) or transmutations; or extra chromosomal material, as in XXY syndrome. However, children with rare genetic abnormalities may have an increased risk for developing ADHD, such as those with veliocardiofacial syndrome, although they may have a different profile of ADHD symptoms and risk for comorbid psychiatric disorders than do children with idiopathic ADHD (Antshel et al., 2007). Children with fragile X syndrome also may have even higher rates of ADHD inattention symptoms than do control children (93 vs. 38%) (Farzin et al., 2006; Tranfaglia, 2011). Likewise, 24% of children with Turner syndrome have ADHD in comparison to 1.3% in the control children (Russell et al., 2006). While children with such chromosomal abnormalities may have more problems with attention, and even higher than typical rates of ADHD, such genetic abnormalities are very uncommon in children with ADHD. By far, most research evidence suggests that ADHD is highly hereditary in nature, making heredity one of the most substantiated etiologies for ADHD.

Family Aggregation Studies

Multiple lines of research support such a conclusion. For more than 40 years, researchers have noted the significantly higher prevalence of psychopathology generally, and ADHD specifically, in the parents and other relatives of children with ADHD (Cantwell, 1972; Morrison & Stewart, 1971). In particular, higher rates of ADHD, conduct problems, substance abuse, and depression have been repeatedly observed in studies of these families dating back several decades (Barkley, DuPaul, & McMurray, 1990; Biederman et al., 1992). Research indicates that parents of children with ADHD are two to eight times more likely to have the disorder than parents of control children. Between 10 to 35% of the immediate family members of children with ADHD are also likely to have the disorder; the risk to siblings of children with ADHD is approximately 32% (Biederman et al., 1992; Biederman, Keenan, & Faraone, 1990; Faraone et al., 1992; Levy & Hay, 2001; Welner, Welner, Stewart, Palkes, & Wish, 1977). Not only do children of parents with ADHD have a higher risk for ADHD, but both the type and severity of the children's ADHD and other externalizing disorders are significantly correlated with the severity of their parents symptoms and disorders (Bornovalova, Hicks, Iacono, & McGue, 2010; Macek, Gosar, & Tomori, 2012), especially those of the father (Macek et al., 2012), and this tendency is genetically transmitted from parent to child (Bornolova et al., 2010). Higher than expected rates of family aggregation of the disorder have been found in African American families similar to rates reported in families of European American children (Samuel et al., 1999). And these higher rates are equally evident in the families of both girls and boys with ADHD (Faraone et al., 2000; Faraone & Doyle, 2001). Even more striking, research shows that if a parent has ADHD, the risk to the offspring is 57% (Biederman et al., 1995). Further evidence for the familial clustering of ADHD in families of affected children came from a study in which Smalley and associates (2000) identified families having at least two affected children with ADHD (n = 132). They then assessed the 256 parents in these families for various psychiatric disorders and found that 55% of the families had at least one parent with a lifetime diagnosis of ADHD. Thus, ADHD clusters far more than expected, as demonstrated by the higher rate of the disorder among biological relatives of children or adults with the disorder than is the base rate for the typical population, strongly implying a hereditary basis to this condition.

Interestingly, research by Faraone and Biederman (1997) at Massachusetts General Hospital suggests that depression among family members of children with ADHD may be a nonspecific expression of the same genetic contribution that is related to ADHD. This is based on their findings that family members of children with ADHD are at increased risk for major depression, whereas individuals who have major depression have first-degree relatives at increased risk for ADHD.

Some research suggests that ADHD with conduct disorder (CD) may be a distinct familial subtype of ADHD (Faraone, Biederman, Mennin, Russell, & Tsuang, 1998). Using sib-pairs in which both siblings had ADHD, Smalley and colleagues (2000) supported this view; they found that CD significantly clustered among the families of only those sib-pairs with CD. Nadder, Ritter, Silberg, Maes, and Eaves (2002) likewise supported this view by finding that while ADHD and oppositional defiant disorder (ODD)/CD have a shared genetic contribution, there are additional unique genetic contributions to ODD/CD. These conclusions are buttressed by support from twin (Thapar, Harrington, & McGuffin, 2001; Wood, Rijsdijk, Asherson, & Kuntsi, 2009) and molecular genetic (Hamshere et al., 2013) studies. This may also be the case for children with ADHD and bipolar disorder; evidence implies that this comprises a distinct familial subtype (Faraone, Biederman, & Wozniak, 2012). These family studies also indicate that ADHD is independent genetically from anxiety disorders (Biederman, Faraone, Keenan, Steingard, & Tsuang, 1991) and learning disabilities (Del'Homme, Kim, Loo, Yang, & Smalley, 2007; Doyle, Faraone, DuPre, & Biederman, 2001).

Some research has also suggested that girls who manifest ADHD may require a greater genetic loading (higher prevalence of family members) than do males with ADHD in order to express the disorder (Smalley et al., 2000; Faraone & Doyle, 2001). Further research using twins supports this view that females appear to have a higher threshold for expression of the disorder than do males with ADHD (Rhee, Waldman, Hay, & Levy, 1999).

Adoption Research

Adoption studies provide natural experiments concerning the likely hereditary transmission of disorders given that adoptive parents do not share genes with their adopted offspring. More than 40 years ago, both Cantwell (1972) and Morrison and Stewart (1973)

reported higher rates of hyperactivity in the biological parents of hyperactive children than in adoptive parents with such children. Both studies suggest that hyperactive children are more likely to resemble their biological parents than their adoptive parents in their levels of hyperactivity, thus supporting a likely hereditary basis to the disorder. Yet both studies were retrospective, and both failed to study the biological parents of the adopted hyperactive children as a comparison group (Pauls, 1991). Later, Cadoret and Stewart (1991) studied 283 male adoptees and found that if one of the biological parents had been judged delinquent or had an adult criminal conviction, the adopted-away sons had a higher likelihood of having ADHD. This is not surprising given that antisocial behavior is quite high among people with ADHD (see earlier chapters) and that a sizable percentage of adults with antisocial behavior have ADHD. In a later study, van den Oord, Boomsma, and Verhulst (1994), using biologically related and unrelated pairs of international adoptees, identified a strong genetic component (47% of the variance) for the Attention Problems dimension of the Child Behavior Checklist, a rating scale commonly used in research in child psychopathology. This particular scale has a strong association with, but is certainly not equivalent to, a diagnosis of ADHD (Biederman, Milberger, Faraone, Guite, & Warburton, 1994) and is often used in research to select subjects with the disorder. Other researchers compared the rates of ADHD in the first-degree adoptive relatives of 25 adopted children with ADHD, nonadopted children with ADHD and their families, and nonadopted control children without ADHD (Sprich, Biederman, Crawford, Mundy, & Faraone, 2000). They found that just 6% of the relatives of the adopted children with ADHD had ADHD—a figure very close to the prevalence of ADHD in adults in the population, suggesting that the children's ADHD did not arise from family environmental transmission. However, 18% of the relatives of the nonadopted children with ADHD were diagnosed with ADHD, compared to 3% for the control group. Thus, like the family association studies discussed earlier, results of adoption studies point to a strong possibility of a significant hereditary contribution to hyperactivity and ADHD.

Twin Studies

Studies of twins provide yet another avenue of evidence for a genetic contribution to ADHD. The evidence for the remarkably high heritability of ADHD traits is even more substantial in scope now than was the case in the prior edition of this volume, and it is now overwhelming. Relative to other psychological traits, the genetic contribution to individual differences (human variation) in ADHD symptoms is striking in magnitude and accounts for the majority of variance. As long as 40 years ago, studies demonstrated a greater agreement (concordance) for symptoms of hyperactivity and inattention between monozygotic (MZ) compared to dizygotic twins (DZ) (Goodman & Stevenson, 1989; O'Connor, Foch, Sherry, & Plomin, 1980; Willerman, 1973). Numerous large-scale twin studies were subsequently conducted. They were reviewed in a meta-analysis of 79 twin and adoption studies. Nikolas and Burt (2010) found that the majority of variance in the traits of ADHD are a result of genetic factors (averaging 71–73% for ADHD inattention and hyperactive-impulsive symptoms, respectively; ranging from 55 to 98%). Other reviews involving many studies find a heritability exceeding .8 (Boomsma, Cacioppo, Muthen, Asparouhov, & Clark, 2007; Grant et al., 2007). The genetic contribution may increase, the more extreme the scores along this trait happen to be, although this latter point is debatable. Thus, twin studies indicate that heritability explains the majority of variation in ADHD symptoms and can be even higher than 70–80% when clinical diagnostic criteria serve as the basis for determining ADHD (Rhee et al., 1999). Different genetic pathways may affect the two ADHD symptom dimensions differently (Kuntsi et al., 2014). Dominant genetic effects are larger for the inattention symptoms, whereas additive genetic effects make a greater contribution to the hyperactive–impulsive symptoms (Nikolas & Burt, 2010). This research adds substantially more evidence to that already found in family and adoption studies supporting a strong genetic basis to ADHD and its behavioral symptoms.

But twin studies can also tell us as much about certain general environmental contributions as they do about genetic factors affecting the expression of a trait, as was noted nearly 20 years ago (Faraone, 1996; Pike & Plomin, 1996; Plomin, 1995). Across the twin studies conducted to date, the results have been reasonably consistent in demonstrating that the shared environment contributes negligible, if any, individual variation in the traits underlying ADHD, and accounts for a range of 0–13% of the variance among individuals and is not statistically significant (see meta-analysis by Nikolas & Burt, 2010; also see Burt, Larsson, Lich-

tenstein, & Klump, 2012). Similar findings were noted earlier for other forms of child psychopathology (Pike & Plomin, 1996). Such shared environmental factors may include social class and family educational/occupational status, the general home environment, parental and child-rearing characteristics that are common or shared across children in the family, and other such nongenetic factors that were common to the twins under investigation in these studies. In their totality, such shared environmental factors on average seem to account for just 0–5% of individual differences in the behavioral trait(s) related to ADHD and are effectively zero (Burt et al., 2012). It is for this reason that I give little attention here to a discussion of purely social factors involved in the causation of ADHD. The numerous twin studies have not been able to verify that such common environmental factors contribute much of significance to individual differences in symptoms of ADHD.

The twin studies cited earlier also indicate the extent to which individual differences in ADHD symptoms are the result of nonshared (unique) environmental factors. Such factors affect the proband child but not the siblings. They include not only those factors typically thought to involve the social environment but also all biological factors that are nongenetic in origin and have the potential differentially to affect the child but not others in the family. Besides biological hazards or neurologically injurious events that may befall only one member of a twin pair, the nonshared environment also includes differences in the manner in which parents may have treated each child. Parents do not perfectly interact with all their children in an identical fashion, and such unique parent–child interactions conceivably might contribute more to individual differences among siblings than do factors related to the home and childrearing that are common to all children in the family. Yet, as noted earlier, one also has to consider biological hazards, for which there is ample evidence of involvement in ADHD (see below). Twin studies to date have suggested that approximately 9–20% of the variance in hyperactive–impulsive and inattentive behavior or ADHD symptoms can be attributed to such nonshared environmental (nongenetic) factors (see the meta-analysis by Nikolas and Burt, 2010). Research suggests that the nonshared environmental factors also contribute disproportionately more to individual differences in other forms of child psychopathology than do factors in the shared environment (Pike & Plomin, 1996). Thus, if researchers are interested in identifying environmental contributors to ADHD, then these twin studies suggest that such research should focus on biological, interactional, and social experiences that are specific and unique to the individual with ADHD and not part of the common environment to which other siblings have been exposed.

Molecular Genetic Research

The remarkably high heritability of ADHD would certainly encourage investigators to search for the specific gene variants that convey the risk for the disorder and account for the variation in symptoms in the population. This has certainly been the case since publication of the previous edition of this book. Quantitative genetic analyses of large samples of families show that a single gene does not account for the expression of the disorder (Faraone et al., 1992). Instead, like all complex traits, ADHD is polygenic. Several forms of gene variants have been investigated: microsatellite polymorphism (MSP), variable number of tandem repeat (VNTR) polymorphisms, single-nucleotide polymorphisms (SNPs), and copy number variants (CNVs). Faraone (personal communication, October 15, 2013) makes the point that distinguishing SNPs from CNVs is important to understanding this field of ADHD research. He describes SNPs as single base pair changes in the DNA sequence. CNVs are much larger deletions and duplications that remove or add segments of genes. They are typically rare, occurring in less than 5% of the population (and often much rarer); they usually change the function of a gene and therefore have etiological significance. While SNPs usually have no functional significance, studies that find them may indicate that functional gene variants contributing to ADHD will be nearby on the chromosome.

Research initially focused on dopamine regulating genes (*DAT1, DRD4, DRD5, DBH,* etc.) given the positive response of ADHD cases to dopamine agonists and reuptake inhibitors, as well as the large role of dopamine in the striatum and frontal cortex—two regions implicated in ADHD. But more recent research has used genomewide scans to identify multiple genetic sites in the genome that appear to be linked to variation in ADHD traits. While some of the potential gene variants identified are related to dopamine and norepinephrine function in the brain, other genes are related to brain growth, migration of cells, endpoint terminations, and cell support, among other important factors in brain development. So prolific has been the field of

molecular genetics of ADHD in the past decade that numerous reviews (Faraone & Mick, 2010; Franke et al., 2011) and meta-analyses now exist (Gizer, Ficks, & Waldman, 2009). Across multiple studies, at least eight gene variants have been identified as reliably linked to ADHD. These include such genes as the serotonin transporter gene (*5HTT*), the dopamine transporter gene (*DAT1*), the D4 dopamine receptor gene (*DRD4*), the D5 dopamine receptor gene (*DRD5*), the serotonin 1B receptor gene (*HTR1B*), and a gene coding for a synaptic vesicle regulating protein known as *SNAP25*. The contribution of each gene to trait variation is fairly small, such that having just one of these variants might elevate risk for the disorder by less than 50%. Results to date for the various linkage studies suggest that gene variants that have large effects on risk for the disorder probably do not exist (see Zhou et al., 2008). The results of studies to date suggest that the following genes may hold promise as contributors to variation in ADHD traits, but that they likely act in concert with each other to explain perhaps 20–30% of the variation in the disorder (Smoller et al., 2013; Yang et al., 2013): *SNAP25, DRD4, SLC6A3, HTR1B, SLC6A4, DBH, NR4A2, PER2, SLC6A1, DRD3, SLC9A9, HES1, ADRA2C, ADRB2, ADRA1B, DRD1, HTR1E, DDC, ST'X1A, ADRA1A, NFIL3, ADRA2A, ADRB1, SLC18A2, TPH1, BDNF, FADS1, FADS2, ADRBK1, ARRB1, DRD2, HTR3B, TPH2, SYT1, HTR2A, SLC6A2, ARRB2, PER1, PNMT, CHRNA4, COMT, ADRBK2, CSNK1E, MAOA, MAOB,* and *HTR2C* (Brookes et al., 2006). Genomewide association (GWA) scans require thousands of patients and family members to identify genes of significance, and this threshold, even when studies are combined, has not yet been adequately reached. But early results hold promise that sufficiently large samples will soon reveal the nature of the genes contributing to the disorder. For instance, some GWA studies identified additional candidates that warrant follow-up, including one that is under a genomewide significant linkage peak in a meta-analysis, *CDH13* (Lasky-Su et al., 2008). This gene is expressed in nicotinic receptors and neurite outgrowth (Canino & Alegria, 2008). As of this writing, an international consortium has assembled 15,000 children with ADHD and control children for an analysis of nonsynonymous (functional) variants, most of which are relatively rare in the population. Sequencing is just getting under way as of 2014 but is expected to identify some rare causal variants, similar to what is already going on in autism research (J. Nigg, & Barkley, 2014).

Rather than look for single genes, researchers are now looking for sets of genes that contribute to various functional networks or pathways known to exist in the brain. For instance, Poelmans, Pauls, Buitelaar, and Franke (2011) identified a coherent network related to nicotinic receptors (already one of the biochemical theories of ADHD) and to neural growth (relevant to newer theories of neurodevelopmental delay). Additionally, Stergiakouli and colleagues (2012) identified metabolic systems related to central nervous system (CNS) development and choleresterol metabolism (essential for neural development) as likely promising in ADHD research. Such efforts provoke new ideas about pathophysiology of ADHD. It is likely that more gene-pathway-based approaches will be fruitful in the future.

The most recent studies in this field suggest that rare CNVs may also contribute to risk for the disorder, and that ADHD cases are likely to involve more such rare CNVs than are found in general population samples (Elia et al., 2009, 2012; Lionel et al., 2011, Williams et al., 2010, 2012; Yang et al., 2013). This evidence of an increased burden of rare CNVs in ADHD implies that one genetic cause of ADHD is the accumulation of rare, large, deleterious deletions and duplications across the genome. For example, one initial study of this method found evidence of a rare CNV at a locus related to ADHD on chromosome 15 at q13.3 in a little under 1% of the population that doubles the risk of ADHD (Williams et al., 2012). Another study using a similar approach concluded that the *PARK2* gene (a gene associated with Alzheimer's disease) has a rare variant occurring in less than 1% of the population that is overrepresented in ADHD (Jarick et al., 2014). More studies of this type are likely to emerge.

Gene by Environment Interactions

As Nigg and I recently summarized (Nigg & Barkley, 2014), in the past decade, studies of gene by environment (G × E) effects have become the norm in psychiatric research. Most of these studies examine one or two selected genetic markers (candidates) in relation to selected measures of the environment. The hazards in such studies are many. In particular, (1) the environmental measure may itself be influenced by variation in unmeasured genes, and (2) if variables are not properly scaled, artifactual or "false-positive" effects are easily found. Nonetheless, initial efforts in this area have been interesting. A recent meta-analysis (Nigg, Nikolas, & Burt, 2010) indicated reliable and consistent in-

teractions of psychosocial distress measures and genotype, particularly for dopamine transporter (*DAT1*) and serotonin transporter, in predicting ADHD. However, these effects remain reliant on a few small studies and could still be overturned. Yet more work on G × E interaction in ADHD is likely to be of considerable interest in coming years.

Furthermore, recent years have seen exciting developments in "epigenetics," that is, the recognition that experience can alter the genome—and therefore the phenotype—sometimes dramatically. This occurs through methylation (modification of chromatin, the material in which DNA is "housed") to alter gene expression; that is, the expression of much of human variation may depend not only on DNA structure but also the regulatory markings that control whether and how a gene is expressed.

These two insights (the importance of G × E and the importance of epigenetic effects) have sparked a renaissance in studies of environmental contributions to ADHD (as well as several other psychiatric conditions) via interactions with genetic susceptibility to disorder.

Environmental Risks and Triggers

When G × E and epigenetic mechanisms are recognized, many possible environmental contributors to the etiology of ADHD emerge as potentially important. A fruitful way to think about the etiology of ADHD is to consider structural DNA (the part that, as far as we know, cannot be changed except by mutations) as conveying liability or susceptibility to ADHD. Experiences then activate the condition, either by causing direct changes in the brain or physiology, or via epigenetic markings that change gene expression. This model suggests that a given environmental risk will not affect all children: Some are "immune" to the effect, but others will be susceptible and develop ADHD in the presence of this risk.

NEUROLOGICAL FACTORS

A variety of etiologies have been proposed for ADHD. Brain damage was initially proposed as a chief cause of ADHD symptoms (see Chapter 1), resulting from known brain infections, trauma, or other injuries or complications occurring during pregnancy or at the time of delivery. Several studies show that brain damage, particularly hypoxic–anoxic types of insults, are associated with greater attention deficits and hyperactivity (Cruikshank, Eliason, & Merrifield, 1988; O'Dougherty, Nuechterlein, & Drew, 1984). ADHD symptoms also occur more in children with seizure disorders (Hesdorffer, Ludvigsson, Olafsson, Gudmundsson, Kjartansson, & Hauser, 2004; Holdsworth & Whitmore, 1974) and with focal stroke to the putamen (Max et al., 2002). However, most children with ADHD have no history of significant brain injuries, and such injuries are unlikely to account for the majority of children with this condition (Rutter, 1977, 1983).

Throughout the century, investigators have repeatedly noted the similarities between symptoms of ADHD and those produced by *lesions or injuries to the frontal lobes more generally and the prefrontal cortex specifically* (Benton, 1991; Levin, 1938; Mattes, 1980; see Chapter 1). Both children and adults suffering injuries to the prefrontal region have long been known to demonstrate deficits in sustained attention, inhibition, regulation of emotion and motivation, and the capacity to organize behavior across time (Fuster, 1997; Grattan & Eslinger, 1991; Stuss & Benson, 1986) that represent an ADHD-like syndrome sometimes called a "dysexecutive syndrome" or "frontal lobe disorder." The similarity of symptoms to those of ADHD do not seem coincidental given the abundant, reasonably definitive evidence that ADHD, at least in part, arises from structural and functional abnormalities in the frontal lobes and its interconnections with the anterior cingulate, basal ganglia, cerebellum, and anterior aspect of the corpus callosum (splenium), to be discussed more below. The difficulties in the networks implicating the anterior cingulate and basal ganglia would therefore imply some difficulties with emotion regulation given the linkage of these structures to the amygdala specifically and therefore the limbic system more generally.

NEUROPSYCHOLOGICAL EVIDENCE

Much of the neuropsychological evidence pertaining to ADHD has been reviewed in other chapters (Chapters 4, 10, and 15) and is not reiterated here in any detail. Such evidence does provide support, albeit very indirectly, for the neurological basis of ADHD given that such tests are linked to a greater or lesser extent with variation in functioning in certain brain regions. Suffice to say that a large number of studies have used neuropsychological tests of frontal lobe functions, perhaps as many as 500 at last count (see Chapter 15).

Results have often found deficits on tests believed to assess executive functions (EFs), such as response inhibition, response variability, working memory (both verbal and nonverbal, but especially nonverbal), both verbal and particularly nonverbal fluency (generating of novel options), timing-related behavior (especially time reproduction), temporal discounting and, to a lesser extent, planning and problem solving. And so by inference, these results would implicate the structures contributing to these EFs (frontal lobes, basal ganglia, and cerebellum, etc.). As discussed in Chapters 4 and 10, and later in Chapter 16, problematic in these studies is that although they identify significant differences between groups of patients with and without ADHD in their mean scores, at least half or more of these patients were not found to be impaired on the tests, implying that many patients with ADHD do not show a dysexecutive syndrome and may not have problems with functioning in the brain structures underlying these neuropsychological functions. In contrast, rating scales of EF routinely find that the vast majority of children and adults with ADHD place in the impaired range (93rd percentile or greater) on all dimensions of EF in daily life. Such results seem to support the view that ADHD is an EF deficit disorder (EFDD), a type of frontal lobe dysexecutive syndrome. They implicate problems in the functioning of prefrontal regions and their networks to other structures (basal ganglia, cerebellum, anterior cingulate, etc.). Moreover, research shows that not only do siblings of children with ADHD who themselves have ADHD show similar EF deficits but also those siblings of children with ADHD who do not actually manifest ADHD appear to have milder yet significant impairments in these same EFs (Seidman, Biederman, Faraone, Weber, & Ouellette, 1997). Such findings imply a possible genetically linked risk family phenotype for EF deficits in families of children with ADHD, even if symptoms of ADHD are not fully manifest in those family members. Evidence does suggest that the EFs apparent in ADHD arise from the same and substantial shared genetic liability as ADHD symptoms themselves (Coolidge, Thede, & Young, 2000).

NEUROLOGICAL EVIDENCE

Far more direct research evidence implicating brain structures and functions in causing ADHD is now available from various studies of the neurological integrity of patients with ADHD. These findings definitively support the view of ADHD as a neurodevelopmental disorder, as it is now classified in DSM-5 (Chapter 2).

Structural Brain Abnormalities in ADHD

Very early studies of the gross structure of the brain as portrayed by coaxial tomographic (CT) scan did not show differences between children with ADHD and typically developing children (B. A. Shaywitz, Shaywitz, Byrne, Cohen, & Rothman, 1983), but greater brain atrophy was found in adults with ADHD who had a history of substance abuse (Nasrallah et al., 1986). The substance abuse, however, seems more likely to account for these results than does the ADHD. CT methods are now considered relatively crude indices of brain structure and have largely been replaced by the approach to measurement I discuss next.

Nearly 20 years ago, researchers began using MRI to explore structural abnormalities in ADHD, and they were not disappointed. Research from that era routinely indicated differences in the structure (mainly size) of selected brain regions in those with ADHD relative to control groups (Tannock, 1998). These and later studies continued to find significantly smaller anterior right frontal regions, smaller size of the caudate nucleus, possibly reversed asymmetry in the size of the head of the striatum (caudate), and smaller globus pallidus regions in children with ADHD compared to control subjects (Aylward et al., 1996; Castellanos et al., 2002; Filipek et al., 1997). The putamen, however, has not been found to be smaller in children with ADHD (Aylward et al., 1996; Castellanos et al., 1996). Besides reduced size, there is some evidence of reduced neurometabolite activity in the right frontal region (Yeo et al., 2003), and the degree of this activity is associated with the degree of attention problems on a continuous-performance test. The size of the basal ganglia and right frontal lobe has also been shown in other studies to correlate with the degree of impairment in inhibition and attention in children with ADHD (Casey et al., 1997; Semrud-Clikeman et al., 2000). Filipek and colleagues (1997) did find smaller posterior volumes of white matter in both hemispheres in the regions of the parietal and occipital lobes, which might be consistent with the earlier studies showing smaller volumes of the corpus callosum in this same area. Castellanos and colleagues (1996) suggested that such differences in corpus callosum volume, particularly in the posterior regions, may be more related to the learning disabilities often found in a large

minority of children with ADHD than to ADHD itself. Numerous subsequent studies (Castellanos et al., 1996, 2001, 2002; Durston et al., 2004) also indicated smaller cerebellar volume in those with ADHD, especially in a central region known as the vermis. This evidence is consistent with the view that the cerebellum plays a major role in EF and the motor-presetting aspects of sensory perception that derive from planning and other executive actions (Akshoomoff, & Courchesne, 1992; Diamond, 2000; Houk & Wise, 1995), and that these functions may be deficient in children with ADHD.

So abundant now is the evidence from structural neuroimaging that multiple meta-analyses of these studies have been published since 2007. In their initial meta-analysis, Valera, Faraone, Murray, and Seidman (2007) found the largest differences between subjects with ADHD and controls in the cerebellum (especially the posterior inferior region of the vermis), followed by the splenium, right and total cerebral volume, and the right caudate. Bilateral prefrontal gray-matter and right globus pallidus volumes were modestly associated with ADHD, but the results were not statistically significant after researchers corrected for multiple comparisons. Subsequent meta-analyses that included new research since 2007 likewise found similar results. For instance, the article by Ellison-Wright, Ellison-Wright, and Bullmore (2008) reported a decrease in area in the right putamen/globus pallidus region linked to ADHD. This region is known to have sizable projections to prefrontal cortex. A meta-analysis by Nakao, Radua, Rubia, and Mataix-Cols (2011) included 14 separate studies across which findings revealed decreased global gray-matter volumes. There were smaller gray-matter volumes, associated with ADHD, in the caudate nucleus but larger gray-matter volumes in the left posterior cingulate cortex/precuneus. A subsequent meta-analysis by Frodl and Skokauskas (2012) that included 11 studies focusing on brain volume replicated the earlier results regarding reduced volumes in the right globus pallidus and putamen, and bilaterally in the caudate.

One of the more fascinating and revealing studies of neurological involvement in ADHD, initially published in 2006 by Shaw and colleagues, was a multisite, longitudinal study of large samples of children with ADHD and controls who were periodically rescanned over approximately a 10-year period (Shaw et al., 2006, 2007). This first serial developmental neuroimaging study of ADHD was able to compute the degree of delay in brain maturation across various brain areas associated with the disorder. The authors defined "cortical maturation" as that age at which peak cortical thickness was achieved. The median age at which half of the cortical points achieved their peak thickness was strikingly dissimilar between children with ADHD and controls (10.5 vs. 7.5 years, respectively). The prefrontal cortex showed the greatest magnitude of delay in development, as had been suggested in many of the earlier MRI studies noted earlier. Such findings are also highly consistent with the view that ADHD is a disorder of EF given that the prefrontal cortex is the executive brain (see Chapter 16). In a later report from this project, Shaw and colleagues (2011) noted a different relationship between hyperactivity–impulsivity symptoms and a slower rate or delay in the normal process of cortical thinning that is typical in later stages of brain development. The findings were discussed as underscoring a dimensional view of ADHD as the extreme end of the distribution of a normal trait(s) within the population given that thinning is linked to the number of ADHD symptoms, even in the control children. In short, ADHD is linked to a delay in brain maturation.

More recently, development of yet another form of imaging provides a means to analyze brain structure in ADHD, focusing on white-matter tracts that connect cortical and subcortical structures. This is known as diffusion tensor imaging (DTI), which is capable of detecting cumulative axonal membrane circumference, axonal density, and the thickness of the myelin sheath in brain white matter. Various approaches to analyzing fMRI findings in this way have been developed, but all are believed to index the axonal integrity and organization of the white-matter structures (van Ewijk, Heslenfeld, Zwiers, Buitelaar, & Oosterlaan, 2012). Enough studies (at least 15 to date) using this methodology have now been done to warrant meta-analysis of all of their findings. The results of just such a review revealed that white-matter integrity was significantly decreased in participants with ADHD, whether they were children, teens, or adults. The differences between participants with ADHD and control groups are as or more striking than those noted in the MRI studies concerning various gray-matter structures. The findings indicate that white-matter regions and tracts are also implicated in ADHD, such as in the inferior and superior longitudinal fasciculus, anterior corona radiata, corticospinal tract, cingulum, corpus callosum, internal capsule, caudate nucleus, cerebellum, uncinate fasciculus, forceps minor, areas within the basal ganglia, and there are widespread differences in the frontal, temporal, parietal, and occipital lobes. Some have con-

cluded that these results indicate that the myelination of these areas is delayed in children with ADHD (van Ewijk et al., 2012) and may remain maldeveloped in adults. Observing the results of these imaging methods leaves one in awe of not just the advances in modern imaging technology but also the widespread adverse maldevelopment with which ADHD is associated in brain development.

Functional Brain Abnormalities in ADHD

It is true that early studies using *psychophysiological measures* of nervous system (central and autonomic) electrical activity, variously measured (electroencephalograms, galvanic skin responses, heart rate deceleration, etc.), were inconsistent in demonstrating group differences between ADHD and control children. But when differences from normal were found in those studies, they consistently were in the direction of *diminished arousal or reactive arousal* in those with ADHD (see Ferguson & Pappas, 1979; Hastings & Barkley, 1978). More consistent evidence of these arousal disturbances comes from later research (Beauchaine, Katkin, Strassberg, & Snarr, 2001; Borger & van der Meere, 2000).

Far more consistent are the results of *evoked response potential* (ERP) and *quantitative electroencephalographic* (QEEG) measures often taken in conjunction with performance of vigilance tests (El-Sayed, Larsson, Persson, & Rydelius, 2002; Loo & Barkley, 2005; Loo & Makieg, 2012). Although results have varied substantially across these studies, the most consistent pattern for QEEG research is increased slow wave, or theta, activity, particularly in the frontal lobe, and decreased beta activity (Loo & Makieg, 2012). Children with ADHD have been found to have smaller amplitudes in the late positive components of their ERPs. These late components are believed to be a function of the prefrontal regions of the brain, are related to poorer performances on vigilance tests, and are corrected by stimulant medication (Johnstone, Barry, & Anderson, 2001; Pliszka, Liotti, & Woldorff, 2000). The EEG improvements from stimulant medication have been shown to be partly a function of the *DAT1* gene allele, particularly in its 10-repeat form (Loo et al., 2003) that may be overrepresented in ADHD (see earlier section on genetics). Thus, although the evidence is far from conclusive, EEG and ERPs related to sustained attention and inhibition suggest an underresponsiveness of children with ADHD to stimulation that is partially or wholly corrected by stimulant medication and that implicates involvement of the prefrontal cortical regions and related networks.

Numerous studies have now used various methods of functional neuroimaging. One such approach is assessing *cerebral blood flow* using single-photon emission computed tomography (SPECT) in children with ADHD and typically developing children (see Hendren, DeBacker, & Pandina, 2000, for an early review). Studies have consistently shown decreased blood flow to the prefrontal regions (particularly in the right frontal area) and pathways connecting these regions to the limbic system via the striatum, specifically its anterior region known as the caudate, and with the cerebellum. Degree of blood flow in the right frontal region has been correlated with behavioral severity of the disorder and reduced EEG activity, while that in more posterior regions and the cerebellum seems related to degree of motor impairment (Gustafsson, Thernlund, Ryding, Rosen, & Cederblad, 2000). Using this method, accompanied by injections of radiopharmaceutical iodine–123-labeled altropane that selectively binds to the dopamine transporter in the striatum, Dougherty and colleagues (1999) reported that the dopamine transporter was elevated by about 70% in adults with ADHD. More recently, a meta-analysis of nine studies using SPECT and other imaging methods targeting this transporter density indicated that these differences associated with ADHD were significant (Fusar-Poli, Rubia, Rossi, Sartori, & Balottin, 2012) but not of the magnitude initially estimated in the Dougherty and colleagues study. Those differences were closer to a 14% elevated density being linked to ADHD. Blood flow, and therefore brain activity, in these regions appears to be affected by methylphenidate, a stimulant often used to treat ADHD (Langleben et al., 2002).

Another approach to studying brain function that employs *PET* to assess cerebral glucose metabolism has indicated diminished metabolism in adults, particularly in the frontal region (Schweitzer et al., 2000; Zametkin et al., 1990). Using a radioactive tracer that indicates dopamine activity, Ernst and colleagues (1999) were able to show abnormal dopamine activity in the right midbrain region of children with ADHD; severity of symptoms was correlated with the degree of this abnormality. Ernst and colleagues (2003) later studied adults with ADHD during a decision-making task and found them to be less likely to activate the hippocampal and insular regions, and more likely to use the right anterior cingulate than did healthy controls. These demonstrations of an association between the metabolic activity

of certain brain regions and symptoms of ADHD and associated EF deficits are critical to proving a causal connection between findings that pertain to brain activation and the behavior comprising ADHD.

Important in the previously discussed research on brain structure is to show not only group differences in brain structure and development but also that such differences were correlated with functional difficulties in these regions, and that those difficulties are linked to the severity of ADHD symptoms. This makes a more convincing case that the findings on brain structure are actually related to problematic functioning in the brain that gives rise to the ADHD symptoms. Early research using *fMRI* indicated that children with ADHD had more abnormal patterns of activation during attention and inhibition tasks than did typically developing children, particularly in the right prefrontal region, basal ganglia (striatum, globus pallidus, and putamen), and the cerebellum (Rubia et al., 1999; Yeo et al., 2003). Again, the demonstrated linkage of brain structure and function with psychological measures of ADHD symptoms and executive deficits is exceptionally important in such research, permitting causal inferences about the role of these brain abnormalities in the cognitive and behavioral deficits comprising ADHD. In a later study, Durston and colleagues (2004) suggested that the reduced size of the brain (about 3–5%), particularly in the right frontal area, found in ADHD may be evident as well in siblings of children with ADHD who themselves do not have ADHD, which perhaps is consistent with the increased familial risk for the disorder and a spectrum of the phenotype for ADHD within these families. But the reduced volume of the cerebellum was also found to be specific only to the child with ADHD and not the unaffected siblings, implying that this region may be directly related to the pathophysiology of the disorder itself. Later, a meta-analysis of 16 fMRI studies (Dickstein, Bannon, Castellanos, & Milham, 2006) noted results highly consistent with those from the previously discussed structural MRI studies. The authors observed that ADHD involved widespread frontal hypoactivity. Also noteworthy was reduced activity in the basal ganglia and parietal cortices.

A more recent approach to studying brain functioning in ADHD involves *proton magnetic resonance spectroscopy* (MRS). This technique measures neurochemicals associated with neurotransmission in the brain. Perlov and colleagues (2009) published a meta-analysis of 16 studies using MRS to evaluate the anterior cingulate cortex, prefrontal cortex, basal ganglia (mostly striatum) and the frontal–striatal/thalamic–frontal circuits. The most consistent result was that choline compounds, such as the ratio of *N*-acetyl-aspartate and glutamate/glutamine to creatine, were significantly abnormal in the disorder.

Yet another approach to evaluating brain functioning uses more complex methods of analyzing fMRI results to investigate *functional interregional interconnectivity*. Several researchers who have done so found reduced functional connectivity in networks in the frontostriatal, frontoparietal, and frontocerebellar zones (Cubillo & Rubia, 2010; Tian et al., 2006; Zang et al., 2007). There was also evidence implicating the sensorimotor cortex and cerebellum (Tian et al., 2006; Zang et al., 2007). These functional abnormalities have been shown to improve with administration of the ADHD drug, methylphenidate (An et al., 2013).

All of these more recent results, published since the publication of the previous edition of this volume, have proven to be quite consistent with earlier reviews of this literature that reached similar conclusions—that abnormalities in the development and functioning of the frontal–striatal–cerebellar regions underlie ADHD (Arnsten, Steere, & Hunt, 1996; Benton, 1991; Fassbender & Schweitzer, 2006). These regions are shown in Figure 14.1.

Neural Networks Implicated in ADHD

Since publication of the previous edition of this volume, various authors have proposed models of ADHD based on these neurological findings, and others concerning neurochemical pathways and ADHD medication effects, that suggest at least three or four networks are involved in the disorder (Castellanos, Sonuga-Barke, Milham, & Tannock, 2006; Makris et al., 2009; Nigg & Casey, 2005; Sagvolden, Johansen, Aase, & Russell, 2005). For instance, Makris, Biederman, Monuteaux, and Seidman (2009) hypothesize that four networks are involved, and that they vary in their involvement across individuals with ADHD, perhaps accounting for some of the heterogeneity in the symptoms of the disorder across patients. Like the other theorists noted earlier, Makris and colleagues propose that there exists a motor activity–regulation network involving the dorsolateral prefrontal cortex, caudate, dorsal anterior cingulate cortex, cerebellum, and the supplementary motor area that give rise to the hyperactive symptoms of the disorder. The second network, which includes the cerebellar hemisphere, dorsal anterior cingulate cortex, dorsolateral prefrontal cortex, thalamus, and the inferior parietal lobule, and their inter-connections, under-

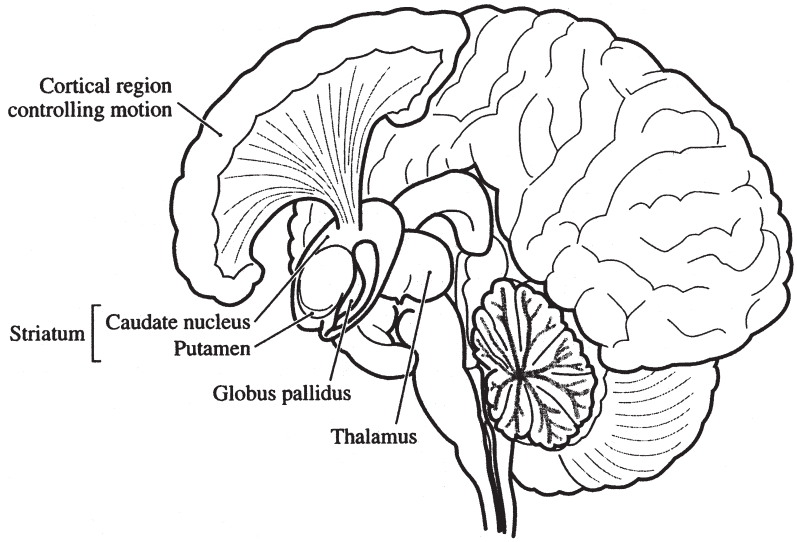

FIGURE 14.1. Diagram of the human brain showing the right hemisphere, and particularly the location of the striatum, globus pallidus, and thalamus. Most of the left hemisphere has been cut away, up to the prefrontal lobes, to reveal the striatum and other midbrain structures. Adapted with permission from an illustration by Carol Donner from page 53 of the article by M. B. H. Youdin and P. Riederer (1997). Understanding Parkinson's disease. *Scientific American*, *276* (January), pp. 52–59. Copyright by *Scientific American*, 415 Madison Avenue, New York, NY 10017-1111.

lies the attention or cold EF (so named for its role in holding information in working memory that guides behavior toward goals) deficits evident in ADHD (e.g., in working memory, planning, and problem solving). Yet a third network, which includes the cerebellar vermis, perigenual anterior cingulate cortex, orbitofrontal cortex, and nucleus accumbens, mediates impulse control, thus explaining the poor inhibition associated with ADHD. The fourth network would explain the deficits in emotional self-regulation evident in the disorder (see Chapter 3; Banaschewski et al., 2012; Barkley, 2010; Biederman et al., 2012b; Kuntsi et al., 2014; Surman et al., 2011) because it involves not only the cerebellar vermis, but also the perigenual anterior cingulate cortex, frontal–orbital cortex, and amygdyla, which can be thought of as the gateway into the emotional brain or limbic system. This is sometimes referred to as the "hot" executive network because of its involvement in emotion, particularly the top-down regulation of affective and motivational states in the service of goal-directed behavior, as well as the bottom-up impact of emotional/motivational states on higher executive functioning (Castellanos et al., 2006; Nigg & Casey, 2005).

Impact of ADHD Medication Treatment on Brain Development

Critics have argued that these differences in brain structure and functioning arise because many of the children in the earliest studies had a history of treatment with stimulant medication, which caused these abnormalities. But many, more recent studies that used treatment-naive patients found identical results, thus refuting the claims of such critics (Frodl & Skokauskas, 2012; Nakao et al., 2011; Paloyelis, Mehta, Kuntsi, & Asherson, 2007). But in point of fact, 29 studies to date have noted that the brain development of children treated with stimulant medications is closer to that of controls who do not have ADHD than to children with ADHD never treated with stimulants (Ivanov, Murrough, Bansal, Hao, & Peterson, 2014; Paloyelis et al., 2007; Sobel et al., 2010). These findings have been observed now in meta-analyses of this body of studies, which makes this a very robust result (Frodl & Skokauskas, 2012; Spencer et al., 2013). Such a result suggests that treating ADHD with medication may result in a greater normalization of brain development,

known as a "neuroprotective effect" (a better term is a "growth-enhancing effect"), than if the medications had never been used. This makes some sense given that clinically effective doses of stimulants increase brain activity in the very regions that have been found to be underactive in the various functional neuroimaging studies discussed earlier (i.e., caudate, prefrontal cortex, cingulate and cerebellum) (Shafritz, Marchione, Gore, Shaywitz, & Shaywitz, 2004; Vaidya et al., 1998). So such a continuous increase in functioning sustained over months or years of development from daily medication use could result in a near-normalization of functioning. In a related study, researchers have found that this effect is also evident on neuropsychological tests of EF in which stimulant-treated children with ADHD have results closer to those of the typically developing control group than to untreated children with the disorder (Semrud-Clikeman, Pliszka, & Liotti, 2008). Although these past studies have been limited to the category of stimulant medications, it is not unreasonable to assume that similar results might arise in response to nonstimulant medications, such as atomoxetine, that overlap in the brain regions they affect by as much as 70% or so with those regions affected by stimulant treatment (Shulz et al., 2012). Granted, these findings cannot be considered definitive evidence of neuroprotection or growth or functional acceleration in the brain development of children with ADHD because children were not randomized to treated and untreated groups, then serially scanned over years of time. But such studies are impossible to do because of ethical, not to mention financial, constraints, so we are left with the evidence that can be obtained from these existing studies that compare treated and untreated cases of ADHD. Undoubtedly, this is one of the most extraordinary findings from neuroimaging studies of the past 15 years, even if it is presently unheralded in the media and runs counter to the prevailing view of those who hold that ADHD medications may be detrimental to brain development and functioning over the course of development.

Neurotransmitter Deficiencies

For many years, possible neurotransmitter dysfunctions or imbalances have been proposed (Arnsten, 2009; Tripp & Wickens, 2008), resting chiefly on the responses of children with ADHD to ADHD medications known to act through dopamine and noradrenergic reuptake inhibitors and agonists (Pliszka, McCracken, & Maas, 1996). Substantial evidence now indicates that three types of medications are effective for managing ADHD symptoms. The first type includes the stimulants, methylphenidate and amphetamine. Both methylpheniate (Volkow et al., 2012) and amphetamine (Riccardi et al., 2008) have been shown through neuroimaging research to increase dopamine (DA) availability (Swanson, Baler, & Volkow, 2011; Volkow et al., 2012), especially in striatum, likely via blockade of the DA transporter (Spencer et al., 2006) that may ultimately increase the rate of DA release. Amphetamine may also increase DA release. Both agents also increase DA and norepinephrine (NE) availability in the hippocampus and prefrontal cortex. Another effective agent for ADHD management is atomoxetine, which is known to block the NE transporter (Takano, Gulyas, Varrone, Maguire, & Halldin, 2009) and is therefore a reuptake inhibitor. More recently, evidence indicates that the ADHD medication guanfacine extended release (XR), originally an antihypertensive medication, acts as an alpha$_2$ receptor agonist serving to more finely tune neural signal transmission (Arnsten, 2009; see Chapters 27 and 35 for more on ADHD medications).

Given the long-standing findings that normal children show a positive, albeit lesser response to stimulants (Rapoport et al., 1978), however, evidence of drug response by itself cannot be used to support a neurochemical abnormality in ADHD. Even so, given that the magnitude of the response to medication is far greater in ADHD than in control cases, some inferences can still be made about the role of neurochemistry and cellular mechanisms in ADHD. Specifically, it is not unreasonable to investigate the involvement of these two neurotransmitters and the DA and NE transporters in the etiology of ADHD.

Early direct evidence from studies of cerebrospinal fluid in children with ADHD and typically developing children, collected before such studies would have failed to pass review by institutional review boards, indicated decreased brain DA in children with ADHD (Halperin et al., 1997; Raskin, Shaywitz, Shaywitz, Anderson, & Cohen, 1984). Evidence from other studies conducted nearly 30 years ago using blood and urinary metabolites of brain neurotransmitters indicates conflicting results (S. E. Shaywitz, Shaywitz, Cohen, & Young, 1983; S. E. Shaywitz et al., 1986; Zametkin & Rapoport, 1986). What limited evidence there was in those early studies seemed to point to a selective deficiency in the availability of both DA and NE, but this evidence was hardly conclusive. But more recent research using

peripheral indicators of catecholamine systems does imply some dysregulation in these neurotransmitters in ADHD (Halperin et al., 1997). Scassellati, Bonvicini, Faraone, and Gennarelli (2012) reviewed 71 studies of such indices and concluded that four of them were reliably implicated in ADHD: increased NE, decreased MHPG (3-methoxy-4-hydroxyphenylethylene glycol), decreased phenlyethylamine, and decreased MAO (monoamine oxidase); the latter was interpreted in this review as possibly impairing the degradation of NE and therefore lowered MHPG levels in individuals with ADHD, which might be a compensatory mechanism for the reduced noradrenergic activity in synapses of patients with the disorder.

Results from numerous animal studies and studies of typically developing humans suggest that these two neurotransmitters may be involved in ADHD, and that it is the cortical–striatal–pallidal system that may mediate ADHD symptoms (Papa, Berger, Sagvolden, Sergeant & Sadile, 1998); increased DA transporter density in the brain may be one cellular mechanism in the disorder (Roessner et al., 2011), as was indicated in the earlier neuroimaging studies using altropane to evaluate transporter density in adults with ADHD. These and other findings led Sagvolden and colleagues (2005) to propose a dynamic neurodevelopmental theory of ADHD (combined and hyperactive types) based on altered DA function that can arise from hypofunctioning in one of three DA branches identified in the brain. Low functioning in a mesolimbic pathway in the brain produces an altered sensitivity to reinforcement and deficient extinction of previously reinforced behavior that could give rise to the delay aversion, hyperactivity, impulsiveness, and poor sustained attention. Low functioning in the mesocortical DA pathway could also give rise to deficient attention toward a target, as well as poor planning and executive functioning. Finally, low functioning in the nigrostriatal DA pathway results in impaired modulation of motor behavior and deficient learning and memory, thereby giving rise to the motor delay, clumsiness, and poor motor inhibition seen in ADHD. Predispositions to low functioning in these DA pathways are hypothesized to interact with each other and with surrounding environmental factors to amplify or alter these initial predispositions. The theory provides a more comprehensive explanation of symptoms and deficits associated with ADHD (see Chapters 2 and 3), while presenting some testable hypotheses concerning which of these should be associated with hypofunctioning in particular pathways. As noted earlier, Makris and colleagues (2009) and others have proposed a similar set of pathways as being involved in ADHD.

PREGNANCY AND BIRTH COMPLICATIONS

As noted in the earlier discussion about genetic contributions to ADHD, genetic factors clearly play a large role in the structural and functional maldevelopment of the brain in ADHD. But a significant minority of cases, perhaps 35%, may develop from adverse environmental factors affecting brain development, resulting in acquired cases of ADHD (Nigg, 2006). One such avenue for environmental adversity to have such an impact is during early fetal brain formation during pregnancy. For more than 50 years, researchers interested in ADHD have examined the extent to which various pregnancy, birth complications, and maternal characteristics during pregnancy may be associated with or cause the disorder. A few early studies did not find a greater incidence of pregnancy or birth complications in children with ADHD compared to typically developing children (Barkley et al., 1990), but others have (e.g., Sprich-Buckminster et al., 1993). For instance, Claycomb, Ryan, Miller, and Schnakenberg-Ott (2004) found that the mother's age at delivery (younger), educational level (lower), time between onset of labor and birth (longer), and presence of delivery complications accounted for 42 percent of the variance in ADHD. The study, however, did not control for maternal ADHD symptoms, which may have resulted in the younger age at delivery and lower educational level of the mothers, given the risk that ADHD poses for earlier risky sexual behavior and limited education (see Chapters 11 and 12). These latter maternal characteristics may simply be markers for maternal ADHD and so explain their being associated with child ADHD.

Some early studies indicate a slightly higher prevalence of unusually short or long labor, fetal distress, low forceps delivery, and toxemia or eclampsia (Hartsough & Lambert, 1985; Minde, Webb, & Sykes, 1968). In keeping with these early findings, a recent very large population-based study found that *in utero* ischemic–hypoxic events, especially birth asphyxia, respiratory distress, and preeclampsia, were independently linked to risk for ADHD (Getahun et al., 2012). But another recent large-scale study of twins did not find pregnancy and delivery factors to be specifically linked to ADHD, with the exception of low birthweight (LBW; Wagner, Schmidt, Lemery-Chalfant, Leavitt, & Goldsmith,

2009). So the evidence for perinatal adversities other than LBW is rather mixed. In fact, in a recent, large study of children with ADHD, Owens and Hinshaw (2013) found that perinatal complications were related to the development of comorbid depression even after they controlled for maternal ADHD, depression, and other factors.

But the evidence for LBW specifically seems quite convincing as a risk factor for ADHD. Early on, Nichols and Chen (1981) found that LBW was associated with an increased risk of hyperactivity, inattention, disruptive behavior, and poor school adjustment. These results for LBW have subsequently been replicated in other studies reviewed over the next 15 years (Breslau et al., 1996; Schothorst & van Engeland, 1996; Sykes et al., 1997; Szatmari, Saigal, Rosenbaum, & Campbell, 1993). And more recently, after controlling for other factors that may be associated with LBW and ADHD (maternal smoking, alcohol use, ADHD, social class, etc.), Mick, Biederman, Faraone, Sayer, and Kleinman (2002) found that LBW was three times more common in children with ADHD than in control children, perhaps accounting for nearly 14% of all ADHD cases. Moreover, twin studies indicate that LBW has a causal relationship with attention problems (Groen-Blokhuis, Middeldorp, van Beijsterveldt, & Boomsma, 2011), and that this relationship to ADHD (and other psychiatric disorders) persists into young adulthood in LBW children (Elgen, Holsten, & Odberg, 2013). It is not merely LBW that seems to pose the risk for symptoms of ADHD or the disorder itself, among other psychiatric disorders, but the extent of white-matter abnormalities due to birth injuries, such as parenchymal lesions, intracerebral bleeding, and/or ventricular enlargement (Whittaker et al., 1997). Although LBW (< 2,500 grams) is a specific risk factor for inattention–hyperactivity and certain learning and motor problems (but not other behavioral or emotional problems) at age 6 (Willcutt, 2012), LBW is itself multiply determined by factors such as maternal health and nutrition, maternal smoking, maternal weight, low socioeconomic status (SES), stress, and other factors, making identification of specific biological mechanisms difficult (Nigg, 2006).

Another pregnancy factor that may be linked to ADHD risk is increased placental size, which may signal the occurrence of disturbances in the maternal environment during pregnancy (Khalife et al., 2013). The placenta may respond to such adversities in the maternal environment with structural and functional changes that can affect the fetus via altered nutrient and hormone supply. Prospective data from this large population-based study in Finland indicated a significant relationship between increased placental size and risk for ADHD, antisocial behavior, and psychopathology more generally, but this risk was limited to boys but not girls.

Among the strongest pregnancy factors associated with risk for ADHD are more chronic influences such as neurotoxins. Among these, the greatest evidence exists for the role of maternal tobacco smoking and, to a lesser extent, alcohol consumption (see Langley, Rice, van den Bree, & Thapar, 2005, for a meta-analysis), both of which can independently increase risk for ADHD about 2.5 times. These are further discussed next in "Environmental Toxins." One study suggests that maternal iodine deficiency may also contribute to risk for ADHD (Vermiglio et al., 2004). Also, other chronic factors, such as maternal toxemia (and also maternal smoking; Fielding, 1985), eclampsia, and genitourinary infections (Mann & McDermott, 2011) have been implicated rather than acute factors, such as traumatic or brief events, such as consuming a single alcoholic beverage or smoking a single cigarette. Another potentially chronic pregnancy factor related to attention and emotional regulation problems may be maternal obesity at the time of conception (Rodriguez, 2010) that may affect prenatal brain development via energy support and disturbed maternal metabolic functioning.

In one very unusual study, Sharp and colleagues (2003) studied possible pregnancy and environmental contributions to ADHD by identifying MZ twins in which only one twin was affected by ADHD. Given the striking genetic contribution to ADHD and the very high concordance rate for it in MZ twin pairs, such a study might find it difficult to find twin pairs in which only one twin was affected. This is precisely the case, in that out of 297 pairs of MZ twins initially screened for the study, just 10 pairs were eventually found in which the twins were discordant for the disorder. Consistent with the authors' hypothesis that the affected twin would be more likely to have birth complications (a nongenetic explanation for the twins' discordance on ADHD), the study found that the affected twin was smaller at birth and more likely to have experienced a breech delivery.

Several studies suggest that mothers of children with ADHD conceive these children at an age younger than that of mothers of control children, and that such pregnancies may have a greater risk of adversity (Claycomb et al., 2004; Denson, Nanson, & McWatters, 1975;

Hartsough & Lambert, 1985; Minde et al., 1968). Because pregnancy complications are more likely to occur in young mothers, mothers of children with ADHD may have a higher risk for complications that may act neurologically to predispose their children toward ADHD. However, the complications that have been noted to date are rather mild and are hardly compelling evidence of pre- or perinatal brain damage as a cause of ADHD. Furthermore, large-scale epidemiological studies have generally not found a strong association between pre- or perinatal adversity and symptoms of ADHD once other factors are taken into account, such as maternal smoking and alcohol use (see later), as well as socioeconomic disadvantage, all of which may predispose children to perinatal adversity and hyperactivity (Goodman & Stevenson, 1989; Nichols & Chen, 1981; Werner et al., 1968).

A few researchers have also examined stress during pregnancy, and their results have been inconclusive. They suggest a modest contribution of stress and anxiety to ADHD symptoms in the offspring of these mothers, but this finding is hardly definitive given the many methodological problems evident to date (for a review, see Linnet et al., 2003). For instance, Van den Bergh and Marcoen (2004) evaluated mothers and their firstborn children, and found that maternal state anxiety during pregnancy explained 22% of the variance in symptoms of ADHD in the offspring, with maternal anxiety during weeks 12–22 being specifically implicated. More recently, Clavarino and colleagues (2010) found that persistent anxiety antenatally in mothers was significantly related to attention problems at ages 5 and 14 years in the follow-up of children born to these mothers. And persistent severe psychosocial stress in mothers has been linked to risk for ADHD independently of other factors that may be related to such distress, such as higher levels of maternal smoking (Motlagh et al., 2011). Such anxiety or stress during pregnancy may result in some programming effect on the fetal brain, perhaps via increased secretion of stress-related hormones into the maternal bloodstream, with concomitant increased exposure for the fetus. Nevertheless, children in most of these studies did not have clinically diagnosed ADHD, and no attempt was made to control for maternal ADHD and its genetic contribution to the level of child ADHD. Anxiety and greater stress is related to ADHD in adults (Chapter 13); thus, it is not far-fetched to think that anxiety in the mothers may have been a proxy for maternal ADHD. Indeed, one recent large-scale study indicated that the relationship of maternal anxiety and depression to offspring ADHD was a genetic one rather than a direct effect causing child ADHD (Van Batenburg-Eddes et al., 2013). Nor was evidence of actual neural programming evaluated in these earlier studies. Therefore, the results remain merely correlational, suggesting some association of maternal anxiety and stress to child ADHD but not clarifying the direction of effect or whether the presence of a third variable, most likely genetic transmission of ADHD risk from the anxious or depressed parent to the child, explains (confounds) these results.

Mick, Biederman, and Faraone (1996) found that the season of a child's birth was significantly associated with risk for ADHD, at least among subgroups that also had learning disability or did not have any psychiatric comorbidity. Birth in September was overrepresented in this subgroup of children with ADHD. The authors conjectured that the season of birth may serve as a proxy for the timing of seasonally mediated viral infections to which these mothers and their fetuses may have been exposed, and that this may account for approximately 10% of cases of ADHD.

ENVIRONMENTAL TOXINS

As the twin and quantitative genetic studies suggest, the environment may play some small role in individual differences in symptoms of ADHD, but these environmental adversities usually involve biological events, not just family/parenting influences or those influences within the social realm. As noted previously, variance in the expression of ADHD that may be a result of environmental sources means all nongenetic sources more generally. These include pre-, peri-, and postnatal complications and malnutrition, diseases, trauma, and other neurologically compromising events that may occur during the development of the nervous system before and after birth. Among these various biologically compromising events, several toxins have been repeatedly linked to risks for inattention and hyperactive behavior.

One such event is exposure postnatally to environmental toxins, and specifically lead. For more than 40 years, *elevated body lead* burden has been shown to have a small but consistent and statistically significant relationship to ADHD symptoms (Nigg et al., 2008; Nigg, Nikolas, Knottnerus, Cavanagh, & Friderici, 2010; Roa et al., 1994). However, even at relatively high levels of lead, less than 38% of children were rated as being hy-

peractive on a teacher rating scale (Needleman et al., 1979), implying that most lead-poisoned children do not develop symptoms of ADHD. And most children with ADHD likewise do not have significantly elevated lead burdens, although one study did indicate that their lead levels may be higher than that in control subjects (Gittelman & Eskinazi, 1983). Researchers who have controlled for the presence of potentially confounding factors in this relationship (as well as type of assay method to detect lead; e.g., hair vs. blood or dentition), as well as a recent meta-analysis (Goodlad, Marcus, & Fulton, 2013), found the association between body lead burden and symptoms of ADHD to be small but still significant (.10–.19) with the more factors controlled, the more likely the relationship falls below .10 This finding suggests that 4% (at best) of the variance in the expression of ADHD symptoms in children with elevated lead is explained by their lead levels. Moreover, one serious methodological issue plagues even the better-conducted studies in this area: None of the studies assessed for the presence of ADHD in the parents and controlled its contribution to the relationship. Given the high heritability of ADHD, this factor alone could attenuate the already small correlation between lead and symptoms of ADHD by as much one-third to one-half its present levels. Interestingly, in a recent large-scale study in Korea, Byun and colleagues (2013) suggested that excess mobile phone use in children with excess body lead burden is linked to severity of ADHD symptoms. Yet the study acknowledges that reverse causality may be at work here, in which children with ADHD are more likely both to experience higher body lead burdens and to use mobile phones more than children without ADHD.

As noted earlier, two other types of prenatal environmental toxins found to have some relationship to inattention and hyperactivity are *prenatal exposure to alcohol and tobacco smoke* (Mick et al., 2002; Milberger, Biederman, Faraone, Chen, & Jones, 1996; Nichols & Chen, 1981; Streissguth, Bookstein, Sampson, & Barr, 1995). The relationship between maternal smoking during pregnancy and ADHD remains significant even after researchers control for symptoms of ADHD in the parent (Mick et al., 2002; Milberger et al., 1996) and shows the strongest association with risk for ADHD. Indeed, in a comprehensive review of the literature on maternal smoking and ADHD, Thakur and colleagues (2012) concluded that such cases represent a more severe form of the disorder than cases of ADHD in which maternal smoking was not implicated. There is some evidence as well that passive secondhand smoke exposure after birth may contribute to disruptive behavior disorders generally (Gatzke-Kopp & Beauchaine, 2007) and risk of having ADHD specifically (Max, Sung, & Shi, 2013). But it may be that the effect of maternal smoking during pregnancy on risk for ADHD is an indirect one, contributing to fetal risk for LBW that is then the more direct contributor to risk for ADHD (Kline, Stein, & Susser, 1989; Thapar et al., 2009). Or it is possible that a G × E interaction is operative here, in which children having risk genes for ADHD are more susceptible during fetal development to the adverse effects of prenatal smoking exposure (Neuman et al., 2007).

Maternal alcohol consumption, at least at moderate levels, may make some contribution to risk for ADHD. The evidence here is less conclusive than that for a relationship between maternal smoking and ADHD (see Linnet et al., 2003, for a review). The relationship of maternal alcohol consumption to ADHD may be due to its link to maternal smoking (Rodriguez et al., 2009): Women who smoke more tend to consume more alcohol during their pregnancies. Even so, other researchers suggest that alcohol consumption may still make a contribution to ADHD risk even if they control for other factors (O'Malley & Nanson, 2002). And recent neuroimaging researchers have suggested that fetal exposure to smoking (Ekblad et al., 2010; de Zeeuw, Zwart, Schrama, van Engeland, & Durston, 2012) and alcohol is associated with reduced brain volume in children with ADHD, especially in the cerebellum, even relative to children with ADHD who have not been exposed (de Zeeuw et al., 2012).

There has been far less research on prenatal cocaine exposure and risk for ADHD than on alcohol or tobacco smoking exposure. It has been suggested that such cocaine use by pregnant women may result in maternal hypertension, decreased uterine blood flow, fetal vasoconstriction and hypoxemia, and nutritional deficiency, among other effects. But some evidence does suggest a dose–response relationship between such maternal cocaine use and risk for a diagnosis of ADHD (Morrow et al., 2009).

Boucher and colleagues (2012) recently observed that placental cord levels of mercury at the time of birth are linked to an elevated level of ADHD symptoms at school age. This suggests yet another possible prenatal environmental toxin that may be implicated in the disorder, although replication of these findings is certainly in order.

Prospective population studies implicate household and outdoor pesticide exposures during critical periods in pregnancy as predictive of ADHD (Goldman et al., 1997; Sagiv et al., 2010; see Nigg, 2006, for a review). In a recent review of the literature, Polanska, Jurewicz, and Hanke (2013) likewise suggested a potential adverse effect of pesticide exposure to risk for ADHD. And a cross-sectional study of a large population sample revealed that children with elevated pesticide metabolite concentrations in urine were nearly twice as likely to have ADHD as were children without such elevated concentrations (Bouchard, Bellinger, Wright, & Weisskopf, 2010). But more research is needed to determine if this is merely a correlate of other causal processes or it is itself a cause of ADHD via neurotoxicity. A recent review of the literature on other such environmental toxins found the greatest evidence for maternal smoking, as discussed earlier. It found far less and inconsistent evidence for chemicals such as phthalates (often found in plastics), bisphenol-A (used in plastics and resins), polycyclic aromatic hydrocarbons (an organic pollutant related to fossil fuels and other carbon-based fuels), and polyfluoroalkyl (used in numerous consumer products, packaging, labeling, coatings, paper, and even personal care products) (Polanska et al., 2013). There is some initial, albeit preliminary, evidence that excess exposure to manganese found in soy-based infant formula may be neurotoxic at high levels and linked to ADHD symptom risk (see review by Crinella, 2012), but far more evidence is required to confirm and understand the nature of this potential relationship.

A crucial challenge in such research is to determine whether such correlates, even though they emerge in prospective population-based studies, are causal. Although G × E interactions, as well as gene–environment correlation can mask environmental effects, they can also mask genetic effects. Teratogens and toxins could be proxies for genetic risk because of gene–environment correlation. This cautionary remark applies to the previously discussed evidence for maternal smoking during pregnancy, secondhand smoke after birth, and even paternal smoking, in which such factors may be proxies for elevated ADHD in the parents that conveys a genetic ADHD risk to offspring (Langley, Heron, Smith, & Thapar, 2012). Moreover, some researchers suggest that such early environmental insults (e.g., pregnancy complications) and toxins may not operate directly on brain maldevelopment but may produce their effects via epigenetic mechanisms that become dysregulated, resulting in altered and abnormal genetic expression (Mill & Petronis, 2008).

Elevated levels of phenylalanine in mothers with PKU (phenylketonuria) have been associated with higher levels of hyperactive–impulsive symptoms in their offspring, whereas when children experience PKU it is more likely to be associated with symptoms of inattention (Antshel & Waisbren, 2003). This study implies that phenylalanine may contribute to some degrees to ADHD in some children, and that the timing of exposure to high levels of phenylalanine affects the two symptom dimensions of ADHD differently.

Other dietary factors that to a small extent may contribute to variation in ADHD traits are discussed in Chapter 25 on dietary treatments.

STREPTOCOCCAL INFECTION

Some earlier researchers suggested that obsessive–compulsive disorder (OCD) and Tourette syndrome may be a consequence of prior exposure to streptococcal infection (Kiessling, Marcotte, & Culpepper, 1993; Singer et al., 1998). In some individuals, such infections may result in autoimmune system antibodies that cross-react with and compromise neural proteins, particularly in the basal ganglia of the brain. Peterson and colleagues (2000) examined 105 individuals with OCD, chronic tic disorder, or ADHD and 37 controls without any disorder. Levels of antistreptococcal antibody titers in blood were measured, as was the integrity of the basal ganglia using MRI. Results indicated that ADHD is significantly related to such antibodies, even after researchers controlled for the effects of OCD and tic disorders, and these antibodies are related to basal ganglia volume. Additional evidence of the possible role of strep infection in ADHD came from the later study in which children with tic disorder, Tourette syndrome, or OCD were more likely to have had a strep infection than control cases, and that these children were also more likely to have ADHD (Leslie et al., 2008). Maddalena and colleagues (in press) found that children with ADHD alone (and no comorbidity) manifested higher antibasal ganglia antibodies and serum antistreptoylsin titers, both of which are indicative of strep infection-induced damage to the basal ganglia, than did control children. These studies suggest that some cases of ADHD may arise from or be exacerbated by streptococcal infection, perhaps via the damaging effects this may have on basal ganglia.

TRAUMATIC BRAIN INJURIES

For many years, dating back to the classic case of Phineas Gage (Harlow, 1848), research on the frontal lobes has indicated that traumatic injuries to the brain and specifically to this region are likely to result in symptoms resembling ADHD, among other disorders. Research in the past decade has shown that children who survive traumatic brain injuries (TBIs) sufficiently to warrant treatment at hospital trauma centers are likely to develop significant attention problems and clinically diagnosed ADHD as a consequence of their TBI. Yeates and colleagues (2005) compared children with severe TBI to those with moderate TBI and a control group with orthopedic injuries on ratings of inattention and neuropsychological tests of attention and EF. They found that the severe TBI group had worse inattention than the moderate TBI group at 4-year follow-up. By that follow-up, 46% of the severe TBI group had significant attention problems compared to 26% of the children with orthopedic injuries. More attention problems were associated with greater EF deficits on testing. Max and colleagues (2005a) found that 16% of children with TBI who had not previously had a diagnosis of ADHD developed ADHD within 6 months of their TBI. Importantly, the development of ADHD was associated significantly with evidence of injury to the orbitofrontal gyrus (Max et al., 2005a). Further follow-up of these children indicated that 15% had developed ADHD between 6 and 12 months of injury, and 21% had developed it in their second year after injury, suggesting a rising risk for ADHD over the 2 years after TBI (Max et al., 2005b). This study reported that secondary ADHD was associated with postmorbid personality changes and new-onset disruptive behavior disorders. In contrast to children developing ADHD within 6 months of TBI, development of ADHD later was predicted by children's lower preinjury adaptive functioning and higher levels of preinjury psychosocial adversity, not by injury location in the brain. In a larger subsequent study, Levin and colleagues (2007) compared samples of children who had preinjury ADHD and those who did not, all of whom had been treated for acute TBI. Among those with no preinjury ADHD, 14.5% had developed ADHD within 6 months and 18.3% by 24 months after TBI. Of the children with preinjury ADHD, 96% had the diagnosis at 6-month follow-up, and 85% at 12-month follow-up. TBI exacerbated the symptoms of ADHD in those who had the disorder preinjury and resulted in a more variable course of ADHD symptoms after injury in those with no history of ADHD before their TBI. Low SES prior to injury was the only predictor of risk for ADHD from TBI at follow-up in the group without preinjury ADHD. Treatment with stimulant medication prior to TBI in children with preinjury ADHD was associated with less exacerbation of their symptoms following TBI. Children who develop secondary ADHD from TBI may have a different profile of inhibitory problems than do children with primary ADHD (not secondary to TBI; Sinopoli, Schachar, & Dennis, 2012). Eme (2012) reviewed this literature, comprising seven separate studies, and concluded that the rate of ADHD secondary to TBI is at least 30% (ranging from 19 to 48%) and that rates may be even higher in those suffering severe TBI because many of these TBI cases were collapsed together regardless of severity. Indeed, ADHD, ODD, and organic personality disorder may be the most common psychiatric diagnoses given to children following TBI. Eme concluded that many more children having TBI are likely to experience emotional dysregulation than is recognized in these rates of formal DSM diagnoses of ADHD that do not incorporate symptoms of emotion regulation problems as part of their criteria (see Chapter 3). All this makes clear that ADHD can be caused by TBI at any stage of child development (and likely across adulthood).

PSYCHOSOCIAL FACTORS

Even the first medical references to an ADHD-like condition in the literature proposed that poor child-rearing and poor teaching, among other social factors, might contribute to ADHD (Crichton, 1798 [also see Palmer & Finger, 2001]; Weikard, 1775 [also see Barkley & Peters, 2012]; see Chapter 1). A few environmental theories of ADHD that were proposed nearly 30 years ago (Block, 1977; Willis & Lovaas, 1977) have neither shown any consistency with the subsequent scientific literature nor received much direct research attention. Willis and Lovaas (1977) claimed that hyperactive behavior is the result of poor stimulus control as a result of weak maternal commands, and that this poor regulation of behavior arises from poor parental management of the children. But if this were the case, there would be a substantial contribution to ADHD from shared or rearing environments in the numerous twin studies conducted to date (discussed earlier), and the exact opposite has been the result. As noted earlier in

"Twin Studies," there is no significant contribution of the rearing or common environment to the behaviors that comprise ADHD.

Others have also conjectured that ADHD results from difficulties in the parents' overstimulating approach to caring for and managing the child, as well as parental psychological problems (Carlson, Jacobvitz, & Sroufe, 1995; Jacobvitz & Sroufe, 1987; Silverman & Ragusa, 1992). But these theories have not clearly articulated just how deficits in behavioral inhibition and other cognitive deficits commonly associated with clinically diagnosed ADHD (Chapters 2, 4, and 10) could arise from such social factors. Moreover, many of these researchers claiming to have evidence that parental characteristics potentially cause ADHD did not use clinical diagnostic criteria to identify their children as having ADHD; instead, they relied merely on elevated parental ratings of hyperactivity or laboratory demonstrations of distractibility to classify the children as ADHD (Carlson et al., 1995; Silverman & Ragusa, 1992). Nor have these theories received much support in the available literature on clinically diagnosed children with ADHD (Danforth, Barkley, & Stokes, 1991; Johnston & Mash, 2001; see Chapter 7). Again, in view of the previously discussed twin studies that show no significant contributions of the common or shared environment to the expression of symptoms of ADHD, theories based entirely on explanations of shared social experiences to explain the origins of ADHD are difficult to take seriously, especially when they do not explain the precise social mechanism by which this effect is supposed to occur. Nor do they involve genetically informed study designs that could easily tease out genetic effects from shared environmental effects. When that is done, the shared environment contributes no significant variance to symptoms of the disorder (see the earlier section on genetics).

Despite the large role heredity seems to play in ADHD symptoms, they may be associated with or even shaped by unique environmental influences, psychosocial adversities, and nonshared social learning. The actual severity of the externalizing symptoms generally, the continuity of those symptoms over development, the types of secondary symptoms, and the outcome of the disorder are related in varying degrees to environmental factors such as psychosocial adversities (parental mental illness, substance abuse, and criminality; family violence, psychical abuse, sexual abuse, and neglect; divorce, and lower SES, among others) (Biederman et al., 1995, 1996; Green et al., 2010; Lingineni et al., 2012; Milberger, Biederman, Faraone, Guite, & Tsuang, 1997; van den Oord & Rowe, 1997; Weiss & Hechtman, 1993). Yet even here care must be taken in interpreting these correlational findings because such adversities are not specific to any single disorder; they are associated with a general risk for psychopathology or disruptive behavior disorders, not ADHD specifically; and they cluster together and are subject to recall bias (Green et al., 2010). Moreover, the direction of causation here is not usually specifiable from the findings (i.e., correlation is not evidence of causation). And some evidence suggests that such childhood adversities are more related to the onset of mood and anxiety disorders, as well as substance use disorders, than to disruptive behavior disorders such as ADHD, yet even here the amount of variance explained is modest (McLaughlin et al., 2010). These studies are also not evidence of a purely social-environmental contribution of childhood adversities to ADHD because many measures of family function and adversity are genetically influenced, which is to say that they also have a strong heritable contribution, largely owing to the presence of similar symptoms and disorders in the parents and the children who share the genes for these disorders and not just their social environments (Pike & Plomin, 1996; Plomin, 1995). Thus, there is a genetic contribution to the family environment, and that genetic contribution may account for the largest degree of risk for ADHD specifically and psychopathology more generally in the children, a fact that often is overlooked in studies of family and social factors involved in ADHD.

Moreover, concerning the role of parenting as a cause in ADHD, as noted in Chapter 7 (see also Danforth et al., 1991), several investigators attempted to evaluate the direction of effects within parent–child interactions. They did so by investigating the effects of stimulant medication and placebo on these mother–child interactions. The studies consistently found that the medications resulted in significant improvements in child hyperactivity and compliance. There was a corresponding reduction in mothers' use of commands, direction, and negative behavior when the children were on medication, indicating that much of the negative behavior of the mothers appeared to be in response to the difficult behavior of these children.

Taken together, these findings suggest that the overly critical, commanding, and negative behavior of mothers of hyperactive children is most likely a reaction to the difficult, disruptive, and noncompliant behavior of these children rather than the cause of it.

And such disrupted parenting is not only likely to be a reaction to the child's behavioral control problems, but it may also arise from the parents' own ADHD and the parents' higher likelihood of experiencing other psychological disorders, such as depression, anxiety, antisocial disorders, and substance dependence/abuse. This is not to say that the manner in which parents attempt to manage their children's ADHD behavior cannot exacerbate it or maintain higher levels of conflict between mother and child over time. Studies have shown that the continuation of hyperactive behavior over development, and especially the maintenance of oppositional behavior in these children, is related in part to parents' use of commands and criticism, and an overcontrolling and intrusive style of management. But all this tells us is that comorbid ODD, when seen in ADHD may, in part, be a function of parental management practices, which is quite consistent with theories of ODD (Barkley, 2013). Yet this does not mean that the child's ADHD is a result of those practices. Indeed, recent twin studies suggest that the high association of ADHD with ODD/CD is likely the result of a shared genetic liability for these two disorders, with ODD/CD also being influenced by additional genetic factors (Nadder et al., 2002). Theories of the causation of ADHD can no longer be based solely or even primarily on social factors, such as parental characteristics, caregiving abilities, child management, or other family environmental factors.

There is no question that parenting is related to risk for ODD and CD (see Barkley, 2013, for a review). And because those disorders are often comorbid with ADHD, researchers may carelessly attribute family, parenting, or social correlates of ADHD as causes of ADHD itself. But in fact, these psychosocial adversities are related to and likely cause the comorbidity of these other disruptive, externalizing disorders that often coexist with ADHD. Researchers who do not take such relationships into account risk misattributing psychosocial risk factors, such as parenting, as correlates or causes of ADHD when they are either the result of ADHD in the child, shared genetic risk with parental mental disorder, or contributors to comorbid disruptive disorders instead.

Block (1977) proposed that an increase in "cultural tempo" in Western civilization may account for the prevalence of hyperactivity in these countries. Precisely what is meant by "cultural tempo" is not operationally defined, nor is evidence presented to suggest that underdeveloped or Eastern cultures have less hyperactivity than do more developed cultures. This theory and its modification by Ross and Ross (1982) remain speculative to this day and seem almost to be scientifically untestable. Moreover, such theories, once again, conflict with a wealth of information on genetics and heritability of this behavior pattern and disorder, and the nonsignificant role of the common environment (of which this tempo would be considered a part) argues against these theories as explanations for the occurrence of most ADHD in children.

One psychosocial factor that periodically receives attention in the popular media as a cause of ADHD is the degree of children's exposure to television or other televised media (video games). Various commentators imply that ADHD, or at least its attention symptoms, could arise from watching too much television in early childhood. And they admonish parents about this detrimental effect and warn them to reduce TV viewing so as to reduce the risk of their child having ADHD. The occasional research study appears to support these critics, but a closer look often indicates that the article overstates the results or, more likely, confuses a correlation with a cause: Children with ADHD watch more TV; therefore, TV causes ADHD

For instance, Christakis, Zimmerman, DiGiuseppe, and McCarty (2004) examined the relationship between hours of television viewed at ages 1 and 3 years, and attention problems at age 7 years, as measured by five items from the Hyperactivity subscale of the Behavior Problems Index. They defined children as having an attention problem if they were 1.2 standard deviations above the mean on this set of five items—a very generous definition of attention problems. Ten percent of their sample was so classified at age 7 years. After statistically controlling for a number of other variables, the authors found that number of hours of television exposure at ages 1 and 3 years was slightly yet significantly associated with being classified as having attention problems at age 7 years. The hypothesis was that early TV viewing shortens children's attention spans because of the unrealistic pace with which TV events unfold relative to real life. The exact mechanism for this causal influence is suggested to be exposure to TV during critical periods of synaptic development in brain neurons. Measures of synaptic development were not taken in the study. While the authors claimed that their findings supported their hypothesis, they did note that their study design permitted no causal inferences from these associations. They then quickly forgot this limitation by stating that they have now added inatten-

tion to the list of detrimental effects of excessive television viewing (such as violent behavior and obesity). They even recommended implementing preventive strategies based on this causal conclusion even going so far as to say that reducing TV exposure might reduce the later risk of developing ADHD.

This is not the only study to find such an association (Landhuis, Poulton, Welch, & Hancox, 2007; Mehmet-Radji, 2004; Miller et al., 2007; Nathanson, Alade, Sharp, Rasmussen, & Christy, in press; Swing, Gentile, Anderson, & Walsh, 2010). But when affirmative results are found, they are small (< 5% of variance explained) even if significant (usually resulting from the use of large sample sizes), and may be found just on teacher-rated inattention, not parent-rated inattention (Miller et al., 2007). And as Miller and colleagues noted, they cannot determine the causal direction from their findings.

Several problems are remarkable about the Christakis and colleagues (2004) study (and others). If, as the authors rightly noted, this study can say absolutely nothing about early TV exposure *causing* attention problems in children, much less ADHD, then why go on to make such sweeping conclusions based on so weak a correlation? One could just as easily say that attention problems lead children to watch more TV. Critically missing in these affirmative studies is the very real possibility that genetics may actually mediate this relationship. Nowhere do these various studies and their authors even acknowledge that genetic effects may mediate these relationships of inattention to TV viewing. Attention problems such as found in ADHD have a strikingly high genetic influence (average heritability of 71–73% across studies and higher in studies using DSM symptom lists; discussed earlier). This means that much of the variation in attention problems in children is the result of genetic effects. Even more important, twin studies have found no compelling evidence that the shared or rearing environment makes any contribution to these symptoms (discussed earlier). TV viewing within families is often a shared event among children in the family, not a unique event that is specific to just one of them.

This is not just conjecture. In another study of this issue, Reitveld, Hudziak, Bartels, van Beijsterveldt, & Boomsma, 2004) assessed children's attention problems at age 3, then later at 7—the same time frame as in the Christakis and colleagues (2004) study. (Reitveld et al. also assessed them again at ages 10 and 12 years of age.). The beauty of their study is that it was a longitudinal examination of 11,938 twins, so it tells us a great deal about genetic and environmental factors that explain the variation in attention problems. Reitveld and colleagues (2004) found that average heritability across ages for attention problems ranged from 70 to 74%, as expected from numerous other twin studies. All residual variation in this trait was due entirely to unique environmental effects (events not shared among twins or siblings). *None was accounted for by shared events* (!). The authors concluded as well that the stability of attention problems in these twins was also accounted for by genetic factors. This suggests that the children with attention problems at age 7 were highly likely to have already had attention problems at age 3 (and probably age 1), and that the presence of those problems at both ages and their persistence over development are largely explained by genetic factors, along with a more modest contribution of unique events that befall these children. They are not due to any shared event such as TV viewing within the family.

Furthermore, children and adults with ADHD are known to be more likely to watch TV and to watch more TV (Acevedo-Polakovich, Lorch, & Milich, 2007; Barkley, Murphy, & Fischer, 2008; Lingineni et al., 2012) and report enjoying it more than do children without ADHD (Acevedo-Polakovich et al., 2007). This makes it just as likely that the opposite causal direction is operative here: Children with more attention problems prefer and watch more TV than do less inattentive children or those without ADHD and the more severe their inattention the more likely they are to do so over time. Inattentive children also likely have more inattentive parents who may permit their children more time viewing TV than would more attentive parents.

Does content of the programs matter or is it merely quantity of exposure that is so harmful? Interestingly, Christakis and colleagues (2004) say nothing about this issue, yet they confide that educational programs might well have had a *beneficial* effect and moderated their results, thereby making those results a conservative estimate of TV's potential harm to children's attention spans. This is having your cake and eating it too: TV is bad for your children's attention, they want to say, but they are quick to dismiss educational TV from this indictment. In a later study, Zimmerman and Christakis (2007) examined TV content and found that time watching educational TV was not related to attention problems later in childhood, but that watching entertainment programs before age 3 was so related. Interest-

ingly, TV viewing at age 4 or 5 was not related to later attention problems. Likewise, later research indicated that viewing child-directed TV programs from ages 1–4 years was not linked to problems with attention or EF, but watching adult-directed content was so related.

However, efforts to replicate these findings failed to find the reported relationship of early TV exposure and later attention problems. Some of these studies used the same database as Christakis and colleagues (2004), whereas others used even larger and more representative samples, and more appropriate statistical analyses (e.g., Ferguson, 2011; Mistry, Minkowitz, Strobino, & Borzekowski, 2007; Stevens, Barnard-Brak, & To, 2009; Stevens & Muslow, 2006). Others who reviewed this literature have reached the same conclusions as I have here about the lack of convincing evidence on the causal role of televised media exposure and ADHD (e.g., Banerjee, Middleton, & Faraone, 2007). Some suggest a more complicated relationship and indicate that the influence here could be bidirectional, at least for playing video games, which young children who are more inattentive and impulsive play more, and playing games feeds back to exacerbate inattentive and impulsive behavior (Gentile, Swing, Lim, & Khoo, 2012). This suggests that the affirmative studies provide examples of how investigator bias toward correlational findings, inappropriate methods and statistics, a media propensity for sensational findings, and especially a lack of genetically informed methodology can all lead to inappropriate conclusions about the causes of ADHD and to feeding the public an exceptionally mistaken impression—that TV causes ADHD. Of course, the trade media failed to report on these failures to replicate the initial findings. It is little wonder that the vast majority of reports in the media on scientific discoveries are never subsequently replicated given this bias to report mainly on sensational and unexpected findings, without regard to their methodological shortcomings (Gutting, 2013).

SUMMARY

It should be evident from the research reviewed here that neurological and genetic factors make a substantial contribution to symptoms of ADHD and the occurrence of the disorder. A variety of genetic and neurological etiologies (e.g., pregnancy and birth complications, acquired brain damage, toxins, infections, and genetic effects) can give rise to the disorder, likely through some disturbance in a final common pathway in the nervous system. That final common pathway appears to date to be the integrity of the prefrontal cortical–striatal–cerebellar network. It now appears that hereditary factors play the largest role in the occurrence of ADHD symptoms in children. Yet the condition can also be caused or exacerbated by pregnancy complications, exposure to toxins, or neurological disease. Social factors alone are not supported as causal of this disorder, but such factors may possibly interact with genetic risks for the disorder and may contribute to the forms of comorbid disorders associated with ADHD. Cases of ADHD can also arise without a genetic predisposition to the disorder provided the child is exposed to significant disruption or neurological injury to this final common neurological pathway. But this would seem to account for only a small minority of children with ADHD. In general, then, research conducted since the previous edition of this volume was published has further strengthened the evidence for genetic and developmental neurological factors, along with biohazardous events during brain development, as likely causing this disorder, while greatly reducing the support for the role of purely social factors in causing ADHD.

KEY CLINICAL POINTS

- ✓ There is no current, credible scientific theory of ADHD that can account for its existence by purely social means.
- ✓ The totality of evidence indicates that genetic factors related to brain development and functioning, as well as nongenetic neurological factors, play a substantial and majority role in the origins and expression of this disorder.
- ✓ Family studies show a markedly elevated risk of ADHD among the biological relatives of children with ADHD (10–35%), rising to as much as 55% risk to at least one parent in families with two affected children. Parental ADHD coveys a risk of up to 57% to offspring. Adoption studies indicate no increased risk of ADHD among adoptive parents of adopted children with ADHD, further supporting a genetic contribution to ADHD.
- ✓ Numerous studies of large samples of twins in many countries find a genetic contribution accounting for 50–95% of the variation in the traits comprising ADHD

averaging 70–80% or higher. No significant contribution of shared or common environmental factors (rearing environment) has been evident, while nonshared or unique environmental factors make a small but significant contribution to variation in these traits. The strong genetic contribution to ADHD is now a "fact in the bag," and the consistent absence of a rearing environmental contribution rules out within-family factors as contributing to the expression of the disorder but suggests some role for unique events in the lives of children (pregnancy complications, biohazards, developmental risks, and possibly unique social effects).

- ✓ Molecular genetic studies indicate that multiple gene sites are likely to contribute to ADHD risk, with up to 26 candidate genes implicated across studies and possibly as many as 40+ sites in the genome being possible gene polymorphisms contributing to the disorder. Genomewide scans have yet to find evidence for specific genes, largely owing to small sample sizes available at this time that preclude adequate power to detect genes having small effects on the traits of the disorder. Larger samples are now being accumulated that permit more powerful tests for risk genes to ADHD. Meanwhile, some promise is evident in identifying suites of genes that contribute to neurotransmitter pathways. Other studies suggest that some gene polymorphisms, as well as accumulated rare CNVs, may contribute to risk for the disorder.
- ✓ Neuropsychological research finds substantial evidence of deficits in behavioral inhibition, sustained attention (task persistence), resistance to distraction, and EF (the internalization of speech and verbal working memory, temporal-sequential working memory, motor coordination, and the timing of fine-motor movements, emotional and motivational self-regulation, verbal fluency, and planning; see Chapter 3). The EFs are mediated by the prefrontal cortex and its networks with the basal ganglia, anterior cingulate cortex, and cerebellum, among other structures, suggesting that these regions may play a prime role in underlying ADHD.
- ✓ Psychophysiological research demonstrates reduced arousal in response to stimulation (particularly on averaged evoked responses), diminished sensitivity to reinforcement, and increased slow-wave (associate with drowsiness and poor focus of attention) or theta activity, and often decreased beta or fast-wave activity (associated with decreased concentration and persistence) on EEG.
- ✓ Studies of cerebral blood flood indicate reduced flow to the frontal lobes, striatum, and cerebellum, consistent with underactivity in these regions.
- ✓ PET scan studies are inconsistent but suggest some reduced activation in the insular and hippocampal regions and greater activation in the right anterior cingulate during decision-making tasks.
- ✓ MRI studies indicate smaller total brain size, with greatest reductions in brain volumes of the anterior frontal lobes (mainly on the right), the basal ganglia, anterior cingulate cortex, cerebellar vermis (mainly on the right), and corpus callosum (mostly the splenium or frontmost section).
- ✓ fMRI indicates differences from normal brain activity in the frontal region, basal ganglia, anterior cingulate cortex, and cerebellum, among other regions.
- ✓ Deficits in specific neurotransmitters have not been definitively established, but a clear role for DA and NE is suggested by the positive response of those with ADHD to stimulants (DA reuptake inhibitors and agonists) and atomoxetine (noradrenergic reuptake inhibitors), by the distribution of these two transmitter networks in the brain regions implicated in ADHD, and by some peripheral metabolite indices of these neurotransmitters.
- ✓ Pregnancy complications are associated with risk for ADHD, especially maternal smoking, maternal alcohol consumption, LBW and associated minor brain hemorrhaging, possibly maternal phenylalanine levels, and an arguable role for stress/anxiety during pregnancy.
- ✓ Several prenatal toxins have been associated with risk for ADHD, two of which are maternal smoking and alcohol consumption, as noted earlier. Another environmental toxin is postnatal elevated body lead burden during the first 2–3 years of child development.
- ✓ One study suggests a potential contribution of streptococcal infection to some cases of ADHD, wherein the infection triggers an immune response of antibodies that destroy cells of the basal ganglia.
- ✓ Evidence for a contribution of psychosocial factors to ADHD is weak. The recent suggestion from several studies of a role for television viewing during early childhood contributing to ADHD seems to have been overstated, may be more a correlate of attention problems than a cause, did not employ genetically informed designs, and has not been replicated in some studies.

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CHAPTER 15

Theories of ADHD

Erik G. Willcutt

The next chapter clearly describes a strong theoretical model of executive functioning (EF) and self-regulation as forming a phenotype that extends outward into the physical and social ecology to produce adaptive effects at a considerable distance over space and time from the individual. It then extrapolates that theory to understanding attention-deficit/hyperactivity disorder (ADHD) and its management (Chapter 16; Barkley, 2012). My overarching objective in this chapter is to describe and evaluate several other theoretical models of ADHD that have been proposed in the literature. The most prominent competing theories hypothesize that ADHD may arise from more general weakness in EFs—specifically, inhibition and working memory (e.g., Pennington & Ozonoff, 1996), pronounced aversion to the experience of delay (e.g., Sonuga-Barke, Taylor, Sembi, & Smith, 1992), dysfunctional responses to reward and/or punishment contingencies (e.g., Luman, Oosterlaan, & Sergeant, 2005), increased intraindividual variability in response time due to attentional fluctuations (e.g., Sergeant, Geurts, Huijbregts, Scheres, & Oosterlaan, 2003; Sonuga-Barke & Castellanos, 2007), or overall slow cognitive processing speed (e.g., McGrath et al., 2011; Shanahan et al., 2006).

META-ANALYSES OF NEUROPSYCHOLOGICAL STUDIES OF ADHD

The neuropsychological literature on ADHD is immense. As of December 2013, over 500 studies had examined some aspect of the neuropsychological correlates of ADHD, and many of these studies were published after the previous edition of this book was completed (Barkley, 2006). This rapid accumulation of new knowledge has not only provided a key resource to facilitate continued refinement of theoretical models of the pathophysiology of ADHD, but it has also underscored the complexity of the neuropsychological dysfunction that is associated with ADHD.

"Meta-analysis" is a statistical procedure that is used to combine effect sizes from multiple studies to compute a single pooled estimate of the overall effect size in the population. Over the past 10 years our group and several others have completed a series of metaanalyses of different aspects of the neuropsychology of ADHD (e.g., Frazier, Demaree, & Youngstrom, 2004; Hervey, Epstein, & Curry, 2004; Huang-Pollock, Karalunas, Tam, & Moore, 2012; Lansbergen, Kenemans, & Van, 2007; Lijffijt, Kenemans, Verbaten, & van Engeland,

2005; Martinussen, Hayden, Hogg-Johnson, & Tannock, 2005; Pauli-Pott & Becker, 2011; Schoechlin & Engel, 2005; Sonuga-Barke, Sergeant, Nigg, & Willcutt, 2008; van Mourik, Oosterlaan, & Sergeant, 2005; Willcutt & Bidwell, 2011; Willcutt, Brodsky, et al., 2005; Willcutt, Doyle, Nigg, Faraone, & Pennington, 2005; Willcutt, Sonuga-Barke, Nigg, & Sergeant, 2008; Woods, Lovejoy, & Ball, 2002), culminating in our recent meta-analysis of all available neuropsychological studies of DSMIV ADHD symptom dimensions and subtypes (Willcutt et al., 2012). For the purposes of this chapter, all effect sizes have been converted to Cohen's *d*, a widely used measure that reflects the mean difference between groups with and without ADHD, divided by the pooled standard deviation (Cohen, 1988). Therefore, an effect size of 0.5 would indicate that the ADHD and control group differ by half of a standard deviation.

In the subsequent section, I first describe each competing theoretical model and provide a streamlined summary of the neural circuits that are implicated by the theory (because the inhibition–EF extended phenotype model proposed by Barkley is described in detail in Chapter 16, it is described only briefly here). I then summarize results from the latest meta-analyses to evaluate of the validity of each competing model as a theoretical explanation of ADHD. In the penultimate section of the chapter, I discuss the important constraints that are provided by these results for current and future theoretical models of ADHD, then examine the strengths and weaknesses of a recent shift toward theoretical models that explicitly hypothesize that ADHD arises from weaknesses in two or more domains. Finally, I conclude the chapter by describing several key directions for future research that are needed to facilitate the continued development and refinement of a comprehensive theoretical model of ADHD.

COMPETING THEORETICAL MODELS OF ADHD

EFs and Behavioral Inhibition

In addition to Barkley's extended phenotype theory of EF and its application to ADHD (Chapter 16), several other theoretical models have proposed that symptoms of ADHD arise from a more general weakness in the EFs, cognitive processes that facilitate the maintenance of an optimal problem-solving set to attain a future goal (e.g., Barkley, 1997; Pennington & Ozonoff, 1996). In a simplified model of cognitive control and decision-making processes, EFs represent "top-down" cognitive inputs that facilitate decision making. They do so by maintaining information about possible choices in working memory, suppressing irrelevant information, and inhibiting responses that are maladaptive or simply off-task. This knowledge is then integrated with information about reinforcement probabilities to select the optimal action for the situation. Although executive control processes involve several distributed brain networks, studies of primates and neuropsychological, neuroimaging, and lesion studies of humans suggest that the primary neural circuit(s) include the thalamus, basal ganglia, and dorsolateral and ventrolateral regions of the prefrontal cortex (Chapter 14; also see Pennington, 2002).

Although initial studies often described EF as a single unitary construct, subsequent exploratory and confirmatory factor analyses and more recent functional neuroimaging studies suggest that EFs are more accurately characterized as a collection of at least six related but separable processes. These include response inhibition, verbal and spatial working memory, set shifting, planning, and interference control (e.g., Collette et al., 2005; Friedman et al., 2008; Miyake et al., 2000; Willcutt, Pennington, Olson, Chhabildas, & Hulslander, 2005).

Meta-analyses of studies of both children and adults indicate that ADHD is associated with significant weaknesses on measures of most EF domains (Figure 15.1). Results of individual studies indicate that these group differences remain significant when any group differences in intelligence, reading ability, and symptoms of other disorders are controlled (e.g., Barkley, Murphy, & Bush, 2001; Nigg, Hinshaw, Carte, & Treuting, 1998; Willcutt, Betjemann, et al., 2010; Willcutt, Pennington, et al., 2005). Pooled effect sizes were largest for measures of response inhibition ($d = 0.73$; Figure 15.1). This effect size falls in the range that Cohen (1988) classified as a medium to large effect, and suggests that poor inhibitory control accounts for approximately 10% of the variance in ADHD symptoms in the population. Effect sizes were similar but slightly lower on measures of working memory and planning, whereas substantially smaller effects were reported for measures of other EF constructs such as set shifting and interference control on the Stroop task (labeled "Other EF" in Figure 15.1; for meta-analytic reviews of these constructs, see van Mourik et al., 2005; Willcutt, Doyle, et al., 2005).

These phenotypic results are also consistent with results from twin studies, which suggest that the mod-

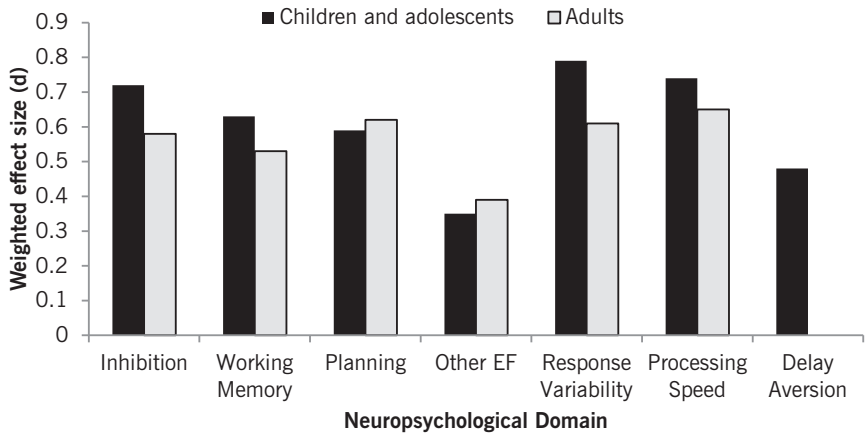

FIGURE 15.1. Weighted mean effect sizes of the difference between groups with and without ADHD on measures of competing theoretical models of ADHD. Effect sizes are based on studies included in previous meta-analyses of neuropsychological studies of children and adults with ADHD, updated to include papers published after the latest meta-analysis (e.g., Frazier et al., 2004; Hervey et al., 2004; Huang-Pollock et al., 2012; Lansbergen et al., 2007; Lijffijt et al., 2005; Martinussen et al., 2005; Pauli-Pott & Becker, 2011; Schoechlin & Engel, 2005; Sonuga-Barke et al., 2008; van Mourik et al., 2005; Willcutt & Bidwell, 2011; Willcutt, Brodsky, et al., 2005; Willcutt, Doyle, et al., 2005; Willcutt et al., 2008, 2012; Woods et al., 2002).

erate covariance among response inhibition, working memory, and ADHD symptoms is primarily explained by common genetic influences (e.g., Willcutt, Betjemann, et al., 2010). However, in addition to the subset of etiological influences that are shared, twin analyses indicate that the majority of the genetic and environmental influences on ADHD symptoms are independent of the influences that lead to weaknesses in inhibition and other aspects of executive control.

In summary, meta-analyses of over 250 studies provide strong support for the theory that ADHD is due at least in part to weaknesses in inhibitory control and other aspects of EF. However, the overall effect sizes from meta-analyses of studies of children and adults indicate that each specific EF weakness accounts for no more than 10% of the variance in ADHD symptoms, suggesting that none of these weaknesses is necessary or sufficient to cause ADHD in isolation.

Motivational Dysfunction

Motivational explanations of ADHD have several variants, but all of these theories suggest that ADHD is attributable to a dysfunctional response to reward and/or punishment contingencies (see Luman et al., 2005, for a comprehensive review). Damage to a neural circuit that includes ventromedial prefrontal cortex, the amygdala, and other limbic structures often leads to difficulty in learning from mistakes and monitoring subtle shifts in reward and punishment probabilities to maximize the short- and long-term benefits of a choice (Bechara, Damasio, & Damasio, 2000; Rolls, 2004). Although only a handful of neuroimaging studies have examined this network in individuals with ADHD, two studies did find a significant correlation between reduced ventromedial prefrontal cortex volume and ADHD symptomatology (Carmona et al., 2009; Hesslinger et al., 2002).

Studies of ADHD that manipulated reward and punishment contingencies have reported mixed results. Because the wide range of tasks and study designs precluded a meta-analysis of these results, I provide a qualitative summary of these results rather than a meta-analysis. One subset of studies found that response contingencies improved and even normalized task performance in individuals with ADHD (e.g., Carlson & Tamm, 2000; Slusarek, Velling, Bunk, & Eggers, 2001). In contrast, several others found a main effect of reinforce-

ment or response cost on the task performance of all children, but no differential effect on individuals with ADHD (e.g., Scheres, Oosterlaan, & Sergeant, 2001; Shanahan, Pennington, & Willcutt, 2008). Finally, a set of studies found that when both reward and punishment were possible outcomes depending on an individual's behavior, individuals with elevations of ADHD symptoms exhibited higher rates of impulsive behavior than they did in a condition with response cost alone (Farmer & Rucklidge, 2006; Hartung, Milich, Lynam, & Martin, 2002).

Taken together, these results suggest that additional research is warranted to clarify the inconsistent results of studies of the impact of motivational contingencies on children with ADHD. However, with the possible exception of the delay aversion model described in the subsequent section, existing data provide minimal support for theoretical models that suggest ADHD is due primarily to a motivational dysfunction.

Delay Aversion

The delay aversion theory suggests that individuals with ADHD find the experience of delay extremely aversive and frustrating, which then leads to choices that minimize delay even when an alternative option would result in a larger reward after a longer delay (e.g., Bitsakou, Psychogiou, Thompson, & Sonuga-Barke, 2009; Sonuga-Barke, 2002; Sonuga-Barke et al., 1992). Individual differences in the capacity to tolerate delay are hypothesized to reflect activity in a neural circuit that includes feedback loops between ventromedial prefrontal cortex, limbic structures, and other areas of the prefrontal cortex (e.g., Sonuga-Barke, Dalen, & Remington, 2003; Sonuga-Barke & Sergeant, 2005). Dopamine, a key neuromodulator for this circuit, is implicated in signaling of rewards, coding incentive value, and coordinating interactions between motivation and cognition during decision-making processes (e.g., Rolls, 2004; Schultz, Tremblay, & Hollerman, 2000).

A number of studies have tested the delay aversion theory by assessing how often individuals with and without ADHD select a small immediate reward rather than a larger delayed reward during a laboratory task. The weighted mean effect size for delay aversion is significant and medium in magnitude, although slightly smaller than the effect sizes for response inhibition and working memory (Figure 15.1). Interestingly, a meta-analysis of studies of preschool children suggested that effect sizes for delay aversion are medium to large in early childhood, then tend to decline over time (Pauli-Pott & Becker, 2011). In contrast, a recent functional neuroimaging study found that experience of a delay was associated with increased right amygdala activity in adults with ADHD but not in adults without ADHD (Wilbertz et al., 2013). This result provides important evidence that adults with ADHD continue to experience negative arousal when exposed to delay, suggesting that additional research is needed to clarify the relation between delay aversion and ADHD across development.

Despite some inconsistent results across studies, the overall literature suggests that delay aversion is one component of a comprehensive neuropsychological model of ADHD. Delay aversion appears to be distinct from general cognitive ability and inhibitory control (e.g., (Karalunas & Huang-Pollock, 2011; Solanto et al., 2001; Sonuga-Barke et al., 2003), but additional studies are needed to test the relations between delay aversion and other cognitive processes such as working memory (Karalunas & Huang-Pollock, 2011) and response variability (e.g., Sonuga-Barke, Wiersema, van der Meere, & Roeyers, 2010).

Response Variability

One of the most consistent results in cognitive studies of ADHD is the finding that the responses of individuals with ADHD are slower and more variable than those of individuals without ADHD (e.g., Alderson, Rapport, & Kofler, 2007; Sergeant et al., 2003; Willcutt et al., 2008, 2012). Meta-analyses of studies that compared groups of children and adults with and without ADHD yielded medium to large effect sizes that are similar in magnitude to the effects that have been reported for response inhibition (Figure 15.1).

Although theoretical models of response variability were initially slow to develop, several theories have recently been proposed. One key theoretical issue concerns whether increased response variability is a unique weakness that is separable from the other cognitive weaknesses associated with ADHD, or whether response variability occurs as a secondary consequence of dysfunction in another cognitive process. For example, an initial parsimonious explanation suggested that increased response variability might be a simple consequence of slow overall reaction time (RT), but most subsequent studies have found that the association between ADHD and reaction time variability remains significant when simple reaction time is controlled (but see Karalunas, Huang-Pollock, & Nigg, 2012).

Studies that used sophisticated statistical models of RT distributions suggest that increased response variability is attributable to a relatively small number of trials with extremely long RTs rather than systematically slower and more variable responses across all trials (Hervey et al., 2006). These sporadic slow responses may potentially reflect attentional lapses due to weak executive control, inconsistent regulation of arousal during lengthy tasks, or dysfunction in short-duration timing mechanisms that are mediated by cerebellar circuits (e.g., Castellanos & Tannock, 2002; Johnson et al., 2007; Sergeant et al., 2003; Toplak & Tannock, 2005).

In summary, while additional research is needed to test competing theoretical models of response variability, the available literature clearly indicates that increased response variability is a robust correlate of ADHD. Of particular importance in the future are studies that administer measures of response variability along with measures of key constructs from other theoretical models of ADHD. By including measures of EF, motivational processing, delay aversion, and processing speed in the same battery as measures of response variability, future studies will be able to test directly whether response variability is independent from these other processes, interacts with weaknesses in one or more of these domains, or is best understood as a secondary consequence of another dysfunctional process.

Processing Speed

Finally, recent evidence from our laboratory and others indicates that groups with ADHD exhibit large and consistent deficits on measures of naming speed and general processing speed (e.g., McGrath et al., 2011; Rucklidge & Tannock, 2002; Shanahan et al., 2006). Effect sizes are medium to large in studies of both children and adults with ADHD (Figure 15.1), and remain significant when group differences in intelligence, reading achievement, and symptoms of other disorders are controlled (e.g., Willcutt, Pennington, et al., 2005).

To my knowledge, no theoretical models have proposed that slow processing speed is a primary weakness that is necessary and sufficient to cause ADHD. However, theoretical models of processing speed have received less attention than the models of the other processes discussed in this chapter. Nonetheless, the robust and consistent effect sizes reported for a range of measures of processing speed indicate that a comprehensive theoretical model of ADHD must account for this specific weakness and clarify its relation with measures of executive control, aversion to delay, and response variability.

DIAGNOSTIC ACCURACY OF MEASURES OF COMPETING THEORETICAL MODELS

The results summarized earlier clearly confirm that groups with ADHD differ from groups without ADHD on measures of multiple neuropsychological processes. To assess the clinical relevance of these group differences it is also important to examine the ability of each measure to predict categorical ADHD diagnostic status. To address this question for this chapter, I completed a series of analyses to calculate the proportion of individuals with ADHD who exhibited significant impairment on measures of each of the competing theoretical models of ADHD.

Analyses were completed in three independent samples in our laboratory. As part of their participation in the Colorado Learning Disabilities Research Center (CLDRC), a sample of 505 participants with ADHD between 8 and 15 years old completed an extensive test battery that includes multiple measures of response inhibition, working memory, response variability, delay aversion, and processing speed (e.g., Willcutt, Betjemann, et al., 2010; Willcutt, Pennington, et al., 2005). A similar battery was administered to a separate population-based sample of 350 children with ADHD who were between 7 and 13 years of age at the time of the assessment (e.g., Willcutt, 2012; Willcutt et al., 2011), and a sample of 190 young adults with ADHD completed all of these measures with the exception of delay aversion (e.g., Willcutt & Bidwell, 2011). In each sample, the 10th percentile of the control group was used as a cutoff score to define significant neuropsychological impairment on each measure, a procedure that is consistent with the approach used in previous analyses by our group (e.g., Nigg, Willcutt, Doyle, & Sonuga-Barke, 2005; Willcutt, 2009) and others (e.g., Sjowall, Roth, Lindqvist, & Thorell, 2013; Wahlstedt, Thorell, & Bohlin, 2009).

Diagnostic Sensitivity

With the exception of a marginally significant finding for delay aversion in the CLDRC sample, participants with ADHD were significantly more likely than individuals without ADHD to exhibit deficits in all five do-

mains (Figure 15.2), indicating that each neuropsychological weakness had significant sensitivity. However, fewer than half of the individuals who met full diagnostic criteria for ADHD exhibited a significant deficit on any specific measure, a pattern that is consistent with earlier results from our studies and other samples of children and adolescents (e.g., Nigg et al., 2005; Sjowall et al., 2013; Wahlstedt et al., 2009; Willcutt, 2009; Willcutt, Brodsky, et al., 2005).

Consistent with the significant group deficits across a wide range of measures in the meta-analyses, these analyses suggest that no single weakness provides a necessary or sufficient explanation of all cases of ADHD. These results also illustrate why neuropsychological measures have limited clinical utility despite the large and consistent effect sizes observed in comparisons of groups with and without ADHD (as discussed in Chapters 4, 10, and 18). Although a higher proportion of individuals with ADHD have each weakness than would be expected by chance, analyses of each measure indicated that over half of the individuals who met full criteria for ADHD did not exhibit significant impairment on that measure.

Diagnostic Specificity

Another important complicating factor for theoretical models of ADHD is the high rate of comorbid disorders in individuals with ADHD. Over 80% of children, adolescents, and adults with ADHD also meet criteria for another disorder, and many meet criteria for multiple additional diagnoses (Chapters 5 and 13; also Faraone, Biederman, Weber, & Russell, 1998; Willcutt, Pennington, Chhabildas, Friedman, & Alexander, 1999). In a comprehensive meta-analysis of studies of DSM-IV ADHD, Willcutt and colleagues (2012) found that children and adults with ADHD combined type were 10 to 15 times more likely than individuals without ADHD to meet criteria for oppositional defiant disorder (pooled estimate from the meta-analysis = 52%) and nearly 20 times more likely to meet criteria for conduct disorder (pooled estimate = 22%; e.g., Hinshaw, 2002; Volk, Neuman, & Todd, 2005). Rates of comorbidity were significant but lower between ADHD and anxiety disorders (12–20%), depression (10–15%), and bipolar disorder (7–12%). Adults with ADHD were three times more likely than adults without ADHD to meet criteria for a substance use disorder (pooled estimate = 41%; e.g., Murphy, Barkley, & Bush, 2002; Sobanski et al., 2008). Finally, children with ADHD were three to four times more likely than children without ADHD to meet criteria for a speech–language disorder (15%) or learning disorder (24%; e.g., Hinshaw, 2002; Willcutt, Betjemann, et al., 2010).

The nearly ubiquitous comorbidity exhibited by individuals with ADHD initially called into question whether the significant neurocognitive weaknesses observed in groups with ADHD might be associated

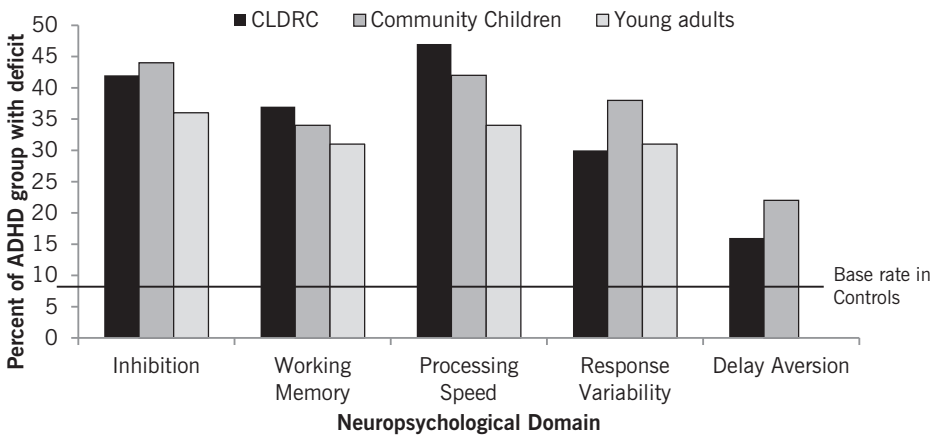

FIGURE 15.2. Percentage of children and adults with ADHD who scored below the 10th percentile of the comparison sample on each neuropsychological composite measure.

with one of the comorbid disorders rather than ADHD per se. However, subsequent studies showed that differences between ADHD and control groups on measures of response inhibition, working memory, delay aversion, response variability, and processing speed all remained significant when symptoms of most comorbid disorders were controlled (e.g., Rucklidge & Tannock, 2002; Solanto et al., 2007; Willcutt, Betjemann, et al., 2010). These results provide important evidence that each of these neuropsychological weaknesses is independently associated with ADHD, and is not simply a consequence of a comorbid disorder.

On the other hand, a meta-analysis of neuropsychological studies of nine childhood disorders indicated that none of these neuropsychological weaknesses are specific to ADHD (Willcutt et al., 2008). Weaknesses in response inhibition, working memory, processing speed, and response variability have been reported consistently in studies of children with pervasive developmental disorders (autistic spectrum disorders; e.g., Geurts, Verte, Oosterlaan, Roeyers, & Sergeant, 2004; Ozonoff & Jensen, 1999; Verte et al., 2005), childhood-onset psychosis (e.g., Karatekin, Bingham, & White, 2009), learning disabilities (e.g., Purvis & Tannock, 2000; Willcutt, Betjemann, et al., 2010; Willcutt et al., 2001), and mood disorders (Bearden et al., 2007; Rucklidge, 2006), suggesting that dysfunction in each of these processes may be a shared risk factor for ADHD and other disorders.

SUMMARY OF FINDINGS ON COMPETING THEORETICAL MODELS

The results summarized in this chapter clearly indicate that ADHD is associated with weak inhibitory control, providing strong support for one the key predictions of the theoretical model proposed by Barkley (1997) over 15 years ago and updated in Chapter 16. In addition to this specific weakness in response inhibition, ADHD is also associated with significant weaknesses on measures of working memory, delay aversion, response variability, and processing speed.

Meta-analyses of studies of both children and adults reported medium to large (d = 0.50–0.70) effect sizes for each of these measures when groups with and without ADHD were compared. However, all of these effects are far smaller than the effect size of the difference between groups on measures of ADHD symptoms (d = 3.0–4.0), and each weakness accounts for a maximum of 10% of the variance in ADHD symptoms. Similarly, whereas individuals with ADHD are three to four times more likely than individuals without ADHD to exhibit a clinically significant weakness on measures of each of these constructs, fewer than half of all individuals with ADHD exhibit a significant deficit in any specific domain. Taken together, these results suggest that no neuropsychological weakness is necessary or sufficient to cause all cases of ADHD, and they underscore the neuropsychological heterogeneity of ADHD.

A SHIFT TOWARD MULTIPLE-DEFICIT MODELS

The converging results summarized in this chapter have precipitated a significant reconceptualization of theoretical models of ADHD (Pennington, 2006; Willcutt et al., 2008). Rather than attempting to identify a single neuropsychological weakness that is necessary and sufficient to cause ADHD, recent theoretical models explicitly hypothesize that ADHD is a complex and heterogeneous disorder that is associated with weaknessess in multiple domains (Nigg, 2006; Sonuga-Barke, 2005; Sonuga-Barke et al., 2003; Willcutt et al., 2008). To illustrate some of the different approaches that may be used to conceptualize these more complex models, I describe in the remainder of this chapter two broad types of theoretical models that incorporate multiple neurocognitive deficits.

Multiple-Pathway Models

Independent pathway models suggest that dysfunction in any of two or more pathophysiological substrates may independently lead to the same final behavioral manifestation of a disorder. In other words, these models propose that there are distinct *neuropsychological subtypes* of ADHD (e.g., Nigg et al., 2005). For example, Sonuga-Barke (2002, 2003) and his colleagues have proposed a dual-pathway model in which some individuals exhibit ADHD symptoms due to significant aversion to delay, whereas others have ADHD due to weak inhibitory control. This model received some support from studies in which delay aversion and response inhibition predicted ADHD symptoms independently (e.g., Solanto et al., 2001; Sonuga-Barke et al., 2003). However, other studies found that a significant subset of individuals with ADHD exhibit weaknesses in both domains, suggesting that these pathways may not be entirely independent (Sonuga-Barke et al., 2008).

Multiple-Deficit Models

In contrast to the distinct neuropsychological subtypes proposed by independent pathway models, multiple-deficit models suggest that ADHD symptoms arise from the additive and interactive combination of multiple neuropsychological weaknesses, none of which is necessary or sufficient to cause the disorder when it occurs in isolation (e.g., Pennington, 2006; Willcutt, Betjemann, et al., 2010). The specific cluster of weaknesses may then differ across individuals, providing a potential explanation for clinical heterogeneity. For example, individuals with weaknesses in executive control and processing speed may be most likely to exhibit significant inattention and comorbid learning difficulties, whereas EF weaknesses coupled with aversion to delay or disruption in other motivational processes might lead to impulsive behaviors and comorbidity with other disruptive disorders, such as conduct disorder.

To illustrate further these two competing models, I conducted a final set of analyses for this chapter using the three samples that were described previously (to simplify the presentation of the results, the two samples of children were combined for this analysis). Figure 15.3 summarizes the total number of neuropsychological deficits exhibited by individuals in the ADHD and comparison groups. Nearly 30% of the individuals who met full DSM-IV diagnostic criteria for ADHD did not exhibit a significant weakness on any of the measures in our test battery, a pattern that is highly consistent with the results reported by others who have completed similar analyses (e.g., Karalunas & Huang-Pollock, 2011; Sjowall et al., 2013; Sonuga-Barke et al., 2008). This subgroup may potentially have subclinical weaknesses on one or more of the dimensions assessed in this study, or a weakness in a process that we simply did not measure despite the extensive neuropsychological batteries that were administered in each study.

Slightly more than 70% of the individuals with ADHD exhibited at least one neuropsychological weakness, and 25–30% exhibited significant impairment in a single domain (Figure 15.3). The cases with a single deficit included subsets of individuals who were only impaired on measures of delay aversion, response variability, or response inhibition. These results are consistent with the predictions of the independent pathway model, which hypothesized that the overarching diagnosis of ADHD would include multiple subgroups with separate neurocognitive weaknesses.

Finally, approximately 40% of the individuals with ADHD exhibited significant impairment in at least two domains, and over 20% exhibited three or more weaknesses. These results are consistent with a recent study in which 27% of participants with ADHD had weaknesses in two or more neurocognitive domains, with 22% exhibiting weaknesses in EF and response variability (Sjowall et al., 2013). The pattern of results in both of these samples is consistent with the expectations of the multiple-deficit model.

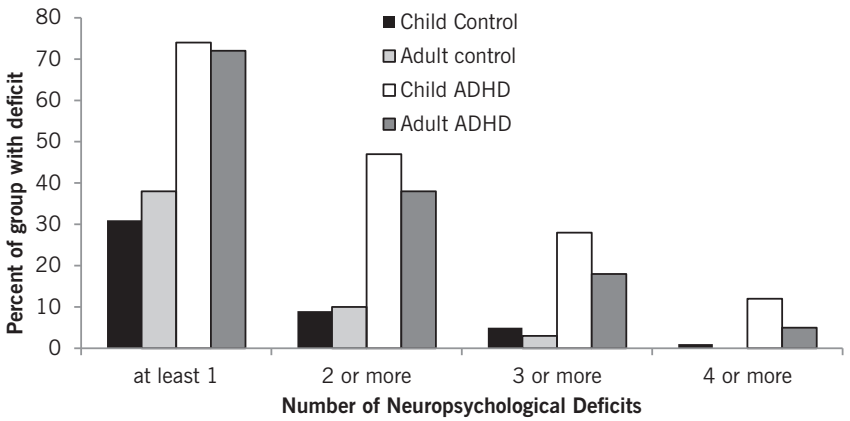

FIGURE 15.3. Percentage of children and adults with and without ADHD who exhibited significant weaknesses in at least one, two, three, or four or more neuropsychological domains.

Summary

A growing literature suggests that a comprehensive neurocognitive model of ADHD is likely to involve dysfunction in multiple cognitive processes that are mediated by neural networks distributed across many locations in the brain. Further research is needed to test whether dysfunction in these different networks leads to distinct neuropsychological subtypes within the overall population of individuals with ADHD, or whether multiple dysfunctional processes act in combination to increase susceptibility to ADHD in an individual. Perhaps the most likely scenario is a hybrid of these two models, in which some cases of ADHD are attributable to a primary deficit in a relatively specific neurocognitive process, whereas others may be caused by the combined effects of dysfunction in multiple substrates.

KEY ISSUES AND FUTURE DIRECTIONS

The transition from theories positing a single primary deficit to multiple-deficit models represents an important shift in the conceptualization of theoretical models of ADHD (Pennington, 2006). In this final section, I summarize several key remaining issues for the field and highlight important future directions for studies that test theoretical models of ADHD within a multiple-deficit framework.

Diagnostic Heterogeneity

Although this book focuses on the DSM diagnosis of ADHD and neuropsychological hetereogeneity within this group, ADHD is also clearly heterogeneous at the level of behavioral symptoms. The importance of this behavioral heterogeneity is underscored by the results of our comprehensive meta-analysis of the DSM-IV symptom dimensions and diagnostic subtypes (Willcutt et al., 2012). As expected, pooled results across studies indicated that zero-order correlations were significant between both inattention and hyperactivity–impulsivity symptoms and a wide range of neurocognitive measures. However, nearly all of these measures were more highly correlated with inattention than hyperactivity–impulsivity, and none were more highly correlated with hyperactivity–impulsivity.

Analyses of the DSM-IV subtypes indicated that the combined and inattentive types performed significantly worse than comparison groups without ADHD on measures of response inhibition, planning, vigilance, processing and naming speed, and some aspects of motor functioning. In contrast, there were few differences between the combined and inattentive types on these measures. The absence of any prominent differences between the combined and inattentive subtypes was unexpected based on initial hypotheses proposed by our group and others (e.g., Chhabildas, Pennington, & Willcutt, 2001; McBurnett, Pfiffner, & Frick, 2001; Milich, Balentine, & Lynam, 2001), but it is consistent with the dimensional results that suggested that most neuropsychological weaknesses are primarily driven by symptoms of inattention.

In contrast to the consistent neuropsychological weaknesses associated with the inattentive and combined types, effect sizes were much smaller and often not significant when the hyperactive–impulsive type was compared to a group without ADHD. Furthermore, the hyperactive–impulsive type performed significantly better than the inattentive and combined types on most neuropsychological measures. Taken together, these results suggest that the inattentive and combined types (now presentations in DSM-5; American Psychiatric Association, 2013) may share a common neuropsychological etiology, whereas the minimal evidence of neuropsychological dysfunction underscores ongoing questions regarding the diagnostic validity of the hyperactive–impulsive type.

Direct Tests of Competing Theoretical Models

Most previous studies have examined the neuropsychological correlates of ADHD with a limited test battery that is designed to assess constructs derived from a single theoretical perspective. While the primary effect sizes from studies of different theories can be compared to provide a preliminary appraisal of the relative merits of different theoretical models, interpretation of these comparisons is often compromised by important differences in study design or sampling procedures. To continue to move the field forward, future studies should be explicitly designed to facilitate direct comparisons of alternative theoretical models in the same sample. As summarized earlier in this chapter, studies that have adopted this approach have already had an important impact on our understanding of competing theoretical models of ADHD (e.g., Karalunas & Huang-Pollock, 2011; Solanto et al., 2001; Sonuga-Barke et al., 2003; Willcutt, Pennington, et al., 2010).

Clinical Utility

I realize that the debate about major and minor details of competing theoretical models may seem esoteric and far removed from any concrete clinical application. But the overarching long-term objective of each of these theoretical models and all neuropsychological research is to improve clinical diagnostic procedures and subsequent interventions for children, adolescents, and adults with ADHD. Because only a minority of individuals with ADHD exhibits a significant weakness on any of the measures of competing theoretical models that have been proposed to date, no measures that are currently available have sufficient sensitivity or specificity to be useful for clinical diagnosis. Furthermore, the clear evidence of pervasive neuropsychological and behavioral heterogeneity in individuals with ADHD suggests that it is likely to remain difficult or impossible to develop a single diagnostic test with positive and negative predictive power that is adequate for diagnostic purposes.

Even if it is not possible to use measures based on the competing theoretical models for diagnostic purposes, results of neuropsychological studies are still likely to have several important clinical implications and utility (Willcutt & Bidwell, 2011). For example, although it appears unlikely that a single neuropsychological measure will have sufficient diagnostic utility in isolation, it may eventually be possible to combine neuropsychological data with information about an individual's genetic background, family history, environmental circumstances, and other etiological factors to create a probabilistic measure of overall risk for ADHD. This profile could then be used to supplement parent and teacher ratings to identify individuals who are at high risk for ADHD, facilitating primary prevention or early intervention.

Neuropsychological measures may also facilitate the identification and implementation of interventions that target the specific areas of weakness that are most impairing for a particular individual. This might involve pharmacological interventions that target dysfunction in a specific neural substrate or environmental interventions designed to remediate or accommodate the specific pattern of weaknesses exhibited by an individual. For example, an individual with significant EF difficulties might benefit from a structured behavioral and organizational system that provides consistent reminders about appointments and tasks that must be completed, whereas an individual with a pronounced elevation of response variability might require frequent breaks to maintain an optimal response set when completing lengthy assignments. Similarly, an individual with severe processing speed difficulties might benefit from increased time to complete tests and assignments, so that slow processing speed does not compromise his or her ability to demonstrate mastery of the material.

CONCLUSIONS

The neuropsychology of ADHD is complex and multifactorial, with no single deficit that is necessary or sufficient to explain all cases of ADHD. A comprehensive theoretical model of ADHD must account for weaknesses in response inhibition and other aspects of executive control, increased aversion to delay, and slower and more variable response speed. Additional research is needed to clarify the relations among these complex cognitive processes and to determine the best approach to describe the behavioral and neuropsychological heterogeneity that characterizes ADHD.

KEY CLINICAL POINTS

- ✓ Several theoretical models of ADHD exist in the literature: the extended phenotype model of executive functioning and behavioral inhibition (see Chapter 16); the concept of ADHD as a motivational dysfunction; the theory of ADHD as comprising delay aversion (Sonuga-Barke et al., 2008); and conceptualizations focusing on excessive response variability (Kinsbourne, De Quiros, & Rufo, 2001), deficient arousal regulation (Sergeant, 2005; Van der Meere & Stemerdink, 1999), and impaired processing speed, among others.
- ✓ Each is briefly reviewed along with the evidence that exists to support it.
- ✓ It seems clear that no single theory or concept can account for all of the findings in the neuropsychological literature on ADHD, in that some subsets of ADHD have deficits in one or more of these concepts, while others do not or have them in different domains.
- ✓ Neuropsychological tests that are purported to assess these various concepts are not sufficiently accurate for use in diagnosis. Deficits on these tests also are often shared with other psychiatric disorders.
- ✓ The findings to date support the need for a multiple-

pathways or multiple-deficits model of ADHD, in which its symptoms and neuropsychological deficits can arise from a variety of cognitive origins that would more easily explain the heterogeneity of findings across cases and studies. Results of research to date appear to be more supportive of a multiple-deficits model of ADHD than of subtypes of ADHD that arise via different pathways.

- ✓ Future research should focus on direct tests of the various competing models of ADHD and identifying tests that may have greater diagnostic accuracy for the disorder than do existing ones.

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CHAPTER 16

Executive Functioning and Self-Regulation Viewed as an Extended Phenotype

Implications of the Theory for ADHD and Its Treatment

Russell A. Barkley

As explained in Chapter 2, attention-deficit/hyperactivity disorder (ADHD) is currently viewed clinically and in official taxonomies as involving developmentally inappropriate degrees of inattention and hyperactive–impulsive behavior. In Chapter 3, I made the case for also including problems with emotional impulsiveness and poor emotional self-regulation in these behavioral symptom dimensions. Here I broaden my argument to show that these symptom dimensions represent "executive functioning"—a suite of mental abilities used for self-regulation over time to accomplish goals largely within a social context and often using social and cultural means so as to maximize one's later welfare (Barkley, 2012b). This suite of faculties underlies human choices between a current state and a future one because it relies on an ability to inhibit automatic behavior and to generate imagined hypothetical futures. This theory was first proposed in rough form in my chapter in an edited volume (Barkley, 1994), followed by a more detailed theoretical article in *Psychological Bulletin* (Barkley, 1997b) and expanded shortly thereafter into a book (Barkley, 1997a). In that theory, I initially argued that behavioral inhibition is a key foundational executive function (EF) that, when deficient in ADHD, would create a cascade of deficits into other EFs, such as working memory, emotional and motivational self-regulation, and planning–problem solving. In a subsequent 2001 paperback edition of the original text (Barkley, 1997a), I amended this view to acknowledge that deficits in working memory can coexist with those of behavioral inhibition in ADHD, in which both are primary interacting contributors to the disorder and, while interdependent, are also only partially uncoupled. This theory has been recently updated and expanded to incorporate the concept from evolutionary biology of "extended phenotypes"—genetic effects at a distance that are of consequence to the survival and welfare of the organism (Barkley, 2012b). In this chapter, an adaptation and expansion of one of my chapters in the previous edition (Barkley, 2006) and subsequent ones (Barkley, 2011a), I briefly summarize this theory and some of its implications for understanding and managing ADHD (not to mention other executive or frontal lobe disorders).

THE PROBLEM WITH THE CURRENT CLINICAL VIEW

Numerous psychological deficits have been identified that do not fit neatly into the clinical view of ADHD as principally a disorder of attention and inhibition–

activity level (see Chapters 4, 10, and 15). These problems also cannot be easily accounted for by competing and far more circumscribed theories of ADHD, as discussed in Chapter 15, such as theories involving delay aversion, resource allocation, or motivation (Coghill, Nigg, Rothenberger, Sonuga-Barke, & Tannock, 2005; Nigg & Casey, 2005; Sergeant, 2005). Among these, are difficulties with (1) physical fitness, gross and fine motor coordination, motor sequencing (Harvey et al., 2007; Kadesjö & Gillberg, 2001), and motor speed (Chapter 15); (2) cold cognitive components of EF (working memory, planning and problem solving, fluency, etc.; Frazier, Demareem, & Youngstrom, 2004; Hervey, Epstein, & Curry, 2004); (3) effort allocation (Douglas, 1983; Nigg & Casey, 2005); (4) developing, applying, and self-monitoring organizational strategies (Hamlett, Pellegrini, & Connors, 1987; Purvis & Tannock, 1997); (5) the internalization of self-directed speech (Berk & Potts, 1991; Winsler, Diaz, Atencio, McCarthy, & Chabay, 2000); (6) adhering to instructions (Danforth, Barkley, & Stokes, 1991); (7) self-regulation of emotion and motivation (Chapter 3); and (8) time reproduction, temporal discounting, and time management (Barkley & Fischer, 2011; Barkley, Koplowicz, Anderson, & McMurray, 1997; Barkley, Edwards, Lanieri, Fletcher, & Metevia, 2001; Barkley & Murphy, 2011; Demurie, Roeyers, Baeyens, & Sonuga-Barke, 2013. Some research has also shown that ADHD may be associated with less mature or diminished moral development (Hinshaw, Herbsman, Melnick, Nigg, & Simmel, 1993). Many of these difficulties appear to be relatively specific to ADHD and cannot be explained as a function of comorbid disorders, such as learning disabilities, depression, anxiety, or oppositional/conduct disorder (Barkley, Murphy, & Fischer, 2008; Klorman et al., 1999; Nigg, 1999), yet they are not accounted for in any competing theories of ADHD, as discussed in Chapter 15.

What these seemingly disparate abilities share, in my opinion, is that nearly all of them, except motor speed and coordination, have been considered to fall within the domain of "EFs" in the field of neuropsychology (Chapters 4, 10, and 15; also see Barkley, 1997a; Denckla, 1996) or "metacognition" in developmental psychology (Welsh & Pennington, 1988), or to be affected by these functions. All seem to be mediated, at least in part, by the frontal cortex, and particularly the prefrontal lobes. And they are related to at least three or more neural networks that are implicated in the neuropsychology of ADHD (Castellanos, Sonuga-Barke, Milham, & Tannock, 2006; Fuster, 1997; Nigg & Casey, 2005; Sagvolden, Johansen, Aase, & Russell, 2005). Theorists and clinical scientists have long-speculated that problems with EF, or the cross-temporal organization of behavior specifically and self-regulation more generally, are at the heart of this disorder and give rise to the more superficial and surface symptoms represented in clinical diagnostic criteria (Barkley, 1994, 1997a, 1997b, 2012b; Douglas, 1983; Pontius, 1973; Still, 1902).

EVIDENCE THAT ADHD IS EF DEFICIT DISORDER

The view of ADHD as EFDD—executive function deficit disorder—is highly consistent with clinical observations of the disorder that bear a striking similarity to patients with prefrontal lobe (executive) injuries (Fuster, 1997; Luria, 1966; Stuss & Benson, 1986), including the classic case of Phineas Gage (Harlow, 1848, 1868). And this view of ADHD as EFDD is also consistent with the results of research employing rating scales of EF in daily life. These studies show that ratings of ADHD symptoms in adults are highly correlated with those ratings of EF in daily life (.80–.91 for inattention; .68–.84 for hyperactivity–impulsivity; .91 for total scores in clinical samples; and .65–.83, .46–.63, and .81 for general population samples, respectively; Barkley, 2011b). Similar findings are evident in children (.77–.87, .60–.74, and .87, respectively, for general population samples; Barkley, 2012a). Indeed these correlations are so high that they suggest ratings of ADHD and EF are colinear or they assess the same construct. The lower correlations most likely arise because ADHD symptom lists do not include problems with EFs such as emotional self-regulation, time management, self-motivation, and so forth.

Also consistent with this interpretation of these findings are the results of factor analyses of ADHD symptom ratings with EF ratings. They indicate that these symptoms form a single factor whether one uses clinical samples of adults with ADHD or a general population sample of U.S. adults (Barkley, 2011b). When clinically diagnosed adults with ADHD are classified as being impaired in EF ratings (placing at or above the 93rd percentile; a rather strict criterion), 86–98% of them are so classified across the various EF dimensions (Barkley & Murphy, 2011). In children diagnosed by ADHD research criteria, 68–78% place in this same range of impairment across EF dimensions (Barkley, 2012a). Far higher rates of impaired individuals would be evident

if one used a more relaxed traditional threshold of impairment, such as the 10th–15th percentile. Effect size differences across EF dimensions between samples with and without ADHD are a staggering 1.93 to 2.86 in that report (Barkley, 2012a). So at least where rating scales of ADHD and EF are concerned, one can rightly conclude that ADHD symptoms and EF deficits are highly similar, if not identical, constructs and that ADHD *is* probably EFDD.

THE SERIOUS PROBLEMS WITH NEUROPSYCHOLOGICAL TESTS OF EF

Then why are these findings not evident in traditional research studies on EF in ADHD, as discussed in Chapter 15? Indeed, why are the results in those studies nearly diametrically opposed to the findings I have just discussed? As is made clear in Chapter 15, only a small minority of patients with ADHD are deficient in any of the many constructs of EF that have been assessed in such research. Indeed, according to reviews of that literature, ADHD cannot be EFDD because most people with ADHD do not exhibit clinical impairment on EF test batteries (see Barkley, 2011b, 2012a, 2012b). How is this possible if one is discussing the same construct of EF as measured by these different methods? For one thing, it is because EF tests and EF ratings are not significantly correlated with each other or, if significant, they are so poorly related that they share less than 10% of their variance, leading reviewers to conclude that these methods do not assess the same construct (Toplak, West, & Stanovich, 2013). These contrasting results pose serious conceptual and methodological problems for EF research. Both approaches cannot be right.

As I have discussed in detail elsewhere (Barkley, 2012b), the information-processing views of EF and ADHD have elevated laboratory neuropsychological tests to the august status of being the "gold standard" by which EF is to be measured. As noted in Chapter 15, of the more than 500 studies of EF conducted in the field of ADHD, nearly all have relied on psychometric tests or batteries of such tests to measure EF. The results are then analyzed and even meta-analyzed in order to get a clearer picture of whether ADHD involves deficiencies in EF (or other psychological abilities) and, if so, which components. From these results, various models of EF (and ADHD) may be constructed and contrasted against each other to determine which provides the best account of the disorder, if any. As is made clear in Chapter 15, people with ADHD show a variety of domains of cognitive deficits on such tests, but only a minority have deficits in any single domain, be it response inhibition, working memory, delay aversion, response variability, or motivation. Thus, reviewers often conclude that ADHD is not EFDD because the majority of patients with ADHD do not share some common EF deficit (Boonstra, Oosterlaan, Sergeant, & Buitelaar, 2005; Jonsdottir, Bouma, Sergeant, & Scherder, 2006; Marchetta, Hurks, Krabbendam, & Jolles, 2008; Nigg, Wilcutt, Doyle, & Sonuga-Barke, 2005; Willcutt et al., 2005).

What usually goes unnoticed or unchallenged here is the unquestioned and often inchoate premise that neuropsychological testing is the best (or only) means of evaluating EF. If that premise is false, the results of those hundreds of studies of EF in ADHD that have relied on it are far, far more limited in their conclusions and the light they shed on EF and on ADHD than has been acknowledged by most scientists working in this field. Consider that these tests have little or no correlation (1) with rating scales of EF in daily life activities, as noted earlier; (2) with adaptive functioning; or (3) with direct observations of EF behavior collected in natural settings; and (4) these tests are rather poor at predicting impairments in major life activities that are rife with EF (see Chapters 4 and 10, also see Barkley, 2012b), if they predict them at all. One therefore has solid grounds on which to challenge the primacy of EF tests as the "gold standard" of EF measurement. Rating scales of EF, in contrast, are superior to EF tests and their batteries in these respects (Barkley, 2011b, 2012a), yet the results of such ecologically valid observations by others are rarely if ever used in evaluating the involvement of EF in disorders such as ADHD in research studies.

There are obvious logical fallacies evident in EF research based on EF tests, including such research in the field of ADHD. They are striking if one simply takes the time to notice them. Consider the following propositions:

1. The prefrontal cortex (PFC) is the "executive" brain (Pribram, 1973) and the principal site underlying the EFs (Denckla, 1996).
2. ADHD is clearly a disorder of prefrontal cortical networks (see Chapter 14).
3. But on EF tests we find only a minority of ADHD cases with impaired EF (Chapters 4, 10, and 15).
4. Therefore, ADHD is not a disorder of EF (Boonstra et al., 2005; Jonsdottir et al., 2006; Marchetta et al., 2008; Nigg et al., 2005; Willcutt et al., 2005).

The latter conclusion is logical only if EF tests are viewed as the most valid means of evaluating EF. But, logically, this conclusion must be wrong given propositions 1 and 2.

Or consider the following example, which makes the logical fallacy just as evident:

1. Patients with ADHD are selected by virtue of having serious symptoms of inattention and hyperactivity–impulsivity (poor inhibition; see Chapter 2).
2. The majority of them do not manifest deficits on tests of inattention and response inhibition (continuous-performance tests, reaction time tasks and reaction time variability, color–word tests, etc.; see Chapters 4, 10, and 15).
3. Therefore, ADHD is not principally a disorder of inattention or inhibition.

So which is it? Is proposition 1 correct or proposition 3? All patients are selected for the studies because they have high levels of inattention and inhibition, as reported by themselves or others. But then this is found not to be so, based on these EF tests. Either the observations and ratings that serve as the basis for proposition 1 are correct and the tests that are the basis of proposition 3 are wrong, or vice versa. One of these two approaches to symptom ascertainment must be wrong. Such a fallacy based on viewing the EF tests as the "gold standard" for evaluating EF has led researchers not only to falsely conclude that ADHD largely does not involve EF but also to then chase multiple pathway models of cognitive deficits for ADHD when the problem here is not theoretical or conceptual but a problem of measurement. Scientists must stop relying on EF tests as the sole method and "gold standard" for measuring EF deficits (and ADHD symptoms)!

EF tests have numerous conceptual (as well as psychometric) flaws, discussed elsewhere (Barkley, 2012b), that clearly show why they capture so poorly the nature of EF as it is used by people in daily life. Suffice to say here that if EF is the cross-temporal organization of behavior toward the future or goal-directed action, it is not clear how current EF tests capture this incredibly important domain of human life. Just what do tests of digit span backwards, detecting X's from O's, building towers on spindles, generating various words beginning with the letter F, or sorting cards into categories have to do with this definition of EF, or with the serious and pervasive deficits that people with prefrontal lobe disorders manifest in daily life? Are these the most important mental faculties that the classic case of Phineas Gage lost in his massive frontal lobe injury in 1848? And are these the most important domains of impairment likely to arise from such injuries or from developmental disorders of the prefrontal executive brain in cases such as ADHD? Obviously not.

The PFC is responsible for the human capacity to contemplate a future state and juxtapose it with a current one, and so engage in choices between these two. One then chooses and acts, thereby engaging in the construction of cross-temporal behavioral structures to strive to achieve or avoid that future state. This is the highest faculty of humanity and likely the principal mental adaptation by which it survives (Barkley, 2012b). The capacity to contemplate the future is the basis of social reciprocity, friendships and social networks, cooperation; educational and occupational functioning, cohabitation and childrearing; economics, finances and marketplaces; ethics, law and government; among other major human life activities. It is also the basis of culture and the capacity to utilize contemplation for goal-directed action (Barkley, 2012b). These are the capacities that are lost when one sustains injuries to the prefrontal executive brain, and not just performance on an *n*-back, spatial memory, or "go/no-go" tasks, among other EF tests.

Thus, studies using EF tests to evaluate the validity of theories of EF and of ADHD do not provide definitive or even very enlightening answers to the question of how much ADHD is a disorder of EF. What little information they provide about EF is highly limited in scope, if they are able to provide such answers at all. And the results of such studies cannot be used to build a comprehensive theory of EF (or of ADHD) alone, no matter how many individual studies, measures, and their meta-analyses are done. In short, we cannot judge the status of EF and its deficits in ADHD by tests alone, and any efforts to build theories of ADHD (or EF) from such tests will be seriously limited in terms of the validity of their constructs and conclusions. I now turn to the construction of a theory of EF that can serve to bridge the cognitive "instrumental" level of EF with how it is applied to daily adaptive and social functioning.

DEFINING INHIBITION, SELF-REGULATION, AND EF

Serious theory building involving ADHD and EF necessitates that one (1) define operationally the terms or constructs to be used, (2) give a reasonable account

of how normal EF and self-regulation develop in children, and (3) explain just how ADHD acts to disrupt that normal developmental process. These necessities have led me to spend the better part of the past 20 years conceptualizing and investigating the nature of self-control in normally developing children and adults and those with ADHD. And as one who favors evolutionary psychological views of mental faculties, I have also conjectured about the possible adaptive advantages that psychological modules for self-regulation and EF may have served in the course of human evolution (Barkley, 2001, 2012). Such conjectures can serve to suggest testable hypotheses for future research concerning the nature and purposes of self-regulation and broaden the scope of social domains that developmental disorders such as ADHD, or acquired disorders such as frontal lobe injuries, may disrupt. Space constraints in this chapter limit me to providing merely an overview of the theory of self-control and EF I have developed, then extrapolated in an attempt to explain that disorder.

Good science and scholarship demand that we define our terms in as operational a way as possible, if we are to avoid conceptual or semantic confusion. Of relevance to a theory of EF, and therefore of ADHD, are operational definitions for the terms "behavioral inhibition," "self-control," and "EF" that have been employed frequently in the fields of developmental psychology, neuropsychology, and child psychopathology.

In this theory, behavioral or response inhibition, self-control or self-regulation, and EF are overlapping concepts that comprise mental faculties that are essential for the contemplation of hypothetical future states and the cross-temporal organization of behavior for the attainment of those future goals. Defining them carefully shows their conceptual overlap and interconnectedness. The stance of this theory is that the initial and overarching purposes of self-control and the EFs that support it are inherently social ones: Humans engage in choices between current and hypothetical future states within a social context, and in reciprocal social exchanges and cooperative (united) activities, often relying on cultural products as means to their survival. People must both track such prior exchanges with others, and anticipate and prepare for such future interactions with others. These purposes arose out of the group-living niche that humans occupy—social groups that comprise genetically unrelated or distantly related individuals who came to depend on forms of reciprocal exchange, or self-interested altruism, and the formation of cooperative coalitions for orchestrating non-zero-sum activities on which their survival depended. It is the essence of such coalitions aimed at joint non-zero-sum activities that they attain economic and other survival benefits that cannot be achieved by individuals acting alone or purely selfishly as in zero-sum interactions (Wright, 2001). From this perspective, nonsocial organisms that live relatively independently of other members of their species (other than for mating/reproductive activities) do not need self-control or the EFs that permit it.

Defining Our Terms

"Response inhibition," as I use it here, refers to three overlapping yet somewhat distinct and separately measurable processes:

1. Inhibiting the initial prepotent (dominant) response to an event so as to create a delay in responding (the response is now temporarily decoupled from the stimulus that served to elicit it).
2. Interrupting an ongoing response that proves to be ineffective, thereby permitting a delay and reevaluation of the decision to continue responding (a sensitivity to error).
3. Protecting the self-directed (executive) responses that occur within these delays, as well as the goal-directed behavior they generate, from disruption by competing events and responses (interference control or resistance to distraction).

I view the first of these as the most important, for without a delay in the prepotent response (stopping), any thinking and related goal-directed actions pertinent to that situation are pointless, if they can occur at all (Barkley, 1997a; Bronowski, 1977). It is not only the response that is delayed but also the decision about a response (Bronowski, 1977). The prepotent response is that response for which immediate reinforcement (positive or negative) is available within a particular context or that has been previously associated with that response in that context (Barkley, 1997b). Both forms of reinforcement—positive and negative—must be considered in defining a response as being prepotent. For instance, while some forms of impulsive behavior function to achieve an immediate reward, others serve to escape or avoid immediate aversive, punitive, or otherwise undesirable events (negative reinforcement). Such escape/avoidance responses are just as much a part of immediate gratification as are responses that result in immediate reward. Both forms of prepo-

tent response require inhibition if EF, or thinking, and self-regulation are to occur and be effective.

I employ the definition of "self-control" that is used in behavior analysis: Self-control is a response (or series of responses) by the individual that functions to alter the probability of their subsequent response to an event and in so doing thereby changes the likelihood of a *later* consequence related to that event (Barkley, 1997a, 1997b; Kanfer & Karoly, 1972; Mischel, 1983; Mischel, Shoda, & Rodriguez, 1989; Skinner, 1953). In other words, self-control is any action by an individual directed toward changing one's behavior and therein altering future rather than merely immediate consequences. Some considered self-control to be the choice of a delayed, larger reward over a more immediate, smaller one (Ainslie, 1974; Burns & Powers, 1975; Logue, 1988; Mischel, 1983; Navarick, 1986). But this ignores the self-directed actions in which the individual must engage (e.g., visual imagery and foresight) so as to value the delayed over the immediate reward and to pursue that delayed consequence. It is useful to make explicit the four minimum steps that self-regulation requires: (1) the inhibition of the prepotent response directed toward some environmental event; (2) the directing of actions (both cognitive and motoric) toward oneself, (3) resulting in the alteration of the subsequent response from what it would have been had none of these self-directed actions been enacted [a different response will be enacted as a consequence of these self-directed actions that replaces the originally prepotent response]; and (4) a change in the likelihood of a delayed (future) consequence that arises as a function of this change in the behavior employed.

What then is EF? Neuropsychology seems to view it as largely comprising unobservable "cognitive" or mentalistic events accomplished chiefly by the PFC (Barkley, 2012a). Probably the most common definition of EF, as seen in Chapter 15, is those mental abilities needed to sustain problem solving toward a goal. The literature on EF is typified by descriptions of various other constructs thought to be included under the metaconstruct of EF, while the metaconstruct itself remains vague or undefined. For instance, literature reviews, EF scale developers, and research articles may define EF by listing its component features, such as inhibition, working memory, planning, emotional or motivational regulation, strategy development and use, flexible sequencing of actions, maintenance of behavioral set, resistance to interference, and so forth (i.e., Denckla, 1996; Gioia, Andrews-Epsy, & Isquith, 2003; Gioia, Isquith, Guy, & Kenworthy, 2000; Thorell & Nyberg, 2008), or just listing measures believed to reflect EF (Biederman et al., 2007; Huizinga, Dolan, & van der Molen, 2006). Others simply conclude that EF encompasses all future-directed behavior (Huizinga et al., 2006) or what the frontal lobes do (Stuss & Benson, 1986). The underlying theme of EFs seems to be this future orientation as proposed by Denckla (1994) and which the philosopher Daniel Dennett (1995) has called "the intentional stance."

Goal-directed behavior requires a capacity for understanding the temporal ordering of both events and the requisite responses to them (Shimamura, Janowsky, & Squire, 1990), including the hierarchical staging of behavior into arrangements of goal–subgoal components (Goel & Grafman, 1995) that seems to map onto a rostral–caudal organization of the frontal cortex (Badre, 2008). These arrangements may form part of a larger capacity for the formation of social scripts (Sirigu et al., 1995) that involve the generation of the sequential steps needed to complete a social goal. Yet all such efforts to describe the EFs fall short of providing a clear, simple, and useful operational definition of the construct (or metaconstruct). All fail to address the essential question of just what, specifically, makes a cognitive or behavioral action *executive* in nature (Barkley, 2012b).

Neuropsychology has opted for a "cognitive" or information-processing view of EF founded on the computer as a metaphor for brain–behavior functioning and rather vaguely defined constructs of EF sampled exclusively by psychometric tests. This view is evident in the discussion of the competing theories of ADHD described in Chapter 15. I believe it is incorrect for various reasons, not the least of which is that *EFs comprise the principal classes of behavior in which we engage for purposes of self-regulation (changing our current behavior so as to change our future).* An executive act is any action one takes that functions to modify one's own behavior so as to likely change one's future outcomes. EFs represent a specific type or class of self-control. Such actions may be covert but need not be so to be classified as "executive" actions here. The term "covert" merely means that the outward, publicly observable (muscular–skeletal) manifestations of such behavior over the course of evolution (and human development) have become very difficult for others to detect. But these actions still occur and may still be thought of as forms of behavior, albeit behavior to modify the self. These are going to be incredibly difficult to detect using traditional psychometric tests of EF.

Developments in the technology of neuroimaging and the fine-grained recording of shifts in muscle potential now suggest that this covert behavior to change the self is capable of being measured (D'Esposito et al., 1997; Livesay, Liebke, Samaras, & Stanley, 1996; Livesay & Samaras, 1998; Ryding, Bradvik, & Ingvar, 1996). As these studies suggest, when we engage in verbal thought (covert self-speech) and imagined actions, the peripheral muscles and brain substrates ordinarily associated with the outward or public display of these same actions continue to be activated. But the movements of the peripheral muscles are being centrally suppressed, which makes them largely imperceptible to others. Yet the actions to change the self may still be detected through small changes in muscle electrical potentials at those peripheral muscle sites. In short, EF is viewed here as behavior to change the self and develops in such a way that by adulthood, the peripheral muscular–skeletal apparatus associated with such actions is being largely inhibited so as to create a private form of behavior.

The information-processing view of EF and the computer metaphor on which it is based is both a passive and theoretically sterile one; it makes no effort to understand the evolutionary basis for this complex neuropsychological adaptation considered to be EF. By asking the question "Why have EF?" one must take an evolutionary stance to the construct. Only evolution can explain the origin of complex adaptations. Those adaptations arise in species to address adaptive problems they experience in the particular niche in which they have evolved. It forces one to ask what are those problems and opportunities the EF system evolved to address and exploit. In so doing, it also provides valuable insights into what functions may be lost in those who have impairment in EF.

Linkage of Inhibition, EF, and Self-Regulation

The conceptual linkage of inhibition with self-regulation and of both of these constructs with EF is obvious given the foregoing definitions. Response inhibition is a prerequisite to self-regulation because one cannot direct actions or behavior toward oneself if one has already responded impulsively to an immediate event. The EFs are the general forms or classes of self-directed actions that humans use in self-regulation. I have identified at least four such classes besides inhibition below. The EFs and the general self-regulation they create produce a net overall maximization of social consequences when considering both the immediate and delayed outcomes of certain response alternatives. Self-regulation and the EFs that comprise it, in short, function to strive to maximize future consequences over immediate ones and are instrumental to purposive, intentional behavior. As I argued earlier, the hypothetical or anticipated future toward which they are directed is a social one (Barkley, 2012b). This resembles the view of Lezak (1995), who described the EFs as "those capacities that enable a person to engage successfully in independent, purposive, self-serving behavior" (p. 42) or that of Denckla (1994), noted earlier, as attention and intention toward the future. Regrettably, neither author specified the nature of these EFs in any operational way as I have done here.

Often unstated in discussion of self-control and EFs is that they make little or no sense if there is not some means by which the individual is capable of perceiving and valuing future over immediate outcomes. This contrast between the present state and the conjectured and desired later state is the basis of human choice or decision, and hence human action, which can also be thought of as the basis for economics (the nature of human decisions to act; von Mises, 1948/1990). In short, if there is no sense of the future, there is no choice to be made and therefore no self-control because there is no need for it. A long-term outcome may have greater reward value than a short-term reward if the two are compared to each other without regard to time. But arranged temporally as they are, the reward value of the long-term outcome will be hyperbolically discounted as a function of the length of the temporal delay involved to get it (Mazur, 1993). Humans demonstrate a remarkable shift over the first three decades of life toward a greater preference for larger delayed rewards over smaller more immediate ones (Green, Meyerson, Lichtman, Rosen, & Fry, 1996). Humans discount future outcomes less steeply in older individuals in comparison to younger individuals as well as when both are compared to other species. As noted earlier, this requires some neuropsychological capacity to sense the future or the later desired outcome, that is, to construct hypothetical futures, particularly for social consequences. It simultaneously involves weighing alternative responses and their temporally proximal and distal outcomes—a calculation of risk–benefit ratios over time. Some neuropsychological mechanism(s) must have evolved that permitted this relatively rapid construction of hypothetical social futures, while simultaneously engaging in an economic analysis intertemporally or across time, contrasting immediate versus delayed outcomes. With-

out such an evolved mental mechanism, self-control would not occur. As I indicate below, the first EF to develop in children provides the capacity for just such a cross-temporal economic spreadsheet: It is visual imagery. Imagery offers a means of iconically representing past transactions and recalling them as needed in evaluating the ongoing stream of social interactions in which an individual participates.

A THEORY OF EF AND SELF-CONTROL

I originally proposed that humans have at least five means of self-control, that is, five classes of action that they direct toward themselves to change themselves to improve their future. The details of this original "hybrid" model of EF can be found in previous publications (Barkley, 1997a, 1997b), along with the evidence that seems to support their existence. That evidence comes from developmental psychology, neuropsychological studies into the underlying factors or dimensions of EF, and neuroimaging research on the apparent localization of these EFs, largely within the prefrontal lobes. It also comes from a substantial amount of research on EF in children and adults with ADHD, a disorder of inhibition and EF that originates in the prefrontal–striatal–cerebellar networks (Bush et al., 2005; Hutchinson et al., 2008; Mackie et al., 2007; Paloyelis, Mehta, Kuntsi, & Asherson, 2007; Valera, Faraone, Murray, & Seidman, 2007).

The initial structure of this model (Barkley, 1994) was taken from Bronowski (1977), who first proposed it in his discussion of the unique properties of human language that he attributed to the PFC. I further elaborated this framework by drawing heavily from Fuster's (1995, 1997) insights into the functioning of the PFC. To this, I added the findings of Goldman-Rakic (1995) and others on working memory, and also that of Damasio (1994, 1995) on the somatic marker system and the rapid economic (motivational) analysis of hypothetical outcomes it affords. The original model of EFs is thereby a "hybrid" one. I have since broadened this model slightly by separating out a likely sixth EF that represents self-awareness, or the self-direction of attention (Barkley, 2012b). That function was originally placed within the nonverbal working memory system, described as self-directed sensing and largely comprised of visual imagery (Barkley, 1997b).

In both the original and this slightly revised model, inhibition sets the occasion for the occurrence of the other EFs and provides the protection from interference that they will require so as to construct hypothetical futures and direct behavior toward them. Despite being relatively distinct, the inhibitory functions and the other five EFs are interactive in their natural state and share a common purpose. That purpose is to "internalize" or make private certain self-directed behavior so as to anticipate and prepare for the future, especially the social future. I discuss later why such self-directed behaviors had to become covert in form. For now, the ultimate utility of the EFs is to maximize net long-term versus short-term social outcomes.

I view inhibition and the other EFs as developing by a common process. That process was borrowed from Vygotsky's theory for the internalization of speech (Diaz & Berk, 1992; Vygotsky, 1978, 1987; Vygotsky & Luria, 1994), which I proposed as being the basis for the verbal working memory system of EF. I then proposed that such a process of internalization of self-directed action was a more general one and so extended it to the other EFs. All are now seen as forms of behavior that become self-directed, self-guiding, and eventually covert or internalized. All EFs represent private, covert forms of behavior. I proposed that at one time in early child development (and in human evolution), they were entirely publicly observable and initially directed toward others and the external world at large. With maturation, this outer-directed behavior is turned on the self as a means to change and control one's own behavior and guide it toward a future desired state.

Such self-directed behavior then becomes increasingly less observable to others as the suppression of the public muscular–skeletal aspects of the behavior progresses. This progressively greater capacity to suppress the publicly observable aspects of behavior is what I mean here by the terms "covert, privatized, or internalized." Privatization permits the brain functions that allow those initially publicly observable, self-directed actions to continue unabated while the peripheral expressions of those brain activities are being largely inhibited from public display. In other words, the brain activities that create self-directed action are prevented from being released into the spinal column for execution and therefore remain in the brain. A private form of self-directed activities arises and probably represents the conscious mind. This prevention of externally executed behavior most likely involves an inhibitory switching mechanism, probably in the basal ganglia and associated neural networks (see Saint-Cyr, 2003), which serves to suppress the execution of motor ac-

tions during cortical preparations to act. The process of privatization grants the individual a capacity to generate actions in the brain (mind) without acting them out in the environment, thus forming a type of simulation of behavioral trials. Perhaps it also explains why disruption of the basal ganglia, as seen in Tourette syndrome, results in an involuntary release of cortically activated motor and verbal behavior. Regardless of the neurological mechanism responsible for privatization (an interesting research question in its own right), it creates a conscious private or mental life for the individual, one based on private sensorimotor and linguistic activities. The individual possesses a set of six "mind tools" that can be used privately or cognitively for mentally simulating reality and their potential actions within it. Mental simulations are imaginary constructions that can be used to contemplate and mentally test out various rearrangements of the material world and one's own behavior within it before selecting the one that seemingly best achieves the desired state or its intermediary states.

Note here that the EFs, and the self-regulation toward the future they permit, involve three important processes that arise during human development: *self-direction, internalization,* and *self-regulation*. All three seem deserving of far more research attention in developmental psychology than they have received to date. Although I discussed self-direction and internalization (privatization) earlier, it is important to note that these are of little consequence unless the self-directed actions come to actually regulate the behavior of the individual. Such self-regulation involves not only effectively stopping or precluding the initial automatic response to events but also developing and effectively guiding subsequent action plans toward the desired later state (the future). As Vygotsky and Luria (1994) have noted, for instance, speech may become self-directed in early childhood but may not have any effective guiding or controlling function over behavior until age 5 years or older. This self-guiding or self-controlling function of self-directed actions seems to overlap with the progressive internalization of that mode of self-directed action. Below, I briefly describe the six forms of EF or self-directed actions as portrayed in my most recent textbook on this theory (Barkley, 2012b).

Self-Directed Attention (Self-Awareness)

Self-awareness is vital as the starting point for EF. It has been acknowledged so in prior reviews of PFC functions because the pinnacle EF or central executive and may arise out of more than just self-directed sensing. The self-direction of attention comes to create self-awareness. Logic alone requires that it precede the existence of the other EF components because they all presuppose a sense of self. One cannot direct an action back on the self for self-regulation if one has no sense of self. It must be the first to arise in development, and it may well be the most important because it serves as a precursor to all other forms of self-regulation. It is here that the individual becomes conscious or aware of the entirety of his or her internal and external states, drives, wants, and actions, and so achieves an organized, integrated unity or sense of self. Its importance explains why I have now separated it out to form a separate EF in this most recent version of my theory.

Self-Restraint (Executive Inhibition)

The second EF component in importance must be self-restraint or executive inhibition. This is so because no further egoistic actions can occur until the individual ceases directing action toward the environment, however briefly. One cannot respond to the environment with action and self-direct an action simultaneously. There is a *separation* of the external event from the eventual sensorimotor responses that will be enacted in response to it. This creates a decoupling of the stimulus from its response that was typical of the earlier, automatic form of behavior so well studied in operant conditioning. The capacity to delay action likely codevelops with the capacity to prolong the sensory impression of the stimulus (see below) because neurons for both appear to be adjacent in the PFC (Goldman-Rakic, 1995). This separation or decoupling requires inhibition of the prepotent motor response that such sensory events typically generate at the automatic level of brain functioning This appears to be largely localized to the frontal–striatal circuitry and an indirect routing aspect of the basal ganglia (Saint-Cyr, 2003). It would also require inhibition of attention to unrelated events and their sensory impressions (a resistance to distraction) at the time the primary sensory representation is being prolonged or reimagined.

For goal-directed behavior to arise, attention must be shifted away from the moment and external reality and turned toward the self (the first EF, discussed earlier) and the mentally contemplated future for that self—the goal (the next EF, discussed below). The temporal gap created by the deferred response provides the

opportunity for further self-regulatory actions and the eventual goal-directed behavior they will actually initiate.

Sensing to the Self (Nonverbal Working Memory)

This EF has been called nonverbal working memory. It is akin to Baddeley's (1986; Baddeley & Hitch, 1994) visual–spatial sketchpad in his information-processing rendition of working memory. I prefer to understand it in Vygotsky-like terms as a type of self-directed human action. In this case, it is largely the use of self-directed vision that is visual imagery. A person literally resees images, typically engaging the right dorsolateral PFC, as well as the posterior visual association areas (D'Esposito et al., 1995, 1997). This is done not with just vision but with all of the senses, such as rehearing, resmelling, refeeling, and so forth. Done jointly, the mental representation comprises not just an image alone but a fully integrated sensory representation. This is essentially the privatization of self-directed sensorimotor actions—it is sensing and acting to the self (literally, resensing to the self). The most important of the senses to humans are vision and hearing, so this EF may chiefly comprise visual imagery and covert audition—reseeing and rehearing to the self.

This EF has both retrospective (sensory or resensing) and prospective (preparatory motor) elements (Fuster, 1997; Goldman-Rakic, 1995). Here, then, arises the mental module for sensing the hypothetical future from the experienced past. This serves to generate the private or mental representations (images, auditions, etc.) that bridge the cross-temporal elements within a contingency arrangement (event–response–outcome) that is so crucial for self-control across time toward the future. Pierce (1897/1955), and later Deacon (1997) and Donald (1993), noted that such private sensorimotor representations are prerequisites for symbolization and language because they form the mental icons to which otherwise arbitrary terms in language refer.

The person can now use this reimagining of past sensorimotor events to self-elicit action in the absence of the primary stimulus typically eliciting it. This permits the person to reenact the behavior repeatedly. Behavioral reenactments, or mimesis, being triggered by mental representations of sensory stimuli, can become means of practice and rehearsal. They can become means to perfect further the response to the actual environmental event the next time it appears in that external world. This initial rehearsal is publicly observable, yet it is incredibly advantageous for improving subsequent behavior. Humans often demonstrate public rehearsals of actions they intend to use later under real circumstances. When this behavioral rehearsal becomes internalized, it is a means for the private simulation of human actions and may be the basis for human rituals (Rossano, 2011). This internalization of sensorimotor actions also explains the importance of the cerebellum in EF or "higher cognition" (Diamond, 2000; Houk & Wise, 1995); given its importance for the execution of public behavior it would be just as essential for its private simulation. Some research suggests that such "working memory" may be the function that provides the foundation for some of the other EF components, such as the mental manipulation of information and problem solving (McCabe, Rodeiger, McDaniel, Balota, & Hambrick, 2010).

The first three EF components—self-awareness, self-restraint (inhibition), and self-directed sensorimotor actions (imagination)—create the base of a unified EF system. How or why each exists cannot be logically understood without reference to the other two. Sensing the future cannot arise without self-awareness and self-inhibition, but the reason the latter two functions exist is to facilitate the third; alone, these two would have no functional significance. Why be aware of and even stop oneself from doing something if there is no other course of action to be contemplated? The remaining EF components arise out of this triadic foundation—the development of speech and its eventual self-direction, the self-regulation of emotion and motivation, and reconstitution or play to the self for planning and goal-directed innovation.

Speech to the Self (Verbal Working Memory)

The fourth EF has been called verbal working memory. It is similar to Baddeley's (1986) construct with the same name. This EF can be better understood using, once again, Vygotsky's model of the developmental internalization of speech. The individual is capable of activating the central or cortical aspects of speech without engaging the actual motor execution of that speech. One can literally talk to oneself without moving the face or activating the larynx to any appreciable degree. The individual is using the same speech areas of the brain for this activity that he or she uses when speaking publicly, except that the primary motor areas related to external or public speech are being suppressed (Ryding et al., 1996). Such self-speech permits self-description and reflection, self-instruction, self-

questioning, and problem solving, as well as the invention of rules and metarules to be applied to oneself (Diaz & Berk, 1992). It therefore not only contributes to a major form of self-control via language, but it also likely provides the basis for moral conduct (internalizing socially prescribed rules of conduct). In addition, it makes possible reading comprehension through silent reading (self-speech) that must be held in mind for the extraction of its semantic (nonverbal) content.

Self-Directed Appraisal (Emotion/Motivation to the Self)

This EF may occur initially as a mere consequence of the first four EFs (discussed earlier). Those EFs involve covertly representing forms of visual and verbal information to oneself. These mentally represented events have associated affective and motivational properties or valences, which Damasio (1994, 1995) called "somatic markers." Initially those affective and motivational valences may have publicly visible counterparts, such as emotional displays when we laugh out loud in response to a mentally visualized incident. Eventually, though, these affective displays are kept private or covert in form. Here originates, I believe, the next EF of private affect and its motivational properties. In brief, it is feeling (emoting/motivating) to the self. This model argues that this EF forms the wellspring of intrinsic motivation (willpower) so necessary to support future-directed behavior, especially across large delays in schedules of reinforcement or when external consequences for such future-directed action are otherwise not available in the immediate context. It provides the motivational basis for persistence (sustained attention) toward attainment of future goals.

Once humans developed capacities to inhibit prepotent reactions to events and to contemplate "the later" against "the now," they automatically encountered choice. Such a choice is inherently a conflict. A future state is being contrasted against the current one, and a decision must be made as to which is to be pursued. All such comparisons involve a calculation of costs and benefits for the individual. The goals (changes in uneasiness) to be gained from the possible choices are computed (valued) and then compared. The one producing a net maximization of value for the individual is then pursued. The decision is personal, reflecting the individual's appraisal of the subjective use value of the means and ends. Therefore, a mental mechanism for the conscious executive appraisal involved in these comparisons must exist. It is argued here to be a component of EF. It provides for a rapid cost–benefit analysis of means and ends, and appears to arise from bidirectional networks linking the dorsolateral PFC, orbitofrontal cortex, anterior cingulated cortex, and amygdala (hence, the limbic system; Damasio, 1994, 1995; Etkin, Egner, Peraza, Kandel, & Hirsch, 2006; Ochsner & Gross, 2005, 2008; Ochsner et al., 2009; Rushworth, Behrens, Rudebeck, & Walton, 2007).

The first four EF components can also be used for emotional self-regulation; the two step-process of initially inhibiting strong emotions, then down-regulating or otherwise moderating them (Ochsner & Gross, 2005, 2008; Ochsner et al., 2009). Various disorders of the PFC result in an inability to self-regulate displays of strong emotions that are not in the longer-term self-interests of the individual. Raw emotional displays, unmodified relative to their appropriateness to a given social context and poorly moderated, are likely to impair social relationships, if not lead to outright rejection by others, as discussed in Chapter 3. Humans possess means by which they can inhibit the initial displays of strong emotions. They employ these other EF components to replace the initial strong emotion with alternative emotional responses that are more consistent with social demands and the individual's goals and longer-term welfare. For humans, emotions are not merely environmentally provoked reactions that must be dealt with appropriately; they are also states that can be created de novo as needed in the service of one's goals (Izard, Stark, Trentacosta, & Schultz, 2008). The self-regulation of emotion is probably based on the same bidirectional network that underlies the appraisal system for mentally contemplated goals and their means (discussed earlier).

Because emotions are motivational states, this EF of creating private emotion and motivation probably also provides the capacity for self-motivation—the drive states needed to initiate and sustain action toward the future. Research suggests that the emotion regulation and motivation regulation aspects of this unit may be partially separable both neuroanatomically (Murray, 2007; Rushworth et al., 2007) and behaviorally (Barkley, 2011b). Even so, they are treated as a single unit here for both simplicity of presentation and because of their substantial neurological and functional overlap.

Self-Play (Reconstitution)

The last EF is self-directed private (covert) play, or reconstitution, a process of initially taking apart (analysis) then recombining information (synthesis) to form

novel and potentially useful recombinations. "Fluency," "flexibililty," and "generativity" are other terms by which this EF is known in neuropsychology. This EF serves to generate a diversity of new combinations of behavioral units out of old ones and is therefore the source of behavioral innovation (problem solving) during goal-directed actions. Analysis and synthesis are applied to the mental contents being held in the working memory systems (self-sensing and self-speech systems). In analysis, old behavior sequences are broken down into smaller units. These units are then recombined (synthesized) into new sequences that can be tested against the requirements of the problem to be solved (Corballis, 1989; Fuster, 1997). It is hypothesized to arise from the internalization of play (both sensorymotor and symbolic) and serves to create novel future-directed actions. Such novel actions will be needed when obstacles to a goal are encountered (problems) in order to overcome them and successfully attain the goal. The generation of such novel responses has been shown to be especially problematic for patients with frontal lobe injuries (Godefroy & Rosseaux, 1997). It has been blamed on their inability to form and sustain mental referents from instructions so as to manipulate them to discover a means to achieve a goal. And that, as I have argued, is simply covert play to one's self.

This EF may be subdivided further into verbal and nonverbal components comparable to the working memory system (verbal or nonverbal) on which it acts. Neuroimaging studies suggest that verbal and nonverbal (design) fluencies are mediated by separate (left vs. right) regions of the dorsolateral PFC (Lee et al., 1997; Stuss et al., 1998). This implies that a bivariate subdivision of this EF might be useful. However, prior factor-analytic studies of EF measures have found only a single dimension representing both verbal and nonverbal fluency (Levin et al., 1996).

Problem solving is often needed to begin a process of planning goal-directed action. That is because goal-directed action most often arises when there is a conflict between a present state (what is) and a desired state (what is wanted). Such a conflict, by definition, is a problem. Hence problem solving may be invoked at the very start of creating goal-directed action. The problem-solving capacity provides for mentally creating and testing options for their likely ability to achieve the goal. This is predominantly a process of action fluency (Piatt, Fields, Paolo, & Troster, 1999), not so much object naming fluency. In short, the individual can conceive of a variety of ways of doing something and select that which most likely will attain the goal given his or her experience. Evidence suggests that the orbitofrontal cortex and anterior cingulate cortex play distinct yet interacting roles in this process of generating and testing out a variety of response options (Rushworth et al., 2007).

Further Implications of the Theory

Each EF is also hypothesized to contribute to the following developmental shifts in the sources of control over human behavior:

- From external events to mental representations related to those events.
- From immediate reinforcement to delayed gratification.
- From the temporal now to the conjectured social future.
- From control by others to control by the self.
- From largely noncultural to cultural (shared information) means.

With maturation, the individual progressively comes to be guided more by covert representations that permit self-control, deferred gratification, and goal-directed actions toward conjectured social futures often using social and cultural means (Barkley, 2012b).

Briefly put, the six EFs provide an exceptionally powerful set of mind tools that greatly facilitate adaptive functioning in anticipation of the future. In a sense, these EFs permit the private simulation of actions within specific settings that can be tested out mentally for their probable consequences (somatic markers) before a response is selected for eventual public execution. This, as Karl Popper noted, allows our ideas to die in our place should they prove not to be correct or suitable in such mental simulations (cited in Dennett, 1995). It constitutes a form of mental trial-and-error learning that is devoid of real-world consequences for one's mistakes.

The Social Importance of EF and Self-Regulation

Among several possible adaptive advantages of self-control and an executive system that might be useful are the following three (see Barkley, 2001, for others), which I subsequently used to create a multilevel (hierarchical) model of EF as an extended phenotype (to be described later).

Imitation (Vicarious) Learning

Though rarely mentioned in discussions of EF, particularly those of nonverbal working memory, the capacity to engage in imitation, particularly delayed imitation, is probably one of the most important capacities for a group-living social species such as humans. Many species, as Darwin (1871/1992) noted, are capable of mimicry or even immediate imitation of particular acts. For many reasons, immediate mimicry or imitation is a good adaptive strategy, and other species have converged on it. Delayed imitation, however, especially in generalized form is a notably human achievement (Donald, 1991, 1993). Our species has an early-developing instinct, indeed, nearly a compulsion to do it (Meltzoff, 1988).

Imitation, especially delayed imitation, clearly depends on three cognitive capacities: (1) the inhibition of prepotent responses, (2) an evolved mental mechanism for carrying past sensory perceptions of others' behavior forward in time across a delay interval, and (3) a capacity to construct motor responses on the basis of those mentally reperceived actions of others. The latter two requirements are obviously the retrospective and prospective aspects of the nonverbal working memory system. Initially, it seems likely that the initial delay between the act and its imitation was undoubtedly brief, perhaps owing to the initially fleeting afterimages derived from primary sensory impressions. Regardless of how it originated, the capacity to inhibit prepotent responses and to carry forward in time past perceptions (retrospection) that create the template for the later imitative motor act (prospection) form the foundation of self-regulation, as noted earlier. The more highly developed the nonverbal working memory capacity, the lengthier and more hierarchically complex the sequence of actions that can be held in mind for later imitation, the longer the delay over which it can be carried into the future, and the greater the demand for response inhibition during the period when such imitative responses are being programmed and eventually executed. The more complex the sequence, the more its syntax and timing must also be held in mind. The holding of a sequence of events in mind may also form the beginnings of a subjective or psychological sense of time (Davies, 1995).

Imitation involves the reproduction of another person's behavior following its observation. Vicarious learning is a more advanced form of imitation. It involves not only imitation (doing what gained reinforcement for others) but also inverse imitation, that is, not doing what another person does (avoiding what actions led to aversive, painful, or even mortal outcomes for others). Note the requirement for oppositional action involved in vicarious learning. The amount of social learning that occurs in humans through imitation and vicarious learning is substantial, to say the least. It is undoubtedly far more than the learning that could occur by operant conditioning or by trial-and-error learning alone. Imitation develops very early in childhood; in fact, rudiments of it are present in infants by age 9 months (Meltzoff, 1988). Its development seems to parallel the development of representational memory, especially visual imagery (Kopp, 1982; Meltzoff, 1988).

There is no other species that comes close to the human capacity for this form of learning. Evolutionary theory demands that explanations for such adaptations initially be considered from a self-interested perspective (the good of the individual or his or her genes) before giving credence to explanations at the group level (for the good of others) (Dawkins, 1976, 1997; Williams, 1966/1996). From that self-interested perspective, vicarious learning constitutes a form of *experiential plagiarism* or ideational theft that is clearly in the imitator's self-interests. Through imitation and vicarious learning, the individual profits from the experiences that others may have with real-world contingencies, without the costs, penalties, pitfalls, morbidity, and mortality that can be associated with those contingencies. The vicarious learner gains a considerable adaptive advantage in a group-living species because he or she appropriates the experience of another person at minimal cost to him- or herself. From that vantage point, imitation and vicarious learning are incredibly useful self-interested adaptations.

Imitation also provides for the development of tool manufacture, as well as social communication via gesture (Blackmore, 1999; Donald, 1993). The origin of imitation and later vicarious learning would have set up selection pressure for humans to evolve a covert form of behavioral rehearsal so as to keep others from copying (plagiarizing) their behavior while it was being rehearsed and further perfected. Though speculative, this may have initiated the need for the internalization or privatization of one's behavior-to-the-self that became the basis for the EFs. Interestingly, this resensing of one's past experiences may also be the origin of "autonoetic awareness," or the awareness of self across time (Barkley, 1997a; Kopp, 1982; Wheeler, Stuss, & Tulving, 1997).

Self-Reliance and Social Self-Defense

The basic background of any social ecology is competitive and predatory, whether it is between members of different species or those of the same species. Self-interest prevails in all species as an inherent feature of both genetics itself (self-replication; Dawkins, 1976) and of the organisms created by those genes (survival, reproduction, and personal welfare). This theory of EF accepts and respects intraspecies competition and predation as the background of humans, just as it exists for all other organisms. Competition needs little explaining or justification; it is self-evident across evolution in all species. What requires explaining is the possibility that later levels of reciprocity and cooperation may emerge out of such interpersonal competition and inherent short-term self-interest (Axelrod, 1997).

Social existence guarantees competition for limited resources. Competition means that individuals will attempt to influence others for their own one-sided benefit and self-interests. It is social parasitism or predation. This occurs very frequently in the biological world within and between species. Human communication systems such as gesture and language serve just this purpose—to alter the mental representations and behavior of others for one's own ends or purposes. Not all efforts at social influence are parasitic or detrimental to the person targeted. For example, communication among genetically related individuals tends to promote inclusive fitness of the shared genotype (genetic self-interests), as would be expected from selfish gene theory (Dawkins, 1976). But we must accept the fact that in the biological world, many such communicative systems are parasitic or one-sided in promoting only the self-interests of the signaler to the detriment of the recipient. Thus, some means of resisting, repelling, or just delaying and further evaluating such influences are essential to the individual's welfare.

Mental mechanisms, such as self-regulation and other forms of resistance to unwanted, detrimental social influences, are needed to counter this manipulative aspect of human social life. The efforts of people to influence others into giving up valuable resources are evident not only in advertising, marketing, and salesmanship, but also in such activities as theft, embezzlement, and financial scams. Advances in telecommunications such as the Internet have only broadened the opportunity to communicate, sell, persuade, swindle, and otherwise prey on others for one's own one-sided benefit. Given this background of predation and competition, the EF system may have evolved in part to permit greater self-reliance (adaptive functioning) and social self-defense.

Reciprocal Altruism (Social Exchange) and Coalition Formation (Cooperation)

Among human universal social attributes, reciprocal altruism with nonkin (others with whom one does not share genetic self-interest) stands out as one of our most unique behavioral features relative to other species. Humans exchange goods or services now for other ones later, despite having no common genetic self-interests with those with whom they engage in such exchanges. They do it nearly all the time, forming the backbone of human economic systems (Ridley, 1997). While Williams (1966/1996) prefers the less emotive term "social donors" to describe those engaged in this practice to Haldane's term "altruism," the point is the same (see Williams, 1966/1996, for Haldane's view). Genetically unrelated humans live within a social group and frequently exchange benefits and costs now for benefits and costs later. The exchanges are reciprocated, and those reciprocations are delayed in time. Such a delayed exchange of costs and benefits between nonkin constitutes a promise or a social contract. Darwin (see Williams, 1966/1996, p. 94) was apparently well aware of the fact that a group-living species might well come to evolve a form of social exchange (what he termed "the lowly motive"). He also appreciated that such an exchange was an important factor to consider in understanding the evolution of not only human mental functions but also friendship and culture.

Reciprocal exchange, particularly when it is delayed, constitutes a prime candidate for one of the initial adaptive PFC functions. It requires both inhibition and a representational memory system for sensing past and future occasions—the foundation of self-control, as discussed earlier. Just as with any other form of adaptation, the mental mechanisms affording self-control exact a biological cost to the individual. That cost must be outweighed by some benefit, and such a benefit need not be for the good of the species or even the group in order to evolve. It must be for the good of the individual and specifically the individual's genes. Yet humans voluntarily subject themselves to periods of self-deprivation (e.g., sharing or even dieting), deferred gratification (e.g., saving, investing, and education), and even aversiveness (e.g., getting inoculations against diseases). From the standpoint of selfish gene theory and its re-

lated kin selection theory (Ridley, 1997), these actions make little sense in the context of the moment. According to these theories, individuals should seek as much benefit and advantage now for themselves and their genetic relatives, if only because others will do so if they do not, leaving the former at a disadvantage. Such personal greed is certainly evident in humans and can result in a sort of "tragedy of the commons," whereby publicly held resources are depleted by self-interested individuals even if the long-term depletion of the asset is not in their best interests (Ridley, 1997). In such instances, acts of self-control are losing strategies. The costs of reciprocal altruism and self-control can be substantial, and the individual employing them can easily be cheated or outcompeted in acquiring the immediate resources. The existence of reciprocal altruism requires that there be some advantage to the self-interested motives of those individuals involved in those exchanges.

Delayed reciprocal exchange requires a capacity to perceive long-term sequences of events and their outcomes for oneself and for others with whom one is trading. Even rudimentary, little delayed forms of reciprocal exchange would create selection pressures for the evolution of an increasingly longer sense of past and future (nonverbal working memory) to evaluate those longer-term consequences of the trade. It has been suggested that in the environment of prehistoric humans, such as the grasslands of central Africa, food sources and other resources showed cyclical patterns of availability, as they do even today (Ridley, 1997). Periods of plenty were punctuated by periods of famine. Under such conditions of large swings in resource availability, sharing, and its associated reciprocal exchange would have brought great advantage to individuals living in groups as a means of mediating or modulating the personal risks and costs associated with these cycles of feast and famine. In such circumstances, it would pay those who had been lucky in hunting or scavenging to give up some of their excess bounty to others in exchange for the same sort of reciprocation later, when those others were more fortunate and the previously successful hunters were not. Like a group insurance pool today, individuals would chip in resources they did not require at the moment to those who needed them in exchange for the same treatment later in their own time of need—resulting in a sort of Golden Rule. A group of selfish cooperators would evolve provided that the consequences for cheating on the contracts were made sufficiently harsh by the group so as to make reneging on those exchanges costly (Barkley, 2012b; Ridley, 1997). Indeed, in some modern hunter–gatherer groups, such as Eskimos, it seems that on some occasions, successful hunters who failed to share when their turn came could lose their lives (Dugatkin, 1999; Ridley, 1997).

In essence, social exchange requires a sort of mental spreadsheet that calculates temporal sequences of exchange for which the executive system seems ideally designed. Where social exchanges occur frequently between two selfish cooperators, those exchanges can become the foundation for building not only friendships but also social coalitions for cooperating with or acting against other individuals and coalitions. Such coalitions also may accomplish goals that no individual reciprocator could do alone (cooperative ventures). The EFs seem to be well-designed mental modules for mediating this adaptive strategy of social exchange and cooperative coalition formation for greater adaptive advantage. If so, the implication is that one of the major detrimental effects of ADHD (and of other frontal lobe injuries) for daily adaptive functioning is the diminution of the capacity for effective social exchange and its attendant cooperative coalition formation in daily social life.

EXTENDING THE MODEL INTO EVERYDAY HUMAN LIFE ACTIVITIES: THE EXTENDED PHENOTYPE

In the most recent revision and expansion of this theory of EF, I have cast all of the previously discussed characteristics in the form of a multilevel hierarchy (Barkley, 2012a). I did so in large part to examine how the initial, self-directed, and cognitive levels of the EFs extend outward like a series of concentric rings to impact important major domains (levels) of human social activity. As I noted earlier and discussed in detail elsewhere (Barkley, 2012b), the human EF system does not exist to sort cards, detect between X's from O's on computer screens (as in continuous-performance tests), recite digit spans backwards, or generate multiple words with the initial letter F, as may often occur in neuropsychological batteries purporting to measure EF (see Chapters 4 and 10). These are not the most important purposes of the EF system because they are not the crucial faculties lost in people with frontal lobe disorders, such as the classic case of Phineas Gage (Harlow, 1848, 1868). Using the notion of an extended phenotype developed by Dawkins (1982) in understanding

the evolution of various adaptations in species' phenotypes, I have attempted to show how the EF mind tools can produce effects that radiate outward, like a series of concentric rings from a drop in a water basin, into physical, social, and cultural space to produce important evolutionary (selective) effects at a considerable distance from the individual. Such effects-at-a-distance can be considered part of the species' phenotype if they feed back eventually to impact the survival, reproductive fitness, and general welfare of the individual.

The new model is depicted in Figure 16.1. Briefly stated, the theory now includes the self-directed, internalized EFs described earlier as forming the initial *instrumental–self-directed* level, so named because the EFs are not merely self-directed as an end in themselves but are a means to other ends (and so are instrumental). As they develop, they contribute to survival benefits at the next level of the extended phenotype of EF, the *methodical–self-reliant* level, so labeled because the EFs are used to create short-chain behaviors that accomplish near-term goals (methods) having to do with immediate survival, welfare, independence from others, and social self-defense. This level permeates or comprises daily human adaptive functioning.

Over time, there emerges a third ring or level of extended phenotypic effects that have to do with social reciprocity and exchange, which form the basis of human friendships and extended social networks. This level is entitled the *tactical–reciprocal* level, for obvious reasons. The term "tactical" here implies not only mili-

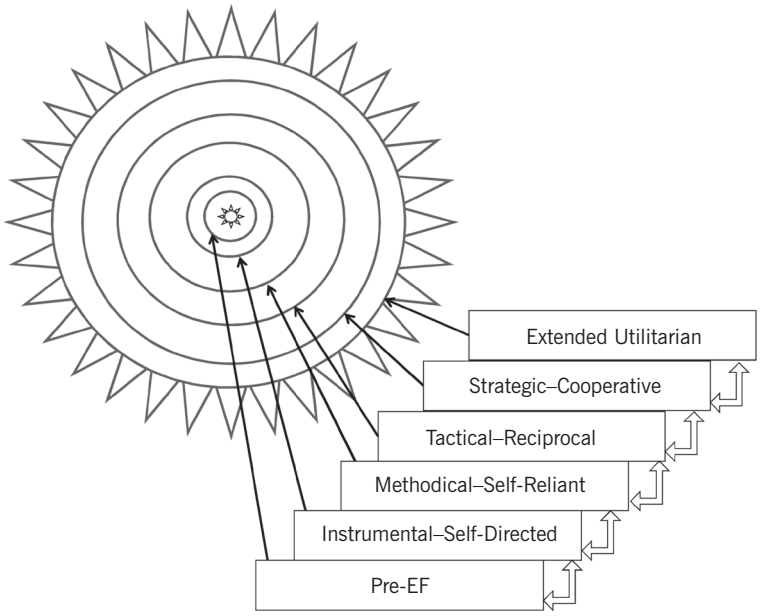

FIGURE 16.1. Two different ways of illustrating the extended EF phenotype. The concentric rings at the left indicate the outwardly radiating nature of the phenotype (Pre-EF, Instrumental–Self-Directed, Methodical–Self-Reliant, Tactical–Reciprocal, Strategic–Cooperative). The final sunburst edging of this diagram reflects the extended consequences or utility of employing EF across these levels and one's lifetime, known as the Extended Utilitarian zone. The stacked boxes at the right indicate the hierarchical arrangement of these six phenotypic levels and the ultimate utility (effects-at-a-distance) of using them. The bidirectional arrows to the right of each box are intended to convey the bidirectional flow of information between the levels. Information from the lower level flows upward to the higher level, while management of the lower level may be exerted downward by the next higher level. From R. A. Barkley (2012). Copyright 2012 by The Guilford Press. Reprinted by permission.

tarily adjusting one's actions as a function of the actions of others but also represents the nesting of methods (short-chain, goal-directed behavior) into longer-chain activities that accomplish goals further distant in time.

With further development, I argue that another, higher level or extended ring of the EF phenotype emerges in which people join together to accomplish joint goals that none could do alone and so share in the outcomes of those joint activities. Cooperative ventures such as these require the existence of very special situational factors that cause them to emerge and are rather fragile, deteriorating back to lower levels of self-interested reciprocal or even predatory forms of social behavior if those conditions deteriorate (see Barkley, 2012b, for these conditions). This level of the extended EF phenotype is called the *strategic–cooperative* level, not only because it is the level or concentric zone in which cooperation takes place but also because it incorporates the two meanings of the word "strategic." In its obvious sense, this term refers to sets of tactics being used to accomplish a longer-term goal in the context of others and using others, which form strategies. In another sense, the term means a nested set of tactics, each of which accomplishes an intermediate term goal that when strung together can accomplish far longer-term goals.

Note then not only the social and cultural aspects of these levels of concentric phenotypic rings of effects but also the nesting of instrumental cognitive, self-directed actions within strategies that result in short-term within midterm within longer-term goals that create highly complex forms of human activities. Within the *strategic–cooperative* level may even arise under special sociocultural conditions a higher level of human EF activities, known as *principled–mutualistic,* in which not only groups of individuals accomplish longer-term shared goals (and benefits) but also each individual looks out for the welfare of others in the group, and much higher level rules guide social and cultural conduct (principles), such as abstract laws, norms, rules, and so forth.

At each new level of EF, consequences (advantages mostly) accrue to the individual; otherwise, the extension into that zone of effects would not have happened or it would have been a mere by-product with no selection effect in evolution and therefore would not be considered part of the extended EF phenotype at all. Recall from earlier discussion that by-products (extended effects) of an adaptation must have effects that alter survival, reproduction, inclusive fitness, or self-interests generally or they are not considered extended phenotypic effects. Therefore, each new ring of the extended phenotype is surrounded by a feedback zone of adaptive effects. In short, extended phenotypes produce extended consequences. I refer to this outermost zone of consequences as the *extended utilitarian* level. These consequences are part of the phenotype because they are the feedback mechanism—the consequences for human survival and welfare as a result of using EF.

Should individuals have developmental or acquired disorders of the PFC and the EF system it supports, any and all of these concentric or hierarchically arranged levels of this EF extended phenotype are put at risk for dysfunction.

Implications of the Theory for ADHD

Within the *instrumental–self-directed* level of EF, a central set of problems occurs in those with ADHD. This is the capacity for behavioral inhibition (Barkley, 1997a; Nigg, 2001; Quay, 1997), self-awareness, and working memory (Rapport et al., 2008). Given the importance of such self-awareness, self-restraint, and self-directed sensing that form mental representations to the other instrumental EFs as described earlier, a deficit in any or all of these foundational EFs results in a cascade of secondary deficits into the remaining EFs. As extrapolated to those with ADHD, the model predicts that deficits in behavioral inhibition, self-awareness, and nonverbal working memory, including (1) particular forms of forgetfulness (forgetting to do things at certain critical points in time); (2) impaired ability to organize and execute actions relative to time (e.g., time management); (3) reduced hindsight and forethought; (4) a reduction in the creation of anticipatory action toward future events; along with (5) reduced self-monitoring and awareness. Consequently, the capacity for the cross-temporal organization of behavior in those with ADHD is diminished, disrupting the ability to string together complex chains of actions (instrumental–methodical–tactical–strategic) directed, over time, to an ever more distant future goal. The greater the degree to which time separates the components of the behavioral contingency (event, response, consequence), the more difficult the task will be for those with ADHD who cannot bind the contingency together across time so as to use it to govern their own behavior, as well as that of others.

Research demonstrates some of these deficits in people with ADHD, such as nonverbal working memory,

timing, and forethought (Chapters 4 and 10; also see Barkley, 1997a; Barkley et al., 2008; Frazier et al., 2004; Hervey et al., 2004). Only cursorily studied is the prediction from this theory that children with ADHD will be more delayed in making references to time, the past, and the future in their verbal interactions with others than do typically developing children in relation to their development of sense of time, hindsight, and foresight. Yet this seems to be the case (Barkley et al., 1997; Houghton, Durkin, Ang, Taylor, & Brandtman, 2011).

For those with ADHD, delayed privatization of speech should result in greater public speech (excessive talking), less verbal reflection before acting, less organized and rule-oriented self-speech, a diminished influence of self-directed speech in controlling one's own behavior, and difficulties following the rules and instructions given by others (Barkley, 1997a). Substantial evidence supports this prediction of delayed internalization of speech (Berk & Potts, 1991; Landau, Berk, & Mangione, 1996; Winsler, 1998; Winsler et al., 2000). Given that such private self-speech is a major basis for verbal working memory (Baddeley, 1986), this domain of cognitive activity also should be impaired in ADHD. Evidence suggests that this is so. Children with ADHD have difficulties with tasks such as digit span backwards, mental arithmetic, paced auditory serial addition, paired associated learning, and other tasks believed to reflect verbal working memory (Barkley, 1997a; Frazier et al., 2004; Hervey et al., 2004; Kuntsi, Oosterlaan, & Stevenson, 2001).

The impairment in the internalization and self-direction of emotion arising from ADHD leads to the following predictions, which are well supported in the review in Chapter 3. Those with ADHD should display (1) greater emotional expression in their reactions to events; (2) less objectivity in the selection of a response to an event; (3) diminished social perspective taking because they do not delay their initial emotional reaction long enough to take into account the views and needs of others; and (4) diminished ability to induce drive and motivational states in themselves in the service of goal-directed behavior. Those with ADHD remain more dependent than others on the environmental contingencies within a situation or task to determine their motivation (Barkley, 1997a).

The model further predicts that ADHD will be associated with impaired reconstitution, or self-directed play, which is evident in a diminished use of analysis and synthesis in the formation of both verbal and nonverbal responses to events. The mental capacity to visualize, manipulate, then generate multiple plans of action (options) in the service of goal-directed behavior and to select from among them those with the greatest likelihood of succeeding should therefore be reduced. This impairment in reconstitution is evident in everyday verbal fluency when the person with ADHD is required by a task or situation to assemble rapidly, accurately, and efficiently the parts of speech into messages (sentences) to accomplish the goal or requirements of the task. It is also evident in tasks in which visual information must be held in mind and manipulated to generate diverse scenarios to solve problems (Barkley, 1997a). Evidence for a deficiency in verbal and nonverbal fluency, planning, problem solving, and strategy development more generally in children with ADHD is limited, but what exists is consistent with the theory (Barkley, 1997a; Clark, Prior, & Kinsella, 2000; Klorman et al., 1999).

In general, ADHD is predicted to disrupt the various levels of this extended phenotype, depending on its severity, resulting in a constriction or even contraction of these levels that adversely impacts the five transitions noted earlier in the sources of control over behavior. The child with ADHD will be more under the control of external events than mental representations about time and the future; more influenced by others than acting to control him- or herself, pursuing immediate gratification over deferred gratification; more under the influence of the temporal now more than of the probable social futures that lie ahead; and less likely to deploy cultural methods and devices for his or her own self-regulation and goal-attainment.

From this vantage point, ADHD is not a disorder of attention, at least not relative to the moment or to the external environment, but more of a disorder of intention, that is, attention to the future and what one needs to do to prepare for its arrival. It is also a disorder of time management specifically, in that the individual manifests an inability to regulate his or her behavior relative to time and future welfare, as well as that of others at his or her developmental level. This creates a sort of temporal myopia, or time blindness, in which the individual responds to or prepares only for events that are relatively imminent, not the ones that lie further ahead in time and for which others of their age are preparing so as to be ready for their eventual arrival (Barkley, 1997a).

By implication, this view of ADHD, combined with an evolutionary perspective on the disorder, suggests that the disorder will interfere with the four larger so-

cial purposes for which EF and self-control may have evolved—vicarious learning and the adoption of culture for self-improvement and long-term welfare, self-reliance and social self-defense, reciprocal altruism (social exchange), and social cooperation. These predictions remain to be directly tested in research on children with ADHD, but the evidence reviewed in all of the prior chapters on families, education, social, occupational, and adaptive domains of functioning would be consistent with this extended phenotype model of EF as applied to ADHD.

Implications of the Theory for the Treatment of ADHD

The numerous implications of this theory relative to EF and its assessment can be found in my earlier texts (e.g., Barkley, 2012b), but of equal or even more importance are the implications of this perspective for the management of ADHD or other disorders of EF. Some of these implications from earlier texts are discussed below (Barkley, 1997a, 2006, 2012; Barkley et al., 2008).

If EF deficits are viewed as specific cognitive difficulties in tasks such as card sorting by categories and repeating digit sequences forward and backward, then rehabilitation would comprise retraining of these sets of skills and related abilities. More is to be gained by understanding that EF is self-regulation (SR) and extends as a phenotype upward through a hierarchical structure of increasingly complex behavior and outward to involve increasingly larger social networks assisted by increasingly complex cultural scaffolding. The latter perspective involves deficits in the EF/SR dimensions of self-management relative to time, self-organization and problem solving, self-restraint, self-motivation, and self-regulation of emotion, as suggested in recent EF rating scales evaluating these levels (Barkley, 2011a; Barkley & Murphy, 2011). It also includes the social activities of dyadic reciprocity, social exchange, group cooperative ventures, and even community mutualism not evident at all in the cognitive view of EF. Efforts aimed at accommodating EF deficits in time management, self-organization and problem solving, self-restraint, self-motivation, and self-regulation of emotions would not arise from the cognitive model of EF, but they *would* spring from the extended phenotype model of EF/SR and its inclusion of daily adaptive and social spheres of EF functioning.

Another distinction between traditional EF models and the extended phenotype model that is relevant to treatment arises from their widely disparate views on the origin and purpose of EFs. The traditional cognitive model sees EF as a catalog of mental modules that process information of various types. The modules then pass on their processed information to other modules, all of which appear to be routed and scheduled by some "central executive" that remains unspecified yet directs the action. How does such a view lend itself to developing treatment recommendations for a patient with EF deficits? The extended phenotype view does lead to recommendations. The instrumental EFs represent pre-EF actions that have been *self-directed and internalized* over development (and evolution) to give rise to "mental" information that is being actively held in mind so as to guide behavior across time. All recommendations flow from acknowledging that people with EF deficits such as those in ADHD, in which the self-directed and internalized form of EF is weak or deficient, cannot govern their behavior as well as others are able to do. Therefore, going backward in the developmental sequence, there is a need for greater reliance on external forms of overtly self-directed actions (e.g., out-loud verbal self-speech) and even a greater reliance on external props and prompts within the sensory fields to help facilitate self-regulation.

The psychometric view of EF sees EFs as mental modules passively processing information and exchanging it along pathways with other modules. The extended phenotype model sees EF as conscious, effortful, self-initiated, and self-directed activities that strive to modify otherwise automatic behavior so as to alter the likelihood of future consequences (longer-term goals and desires). It views these self-directed activities as largely comprising self-directed attention, self-restraint, sensorimotor action to the self using chiefly visual imagery, speech to the self, emotion to the self and self-motivation, and self-directed play, all of which make it crystal clear just what humans are doing to themselves when they engage in EF/SR. This view would encourage individuals wishing to develop or rehabilitate their EF/SR further, as in the case of ADHD, to practice the following repeatedly: self-monitoring, self-stopping, seeing the future, saying the future, feeling the future, and playing with the future so as to effectively "plan and go" toward that future.

The extended phenotype view of EF argues that the problems posed for those with EF deficits in major life activities have more to do with not using what they know at critical points of performance in their natural environments than with not knowing what to do

(Barkley, 1997a, 1997b). In short, information is not self-regulation. Just knowing about self-regulation will not automatically translate into actual self-regulation. The EF system is largely a motor or performance system rather than a sensory processing system. It is a system in which what one knows (skills and knowledge) is applied to daily life across time. To use the knowledge one has acquired in life, one must stop responding impulsively to immediate events and pause the ongoing action. This pause permits the executive system to generate the mentally represented information needed to guide a more appropriate response in that situation.

As I have discussed in detail elsewhere (Barkley, 1997a, 2012; Barkley et al., 2008), treatments for EF deficits, such as ADHD or PFC injuries, should focus on several key recommendations that stem from the self-regulatory extended phenotype model of EF. All of these will be most helpful when they assist with the performance of a particular behavior at the *point of performance* in the natural environments, where and when such behavior should be performed. A corollary of this is that the further away in space and time a treatment is from this point of performance, the less effectively it is likely to assist with the management of EF deficits. Not only is assistance at the "point of performance" going to prove critical to treatment efficacy, but so also is assistance with the time, timing, and timeliness of behavior, not just in the training of the behavior itself. If such assistance is summarily removed within a short period of time once the individual is performing the desired behavior, then maintenance of treatment effects will be unlikely. The value of treatment lies not only in eliciting behavior likely to exist already in the individual's repertoire at the point of performance, where its display is critical, but also in maintaining the performance of that behavior over time in that natural setting.

Disorders of EF pose great consternation for the mental health, rehabilitation, and educational areas of service because they create disorders of mainly performance rather than knowledge or skills. Mental health and education professionals are more expert at conveying knowledge and skills—how to change and what to do; far fewer are expert in ways to engineer environments to facilitate performance—where and when to change. At the core of such problems is the vexing issue of just how one gets people to behave in ways that even they know may be good for them, which they seem unlikely, unable, or unwilling to perform. Conveying more knowledge does not prove as helpful as altering the parameters associated with the performance of that behavior at its appropriate point of performance. Coupled with this is the realization that such changes in behavior are likely to be maintained only so long as those environmental adjustments or accommodations are maintained as well. To expect otherwise would be to approach the treatment of EF deficits with outdated or misguided assumptions about the essential nature of EF and its impairments.

Principles of EF Deficit Management

Some of the principles of EF deficit management for ADHD that arise from the extended phenotype model are briefly mentioned below.

Externalize Information

If the process of regulating behavior by internally represented forms of information (working memory or the internalization of self-directed behavior) is impaired or delayed in those with EF deficits, then they will be best assisted by "externalizing" those forms of information; the provision of physical representations of that information will be needed in the setting at the point of performance. Since covert or private information is weak as a source of stimulus control, making that information overt and public may assist with strengthening control of behavior by that information. It must be made physical outside of the individual, as it has to have been in earlier development. The internal forms of information generated by the executive system, if they have been generated at all, appear to be extraordinarily weak in their ability to control and sustain the behavior toward the future in those with EF deficits. Self-directed visual imagery, audition, covert self-speech, and the other covert resensing activities that form nonverbal working memory do not have sufficient power to control behavior in many EF disorders. That behavior remains largely under the control of the salient aspects of the immediate context.

The solution to this problem is not to nag those with EF difficulties simply to try harder or remember what they are supposed to be working on or toward. Instead, the solution is to fill the immediate context with physical cues comparable to the internal counterparts that are proving so ineffective. In a sense, clinicians treating those with EF deficits must beat the environment at its own game. Whenever possible, minimize sources of high-appealing distracters that may subvert, distort, or disrupt task-directed mentally represented informa-

tion and the behavior it is guiding. In their place should be cues, prompts, and other forms of information that are just as salient and appealing yet directly associated with or an inherent part of the task to be accomplished. Such externalized information serves to cue the individual to do what he or she knows.

For instance, if the rules that are understood to be operative during educational or occupational activities do not seem to be controlling the adult's behavior, they should be externalized. Adults can externalize them by posting signs about the school or work environment and its rules, and frequently refer to them. Having the adult verbally self-state these rules aloud before and during individual work performances may also be helpful. One can also record these reminders on a digital recorder that the adult listens to through an earphone while working. It is not my intention in this chapter to articulate the details of the many treatments that can be designed from this model. That is done in other textbooks. All I wish to do here is simply demonstrate the principle that underlies them: Put external information around the person within his or her sensory field to better guide their behavior in more appropriate activities. With the knowledge this model provides and a little ingenuity, many of these forms of internally represented information can be externalized for better management of the child or adult with EF deficits, as seen in ADHD, for instance.

Externally Represent or Remove Gaps in Time

The organization of the individual's behavior both within and across time is one of the ultimate disabilities rendered by PFC injuries and other EF disorders. EF deficits create problems with time, timing, and timeliness of behavior, such that they are to time what nearsightedness is to spatial vision. They create a temporal myopia in which the individual's behavior is governed even more than normal by events close to or within the temporal now and the immediate context rather than by internal information that pertains to longer term, future events. This helps us understand why adults with EF deficits make the decisions they do, shortsighted as they seem to be to others around them. If one has little regard for future events, then much of one's behavior will be aimed at maximizing the immediate rewards and escaping from immediate hardships or aversive circumstances, without concern for the delayed consequences of those actions. One might assist those with EF deficits by representing time itself more externally, by reducing or eliminating gaps in time among the components of a behavioral contingency (event, response, outcome). Caregivers and others can also help to bridge such temporal gaps related to future events.

Another solution is to reduce or eliminate these problematic time-related elements of a task when feasible. The elements should be made more contiguous. Rather than tell the person that a project must be done over the next month, help him or her to take a step a day toward that eventual goal, so that when the deadline arrives, the work has been done but in small daily work periods with immediate feedback and incentives for doing so.

Externalize Motivation

The model also hypothesizes that a deficit will exist in internally generated and represented forms of motivation needed to drive goal-directed behavior. Complaining to these individuals about their lack of motivation (laziness), drive, willpower, or self-discipline will not suffice to correct the problem. Pulling back from assisting them to let the natural consequences occur, as if this will teach them a lesson that will correct their behavior, is likewise a recipe for disaster. Instead, artificial means of creating external sources of motivation must be arranged at the point of performance in the context in which the work or behavior is desired. For instance, the provision of artificial rewards, such as tokens, may be needed throughout the performance of a task or other goal-directed behavior when there are few or no immediate consequences associated with that performance. Such artificial reward programs become for the person with EF deficits what prosthetic devices are to the physically disabled, allowing them to perform more effectively in some tasks and settings with which they otherwise would have considerable difficulty. The motivational disability created by EF deficits makes such motivational prostheses essential for most children deficient in EF, and they can be useful for adults with EF deficits as well.

The methods of behavior modification are particularly well suited to achieving these ends. Many techniques within this form of treatment can be applied to children and adults with EF deficits. What one first needs to recognize, as this model of EF stipulates, is that (1) internalized, self-generated forms of motivation are weak at initiating and sustaining goal directed behavior; (2) externalized sources of motivation, often artificial, must be arranged within the context at the point

of performance; and (3) these compensatory, prosthetic forms of motivation must be sustained for long periods. If the external motivation is removed, the behavior will not be further sustained, and the individual will regress to more erratic goal-directed behavior, with less ability to sustain actions toward tasks and goals.

In general, there are two reasons to teach behavior management to anyone: for informational training and for motivational sustaining. The former is for individuals who have not yet acquired a skill. Once the skill is taught through behavioral or other pedagogical methods, those methods can be withdrawn and the behavior is sustained presumably by contact with the natural contingencies. But in persons with EF disorders, the issue is not ignorance or lack of knowledge of a skill; the problems are with the skill's timing and execution at key points of performance, and with the self-motivation needed to sustain the performance. Behavioral treatments can provide the motivational or behavior-sustaining assistance. Removing a person's external motivation after improvement in task performance will result in a loss of motivation and a return to the baseline state of limited self-motivation and an inability to sustain actions toward goals.

By equating EF with SR, and by viewing the SR of emotion as described by Gross (1998, 2007) as but a specific form of a more generalized process of SR, the extended phenotype model of EF illustrates at least five vectors through which EF/SR can influence goal-directed activities: situation selection, situation modification, attentional control/redirection, reappraisal, and response modification/suppression. In attempting to assist individuals with rehabilitating or at least compensating for their EF deficits, these five vectors offer opportunities in which clinicians can strive to improve such deficits. While this can be done by working directly with the patient, it is likely to be greatly assisted by advising caregivers or significant others to assist the individual with these five pathways of SR. Modifying the "point of performance," as I discuss in more detail below, readily fits into the situation modification vector of SR. Various cognitive-behavioral therapies may prove useful with the reappraisal vector. The point here is not to map out all possible ways by which these five vectors of SR could be used to boost EF in those with EF deficits but to make clinicians cognizant that such pathways are available for doing so.

Related to this idea of motivational deficits accompanying EF disorders is the literature on self-regulatory strength and the resource pool of effort (willpower) associated with activities of SR. The abundant literature on this topic has been overlooked by neuropsychologists studying EF, yet it has a direct bearing given that EF is viewed as SR. As nicely summarized by Bauer and Baumeister (2011), research indicates that each implementation of SR (hence, of EF) across all types of SR (working memory, inhibition, planning, reasoning, problem solving, etc.) depletes this limited resource pool temporarily, such that protracted SR may greatly deplete the available pool of effort. This can result in an individual being less capable of SR (EF) in subsequent situations or immediately succeeding time periods. He or she is therefore more likely to experience problems or fail outright in efforts at EF/SR and resistance to immediate gratification. Such temporary depletions may be further exacerbated by stress, alcohol or other drug use, illness, or even low levels of blood glucose. Research also indicates that the following factors may serve to replenish the resource pool more rapidly:

- Routine physical exercise.
- Taking 10-minute breaks periodically during SR strenuous situations.
- Relaxing or meditating for at least 3 minutes after such SR exerting activities.
- Visualizing the rewards or outcomes while involved in EF/SR tasks.
- Arranging for periodic small rewards throughout the tasks for SR-demanding settings.
- Engaging in self-affirming statements of self-efficacy prior to and during such tasks.
- Generating positive emotions.
- Consuming glucose-rich beverages during the task.

Some research further suggests that the actual capacity of the resource pool may be boosted by routine physical exercise and routine practice of tasks involving self-regulation daily for 2 weeks. From the extended phenotype view of EF as SR, these findings from the psychological literature on SR are directly pertinent to EF and its disorders.

Intervene at the Point of Performance in Natural Settings

Given previous list of considerations, clinicians should likely reject most approaches to intervention for adults with EF deficits that do not involve helping patients with an active intervention at the point of performance and across the extended EF phenotypic levels that are

impaired. Once-per-week counseling is unlikely to succeed with the patient with deficient EF without efforts to insert accommodations at key points of performance in natural settings to address the impaired domains of major life activities. This is not to say that extensive training or retraining at the instrumental level of EF, as with working memory training, may not have some short-term benefits. Such practice has been shown to increase the likelihood of using EF/SR and of boosting the SR resource pool capacity in normal individuals, at least temporarily (Bauer & Baumeister, 2011).

An implication for the management of EF deficits from the extended phenotype theory is that only a treatment that results in improvement or normalization of the underlying neurological and even genetic substrates of EF is likely to result in an improvement or normalization of the phenotypic deficits. To date, the only treatment that has any hope of achieving this end is medication (e.g., stimulants or nonstimulants such as atomoxetine or guanfacine XR [extended release]) that improves or normalizes the neural substrates in the prefrontal regions and related networks that likely underlie some of these deficits, such as those associated with ADHD. Evidence to date suggests that this improvement or normalization in ADHD-related EF deficits may occur as a temporary consequence of active treatment with stimulant medication, yet only during the time course the medication remains within the brain. For instance, research shows that clinical improvement in behavior occurs in as many as 75–92% of those with ADHD and results in normalization of behavior in approximately 50–60% of these cases, on average. The model of EF developed here, then, implies that medication is not only a useful treatment approach for the management of certain EF deficits in ADHD but it may also be a predominant treatment approach among currently available treatments because it is the only treatment to date that is known to produce such improvement/normalization rates, albeit temporarily, for ADHD-related EF deficits.

Approach ADHD and Its EF Deficits as a Chronic Condition

The forgoing leads to a much more general implication of this extended phenotype model of EF: The approach taken to its management must be the same as that for other chronic medical or psychiatric disabilities. Diabetes is an analogous condition to many forms of EF deficits. At the time of diagnosis, all involved must realize that there is currently no cure for the condition. Still, multiple means of treatment can provide symptomatic relief from the deleterious effects of the condition, including taking daily doses of medication and changing settings, tasks, and lifestyles. Immediately following diagnosis, the clinician works to educate the patient and family on the nature of the chronic disorder, then designs and implements a treatment package for the condition. This package must be maintained over long periods to maintain the symptomatic relief that the treatments initially achieve. Ideally, the treatment package, so maintained, will reduce or eliminate the secondary consequences of leaving the condition unmanaged. However, each patient is different, as is each instance of the chronic condition being treated. As a result, symptom breakthroughs and crises that are likely to occur periodically over the course of treatment may demand reintervention or the design and implementation of modified or entirely new treatment packages. Changes to the environment that may assist those with the disorder are not viewed as somehow correcting earlier faulty learning or leading to permanent improvements that can permit the treatments to be withdrawn. Instead, the more appropriate view of psychological treatment is the design of a prosthetic social environment that allows the patient to cope better and to compensate for the disorder. Behavioral and other technologies that assist people with EF deficits are akin to hearing aids, wheelchairs, ramps, and artificial limbs and other prostheses that reduce the handicapping impact of a disability and allow the individual greater access to and better performance of their major life activities. Those methods provide the additional social and cultural scaffolding around the person with EF deficits, so that performance in that specific setting can be more effective.

Besides these recommendations, three EF-based cognitive-behavioral training approaches related to the phenotype model of EF/SR have been recently developed, researched, and published in manual form for clinicians (see Chapter 31; Ramsay & Rostain, 2007; Safren, Perlman, Sprich, & Otto, 2005; Solanto, 2010; Solanto et al., 2010). All three focus on addressing the types of EF problems associated with adult ADHD, such as poor time management, self-organization, and emotional self-regulation (Barkley, 2011b). Yet they are just as applicable to other EF disorders, with modifications for the specific EF deficits evident in any given case. All are related in one form or another to my earlier self-regulatory model of EF (and therefore tac-

itly, though not explicitly, to the extended phenotype model developed here). Their contents could easily be extended to many other patient groups in which EF deficits are of sufficient concern to warrant the use of a psychosocial training program. These training programs adopt a view of EF that is more similar to the model proposed here than to the traditional cognitive psychometric view of EF. They go far beyond simply exercising the cognitive components of EF that often are targeted in purely cognitive rehabilitation training programs. Instead, the focus is on skills that would have much more of an effect on the five dimensions of EF behavior discussed previously for the methodical–self-reliant and higher levels of EF in the present model (time management, self-organization, problem solving, emotional self-control, self-motivation, etc.).

Intervene at the Most Disrupted Level

Of further importance to intervention is the multilevel nature of EF/SR proposed here and the need to intervene at those levels most disrupted or adversely affected by damage or disorder. For instance, deficits at the instrumental–self-directed (or cognitive) level of EF might require training in self-directed inhibition, imagery, audition, and speech, among other areas, that are often the focus of cognitive rehabilitation (often computer-based) training programs. Although these may boost the initial low capacity of the individual in terms of inhibition, nonverbal and verbal working memory, planning, and problem-solving abilities, among others, evidence suggests that these capacities may decline after treatment has ceased, and that retraining may be needed periodically to sustain initial gains. Adverse effects at the methodical–self-reliant level may need to focus more on helping the individual to reorganize his or her external environment to facilitate performance of EF, self-care, and general adaptive functioning. This might also be facilitated and amplified by artificial devices such as digital memory recorders, computers, personal data assistants, cell phones to which periodic prompts and reminders are sent, and other such environmental prostheses. Also, the cognitive-behavioral therapy (CBT) programs discussed earlier may be particularly applicable to this level of EF deficits given that they, too, focus on rearranging the physical and social environment to facilitate and amplify EF/SR in specific settings. There is also the need to help patients deal with potential social parasitism or predation from others, perhaps through more direct supervision; closer or more highly supervised living arrangements; or, in extreme cases, making the patient a ward of a normally functioning relative. Deficits at the tactical–reciprocal and strategic–cooperative levels will likely require training and ongoing assistance with social skills, etiquette, emotional self-regulation in social settings, and other therapies that address the social nature of these levels (reciprocity, cooperation, mutualism). Legal assistance may also be periodically required to address problems with social contracting, obeying laws, and conforming one's actions more generally to regulatory rules in specific settings. It may also be needed to protect the individual from serious breaches of performance at these levels and even the principled–mutualistic stage, as needed. In short, deficits at each level need to be catalogued, and interventions specific to each level should result in the design of a prosthetic environment around the EF-impaired individual that would not have been at all evident from a merely psychometrically based cognitive view of EF.

CONCLUSION

I hope that the previous discussion has shown that there is obviously much promise in viewing ADHD as a disorder of EF and SR from the vantage point of this extended phenotype theory of EF. It encourages psychopathologists to develop more fully the models of how normal self-control arises across childhood and even into adulthood, and to indicate where in these models disorders such as ADHD disrupt the normal structure and processes of self-regulation to produce what is known about the disorder. Moreover, such model building also suggests new hypotheses that can be pursued in not only testing the model but in providing a greater understanding of what is disrupted by the disorder. I have also argued here that taking an evolutionary or adaptive perspective toward self-control and its associated EFs can further enlighten us on the nature of these relatively unique human abilities and what larger domains of social functioning may be deficient in those with ADHD. That perspective implies that self-control may have arisen for a set of largely social functions, such as self-reliance–social defense, reciprocal exchange, cooperative coalitions, and vicarious learning (and culture). This perspective provides further grounds for the development of testable hypotheses about not only self-control but also the social deficiencies that arise in disorders of self-regulation such as ADHD.

As I have stated before (Barkley, 2012b), no theory is ever perfect as proposed, and that is certainly true of this theory. All one asks of a theory is that it serve as a useful, albeit time-limited tool—as a means to an end, which is to provide a better explanation for what may already be known and to suggest further hypotheses and implications than have heretofore been the case with alternative or prior theories. The standard for judging any theory is utility: Does it help us understand the material world better than prior explanations? Does it better serve us as we strive to improve our methods of survival and welfare, and those of our descendants? To paraphrase Durham (1991) on theorizing, one simply seeks to build a better ship that can be floated for a time, and from the results we can then build an even better ship. It is trial and error with retention and criticism—theories evolve. All one can ask of the extended phenotype theory of EF offered here is that it be an improvement over existing theories of EF and especially of ADHD—imperfect but temporarily useful.

KEY CLINICAL POINTS

- ✓ ADHD is now viewed as involving far more than just its diagnostic symptoms of inattention, impulsivity, and hyperactivity. It is a disorder of SR and EF. The EFs are essential for the contemplation of the future juxtaposed against the here and now, and for the cross-temporal organization of behavior needed to attain that desired future.
- ✓ Numerous studies comparing groups of patients with ADHD and controls typically find those with ADHD to have impaired response inhibition, nonverbal and verbal working memory, and nonverbal fluency. This has not been the case at the individual level of analysis. Despite the inability of neuropsychological tests of EF to indicate impairment of persons with ADHD, multiple studies using rating scales of EF in daily life clearly attest to the fact that the vast majority of patients with ADHD are impaired in the major EF domains of time management, self-organization and problem solving, self-restraint, self-motivation, and self-regulation of emotions. Thus, ADHD is clearly a disorder of EF, as demonstrated by these group studies, rating scales of EF, and neuroimaging research.
- ✓ The theory of self-regulation and EF set forth here views EF as an extended phenotype—a suite of neuropsychological abilities that produce significant adaptive effects at considerable distances from the individual and over considerable time periods. This phenotype is viewed as involving at least five hierarchically organized levels of functioning or concentric rings of radiating activities that produce effects across space and time that impact the individual's long-term welfare.
- ✓ These levels are the instrumental–self-directed, the methodical–self-reliant, the tactical–reciprocal, the strategic–cooperative, and the extended utilitarian stages of EF as it is deployed in everyday life.
- ✓ At the instrumental–self-directed level, EF is viewed as arising from the self-direction, internalization, and self-regulation of behavior across development. The six forms of actions directed at the self involve self-awareness (self-directed attention), self-restraint, self-directed sensorimotor actions (nonverbal working memory), self-directed speech (verbal working memory), the self-direction of emotions and motivations, and self-directed play (for planning and problem solving). These self-directed actions form a suite of mind tools that across time are deployed for self-regulation to achieve desired goals or accomplish assigned tasks.
- ✓ The self-directed, largely cognitive forms of EF are then used at the methodical–self-reliant level to facilitate vicarious learning, social self-defense, and adaptive functioning (self-care). EF at this level involves time management, self-organization and problem solving, self-restraint, self-regulation of emotions, and self-motivation, as reflected in rating scales of EF in daily life.
- ✓ Over time, the tactical–reciprocal level of EF emerges, in which the individual uses EF to engage selectively in social reciprocity or exchange, forming the basis for economics (markets), social relations, friendships, and so forth.
- ✓ Eventually, the earlier stages of EF culminate in the strategic–cooperative level, in which individuals band together to accomplish shared goals and to share in the outcomes that none could accomplish alone. This forms the basis for social groups and communities. Within this stage, under special circumstances, another level of the EF phenotype may emerge, the principled–mutualistic stage, in which individuals use higher level abstract rules to form societies and in which members of the group not only cooperate but also look out for each other's welfare and interests.

- ✓ These levels of EF expand into spheres of human social life and involve the use of others and culture to contemplate, plan for, and execute complex changes of actions to accomplish future goals or attain future desired states.
- ✓ ADHD and other disorders of EF result in dysfunction within and across these levels of the EF phenotype, causing a contraction or even, if severe enough, a collapse in the EF hierarchy. The individual is therefore at risk of impairment in educational, social, occupational, and other forms of major life activities that require effective deployment of EF. Because the EF system is largely one of performance (doing what one knows) rather than knowledge (knowing what to do), ADHD is far more of a performance disorder than one of knowledge or skill.
- ✓ Treatment of ADHD and EF disorders involves altering key elements of the environment (creation of social scaffolding) at crucial points of performance where problems in functioning exist, so as to help people use what they know to achieve better adaptive functioning. It may also involve using medications that improve the EFs and therefore reduce the likelihood of impairment in these various domains of daily life.

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CHAPTER 17

Concentration Deficit Disorder (Sluggish Cognitive Tempo)

Russell A. Barkley

This chapter reviews the evidence for a second attention disorder that is distinct from attention-deficit/hyperactivity disorder (ADHD) yet overlaps with it. Although this condition has been called sluggish cognitive tempo (SCT) since the 1980s, I have recently recommended that the name be changed to concentration deficit disorder (CDD) for various reasons, not the least of which is that SCT can be viewed by the public as pejorative, derogatory, or frankly offensive (Barkley, 2014; Saxbe & Barkley, 2014). Although some prior reviewers of the evidence have suggested that the disorder be called attention deficit disorder (ADD; Diamond, 2005; Milich & Roberts, 2013), and many clinicians have adopted this term to describe people who are primarily inattentive and have little or no evidence of hyperactive or impulsive behavior, it is not advisable to do so given that ADD is the older term for ADHD dating back to DSM-III (American Psychiatric Association, 1980), so resurrecting it as the name for a second attention disorder merely creates unnecessary confusion between these conditions which, as will be shown below, differ quite markedly in a number of important features. The term SCT also implies that neurocognitive dysfunction underlying the condition is well known and supported by empirical evidence, and this is very far from the case at the moment. The same criticism can be applied to other, suggested terms for this condition, such as primary disorder of vigilance (PDV; Weinberg & Brumback, 1990, 1992; Weinberg & Harper, 1993), although PDV was asserted to be an alternative to ADHD and not just the identification of another attention disorder. Obviously, if vigilance is defined as alertness and "sustained attention or efficiency then, by definition, ADHD can automatically be redefined as representing PDV, with the label ADHD now relegated to mythical status by those authors (Weinberg & Brumback, 1992). By tossing into the definition, as they did, problems with wakefulness and focus of attention, one pretty much covers both disorders under discussion here, without having to engage in anything more scholarly than pontification or do anything more scientific than dredge up five clinical cases from one's practice, while summarily dispensing with 200 years of medical commentary and scientific research on ADHD (Weinberg & Brumback, 1990, 1992; Weinberg & Harper, 1993). CDD seems to be a reasonable option for various reasons: (1) It keeps the focus of the label on an attention problem yet makes it distinct from ADHD; (2) it is not as offensive or pejorative to patients and family members as SCT; (3) it does not imply we know more than we do about the underlying cognitive dysfunction, as do the terms SCT and PDV; and (4) it may suggest some overlap with ADHD,

which is the case (see below). Moreover, the term "concentration" does not appear in the symptom lists in DSM-5 for ADHD (American Psychiatric Association, 2013) or in those for SCT (Penny, Waschbusch, Klein, Corkum, & Eskes, 2009), so it is less likely to create unnecessary semantic confusion. For these reasons, throughout the remainder of this chapter I refer to this condition as CDD (SCT).

HISTORY OF CDD (SCT) VERSUS ADHD

Cases of CDD (SCT) have likely existed within the human population at least throughout the past two centuries, if not longer. Descriptions of individuals with "low power" of attention or arousal, who appear to stare or to daydream frequently, and otherwise seem too inattentive or sluggish and erratic to process information accurately, seem to have first appeared in the medical literature in Crichton's description of two disorders of attention in his medical textbook (Crichton, 1798/1976). Certainly Weikard (1775) did not seem to mention this condition in his earlier treatise on attention deficits (Barkley & Peters, 2012). The first attention disorder noted by Crichton was one of distractibility; frequent shifting of attention, or inconstancy; and lack of persistence or sustained attention. It nicely aligns with the attention disturbance assigned now to ADHD (Palmer & Finger, 2001). The second was a disorder of diminished power or energy of attention that seems more like the attention problem evident in CDD (SCT). Crichton had little to say about the second disorder of attention other than that it may be associated with debility or torpor of the body, which, he reasoned, weakens attention, causing individuals to be retiring, unsocial, and to have few friendships or attachments of any kind. What friendships may exist are seldom of a durable nature. Crichton further argued that the faculty of attention could be so weakened as to leave an individual insensible to external objects or to impressions that ordinarily would awaken social feelings. This seems to overlap with current depictions of CDD (SCT) (Milich, Ballentine, & Lynam, 2001; Penny et al., 2009) in some respects. However, Crichton's description could also be ascribed to autistic spectrum disorders or even schizoid or schizotypal personality disorders if not psychopathy.

Other than these historical curiousities, the contemporary period of research on CDD (SCT) began in 1980. It was a clear consequence of the proposal to create two types of ADHD in DSM-III (American Psychiatric Association, 1980): those having ADD with (+H) and without hyperactivity (–H). As I recall from those meetings, this bifurcation was largely predicated on some anecdotes of clinician members of the committee who saw such cases of ADD – H in their practices and wished to have a means to identify them in the official taxonomy of childhood disorders. At first, the DSM-III mistakenly placed impulsiveness in with the inattentive symptoms, creating this dichotomy on the basis of hyperactivity alone. Studies soon indicated that the impulsive symptoms were more closely linked to the hyperactive ones than to those of inattention (Carlson, 1986; Lahey, Schaughency, Strauss, & Frame, 1984; Milich et al., 2001), as was later indicated in DSM-IV (American Psychiatric Association, 1994) and now in DSM-5 (American Psychiatric Association, 2013). Subsequently, researchers corrected this error and proceeded to do numerous studies comparing individuals with ADD + H and ADD – H (persons high in symptoms of both inattention [I] and hyperactivity–impulsivity [HI], and those high in I but very low in HI symptoms).

Probably the first article to examine the existence of ADD – H was that by Maurer and Stewart (1980). Out of a review of 297 patients, they identified 52 as likely having ADD. More than half of them (31) had conduct disorder, and 11 others were diagnosed with various other psychiatric disorders. They found only nine children who appeared to be pure cases of ADD – H, and characterized these children as principally having significant learning disabilities and "lack of motivation." The authors concluded that ADD – H did not appear to be an independent syndrome. This was followed shortly by a study in which Pelham, Atkins, and Murphy (1981) screened 610 children between kindergarten and fifth grade, and distinguished between those with ADD + H and those with only ADD – H. As usual, the ADD + H group had higher ratings of conduct problems. Girls with ADD – H were rated by teachers as significantly more inattentive–passive and immature, and by peers as more withdrawn than girls with ADD + H. This was probably the first article to identify conduct problems as being differentially associated with ADD + H rather than ADD – H. Many other researchers would subsequently replicate this finding. In 1984, Lahey and colleagues published an article in which they compared 10 children with ADD + H and 20 children with ADD – H. Like the earlier researchers, they found that the ADD + H group had significantly

higher levels of aggressive behavior and conduct problems, bizarre behavior, and lack of guilt, and were quite unpopular. They also performed poorly in school. In comparison, the ADD – H group was more likely to be anxious, shy, socially withdrawn, and moderately unpopular; to do poorly in sports; and to have poor school performance. Both groups manifested significant problems with depression and poor self-concepts, but they differed in the areas of low self-esteem they reported. The ADD + H group reported problems with academic status, behavior, and popularity. The ADD – H group reported self-concept concerns regarding physical appearance, anxiety, and general happiness. This article was most likely the origin of the term SCT for this subset of children with ADD – H and symptoms of drowsiness, sluggishness, and daydreaming (C. L. Carlson, personal communication, November 20, 2013).

In a subsequent article, Lahey, Schaughency, Frame, and Strauss (1985) compared 20 children with ADD + H to 20 with ADD – H and found the usual differences noted earlier concerning greater symptoms of sluggishness, drowsiness, and daydreaming in contrast to the impulsive, distractible, and overactive pattern found in ADD + H. They argued that those having CDD (SCT) symptoms had a different type of attention disorder than ADD + H; they were not subtypes of the same ADD disorder at all and did not share the same underlying attention disturbance. In 1985, according to Carlson (1986), her then graduate student, Neeper, conducted a cluster analysis in order to subtype children with learning disabilities (LD) on the basis of their behavior. Using 75 children with LD, the author used cluster analysis on the Child Behavior Rating Scale, a rating scale that was new to this author, in which he identified a separate group of 11 children having high scores on an inattention–disorganization factor and low scores in motor hyperactivity. This group had significantly higher ratings of anxiety–depression and higher ratings on a factor he named "sluggish tempo factor," which comprised items related to apathetic, lethargic, sluggish, and drowsy behaviors. The children with ADD + H were, once again, found to have significantly higher ratings of conduct disorder than the children with ADD – H. The two subtypes differed somewhat on a battery of cognitive tests (Carlson, Lahey, & Neeper, 1986). In that study, 20 children with ADD + H were contrasted against with ADD – H. Both ADD groups scored significantly lower on intelligence testing. The ADD – H group had lower full-scale IQ scores than the ADD or control groups. Both ADD groups did poorly on tests of spelling and reading, but the ADD – H group performed more poorly on math achievement. Problems with visual matching were greater in the ADD – H than in the ADD + H group. The groups did not differ in accuracy on the Stroop task, rapid naming, measures of receptive and expressive language, visual–motor integration, or sustained visual attention. Thus, it appears that these two disorders of attention differ more in ratings of behavior, social relations, self-esteem, and internalizing symptoms, but less so on cognitive measures. Noteworthy is that this may be the first report of an association of ADD with difficulties with math performance that appeared again in subsequent studies of CDD (SCT). Despite these initial successes at identifying differences between these supposed subtypes of ADD + H and ADD – H, other studies produced quite mixed results or found just minor differences (King & Young, 1982; Maurer & Stewart, 1980).

Carlson's 1986 review article provides a more comprehensive summary of the research on ADD + H and ADD – H up to that time. It concluded that ADD – H can be distinguished from ADD + H based on its behavioral characteristics and impairments. Both groups show poor academic functioning, but the peer problems aligned with ADD – H comprise problems with anxiety, shyness, and social withdrawal. Their attention deficit symptoms ran more toward the sluggish, drowsy, and apathetic sort. These children were less likely to show conduct problems, peer unpopularity, and social rejection than were children with ADD + H. In contrast, the children with ADD + H were more socially rejected, displayed more aggression and conduct problems, and were more distractible and impulsive than children with ADD – H or controls. Milich and colleagues (2001) reached the same conclusions in their review years later.

Eventually, DSM-III-R (American Psychiatric Association, 1987) would abolish the +H and –H in view of the limited research supporting such subtyping, yet it called for continuing research on the ADD – H group, now termed "undifferentiated ADHD" and placed in the appendix of that manual. Researchers' continued exploration of differences between these subtypes for a few years thereafter (Barkley, DuPaul, & McMurray, 1990) suggested a greater manifestation of CDD (SCT)-like symptoms in the ADD – H than in the ADD + H group and possibly a reduced response to stimulant medication (Barkley, DuPaul & McMurray, 1991).

With the advent of DSM-IV (American Psychiatric Association, 1994), these +H and –H subtypes would

reappear in the official taxonomy as ADHD combined type (ADHD-C) versus ADHD inattentive type (ADHD-I). Once more, official sanctioning of this subtyping would foster numerous studies comparing them well into the 1990s and beyond. That same year, Wheeler and Carlson (1994) reviewed what was known about ADD +H and –H differences concerning children's social functioning and argued that these differences could be extended to the newly created ADHD-I and ADHD-C types in DSM-IV.

Seven years later, in 2001, a very influential review of the literature concerning these two attention disorders was published by Milich and colleagues (2001). The authors comprehensively reviewed research regarding the subtypes and concluded that ADD – H, or what was now ADHD-I type, was a distinct and unrelated disorder to ADD + H, or ADHD-C type. In addition to the evidence reviewed earlier, the authors also reported the results of studies using factor analysis to compare ADHD symptoms and those of SCT. Those analyses revealed three distinct factors, two of which characterized ADHD: I and HI symptoms. CDD (SCT) symptoms formed a factor that was distinct from these other two dimensions. This was also found to be the case in a factor analysis of the direct observation form of the Child Behavior Checklist by McConaughy and Achenbach (2001). The authors argued that a subset of children characterized as having the ADHD-I type might have a separate disorder entirely and be best distinguished by their symptoms of CDD (SCT). The reviewers also noted, however, that contained within the ADHD-I type would be children who simply had a milder form of the ADHD-C type but with barely sufficient HI symptoms to be so classified. Hence, the latter children were being incorrectly classified as being the ADHD-I type and would contaminate any efforts to find differences between the ADHD-I and ADHD-C types if not removed.

Also in 2001, McBurnett and colleagues (2001) conducted a factor analysis of 692 children referred to a specialty pediatric clinic for ADHD and found, as did Neeper (see Carlson, 1986) earlier, that symptoms of CDD (SCT) form a dimension that is distinct from the two traditional ones comprising ADHD. Three years later, Todd, Rasmussen, Wood, Levy, and Hay (2004) factor-analyzed data from 2,894 twin pairs and also found a separate factor for CDD (SCT), distinct from those for ADHD. It should be noted here that symptoms of CDD (SCT) were included in the DSM-IV field trial (see McBurnett et al., 2001). But they were found to have little or no value in identifying cases of ADHD, particularly ADHD-C, and so were omitted from further consideration. Now we know why, of course. The symptoms were actually identifying a distinct disorder of attention, separate from ADHD.

In the past decade, because of the mixed pattern of findings to date on the distinction, researchers decreased their efforts to study this C type (ADD + H) versus I type (ADD – H) distinction in favor of studying those children specifically identified with high levels of CDD (SCT) symptoms in comparison to those with ADHD-C. Some studies have estimated that as many as 30–63% of cases of the ADHD-I type have high levels of CDD (SCT) (Carlson & Mann, 2002; Garner, Marceaux, Mrug, Patterson, & Hodgens, 2010; McBurnett et al., 2001). One of the first to separate out children having CDD (SCT) symptoms from within the ADHD-I type was Carlson and Mann (2002). They compared two sets of children with ADHD-I type based on whether they were high or low in CDD (SCT) symptoms. Both groups had similar levels of learning problems and inattention. But children with CDD (SCT) had less externalizing symptoms and higher levels of unhappiness, anxiety/depression, withdrawn behavior, and social dysfunction. They argued for the use of CDD (SCT) symptoms to identify a more homogeneous group of inattentive children who were distinct from those having ADHD. Subsequently, many researchers did so (Barkley, 2012b, 2013; Carlson & Mann, 2002; McBurnett et al., 2001; Garner et al., 2010; Penny et al., 2009; Skirbekk, Hansen, Oerbeck, & Kristensen, 2011). Indeed, Penny and colleagues (2009) went so far as to compile a comprehensive set of CDD (SCT) symptoms according to experts they surveyed and a review of research papers, then subjected them to further analysis, ultimately creating a rating scale of the most useful set. By 2012, I had developed the first CDD (SCT) rating scale for adults and published the results of the first study of adult CDD (SCT) based on a representative U.S. sample of adults ages 18–92 (Barkley, 2012b), discussed further below.

In summation, the construct of CDD (SCT) grew out of efforts to identify differences between subtypes of ADD and subsequently children with ADHD. While differences between those subtypes proved mixed and unconvincing relative to any substantial or qualitative differences, research focusing specifically on children having CDD (SCT) proved more promising. Even so, as I have stated elsewhere (Barkley, 2014), CDD (SCT) remains a highly understudied construct and associated

pattern of symptoms (and disorder) within the field of clinical psychology and psychiatry—a situation which I attempted to address in a special issue of the *Journal of Abnormal Child Psychology* (Barkley, 2014). More than 10,000 articles exist on ADHD (more than 4,000 of which have been published since 2007). But I believe that fewer than 50 articles on CDD (SCT) specifically currently exist. Substantially more research needs to be directed at all aspects of CDD (SCT) (vs. ADHD and related disorders, as well as typically developing people) including demographics, correlates, comorbidity, families, and especially etiologies, interventions, and life course risks. As I have previously noted (Barkley, 2014; Saxbe & Barkley, 2014), students now entering the profession could easily make a successful clinical research career specializing in the study of CDD (SCT) given the paucity of research and the great promise of distinct findings foreshadowed by the results of current research. Demand for such empirically based knowledge is likely to increase due to increasing clinical referrals of individuals with this condition, driven by increased awareness of the general public about CDD. The fact that CDD does not yet exist in any official taxonomy of psychiatric disorders does not alter the situation. The increasing information on CDD (SCT) at various widely visited Internet sites such as YouTube and Wikipedia, among others, will ensure a growing public demand for more scientific knowledge about CDD (SCT) and its management.

WHAT DO WE KNOW ABOUT THE NATURE OF CDD (SCT) COMPARED TO ADHD?

Symptom Dimension Differences

There is no official diagnostic term for children whom researchers have labeled as having CDD (SCT). There are no official criteria available for its clinical recognition. However, researchers have identified the most salient symptoms of CDD (SCT) (Barkley, 2012b, 2013; Carlson & Mann, 2002; Garner et al., 2010; McBurnett et al., 2001; Penny et al., 2009): (1) daydreaming; (2) trouble staying awake/alert; (3) mentally foggy/easily confused; (4) stares a lot; (5) spacey, mind is elsewhere; (6) lethargic; (7) underactive; (8) slow-moving/sluggish; (9) does not process questions or explanations accurately; (10) drowsy/sleepy appearance; (11) apathetic/withdrawn; (12) lost in thoughts; (13) slow to complete tasks; and (14) lacks initiative/effort fades. The last two symptoms, however, are as likely to be associated with ADHD as with CDD (SCT) in children or adolescents, so they are not recommended for assisting with differential diagnosis between these two types of attention disorders (Barkley, 2013; Burns, Servera, Bernad, Carrillo, & Cardo, 2013; Lee, Burns, Snell, & McBurnett, 2014). But the remaining 12, among others (Penny et al., 2009), appear to be highly useful for making such distinctions.

The findings from research using at least 10 or more of these listed symptoms indicate that at least two dimensions seem unique to CDD (SCT) yet are intercorrelated sufficiently to be combined in this disorder. One is a daydreaming/slow dimension or factor and the other is a sleepy/sluggish/underactive dimension or factor (Barkley, 2013; Burns et al., 2013; Jacobson et al., 2012; Penny et al., 2009). Sometimes a third factor is found for the low initiative/impersistence items, but as I just noted, these seem more related to ADHD-I symptoms and are therefore not much help in differential diagnosis (Barkley, 2013). Interestingly, as with ADHD, there is a cognitive–inattentive dimension and a behavioral–motor dimension to CDD (SCT), yet both are reasonably distinct from those evident in ADHD. These distinct factors are evident across all of the various approaches to measurement studied to date. These include parent and teacher ratings (Barkley, 2013; Bauermeister, Barkley, Bauermeister, Martinez, & McBurnett, 2011; Becker, Luebbe, Fite, Stoppelbein, & Greening, 2014; Burns et al., 2013; Garner et al., 2010; Hartman, Willcutt, Rhee, & Pennington, 2004; Jacobson et al., 2012; Lee et al., 2014; McBurnett et al., 2014; Penny et al., 2009; Willcutt et al., 2014), observations of behavior at school (McConaughy, Ivanova, Antshel, Eiraldi, & Dumenci, 2009), and observations of behavior in clinical settings (McConaughy, Ivanova, Antshel, & Eiraldi, 2009). CDD (SCT) symptoms are also found to be separate from those for ADHD in adult self-reports (Barkley, 2012b).

CDD (SCT) symptoms are significantly but moderately correlated with the ADHD symptom dimensions, particularly so for the ADHD-I dimension. Moreover, these symptoms identify a unique group of children even within samples that have ADHD-I type (Capdevila-Brophy et al., 2012; Marshall, Evans, Eiraldi, Becker, & Power, 2014). Yet CDD (SCT) symptoms are substantially less correlated with ADHD symptoms than are the two CDD (SCT) dimensions to each other or the two ADHD symptom dimensions with each other (Barkley, 2012b, 2013; Penney et al., 2009). A number of studies indicate that CDD (SCT) symptoms

demonstrate a far lower relationship to HI symptoms than they do to ADHD-I symptoms (Barkley, 2012a, 2012b; Burns et al., 2013; Hartman et al., 2004; Garner et al., 2010; Jacobson et al., 2012; Penny et al., 2009; Wahlstedt & Bohlin, 2010). In fact, this relationship of CDD (SCT) to HI symptoms may become negative once the overlap of ADHD-I with CDD (SCT) is statistically removed (Lee et al., 2014; Penny et al., 2009). All of this is to say that the structure of CDD (SCT) symptoms is not merely a reflection or broadening of the ADHD symptom dimensions, as might be expected from the CDD (SCT)-as-ADHD-subtype hypothesis. Instead, CDD (SCT) symptoms are as independent or partially coupled to ADHD symptoms, as are other symptoms dimensions of child and adult psychopathology to each other.

The totality of evidence shows that CDD (SCT) symptoms have a clear separation in their dimensional structure (usually via factor analysis) from the two dimensional structure of ADHD. While correlated to a low to moderate degree with the ADHD symptom dimensions, the two (or more) dimensions of CDD (SCT) are more highly correlated with each other than with those of ADHD. The relationship between CDD (SCT) and ADHD dimensions is similar to that found for other dimensions of psychopathology that are semi-related yet also rather distinct from each other, such as the relation between anxiety and depression or between oppositionality and ADHD.

Demographic Differences

Only a handful of prior studies examined parental/family demographic characteristics of CDD vs ADHD. Several studies (e.g., Garner et al., 2010; Jacobson et al., 2012) found that CDD (SCT) was not related to child age, gender, or minority status. This same pattern was evident in my two large epidemiological studies of representative samples of U.S. children (Barkley, 2013) and adults (Barkley, 2012b) across ages 6–89 years. In ADHD, however, the symptoms decline across childhood with age, as discussed in Chapter 2. In the study of children (Barkley, 2013), I found that those with CDD (SCT) were older than those with ADHD, which implies a somewhat later age of onset for the former symptoms.

ADHD symptoms occur more often in boys than in girls during childhood and adolescence but come close to equalizing in adulthood (Chapter 2; also Barkley, 2012b, 2013; Burns et al., 2013). This is not the case for CDD (SCT), in which males have only slightly more symptoms than females in childhood and no evident sex differences by adulthood (Barkley, 2012b, 2013; Burns et al., 2013). This lack of association of CDD (SCT) with age and sex was also evident in the recent study by Lee and colleagues (2014), who noted no sex differences and no effect of age on teacher ratings, and only a very small difference due to those demographic factors in parent ratings.

Some studies have found ADHD symptoms to be slightly but significantly associated with some ethnic groups (Hispanic–Latino) more than others, whereas this is not the case for CDD (SCT) symptoms in those same nationally representative samples (Barkley, 2012b, 2013). Likewise, earlier studies of CDD (SCT) also failed to find any association with age, sex, and ethnicity (Garner et al., 2010; Jacobson et al., 2012).

In my national survey of children (Barkley, 2013), I noted that CDD (SCT) was linked to lower parental education, lower annual household income, and greater likelihood of a parent being out of work due to disability. My survey of U.S. adults (Barkley, 2012b) also indicated that those classified as CDD (SCT) also had less education and less annual income. In those instances where CDD (SCT) was comorbid with ADHD in the adult survey (Barkley, 2012b), individuals were more likely to be unmarried and to be out of work on disability than were adults with ADHD. Such findings intimate that CDD (SCT) might be more strongly associated with psychosocial adversity or stressors than is ADHD.

To summarize, emergent patterns in results to date indicate that the demographic correlates associated with CDD (SCT) may be different from those evident in ADHD.

Neuropsychological Differences

Initial studies on cognitive differences between ADD + H and ADD – H, such as those by Carlson and colleagues (1986), do not clearly inform the issue about comparing CDD (SCT) and ADHD directly. That is because, as noted earlier, ADD – H groups are contaminated with children who are really subthreshold ADD + H (or ADHD-C type) cases. Also, the ADD – H groups, like the later ADHD-I type groups studied in research, were not selected directly for having CDD (SCT). Moreover, all cases were chosen from referrals for ADD or ADHD, which makes it appear as if the ADD – H, ADD-I type, or even CDD (SCT) cases are

a subset and hence a subtype of ADHD. To determine whether CDD (SCT) is a different disorder from ADD or ADHD, the selection of cases with CDD (SCT) must be done directly from either general clinical referrals or preferably general community samples screened specifically for CDD (SCT). Just as did research comparing ADD – H and ADD + H cases, research comparing the ADHD-C to ADHD-I types found patterns of cognitive differences that were rather weak, if they appeared at all (Solanto et al., 2007). This makes it appear that these two disorders of attention differ less in cognitive patterns and more in ratings of disruptive behavior (higher in ADD + H), social relations (less popular yet less withdrawal in ADD + H), self-esteem (lower in ADD – H), and internalizing symptoms (higher in ADD – H) (Milich et al., 2001). As already noted, the study by Carlson and colleagues (1986) seems to be the first report of an association of ADD – H with difficulties with math performance, a pattern that would become evident in later, specific studies of CDD (SCT) (Bauermeister et al., 2012).

In general, there has been vastly less research on the neuropsychological deficits associated with CDD (SCT) compared to ADHD, in which the research literature is abundant (Frazier, Demaree, & Youngstrom, 2004; Hervey, Epstein, & Curry, 2004; Willcutt, Doyle, Nigg, Faraone, & Pennington, 2005). A few researchers imply that CDD (SCT) may have problems with early information processing or selective attention, which is not typical of ADHD (e.g., Huang-Pollack, Nigg, & Carr, 2005). But this must be replicated in more research before being viewed as a correlate of CDD (SCT). Likewise, slower motor speed has been linked to CDD (SCT) in some studies (e.g., Adams, Milich, & Fillmore, 2010; Garner et al., 2010), consistent with its symptom profile. Others (Bauermeister et al., 2012), however, did not replicate this finding. Variability of spatial memory performance was specifically linked to CDD (SCT) but not ADHD in the Skirbekk and colleagues (2011) study, even after the researchers controlled for IQ, ADHD inattention, and other variables. Again, replication of such findings is essential before one can have confidence in their linkage to CDD (SCT).

Only a few studies using psychometric tests of executive functioning (EF) have been done with cases selected for CDD (SCT). Unlike ADHD, results indicate that CDD (SCT) is not as serious and pervasive a disorder of EF (Bauermeister et al., 2012; Wahlsted & Bohlin, 2010). Ubiquitous research indicates that in ADHD, for instance, there are deficits on tests of inhibition and working memory, especially nonverbal working memory (Willcutt et al., 2005). In contrast, this is not seen in CDD (SCT) (Bauermeister et al., 2012). But EF tests have low or no ecological validity and low or no relationships to various domains of impairment in contrast to ratings of EF (Barkley, 2012a; Barkley & Fischer, 2011; Barkley & Murphy, 2010). So EF ratings may provide a different pattern of results for SCT than do EF tests.

Just a few studies have used EF ratings to study cases of CDD (SCT). For instance, in my own large studies (Barkley, 2012b, 2013) I used my rating scale of EF in daily life with large epidemiologically derived samples of children and adults having CDD (SCT), ADHD, or both. Results indicated that CDD (SCT) had only very weak relationships to four of the five EF deficit dimensions (< 1% shared variance) when its association with ADHD symptoms, especially the inattention dimension, was statistically controlled. On one dimension (Planning and Problem Solving) there was a slightly higher contribution (< 5%) after such statistical control. Overall, it is the inattentive dimension of ADHD that contributes to the vast majority of variance across most EF dimensions on my scales, with the HI dimension accounting for a lesser but still significant degree of variance, especially in the EF dimensions of Self-Restraint (inhibition) and Emotional Self-Regulation. From these results, I concluded that CDD (SCT) is not a disorder of EF but that ADHD is massively so. Recently, McBurnett and colleagues (2001) found some items related to the EF component of working memory to be a useful additional dimension of CDD (SCT) symptoms, beyond those usually found in community studies. But I have reservations about such findings given that the sample used in that study was a large group of children with ADHD-I referred for a psychosocial treatment program. As I noted earlier, this procedure can lead to a rather heterogeneous group, only a subset of which can be expected to have CDD (SCT) and none purely so. Moreover, others may well be subthreshold variants of ADHD-C that fall just shy of the necessary six HI symptoms, as McBurnett and colleagues noted. These cases should be construed as mild versions of ADHD-C. The results of the study by McBurnett and colleagues may be an artifact of recruitment that may not replicate in community samples from which CDD (SCT) cases were directly sampled.

As I found in my studies on CDD (SCT), Becker and Langberg (2013) likewise found a smaller contribu-

tion of CDD (SCT) to the metacognitive factor on the Behavior Rating Inventory of Executive Functioning in comparison to the inattentive symptoms of ADHD. So did Jiménez, Ballabriga, Martin, Arrufat, and Giacobo (in press), even after controlling for ADHD inattention. This small link of CDD (SCT) to EF-like problems was also evident in the study by Langberg, Becker, Dvorsky, and Luebbe (in press), but only for parent-reported organizational problems. Yet only ADHD inattention symptoms linked up with organizational problems as rated by teachers. It is possible that problems with certain aspects of working memory may be weakly related to or possibly secondary to the cognitive CDD (SCT) daydreaming dimension. Yet I believe those working memory/organizational problems hardly compare to the more severe and pervasive EF deficits so evident in ratings of daily life in children and adults with ADHD (Barkley, 2012a, 2013). Moreover, it is clear across all of these studies utilizing EF rating scales that SCT has no significant association with EF inhibitory problems, whereas those problems are substantial in ADHD.

Mostly what has been found so far is an apparent dissociation of CDD (SCT) from most EF deficits in daily life that are so striking in ADHD. This pattern implies that the cognitive dysfunctions underlying CDD (SCT) symptoms are not like those involved in ADHD. Consequently, CDD (SCT) is really not a subtype of ADHD.

Overlap of ADHD and CDD (SCT)

Selection of individuals for the majority of research on CDD (SCT) was from children referred to clinics for concerns about ADHD; indeed, in some, a diagnosis of some type of ADHD (via DSM-IV criteria) was the starting point. As noted earlier, this can automatically make it seem as if CDD (SCT) is a subtype of ADHD in the results of such research if any differences emerge at all. It also means one cannot study the overlap or independence of the disorders. But if individuals with CDD (SCT) are selected from general population or clinic samples, there is the opportunity for CDD (SCT) to be seen independently of ADHD, so the comorbidity between the two can be studied. I did so in my two national surveys (Barkley, 2012b, 2013), in which I found that more than half (59%) of the children qualifying for a research diagnosis of CDD (SCT) met research criteria for having ADHD. It was mostly among those ADHD subtypes having significant I-type symptoms rather than among those with HI-type, as others have found (Garner et al., 2010; Penny et al., 2009; Skirbekk et al., 2011). While such overlap could mean that CDD (SCT) is a form of ADHD, other findings discussed earlier seem to rule against that conclusion. Only 39% of the children qualifying for ADHD of any type also qualified for CDD (SCT). Again, these findings agree with prior studies of children (Garner et al., 2010; Hartman et al., 2004) and adults (Barkley, 2012b). For instance, in a recent survey of U.S. adults (Barkley, 2012b), 5.8% of the sample met criteria for high CDD (SCT) symptoms. Although approximately half (54%) of those participants qualifying for CDD (SCT) had ADHD, nearly half did not. The overlap arose mostly with those subtypes having significant ADHD-I type symptoms. Similarly, approximately half of individuals qualifying for ADHD of any type (46%) also qualified for CDD (SCT). Once more, the overlap with CDD (SCT) mainly involved individuals having high symptoms of the ADHD-I type, as would be expected given the moderate correlation between these two symptom dimensions. It seems here that the relationship of CDD (SCT) to ADHD is one of comorbidity between two relatively distinct but related or partially coupled disorders, such as exists between anxiety and depression, and not one of subtyping within a single shared disorder. More research will help to clarify whether this is in fact the case. Meanwhile, it seems prudent to create a higher-order or meta-category of attention disorders under which one would then break out ADHD and CDD as separate, semidistinct conditions much like what is done now for the supracategory LD that comprises reading, spelling, math, writing, and related disorders that may exist alone but also may be comorbid.

Patterns of Comorbidity

As described earlier, research comparing ADD – H and ADD + H seems to demonstrate that ADD – H cases were more often linked with anxiety, low self-esteem, social withdrawal, and ratings of internalizing symptoms more generally. Later, this seemed to be true for studies contrasting ADHD-I and ADHD-C types. This pattern seems to be even more evident in comparisons of CDD (SCT) to ADHD. Repeatedly, CDD (SCT) symptoms are more often linked to elevated ratings of internalizing symptoms generally than are ADHD symptoms (Bauermeister et al., 2012; Becker & Langberg, 2013; Becker, Luebbe, et al., 2014; Capdevila-Brophy et al., 2012; Carlson & Mann, 2002; Garner et al., 2010; Hartman et al., 2004; Penny et al., 2009),

even after researchers control for the contribution of ADHD symptoms (Bauermeister et al., 2012; Becker & Langberg, 2013; Burns et al., 2013; Lee et al., 2014; Penny et al., 2009; Willcutt et al., 2014). When the inverse is done and CDD (SCT) symptoms are statistically removed, the ADHD-I dimension may be less or even be unrelated to internalizing symptoms (Lee et al., 2014; Penny et al., 2009) or ratings of social problems (Becker, Luebbe, et al., 2014). Worth noting is that whereas the relationship of CDD (SCT) to ODD is not significant or it is even negative (see below), the relationships of CDD (SCT) to anxiety and depression are positive (Lee et al., 2014). This pattern is different than that seen in ADHD, in which relationships to ODD and internalizing symptoms are both positive (Burns et al., 2013). CDD (SCT) may predict each of these internalizing dimensions (anxiety, depression) even after researchers control for the overlap of the latter dimensions with each other (Becker, Luebbe, et al., 2014), and this association of CDD (SCT) to depression remained even after they controlled for parental internalizing dimensions. While a few exceptions exist in this literature (Burns et al., 2013; Harrington & Waldman, 2010; Wahlstedt & Bohlin, 2010), the weight of the evidence finds CDD (SCT) to be more closely related to internalizing symptoms (anxiety, depression, withdrawal) than is ADHD. The pattern here of a double dissociation between the two disorders in their linkage to internalizing symptoms is evidence that they are each distinct conditions, not subtypes of a common disorder.

ADHD is routinely linked to a higher risk for comorbidity for the externalizing symptom dimension generally; consider that oppositional defiant disorder (ODD) is 11 times more likely to occur with ADHD than it does in the general population (Angold, Costello & Erkanli, 1999). In contrast, there is no association or even a negative association of CDD (SCT) with ODD (Barkley, 2013; Burns et al., 2013; Lee et al., 2014 ; Penny et al., 2009). Because of this lack of association with ODD, it can be reasoned that CDD (SCT) also would have little or no associations with conduct disorder (CD), substance use disorders, or adult antisocial personality disorder, all of which are linked to varying degrees with ODD. Further evidence for this lack of association or even negative association with externalizing disorders is evident in a study using direct observations of disciplinary actions [time-outs] on an inpatient unit (Becker, Luebbe, et al., 2014). Such disciplinary actions are often in response to disruptive or aggressive behavior and were positively linked to the HI symptoms of ADHD but negatively associated with CDD (SCT) symptom severity. This is yet another double dissociation supporting the distinctiveness of CDD (SCT) from ADHD.

One prior study examined the relationship of CDD (SCT) and ADHD to specific professional diagnoses of 17 different learning, developmental, and psychiatric disorders as reported by parents concerning past professional diagnoses their children had received (Barkley, 2013). Both CDD (SCT) and ADHD were associated with elevated rates of comorbidity for 11 of the 17 disorders. But CDD (SCT) was not associated with higher rates of reading or math disorders, hearing impairment, ODD, anxiety disorder, or bipolar disorder diagnoses than controls. ADHD was linked to higher rates for all of these disorders except hearing impairments. Unlike ADHD, the CDD (SCT) group had a higher rate of depression than either the controls or those with ADHD. The comorbidity of ADHD + CDD (SCT) was associated with higher rates of comorbidity for most disorders than was either disorder alone. This implies an additive effect of each disorder when it exists with the other, as if each were a distinct disorder that rendered greater risks when comorbid. Or this pattern could have arisen merely as a function of symptom severity: Comorbid cases had more symptoms of both disorders than was the case for each specific disorder group.

Domains of Impairment

For a condition to rise to the level of a mental disorder, there must be evidence of impairment or harm to (adverse consequences for) the individual from those symptoms (American Psychiatric Association, 2013). We think of "symptoms" as the cognitive and behavioral expressions of a disorder, while "impairment" represents the consequences that flow from such symptoms. As discussed earlier, ADD – H, as well as its subsequent iteration as ADHD-I type had been routinely associated with social withdrawal. Studies of symptoms of CDD (SCT) more specifically have shown it to be linked to social problems generally and social withdrawal specifically (Becker & Langberg, 2013; Becker, Luebbe, et al., 2014; Burns et al., 2013; Capdevila-Brophy et al., 2012; Garner et al., 2010; Marshall et al., 2014; Willcutt et al., 2014) even in the presence of high ADHD-I symptoms (Capdevila-Brophy et al., 2012). Such findings may be more apparent in teacher ratings than in parent ratings (Bauermeister et al., 2012; Becker & Langberg, 2013).

Mikami, Huang-Pollock, Pfiffner, McBurnett, and Hangai (2007) provide the only study to date using detailed observations of the social interactions of children with CDD (SCT) in a simulated chat room with children with ADHD and controls. They statistically controlled for ADHD type, IQ, reading ability, and typing skill in their analyses. CDD (SCT) was noted to independently predict fewer total responses in the chat room, less perception of subtle social cues, less memory for the conversation, and a smaller proportion of hostile responses. While these findings agree with the more general findings earlier that children with CDD (SCT) are more socially withdrawn, it also suggests a role of CDD (SCT) in attention and an encoding dysfunction that accounts for impairment in critical social behaviors of a different sort than those seen in ADHD (social intrusion, aggression, bossiness, excessive speech, etc.).

Noteworthy is that the association of CDD (SCT) to social impairment or withdrawal remains even after statistical removal of ADHD symptoms, as well as those of ODD, CD, generalized anxiety disorder, major depressive disorder, and even IQ (see Barkley [2014] and other studies in the special issue of the *Journal of Abnormal Child Psychology* on CDD [SCT]; also Burns et al., 2013). CDD (SCT) and the ADHD-I dimension contribute to variance in social problems and apparently peer neglect, yet their contributions are independent or additive, not redundant (Burns et al., 2013; Willcutt et al., 2014). Similarly, Becker, Luebbe, and colleagues (2014) found that the positive association between CDD (SCT) and general social problems was apparently not due to disruptive social problems given the association noted earlier with significantly lower rates of discipline in inpatient children. This relationship of CDD (SCT) to social withdrawal persists even after researchers control for demographic factors and comorbidity (Barkley, 2014). Thus, CDD (SCT) contributes unique variance to certain areas of social impairment independent of other disorders, including ADHD.

Another domain of impairment linked to ADD – H or the later ADD-I type and probably to the more specific disorder of CDD (SCT) is poor academic performance (Carlson, 1986; Milich et al., 2001). ADD – H has been linked repeatedly across studies with academic performance difficulties, and possibly math difficulties specifically, even if it is not as strongly associated with disruptive behavior in school as is ADHD. Bauermeister and colleagues (2012) found that both CDD (SCT) and ADHD-I were each significantly and independently associated with lower academic achievement scores on testing after they controlled for the other set of symptoms, whereas HI symptoms showed no such relationship. And, as noted earlier, CDD (SCT) symptoms were uniquely associated with deficient math performance. Similarly, Burns and colleagues (2013) found that CDD (SCT) was significantly associated with ratings of academic impairment even after they controlled for ADHD-I symptoms. In contrast, three studies (Becker & Langberg, 2013; Langberg, Becker, & Dvorsky, 2014; Watabe, Owens, Evans, & Brandt, 2014) did not find an association of CDD (SCT) with academic achievement tests after researchers controlled for IQ and ADHD symptoms, or they found it to be rather weak.

Why the disparity across studies? It may arise from the fact that some researchers selected their samples for ADHD first, then within such samples examined those high and low in CDD (SCT) symptoms. This can contaminate any findings for CDD (SCT) with those known to be related to ADHD. Even so, when symptoms of ADHD are statistically removed, CDD (SCT) appears to add unique variance to the prediction of academic problems (Barkley, 2013) and may make unique contributions to written language and reading problems, organization problems, and homework specifically, beyond the contribution of ADHD-I symptoms (Langberg, Becker, & Dvorsky, 2014; Marshall et al., 2014; Willcutt et al., 2014). Difficulties with math performance may also be more evident in CDD (SCT) than in ADHD, although this finding, too, requires replication to be considered reliable (Bauermeister et al., 2012).

In addition to social and academic domains, my own national surveys of children and adults included a measure of 15 domains of impairment (Barkley, 2012b, 2013). Children were sorted into those with CDD (SCT) only, those with ADHD only, and those with both conditions, with the remainder serving as the community control group. Figure 17.1 shows the results. Children with CDD (SCT) were more impaired than controls in all domains; they had more difficulties in community–leisure domains than in home–school (work) domains. In contrast, although children with ADHD were also impaired across all domains, their greatest difficulties occurred in home–school domains. Moreover, ADHD was associated with more pervasive impairment. That is, both ADHD groups (ADHD alone and combined with CDD) experienced impairment in at least twice as many of the 15 domains as did CDD (SCT) cases. The results also indicated that

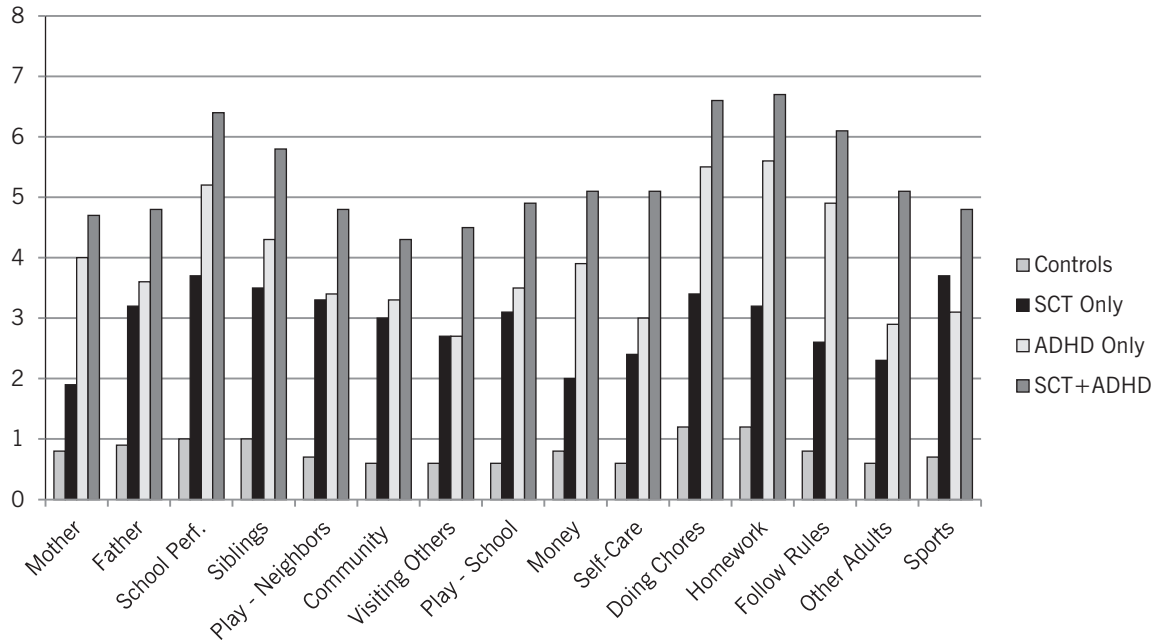

FIGURE 17.1. Comparisons of children with SCT only, children with ADHD only, those with both disorders (SCT + ADHD), and a control group. The figure was created from the results reported in Barkley (2013), from Saxbe and Barkley (2014). Copyright 2014 by Physician's Postgraduate Press, Inc. Reprinted by permission.

ADHD symptom dimensions, especially inattention, contributed markedly more variance to impairment in the home–school domains than did HI or CDD (SCT) dimensions. By contrast, the HI dimension contributed more variance to community–leisure impairments, as did CDD (SCT) but to a far lesser extent. CDD was not found to be more impairing than ADHD in educational settings, at least as rated by parents, consistent with other research discussed earlier. When ADHD and CDD symptoms were regressed onto the community–leisure and home–school impairment summary scores, results indicated that both contributed uniquely to impairment, although ADHD accounted for a greater proportion of variance in each summary score.

The adult survey (Barkley, 2012b) also used a rating scale of impairment in 15 domains more appropriate to adults. The findings appear in Figure 17.2. Both the CDD (SCT)-only and ADHD-only groups were more impaired than the control group but did not differ in this respect in overall mean impairment. A somewhat different pattern was evident for the percentage of domains in which impairment occurred (pervasiveness). Here, both of the ADHD groups (ADHD alone, ADHD + SCT) were impaired in more domains than was the CDD (SCT)-only group and the control group. The results further revealed that the CDD (SCT)-only group was also impaired in more domains than the control adults, but not to the degree that was evident in the ADHD groups. These results are consistent with numerous studies showing that ADHD adversely affects many domains of major life activities relative to clinical and community control groups (Barkley, Murphy, & Fischer, 2008). But they also show that CDD (SCT) is an impairing disorder in adults even if not as much or as pervasively as ADHD. In both of my studies, when comorbid, CDD (SCT) + ADHD disorders were additive; that is, the combination of disorders resulted in far more severe impairment and more domains of impairment than did either disorder alone.

Combs and colleagues have also studied the linkage of CDD (SCT) to some aspects of impairment in large adult community samples (Combs, Canu, Broman,

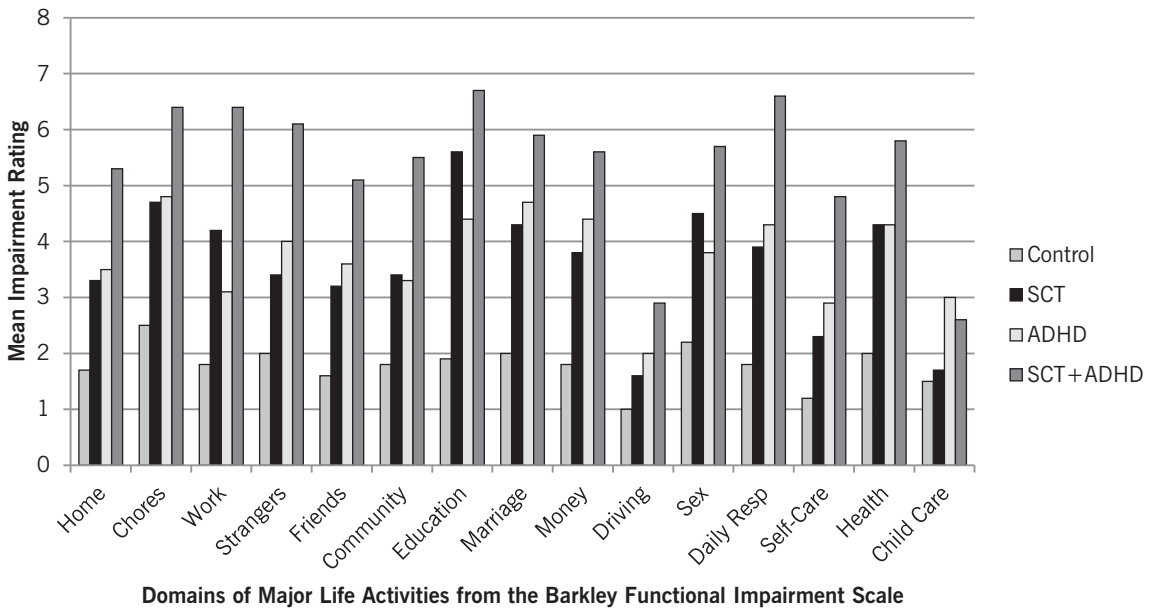

FIGURE 17.2. Comparisons of adults with SCT only, adults with ADHD only, those with both disorders (SCT + ADHD), and a control group. The figure was created from the results reported in Barkley (2012b), from Saxbe and Barkley (2014). Copyright 2014 by Physician's Postgraduate Press, Inc. Reprinted by permission.

& Nieman, in press; Combs, Canu, Broman-Fulks, Rocheleau, & Nieman, 2012). In one study (Combs, Canu, Broman, & Nieuman et al., in press), the authors evaluated the contribution of both ADHD and CDD (SCT) symptoms to a quality-of-life (QOL) measure. The authors found that each set of symptoms contributed unique variance to negative QOL ratings after they controlled for the other set of symptoms, as well as for anxiety, depression, and some demographic factors. The other study (Combs, Canu, Broman-Fulks, et al., 2012) had much the same results for the association of ADHD and SCT with self-reported stress in adults. All of the this suggests that CDD (SCT) is associated with some distinct impairments in various domains of functioning from those associated with ADHD and contributes unique effects to impairment beyond that accounted for by ADHD.

Etiology

Very few studies have examined the etiology of CDD (SCT). A recent study by Moruzzi, Rijsdijk, and Battaglia (2014) examined the heritability of CDD (SCT) using a small set of items of CDD (SCT). CDD (SCT) was found to be substantially heritable, and it shared about half of its genetic contribution with that of ADHD. But CDD (SCT) was less heritable and involved a somewhat greater contribution of unshared or unique environmental factors than did ADHD. Another study indicated that CDD (SCT) may be associated with prenatal alcohol exposure (Graham et al., 2013). It has also emerged as a treatment side effect, along with lower IQ and lower academic achievement in acute lymphoblastic leukemia (Reeves et al., 2007). The demographic factors shown earlier to be linked to CDD (SCT) imply that there may be a greater contributing role for social adversities than may be the case for ADHD. And so it seems that, like ADHD, CDD (SCT) may turn out to have multiple etiologies. Most causes fall in the realm of neurobiological and genetic factors but less strongly than causes for ADHD. We sorely need neuroimaging research, as well as more behavioral genetic and molecular genetic studies on the nature of CDD (SCT) in comparison to other disor-

ders, especially ADHD. However, researchers must take care to control for the overlap of CDD (SCT) with ADHD. Not doing so will contaminate any findings with ADHD-related results.

What Is the Underlying Mental Dysfunction in CDD (SCT)?

As I have discussed elsewhere (Barkley, 2014), it is possible that CDD (SCT) represents a dysfunction in the focus/execute component of attention in Mirsky's (1996) model of attention components or in the vigilance component, as I noted earlier in my discussion of PDV. It is also possible that it is a form of hypersomnia or arousal disorder given that some dimensions of CDD (SCT) identified in past research include symptoms of sleepiness, low arousal or energy, or drowsiness (Penny et al., 2009). But this seems unlikely in view of recent evidence that whereas both CDD (SCT) and ADHD were significantly associated with daytime sleepiness in college students, such sleepiness formed a factor distinct from those representing CDD (SCT) and ADHD (Langberg, Becker, Dvorsky, & Luebbe, in press); so CDD (SCT) is not just another label for hypersomnia, but it does have a significant association with daytime sleepiness even after researchers control for ADHD, anxiety, and depression symptoms.

Might CDD (SCT) be a form of pathological mind wandering (Adams et al., 2010)? Past research suggests that mind wandering is commonplace and advantageous under certain conditions. It arises when performance of a primary task demands little EF capacity and therefore allows the contemplative or problem-solving capacity of the EF system to focus on more salient personal concerns. The latter then becomes a secondary task that is engaged while the individual performs the relatively automatic actions toward familiar goals (primary task) in the environment (Smallwood & Schooler, 2006). When poorly regulated, however, mind wandering can lead to adverse effects on performance of EF tasks (perhaps due to reduced meta-awareness or self-monitoring of goal pursuit, diminished working memory capacity available for pursing the external goals, etc.) (Smallwood & Schooler, 2006). Excessive mind wandering can adversely affect academic performance (Smallwood, Fishman, & Schooler, 2007). It would be worthwhile for future research to investigate this possibility of CDD as a disorder of mind-wandering.

Other possibilities exist. CDD (SCT) could arise from a ruminative/obsessional disorder and perhaps may be a milder variant of obsessive–compulsive disorder. Excessive and recurrent focusing on maladaptive thoughts might well lead to an attentional problem resembling CDD (SCT). Or CDD (SCT) could represent a deficit in motivation, in which the person lacks not only energy but also initiative or self-motivation. I think this is unlikely given that research has not linked CDD (SCT) to deficits in self-motivation, as reflected on EF rating scales in children or adults, once the overlap with ADHD symptoms is statistically removed (Barkley, 2012a, 2013).

DIAGNOSING CDD (SCT)

During the initial evaluation of a child or adult, the suspicion of CDD (SCT) may arise when there are complaints of inattention in the context of few or no symptoms of hyperactivity or impulsivity, and when symptoms of passivity, hypoactivity, and even social withdrawal are evident (Saxbe & Barkley, 2014). Clinicians can also use rating scales that directly assess CDD (SCT) symptoms (for adults: Barkley, 2011a; for children: Penny et al., 2009). There are no official diagnostic criteria for CDD (SCT), but my own research (Barkley, 2013) suggests that if parents endorse at least three or more of the 12 symptoms of CDD (SCT) discussed earlier, and they occur often or more frequently, this represents the 93rd percentile for the population. That is a traditional index of clinical significance and statistical deviance. This, combined with evidence of impairment from the symptoms, could be used for the time being as diagnostic criteria for CDD (SCT) in children. In the case of an adult, the symptom threshold would be five of the nine symptoms used in my study of adults (Barkley, 2012b). When coupled with evidence of impairment in one or more major life activities, such as may be shown on normed rating scales of impairment (Barkley, 2011b, 2012a), one can make a diagnosis of CDD (SCT).

In discussing the diagnosis with a patient or family, it may be helpful to describe the situation between ADHD and SCT as presented earlier. Describe a higher order category of attention disorders under which one can distinguish ADHD and CDD as separate, semidistinct conditions, much as is done now for the supracategory of LD that comprises reading, spelling, math, writing, and related disorders that may exist alone yet also be comorbid. But patients and families should also be told that CDD is not an officially recognized

mental disorder, as yet, and that other professionals are unlikely to be aware of the term or the nature of the condition. They should also be told that much more research is needed before CDD will be admitted into official taxonomies of mental disorders.

TREATMENT OF CDD (SCT)

As with the etiology of CDD (SCT), there exist only a few studies on possible treatments for CDD (SCT). Early studies on stimulants (e.g., methylphenidate, or MPH) for treating ADHD-I type cases did not find them to be particularly effective in improving the inattention linked to CDD (SCT) (Milich et al., 2001). My own study found a modest positive response to MPH, mainly at low doses, but with only 20% of cases remaining on this medication after a double-blind, placebo-controlled trial compared to the vast majority of children with ADHD-C in whom the degree of improvement was greater (Barkley et al., 1990). But no stimulant medication studies have specifically examined children with CDD (SCT).

Only one drug study to date has specifically examined a nonstimulant ADHD medication for treating CDD (SCT) symptoms. Wietecha and colleagues (2013) found that the norepinephrine reuptake inhibitor *atomoxetine* was effective at reducing CDD (SCT) symptoms in patients with both ADHD and dyslexia, ADHD only, and dyslexia only. The reduction in CDD (SCT) symptoms remained evident even after they statistically controlled for the overlap of CDD symptoms with those of ADHD symptoms. The medication also improved CDD (SCT) symptoms in the group with dyslexia only.

What other medications might work? Given the overlap between CDD (SCT) and anxiety and depression, selective serotonin reuptake inhibitors (SSRIs) might be a possible treatment. Would an activating antidepressant (e.g., fluoxetine, sertraline, venlafaxine, or bupropion) reduce the observed sluggishness and boost alertness? No evidence is available yet to address that hypothesis. Some clinicians have used Luvox for management of pathological mind wandering given its effects on obsessional thinking, but it is not clear that such thinking is the case in CDD (SCT). Given that CDD (SCT) is associated with hypersomnia or daytime sleepiness, should one consider investigating the use of antinarcoleptics, such as modafinil? Perhaps but again evidence is lacking. It seems to me that the alpha$_2$ agonist guanfacine XR (extended release) used for management of ADHD might be worth investigating for CDD (SCT), yet its side effects of sleepiness might be counterproductive given the sluggish/sleepy features seen in CDD (SCT).

Just a single study of behavior modification methods has been done to date, and that only with children. Pfiffner and colleagues (2007) found that children with CDD (SCT) symptoms responded well to traditional home and school behavior management methods when specific symptoms of CDD (SCT) were targeted. Although Antshel and Remer (2003) did not use children with CDD (SCT) specifically, their one study of social skills training found that assertion skills of children with ADHD-I type (who are more likely to have CDD [SCT]) improved more than did those of children with ADHD-C type. Yet neither ADHD type improved in other domains of social skills. Cognitive-behavioral therapy has not been shown to be useful for ADHD in children (Abikoff, 1987), but it has proven to be useful for cases of anxiety and/or depression. I believe it may be worth exploring as a possible intervention for CDD (SCT) given the higher than expected comorbidity between these disorders. In view of the distinct symptoms and impairments of CDD (SCT) relative to ADHD, treatments for ADHD cannot be automatically assumed to work for CDD (SCT), nor can those treatments that have failed for ADHD be ruled out for CDD (SCT).

KEY CLINICAL POINTS

- ✓ CDD (SCT), an impairment of attention in hypoactive-appearing individuals, first presents in childhood. It is characterized by a cognitive dimension of symptoms that comprises daydreaming, sleepiness, staring, "spaciness," and mental fogginess and confusion, along with a motor dimension of slow movement, hypoactivity, lethargy, and passivity.
- ✓ The symptom dimensions forming CDD (SCT) are distinct from yet partially correlated with those forming ADHD.
- ✓ To avoid giving offense to patients with the condition and to avoid implying that the cognitive deficit in CDD (SCT) is known, the condition should be called concentration deficit disorder, or CDD.
- ✓ The history of CDD (SCT) in the medical literature

probably dates as far back as Alexander Crichton in 1798 or at the very least, to 1980 and the creation of ADD without hyperactivity in DSM-III (American Psychiatric Association, 1987).

- ✓ At this time, it exists only as a research entity that has yet to debut in any official diagnostic taxonomies of mental disorders.
- ✓ CDD (SCT) is associated with significant impairment, mostly in social impairment, primarily social withdrawal. It also makes some contribution to difficulties with academic performance in children, and even more so in adults. It is associated in adults with impairment in occupational functioning.
- ✓ CDD (SCT) is also significantly associated with risk for internalizing symptoms, especially anxiety and depression.
- ✓ It has no or even a negative relationship to ODD (hence, there is likely no relationship to CD, substance use disorders, or antisocial personality disorder).
- ✓ The etiologies of CDD (SCT) are not well studied, but some evidence suggests a strong heritability to the disorder, but not as much as that seen in ADHD. CDD (SCT) may also be associated with fetal alcohol exposure and with the treatment of acute lymphoblastic leukemia.
- ✓ Evidence supports the view that CDD (SCT) is distinct from ADHD and not a subtype of it. But the two conditions can overlap in nearly half of all cases of each.
- ✓ Future diagnostic taxonomies, such as the DSM, should create a higher order meta-category of attention disorders (ADs) under which one would then break out ADHD and CDD as separate, semidistinct conditions, much like what is done now for the supracategory of LD, rather than continue the mistaken view that CDD/SCT is a subtype of ADHD.
- ✓ Very little research has been done on treatments for CDD (SCT).

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PART II

Assessment of ADHD

CHAPTER 18

Psychological Assessment of Children with ADHD

Russell A. Barkley

My intent in this chapter is not to provide a detailed review of the manner in which a thorough clinical evaluation should be conducted for children with attention-deficit/hyperactivity disorder (ADHD) and their families. Instead, this chapter highlights the major topics that should be covered and several methods that may be used in conducting a psychological assessment. Many such methods exist. Only those approaches that I recommend receive emphasis here.

ASSESSMENT ISSUES

The psychological evaluation of children for ADHD incorporates multiple assessment methods that rely on several informants concerning the nature of children's difficulties (and strengths!) across multiple situations. To accomplish this, parent, child, and teacher interviews should be conducted, parent and teacher rating scales of child behavior and rating scales or surveys of child adaptive functioning should be obtained, and parent self-report measures of relevant psychiatric conditions and of parent and family functioning also should be collected. Some clinicians may wish to collect laboratory measures of ADHD symptoms, as well as direct observations of parent–child interactions, if parent–child conflicts or oppositional defiant disorder are considered to be present. Of course, children with suspected intellectual or developmental delays or learning disabilities (LD) should undergo psychological testing of these domains, if they have not already done so.

There are several goals to bear in mind in the evaluation of children with ADHD. A major goal of such an assessment is to determine not only the presence or absence of psychiatric disorders but also to differentiate between diagnosis of ADHD and other childhood psychiatric disorders. This requires extensive clinical knowledge of these other psychiatric disorders, and the reader is referred to Mash and Barkley's (2014) text on child psychopathology for a review of the major childhood disorders. In evaluating children for ADHD, it may be necessary to draw on measures that are normed in a particular country of residence, with a representative sampling of the various ethnic backgrounds in that general population, if such instruments are available, so as to preclude the over-diagnosis of minority children when diagnostic criteria developed for white children are extrapolated to them.

A second purpose of the evaluation is to begin delineating the types of interventions needed to address the psychiatric disorders and psychological deficits, and academic, adaptive, and social impairments identified

in the course of assessment. As I note later, these interventions may include individual counseling, parent training in behavior management, family therapy, classroom behavior modification, psychiatric medications, and formal special educational services, to name just a few. For a more thorough discussion of treatments for ADHD, see Part III of this text; for treatments for other childhood disorders, the reader is referred to Mash and Barkley (2005).

Another important purpose of the evaluation is determination of comorbid conditions and whether these may affect prognosis or treatment decision making. For instance, high levels of physically assaultive behavior by the child may signal that a behavioral parent training program may be contraindicated, at least for the time being, because of the likelihood of temporarily increasing child violence toward parents when limits on noncompliance with parental commands are established. Or consider the presence of high levels of anxiety specifically, and internalizing symptoms more generally in children with ADHD. Research has shown such symptoms to be arguably a predictor of poorer responses to stimulant medication (see Chapter 27; also Moshe, Karni, & Tirosh, 2012) or of a partial response seen in nonanxious children with ADHD (Blouin, Maddeaux, Firestone, & van Stralen, 2010). Similarly, the presence of high levels of irritable mood, severely hostile and defiant behavior, and periodic episodes of serious physical aggression and destructive behavior may be early markers for later severe disruptive mood dysregulation disorder (DMDD) or, if accompanied by mood swings toward mania, even childhood bipolar disorder (BP; manic–depression) (see Chapter 5). Symptoms of ADHD are almost universal in juvenile-onset BP (Carlson & Meyer, 2006). Such a disorder will likely require the use of psychiatric medications in conjunction with a parent training program that focuses on the management of the ADHD symptoms and especially the high levels of aggressive behavior that may exist in children with DMDD or BP.

A further objective of the evaluation is to identify the pattern of the child's psychological strengths and weaknesses and to consider how these may affect treatment planning. This may also include gaining an impression of the parents' own abilities to carry out the treatment program, as well as the family's social and economic circumstances and the treatment resources that may (or may not) be available within their community and cultural group. One may also need to determine the child's eligibility for special educational services within his or her school district, if eligible disorders, such as intellectual disability, motor developmental delay, LD, or other conditions, are present.

As the previous discussion illustrates, the evaluation of the presence of ADHD is but one of many purposes of the clinical evaluation of children referred for that condition. A brief discussion now follows of the different methods of assessment that may be used in the evaluation of children for ADHD.

ASSESSMENT METHODS

Prior to the Evaluation

When parents call a clinic for an evaluation, a form that may be completed by the receptionist that gathers important demographic information about the child and parents, the reason for the referral, and insurance information that will be cross-checked with the insurance company, when necessary. This form is then reviewed by the billing agent for the clinic and the clinician who receives the case. Depending on the clinician's area of specialization, some types of referrals that may be inappropriate for the clinician's practice can be screened out at this time for referral to more appropriate services.

One can then send out a packet of questionnaires to parents and teachers following the parents' call to the clinic but in advance of the scheduled appointment. In fact, the parents of children referred to clinics I have supervised are not given an appointment date until these packets of information are completed and returned to the clinic. This ensures that the packets will be completed reasonably promptly and that the information is available for review by the clinician prior to meeting with the family, which makes the evaluation process far more efficient in its collection of important information. In these days of increasing cost consciousness concerning mental health evaluations, particularly in managed care environments, the efficiency of the evaluation is paramount, and time spent directly with the family is often limited and at a premium. This packet of information can include a form cover letter from the professional asking the parents to complete the packet of information and informing them that the appointment date will be given when this packet is returned. The packet also contains the General Instructions sheet, a Child and Family Information Form, and a Developmental and Medical History Form, all of which can be found in the clinical workbook by Barkley and Murphy (2006).

These forms and others are also available in Spanish from the publisher. In addition, the packet includes a reasonably comprehensive child behavior rating scale that covers the major dimensions of child psychopathology, such as the Child Behavior Checklist (CBCL; Achenbach, 2001) or the Behavior Assessment System for Children, Second Edition (BASC-2; Reynolds & Kamphaus, 2004). Also in this packet is a copy of the ADHD Rating Scale–IV (DuPaul, Power, Anastopoulos, & Reid, 1998) and the Barkley Functional Impairment Scale—Children and Adolescents (BFIS-CA; Barkley 2012a). The latter is used to evaluate the degree of psychosocial impairment of the child across the 15 domains sampled by this scale. If executive functioning (self-regulation) is an issue in the referral concerns, clinicians can screen this domain using the Barkley Deficits in Executive Functioning Scale (Barkley, 2012a) or the Behavior Rating Inventory of Executive Functioning (Gioia, Isquith, Guy, & Kenworthy, 2000). Finally, parents are sent the Home Situations Questionnaire (HSQ; Barkley & Murphy, 2006) in this packet so as to give the clinician a quick appreciation for the pervasiveness and severity of the child's disruptive behavior across a variety of home and public situations. Such information is of clinical interest because it not only indicates pervasiveness and severity of behavior problems, but it also focuses discussion around these situations during the evaluation and subsequent parent training program. These rating scales are discussed below.

A similar packet of information is sent to the child's teachers, with parental written permission obtained beforehand, of course. This packet does not contain the Developmental and Medical History Form. This packet contains the Teacher version of the CBCL or BASC-2, the School Situations Questionnaire (SSQ; Barkley & Murphy, 2006), and the Teacher version of the ADHD Rating Scale–IV. The Social Skills Rating Scale (Gresham & Elliott, 1990) may also be included and can be informative about the child's social problems in school, as well as academic competence, as quickly screened by this relatively brief scale. If possible, it is quite useful to contact the child's teachers by telephone for a brief interview prior to meeting with the family. Otherwise, this can be done following the family's appointment.

Once the parent and teacher packets have been returned, the family should be contacted by telephone and given an appointment. One can then send out a letter confirming this appointment date, with directions for driving to the clinic. This letter may be accompanied by a detailed instruction sheet entitled "How to Prepare for Your Child's Evaluation" (Barkley & Murphy, 2006), which gives family members some information about what to expect on the day of the evaluation and may set their minds at ease if a mental health evaluation is disconcerting or anxiety-inducing for them.

This preparation leaves the following to be done on the day of the appointment: (1) parental and child interview, (2) completion of self-report rating scales by the parents, and (3) any psychological testing that may be indicated by the nature of the referral (intelligence and achievement testing, etc.).

Parental Interview

Although often criticized for its unreliability and subjectivity, the parental (often maternal) interview remains an indispensable part of the clinical assessment of children. If one were limited to just a single method for psychological evaluation of a child, the parental (maternal) interview, unhesitatingly, would be the method of choice. Whether wholly accurate or not, parental reports provide the most ecologically valid and important source of information concerning children's difficulties. It is frequently the parents' complaints that have led to the referral of the children, that will affect the parents' perceptions of and reactions to the children, and that will influence parents' adherence to the clinician's treatment recommendations. Moreover, the reliability and accuracy of the parental interview hinge on the manner in which it is conducted and the specificity of the questions posed by the clinician. Diagnostic reliability is greatly enhanced by interviewing that includes highly specific questions about symptoms of psychopathology that have been empirically demonstrated to have a high degree of association with particular disorders. The interview must also focus on the specific complaints about the child's psychological adjustment and any functional parameters (eliciting and consequential events) associated with those problems, if psychosocial and educational treatment planning is to be based on the evaluation.

Demographic Information

If not obtained in advance, the routine demographic data concerning the child and family (e.g., ages of child and family members; child's date of birth; parents' names, addresses, employers, occupations, and

religion(s); and the child's school, teachers, and physician) should be obtained at the outset of the appointment. I have also used this initial introductory time period to review with the family any legal constraints on the confidentiality of information obtained during the interview, such as the clinician's legal duty (as required by state law) to report to state authorities instances of suspected child abuse, threats that the child (or parents) may make to cause physical harm to other specific individuals (the duty to inform), and threats that the child (or parents) may make regarding self-harm (e.g., suicide threats).

Major Parental Concerns

The interview then proceeds to the major referral concerns of the parents and of the professional who referred the child, when appropriate. General descriptions of concerns by parents must be followed by specific questions from the examiner to elucidate the details of the problems and any apparent precipitants that can be identified. Such an interview probes for not only the specific nature, frequency, age of onset, and chronicity of the problematic behaviors, but also the situational and temporal variations in the behaviors and their consequences. If the problems are chronic, which they often are, determining what prompted the referral at this time reveals much about parental perceptions of the children's problems, current family circumstances related to the problems' severity, and parental motivation for treatment. A form for collecting such information can be found in the clinical manual by Barkley and Murphy (2006).

Review of Major Developmental Domains

Next, one should review with the parents potential problems that might exist in the child's developmental domains of motor, language, intellectual, thinking, academic, emotional, and social functioning. Such information greatly aids in the differential diagnosis of the child's problems. To accomplish this requires that the examiner have an adequate knowledge of the diagnostic features of other childhood disorders, some of which may present with attention problems or clinical ADHD. For instance many children with autism spectrum disorders or early BP may be viewed by their parents as having ADHD because the parents are more likely to have heard about ADHD and recognize some of the qualities in their children. Questions about inappropriate thinking, affect, social relations, and motor peculiarities may reveal a more seriously and pervasively disturbed child.

School, Family, and Treatment Histories

Information on the school and family histories should also be obtained; the latter includes a discussion of possible psychiatric difficulties in the parents and siblings, marital difficulties, and any family problems centered around chronic medical conditions, employment problems, or other potential stress events within the family. Of course, the examiner will want to obtain some information about prior treatments received by the child and his or her family members for these presenting problems. When the history suggests potentially treatable medical or neurological conditions (allergies, seizures, Tourette syndrome, etc.), a referral to a physician is essential. Without evidence of such problems, however, referral to a physician for examination usually fails to reveal any further useful information for treatment of the child. An exception to this occurs when use of psychiatric medications is contemplated, in which case a referral to a physician is clearly indicated.

Review of Childhood Disorders

As part of the general interview of the parents, the examiner needs to cover the symptoms of the major child psychiatric disorders likely to be seen in children with ADHD (see Chapter 5). These are set forth in the major childhood disorders section of DSM-5 (American Psychiatric Association, 2013). As one means of partially precluding over-identification of psychopathology in minority children, the following adjustment has been recommended. When reviewing the psychiatric symptoms for the childhood disorders with parents, if the parents indicate that a symptom is present, follow up with the question, "Do you consider this to be a problem for your child compared to other children of the same ethnic or minority group?" Only if the parent answers "yes" to this follow-up question should the symptom to be considered present for purposes of psychiatric diagnosis.

It helps if one appreciates the fact that DSM criteria represent guidelines for diagnosis, not rules of law or dogmatic "religious" proscriptions. Some clinical judgment will always be needed in the application of such guidelines to individual cases in clinical practice. For instance, if a child meets all criteria for ADHD,

including both parent and teacher agreement on symptoms, except that the age of onset for the symptoms and impairment is 13 years rather than the 12 years recommended in DSM-5, should the diagnosis be withheld? Given the lack of specificity for an age of onset of 12 years with ADHD, the wise clinician would grant the diagnosis anyway. Some flexibility (and common sense!), then, must be incorporated into the clinical application of any DSM criteria.

Some clinicians have eschewed diagnosis of children entirely, viewing it as a mechanistic and dehumanizing practice that merely results in unnecessary labeling of children. Moreover, they may feel that it gets in the way of appreciating the clinical uniqueness of each case, unnecessarily homogenizing the heterogeneity out of clinical cases. Some may believe that labeling a child's condition with a diagnosis is unnecessary, and that it is far more important, in planning behavioral treatments, to articulate the child's pattern of behavioral and developmental excesses and deficits than to give a diagnosis. Although there may have been some justification for these views in the past, particularly prior to the development of more empirically based diagnostic criteria, this is no longer the case in view of the wealth of research that went into delineating DSM-5 childhood disorders and their criteria. This is not to say that clinicians should not proceed to document patterns of behavioral deficits and excesses because such documentation is important for treatment planning, only that this should not be used as an excuse to omit diagnosis. Furthermore, given that the protection of rights and access to educational and other services under various federal and state laws and regulations may actually hinge on awarding or withholding the diagnosis, dispensing with diagnosis altogether could well be considered professional negligence. For these reasons, and others, clinicians must review in some systematic way with the parent of each referred child the symptom lists and other diagnostic criteria for various childhood mental disorders.

The parental interview may also reveal that one parent, usually the mother, has more difficulty managing the child than the other. Care should be taken to discuss differences in the parents' approaches to management and any marital problems this may have spawned. Such difficulties in child management can often lead to reduced leisure and recreational time for the parents, and increased conflict within the marriage and often within the extended family, should relatives live nearby. It is often helpful to inquire about what attributions the parents may have about the causes or origins of their child's behavioral difficulties because this may unveil areas of ignorance or misinformation that require attention later, during the initial counseling of the family about the child's disorder(s) and their likely causes. The examiner also should briefly inquire about the nature of parental and family social activities to determine how isolated, or insular, the parents are from the usual social support networks in which many parents are involved. Earlier research by Wahler (1980) has shown that the degree of maternal insularity is significantly associated with failure in subsequent parent training programs. Where present to a significant degree, such a finding might augur well for addressing the isolation as an initial goal of treatment rather than progressing directly to child behavior management training with that family.

The parental interview can then conclude with a discussion of the child's positive characteristics and attributes, as well as potential rewards and reinforcers desired by the child that will prove useful in later parent training on contingency management methods. Some parents of children with ADHD have had such chronic and pervasive management problems that, upon initial questioning, they may find it hard to report anything positive about their children. Getting them to begin thinking of such attributes is actually an initial step toward treatment because the early phases of parent training teach parents to focus on and attend to desirable child behaviors.

At a later appointment, the examiner may wish to pursue more details about the nature of the parent–child interactions surrounding the following of rules by the child if behavioral parent training is to be recommended to the parents. Parents should be questioned about the child's ability to respond to commands and requests in a satisfactory manner in various settings, to adhere to rules of conduct governing behavior in various situations, and to demonstrate self-control (rule following) appropriate to the child's age in the absence of adult supervision. To accomplish this, I have found it useful to follow the format set forth in Table 18.1, in which parents are questioned about their interactions with their child in a variety of home and public situations. When problems are said to occur, the examiner follows up with the list of questions from the table. If the parents have completed the HSQ as part of this evaluation, then their responses on that questionnaire can be used as the starting point for this interview, following up each situation endorsed as a problem on that questionnaire with these same follow-up questions.

TABLE 18.1. Parental Interview Format for Assessing Child Behavior Problems at Home and in Public

Situation to be discussed	If a problem, follow-up questions to ask
Overall parent–child interactions	1. Is this a problem area? If so, then proceed with questions 2–9.
Playing alone	2. What does the child do in this situation that bothers you?
Playing with other children	3. What is your response likely to be?
Mealtimes	4. What will the child do in response to you?
Getting dressed/undressed	5. If the problem continues, what will you do next?
Washing and bathing	6. What is usually the outcome of this situation?
When parent is on telephone	7. How often do these problems occur in this situation?
Child is watching television	8. How do you feel about these problems?
When visitors are in your home	9. On a scale of 1 (*no problem*) to 9 (*severe*), how severe is this problem for you?
When you are visiting someone else's home	
In public places (stores, restaurants, church, etc.)	
When father is in the home	
When child is asked to do chores	
When child is asked to do school homework	
At bedtime	
When child is riding in the car	
When child is left with a baby-sitter	
Any other problem situations	

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Such an approach yields a wealth of information on the nature of parent–child interactions across settings, the type of noncompliance shown by the child (stalling, starting the task but failing to finish it, outright opposition and defiance, etc.), the particular management style employed by parents to deal with noncompliance, and the particular types of coercive behaviors used by the child as part of the noncompliance. This may take 30–40 minutes beyond the parental interview described earlier, but is well worth the time invested when it is possible to do so, especially if parent training in child behavior management is likely to be recommended. When time constraints make this problematic, the HSQ rating scale that was developed to provide similar types of information can be used. After parents complete the scale, they can be questioned about one or two of the problem situations using the same follow-up questions as described in Table 18.1. The HSQ is discussed below.

Child Interview

Some time should always be spent directly interacting with the referred child. The length of this interview depends on the age, intellectual level, and language abilities of the child. For preschool children, the interview may serve merely as a time for the clinician to become acquainted, noting the child's appearance, behavior, developmental characteristics, and general demeanor. For older children and adolescents, this time can be fruitfully spent inquiring about the child's views about the reasons for the referral and evaluation, how he or she sees the family functioning, any additional problems the child may have, how well he or she is performing at school, degree of acceptance by peers and classmates, and changes in the family that the child believes might make life happier at home. As with the parents, the child can be queried as to potential rewards and reinforcers he or she finds desirable, which will prove useful in later contingency management programs.

Children below ages 9–12 years do not provide especially reliable reports of their own disruptive behavior. The problem is compounded by the frequently diminished self-awareness and impulse control that is typical of children with ADHD (see Chapter 2). Such children often engage in little reflection about the examiner's questions and may even lie or distort information in a more socially pleasing direction. Some report that they have many friends, have no interaction problems at home with their parents, and are doing well at school, in direct contrast with the extensive parental and teacher complaints about the inappropriate behavior

of these children (see Chapter 7). Because of this tendency of children with ADHD to underreport the seriousness of their behavior, particularly in the realm of disruptive or externalizing behaviors (Barkley, Fischer, Edelbrock, & Smallish, 1991; Fischer, Barkley, Fletcher, & Smallish, 1993), the diagnosis of ADHD is never based on reports of the child. Nevertheless, children's reports of their internalizing symptoms, such as anxiety and depression, may be more reliable and should therefore play some role in the diagnosis of comorbid anxiety or mood disorders in children with ADHD (Chapter 5; also see Pliszka, 2009).

Although noting children's behavior, compliance, attention span, activity level, and impulse control within the clinic is useful, clinicians must guard against drawing any diagnostic conclusions when children are not disruptive in the clinic or office. Many children with ADHD do not misbehave in clinicians' offices, so heavy reliance on such observations would clearly lead to false negatives in the diagnosis (Sleator & Ullmann, 1981). In some instances, the behavior of children with their parents in the waiting area prior to the appointment may be a better indication of the child management problems at home than their behavior toward the clinician, particularly when this involves a one-to-one interaction between the child and the examiner.

This is not to say that the child's office behavior is entirely useless. When it is grossly inappropriate or extreme, it may well signal the likelihood of problems in the child's natural settings, particularly school. It is the presence of relatively normal conduct by the child that may be an unreliable indicator of the child's normality elsewhere. For instance, using data collected in an earlier study of 205 children ages 4–6 (Shelton et al., 1998), we examined the relationship between office behavior and parent and teacher ratings. Of these children, 158 were identified at kindergarten registration as being 1.5 *SD* above the mean (93rd percentile) on parent ratings of ADHD and oppositional defiant disorder (ODD; aggressive) symptoms. These children were subsequently evaluated for nearly 4 hours in a clinic setting, after which the examiner completed a rating scale of their behavior in the clinic. We then classified the children as falling below or above the 93rd percentile on these clinic ratings using data from a normal control group being tested as part of this project. The children were also classified as falling above or below this threshold on parent ratings of home behavior and teacher ratings of school behavior using the CBCL. We found that there was no significant relationship between the children's clinic behavior (normal or atypical) and the ratings by their parents. However, there was a significant relationship between atypical ratings in the clinic and atypical ratings by the teacher, in that 70% of the children classified as atypical in their clinic behavior were also classified as such by the teacher ratings of class behavior, particularly on the externalizing behavior dimension. Normal behavior, however, was not necessarily predictive of normal behavior in either parent or teacher ratings. This suggests that atypical or significantly disruptive behavior during a lengthy clinical evaluation may be a marker for similar behavioral difficulties in a school setting. Nevertheless, the wise clinician will contact the teacher directly to learn about the child's school adjustment rather than rely entirely on such inferences about school behavior from the child's clinic/office behavior.

Teacher Interview

At some point before or soon after the initial evaluation session with the family, contact with the child's teachers is essential so as to clarify further the nature of his or her problems. This is most likely done by telephone unless the clinician works within the child's school system. Interviews with teachers have all of the same merits as do interviews with parents, providing a second ecologically valid source of indispensable information about the child's psychological adjustment, in this case, in the school setting. Like parent reports, teacher reports are also subject to bias. As always, the integrity of the reporter of information, be it parent or teacher, must always be weighed in judging the validity of the information itself.

Many children with ADHD have problems with academic performance and classroom behavior (see Chapter 6), and the clinician needs to obtain details about these difficulties. While this may initially be done by telephone, when time and resources permit, a visit to the classroom and direct observation and recording of the children's behavior can prove quite useful if further documentation of ADHD and comorbid behaviors is necessary for planning later contingency management programs for the classroom. Granted, this is unlikely to prove feasible for clinicians working outside of school systems, particularly in the climate of increasing managed health care plans that severely restrict the evaluation time that will be compensated. But for those professionals working within school systems, direct behavioral observations can prove very fruitful for di-

agnosis, and especially for treatment planning (DuPaul & Stoner, 2003).

Clinicians should also send teachers the previously mentioned rating scales as a packet prior to the actual evaluation, so that the results are available for discussion with not only the parents during the interview but also the teacher during the subsequent telephone contact or school visit.

The teacher interview also should focus on the specific nature of the child's problems in the school environment, again following a behavioral analysis format. The settings, nature, frequency, consequences, and eliciting events of the major behavioral problems also can be explored. The follow-up questions used in the parental interview on parent–child interactions (shown in Table 18.1) may prove useful here as well. Teachers should be questioned about potential LD in the child, given the greater likelihood of occurrence of such disorders in this population. Where evidence suggests their existence, the evaluation of the child should be expanded to explore the nature and degree of such deficits as viewed by the teacher. Even where LDs do not exist, children with ADHD are more likely to have problems with sloppy handwriting, careless approaches to tasks, poor organization of their work materials, and academic underachievement relative to their tested abilities (Chapter 6). Time should be taken with the teachers to explore these problems.

Child Behavior Rating Scales for Parent and Teacher Reports

Child behavior checklists and rating scales have become an essential element in the evaluation and diagnosis of children with behavior problems. The availability of several scales with excellent normative data across a wide age range of children and having acceptable reliability and validity makes their incorporation into the assessment protocol quite convenient and extremely useful. Such information is invaluable in determining the statistical deviance of children's problem behaviors and the degree to which other problems may be present. As a result, it is useful for the clinician to mail a packet of these scales out to parents prior to the initial appointment, and to ask that they be returned on or before the day of the evaluation, as described earlier. This permits the examiner to review and score them before interviewing the parents, allows for elucidation of vague or significant answers in the interview, and serves to focus the subsequent interview on those areas of atypicality that may be highlighted in the responses to scale items.

Numerous child behavior rating scales exist. Despite their limitations, behavior rating scales offer a means of gathering information from people who may have spent months or years with the child. Apart from interviews, there is no other means of obtaining such a wealth of information for so little investment of time. The fact that such scales provide means of quantifying the opinions of others, often along qualitative dimensions, and comparing these scores to norms collected on large groups of children, are further merits of these instruments. Nevertheless, behavior rating scales are opinions and are subject to the oversights, prejudices, and limitations on reliability and validity inherent in such opinions.

Initially, it is advisable to utilize "broad-band" rating scales that provide coverage of the major dimensions of child psychopathology known to exist, such as depression, anxiety, withdrawal, aggression, delinquent conduct, and, of course, inattentive and hyperactive–impulsive behavior. These scales should be completed by parents and teachers. Such scales include the BASC-2 (Reynolds & Kamphaus, 2004) and the CBCL (Achenbach, 2001), both of which have versions for parents and teachers and satisfactory normative information. (The CBCL can be obtained from Thomas Achenbach, PhD, Child and Adolescent Psychiatry, Department of Psychiatry, University of Vermont, 5 South Prospect Street, Burlington, VT 05401. The BASC-2 can be obtained from American Guidance Service, 4201 Woodland Road, Circle Pines, MN 55014.)

In the initial screening of children, more narrow-band scales should then be employed that focus more specifically on the assessment of symptoms of ADHD. For this purpose, I created the Parent and Teacher versions of the Disruptive Behavior Disorders Rating Scale (DBDRS; Barkley & Murphy, 2006), which obtains ratings of the DSM symptoms of ODD, ADHD, and conduct disorder (CD). The fact that the symptom lists for these disorders did not change with the publication of DSM-5 means that these scales remain relevant for the evaluation of children for these disorders.

The pervasiveness of the child's behavior problems within the home and school settings should also be examined because such measures of situational pervasiveness appear to have as much or more stability over time than do the aforementioned scales (Fischer et al., 1993). The HSQ and SSQ (Barkley & Murphy, 2006)

provide a means for doing so, and normative information for these scales is available. The HSQ requires parents to rate their child's behavior problems across 16 different home and public situations. The SSQ similarly obtains teacher reports of problems in 12 different school situations. Both scales are scored the same way to yield two separate scores. The first is the Number of Problem Settings, calculated simply by counting the number of items answered "yes." The second is the Mean Severity Score, calculated by summing the numbers circled beside the items, then dividing by the number of "yes" answers. Again, using the 93rd percentile (1.5 *SD* above the mean) as an indication of clinical significance, scores at or above the following thresholds would be significant:

	Boys		Girls	
Age (years)	No. of problems	Severity	No. of problems	Severity
Home ratings				
4–5	7.3	3.8	6.1	3.4
6–8	9.1	4.1	8.7	3.9
9–11	8.6	4.2	7.5	3.5
School ratings				
6–8	7.4	4.5	4.0	3.1
9–11	7.6	5.1	4.5	2.6

Both the more specialized or narrow-band scales focusing on symptoms of ODD and ADHD in the DBDRS, as well as the HSQ and SSQ, can be used to monitor treatment response when given prior to and at the end of medication trials and parent training programs. When stimulant medication is to be tried, clinicians can also use the Side Effects Rating Scale (see Barkley & Murphy, 2006).

Self-Report Behavior Rating Scales for Children

Achenbach (2001) has developed a Cross-Informant version of the CBCL rating scale that permits direct comparisons of results among the parent, teacher, and youth self-report forms of this popular rating scale. Research suggests that while such self-reports of children and teens with ADHD indicate more deviance than the self-reports of youth without ADHD, the self-reports of problems by youth with ADHD, whether by interview or the CBCL, are often less severe than the reports provided by parents and teachers (Fischer et al., 1993; Loeber, Green, Lahey, & Stouthamer-Loeber, 1991).

The reports of children about internalizing symptoms, such as anxiety and depression, are more reliable and likely to be more valid than the reports of parents and teachers about these symptoms in their children (Achenbach, McConaughy, & Howell, 1987; Hinshaw, Han, Erhardt, & Huber, 1992). For this reason, the self-reports of children and youth with ADHD should still be collected because they may be more pertinent to the diagnosis of comorbid disorders than to the children's defiant behavior itself.

Adaptive Behavior Scales and Inventories

Research has begun to show that a major area of life functioning affected by ADHD is the realm of general adaptive behavior (Chapter 4; also see Roizen, Blondis, Irwin & Stein, 1994). "Adaptive behavior" often refers to the child's development of skills and abilities that help him or her to become a more independent, responsible, and self-caring individual. This domain often includes (1) self-help skills (e.g., dressing, bathing, feeding, and toileting requirements, as well as telling and using time, and understanding and using money); (2) interpersonal skills (e.g., sharing, cooperation, and trust); (3) motor skills (e.g., fine motor [zipping, buttoning, drawing, printing, use of scissors, etc.] and gross motor [walking, hopping, negotiating stairs, bike riding, etc.] abilities); (4) communication skills; and (5) social responsibility (degree of freedom permitted within and outside the home, running errands, performing chores, etc.). So substantial and prevalent is this area of impairment in children with ADHD that Roizen and colleagues (1994) have even argued that a significant discrepancy between IQ and adaptive behavior scores (expressed as standard scores) may be a hallmark of ADHD.

Several instruments are available for the assessment of this domain of functioning. The Vineland Adaptive Behavior Inventory (Sparrow, Balla, & Cichetti, 2005) is probably the most commonly used measure for assessing adaptive functioning. It is an interview, however, and takes considerable time to administer. The CBCL and BASC-2 completed by parents (discussed earlier) also contain several short scales that provide a cursory screening of several areas of adaptive functioning (Activities, Social, and School) in children, but they are

no substitute for the more in-depth coverage provided by the Vineland.

Psychosocial Impairment

As noted in Chapter 2, it is useful to distinguish between the concepts of symptoms and impairments. "Symptoms" refer to the cognitive and behavioral expressions of a disorder by an individual, as is often rated on various child behavior rating scales or as contained in DSM-5. "Impairment" refers to the ineffectiveness of the individual in meeting demands in major life activities and is reflected in the consequences the individual experiences because of the expression of his or her symptoms. The face pages of the CBCL and BASC-2 completed by parents (discussed earlier) contain several short scales that provide a cursory screening of several areas of impairment (Activities, Social, and School). However, if a more detailed picture of psychosocial impairment is desired, clinicians can now use the BFIS-CA (Barkley, 2012b). The scale provides parents a means to rate their child's degree of functional ineffectiveness (impairment) in 15 domains of major life activities, along with additional specific follow-up questions about school, social, and community adjustment. The scale is normed on a representative sample of 1,800 U.S. children ages 6 to 17 years and has quite satisfactory reliability and validity (see manual; Barkley, 2012b).

Peer Relationship Measures

As noted earlier, children with ADHD often demonstrate significant difficulties in their interactions with peers (see Chapter 8), and such difficulties are associated with an increased likelihood of persistence of their disorder (Biederman et al., 1996). A number of different methods for assessing peer relations have been employed in research on children with behavior problems, such as direct observation and recording of social interactions, peer- and subject-completed sociometric ratings, and parent and teacher rating scales of children's social behavior. Most of these assessment methods have no norms and so would not be appropriate for use in the clinical evaluation of children with ADHD. For clinical purposes, rating scales may offer the most convenient and cost-effective means for evaluating this important domain of childhood functioning. The CBCL and BASC-2 rating forms described earlier contain items that evaluate children's social behavior. As discussed earlier, norms are available for these scales that permit their use in clinical settings. The Social Skills Rating System (Gresham & Elliott, 1990) also has norms and a software scoring system, which make it useful in clinical contexts for evaluating this domain of impairment.

Parent Self-Report Measures

It has become increasingly apparent that child behavioral disorders, their level of severity, and their response to interventions are, in part, a function of factors affecting parents and the family at large. As noted in Chapter 7, several types of psychiatric disorders are likely to occur more often in family members of a child with ADHD than in typically developing children. That these problems might further influence the frequency and severity of behavioral problems in children with ADHD has been demonstrated in numerous studies over the past 20 years. As discussed earlier, the extent of social isolation in mothers of behaviorally disturbed children influences the severity of the children's behavioral disorders, as well as the outcomes of parent training. Other researchers have also shown that parental psychopathology and marital discord separately and interactively contribute to the decision to refer children for clinical assistance, the degree of conflict in parent–child interactions, and child antisocial behavior (see Barkley, 2013, Figure 1.4). The degree of resistance to parent training is also dependent on such factors. Assessing the psychological integrity of parents, therefore, is an essential part of the clinical evaluation of children for ADHD, the differential diagnosis of their prevailing disorders, and the planning of treatments stemming from such assessments. Thus, the evaluation of children for ADHD often involves a family assessment rather than just assessment of the child. The clinical assessment of adults and their disorders is discussed in subsequent chapters, but I briefly mention here some of the assessment methods that clinicians have found useful in providing at least a preliminary screening of parents for certain important variables in the treatment of children with ADHD.

The instruments that assess the parents' own adjustment (discussed below) can be completed by parents in the waiting room while their child is being interviewed. They should not mailed out in advance with the other rating scales because the clinician will need to introduce the purpose of these self-report scales briefly to

the parents so as not to offend them by requesting such sensitive information. Typically, I have indicated to parents that a complete understanding of a child's behavior problems requires learning more about both the child and the parents. This includes gaining more information about the parents' own psychological adjustment and how they view themselves in their role as parents. The rating scales below are then introduced as one means of gaining such information. Few parents refuse to complete these scales after an introduction of this type. To save time, some professionals may prefer to send these self-report scales out to parents in advance of their appointment, at the same time as the child behavior questionnaires. If so, be sure to prepare a cover letter that sensitively explains to parents the need for obtaining such personal information. For instance, this letter might include the following statement:

> When completing the questionnaires pertaining to yourself and to other aspects of your marriage and family, please keep in mind that we are not trying to evaluate you. Instead, we are trying to learn as much as we can about the home environment in which your child lives. That home environment is very important in helping us to understand the nature of the problems a child may be experiencing. Having such information allows us to make careful and well-informed recommendations about how best to help your child become more successful and better adjusted both at home and at school.

Parental ADHD and ODD

Family studies of the aggregation of psychiatric disorders in the biological relatives of children with ADHD and ODD have clearly demonstrated an increased prevalence of ADHD in the parents of these children (see Chapter 7; Biederman, Faraone, Keenan, & Tsuang, 1991; Faraone et al., 1993). In general, there seems to be at least a 40–50% chance that one of the two parents of children with ADHD will also have adult ADHD (15–20% of mothers and 25–30% of fathers). The manner in which ADHD in a parent might influence the behavior of a child with ADHD specifically, and the family environment more generally, has not been well studied. Adults with ADHD have been shown to be more likely to have problems with anxiety, depression, personality disorders, alcohol use and abuse, and marital difficulties; to change their employment and residence more often; and to have less education and lower socioeconomic status than adults without ADHD (see Chapters 12 and 13; Barkley, Murphy, & Fischer, 2008). Greater diversity and severity of psychopathology in parents was particularly apparent in the subgroup of children with ADHD and comorbid ODD or CD (Lahey et al., 1988). More severe ADHD also seems to be associated with younger age of parents (Barkley et al., 2008), suggesting that pregnancy during their own teenage or young adult years is more characteristic of parents of children with ADHD than of those without children with ADHD. It is not difficult to see that these factors, as well as the primary symptoms of ADHD, could influence the manner in which child behavior is managed within the family, and the quality of home life for such children more generally. Research suggests that when the parent has ADHD, the probability that the child with ADHD will also have ODD increases markedly (see Chapters 3 and 5). Other studies indicate that ADHD in a parent may interfere with the parenting behavior (Chronis-Tuscano, Raggi, et al., 2008; Johnston, Mash, Miller, & Ninowski, 2012) as well as their ability to benefit from a typical behavioral parent training program (see Chapter 7; Chronis-Tuscano et al., 2011; Sonuga-Barke, Daley, & Thompson, 2002). Treatment of the parent's ADHD (with medication) may result in greater success in subsequent retraining of the parent (Chronis-Tuscano, Seymour, et al., 2008). These findings suggest the importance of determining the presence of ADHD in the parents of children undergoing evaluation for that disorder.

The DSM-5 symptom list for ADHD has been cast in the form of a behavior rating scale, and U.S. norms on more than 1,200 adults, ages 17–81 years, have been collected (Barkley, 2011). This rating scale for adults, the Barkley Adult ADHD Rating Scale, is completed twice—the first time to measure their current behavioral adjustment and a second time to test recall of their own childhood behavior between ages 5 and 12 years. Norms for both current and childhood recall scores are provided in the manual. Clinically significant scores on these scales do not, by themselves, ensure the diagnosis of ADHD in a parent, but they should raise suspicion in the clinician's mind about such a possibility. If so, consideration should be given to referral of the parent for further evaluation and, possibly, treatment of adult ADHD, if necessary.

The use of such scales in the screening of parents of children with ADHD would be a useful first step in determining whether the parents had ADHD. If the

child meets diagnostic criteria for ADHD and these screening scales for ADHD in the parents proved positive (clinically significant), then referral of the parents for a more thorough evaluation and differential diagnosis might be in order. At the very least, positive findings from the screening would suggest the need to take them into account in treatment planning and parent training.

Marital Discord

Many instruments evaluate marital discord between parents. The one most often used in research on childhood disorders has been the Locke–Wallace Marital Adjustment Scale (Locke & Wallace, 1959). Marital discord, parental separation, and parental divorce are more common in parents of children with ADHD (see Chapter 7). Parents with such marital difficulties may have children with more severe defiant and aggressive behavior, and such parents may also be less successful in parent training programs. Screening parents for marital problems, therefore, provides important clinical information to therapists contemplating a parent training program for such parents. Clinicians are encouraged to incorporate a screening instrument for marital discord into their assessment battery.

Parental Depression and General Psychological Distress

Parents of children with ADHD are frequently more depressed than those of typically developing children, and this may affect their responsiveness to behavioral parent training programs. A scale often used to provide a quick assessment of parental depression is the Beck Depression Inventory (Beck, Steer, & Garbin, 1988). Greater levels of psychopathology generally, and psychiatric disorders specifically, also have been found in parents of children with ADHD, many of whom also have ADHD. One means of assessing this area of parental difficulties is through the use of the Symptom Checklist–90—Revised (SCL-90-R; Derogatis, 1995). This instrument has scales that not only assess depression in adults but also measure other dimensions of adult psychopathology and psychological distress. Whether clinicians use this or some other scale, the assessment of parental psychological distress generally, and psychiatric disorders particularly, makes sense in view of their likely impact on the course and the implementation of the child's treatments, typically delivered via the parents.

Parental Stress

Research as early as 25 years ago suggested that parents of children with behavior problems, especially those children with comorbid ODD and ADHD, report more stress in their families and their parental role than those of typically developing or clinic-referred children without ADHD (Breen & Barkley, 1988; Fischer, 1990; Johnston & Mash, 2001). One measure frequently used in such research to evaluate this construct has been the Parenting Stress Index (PSI; Abidin, 1995). The current PSI, a 120-item, multiple-choice questionnaire, yields six scores pertaining to child behavioral characteristics (distractibility, mood, etc.), eight scores pertaining to maternal characteristics (depression, sense of competence as a parent, etc.), and two scores pertaining to situational and life stress events. These scores can be summed to yield three domain or summary scores: Child Domain, Mother Domain, and Total Stress. A shorter version of this scale is available (Abidin, 1995), and clinicians are encouraged to utilize it in evaluating parents of children with ADHD.

Psychological Testing

Given the high likelihood of the coexistence of LD and ADHD in children (Chapter 6), the inclusion of some type of short screening test for the major academic achievement skills (reading, spelling, math) in the evaluation of children with ADHD is quite justified. Should the child obtain scores that suggest some deficiency in any domain, a more thorough battery of academic achievement tests could be administered. And given that most children with ADHD have difficulties in the educational environment, it would also be prudent to determine whether intellectual disability (ID) is a contributing factor to their educational problems, beyond what may be accounted for by their ADHD. As noted in prior chapters, children with ADHD are somewhat more likely to place in the low normal, borderline, or even ID range of intelligence relative to typically developing children. It therefore makes sense to include a brief screening scale of intelligence in the initial evaluation of children with ADHD. Here again, if the child places in the deficient range of the screening scale, a more complete intelligence test could be administered to clarify the extent and nature of this deficiency. While it is certainly possible to administer such screening scales as part of the evaluation, clinicians need to make parents aware that such testing can

also be provided at no expense to the family through the child's public school, if that child is having significant problems with school adjustment and academic performance. The delay often involved in obtaining such testing usually prompts parents to at least agree to a screening evaluation of academic achievement and intelligence after which, if necessary, more complete assessments of these domains could be done through the child's school.

Apart from addressing these two issues (LD, ID), there is little or no reason to administer other psychological or neuropsychological tests to children as part of an evaluation for ADHD. Such tests are not sufficiently accurate in the diagnosis of ADHD to recommend their use for clinical diagnostic purposes even if published articles indicate that there are group differences between samples of children with and without ADHD (see Chapter 4). Such differences cannot serve as evidence to support the use of tests for individual classification purposes (e.g., diagnosing ADHD). A review of this literature clearly shows that only a minority of children place in the deficient range on these tests. Consequently the level of false negatives is simply too high to rely on these tests for making a diagnosis. Hence, normal scores cannot be used to rule out the diagnosis of ADHD. This problem prevails across all forms of tests that have been recommended for evaluating children with ADHD, including continuous-performance tests (CPTs) of attention and inhibition, planning tests (e.g., Tower of London or Hanoi tests), tests of set shifting (e.g., card sorting), working memory tests, measures of activity level, projective tests, or tests of executive functioning. Many such tests were reviewed in the prior edition of this text (Barkley, 2006); they require no further review here given that the conclusions have remained unchanged despite subsequent research on them, as well as the promulgation of new tests in this area. There remains no convincing evidence that such tests are reasonably accurate enough in detecting ADHD to warrant their use in clinical practice as diagnostic tools.

Two additional concerns about psychological testing that were raised in the prior edition of this volume remain cogent today:

> We are concerned about two other issues related to routine administration of extensive, multitest batteries. First, the inclusion of many measures raises the possibility of false positive errors. Because of sequential error, the probability is high that at least several test scores from an array of 30 or 40 will be atypical. The likelihood of overidentification of problems increases further because the psychometric properties for these tests have not been well established for child populations. Therefore, the scattershot quality of comprehensive neuropsychological testing almost guarantees some indication of atypicality.
>
> Our other concern is tied more to economics than to methodology: If one accepts the proposition that most, if not all, of the tests administered in a neuropsychological battery are of dubious diagnostic benefit for ADHD-related decisions, routine testing could fairly be judged by third-party payers as frivolous. Given the nature of the U.S. health system, it is not unlikely that psychological testing in general will be unfairly painted with the same brush. Because psychodiagnostic assessment certainly has a legitimate role in the diagnosis of other childhood disorders, we are concerned that the entire enterprise will be tarnished because of overtesting for ADHD. The exception may be in those instances in which evidence from history and imaging studies are suggestive of brain injury. (Gordon, Barkley, & Lovett, 2006, p. 375)

Clinicians may argue that they derive additional insights into child neuropsychological development and daily functioning by administering neuropsychological tests, so these tests have value in evaluation of a child for ADHD even if they cannot be used to diagnose the disorder. This line of reasoning is questionable, however, in view of the fact that neuropsychological tests, such as those of executive functioning, do not correlate significantly with parent and teacher ratings of such functioning in the child's daily life activities (Barkley, 1991; Toplak, West & Stanovich, 2013). While such tests may have some significant relationship to intellectual functioning and academic achievement, they are not proxies for the direct assessment of the latter abilities. The child would be better served in this case if screening tests for IQ and academic achievement were to be given directly because these would be far more indicative of the child's functioning in those domains than other neuropsychological tests with which they have some modest correlation.

Direct Behavioral Observations

As indicated in the previous edition of this volume, a number of studies support the benefit of incorporating structured classroom observations of children into the diagnostic process (see DuPaul & Stoner, 2003). But I continue to believe that those benefits are not enough

to justify the considerable cost and effort they involve. For most clinicians, formal behavior coding is simply impractical; even if clinicians desire to observe a child in a school setting, insurance carriers are not likely to cover this cost. This leaves it to parents to foot the bill out of pocket, which is also highly unlikely. Thus, I do not review classroom observation methods here. Readers who want to consider instituting an informal observational protocol should refer to Gordon (1995). A more formal approach to behavior coding is available in a previous edition of this book (Barkley, 1990).

The clinician's office does provide an observational opportunity, such as during the psychological testing session. Clinicians have long recorded test session behavior while administering standardized tests, but the behavior assessments themselves have rarely been standardized. Now, both a Test Observation Form (TOF) and a Classroom Observation Form are part of the Achenbach System of Empirically Based Assessment (ASEBA; Achenbach, 2014). Observers rate the child on 125 items (e.g., "fidgets"), based on the narrative observations. As in the other ASEBA instruments (e.g., the CBCL), item scores are summed to make subscale scores, which together form a profile of the child's test session behavior. The TOF's syndrome scales are (1) Withdrawn/Depressed, (2) Language/Thought Problems, (3) Anxious, (4) Oppositional, and (5) Attention Problems. In addition, item scores can be used to obtain scores on Internalizing and Externalizing subscales, as well as a DSM-based ADHD scale. For each subscale, the child's scores are compared with a national sample of normative data to derive standardized *T*-scores. The TOF has demonstrated some promise in several studies using observations of school behavior (McConaughy, Ivanova, Antshel, Eiraldi, & Dumenci, 2009) and clinic behavior (McConaughy, Ivanova, Antshel, & Eiraldi, 2009). Both methods can enhance the amount and quality of data from a psychological evaluation (McConaughy, Antshel, Gordon, & Eiraldi, 2010). But these findings do not support its use as a tool for making the diagnosis of ADHD.

Summary of Assessment Methods

It should be clear from the forgoing that the assessment of children for ADHD is a complex and serious endeavor requiring adequate time (approximately 3 hours) and knowledge of the relevant research and clinical literature, as well as differential diagnosis, skillful clinical judgment in sorting out the pertinent issues, and sufficient resources to obtain multiple types of information from multiple sources (parents, child, teacher) using a variety of assessment methods. Screening for IQ and academic achievement skills can also be done using brief psychological tests of those domains. Where time and resources permit, school personnel can engage in direct observation of ADHD behavior in the classroom. At the very least, telephone contact with a child's teacher should be made to follow up on his or her responses to the child behavior rating scales and to obtain greater detail about his or her classroom behavior problems with ADHD. To this list of assessment methods would be added those others necessary to address the specific problems often occurring in conjunction with ADHD in children.

TREATMENT IMPLICATIONS

A multimethod assessment protocol for ADHD in children will certainly reveal a variety of areas of deficits, excesses, and impairments requiring clinical intervention, and perhaps even more detailed behavioral assessment than has been noted here. The subsequent treatments undoubtedly will be based on those deficit areas found to be the most salient, the most significant to the concerns of the referral agent (e.g., parent, physician, teacher, etc.), or to have the greatest impact on present and later adjustment. Such treatment recommendations may range from simple parent counseling about the disorder in those children found to have no impairments, to residential treatment for those children with BP, DMDD, or CD who have severe, chronic, or even dangerous forms of conduct problems or depression. Between these extremes, treatment recommendations may focus on reducing ADHD symptoms through medication or classroom behavioral interventions and reducing oppositional behavior through direct training in effective child management procedures. Many children with ADHD have peer relationship problems that might benefit from individual or group social skills training, using the innovative approaches discussed by Mikami in Chapter 23, provided that such training were implemented within the school, home, or neighborhood settings in which such skills should be used. The evaluation, in most cases, reveals the need for multiple interventions for the child, or even the other family members, to address fully the issues raised therein. Regardless of the treatments indicated from the initial evaluation, ongoing, periodic reassessment using many

of the methods noted earlier will be necessary to document change (or the lack thereof) throughout treatment, maintenance of treatment gains over time after treatment termination, and generalization (or the lack of it) of treatment effects to other problematic behaviors and environments.

LEGAL AND ETHICAL ISSUES

Apart from the legal and ethical issues involved in the general practice of providing mental health services to children, several such issues may be somewhat more likely to occur in the evaluation of children for ADHD. The first of these involves the issue of custody or guardianship of the child and pertains to who can request the evaluation of the child who may have ADHD. Children with ADHD, and especially those with comorbid ODD or CD, are on average more likely to come from families in which the parents have separated or divorced or significant marital discord may exist between the biological parents. As a result, the clinician must take care at the point of contact between the family and the clinic or professional to determine who has legal custody of the child and particularly the right to request mental health services on behalf of the minor. It must also be determined in cases of joint custody, an increasingly common status in divorce/custody situations, whether the nonresident parent has the right to dispute the referral for the evaluation, to consent to the evaluation, to attend on the day of the appointment, and/or to have access to the final report. This right to review or dispute mental health services may also extend to the provision of treatment for the child with ADHD. Failing to attend to these issues before the evaluation may lead to great contentiousness, frustration, and even legal action among the parties to the evaluation that could have been avoided had greater care been taken to iron out these issues beforehand. Although these issues apply to all evaluations of children, they may be more likely to arise in families seeking assistance for ADHD in a child.

A second issue that also arises in all evaluations but may be more likely in cases involving ADHD is the duty of the clinician to report to state agencies any disclosure of suspected physical or sexual abuse or neglect of the child during the evaluation. Clinicians should routinely forewarn parents of this duty to report when it applies in a particular state *before* starting the formal evaluation procedures. In view of the greater stress that children with ADHD or defiance/ODD appear to cause in their parents, as well as the greater psychological distress their parents are likely to report, the risk for abuse of such children may be higher than average. The greater likelihood of parental ADHD or other psychiatric disorders may further contribute to this risk, resulting in a greater likelihood that evaluations of children with disruptive behavior disorders may involve suspicions of abuse. Understanding such legal duties as they apply in a given state or region and taking care to exercise them properly yet with sensitivity to the larger clinical issues likely to be involved are the responsibility of any clinician involved in providing mental health services to children.

Increasingly over the past two decades, children with ADHD have been gaining access to government entitlements and protections, sometimes wrongly described as "legal rights," that make it necessary for clinicians to be well informed about these legal issues if they are to advise the parents and school staff involved in each case properly and correctly. For instance, children with ADHD are now entitled to formal special educational services in the United States under the Other Health Impaired Category as part of the reauthorization of the Individuals with Disabilities in Education Act (1991) (see Chapters 6 and 24), provided, of course, that their ADHD is sufficiently serious to interfere significantly with school performance. This is commonly known throughout the United States. Less commonly understood is that such children also have legal protections and entitlements under Section 504 of the Rehabilitation Act of 1973 or the more recent reauthorization of the Americans with Disabilities Act (2011) as it applies to the provision of an appropriate education to disabled children (for discussions of these and other entitlements/protections, see DuPaul & Stoner, 2003; Latham & Latham, 1992). And should a child with ADHD have a sufficiently severe disorder and reside in a family with low economic means, he or she may also be eligible for financial assistance under the Social Security Act. Space precludes a more complete explication of these legal entitlements here. Suffice it to say that clinicians working with children who may have ADHD need to familiarize themselves with these various rights and entitlements if they are to be effective advocates for the children they serve.

A final legal issue related to children with ADHD pertains to their legal accountability for their actions in view of the argument made elsewhere (Chapter 16; also see Barkley, 1997) that their ADHD is a develop-

mental disorder of self-control. Should defiant children with ADHD be held legally responsible for the damage they may cause to property, the injury they may inflict on others, or the crimes they may commit? In short, is ADHD an excuse to behave irresponsibly without being held accountable for the consequences of one's actions? The answer is unclear and deserves the attention of sharper legal minds than my own. It has been my opinion, however, that ADHD provides an explanation for why certain impulsive acts may have been committed but does not sufficiently disturb mental faculties to serve as an excuse from legal accountability, as might occur under the insanity defense, for example. Nor should ADHD be permitted to serve as an extenuating factor in the determination of guilt or the sentencing of an individual involved in criminal activities, particularly those involving violent crime. This opinion is predicated on the fact that the vast majority of children with ADHD, even those with comorbid ODD, do not become involved in violent crime as they grow up. Moreover, studies attempting to predict criminal conduct within samples of children with ADHD followed to adulthood have either not been able to find adequate predictors of such outcomes beyond simply earlier levels of conduct problems or disorder or they have found them to be so weak as to account for a paltry amount of variance in such outcomes. And those variables that may make a significant contribution to the prediction of criminal or delinquent behavior more often involve measures of parental and family dysfunction, deviant peer affiliations, social disadvantage, and to a lesser degree, if at all, measures of ADHD symptoms. Until this matter receives greater legal scrutiny, it seems wise to view ADHD as one of several explanations for impulsive conduct but not as a direct, primary, or immediate cause of criminal conduct for which the individual should not be held accountable.

THE FEEDBACK SESSION

The feedback session with parents concludes the diagnostic evaluation. This session should take place after all the direct testing with the child is completed and scored, and after the clinician has reviewed all the data and drawn diagnostic conclusions (the family may need to wait while the clinician makes any necessary collateral phone calls to the school, current therapist, etc.). As with the parent interview, children under age 16 are not generally included in the feedback session, but they may be invited in at the end of the session to be given diagnostic conclusions at a level appropriate to their age and cognitive development.

The first step in the feedback session is to inform parents about ADHD. We generally explain to parents that ADHD is defined as a developmental disorder, not as a mental illness or the result of stress in families. The developmental delay affects the child's ability to regulate behavior, control activity level, inhibit impulsive responding, or sustain attention. In other words, the child with ADHD will be more active, impulsive, and less attentive than other children of the same age.

We then explain that there is no direct test for ADHD—no laboratory test, X-ray, or psychological test that definitely tells us that a child has ADHD. What we have to do instead is collect a lot of information and analyze it statistically. Therefore, everything that we have learned about their child has been scored, and we compare these scores with scores collected on hundreds if not thousands of children of the same age. If their child's scores consistently place him or her at or above the 93rd percentile in the areas of activity level, impulse control, or attention span that suggests ADHD, then this means that the child is having more difficulty than 93 of 100 children of the same age. This is the level of "developmental deviance" that must be established.

The second step is to establish a history consistent with the notion of a "developmental" problem. Do these symptoms have a long-standing history that stretches back over time, for at least the past year — not something that cropped up last week or last month, or something that only came about after a trauma occurred in the child's life.

The third step is to rule out any other logical explanation for the problem. Is there anything else going on that would overrule ADHD as a diagnosis or be a better explanation than ADHD for problems the child is having. We then walk parents through the data we obtained about their child, step by step, so they can see clearly how we reached our diagnostic conclusion. These steps include the following:

- Explanation and results of the ADHD Rating Scale
  - Parent interview responses
  - Parent ADHD Rating Scale
  - Teacher ADHD Rating Scale

- Broad-band scale results
  - Parent versions, especially the Attention Problems Scale and/or the Hyperactivity Scale
  - Teacher version
- Teacher rating scales (such as the Connors Rating Scales or ADHD Rating Scale–IV)
- Parenting Stress Inventory
- Social Skills Rating Scale
- Academic Performance Rating Scale
- Clinic-based testing results (e.g., IQ and achievement testing)

Before any discussion of a treatment plan occurs, parents are asked if they have any questions about the diagnostic process or comments about the conclusions that were drawn. Parents are always asked whether they are surprised that their child was (or was not) diagnosed with ADHD.

By walking parents through the data this way, any confusion can be quickly clarified. Parents should leave the diagnostic interview with the impression that the clinician was comprehensive and competent. This sense of security will help them cope with the grief and disappointment they may experience at being told that their child has a developmental disability, as well as the confidence to follow any treatment recommendations that are made.

In closing, a number of implications for clinical practice seem evident from the earlier chapters of this text, particularly that on parent–child relations (Chapter 7):

- The clinical assessment of children with ADHD must incorporate measures that assess not only child behavior and adjustment but also parent–child interactions, parental psychological status, and marital functioning, if a thorough picture of the socioecological fabric of childhood ADHD is to be more fully appreciated.

- Reference must be made to the developmental context in which the findings from this assessment were obtained. The manner in which these levels of the socioecological system have interacted to result in the family as it now presents must be appreciated. Fault finding within such reciprocal systems is often difficult to prove and needlessly judgmental. One can identify those problems within the family that seem primarily attributable to separate child and parent characteristics without the witch-hunt atmosphere that sometimes occurs in such clinical assessments. Great compassion and empathy are far more useful in both discovering these sources of maladjustment and in understanding their direction of effects.

- In counseling the parents of children with ADHD, it is necessary to separate the causes and mechanisms for the children's ADHD from that of hostile–defiant behavior or ODD and CD. The former is clearly a developmental disorder of behavioral disinhibition associated with neuromaturational immaturity and has a strong hereditary predisposition. Parents therefore cannot be held liable for this developmental disorder. ODD and CD, however, are likely to arise within and be maintained by family characteristics, particularly parental psychiatric factors and conditions of social adversity. These characteristics permit both the modeling of aggressive social exchanges with others and the success of garden-variety aggression in escaping these attacks and unwanted task demands made by others. Consequently, parents can and should be held accountable (not blamed) for many, though not all, of these circumstances and should be strongly encouraged to accept this responsibility and seek mental health services to change them. The treatments for ADHD and for ODD and CD are clearly distinct.

- The clinical treatment of ADHD when it coexists with ODD and/or CD must involve more comprehensive interventions that focus, as needed, on parental beliefs and attitudes, psychological distress, communication and conflict resolution skills, and family systems rather than simply using medication or training parents in child management skills alone. Training in child management, when provided, must concentrate on the inconsistent and often noncontingent use of social consequences within these families, and on increasing the availability of rewards and incentives for prosocial conduct. It must also strive to increase parental involvement and particularly monitoring of child behavior both at home and in the neighborhood if it is to prevent escalation to more serious stages of antisocial behavior. My coauthors describe exemplar programs for each of these treatment approaches in Part III of this volume.

- The families of children with ADHD and ODD and/or CD are likely to require more frequent and periodic monitoring via follow-up visits and reintervention, as the case dictates, than other types of childhood psychological disorders if a significant impact is to be made relative to long-term outcomes of these children.

KEY CLINICAL POINTS

- ✓ The ultimate goal of evaluation of the child with ADHD is to determine the interventions that may be needed to address the child's presenting complaints.
- ✓ The evaluation itself is a process driven by the issues that must be addressed, not necessarily by the methods with which the clinician is most comfortable.
- ✓ Key issues involved in most cases will be (1) presenting complaints, (2) history of those complaints, (3) differential diagnosis, (4) establishing developmental deviance, (5) determining domains of impairment (major life activities affected), (6) clarifying possible comorbidities, (7) evaluating the integrity of the information, (8) documenting parental psychological adjustment and motivation to change, and (9) assessing child and family strengths (and weaknesses) and community resources.
- ✓ The evaluation requires integrating information from multiple sources (parents, teachers, other caregivers and professionals), using multiple means of collecting that information (semistructured and structured interviews, standardized behavior rating scales, the medical examination, and psychological testing as indicated) and surveying multiple domains of major life activities (family, peer, school, and community functioning, among others).
- ✓ Useful psychological testing involves screening of intelligence and academic achievement skills, with subsequent, more thorough testing if patients fail the screens. Other psychological tests, such as CPTs or neuropsychological tests, are not currently able to diagnose ADHD accurately or to predict functioning in daily life activities as rated by parents and teachers (see Chapter 4). They might, in some cases, be useful in helping to define some types of impaired cognitive processes in cases in which other developmental delays or losses of functioning may arise from other causes, such as head injury.
- ✓ A medical examination is useful when (1) prior physical exams are unavailable or outdated, (2) history implies a treatable medical condition, (3) another medical disorder may better account for the presenting complaints, or (4) drug treatment of the child is anticipated.
- ✓ Laboratory testing or other medical procedures are usually unnecessary for purposes of diagnosing ADHD.
- ✓ The parental feedback session that concludes the evaluation is the first step in treatment, providing parents with useful scientific information on the nature, course, outcomes, and causes of ADHD, as well as the treatments that are empirically established or that are unproven and need to be avoided.

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CHAPTER 19

Psychological Assessment of Adults with ADHD

J. Russell Ramsay

An accurate diagnosis of attention-deficit/hyperactivity disorder (ADHD) is the first clinical intervention for individuals seeking an assessment of and treatment for this clinical syndrome. Recognizing that one's longstanding coping and functional difficulties stem from heretofore unrecognized ADHD helps make sense of these confounding frustrations. Moreover, the specific features of ADHD, both symptomatic and functional, provide targets for treatment for which there are evidence-supported medical and psychosocial options (Ramsay, 2010), as later chapters in this book attest, all of which provide hope for change.

The assessment of ADHD is complicated by the fact that there is a great deal of overlap between symptoms of ADHD, particularly distractibility and inattention, and various other psychiatric and medical conditions. What is more, adults with ADHD often present with at least one coexisting disorder. Despite these many potential complications, a comprehensive and thorough evaluation of ADHD will yield sufficient clinical data to accurately determine whether or not the clinical picture for a patient is consistent with a diagnosis of ADHD.

My purpose in this chapter is to review the essential components of a comprehensive, "gold standard" diagnostic assessment for ADHD. There is a great deal of controversy in professional and popular circles about the prevalence rates of ADHD and whether it is being either over- or underdiagnosed. The central issue in this controversy is concern about the misdiagnosis of ADHD, which runs the risk of delaying potentially helpful treatment regardless of the direction of the error. Thus, this discussion is relevant insofar as it provides practicing clinicians with a guiding template for an assessment that specifically targets ADHD and examines other diagnostic possibilities in order to increase diagnostic accuracy.

This chapter presents a step-by-step approach to a specialized evaluation for adult ADHD, starting with an initial screening and culminating with the assessment feedback session at which the evaluation findings and treatment recommendations are reviewed (See Table 19.1). Practicing clinicians will undoubtedly adjust this assessment template to their practice settings, though it is recommended that each section of the template be represented in some form. In addition to elaborating on the different assessment steps, I discuss special issues related to "testing" for ADHD, informing patients when the evaluation results do not support a diagnosis of ADHD, and being alert to the possibility of faking symptoms of ADHD.

However, before I describe the components of the evaluation, it is important to review the current official diagnostic guidelines, symptom criteria, and thresholds

TABLE 19.1. Components of a Comprehensive Psychological Assessment of Adult ADHD

- Clinical interview
  - Review presenting problems and goals for the evaluation
  - Developmental history
    - —Family history
      - Current family constellation and family of origin
      - Pregnancy, birth, early developmental milestones, risk factors, etc.
      - Early childhood development and behavior at home
    - —Educational history
      - Adjustment to kindergarten and grade school (or day care, preschool, etc.)
      - Transition to middle school, high school, and/or technical school
    - —College
      - Transition to and experience in college
      - Transition to and experience in graduate or other professional schools
    - —Occupational history
    - —Social and interpersonal history
- Structured diagnostic interview
- Review of clinical inventories
  - Past and current ADHD symptom checklists (self- and other-report)
  - Adult ADHD inventories (self- and other-report)
  - EF inventory (self- and other-report)
  - Other mood, anxiety, and psychiatric symptom inventories
- Feedback session

that provide the framework for making the diagnosis. In the next section I review the most recent revisions to the diagnostic criteria for ADHD and their effects on the assessment of adults.

DIAGNOSTIC CRITERIA AND SYMPTOM DESCRIPTIONS

DSM-5 Diagnostic Criteria for ADHD in Adults

The current diagnostic criteria for ADHD are set out in the fifth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-5; American Psychiatric Association, 2013; see Chapter 2 for these criteria). This edition represents the first substantial update of psychiatric diagnoses in nearly 20 years. ADHD is included in a section on neurodevelopmental disorders, which is a new category devoted to chronic conditions that affect functioning but manifest differently at various points along a developmental trajectory.

As discussed in Chapter 2, the 18 core symptoms defining ADHD and their wording for inattention and hyperactivity/impulsivity are essentially unchanged from the previous edition, with inattention and hyperactivity/impulsivity categories each comprising the same nine respective symptoms as in the previous DSM edition. There are some new parenthetical exemplars of symptoms, specifically to provide age-appropriate illustrations of ADHD in adult life. The term "presentations" is used instead of "types" to distinguish the different symptom constellations (i.e., predominantly inattentive, predominantly hyperactive/impulsive, and combined presentations). This change acknowledges that the different ADHD symptom clusters do not indicate clear, distinct types.

The most long awaited change is that the age-of-onset criterion for symptoms has been raised from 7 years old to 12 years old, with the stipulation that "several" (instead of "some") relevant symptoms be present by then. The requirement is not that full diagnostic criteria must be met or that impairments must necessarily be experienced by that age, but that there is compelling evidence that a cluster of symptoms has emerged. From a clinical standpoint, an evidence-based case can be made that an even older age of onset, at least up to age 16 years (Barkley, Murphy, & Fischer, 2008; Faraone et al., 2006; Polanczyk et al., 2010), can be adopted without sacrificing diagnostic accuracy.

The symptom threshold for diagnosing ADHD in adults has been adjusted downward to be more consistent with the symptom presentations of adults. Whereas DSM-IV required the presence of at least six of nine symptoms of at least one of the subtypes to fulfill diagnostic criteria, regardless of the age of the patient, the current threshold for adults is adjusted downward to five of nine symptoms on at least one of the symptom domains and impairment in multiple settings over the past 6 months. In fact, a threshold of four of nine symptoms has been shown to be sufficient to reliably differentiate between adults endorsing symptoms at a clinically significant level (i.e., 1.5 standard deviations above the mean) and controls (Barkley, 2011a; Barkley, Fischer, Smallish, & Fletcher, 2002; Barkley et al., 2008), though the current modification is a step in the right direction.

Considering that the diagnosis of ADHD and related difficulties frequently plays a role in disability determinations, assessment of the need for academic or work-

place accommodations (see Chapter 32) and for determining level of treatment, the introduction of severity specifications in DSM-5 is a positive development. In addition to identifying the symptom presentation of ADHD, a severity level is assigned, namely, mild, moderate, or severe symptom and functional impairments. ADHD also can be denoted as "in partial remission" to reflect clinically significant residual symptoms in cases where full criteria had previously been met.

DSM-5 guidelines urge evaluators to corroborate self-reports of patients by gathering ancillary information about the childhood onset of symptoms in light of difficulties inherent in this sort of historical recall, as well as significant disparities that can occur in this disorder between self- and other-reports (Barkley, Knouse, & Murphy, 2011). In fact, seeking corroborative information has long been considered a valuable part of the assessment of adult ADHD (McGough & Barkley, 2004). In addition to past school records or reports, feedback from people who know the patient help an evaluator establish the onset and persistence of symptoms in multiple domains of life. Finally, pervasive developmental disorder has been eliminated from the exclusion criteria, thereby recognizing that ADHD and autism spectrum disorders may coexist.

DSM-5 provides the list of symptom criteria with corresponding exemplars to aid clinicians. However, the symptoms of ADHD have wide-ranging effects on most, if not all, domains of adult role functioning (see Chapters 11 and 12). Hence, I provide in the next section a more detailed discussion of symptom manifestations of adult ADHD relevant to the clinical interview.

Assessing Symptoms of ADHD in Adults during the Clinical Interview

There are limitations to existing diagnostic criteria despite DSM-5 revisions. The symptoms themselves are still based on those originally established for children and adolescents ages 4–17 (McGough & Barkley, 2004). It can be argued that a distinct set of symptom criteria for adults should be used. Studies of adults have indicated that there is overlap of some essential symptoms in the extant criteria, such as distractibility. However, separate, developmentally appropriate symptoms for adults have emerged from research, such as driving a car too fast, cognitive inflexibility, and poor emotional management (Chapter 2; also see Barkley et al., 2008; Fedele, Hartung, Canu, & Wilkowski, 2010; Kessler et al., 2010).

Symptoms of executive dysfunction and their implications for ADHD are particularly underrepresented in the official diagnostic criteria. ADHD is increasingly understood as a neurodevelopmental disorder of impaired executive functioning (Chapters 3 and 10; Barkley, 1997; Brown, 2013) and motivational deficits (Volkow et al., 2009), among other downstream difficulties that stem from various brain networks (Castellanos & Proal, 2012). There have been many attempts to define the executive functions (EFs), with most clustering around issues of higher order cognitive skills that aid in problem solving and self-control. Contemporary definitions relevant to ADHD focus on EF as self-regulation (Barkley, 1997, 2012b). Even more relevant for the assessment of ADHD, EFs allow individuals to plan, enact, and organize behaviors over time to achieve personally relevant and desired goals for which there will be benefit in the future but not necessarily immediate rewards (Barkley, 2012b). In fact, many presenting problems that lead individuals to seek out a specialized assessment for adult ADHD represent functional difficulties stemming from EF deficits.

The combination of DSM-5 criteria and EF domains provide an assessing clinician with a framework for identifying examples of ADHD symptoms throughout the evaluation. A good clinical interview is a valuable tool that on the one hand helps bring to light a tapestry of symptoms and difficulties across time and situations in cases of ADHD; on the other hand, it reveals a dearth of these connections in cases that are not consistent with ADHD. The next sections provide examples of symptoms within the different presentation domains, some of which are presented in Table 19.2 (see also Chapters 2 and 10).

Inattention

The domains inattention, distractibility, and poor sustained concentration are the more persistent across development (Larsson, Dilshad, Lichetenstein, & Barker, 2011) and are often the primary source of difficulty for adults with ADHD. Distractibility is the most predictive symptom of ADHD (Barkley et al., 2008), though it is common to other disorders, too. Distractibility is a somewhat different form of inattention insofar as it represents punctuated attention or difficulties screening out interference. Adults with ADHD are sensitive to distraction and have a much harder time reengaging in a task once their focus has been disrupted. The sources of these disruptions are often external stimuli,

TABLE 19.2. Examples of Each of the Common ADHD Symptom Domains

Inattention	Hyperactivity–Impulsivity	Sluggish Cognitive Tempo
Easily distracted by sounds, movements	Fidgety, frequent shifting	Lethargic, slow moving
Problems resuming tasks after distraction	Tapping fingers or pen, wagging foot, playing with items	Underactive, low energy
Poor concentration during conversations, meetings, lectures, etc.	Discomfort with sedentary, confining tasks	Difficulties getting engaged in a setting "mind wandering," "daydreaming"
Difficulties getting started on and following through on tasks, procrastination	Mental restlessness, juggling several ideas but not following through on any	"Sleepy," easily tired
Forgetfulness, misplace or lose items, late for deadlines	Starting projects but not finishing them	Problems staying awake, alert if bored
Difficulties with focus and comprehension when reading	Saying things without thinking or that are inappropriate to setting, disinhibited	Slow mental processing
Daydreamer, "gets lost" in thought	Making promises but then becoming overcommitted, "impulsive compliance"	Easily confused or mentally overwhelmed
Disorganization	Impulsive spending, substance use	Likely to "shut down"
Run out of steam, low attention vigilance	Difficulties managing discomfort, boredom	Anxiety
Require longer than average to complete tasks	Problems "turning off brain"	
Underperformance relative to abilities		

such as distracting noises, witnessing activity outside a window, or any trigger that interrupts and diverts attention. However, distractions can also be internal, such as tangential thoughts, remembering another task, hunger or fatigue, or an internal sense of restlessness. It is not the presence of these distractions that is diagnostic, but persistent difficulties efficiently screening and managing them.

The list of inattention symptoms also includes issues related to poor follow through on tasks, disorganization, and issues related to memory and working memory. These attention difficulties often contribute to the academic difficulties that are common presenting problems for children and adolescents with ADHD and are recalled by adults during an assessment. Even if their grades were solid, if not quite good, adults with ADHD often remember underperforming in school, getting by with inadequate study habits, or devoting inordinate time and effort to keep up with assignments. When assessing for childhood emergence of these symptoms, it is useful to ask about the ability to pay attention in class at different levels of education and focus on and reasonably comprehend assigned readings. Adults with ADHD often remember being described as "daydreaming," "zoning out," and being "spacey." In fact, postsecondary education presents distinct challenges for young adults with ADHD who had not been previously diagnosed and may be the point at which they experience impairment from unrecognized symptoms of ADHD.

Though challenging for students with ADHD, the academic year provides many endpoints for classes or semesters and the opportunity to start with a "clean slate." For "free range" adults, though, there are no such endpoints that provide an opportunity to start over fresh. On the contrary, there are increased expectations of being able to maintain sufficient focus and follow through on tasks over the long haul, such as at work, in relationships, and as a parent. Personal health and financial affairs require similar endurance of effort to manage effectively.

Some of the more common signs of inattention and poor concentration are the inability to engage in reasonably sustained reading, particularly for required or technical reading. Adults describe losing focus or being distracted during conversations and when working on tasks that require a measure of persistence, such as paperwork or household chores. These attention problems interfere with taking steps needed to become engaged in a task, sustaining focus and effort to reach a reasonable stopping point, being able to complete a task or project by a deadline, or some combination of these issues. Poor attention interacts with poor memory to result in forgetting and not following through on promises, which has been cited as a source of conflict in ADHD-affected marriages (Pera, 2008; see Chapter 34).

These aforementioned attention difficulties contribute to the common complaints of procrastination and disorganization. "Procrastination" refers to a maladaptive task delay despite the recognition of negative outcomes (Steel, 2007). This sort of avoidance may stem from poor foresight and planning and likely interacts

with negative anticipations or at least some degree of discomfort associated with a task. This discomfort creates an emotional distraction, which makes it harder to get started. Hence, adults with ADHD describe putting off tasks until the "last minute," relying on a deadline pressure to "motivate" them to follow through. It is common for patients to describe examples of being aware of a task priority, such as a work or school project, but having problems working on it, instead escaping into pleasurable distractions or lower priority errands or chores that make them feel relief in the short-term but avoid their primary objective.

"Disorganization" refers to problems keeping track of plans, items, and time. Thus, common complaints involve misplacing or losing important items. Adults with ADHD often describe some form of "messiness" in their lives, such as desks or offices, purses, briefcases, or book bags, and specific rooms in their homes that are described as a "disaster." These difficulties create impairments, such as late fees for missed payments, replacement of lost items, or devoting inordinate time and stress to looking for missing items, such as keys or cell phones. In addition to generating complications that could have been avoided, disorganization also interferes with problem-solving skills, a combination that leads to a global sense of inefficiency in managing the demands of adult life.

Time management involves organizing and tracking behavior over time. Thus, it is common for adults with ADHD to describe being late for appointments and commitments, and simply "losing track of time" in various ways because of their poor internal sense of time and poor external coordination of time and behaviors. Patients describe having difficulties tracking commitments and being able to enact plans, including social and recreational ones, repeatedly "running out of time" for an opportunity, or becoming overcommitted. Without a firm, external deadline, such as is the state of affairs for many household projects or a dissertation, common complaints include tasks going undone, or at least being completed much later than anticipated, and often under the duress of a looming consequence.

Although treated as a separate symptom domain, inattention and distraction often coexist with restlessness and poor impulse control. In fact, there seems to be a developmental progression in which hyperactivity symptoms are more prominent in childhood and the inattentive symptoms, and probably the impulsivity items, become more pronounced with increased age (see Chapter 9). The combination of these symptoms helps explain why adults with ADHD have such problems managing the distractions and temptations that all people face.

Hyperactivity–Impulsivity

The hyperactivity–impulsivity domain of ADHD symptoms is associated with behavioral disinhibition that is characteristic of the disorder. Hyperactivity is probably considered the quintessential symptom of ADHD, at least in children, but hyperactivity rarely exists without concomitant inattention. *Hyperactivity* refers to excessive activity that is developmentally and contextually inappropriate. For children with ADHD, hyperactivity refers to being "on the go" and having physical restlessness that is disruptive to self and others. Frequently shifting around in one's seat, getting up and moving around, or bouncing legs or tapping fingers are common symptom manifestations that may be present in adults.

Overt motoric hyperactivity diminishes with age due to ongoing brain development, a factor that contributed to the now antiquated view that children with ADHD will "grow out of it" by late adolescence. Although adult hyperactivity does not manifest in the same way it does in children, adults with ADHD often describe an internal sense of restlessness, both physical and mental. There may be more subtle manifestations of overt activity, such as bouncing one's foot, tapping a pen, playing with items in one's hands, or finding excuses to get up and move around that are appropriate to the setting (e.g., get coffee), that still end up being disruptive for the individual. Many adults with ADHD have a propensity to be "busy," "in motion," engage in "thrill seeking," or at least unable to stay with one activity for very long; the key indicator of impairment is that they do not complete tasks or their excess activity diverts time and effort away from higher priority tasks. Moreover, adults with ADHD describe physical discomfort in situations in which they feel confined, such as sitting through a meeting with the internal reaction "I'm going to explode," which interferes with paying attention during the meeting. This restlessness may be more pronounced when they face tasks that are cognitively (i.e., attention) challenging, which results in the tendency to escape into more enjoyable (or at least less uncomfortable) tasks.

"Mental hyperactivity" is often described as contending with numerous or "racing" thoughts and ideas or following an interesting train of thought that is tangential and a distraction from the current situation.

Persons with ADHD describe the impulse to act on a thought or a task while it is in their mind or else they will forget it later. They often report juggling several interesting ideas or projects in their minds but being unable to turn them into action or achieve some sort of outcome. Even when trying to relax, an adult with ADHD may describe the inability to "turn off his or her brain," which is distracting, particularly when one is trying to sleep.

"Impulsivity" can be described as acting (reacting) without thinking. Impulsivity problems often appear to be poor judgment, such as spending money beyond one's budget, making unwise decisions, or not considering the long-range consequences of an action. Impetuous decisions result in procrastination, such as playing video games with a roommate rather than studying for an examination. Verbal impulsivity is an issue for many adults with ADHD, such as talking over other people or saying "the wrong thing" in a situation due to not considering the context and potential consequences. Impulsive compliance, commonly known as the inability to say "No," plays a role in becoming overcommitted and making promises that ultimately are not kept.

Although it is not included in the official diagnostic criteria, emotional management can be considered to be associated with impulse control. That is, an impulse can be thought of as a predominant emotional reaction to a situation. It is often the case for adults with ADHD that it is not the experience or valence of an emotion but rather the expression of the emotion in speech or behavior that is problematic. Anger and frustration management are common emotional challenges for adults with ADHD, particularly in relationships and the workplace. Individuals with ADHD often describe becoming quickly and intensely upset with a situation or a person and reacting strongly but soon thereafter expressing regret for the response in recognition of their overreaction. Persons with ADHD are predisposed to disengage from frustrating though important tasks and instead gravitate to more enjoyable or simply less uncomfortable tasks. Thus, a student with ADHD facing the prospect of going to the library to work on a 10-page essay quickly agrees to drive a roommate to the airport instead, although others are available to do so. On the other hand, positive emotions may trigger impulsive actions that are ultimately self-defeating, such as deciding to skip work or classes to get an early start on a weekend trip.

Although falling under the umbrella of ADHD, there is wide variability in symptom presentations and severity. The next sections focus on additional factors related to ADHD that are pertinent to an assessment.

Sluggish Cognitive Tempo (Concentration Deficit Disorder)

A subset of inattention-type symptoms deemed sluggish cognitive tempo (SCT; see Chapter 17) represents a set of features that is distinct from but overlaps with ADHD (Barkley, 2012a). SCT is characterized by "daydreaminess," sleepiness, low energy, hypoactivity, lethargy, mentally fogginess or being easily confused, trouble staying alert, and slow cognitive processing. Whereas in ADHD inattention appears as disrupted attention or poor vigilance, SCT seems to disrupt the ability to orient or engage one's attention. It might also be considered a problem of poorly regulated "mind wandering" that interferes with attending to the immediate setting, which may contribute to observations that SCT is associated with EF deficits in the domains of self-organization and problem solving in adults (Barkley, 2012a). There is much less, if any, disinhibition and very little evidence of executive dysfunction in SCT than is seen in ADHD; instead, anxiety and social withdrawal are more common. SCT and ADHD commonly coexist, and each makes a distinct contribution to functional impairments.

None of the aforementioned symptoms by itself is sufficient evidence of the diagnosis of ADHD; however, a sustained pattern of multiple examples of these features across time and situations create impairments in functioning that are characteristic of ADHD. As I mentioned earlier, the executive dysfunction model of ADHD provides a scientifically sound and clinically useful framework for understanding the syndrome, which is useful in the assessment process. The next section reviews EFs and adult ADHD.

Executive Functions

As I mentioned earlier, ADHD is increasingly understood as a developmental disorder of impaired EFs (Barkley, 1997, 2012b; Brown, 2013). Moreover, self-regulation (i.e., intact EF) provides the ability to define, organize, and enact plans across time, often in concert with others and using social means, to achieve delayed though personally desirable benefits whose reward is delayed and perhaps even entails a short-term cost (Barkley, 2012b). After hearing this definition of EFs during his ADHD evaluation feedback session, a

patient slapped his knee and said, "That's it! My boss always tells me that if he needs something from me in 10 minutes, he gets it in 5 minutes; if he needs it from me in 2 weeks, it takes me a month."

EFs have been demonstrated to be a reliable diagnostic indicator of adult ADHD (Kessler et al., 2010; also see Chapter 10) although they continue to be underrepresented in the official diagnostic criteria. Although EF is often found to represent a single large factor in factor analyses of various measures (probably self-regulation or future-directed behavior), the semidistinct EF domains that have been identified in ratings of everyday life activities are (1) time management, (2) organization and problem solving, (3) self-control (i.e., inhibition), (4) self-motivation, and (5) emotional management (Barkley, 2011b). These categories help clinicians to target their review of symptoms and impairments during the assessment. That is, many patients with ADHD describe having difficulties organizing and pacing their behavior across time to meet deadlines (time management) or coordinating information and options for handling real-world problems and decisions (organization and problem solving). People with ADHD also report having problems succumbing to impulses and deferring gratification (self-control), getting started on tasks when there is not an immediate or external pressure to do so (self-motivation), and keeping their emotions in check (emotional regulation).

The EF model provides clinicians a framework with which to listen to and conceptualize the presenting complaints of adults seeking an evaluation for ADHD. These domains also provide categories to guide probes about symptoms and functioning to make sure all bases are covered and to differentiate cases of ADHD from those better explained by other factors. Together the symptom and EF descriptions help clinicians make sense of the myriad data that are presented in order to increase diagnostic accuracy. In the next section I review the different steps of the evaluation, from initial screening to the feedback session.

ADULT ADHD EVALUATION: STEP BY STEP

Screening

When a request is made for an adult ADHD evaluation, it is advisable to perform a brief telephone screening to rule out any obvious contraindications. The World Health Organization's Adult ADHD Self-Report Scale (ASRS; Adler, Kessler, & Spencer, 2003) is an 18-item symptom checklist. The first six items of the scale provide a reliable, stand-alone screening measure for ADHD that can be easily administered by phone. Each item is rated on a 5-point Likert scale from 0 (*rarely*) through 4 (*very often*). A total scores of 11 or more is highly predictive of a subsequent diagnosis of ADHD. Scores falling below this threshold should raise questions about the motives behind the request for an ADHD evaluation or at least warrant further probing of the rationale for seeking this sort of evaluation. The screening items of the ASRS (and complete scale) are in the public domain and readily available online.

In addition to looking for at least some indication of the presence of ADHD while speaking by phone, including past diagnoses, assessments, or treatment for ADHD, it is useful to screen for other issues that might preclude an ADHD evaluation on clinical grounds. Explicitly asking about active substance abuse, past and current prescribed medications, psychiatric hospitalizations, self-harming behavior, arrests/legal problems, suicidal ideation, aggression toward others, and symptoms of psychosis provides important clinical information about a caller's assessment needs. This information helps to determine whether it makes sense to proceed with an ADHD evaluation or provide a referral when there are other, priority clinical issues and/or scant evidence of ADHD.

Home Packet

Once an appointment for an ADHD evaluation has been set, it is useful to send out a packet of information about the evaluation (or provide instructions for accessing this information and forms online, if they are available electronically). A cover letter with the date and time of the scheduled appointment, contact information for questions or problems, as well as driving directions or parking advice, addresses common logistical issues.

An adequate assessment of ADHD requires obtaining extensive symptom ratings and developmental information. Hence, self- and observer-report ADHD symptom checklists (both retrospective accounts of childhood and current symptom forms), adult ADHD clinical inventories, additional self-report clinical inventories (e.g., mood, anxiety), and life history questionnaires are sent along in the packet to be completed and brought to the evaluation appointment to help save time during the interview.

Impressions on rating scales completed by people who know the patient provide useful ancillary data. Pa-

tients can obtain these ratings over the phone if family or significant others do not live nearby. Of course, there are cases in which such reporters are unavailable or deceased, or there may be valid clinical reasons that patients do not wish to contact them. Whenever possible, a patient is encouraged to provide copies of previous evaluation reports, school or work records, legal or driving records, or any other pieces of information that provide a "paper trail" of his or her developmental course and particularly of impairments.

Evaluation Appointment

After arriving for the evaluation appointment and completing any remaining administrative tasks or forms, the patient is greeted by the clinician and taken to the consulting room. The clinician quickly peruses the home packet to make sure forms are fully completed. In some cases, the second side of a double-side inventory will be left incomplete or individual items are not answered because the person forgot to go back and finish them, and so forth. If too many forms are incomplete, the patient is encouraged to complete and return them before the evaluation can be considered complete, ideally in the waiting area after the meeting.

It is fairly common for family members or significant others to accompany a patient to the assessment appointment. With the patient's permission, such guests are encouraged to participate in the evaluation. A common format is to have the significant other participate in the open-ended interview during which presenting issues, developmental history, and the goals for the evaluation are reviewed. Parents or guardians accompanying young adults to the evaluation often provide especially useful information about family and developmental histories.

The significant other can be excused during the structured diagnostic interview, though often a patient will invite him or her to stay throughout the process. In the latter case, it is advisable to spend at least a few moments alone with the patient to double-check some important clinical issues that she or he may not wish to disclose in front of family members (e.g., substance use, suicidal ideation).

Clinical Interview and Developmental History

Although it is performed within the context of an evaluation for ADHD, a good clinical interview is the cornerstone of a good psychological assessment of any sort. Patients vary regarding their experience with mental health services and formats, so it is helpful to orient them to the assessment process, the types of questions that will be asked, and any testing that may be performed. Providing a framework for how long the assessment will take, opportunities for breaks, different staff with whom the patient will interact, and time frames for a feedback session and any follow-up documentation will cover most matters, though questions are invited throughout the process.

PRESENTING PROBLEMS

The interview starts with a review of the presenting problems and other circumstances that led the individual to seek the evaluation, including the specific request for an ADHD evaluation. Starting an evaluation with an open-ended question (e.g., "What led you to be here today and how are you hoping I can be helpful to you?") allows the individual and anyone else in attendance to tell the story of the presenting issues. The interviewing clinician explores in greater detail the presenting problems, their history, and previous attempts to address them, either through the patient's own efforts or professional services. The expression of these problems in various domains of life is explored to determine whether they are wide-ranging or reflect circumscribed problems (e.g., "You described that procrastination at work led you to be here today. Are there any other areas of your life where procrastination causes problems for you?"). The clinician gathers important information during this phase and from it generates hypotheses about the presence of ADHD and other diagnoses that will guide later inquiry.

After gaining an adequate understanding of the presenting problems, and before moving on to the developmental history, it is useful to clarify the goals for the evaluation. When the assessment is part of the intake procedure for pursuing treatment, the broad treatment goals commonly voiced by patients are refined into more specific behavioral targets, as much as is possible (i.e., "What changes that you would hope to see in the next 8 weeks or so in your daily life would let you know you are doing better? What is a specific way that you will know you are procrastinating less?"). When the patient is seeking the evaluation for another reason, the goals should be clarified, as should any logistics in terms of when a report can be expected, with whom it will be shared, and so forth.

FAMILY HISTORY

After the presenting issues and goals for the evaluation have been addressed, the clinician moves on to the developmental history, starting with the current family situation. In order to shift into this phase of the assessment, the interviewer can use a transition statement, such as "You have given me a lot of good information about what brings you here. Now, let me now get some important background information about you."

A genogram provides a good way to gather and organize useful information about the family history. The current living situation and family structure offer a good starting point. For young adults, the family includes parents and/or caregivers and siblings; for adults, the discussion mainly focuses on a current partner/spouse, children, and parents. This degree of family background usually provides adequate family medical, psychiatric (including family history of ADHD or learning differences), and substance use histories, and explores any relevant issues that may have influenced the patient.

After obtaining information on family background, the focus returns to the patient's developmental history, starting with information about birth and any prenatal adverse events. Premature birth or, more specifically, associated low birthweight is linked with a higher risk for ADHD (Nigg, 2006). Prenatal risk factors, such as maternal smoking or alcohol and substance use, are explored. Finally, the patient is asked about achieving developmental milestones, as best as he or she can recall them.

A useful first step in assessing for emergence of symptoms during childhood is to ask whether there are any family stories or recollections about the patient's behavior during the preschool years at home or in day care. There may be descriptions of "always getting into things" or being "kicked out of day care" that contribute to the diagnostic tapestry.

EDUCATIONAL HISTORY

Knowledge of the patient's adjustment to preschool, kindergarten, and early grade school offers an opportunity to assess his or her early response to managing initial exposure to the structure and learning environment of a classroom. Inquiries about separating from parents or caregivers, managing basic classroom rules, and dealing with early social situations yield useful information, as do the patient's early learning experiences. In subsequent years, there might be early signs of learning differences or problems managing early schoolwork or more complex assignments that require a degree of organization, such as essays in middle school. There may also be unique learning environments, such as Montessori classes, attending boarding school with structured study times, or homeschooling, that are important to consider in terms of masking ADHD or precluding or reducing the usual impairments from it.

It is important to assess the transition to middle school, particularly for adults whose ADHD was not identified in childhood. There are progressively greater expectations for self-management of behavior and organization of schoolwork during the teenage years. There are similar expectations at home with regard to keeping up with homework and doing chores, as well as organizing one's behavior and activities. Even if the patient reports having earned respectable grades, it is valuable to inquire about *how* he or she handled academics and assignments during this phase of schooling, and to review corroborative information for clues about functioning. Adequate grades can be maintained with inadequate study habits that may foretell later academic problems.

Many adults with ADHD describe "doing enough to get by" during middle school and high school, settling for relatively low, though solid, grades in order to avoid or at least minimize the amount of time spent on tasks with which they struggled, such as assigned readings or essays. Some patients report that they did not complete assigned readings, instead relying on information presented in class, obtained online, or friends' notes; on the other hand, some adults recall that they relied on readings or other means to learn topics because they could not focus in class. It is common to hear descriptions of "getting by" without completing homework or doing it in a rushed fashion. Many patients relied on their parents to supervise their homework completion (or to do it for them, in some cases) or took advantage of other circumstances (e.g., supervised study hall at boarding school, long bus ride) that did not translate into adaptive study skills.

The transition to high school involves many of the same academic and homework difficulties mentioned earlier, along with the increased role of a social life. Some adults recall continued struggles in school, while others report semesters or years in which their performance improved, either in response to the novelty of starting high school or in recognition of the need to make better grades for college applications. Some high school students start working part-time jobs, enter

vocational–technical programs, or have some sort of internship that allow them to identify strengths but also may provide evidence that their organization and self-regulation difficulties follow them to these settings.

The interviewer should inquire about any major disruptions in primary or secondary education, such as having to repeat a grade, failed courses, required summer school, or steps taken to address learning difficulties (e.g., tutoring, special education). Examples of impulsive or oppositional behaviors, such as frequent detentions or suspensions, should be assessed. The clinician should confirm that the patient, in fact, completed high school on time.

COLLEGE FUNCTIONING

Asking about the decision of whether to attend college, and the college application and decision-making process, may yield useful information. For example, some patients may apply to a school simply because a personal statement essay was not required, or because they missed the deadlines for other schools.

The transition to college or other postsecondary program often places significant demands on EFs and results in newfound coping difficulties and impairments for many students with ADHD (see Chapter 12). The evaluator should explicitly ask about the adjustment to college the first year, including the demands of living away from home (or living at home while attending a community college), class attendance, keeping up with assignments, and balancing academics and social life. The issue of alcohol and substance use, as well as any conduct or legal issues on campus (or off), should be explicitly explored. It is not uncommon to hear accounts of dropped or failed courses, poor class attendance, or academic probation during the first 1 or 2 years of college for students with ADHD, particularly those who were previously undiagnosed.

As with previous levels of education, the manner in which the individuals managed academic demands should be explored to determine whether there were looming problems, even if grades were solid. Many students encounter larger classes and have less contact with instructors than they were accustomed in high school. Likewise, less monitoring by others of students' follow through on work in college often results in misjudging the time and effort needed to complete assignments. Some individuals report that they were able to get extensions from professors and dodged the consequences of their disorganization. The ability to pay attention in lectures, track and organize work and assignments, and manage and comprehend assigned readings are other areas of weakness that may exist despite passing grades.

Although many adults with ADHD are able to earn passing grades during college, it is not uncommon for students with ADHD to have taken a leave of absence from college, either forced or by choice. Similarly, adults with ADHD may have a college record sprinkled with several dropped or failed courses and switching majors or colleges, and often require more than the standard 4 years to complete degree requirements or end up discontinuing their education.

It is possible for students in medical, graduate, or other professional school programs to exhibit ADHD and EF problems. There are cases of highly intelligent persons whose degree of executive dysfunction creates difficulties for them (Antshel et al., 2010; Brown, 2013). On the other hand, some graduate students misinterpret difficulties keeping up with the strenuous academic and performance demands as signs of ADHD or, in some cases, might contrive symptoms in order to obtain stimulant medications for a competitive advantage. Areas of difficulty reported among graduate and professional students with ADHD are usually associated with comprehensive and unstructured independent projects, such as a dissertation, clerkships or internships, clinical rotations and residencies, and other situations that reflect the day-to-day organizational demands of a profession. Finally, emergent difficulties attributed to ADHD when facing high-stakes examinations, such as qualifying examinations or professional licensing, comprise another area for which individuals seek an evaluation. However, clinicians should be mindful of the possibility of malingering or at least that the presenting difficulties are not diagnostic of ADHD.

WORKPLACE FUNCTIONING

The workplace is another domain of life that is made more difficult by ADHD (see Chapter 12). Gaining a sense of the progression of jobs and reasons for job changes helps the evaluator identify trends in a person's employment history. Adults with ADHD have performance problems on the job and a history of more frequent job changes, which includes being fired (Barkley et al., 2008). Individuals often express a sense of repeatedly "starting over" and not establishing a direction in their job history, of often having to "find something to get by." In fact, many individuals end up being self-

employed (Faraone & Biederman, 2005), though many adults with ADHD end up in this situation out of necessity rather than by choice. The self-employment option may be a good one for some individuals, inasmuch as it allows for more flexibility in setting one's schedule and the types of projects undertaken. However, the self-employed worker assumes all job responsibilities, including administrative tasks, such as scheduling and billing, which require good organizational and time management skills.

Some individuals have an extended tenure in one position that offers a particularly good fit and/or many compensatory buffers, such as working in a family business or for a particularly supportive boss in a relatively low-demand, manageable position. However, workers with ADHD often report difficulties coping with an increase in responsibilities that might result from downsizing, a promotion, or an upsurge in business. Workers with ADHD may also struggle with the prospect of changing and starting over in a new job in order to seek higher pay or a better benefits package.

Just as students with ADHD who earn solid grades manifest difficulties with ADHD and EF, so too do workers with ADHD, even in stable work situations. Common issues to explore are timely arrival to work or meetings, ability to organize and follow through on deadline-bound projects and tasks, and overall work productivity. Lateness and poor time management are common complaints among workers with ADHD. Similarly, independently organizing, managing, and tracking tasks (often multiple tasks) with future deadlines are common problem areas. Workers with ADHD often report having to work harder and longer than peers in order to maintain expected productivity, often to compensate for periods of inefficiency during the workday. The cumulative effect of these difficulties is lower productivity relative to coworkers, missed deadlines, and more mistakes, including potential safety issues in some jobs (Kessler et al., 2005).

Another job-related area to consider that may have implications for other life domains is interactions and relationships with others. Despite the aforementioned job performance difficulties, which are often reflected in work evaluations of employees with ADHD, these sorts of inefficiencies, for the most part, are tolerated by employers. However, poor interpersonal behaviors at work, including anger, poor impulse control, and argumentativeness, are factors that often result in termination (Barkley et al., 2008; also see Chapters 3 and 12).

SOCIAL HISTORY AND INTERPERSONAL FUNCTIONING

Information about an individual's social history comes up throughout the evaluation. For example, the relationship with the family of origin may be a source of support for the individual with ADHD, though there may be a history of past or ongoing conflict related to functioning and coping issues. For some adults with ADHD there may be ongoing disagreements with family members regarding financial support and dealing with the effects of various educational or occupational interruptions, and so forth.

Some individuals with ADHD describe childhood experiences of rejection and bullying by peers. These problems often improve or remit by late adolescence and young adulthood, but their effects can shape one's self-evaluation and relationships. Many adults with ADHD also describe a history of low-level social anxiety as a result of repeatedly missing social cues due to inattention, or gaffes due to impulsivity. Some individuals with ADHD and coexisting oppositional behaviors may report a history of conflicts with others, including aggression, in some cases. These patients might be described as hot-tempered and argumentative as adults, characteristics that create difficulties in marriage and parenting, as well as in the workplace.

Features of ADHD likely have effects on one's dating and romantic relationship history, though not necessarily in a uniform fashion. There is evidence that adults with ADHD have a pattern of more frequent, short-lasting relationships and becoming sexually active at a younger age (Barkley et al., 2008). Considering the association between ADHD and unsafe sexual behavior (see Chapter 12), it is important to inquire explicitly about sexual activity, history of unplanned pregnancy, sexually transmitted disease, infidelity in relationships, and so forth, which also are relevant for the review of one's medical history.

MEDICAL AND PSYCHIATRIC HISTORY

The review of the patient's medical and psychiatric history helps to complete the developmental history and serves as a transition to the structured diagnostic interview portion of the evaluation. Many of these facts likely came up during the interview, but it is useful to confirm this information, and the repetition provides an opportunity to trigger reminders of about key points.

The review of the patient's medical history may start with a general inquiry (e.g., "How is your physical

health? Do you have any medical problems?"). A list of currently prescribed medications, including psychiatric medications, supplements, and any other medicines, should be documented. Any history of previous medications and response to them is also helpful and lends itself to a discussion of past medical treatment and health issues. Adults with ADHD are at higher risk for obesity, eating pathology, sleeping problems, accidental injuries, and poorer general medical (and likely dental) health (see Chapter 11), so these may be areas to probe in more detail. Some of these factors may have to be ruled out as causes of ADHD symptoms, such as significant head trauma or a chronic primary sleep disorder.

Psychiatric and psychological treatment history is useful to review. Past assessments and diagnoses of ADHD, as well as other diagnoses, or learning difficulties and related issues can be explored. During the review of treatment history, current and past suicidal and homicidal ideation should be assessed, per standard clinical practice. The evaluator should ask explicitly about past suicide attempts, self-harming behaviors, and/or aggression toward others. Although it will be explored during the structured clinical interview, it is useful to ask about past traumatic experiences because individuals with posttraumatic stress disorder (PTSD) often mistake their dissociative and other symptoms as representing ADHD.

Substance use history, another important topic to cover, includes history of abuse, dependence, and treatment (see Chapters 11 and 13). Even in cases of recreational use of alcohol or other substances, it is useful to gain a sense of the patient's pattern of use and whether it seems to serve as self-medication (e.g., nicotine). Substance use also includes nonprescribed prescription medications, including stimulants used to treat ADHD. It is useful to ask about caffeine use (including "energy" drinks) and nicotine use.

Extending the discussion of substance use, the patient is asked whether he or she has ever been pulled over for or charged with drunk driving or driving under the influence of substances. This can be a pathway to asking about his or her driving record, including driver-caused accidents, moving violations, and any of these that occurred but may have been excused and are "not on the books" anywhere. Driving is a particularly high-risk area for adults with ADHD (see Chapters 11 and 29), and frequent excessive speeding in a motor vehicle has been found to be a very good discriminatory symptom of ADHD in adults (Barkley et al., 2008). Simply asking "How is your driving?" often yields useful information; for example, some young adults choose not to drive due to the severity of their inattentiveness. Bear in mind, however, that adults with ADHD may underreport the severity of their deficits in driving (Knouse, Bagwell, Barkley, & Murphy, 2005), so obtaining corroborative evidence through the reports of others or the official driving record may be helpful in gaining a more complete picture of this domain. Finally, any relevant legal history or arrest record is reviewed. Given their problems with impulsivity, as well as significant comorbidity with antisocial personality disorder (see Chapter 13), it is not surprising to find that adults with ADHD commit more antisocial activities and experience more arrests (Chapter 11).

In addition to providing the necessary background information, the developmental history provides a great deal of useful clinical information and hypotheses. This information is used to guide the structured diagnostic interview, which is the focus of the next section.

Structured Diagnostic Interview

For clinicians in search of structured diagnostic interview formats for adults with ADHD, there are a number of reputable, clinically useful protocols, some of which are also used in research (see Adler, Spencer, & Biederman, 2003; Barkley & Murphy, 2006; Brown, 1996; Epstein, Johnson, & Conners, 2001; Kaufman et al., 1997). These interview protocols include the necessary review of childhood and current symptoms of ADHD, as well as other psychiatric conditions. Because ADHD symptoms have not been changed in DSM-5, these interview protocols continue to be relevant inasmuch as they can be easily adapted to the changes in DSM-5, such as symptom thresholds and age-of-onset criterion. With regard to the assessment of comorbid disorders based on DSM criteria, it is anticipated that there will be revisions of relevant sections on some of these protocols and other structured interviews, such as the Structured Clinical Interview for DSM-IV (SCID; First, Spitzer, Gibbon, & Williams, 1997), in order to be consistent with DSM-5.

Regardless of the diagnostic interview format, there are some common diagnostic categories that overlap and may be confused with symptoms of ADHD. To further complicate matters, any one of the conditions can coexist with ADHD given that over 80% of clinic-referred adults diagnosed with ADHD have at least one other comorbid disorder (see Chapter 13). In the fol-

lowing sections I discuss strategies for making differential diagnoses relevant to an adult ADHD evaluation.

DEPRESSION

Major depressive disorder is characterized by excessive feelings of sadness, tearfulness, lack of enjoyment or pleasure (e.g., anhedonia), vegetative symptoms (e.g., changes in sleep, appetite, and energy), and increased guilt and self-criticism. These symptoms are experienced most of every day for at least 2 weeks. Concentration and memory difficulties are common symptoms of depression. What is more, depressive symptoms are commonly seen in adults with ADHD. Symptoms of depression magnify the features of ADHD and make it even more difficult for individuals to sustain concentration on tasks and initiate behaviors.

A key point of difference between depression and ADHD is the course of the respective disorders. Someone who is currently depressed describes his or her attention and memory difficulties as being associated with his or her mood state. There will not be evidence of these symptoms outside of a depressive episode. ADHD, on the other hand, is a neurodevelopmental syndrome, so there will be evidence of symptoms and EF problems that persist across time and situations regardless of mood status. That being said, someone with ADHD who experiences depression will describe a worsening of symptoms during these mood episodes.

ANXIETY

Anxiety is the most common comorbid diagnosis with ADHD (Kessler et al., 2006). Many tasks of daily life are more difficult for individuals with ADHD than they are for those without it. If anxiety is thought of as an emotional reaction to a "threat," then this comorbidity pattern makes sense because many aspects of daily life are challenging to manage for adults with ADHD.

Generalized anxiety disorder is associated with feelings of apprehension, tension, and being "on edge." It is also associated with irritability, worry, restlessness, and difficulty concentrating (e.g., one's mind going blank). Anxiety may also have physical manifestations, such as headaches, gastrointestinal discomfort, and difficulties falling asleep. As with depression, there is some overlap of anxiety and ADHD symptomatology, complicated by the fact that many adults with ADHD experience anxiety.

Individuals with anxiety disorders often experience their symptoms when faced with specific triggers, such as a social situation or phobic stimuli. There also may be discrete episodes of anxiety, for example, when experiencing a panic attack. When not faced with a trigger, however, a person's anxiety symptoms diminish, as do the symptoms that may be confused with ADHD. The anxious person describes functioning normally when not anxious. Even in the case of generalized anxiety, there are times of improved functioning associated with lower anxiety levels. As with depression, an adult with ADHD who is also anxious will experience a worsening of symptoms; however, there will continue to be evidence of ADHD and EF problems even when the person is not anxious. In fact, some patients with ADHD describe not being anxious enough in some situations.

Within the anxiety disorders, it is important to screen for PTSD. The regions of the brain associated with the EFs are affected by childhood trauma, which may produce lasting symptoms of inattention and impulsivity (Karl et al., 2006). The key factor in distinguishing between ADHD and PTSD symptoms is the determination of when the trauma occurred. Those with ADHD are more prone to accidental injuries and to engaging in violence against others or being the subject of such violence (see Chapters 12 and 13), increasing the possibility that they may develop PTSD. When a traumatic event occurred after there is evidence of the onset of ADHD (e.g., late adolescence, young adulthood, or later), the case can be made for comorbidity. When a trauma is traced back to childhood, and before there is sufficient evidence of ADHD, the PTSD diagnosis should be the sole diagnosis, barring compelling evidence to the contrary. Assessors should be aware that adult patients may present for an ADHD evaluation and report symptoms of ADHD but may not disclose a trauma history unless explicitly asked.

BIPOLAR DISORDER

The hallmark trait of bipolar disorder is extreme mood fluctuation or the "swing" between the "highs" of mania and the "lows" of depression, hence the old term manic–depression. Classic bipolar I disorder is defined by at least one full-blown manic episode, which can include psychotic symptoms and suicidality, often requiring hospitalization to stabilize. It is usually the less pronounced manifestations of bipolar spectrum disorders, including bipolar II disorder, that represent

a challenge to evaluators. Hypomania is associated with varying degrees of decreased need for sleep, racing thoughts, impulsivity, and increased activity level, which are similar to features of adult ADHD.

Interepisode functioning provides useful information to tease apart ADHD and bipolar disorder. Symptoms and difficulties associated with ADHD persist across time and mood states, whereas individuals with bipolar disorder report improvements during periods of mood stability. What is more, both ADHD and bipolar disorder consist of emotional management difficulties, including anger and irritability. The manifestation of irritability in bipolar spectrum disorders is more consistent with a mood disorder, inasmuch as these emotional reactions are stronger and longer lasting, whereas the emotional dysregulation in ADHD results in brief impulsive outbursts of anger or other emotions, followed quickly by recognition of the overreaction (see Chapter 3).

SUBSTANCE USE

Chronic substance abuse is known to have negative effects on cognitive and emotional functioning that can mimic the symptoms of ADHD; conversely, untreated ADHD is a risk factor for substance use problems in one's lifetime (Wilens & Fusillo, 2007). In cases with a development sequence in which clear evidence of the onset of ADHD predates the development of a substance abuse problem, there is sufficient evidence of comorbidity. The clinical picture is complicated when substance use problems start before there is clear evidence of ADHD. Most often, for adults seeking an evaluation for ADHD, evidence of ADHD predates the onset of substance abuse. In fact, there may be evidence that substance use patterns are consistent with the "self-medication" hypothesis (e.g., Khantzian, 1985), in which substance use is viewed as an effort to manage symptoms. Alcohol and marijuana are the most commonly reported substances of abuse among individuals with ADHD (e.g., Barkley et al., 2008; Wilens, 2004).

Even when comorbidity of ADHD and substance abuse has been established, a task in the assessment is to consider the relative priority of treatment. That is, in cases of ongoing substance abuse or dependence, a detoxification or rehabilitation program may be required before treatment for ADHD can be started. Even in cases in which ongoing substance use does not require inpatient treatment, there may need to be a period of abstinence or at least reduced use for at least a month before proceeding with pharmacologic interventions.

Concluding the Assessment Meeting

After gathering all the necessary interview information, the clinician concludes the assessment meeting by scheduling the feedback session. Arrangements for gathering any incomplete paperwork or signing consent forms for contacting significant others or treatment professionals also can be made at this time. Patients are often eager to get the clinician's impressions at the end of this meeting. In some cases, a profile consistent with ADHD emerges quickly and clearly; in other cases, the clinical picture remains unclear, even after extended time with a patient. It is usually prudent to defer final conclusions until the feedback session in order for the clinician to have adequate time to score and review the clinical inventories completed by the patient, as well as any other information gathered. The clinical inventories commonly used in an adult ADHD evaluation are reviewed in the next section.

Adult ADHD Inventories

A "gold standard" evaluation for adult ADHD requires a review of DSM-5 symptom criteria. The requirement of emergence of "several symptoms" by age 12 calls for a systematic, retrospective assessment of childhood symptoms. Thus, in addition to information obtained through interview, symptom checklists and other inventories for ADHD provide essential documentation. What is more, the combination of checklists and interviews provides a format in which to make follow-up inquiries about specific symptoms patients may or may not endorse or to probe discordance between interview and checklist responses.

Obtaining observations and ratings of patient functioning from others who know the patient well has always been advised, and DSM-5 guidelines explicitly emphasize the need to do so, whenever possible, as part of the standard diagnostic process. There may be small discrepancies between different reporters (Barkley et al., 2011), and indeed some underreporting of symptoms or impairments by adults with ADHD. But large disparities in which the patient's reports of symptoms greatly exceed that reported by others may signal a need to explore the possibility of malingering. While it is important to maintain a healthy skepticism with regard to such inconsistencies, particularly in cases of

potential malingering, these incongruities occur often when attempting to capture a wider range of symptom reports than can be achieved by a single person. That is, a parent might have difficulties identifying symptoms of inattention in a college-age son (e.g., "Was he reading or daydreaming while staring at the book?") but cite several examples of impulsivity; the student might describe pervasive inattention but is less cognizant of impulsivity or restlessness.

A checklist of DSM-5 symptoms or other relevant symptom criteria, such as the nine-item list of empirically derived symptoms of adult ADHD compiled by Barkley and colleagues (2008; see Chapter 2, Table 2.2), provides clinicians with an easy and accessible means for assessing symptoms. Other available scales that offer additional useful features in a comprehensive assessment are summarized in Table 19.3.

The Barkley Adult ADHD Rating Scales–IV (BAARS-IV; Barkley, 2011a) provide an easy-to-use, norm-based symptom checklist. It includes both self- and observer-report forms for both childhood and adult symptoms of ADHD. The BAARS-IV provides norms for total rating scores, as well as symptom counts for each symptom cluster for Inattention, Impulsivity, and Hyperactivity. A total ADHD score and symptom count also are provided. (For ratings of childhood behavior, a single score for the combination of Hyperactivity–Impulsivity is provided). Respondents rate symptoms on a 4-point scale of severity of symptoms from those that are/were *never or rarely* a problem (1, or minimal) to those that are/were *very often* a problem (4, or severe). According to the scoring instructions, symptoms endorsed as occurring at least *often* (3, or moderately) are considered diagnostic and are counted for each of the subtypes. Thus, the BAARS-IV is useful because diagnostic questions regarding ADHD can be answered in terms of symptom endorsement as defined by DSM-5, as well as using norm-based ratings of symptom severity.

The BAARS-IV includes a measure of SCT symptoms in adulthood. As I mentioned earlier (also see Chapter 17), SCT is characterized by difficulties in orienting and engaging attention, effort, and alertness. Individuals with SCT are described by themselves and others as having problems associated with being "daydreamy," sleepy, lethargic, easily bored, sluggish, and hypoactive; the first two features are the most distinctive factors of SCT (Penny, Wachbusch, Klein, Corkum, & Eskes, 2009). Executive dysfunction is not seen in SCT to anywhere near the same degree as it is in classic ADHD (Barkley, 2012a; see Chapter 17), though levels of impairment are nearly equivalent in children (Bauermeister, Barkley, Bauermeister, Martinez, & McBurnett, 2011) and in certain domains (education, work) may be greater than levels in adults with ADHD (Barkley, 2012a).

TABLE 19.3. Useful Clinical Inventories for the Assessment of Adult ADHD

ADHD symptom checklists
ADHD Rating Scale–IV
Adult Self-Report Scale (first six items are a screener for adult ADHD)
Barkley Adult ADHD Rating Scales–IV
Wender Utah Rating Scale (for childhood symptoms)
Adult ADHD inventories
Brown Attention-Deficit Disorder Scales—Adult Version
Conners' Adult ADHD Rating Scale
EF inventories
Barkley Deficits in Executive Functioning Scale
Behavior Rating Inventory of Executive Function—Adult Version
Functional impairment inventories
Adult ADHD Quality-of-Life Scale
Barkley Functional Impairment Scale
Weiss Functional Impairment Rating Scale

Other symptom rating scales include the ADHD Rating Scale–IV (ADHD-RS-IV; DuPaul, Power, Anastopoulos, & Reid, 1998), which is consistent with DSM-IV criteria. The ADHD-RS-IV assesses each DSM-IV symptom and provides prompts to help discern their presence, and severity is rated on a 0- to 3-point scale, from *never/rarely* to *very often*. The prompts for each item can be adapted for the assessment of both children and adults, and the scale can be used to obtain both self- and observer ratings. Normative information for adults is lacking, however, because the scale was developed for use with children.

The Wender Utah Rating Scale (WURS; Ward, Wender, & Reimherr, 1993) is often used for the recall of childhood symptoms. The WURS is a 61-item self-report measure that includes a subset of 25 items specifically targeting the diagnosis of ADHD, with the remaining items assessing associated features. Items are endorsed on a 0- 4-point scale ranging from *not at all/slightly* to *very much*.

The need to document DSM-5 symptoms is a necessary but not sufficient component of a psychological assessment for adult ADHD. That is, the DSM-5 symptom list does not adequately capture the range of difficulties faced by patients with ADHD. There are several clinician-friendly, standardized adult ADHD rating scales that can be included in a comprehensive assessment. Two commonly used scales are the Conners' Adult ADHD Rating Scales (CAARS; Conners, Erhart, & Sparrow, 1999) and the Brown Attention-Deficit Disorder Scales for Adults (BADDS; Brown, 1996). While complementing DSM symptoms, these scales assess a variety of adult symptoms of ADHD not codified in the official criteria, many of which overlap with items from scales assessing EF.

The CAARS is a self-report inventory that measures a wide range of symptoms of ADHD in adult patients. Respondents rate each item relative to the occurrence of symptoms on a 4-point scale ranging from *not at all, never* to *very much, very frequently*. The CAARS yields a total score and subscale scores measuring a variety of deficits commonly associated with ADHD. Among the subscale scores are three devoted to DSM criteria (DSM-IV Inattentive Symptoms, DSM-IV Hyperactive–Impulsive Symptoms, and DSM-IV ADHD Symptoms Total) and an additional ADHD Index score that is helpful in corroborating clinical data gathered during interviews. The long version of the CAARS includes additional useful subscales. Each respondent's responses are tabulated and plotted on a profile form that is based on compiled norms that are specific to both gender and age of the respondent. The CAARS offers long (66-item), brief (30-item), and screening (28-item) versions, and observer rating forms.

The BADDS Adult version is a 40-item inventory that measures a variety of symptoms of ADHD in adult patients, including many that target EF. The BADDS examines the ability not only to sustain attention but also to get started on work tasks, initiate and sustain attention, maintain effort necessary to complete tasks, regulate moods, and recall information encountered in daily life. It yields a total score and five subscale scores corresponding to the previously mentioned components of the EFs (i.e., Activation, Attention, Effort, Affect, and Memory). Each item is rated for occurrence of symptoms on a 4-point scale ranging from *never* to *almost daily*. Similar to the CAARS, respondents' responses are tabulated and plotted on a profile form that is based on adult norms. The form includes a space for observer ratings of symptoms, but norms are not available for these ratings.

The use of the aforementioned forms and checklists are sufficient steps to document the symptoms of ADHD, though it is important to review the items and scores and to compare them with the information gathered during the interview and other sources. There may be subthreshold ratings in cases of obvious symptoms and impairments associated with ADHD; on the other hand, for ratings that exceed diagnostic thresholds there may be important evidence that suggests something other than ADHD is causing them. Hence, a comprehensive evaluation for ADHD should include an assessment of EF, which provides clinically useful information, as well as another means to assess for self-regulation.

EF Inventories

Kessler and colleagues (2010) and Barkley (2011b; Barkley et al., 2008) reported that symptoms of executive dysfunction are strongly associated with the diagnosis of ADHD and predict associated functional impairments. Furthermore, self-reports of executive dysfunction are a reliable diagnostic indicator and a means to assess level of impairment. Thus, a norm-based scale of EF is a useful component of an ADHD evaluation.

The BADDS represented an early attempt to assess for executive dysfunction in adults with ADHD; its five subscales are consistent with Brown's (1996, 2013) EF model, though it has also been used as a symptom measure. More recent inventories provide a more comprehensive evaluation of EF activities in daily life and have a stronger normative base. The Behavior Rating Inventory of Executive Function (BRIEF®; Roth, Isquith, & Gioia, 2005) is an 80-item self-report scale. Items are rated for frequency of a particular behavior on a 3-point scale (*never, sometimes, often*) and there are nine subscale scores measuring behavioral regulation (i.e., Inhibit, Shift, Emotional Control) and metacognitive (i.e., Initiate, Working Memory, Plan/Organize, Monitor, Organize Materials) facets of EF. There is also a Negativity Scale and an Inconsistency Scale that can be calculated to make sure an inventory is valid. There is also an Observer version of the BRIEF®.

A more recent EF inventory is the Barkley Deficits in Executive Functioning Scale (BDEFS; Barkley, 2011b). The BDEFS provides a norm-based measure of execu-

tive dysfunction with both self- and observer-report forms. The five EF domains that constitute the five subscales of the BDEFS are Self-Regulation to Time, Self-Organization/Problem Solving, Self-Motivation, Self-Restraint, and Self-Regulation of Emotions. Respondents rate items for severity of symptoms on a 4-point scale ranging from those that are/were *never or rarely* a problem (1, or minimal) to those that are/were *very often* a problem (4, or severe). Total scores are calculated for each domain and compared with age- and gender-based norms. A Total EF score is calculated, as well as an ADHD-EF Index score that provides an indication of risk for ADHD.

Functional Impairment Inventories

It is most often the case that a clear link among symptoms of ADHD, executive dysfunction, and presenting problems can be drawn in the course of an assessment. A lifetime history of adult ADHD is associated with a wide variety of problems that interfere with functioning in at least one, if not several, domains of adult life, as is evident from previous chapters. In fact, establishing impairment is a required element of the diagnosis of ADHD (and other DSM-5 diagnoses).

While a direct and causal link between symptoms and impairments may often seem easy to establish, the two constructs are not identical and are only partially correlated (Barkley, 2011c). This means that the presence of one does not ensure that it arises from the presence of the other. Some assessment situations benefit from the use of norm-based measures of functional impairment or at least a means to quantify specific domains of impairment. The Adult ADHD Quality-of-Life scale (AAQOL; Brod, Johnston, Able, & Swindle, 2006) offers a brief, self-report inventory of relative satisfaction with different domains of life and adult role functioning. Respondents rate items on a 5-point scale that ranges from *not at all/never* to *extremely/very often*. Subscale scores are available for Life Productivity, Psychological Health, Life Outlook, Relationships, and a Total score.

The Weiss Functional Impairment Rating Scale (WFIRS; Weiss, 2010) is another assessment that targets different domains of adult life. There are self- and observer-report forms available. Items within each domain are rated by respondents on a 4-point scale that ranges from *never or not at all* to *very often or very much*, yielding subscale scores for Family, Work, School, Life Skills, Self-Concept, Social, Risk, and Total. Norms are not provided for this scale, however.

Finally, the Barkley Functional Impairment Scale (BFIS; Barkley, 2011c) provides a norm-based measure of functioning that is not limited to ADHD. Items are rated by the respondent (self- or other-report) on a 10-point scale of severity of functional difficulties in each of 15 domains of major life activities. A score can be calculated for each domain along with a Total score across all domains and a score reflecting the number of domains in which the individual is impaired. These scores can then be compared with age- and gender-based norms.

Comorbidity Inventories

Considering the high rates of psychiatric comorbidity in adults with ADHD (Chapter 13), as well as the need to consider other potential explanations for presenting symptoms, it is useful to obtain measures of other psychiatric symptoms. The most commonly used measures are those that assess current level of depression and anxiety. With regard to depression, the Beck Depression Inventory–II (BDI-II; Beck, Steer, & Brown, 1996) offers an easy-to-use, well-researched option. The BDI-II is a 21-item self-report scale that monitors current mood symptoms. Patients rate their current level of distress on various symptoms of depression, such as Self-Criticism, Energy Level, and Suicidal Ideation. Each item is rated from 0 (*not a problem*) to 3 (*severe problem*), and the total score is the sum of the ratings.

The Hamilton Depression Scale (HAM-D; Hamilton, 1967) is a 17-item, clinician-administered scale that assesses current level of depression. The symptom categories evaluated include Feelings of Guilt, Insomnia, Somatic Symptoms, and Helplessness.

The Hamilton Anxiety Scale (HAM-A; Hamilton, 1959) is a 14-item, clinician-administered scale that assesses current level of anxiety. The symptom categories evaluated include Tension, Autonomic Symptoms, and Cognitive Difficulties.

The Beck Anxiety Inventory (BAI; Beck & Steer, 1990) is a 21-item, self-report instrument that monitors both physiological and cognitive symptoms of anxiety. Patients rate their current level of distress on various symptoms of anxiety, such as Inability To Relax, Nervousness, and Fear of Losing Control. Similar to the BDI-II, each item is rated from 0 (indicating that it is not a problem) to 3 (indicating a severe problem) and

the Total score is the sum of the ratings. Because the BAI emphasizes many somatic symptoms of anxiety, such as those characteristic of panic attacks, it may underestimate the degree of apprehension and angst about tasks and other demands that is anecdotally reported by adults with ADHD.

The Penn State Worry Questionnaire (PSWQ; Meyer, Miller, Metzger, & Borkovec, 1990) is a reasonable alternative that focuses more on features of generalized anxiety, including the elements of apprehension and uneasiness that are often expressed by adults with ADHD. The PSWQ is a 16-item, self-report inventory that is designed to capture the excessiveness, pervasiveness, and uncontrollability of pathological worry. Items are scored on a 5-point scale (with some items reverse-scored); the sum of these scores is the total score that identifies low, moderate, or high level of worry.

The Symptom Checklist–90—Revised (SCL-90-R; Derogatis, 1977) is a 90-item self-report scale designed to measure psychological symptoms and distress on a 5-point scale of severity, from *not at all* to *extremely,* covering nine symptom domains. In addition to being useful in assessing possible comorbid symptoms, 16 items have been identified and used as a measure of ADHD (Hesslinger et al., 2002). As reported in Chapter 13, virtually all studies of adults with ADHD find that their scores are significantly elevated across most or all of the subscales on this form.

These inventories offer a sample of well-regarded and researched mood and anxiety measures. There are likely others that provide a means to assess clinical symptoms in order to determine the presence of comorbidity or better explain the presenting issues that led to the current evaluation. It is advised to use a scale or scales that provide adequate measures of depression and anxiety, at the very least. These inventories, combined with the other information gathered during the assessment process, are used to develop diagnostic impressions and resultant recommendations to be shared with the patient at the evaluation feedback session.

Feedback Session

The stage is set for the feedback session during the evaluation itself. That is, patients are reminded that the assessment process is, in fact, an assessment of the presenting situation and requires the synthesis of a variety of clinical data. Consequently, even though individuals request an "evaluation for ADHD," it is a useful reminder that the clinician is conducting an evaluation; thus, a potential outcome of an ADHD assessment is that the presence of ADHD may not be supported by the evidence.

We reserve a 1-hour appointment for the feedback session, which is viewed as the final step in the overall assessment. We make every attempt to schedule it during the week following the evaluation appointment in order to provide time to score, review, and contemplate all the inventories and test scores, to review information gathered during the interview and archival records provided by the patient or obtained from officials, and to formulate diagnostic impressions and treatment recommendations.

The feedback session starts with a general check-in with the patient and an invitation to address any questions about or impressions of the evaluation. The process of going through the evaluation often prompts discussion between patients and their loved ones that may yield useful clinical information that is brought up at the feedback session. At times this information has been quite valuable in clearing up what had been unclear from a diagnostic standpoint. In fact, it is good advice to wait until after the feedback session to complete the evaluation report because sometimes information that arises may change an evaluator's conclusion.

Once these introductory matters have been addressed, the evaluator provides an executive summary of conclusions based on the evaluation, including whether the clinical data support a diagnosis of ADHD, as well as any other key issues. The session then proceeds with a thorough review of the clinical data on which the clinician's opinion is based, in order to be transparent about the diagnostic process.

A useful format is to start with a review of past and current ADHD symptom checklists, providing education about the age-of-onset criterion and diagnostic thresholds. The limitations of the symptom criteria are used to transition to a review of the results from various adult ADHD inventories. A visual aid that illustrates a normal distribution with the various standard scores is a useful tool with which to explain norm-based results gained from these inventories and to help patients understand their scores. Additional scales relevant to the diagnosis of ADHD, namely, any EF or functional impairment inventories that were completed, are reviewed next as a way to wrap up the review of ADHD symptoms. The patient's EF profile is reviewed, with areas of difficulty pointed out using norm-based percentile ranks, as well as areas in which there is no evidence of deficits. As with the symptom checklists, self- and

other-reports are compared, and any substantial discrepancies across inventories or reporters are discussed.

At this point, the conclusions regarding the ADHD diagnosis are repeated in the context of the recent review of data and integrated with developmental information gathered during the clinical interview. The patient is asked whether he or she has any questions or needs any clarification about the scales or the results. When the clinical data do not support a diagnosis of ADHD, the relevant factors can be highlighted and discussed, an important issue that I discuss separately in the next section of this chapter.

The next step in the feedback session is to review other clinical inventories, such as mood, anxiety, or other psychiatric symptoms rating scales. The reported levels of depression, anxiety, or other symptoms are reviewed and put in the context of normative scores or degree of severity. These scores are compared with findings from the structured diagnostic interview in a discussion of any coexisting symptoms and their relationship with ADHD, or perhaps how they better explain presenting problems. Other clinically relevant findings that are central to the diagnosis and treatment planning are discussed at this point (substance use, past trauma, sleep, eating pathology, driving, etc.).

Findings from any administered psychological testing results or personality inventories are reviewed next, using a format similar that of the various inventories. The purpose of each test and its relevance within the assessment are reviewed. The patient's performance on each test is reviewed, as well as any limitations to the test, in order to guard against overgeneralizing the implications of a score.

The overall summary of the findings offered at the beginning of the feedback session is repeated in order to wrap up the review. At this point, questions are invited. The main question that most patients have at this point—"What do I do now?"—leads to a discussion of treatment recommendations based on the assessment data. While every case of adult ADHD is different, there are some typical domains of treatment recommendations to consider. Medication treatment for ADHD (see Chapter 35) and/or other psychiatric diagnoses are the first domains to address and review. Referrals for further medical tests, if needed, can be outlined at this point, such as a sleep study to rule out sleep apnea or a comprehensive physical examination that includes blood work to assess overall physical health and rule out possible, if not suspected, medical explanations for symptoms.

Psychosocial treatment for ADHD and/or coexisting psychiatric disorders is another possible recommendation. Counseling (see Chapter 31) and cognitive-behavioral therapy (see Chapter 32) adapted for adult ADHD are evidence-supported psychosocial treatments (Ramsay, 2010). Adult ADHD coaching, relationship treatment (Chapter 34), or group therapy may also be appropriate for some patients.

Some adult ADHD evaluations are bundled within a comprehensive psychoeducational assessment, the findings of which can be used to petition for formal academic accommodations (see Chapter 33). If the diagnosis of ADHD is not sufficient to obtain these accommodations, then the results from clinical assessment and/or testing used in the evaluation may be used to determine whether it makes sense to pursue testing for learning disabilities that may qualify. Functional issues not explained by ADHD may warrant additional neuropsychological or neurological assessment. Referrals for learning support in academic settings can be provided.

Finally, various psychoeducational materials and online resources can be provided to the patient. There are many good and reputable websites that focus on ADHD and provide useful coping suggestions and referral lists.

Evaluations for adult ADHD are viewed as a specialty and are sought when there is a strong suspicion of the presence of ADHD. Some issues that arise are of particular relevance to the assessment of ADHD. Thus, in the next section I review questions about the role of testing for ADHD, the skill of providing feedback when a patient's developmental profile is not consistent with ADHD, and the increasing problem of individuals feigning ADHD symptoms.

SPECIAL TOPICS

The Use of Testing in the Assessment of ADHD

There is an understandable desire to establish a diagnostic test for ADHD so as to place it on a firmer, more objective, and less error-prone footing. There are various examples in medicine of using imaging or laboratory tests to establish the presence of an injury or disease state. As it currently stands, there is no such stand-alone test that reliably and accurately diagnoses symptoms and impairments associated with ADHD, much less any other mental disorder. Any tests that might be used to gather information are administered

in the context of a comprehensive evaluation of the sort outlined in this chapter, comprising just a small part of the totality of information used to render decisions on diagnosis and management.

Recent research has indicated that self-reports of EF problems are not significantly correlated with the results of EF tests (Toplak, West, & Stanovich, 2013). Moreover, additional evidence suggests that the ratings provide a better index of EF-related impairments in daily life than do psychological tests purporting to assess EF (Barkley & Fischer, 2011; Barkley & Murphy, 2010). Considering that EFs likely evolved to help humans adapt to social and contextual milieus, it makes sense that tests of "cold" cognition that comprise tasks designed to approximate these skills that are performed in a quiet testing office would produce inconsistent results. In particular, many individuals with ADHD perform adequately on office-based tests but are found to be impaired in many domains of their lives.

Consequently, the use of testing is not required to establish the presence of impairments associated with ADHD. Psychoeducational testing is still required to establish norm-based evidence of learning disabilities in order to secure formal academic or testing accommodations. It is also essential to rule out intellectual disability as an explanation for or contributing factor to educational and occupational difficulties. However, even when they are components of an evaluation, testing data should not be the sole or even the primary source of evidence of the presence of impairments or even symptoms of ADHD.

That being said, there is clinical utility in the use of testing to assess for some aspects of ADHD and related difficulties. While the absence of evidence of difficulties on tests is not evidence of absence of deficits, the presence of difficulties on tests can be informative. The problem with testing is not so much high rates of false positives as it is false negatives, in that many adults with legitimate ADHD pass the tests. There may be testing data that suggest the presence of learning difficulties or other developmental or neuropsychological problems that affect functioning and warrant further assessment (see Chapters 10 and 11).

Brief tests of auditory working memory (Brown, 2013), spatial working memory, and fluency or computerized continuous-performance tasks (Barkley & Murphy, 2010; Barkley et al., 2008) provide measures of common skills deficits in adults with ADHD. Moreover, testing results, such as subscales of an intelligence test used to gauge overall intellect and to screen for learning differences, may provide evidence of areas of strength for individuals who may discount them in the face of coping difficulties. Observation of behaviors during testing provides as much or more useful, and often qualitative, information than do the actual scores from the test. Finally, it can be helpful to obtain an overall assessment of cognitive functioning, separate from the clinical diagnosis of ADHD. The challenge for evaluators is to construct an assessment that best answers the referral questions.

What If the Patient Does Not Have ADHD?

Clinicians who perform assessments for adult ADHD should be prepared to find themselves in a position of informing patients that the results of an evaluation do not support the diagnosis. For instance, Barkley and colleagues (2008) reported that over 40% of referrals to an adult ADHD clinic were found not to meet full DSM-IV criteria for the disorder, even though all suspected they might have it (these became the clinical control group in their subsequent research study). This process can be difficult for patients who have invested time, money, and their hope for change in obtaining the diagnosis of ADHD. However, an accurate diagnosis and competent treatment plan will help patients make improvements in their lives in the long run rather than acquiescence to their own self-diagnosis.

During the feedback session with a patient for whom the conclusion is that he or she does not have ADHD, it is useful to start the discussion with a clear statement about the diagnosis; namely, that the clinical evidence does not support a diagnosis of ADHD. This statement is followed with an open and collaborative review of the clinical data. Here one explains that many factors can contribute to inattention without rising to the level of clinical ADHD. In short, all inattention is not ADHD, and ADHD is far more than just being inattentive. The clinician can then provide straightforward explanations of diagnostic interpretation of the various data, including management of seeming incongruities (e.g., "You say I don't have ADHD but you also say that this score for inattention is elevated"), in order to be transparent about one's clinical decision making. It is useful to provide psychoeducation about the diagnostic criteria and various requirements (e.g., emergence and persistence of symptoms), as well as how other conditions can look like ADHD but are different.

The review of the clinical data and the clinician's assessment of these data, such as the emergence of "in-

attention" symptoms after a trauma, help affirm the patient's experience of distress and difficulties that led him or her to seek the evaluation. This may also be a time to explain that the symptoms may be more consistent with SCT (Chapter 17) or another disorder (depression). Thus, the clinician acknowledges a patient's distress and puts it into a different framework. Even if a diagnosis of ADHD is not supported by the clinical information, a good evaluation will yield feasible and competent treatment recommendations that address the presenting problems and instill hope for change.

In rare cases in which a patient continues to dispute the conclusions, a clinician can sidestep unproductive arguments and stand by his or her professional opinion, inviting the patient to seek a second opinion and refocus on ways to improve well-being. Most patients leave the feedback session with a better understanding of their difficulties and a direction in terms of the next steps toward increased well-being. Even when the clinical data do not support a diagnosis of ADHD, a comprehensive and thorough assessment provides a formulation and recommendation that resonates for patients.

In some cases, however, events may arise during the feedback session or other parts of the assessment process that trigger concerns about possible malingering by the patient. The issue of patients intentionally "faking ADHD" is discussed in the next section.

Malingering

The accuracy of the psychological assessment of ADHD is an important issue. If nothing else, diagnostic accuracy is essential to direct patients to the clinical and support services that help to improve their functioning and well-being. Assessments in clinical psychology and psychiatry rely on information gathered from patients regarding symptoms and functioning. There will likely be at least some inconsistencies in clinical data gathered during an evaluation for adult ADHD. For the most part, these incongruities are not substantial and are based on good faith efforts by patients and observers to provide helpful information. For example, a patient might recall relatively few symptoms of ADHD in childhood, but a parent completing an observer rating form endorses symptoms with greater frequency and severity; or a spouse's ratings of EF emphasize functioning at home, while the patient's focus on functioning at work provides wider symptom coverage.

A more worrisome problem is the intentional misrepresentation of symptoms or "faking bad" in order to obtain an ADHD diagnosis from a qualified professional (Harrison, Edwards, & Parker, 2007; Quinn, 2003). The issue is particularly relevant for high school and college students without ADHD who are motivated to obtain academic accommodations or prescriptions for stimulant medications for misuse and/or diversion. It can also be seen in instances in which the person derives direct and immediate financial or other benefit from the diagnosis (insurance settlements, lawsuits, criminal sentencing, etc.). It is relatively easy to track down the symptoms of ADHD on the Internet and parrot them back during an appointment. Self-report inventories, including some reviewed in this chapter, are particularly susceptible to faking bad (Sollman, Ranseen, & Berry, 2010; Tucha, Sontag, Walitza, & Lange, 2009). A chart review study indicated that 22% of individuals referred for ADHD evaluations misrepresented symptoms or performance on cognitive testing (Marshall et al., 2010), although there are reasons to suspect that this is an overestimate. The estimated baseline rate of malingering among a general outpatient population was 15% (Rogers, Harrell, & Liff, 1993).

In some ways, a comprehensive assessment for adult ADHD and the multiple steps involved in arranging the appointment, completing inventories, and going through the evaluation visit and feedback session protect against individuals looking for a quick diagnosis. Malingerers are more likely to seek out all-in-one appointments, such as with a primary care physician or a psychiatrist in private practice who will meet with a new patient, conduct an evaluation, and initiate treatment, all in one visit.

The possibility that a patient is feigning symptoms of ADHD must be considered, particularly in situations in which the assessment results are associated with secondary gains, such as obtaining stimulant medications or specialized support services. While there are no surefire means of establishing malingering, there are some clues to look for that raise suspicions about the possibility of faking bad. However, they must be considered in the context of the individual patient.

One warning sign is excessive focus on and agitation about obtaining medications for ADHD, especially at the start of or throughout the evaluation. That is, an individual might be upset to learn that he or she will not be given a prescription for medication at the end of the evaluation or feedback session. Most patients are amenable and understanding when informed that the evaluation process is separate from treatment. Even at

the outset of pharmacotherapy, an informal assessment of drug-seeking behavior is for the physician to suggest starting with an approved nonstimulant medication for adult ADHD in order to gauge a patient's reaction.

Second, attempts to circumvent the typical assessment procedures should give pause. There will be many credible requests for "special treatment," such as someone who asks to be "squeezed in" before a semester starts or for whom it is difficult to arrange a phone screening due to work conflicts. However, people who show up at an office expecting a "drop in" meeting, claim to have a scheduled meeting that is unsubstantiated and strains credibility, or resist completing forms, contacting others for collateral reports, or resist other essential elements of the evaluation may be trying to get around procedures in order to get a "foot in the door."

Third, although it is relatively easy for someone to fake symptoms of ADHD on a rating scale, it is much more difficult to provide specific examples of functional difficulties at different points in their developmental trajectory in a relatively fluent, coherent, and consistent fashion during an interview. The evaluating clinician assesses the onset and manifestation of symptoms and impairments across time and contexts to derive a coherent narrative that is consistent with what is known about ADHD and its impairments and comorbidities, and does not rely solely on symptom checklists and inventories. Adults with ADHD are able to recall examples of difficulties, as well as comments made by others, with little difficulty and in rich detail. People who are faking bad, on the other hand, appear to be searching for answers and produce generic situations without providing common details, such as the name of a teacher, boss, or stories of past frustrations.

The production of corroborative information, such as report cards or observer reports, is the fourth aspect of comprehensive assessment that reduces the likelihood of faking. There may be credible reasons that this information is not available, such as an only child whose parents are deceased, but other archival academic, employment, driving, criminal, medical, and other records are often available to corroborate a history of impairment. The availability of a "paper trail" for ADHD is a good sign of credibility.

Apart from the previous suggestions, there is an increased need for other ways to identify malingering in the assessment of adult ADHD. The use of symptom validity tests (SVTs) in the context of neuropsychological testing as a means to identify symptom exaggeration shows some initial promising results (Jasinski et al., 2011; Marshall et al., 2010).

Extreme exaggeration on behavior rating scales is one clue that suggests potential malingering (Marshall et al., 2010). The conventional cutoff defining clinical elevation on ADHD ratings scales is at least 1.5 standard deviations above the population mean, often expressed as a *T*-score of 65 or higher on many inventories. It makes sense that adults with ADHD will endorse these symptoms in greater number and severity than individuals without ADHD. However, respondents who endorse ADHD symptoms at greater than two standard deviations above the mean for adults with ADHD may be exaggerating them, particularly when there are other indications of potential faking, such as far lower ratings provided by others, lack of impairment consistent with such extreme symptoms in the history and archival records, and so forth. Even reviewing symptoms checklists and other clinical inventories to see if nearly all items seem to be endorsed as *severe* or *always* is a way to screen for possible symptom exaggeration. The CAARS has an Inconsistency Index score that reflects variability in rating different presentations of the same symptom, but this index has not been found to be adequately sensitive and specific to identify malingering.

Marshall and colleagues (2010) reviewed past neuropsychological testing batteries for adult ADHD that included a variety of embedded or stand-alone SVTs. Based on their review of 268 charts, more than one in five adult ADHD evaluations were flagged as symptom exaggeration by meeting at least two of the following criteria: (1) failure on an SVT measure (failing two SVTs is also an indication of feigning), (2) a test score more than two standard deviations worse than the ADHD average, (3) failure on an ADHD inventory validity scale, or (4) major discrepancy between self-report and tester rating of observed ADHD behaviors. Patients seem to be more likely to fake tests viewed as being directly associated with ADHD, such as attention tasks, tests of speed of processing, working memory, memory, and divided attention. Of course, there may be inconsistencies in the ADHD rating scales in cases in which there is compelling clinical information supporting a diagnosis of ADHD.

Prospective studies comparing groups of college students coached to fake ADHD, students with a history of ADHD, and controls indicate that it is easy to fake symptoms of ADHD (Jasinski et al., 2011; Sollman et

al., 2010). Consistent with the findings of Marshall and colleagues (2010), SVTs were helpful in distinguishing between valid and feigned ADHD symptoms. Again, a criterion of failure on two or more SVTs provides an effective threshold with which to identify malingering among college students.

There are several steps clinicians can take in the course of standard practice to be alert to the possibility of individuals who may be faking ADHD. In cases of potential secondary gain, there are formal SVTs that can be employed as a safeguard. Furthermore, Barkley (2011c) discusses a method of "triangulation" of the multiple sources of information against each other to evaluate consistency of findings. One can evaluate the number of gross disparities as an indicator of potential malingering by examining all possible three-way combinations of (1) current and past symptom pictures; (2) history of onset, course, and any remissions; (3) present and past impairments; (4) collateral reports of items 1–3; (5) rating scales; (6) test results, including SVTs; and (7) archival records, when available.

SUMMARY

In this chapter I have outlined a template for a comprehensive diagnostic evaluation for adult ADHD. Such evaluations are specialized inasmuch as there is specific attention paid to and inventories used to identify symptoms and impairments consistent with ADHD, as well as the assessment of the current diagnostic criteria for ADHD in adults. However, many components are found in any competent psychological evaluation in order to consider other possible explanations for the presenting problems. There are cases in which it is helpful to administer additional testing, though it is important to understand the limits of testing, both with regard to making a diagnosis of ADHD and establishing EF deficits. Moreover, there are situations in which the evaluator will have to consider the possibility that a patient is faking ADHD in order to secure secondary gains. Finally, clinicians who perform psychological assessments for adult ADHD must at times be able to communicate negative findings to patients and direct them to more appropriate treatments. The ultimate goal for the evaluation is to increase diagnostic accuracy in order to provide a coherent formulation of presenting difficulties that will inform treatment.

KEY CLINICAL POINTS

- ✓ The evaluation of adults for ADHD includes establishing the presence of not only developmentally inappropriate symptoms or other DSM-5 diagnostic criteria but associated neuropsychological deficits, comorbidity, impairments not represented in DSM-5, other sources of distress, and the potential for malingering.
- ✓ The ideal approach is to employ a multimethod, multisource paradigm that not only permits a comprehensive survey of patient functioning but also contrasts sources for relative consistency with what is known about ADHD in adults.
- ✓ This approach uses unstructured and structured clinical interviews and rating scales, ideally with both the patient and a collateral person who knows the patient well; past evaluations and archival records, if available for education, driving, health, and other domains; academic achievement and intellectual screening tests to rule in or out comorbid learning or intellectual disabilities; and other neurocognitive testing and rating scales of EF if those domains are of interest.
- ✓ However, neuropsychological testing of EF is seriously limited due to poor ecological validity and low accuracy in detecting ADHD in adults, and should not be used alone. Rating scales of EF appear to be more ecologically valid and predictive of ADHD in adults than are tests.
- ✓ Given that inattention is common in many mental disorders, differential diagnosis is critical to distinguish the types of inattention often linked to ADHD (lack of persistence, low resistance to distraction, high variability of task performance, poor task reengagement, impaired working memory) from those seen in other disorders ("daydreamy," spacey, mentally preoccupied, hypervigilant, etc.), and more importantly, the presence of disinhibition and other executive deficits commonly found in the routine daily functioning of adults with the disorder (e.g., problems with time management, problem solving and self-organization, self-restraint, impulsive emotional behavior and poor emotional self-regulation, and self-motivation).
- ✓ ADHD represents a chronic disorder of disinhibition that includes inattention and poor self-regulation, with relatively early onset in contrast to other disorders that dates back to childhood or adolescence and no periods of spontaneous symptom remission.

- ✓ Up to 40% of individuals seeking an evaluation for ADHD may not meet full criteria for the disorder or may be found to have other mental disorders or explanations for their distress instead.
- ✓ In cases involving inattention but no evidence of disinhibition or poor self-regulation, the clinician should seek explanations other than ADHD, such as SCT (concentration deficit disorder, Chapter 17), or comorbid mood or other internalizing disorders, social adversity and associated distress, or health problems.
- ✓ Clinicians must be cognizant of the growing evidence of possible malingering (feigning symptoms) in adults seeking evaluations for ADHD. Various signs in the evaluation may suggest possible malingering and SVTs may be useful in detecting it. But there is no foolproof method for doing so. The "triangulation" of multiple sources of information against each other for consistency may aide clinicians in determining the existence of malingering.

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CHAPTER 20

Diagnosing ADHD in Adults in the Primary Care Setting

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Attention-deficit/hyperactivity disorder (ADHD) is one of the most common psychiatric disorders, affecting 6–9% of children worldwide (Faraone, Sergeant, Billberg, & Biederman, 2003). It is estimated that in up to two-thirds of children with ADHD the disorder persists into adulthood, with an estimated prevalence of 4.4% according to the National Comorbidity Survey Replication (NCS-R). Adult ADHD remains vastly underrecognized and undertreated, with only 10–25% of adults with the disorder diagnosed and adequately treated (Castle, Aubert, Verbrugge, Khalid, & Epstein, 2007). A recent survey of prescription fills revealed that only 10% of individuals with adult ADHD received treatment for the disorder in the previous year (Kessler et al., 2006; Kessler, Adler, Barkley, et al., 2005). As numerous prior chapters attest, the costs to the individual, family, and society from untreated ADHD are substantial; untreated ADHD has been associated with higher rates of academic underachievement, unemployment, underemployment, divorce, marital separation, early-onset substance abuse, cigarette smoking, and motor vehicle accidents (Barkley, 2002; Biederman, Faraone, Spencer, Mick, & Monuteaux, 2006; Biederman, Monuteaux, et al., 2006; Eakin et al., 2004; Mannuzza & Klein, 1999; Wilens & Dodson, 2004). Because many of these associated conditions and impairments are likely to be seen and treated in primary care settings, they underscore the need for clinicians working in those settings to be able to diagnose ADHD accurately in adults. We discuss in this chapter the issues involved in diagnosing adults with ADHD as they apply to primary care providers (PCPs). As such, the subject matter in this chapter of necessity overlaps to some degree with the prior chapter on psychological assessment of adults for ADHD.

HISTORY OF ADHD IN THE DSM

PCPs should understand that ADHD has a long and distinguished history, despite occasional media claims that it does not exist or is a far less serious problem than is in fact the case (see Chapters 1, 10–13). The first mention of ADHD in the *Diagnostic and Statistical Manual of Mental Disorders* (DSM; see Chapter 1), was in DSM-II in 1968, under the classification of "Hyperkinetic Reaction of Childhood." Early definitions of ADHD emphasized the hyperactive behaviors and restless symptoms (American Psychiatric Association, 1968; Biederman, Mick, & Faraone, 2000). The third edition of DSM (DSM-III), published in 1980, changed the name of the disorder to attention deficit disorder

(ADD) and expanded its definition by acknowledging that symptoms of inattention can occur independently of hyperactive and impulsive symptoms. DSM-III also presented two subtypes of the disorder: ADD with hyperactivity and ADD without hyperactivity. The revised third edition of DSM (DSM-III-R; American Psychiatric Association, 1987) changed the name to ADHD and removed the two subtypes found in DSM-III. This edition was the first time that the DSM included an active diagnosis of ADHD in adults.

The fourth edition of DSM (DSM-IV; American Psychiatric Association, 1994) reclassified the symptoms of ADHD into three subtypes based on a series factor analyses published at the time and DSM-IV field trials: predominately inattentive, predominately hyperactive, and a combined subtype that included both inattentive and hyperactive symptoms of ADHD (Lahey et al., 1994). A text revision of DSM-IV was published in 2000 (DSM-IV-TR) but no substantive changes were made to ADHD. The fifth edition of DSM (DSM-5), published in 2013, kept most of the criteria from DSM-IV and DSM-IV-TR, with several important changes, which are detailed below (see also Chapter 2).

ADHD Diagnostic Criteria

PCPs familiar with DSM-IV and more recent DSM-5 understand that few changes occurred across these editions, but those that did are very pertinent to diagnosing adults with ADHD. DSM-IV-TR diagnostic criteria for ADHD were the same for children and adults and can be reliably used to diagnose individuals who are currently experiencing symptoms of the disorder and those who, in the case of older adolescents and adults, have a history of significant symptoms since childhood. Establishing the childhood onset of symptoms in older adolescents and adults can be done by careful questioning of the individual, which is combined with parental or teacher reports for adolescents (Adler & Cohen, 2004). Collateral information, such as retrospective reports of childhood symptoms by parents or older siblings, or behavioral comments in elementary school report cards, can also be quite helpful in this regard (Adler & Cohen, 2004).

According to DSM-IV-TR, individuals must currently have six or more significant symptoms of inattention (IA) and/or six or more symptoms of hyperactivity–impulsivity (HI) (Criterion A) that have persisted for a minimum of 6 months and "to a degree that is maladaptive and inconsistent with developmental level" (American Psychiatric Association, 2000). Some of the symptoms must have been present before age 7 (Criterion B) and occur in at least two settings, such as at home and at school or work (Criterion C) (American Psychiatric Association, 2000). There must also be clear evidence of impairment in "developmentally appropriate social, academic, or occupational functioning" (Criterion D) (American Psychiatric Association, 2000). Finally, the symptoms cannot occur exclusively during the course of a pervasive developmental disorder, schizophrenia, or other psychotic disorder and are not better accounted for by another mental health disorder (Criterion E) (American Psychiatric Association, 2000). The symptoms of IA and HI are shown in Table 2.1 in Chapter 2. A diagnosis of ADHD requires that an individual meet all criteria from A through E.

DSM-IV classified ADHD into three subtypes. Individuals presenting with six or more IA symptoms and less than six HI symptoms meet the criteria for the predominantly inattentive subtype. Individuals presenting with six or more HI symptoms and less than six IA symptoms meet the criteria for the hyperactive–impulsive subtype. Individuals presenting with at least six IA symptoms and at least six HI symptoms meet the criteria for the combined subtype.

ADHD symptom loading tends to change over time, such that in childhood the IA and HI symptoms tend to be equally present; in adulthood, the frank hyperactivity tends to wane (and become much more a sense of internal restlessness); accordingly, the inattentive symptom loading increases with the transition to adulthood (Mick, Faraone, & Biederman, 2004). A survey of 149 clinic-referred adults with ADHD indicated clinically significant HI levels in approximately half of adults diagnosed with ADHD and prominent inattention symptoms in up to 90% (i.e., the IA symptoms were about twice as common as the HI ones; Millstein, Wilens, Biederman, & Spencer, 1997).

The finding of the increased loading of IA symptoms in adults was recently revalidated in an examination of patients in the NCS-R and a survey of a managed care population (Kessler et al., 2010). Significant symptoms of IA occurred in 77% of the sample, while HI symptoms occurred in 54% of the 90 patients examined (Figure 20.1). Furthermore, the diagnostic scale utilized in these studies, the Adult ADHD Clinical Diagnostic Scale (ACDS) version 1.2, employed an expanded symptom set to allow examination of common coexisting ancillary symptoms of executive function (EF) and emotional control (EC) not included in the

core ADHD symptoms noted in DSM-IV. EF symptoms include deficits in working memory, organization, planning, prioritization and time management, while EC symptoms involve changeable mood and emotional overreactivity. A recent analysis of data from these studies (presented by Adler to the Annual Meeting of the American Professional Society of ADHD and Related Disorders, Washington, DC, September 2013) showed that symptoms of IA and EF were equally common and occurred with significantly higher prevalence than the equally low-prevalence HI and EC symptoms (Figure 20.1).

PCPs who are unfamiliar with the newly published DSM-5 need to know that a number of changes were proposed by the DSM-5 workgroup when considering possible changes to the criteria for adult ADHD in the latest version of the manual (American Psychiatric Association, 2010). The suggested symptomatic changes included only requiring at least four IA or HI symptoms (as opposed to six) for those 17 and older, expanding the descriptions and prompts for existing symptoms, and inclusion of four new impulsivity symptoms in the HI subset (namely, "acts without thinking," "impatient," "uncomfortable doing things slowly and systematically," and "difficult to resist temptations or opportunities"). Other possible changes included increasing the age-of-onset criteria to 12 years (from 7 years), expanding the descriptions of settings to allow clinicians to probe for specific impairments, and expansion of the description of impairment (Criterion D) to include "symptoms [that] interfere with or reduce the quality of social, academic, or occupational functioning." The workgroup also considered changing the term "subtype" to "presentation." Furthermore, it suggested changing the combined subtype to require at least four HI and four IA symptoms. The workgroup also suggested splitting the IA subtype into two separate entities: IA restrictive and predominantly IA. The IA restrictive presentation would require at least 6 symptoms of IA (at least four for older adolescents/adults) with at most two HI symptoms. However, for the predominantly IA subtype, while still requiring the six symptoms of IA (or four for older adolescents/adults), the workgroup would limit the HI symptoms to at least three but less than six. Similarly, for older adolescents/adults with predominantly HI subtype, they considered making the criterion at least four HI symptoms, without changing the criterion for children.

The proposal for lowering the symptom threshold was supported by studies indicating that a valid diagnosis of ADHD could be established by a smaller number (than six) of significant symptoms (Barkley, Murphy, & Fischer, 2008). The proposed lowering of the age-of-onset criterion was buttressed by an examination of adults presenting with an older age of onset (into young adulthood) that looked identical to that of a cohort

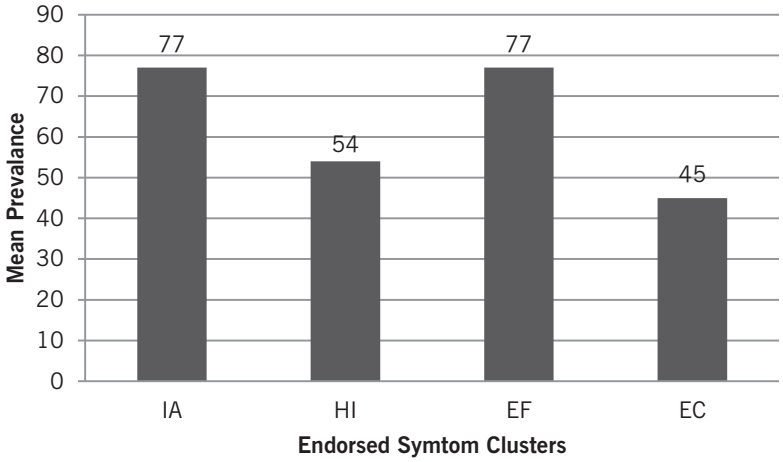

FIGURE 20.1. Inattention drives presentation of ADHD in adults: community-based and managed care samples (Kessler et al., 2010). IA. inattentive; HI, hyperactive–impulsive; EF, executive function; EC, emotional control.

of adults with childhood onset of significant ADHD symptoms in terms of symptom severity, impairment, and comorbidity (Faraone et al., 2006). However, not all of these proposed changes were accepted in the DSM-5, which was published in May 2013. The number of required symptoms for both the IA and HI subtypes was reduced from six to five for individuals 17 or older. Of note, although the required number of symptoms for adults has been decreased from six or more to five or more, the symptom sets were not expanded to include symptoms of EF and EC. The age of onset was increased from 7 to 12 years of age. The DSM-5 also now specifies that several of the symptoms must be present prior to age 12 and that several symptoms must occur in two or more settings. It further specifies that several symptoms must be present in each of the settings (school/work, home or social). The DSM-5 refers now to subtypes as "presentation." Finally, DSM-5 removed comorbid autism spectrum disorders as an exclusion criterion.

DIAGNOSING ADULTS WITH ADHD

Many adults are not diagnosed with ADHD in childhood, perhaps because of the structure of their environments or the minimal demands made on them, among other reasons. It is suggested that these individuals have increased impairment as adults because as children and adolescents they were undiagnosed and untreated for so long (Adler & Cohen, 2004; Millstein et al., 1997; Murphy & Adler, 2004). To accurately evaluate ADHD in adults, PCPs should review childhood onset, persistence, and pervasiveness of current symptoms (Spencer & Adler, 2004). A critical element in making an accurate diagnosis of adult ADHD is taking a comprehensive longitudinal history and reviewing symptoms, impairments, and co-occurring psychiatric disorders; nothing replaces the longitudinal history as the linchpin for making an accurate diagnosis of ADHD in adults because it helps to establish the key diagnostic criteria noted earlier, along with what must be ruled out (Adler & Cohen, 2004; Murphy & Adler, 2004). There are a number of structured diagnostic scales that can assist PCPs in making this diagnosis; these scales include Conners' Adult ADHD Diagnostic Interview for DSM-IV (CAADID; Epstein, Johnson, & Conners, 2001), the ACDS (Adler & Cohen, 2004), the Barkley Adult ADHD Rating Scale–IV (BAARS-IV; Barkley, 2011a), the Brown ADD Diagnostic Form (Brown, 1996), and the Diagnostic Interview for ADHD in Adults (DIVA; Kooij, 2012).

The PCP who confronts a possible case of adult ADHD must assess symptoms from the previous 6 months, as well as symptoms that occurred prior to age 12. Currently, a diagnosis of adult ADHD using DSM-5 criteria cannot occur without demonstrating childhood onset of significant symptoms. Although retrospective self-reports have at times been called into question, studies have shown them to be a reliable method of diagnosis (Murphy & Schachar, 2000). Collateral information can also be quite helpful in establishing current and childhood symptoms, along with current impairment (Murphy & Adler, 2004). This collateral information from childhood can include interviews with family members (e.g., parents or older siblings) who can report on symptoms, behaviors, and impairments or a review of school records, such as old report cards for academic performance and any noted behavioral problems. Similarly, collateral information, which can be helpful in assessing adult symptoms, can be obtained through interviews with significant others (boyfriends/girlfriends, spouses or possibly colleagues/supervisors at work or school (Murphy & Adler, 2004); appropriate medical release must be in place prior obtaining such collateral information. However, the ultimate arbiter of making the diagnosis is the clinician, who has the ability to integrate information from multiple sources; it has been shown that in adults, the clinician ratings of adult ADHD symptoms prior to treatment are stronger predictors of treatment response than self-report (Adler, 2008). In making this assessment, as noted earlier, PCPs need to note that HI symptoms often diminish with age (Biederman et al., 1996, 2000; Kessler, Adler, Ames, et al., 2005; Kessler et al., 2010; Millstein et al., 1997). The motivation for seeking a primary care evaluation may also be a useful piece of information because adults often seek help for impairment in academic, professional, or interpersonal difficulties, or because a family member (often a child) has been diagnosed with ADHD (Murphy & Adler, 2004).

Childhood ADHD is more commonly diagnosed in boys, with earlier estimates of the ratio of boys to girls as high as 4:1 and more recent estimates indicating that ADHD is twice as common in boys (Biederman et al., 1994; Gaub & Carlson, 1997; Ramtekkar, Reiersen, Todorov, & Todd, 2010; Visser et al., 2014). The discrepancy is thought to be due to the relatively higher loading of IA symptoms in girls, while boys have

a relatively higher loading of HI symptoms. Since HI symptoms are more likely to cause increased behavioral disruption in the classroom, it is posited that boys are more likely to be identified as possibly having ADHD than girls, who may be seen as underperforming because of IA symptoms (Biederman, 2004). PCPs need to appreciate that since hyperactive symptoms tend to be less severe in adults, distribution of diagnoses between men and women is much closer to even, at around three to two (Biederman, 2004; Biederman et al., 1994) if not equal (Barkley, 2011a).

Various factor analyses have revealed three subtypes: predominately IA symptoms, predominately hyperactive symptoms, and a combination of IA and hyperactive symptoms; the combined subtype is the most common (Kooij et al., 2005). The decreasing loading of frank hyperactivity in adulthood and increasing prevalence of the IA subtype is reflected in the prevalence rates of ADHD subtypes. In adulthood, the accepted distribution of subtypes is as follows: predominantly IA, 25%; combined, 70%; and HI, 5% or less (Adler & Cohen, 2004).

Establishing Impairment

Establishing impairment is a key element in making the diagnosis of ADHD in adulthood; impairment must be significant in two out of three domains, but it can include relative impairment, such as underperformance (American Psychiatric Association, 1994, 2013); symptom presentation alone is not sufficient. The noted symptoms must cause real-life consequences in terms of the patient's functioning. Two scales have been developed that can assist in the documentation of impairment by PCPs. The Barkley Functional Impairment Scale (BFIS; Barkley, 2011a) assesses impairment in 15 different domains, including Home/Family, Home–Chores, Work, Social–Strangers, Social–Friends, Community Activities, Education, Marriage/Cohabitating/Dating, Money Management, Sexual Relations, Driving, Daily Responsibilities, Self-Care Routines, Self-Maintenance, and Child Rearing. The BFIS was standardized on a normative sample of 1,249 adults and establishes cutoffs for impairment in 15 real-world domains on the basis of severity (0, *none* to 9, *severe*). Establishment of impairment is based on comparison to the normative sample for each of these 15 domains and also for a mean impairment score and percentage of domains impaired.

The Weiss Functional Impairment Rating Scale—Self-Report (WFIRS-S) is another self-rating scale of functional impairment (Canadian ADHD Resource Alliance [CADDRA], 2000a, 2000b). The WFIRS-S is a frequency/severity-based scale, rated from 0 (*never or not at all*) to 3 (*very often or very much*), including an n/a (*not applicable*) designation. The domains of impairment queried include Family, Work, School, Life Skills, Social, Self-Concept, and Risk, with eight to 14 questions in each domain. A mean overall impairment score can be calculated; items scoring 2 (*often or much*) or 3 denote significant impairment because they are two to three standard deviations above the normative sample. Impairment for each of the individual domains noted earlier is established by two items scoring 2 or one item scoring a 3 in that specific domain.

Comorbidities

Because adult ADHD commonly co-occurs (is comorbid) with a number of other psychiatric disorders, such comorbidities may be the main complaint of the adult presenting to a PCP for evaluation. A carefully taken history may reveal that ADHD is an associated condition and likely had an earlier age of onset than the disorder for which the adult is seeking treatment. Other disorders have been reported to occur in up to 50% of adults diagnosed with ADHD (Biederman, 2004; Kessler et al., 2006). As discussed in Chapter 13, common comorbidities include major depression, dysthymia, bipolar disorder, anxiety disorders (generalized anxiety, panic, and obsessive–compulsive disorders), antisocial personality disorder, and substance use disorders. For the PCP, the longitudinal history is again critical in establishing whether noted symptoms and impairment are from ADHD or a comorbid condition. There may be significant symptom overlap between adult ADHD and the comorbid mental health disorder, such as the impulsivity seen in mania, inattention as a result of substance use, or difficulty initiating tasks in major depression (Adler & Cohen, 2004). In making this differential comparison, it is important for the PCP to examine the onset and time course of symptom presentation. For example, mood disorders tend to be episodic, whereas ADHD symptoms have an onset prior to the age of 12 years and are more or less present throughout the lifespan; therefore, the clinician should examine for the presence of ADHD-related symptoms prior to the onset, or at periods or times of quiescence, of the

mood disorder. Similarly, substance use disorders have a specific time of onset–offset, which should be compared to the time course of ADHD-related symptoms and impairments. However, when both conditions are active, the ability to establish the differential contribution of the conditions may be problematic (Murphy & Adler, 2004). Clinically, the differential between adult ADHD and comorbid mental health disorders can be one of the most vexing issues facing a PCP making the diagnosis of ADHD. Taking a careful longitudinal history, and noting onset and persistence of symptoms and impairments is the most critical element in this differential. Finally, given the high rates of co-occurrence of ADHD with other mental health disorders and health conditions (Chapters 11–13), PCPs should be vigilant in assessing whether adult ADHD might be present in patients with these other disorders (bipolar, anxiety, and major depressive disorders; obesity, eating pathology; substance dependence, impaired driving, and marital conflict; etc.), who might not be responding adequately to treatment because the untreated ADHD might be a major contributing factor to the noted insufficient response (Adler & Cohen, 2004).

Screening for Adult ADHD and the Importance of Making the Diagnosis

Given its relatively high prevalence, ADHD is likely to be seen in primary care settings. A survey of 400 PCPs who commonly treated adults with mental health disorders reviewed the understanding, knowledge base, and diagnostic/treatment preference of PCPs regarding ADHD and other mental health disorders (Adler, Shaw, Sitt, Maya, & Morrill, 2009). The PCPs were asked to score the five items on a 5-point scale from lowest (i.e., *poor*) to highest (i.e., *extremely knowledgeable/thorough*): (1) knowledge of the disorders; (2) quality of education and/or training in the disorders; (3) perceptions regarding specific aspects related to the diagnosis and treatment of adult ADHD; (4) perceptions regarding the quality of diagnostic tools available for the target disorders; and (5) perceptions regarding the need for an effective screening tool for adult ADHD. PCPs felt that they were significantly more knowledgeable about, and received better training in depression, bipolar, and anxiety disorders than in adult ADHD; they were twice as likely to refer individuals with adult ADHD as those with suspected bipolar disorder to others to establish the diagnosis. Additionally, 85% of respondents felt a need for a validated screening tool to assist in the diagnosis of adult ADHD. The PCPs' responses to the survey highlight not only the need for improved knowledge and education of PCPs in treating adult ADHD, but also their interest in and the potential utility of a validated screening tool for the disorder.

Screening tests are designed to identify individuals at risk for a particular disorder, in order to determine whether further investigation is necessary to establish the diagnosis in question. Screening tests are commonly used in primary care settings to identify individuals at risk for other psychiatric disorders, including depression (Patient Health Questionnaire [PHQ-9]; Kroenke, Spitzer, & Williams, 2001) and bipolar disorder (Mood Disorder Questionnaire [MDQ]; Miller, Klugman, Berv, Rosenquist, & Ghaemi, 2004).

The Adult ADHD Self-Report Symptom Scale version 1.1 (ASRS v1.1) Screener is a screening tool developed by the workgroup on adult ADHD for the World Health Organization (WHO) to identify individuals at risk for adult ADHD; it is not designed to provide a full diagnosis. The ASRS v1.1 comprises two scales: a six-item Screener for general use and an 18-item Symptom Checklist for patients who might be at risk. The latter contains queries about the 18 symptom domains identified in DSM-IV, with modifications to reflect the adult presentation of ADHD symptoms and provide a contextual basis for the symptoms. Symptoms are rated on a frequency basis, ranging from 0 (*none*) to 4 (*very often*) (Kessler, Adler, Ames, et al., 2005). The six-item ASRS v1.1 Screener is extracted from the full 18-item symptom assessment scale and selects the six items that were most predictive of ADHD based on psychometric factor analyses of the diagnostic interviews of patients with and without ADHD in the NCS-R (Kessler, Adler, Ames, et al., 2005; Kessler et al., 2010). The ASRS Screener has shown good sensitivity and specificity, and has a positive predictive value, between 57 and 93% (Kessler, Adler, Ames, et al., 2005; Kessler et al., 2010).

Neither the 6-item ASRS v1.1 Screener nor the full ASRS 18-item Symptom Checklist are designed to be used as stand-alone diagnostic instruments. The diagnosis of ADHD is predicated on assessment of current symptoms, impairment, childhood onset of symptoms, and ascertaining that the noted symptoms and impairments are a result of ADHD and not another mental health disorder. Four of the six items are IA symptoms and the remaining two are symptoms of HI. The

Screener has been translated into over 20 languages and has been validated as a screening instrument in patients with comorbid substance use disorders (Adler, Guida, Irons, Rotrosen, & O'Donnell, 2009). It has been used on days set aside for public screening for adult ADHD, to identify individuals at risk for the disorder who may need further evaluation to receive a diagnosis (Adler, Ciranni, Shaw, & Paunikar, 2010); PCPs could use the ASRS v1.1 Screener to screen their practices, or segments of their practices (e.g., individuals with depressive or substance use disorders who are at higher risk for ADHD), and establish which patients might merit further evaluation for adult ADHD. However, the ASRS v1.1 Screener has not yet been validated (in terms of full psychometric properties) for assessing the likelihood of making a diagnosis based on DSM-5 adult ADHD criteria (just DSM-IV); validation studies are currently being planned. However, as discussed earlier, the major changes in DSM-5, mostly somewhat liberalize the criteria (increasing the age of onset and decreasing the symptom threshold cutoff); therefore, patients who screen positive for being at risk for adult ADHD based on DSM-IV criteria using the ASRS v1.1 Screener are also likely to be at risk for having adult ADHD based on DSM-5 criteria. Individuals who screen positive or are close to meeting "screen positive" criteria (but still screen negative) given the modest liberalization of the criteria, still merit a full diagnostic evaluation for whether they have adult ADHD based on DSM-5 criteria.

KEY CLINICAL POINTS

- ✓ ADHD is a common psychiatric disorder that is likely to be evident in PCP settings. It is prevalent in adults and is associated with significant impairments.
- ✓ The diagnosis is established on the basis of a thorough longitudinal clinical interview that closely follows the criteria put forth in DSM-5; this remains the "gold standard" based on assessment of current symptoms and impairments, and evaluation of childhood symptoms.
- ✓ It is also imperative that the clinician establish that the noted symptoms and impairments are a result of ADHD and not another mental health disorder.
- ✓ Collateral information from parents, siblings, or significant others can be quite valuable in establishing symptoms and impairments.
- ✓ Several diagnostic scales can assist the clinician in performing the interview. Screening tools, such as the ASRS v1.1 Screener, can be used by PCPs to identify individuals at risk for the disorder, but they cannot be used as stand-alone methods for diagnosis.

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PART III

Treatment of Children and Adolescents with ADHD

CHAPTER 21

Training Parents of Youth with ADHD

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Considerable data clearly attest to the often poor long-term outcomes of youth with attention-deficit/hyperactivity disorder (ADHD) well into adulthood (Klein et al., 2012; see Chapter 9). Interestingly, individuals who have a childhood history of ADHD but no longer meet criteria for ADHD in adolescence/young adulthood continue to suffer more functional impairments than do their nonaffected peers (Lee, Lahey, Owens, & Hinshaw, 2008; see Chapters 9, 11, and 12). As such, ADHD and its associated consequences appear to impact the vast majority of youth negatively throughout life. Given the chronic nature of ADHD, active involvement of parents of youth with ADHD is essential to the successful course of their children's lives across multiple domains of functioning. Unlike any other individuals, parents play a consistent and principal role in decisions regarding pursuit of an assessment for ADHD, decisions regarding (varied) treatments across multiple settings over time, and advocacy for their children, among the many other issues and factors that are part of parenting. Optimal long-term outcomes for a child are achieved when parents' central consideration is their role as proactive, engaged decision makers for their child throughout development to ensure that their child is obtaining any and all appropriate services (educational, medical, psychological, etc.) to maximize functioning. As such, effectively supporting parents of youth with ADHD to manage their child's challenges across development is a critical role played by various professionals. Although there are multiple ways in which a professional can provide support to parents, we focus our attention herein on the delivery of behavioral parent training (BPT), one of the most well-studied psychosocial interventions for ADHD (Evans, Owens, & Bunford, in press; Pelham & Fabiano, 2008).

RATIONALE FOR WORKING WITH PARENTS

Parenting per se has not been implicated in the etiologies of ADHD; however, there are several notable reasons for working directly with parents in BPT. First, youth with ADHD have a significant impact on their parents, siblings, and family life in general (Johnston & Mash, 2001; see Chapter 7). Parents are more stressed, there is greater sibling conflict, and there are higher rates of parent–child and family conflict in the families of children with ADHD. Moreover, parents of youth with ADHD are more likely to exhibit ineffectual parenting (e.g., inconsistent, harsh, lax, overreactive, less

responsive) and lower parenting efficacy/competence, higher levels of coercive management practices, and to utilize maladaptive coping strategies (e.g., increased use of alcohol), and have more negative attributions/perceptions of their child (see Chapter 7). The development of child and parental difficulties may be reciprocal in nature. Parents, in an effort to manage a child's behavior, utilize various harsh and punitive methods (e.g., yelling, threatening, corporal punishment) when more proactive and nonpunitive discipline strategies (e.g., removal of privileges; time-out from positive reinforcement) do not appear to work. The behaviors of both the parent and the child may escalate in a coercive process leading to more challenging child behavior and increased harsh, intrusive, and punitive behavior on the part of the parent. Over time, parental efficacy and coping strategies may deteriorate given the challenges of parenting a child with ADHD, which may further result in lax and inconsistent parenting behavior. This effect of child behavior on the parent and family, as well as this coercive cycle, is particularly true of youth with ADHD and comorbid oppositional defiant disorder(ODD)/conduct disorder (CD). Importantly, BPT often directly addresses these issues (parenting practices; coping strategies, etc.; Barkley, 2013) and has been shown to improve many of these outcomes (Chronis, Chacko, Fabiano, Wymbs, & Pelham, 2004; Lee, Niew, Yang, Chen, & Lin, 2012; Rajwan, Chacko, & Moeller, 2012).

Parenting and parent–child difficulties of youth with ADHD have also been tied to the development of other psychiatric disorders in youth. For instance ADHD may place children at risk for the development of ODD/CD, as well as increase the severity of ODD/CD problems over time (Chronis et al., 2007; Pardini & Fite, 2010). Research indicates that approximately 65–80% of clinic-referred children with ADHD also meet criteria for ODD or CD (Jensen, Martin, & Cantwell, 1997; see Chapter 5). Moreover, youth with ADHD and comorbid CD appear to have worse outcomes than youth with only ADHD or CD (Loeber, Burke, Lahey, Winters, & Zera, 2000). These negative outcomes are likely given the impact of ADHD on parenting behavior (e.g., coercive, punitive, harsh), which is closely associated with the development and persistence of ODD/CD and more antisocial/criminal behavior (Burke, Pardini, & Loeber, 2008; Loeber et al., 2000).

Additionally, there is an association between ADHD and later depression in youth (Biederman, Mick, & Faraone, 1998). Studies have demonstrated that parenting practices (Ostrander & Herman, 2006) and parent–child problems (Humphreys et al., 2013) mediate the relationship between ADHD and later depression. Furthermore, some evidence also suggests that negative parenting (e.g., intrusive) of children with ADHD may also confer risk for childhood anxiety disorders (Kepley & Ostrander, 2007; Pfiffner & McBurnett, 2006). Given this complex predictive and interactive relationship between ADHD and ODD/CD, as well as depression and anxiety, prevention or treatment of factors (i.e., parenting practices and the parent–child relationship) influencing the development of these comorbid disorders is critical. In this regard, BPT is an effective intervention for preventing and treating ODD/CD, as well as improving parenting practices and parent–child relationships (Eyberg, Nelson, & Boggs, 2008; McCart, Priester, Davies, & Azen, 2006).

Collectively, it appears that BPT is warranted for families of youth with ADHD who experience notable challenges in coping with their child's behavior, have significant parent–child relationship difficulties, and experience parental or familial stress. BPT may be particularly warranted for youth with ADHD who have co-occurring oppositional and/or conduct problems. For those youth with ADHD (e.g., primarily inattentive presentation) whose behavior does not impact parent or family functioning, formal enrollment into BPT may not be warranted; however, these families may still benefit from a "low-dose," less structured, less intensive behavioral intervention than formal BPT. As we have experienced clinically, parents gain much more from BPT than just specific strategies, and for many families, the intangible benefits of participating in BPT are perceived as most important. For instance, many parents have noted that obtaining social support from peers (in group-based BPT) and therapist(s), discussing the experience of parenting a child with ADHD, and learning about various approaches to advocate and support their child (among many others issues) are equally important. Given this, it can be argued that all families of youth with ADHD can benefit from the supportive and information-sharing aspects that are often part of BPT.

We argue in this chapter that providing BPT for parents of youth with ADHD addresses not only the short-term, pressing needs that bring families to treatment but also it is an opportunity to prime parents for engaging in clinical care, broadly defined, over the longer-term (e.g., learning to effectively advocate for their child's needs; communicating clearly with providers). In our opinion, given the chronic nature of

ADHD, effective engagement of a parent in a child's immediate treatment with an eye toward preparing the parent for what is often long-term management of ADHD is an important goal. In this chapter we focus on a two-phase framework for providing BPT with parents of youth with ADHD: (1) a preparation phase and (2) an implementation phase. It is important to note that although we frame these two phases as distinct in this chapter, this is merely for the sake of providing some structure rather than a true consistent practical distinction. For some families, content discussed in the preparation phase is often discussed again during the implementation phase.

PREPARATION PHASE

Preparing parents for BPT is essential as many parents who are interested in BPT never attend a single session. In a randomized clinical trial comparing a traditional group-based BPT to an enhanced group-based BPT, Chacko, Wymbs, Chimiklis, Wymbs, and Pelham (2012) found that, despite discussion of BPT (format, content, and process) and parent involvement in BPT (active participation, completion of between session homework, etc.), approximately 28% of families in the traditional group-based BPT never attended a single session of BPT. In the enhanced group-based BPT, which focused on addressing barriers, expectations, and attributions, only 5% of families failed to attend a single BPT session. This finding has at least two implications for preparing parents for BPT. First, given that some families never attend BPT, supporting parents in developing skills that lay the foundation for productive engagement in future interactions with health professionals is an important goal. Second, providing systematic psychoeducation about BPT *and* addressing barriers to engagement (as was done in the enhanced group-based BPT condition) can significantly improve parent attendance to BPT. Moreover, when parents agree to enroll formally in BPT, a clinician should also focus efforts on clear goal setting and progress monitoring, which are essential to determine response to BPT and make the necessary modifications to treatment based on progress. More specifically, a clinician should work with parents to identify and prioritize goals that are clearly defined and can be monitored objectively in order to determine if these goals are being addressed effectively during the course of BPT and determine the extent to which BPT will have to be modified if progress on parent-identified goals are not being achieved. We next discuss these steps toward effective preparation of a parent for BPT.

Developing a Foundation for Productive Parental Engagement in Clinical Care

Given that many parents who express an interest in participating in BPT will not enroll in BPT, we have found that focusing some efforts on supporting parents to be more effective communicators in order to be engaged consumers of care for their child is an important first step. In fact, given that ADHD is chronic and there are likely many episodes of care for youth with ADHD over the course of time, teaching parents effective methods to communicate their needs is important for all families because studies have documented that parents often poorly communicate their needs to health care providers (Brinkman et al., 2011). This is particularly problematic because research indicates that parents of youth with ADHD feel that they are primarily responsible for decisions regarding their child's care, not health care professionals (Davis, Claudius, Palinkas, Wong, & Leslie, 2012). Unfortunately, parents of youth with ADHD report higher levels of stress, which may interfere with cognitive processing and communication skills, limiting their ability to participate effectively as members of their child's treatment team (Brinkman, Sherman, & Zmitrovich, 2009). Additionally, the high rates of ADHD, mood disorders, anxiety, and other disorders in parents of youth with ADHD (Chronis et al., 2003) likely further impair parents' ability to communicate effectively with providers. The sense of responsibility that parents report regarding the care of their child, coupled with the intrapersonal stresses and challenges that may impede effective communication and clinical decision making, suggests that methods for supporting parents in development of effective communication skills is an important goal that may have long-lasting ramifications .

In the treatment literature, there are numerous examples of strategies that model and impart to parents how to organize and communicate information effectively within a time-limited context. Shared decision making in pediatric ADHD research offers several examples of improving parent–provider communication (Brinkman et al., 2009). For example, agenda setting is often an important part of cognitive-behavioral treatment and offers a circumscribed opportunity to identify issues to discuss during an assessment (and treatment) session.

Developing an agenda can play an important role in helping parents to learn critical skills for advocating for their child's and family's needs. In our experience, working with parents to develop an agenda requires (1) identifying key issues/questions/decisions, (2) operationally defining these issues/questions/decisions to get to the "core," (3) prioritizing the issues/questions/decisions in order of importance, and (4) agreeing on the subset of issues/questions/decisions that will be addressed during the session. Although these steps often take time, we have found that through therapist modeling and collaboration, parents often find this structured approach very useful and can ultimately complete these steps independently. Ultimately, a parent who is supported by a provider to be "empowered/activated" (e.g., with questions to ask, decisions to consider, and perspectives to share) is likely to be more engaged in the treatment process. Although not often seen as a treatment goal, in our opinion, supporting parents to be active communicators and decision makers is an important goal considering the chronicity of ADHD and the likelihood that families will engage with multiple providers (e.g., therapists, pediatricians, school staff) over time.

Systematic Psychoeducation about BPT and Addressing Barriers to Engagement

Over the past few decades, there has been literature on the role of various factors as predictors of dropout from treatment, including BPT. For instance, low socioeconomic status (SES) backgrounds, single-parent status, ethnic/minority status, parental psychopathology, parental stress, and severe child psychopathology are all factors associated with poor attendance and premature termination from BPT (Chronis et al., 2004). More recently, there has been an emphasis on understanding the role of more malleable factors related to treatment engagement. For instance, attention has been paid to practical/perceived barriers to treatment and parental cognitions (e.g., attribution for their child's behavior, perceived parental competency, expectations for treatment) as two important factors related to poor engagement to treatment (Chronis et al., 2004; Ingoldbsy, 2010; Morrisey-Kane & Prinz, 1999; Nock & Ferriter, 2005).

Based on the seminal work of Kazdin and colleagues (e.g., Kazdin, Holland, & Crowley, 1997; Kazdin, Holland, Crowley, & Brenton, 1997), practical and perceived barriers to treatment have been found to be predictive of engagement in BPT and contribute uniquely to dropout even after other familial, parental, and child variables had been controlled (e.g., single-parent status, SES). According to the barriers to treatment model, perceived barriers fall into four domains: (1) experience of stressors and obstacles (e.g., conflict with significant others); (2) relationship with the therapist (including perceived lack of support and disclosure from the therapist); (3) treatment relevance; and (4) treatment demands. Perceived relevance of treatment was found to be the best discriminator between treatment dropouts and completers, suggesting that parental perceptions are an important target prior to initiating BPT.

Parental attributions have also been shown to be related to engagement in BPT. For instance, research indicates that some parents of children with ADHD are more likely to attribute causes of their child's behavior to enduring symptoms of the disorder and something that is out of the child's control (Johnston & Freeman, 1997). For these parents, there is lower acceptability of interventions, such as BPT, that emphasize improving child behavior through altering contingencies in the environment (Johnston, Mah, & Regambal, 2010). Parents who assume some responsibility for their child's behavior are more likely to complete BPT (Peters, Calam, & Harrington, 2005). Additionally, although this requires further study, parents must perceive some minimal level of their own competence in parenting and parenting efficacy to find interventions focused on improving parenting behaviors to be acceptable. When parenting competence and/or efficacy are very low, parents may feel that any efforts at improving parenting behavior to improve child outcomes are futile. For instance, Johnston and colleagues (2010) demonstrated that mothers' parenting efficacy is significantly and positively correlated with engagement and acceptability of BPT. Importantly, acceptability of ADHD intervention is related to subsequent treatment initiation (Fiks, Mayne, DeBartolo, Power, & Guevara, 2013). Finally, parental expectancies may also play a key role in a parent's willingness to participate in treatment. Studies indicate that parents' inaccurate expectations regarding the content, format, and process of treatment, as well as the potency, rate, and expected course of therapeutic benefit/change, are related to premature termination of BPT (Morissey-Kane & Prinz, 1999; Nock & Kazdin, 2001).

Given what we know about the importance of parental engagement and attendance, are there strategies to help address factors related to poor engagement in BPT? Our perspective is that much can be done to sup-

port families in becoming more fully engaged in BPT. First, for some parents who may suffer from considerable psychopathology (e.g., depression, considerable substance abuse), it may be most appropriate for them to consider treatment for their own difficulties prior to enrolling in BPT. Importantly, the presence of these types of problems does not contraindicate involvement in BPT; some families can successfully participate and benefit from BPT even with these types of difficulties (Chronis-Tuscano et al., 2013). Also low-cost and minimally intensive (in terms of time and effort) methods to prepare parents for BPT may be offered, including the development of written or audiovisual materials that describe the content and process of BPT, as well as how parents are involved during BPT. These have been included in our work but are best developed for the type of BPT one will implement at a particular setting. As such, utilizing generic psychoeducation material regarding BPT may not as be as helpful (Chacko et al., 2009). Additionally, it is important to know that a one-time discussion of these issues is likely insufficient (Chacko et al., 2009) and that these factors typically need to be readdressed over the course of ongoing therapy. In our work, we have developed a relatively brief (approximately 20–30 minutes, but it may be longer depending on the number of issues and complexity of those issues) adjunctive interview that further clarifies expectations, addresses practical/perceived barriers and attributions for their child's behavior, and attempts to improve perceived parenting efficacy and motivation. Although we have typically administered this interview immediately after a diagnostic intake and during determination of the appropriateness of BPT, others (Nock & Kazdin, 2005) have interspersed these interviews throughout BPT. We agree that clinically it may be best to include portions of this interview over the course of ongoing therapy as structured brief check-in periods, which we often find are necessary for maintaining motivation for BPT over time.

If a parent decides to participate in BPT, we focus our attention on addressing the aforementioned factors in a systematic manner. For instance, at the time of intake, parents are asked about immediate practical concerns and possible barriers to ongoing involvement (child care, transportation, negative opinions about treatment from family members, etc.). Furthermore, we ask parents open-ended questions regarding their expectations about their own involvement in treatment (e.g., "What role do you think you will have in treatment?"), as well as that of their child (e.g., "In what way do you think your child will be involved in treatment?"). Parents also are asked open-ended questions regarding their expectations about the rate and potency of treatment-related improvements for their child (e.g., "How fast do you expect to observe improvements in your child's behavior? How much of an improvement do you expect you and your child to make during the course of treatment?"), and about their attributions regarding the locus of control of their child's behavior and the effect of their parenting (e.g., "What do you think causes your child to misbehave? Do you believe your parenting can make a difference in the way your child behaves?"). Although there are multiple methods to address barriers and maladaptive cognitions (all-or-none thinking, discounting, etc.) that arise as part of the interview process, we primarily utilize a problem-solving orientation (D'Zurilla & Nezu, 2007) within the context of other cognitive-behavioral methods. Clearly, it is well beyond the scope of this chapter to detail the use of such methods for various challenges that arise, but clinicians (particular those with a cognitive-behavioral orientation) often apply these methods in practice. Table 21.1 lists a set of questions we have used in our work. These questions are meant to elicit discussion between the therapist and parent(s) to clarify the reasons for seeking treatment; to help parents appreciate the rationale, course, and expected outcomes following treatment; to develop motivation; to anticipate common challenges experienced by parents; to develop and implement a plan that may address common barriers; and to begin the process of building a therapeutic alliance. Those utilizing such questions are encouraged to tailor this list for their own use.

GOAL SETTING AND PROGRESS MONITORING

Identifying objective and attainable goals that align with the putative effects of BPT prior to parental enrollment in BPT is a critical aspect of preparing for BPT. So, how does one consider developing a list of goals and monitoring progress? Our opinion is that parents should first describe, in clear behavioral terms, the specific behaviors that impair their child. Often, in the case of ADHD symptoms, it is helpful to describe the topography of the behavior (what is looks like to an observer) as well as when, where, and how these symptoms cause daily life problems (finishing homework, getting ready for bed, etc.) rather than focus on ADHD symptoms per se as behaviors to target in BPT.

TABLE 21.1. Questions Used to Elicit Discussion during Enhanced Intake

Reasons for seeking treatment

The goals for this section are to gain a better understanding of the motivation and investment for seeking treatment.

- "Why seek treatment now?"
- "What are the most important reasons you have come to get help for your child?"

History of treatment

The goals for this section are to determine what has been successful and unsuccessful in order to continue to support successful efforts and minimize making mistakes that have occurred in the past.

- "What things have you tried to do with your child to improve his or her behavior?"
- "What have been you experiences with other agencies, doctors?"
  - "What about these agencies or doctors did you like or find useful? What are some things that you didn't find useful?"

Attributions of child behavior

The goals for this section are to gain a better understanding of the locus of control parents have for their child's behavior. An understanding of this will help focus discussion of the appropriateness, possibilities, and limitation of treatment (e.g., BPT) for addressing their child's difficulties.

- "What are the reasons you think your child misbehaves?"
- "Have you had chances to see your child behave better, more politely, or be more friendly, or have you had chances to see him or her actually do what you ask and do it well?"
- "If so, why do you think he or she behaves differently at different times?"
- "What kind of effect do you think your parenting has on your child?"
- "Do you believe your parenting can make a difference in the way your child behaves?"

Attitudes and expectations

The goals for this section are to understand and clarify parent's expectations regarding treatment, rate and extent of benefit from treatment, and alignment of their goals with expected effects of treatment.

- "What do you think this treatment is about?"
- "Why do you think it's important to participate in this program?"
- "What do you think is going to happen during the session?"
- "What do you think about your role/involvement during the session and at home?"
- "What do you think about your child's involvement here and at home?"
- "What goals do you have for your child and yourself?"
- "How long do you think it's going to take before you see changes in your child's behavior?"
- "How much and what kind of changes do you think you will see?"
- "How much effort will it take on your part to support these changes?"

  At some point describe the treatment and its components, especially if the details of the intervention and the roles and responsibilities of the parent, child, and therapist have not been fully discussed. Additional questions include the following:
  - "We may discuss issues such as stress, other family members, your mood, and so forth. How do you feel about talking about these issues?"
  - "Are there certain things you don't want to talk about or would have a difficult time talking about?"
  - "Part of the session includes role plays, working with your child while others watch you and provide feedback, and practicing at home. How do you feel about this? Do you think it will be difficult to do this? Why do you think it may be important? What may get in the way?"
  - "This process is pretty time consuming. It will take X hours every week for X weeks. How do you feel about making this commitment? Is this the right time for you and your family to start treatment?"
- "What do you think my role as a therapist will be?"
- "What about my suggestions? Would you feel comfortable with them?"
- "Say you start treatment and things don't go that smoothly with your child. He or she is not responding well or as quickly as you want. How will you feel about that?"

(continued)

TABLE 21.1. *(continued)*

- "If things go well over a couple of weeks and your child then has a difficult week or two, how would you feel about it? Would you expect something like this to happen?"
- "If things don't go the way you want, do you feel comfortable talking to me about it? What are some things we could do to help you feel more comfortable?"

Practical and perceived barriers

The goals of this section are discuss a range of factors that typically interfere with full engagement to treatment and attempt to address these factors prior to initiating formal treatment.

- "What are some obstacles/challenges that could keep you from coming every week to the program? Is there anything we can do to assist you to keep that from happening?" (see below for common areas where obstacles/challenges may arise)
  - Transportation
  - Child care
  - Talking yourself out of coming
  - Family commitments/family conflict
  - Family beliefs/attitudes/support about seeking treatment
  - Work-related issues
  - Your child doesn't want to come
  - Treatment is too demanding
  - Treatment does not feel relevant/meet your needs

For each identified barrier, problem-solve with the parent strategies they can use now to address these barriers.

Note. These questions/points are meant to elicit a discussion between the therapist and parent(s) to clarify the reasons for seeking treatment; to appreciate that child behavior may be affected by different times of the day, settings, people, etc.; to help parents appreciate the rationale, course, and expected outcomes following treatment; to develop motivation; to anticipate common challenges experienced by parents; to develop and implement a plan that addresses common barriers; and to begin the process of building a therapeutic alliance. These questions are illustrative and should be expanded to address and improve upon the quality of discussion between the therapist and the parent(s).

When there is a list of identified target behaviors, parents should prioritize these problem behaviors. Parents' priorities relative to target behaviors should be a major focus of attention early in treatment given that working on parent-identified goals maximizes not only collaboration between the therapist and parents but also the parents' investment. We have found that spending time on understanding the range of target behaviors, parents' concerns about these behaviors, and supporting parents in decision making about which target behaviors on which to focus immediately sets an important foundation for establishing strong communication in treatment and can help parents perceive the value of BPT for achieving their goals. In addition, Janney and Snell (2000) offer a useful heuristic for categorizing these targeted behaviors based on the severity of the problem or (potential) impact on the child and others. These authors suggest that distracting behaviors include those that are atypical in frequency (e.g., interrupting during dinner) relative to behaviors of other children but do not pose any harm to the child or others. Disruptive behaviors are those that interfere with successful learning in various environments (e.g., noncompliance). Finally, destructive behaviors are those that pose potential harm to the child or others (e.g., aggression). It is evident that destructive behaviors are the highest priority for treatment, followed by disruptive and, finally, distracting behaviors.

Once target behaviors have been operationalized in clear behavioral terms and categorized, the parent and therapist should determine and prioritize which of these behaviors to address within the context of BPT (e.g., aggression at home; noncompliance at home; completion of homework). For behaviors that are unlikely to be improved by participating in traditional BPT (e.g., academic achievement, peer relationships at school), the therapist should work closely with the parent to obtain additional appropriate treatments that targets these areas of functional impairment (see the other chapters in Part III). For target behaviors that are amenable to BPT, clearly describing the goals of BPT in regard to these behaviors is important. At times, the

goal may be a reduction (in severity, frequency, duration, etc.) of the impairing target behavior. At other times, or in addition, goals may also include the "positive opposites" of the target behaviors. In other words, as often as possible, we want parents to focus on identifying behaviors to replace the impairing target behaviors (e.g., expressing frustration by communicating with "I statements" instead of blaming others and yelling). The list of final goals (reduction of impairing behaviors and increase in alternative/replacement adaptive behaviors) should be operationalized in terms of the topography and clear appropriate expectations for the child in terms of shaping procedures. This is particularly important for parents, many of whom expect a change in the target behaviors, such that currently impairing behaviors are completely replaced by the new adaptive behavior (a 180-degree change). While this may happen, it is unlikely that target behaviors will be completely eliminated or replaced by adaptive behaviors all the time and in every context. Aligning expectations of treatment effects with what is likely to occur following treatment (as we discussed in the previous section) is critically important to engagement and satisfaction with BPT.

Progress monitoring should be discussed as part of the pre-BPT preparation interview (discussed within the context of parental involvement in BPT), but it is always helpful to restate the expectation that parents will be involved in monitoring progress in BPT. It is important to state to parents that prospective measurement of the extent to which BPT is affecting selected target outcomes is important for several reasons. First, it is helpful for parents to appreciate the level of difficulties their child is experiencing at baseline. Second, implementing an evidence-based treatment does not guarantee improvement. Outcomes of BPT are moderated by many factors (a point we return to below) and, as such, ongoing prospective measurement of progress allows the parent and therapist to make informed decisions about tailoring ongoing treatment. Third, and likely most important, we want parents to have a framework to utilize when considering the impact of the (various and multiple) treatments in which their child may be engaged over time. Given the cost of treatment in terms of time and resources, parents who are educated to make decisions about the extent to which a treatment is working to meet identified goals will likely be better advocates for their child.

Progress monitoring should entail the use of both of idiographic and nomothetic measurement of progress. Idiographic measures are unique to the child and allow understanding of the extent to which BPT addresses the specific, parent-identified goals of treatment. Essentially, idiographic measurement includes the operationalized targets of treatment identified by the parent in preparing for BPT. As such, these should be the main targets of treatment. In order for these targets to be made more tangible, intermediate goals leading to the final attainment of the target outcome should be identified. For example, if the target outcome is completing homework within 30 minutes from a baseline level of 60 minutes, an initial intermediate treatment goal would be to complete homework in 50 minutes. Once consistently attained, a new intermediate goal would be established (e.g., reducing homework time to 40 minutes) until the target outcome is met.

Nomothetic measures are more general but allow the comparison of a score on a measure from an individual child to that of children in the broader population. For instance, a child's score on a psychometrically validated, normed, nomothetic measure of compliance can be used to determine the extent to which the child's score differs from what is typical for other children of the same age and sex. Additionally, nomothetic measures allow one to determine the extent to which treatment has clinical benefits (i.e., normalizes functioning). Tracking progress by using both idiographic and nomothetic measures allows a therapist–parent to determine the extent to which BPT is impacting the exact difficulties that parents want improved, while also gauging the extent to which treatment is normalizing functioning in key broad areas. The number of factors that a therapist would want to measure and the frequency of such measurement are important. A broad range of measures frequently administered is likely burdensome for many parents and therapists. An important mantra we utilize when considering a broad range of measures to track treatment progress is "good data are better than more data." What we have found best is to focus on having parents complete key child nomothetic measures of adaptive functioning and impairment (using psychometrically validated measures) every three sessions of BPT, while brief, idiographic assessments of parent-identified target goals are completed every session.

IMPLEMENTATION PHASE

There are likely many factors to consider when implementing BPT, which is considered the most well-studied

psychosocial intervention for children's mental health problems (Kazdin, 2005); as such, numerous studies have determined the importance of various parameters of BPT. Moreover, we believe that several important issues require consideration in the implementation of BPT and working with parents in the context of BPT. As such, prior to discussion of the content of BPT, we first address several of these key considerations.

Program Type

Over the past several decades, numerous BPT interventions have been developed, evaluated, and are now commercially available (e.g., Defiant Children [Barkley, 2013]; Community Parenting Education Program [Cunningham, Bremner, & Secord, 2006]; Triple P—Positive Parent Program [Turner, Markie-Dadds, & Sanders, 2010]; Parent Management Training [Kazdin, 2005]; Parent–Child Interaction Therapy [PCIT; McNeil & Henbree-Kigin, 2010]; Incredible Years Parenting Program [Webster-Stratton, 2006]). Although there are differences between some of the parameters of these BPT programs (group vs. individual, parent alone vs. parent and child, etc.), the contents of many evidence-based BPT programs are likely more similar than different (Garland, Hawley, Brookman-Frazee, & Hurlburt, 2008), a point on which we focus in our review of BPT content below. Although some interventions have been developed primarily for younger children (e.g., PCIT; Incredible Years), most of these interventions have been effectively used across the preschool- to school-age range (McCart et al., 2006). Importantly, the actual decision to use a particular BPT program is more likely a function of both therapist and parent preferences, as well as practical issues (e.g., space constraints; insurance reimbursement rates; availability of multiple providers to implement BPT; therapist training and preference).

Age

Although BPT is an important treatment for a wide age range, it is often most effective in preschool- to school-age youth (McCart et al., 2006). Once youth approach late elementary/early middle school, the focus on the relationship between a parent and child shifts from one in which the parent most prominently influences the environment to one in which both parent and child have increasingly shared influence. As such, interventions that focus on supporting communication between parents and children (or, more broadly, between family members) and mutually agreed-upon expectations and consequences for behavior are more likely to be effective for older youth (e.g., Chapter 22). In fact, some studies indicate that BPT may have an iatrogenic effect with an adolescent ADHD population (e.g., Barkley, Guevremont, Anastopolous, & Fletcher, 1992).

Format

As discussed earlier, there are varying formats in which BPT programs have been developed and particular advantages to various types of formats. For instance, groups allow for the benefits of peer support, information sharing, and development of ecologically valid ways in which core BPT skills can be implemented in "everyday life." However, groups follow a fixed schedule, content, and pace. As such, there is inflexibility when conducting a group, which may be problematic when families attend sporadically, do not fully "master" a particular BPT skill, or preceive some content as less relevant (an important predictor of poor engagement to BPT). In contrast, individual formats allow for more flexibility in scheduling, pacing, and delivery of content. This increased ability to tailor BPT in individual formats may be particularly important for families with considerable psychosocial stressors. In a meta-analysis of BPT interventions, Lundahl, Risser, and Lovejoy (2006) found that for families with low-SES backgrounds, where there is often an accumulation of stressors impacting parents, an individual format for BPT was more effective than a group-based format. An understudied method, but one that may potentially offer advantages, is to combine group and individual formats. As an example, a group-based BPT can be the standard format for all families, and individual formats can be offered to families who may require additional support, practice, or tailoring. Mixed group–individual BPT may offer an ideal opportunity, but it is not without practical challenges (insurance billing restriction, coordination between the therapist and each family, therapist and family availability, etc.).

The decision to offer a concurrent child group–individual treatment formats is also an important consideration. Some studies, particularly in the area of oppositional and conduct problems in children, have found that concurrent BPT with a problem-solving/social competence treatment for youth results in better outcomes that either intervention alone (Kazdin, Siegel, & Bass, 1992; Webster-Stratton & Hammond,

1997). Others have found that having a child group increases parental attendance to BPT (Jensen & Grimes, 2010). Additionally, utilizing concurrent child groups and/or simply ensuring that the child is available during BPT allows parents to have *in vivo* opportunities to learn and implement BPT skills, with support and feedback from therapists (Chacko et al., 2009). BPT interventions, such as PCIT, utilize parent–child dyads as the core format of BPT, ensuring that parents have an opportunity to practice skills "in the moment," therefore potentially allowing for greater mastery of the skills. Collectively, there are distinct benefits of different formats of BPT, and no one approach will likely work best. However, parent preference, therapist comfort and skills, and practical issues all need to be considered when considering which format(s) to utilize.

Focus on Behavior Management Only versus Developing Relationships and Behavior Management

BPT has long focused on relationship building as the initial focus of BPT (see PCIT and Incredible Years as exemplars). The rationale has been that parent–child relationships are often strained in families of youth with ADHD, particularly if youth have comorbid ODD or CD. As such, focusing on relationship building is necessary. In addition, positive parent–child relationships have been found to enhance the effect of discipline strategies (e.g., time-out from positive reinforcement; Walle, Hobbs, & Caldwell, 1984), which may be seen as less stressful to implement initially during BPT (Eienstadt, Eyberg, McNeil, Newcomb, & Funderburk, 1993) as this strategy does not require the parent to directly confront challenging behavior. Moreover, parental positive attention is viewed as a powerful consequence for children's behavior, so instructing parents to apply positive attention contingent upon specific behaviors is critical. In contrast, some BPT interventions (e.g., parent management training; Kazdin, 2005) focus directly on improving child behavior through more direct discipline strategies (e.g., response cost systems) in an effort to more intensely concentrate the often limited time and parental resources on directly addressing the child problems that caused parents to seek treatment. As an example, parents focus on improving child compliance, which is likely most immediately improved through discipline strategies (e.g., response-cost) rather than parental positive attention. Some research suggests that there are greater improvements in child behavior and greater satisfaction with treatment when discipline strategies are utilized first, likely given the more direct and immediate impact of discipline strategies on improving problematic behavior (Eienstadt et al., 1993) and, clinically, it may be necessary to provide parents with strategies that more quickly alter problematic behavior as a way to engage them in BPT over the long term. Coles, Bagner, Robb, Helseth, and Hartley (2012) presented preliminary data suggesting that use of discipline strategies (time-out from positive consequence, etc.) rather than positive strategies (praise, parent–child special time, etc.) results in greater BPT attendance, fewer dropouts, and greater satisfaction with BPT in a sample of youth with ADHD. Although one can reasonably argue that all parents can benefit from all components of BPT, and that we do not know the additive and interactive effects of positive and discipline strategies on BPT outcome, we believe the (limited) data suggest that a more thoughtful approach should be applied when considering the initial focus of BPT (positive vs. discipline strategies) for families. Although more definitive answers are unavailable, it is likely that for parents with relatively greater child difficulties or those in a positive parent–child relationship who utilize consistent positive strategies, beginning treatment that focuses on discipline strategies may be more palatable, engaging, and/or effective. For these types of families, it may be that starting treatment with more positive strategies may be perceived as insufficient (for children with relatively more severe problems) or unnecessary (for parents with strong positive relationships who utilize consistent positive strategies). In the case of parents who already use excessive discipline to the exclusion of positively rewarding skills, the opposite may be the case. These parents may benefit from positive parent–child relationship skill-building early in treatment in order to develop the necessary foundation for successful long-term outcomes in BPT. Clearly, more empirical data are required to understand this nuanced clinical issue.

Homework

Homework (HW) can be described as between-session exercises in which the client practices specific skills learned within the session in order to promote skills acquisition, which ultimately leads to improved acute and longer-term therapeutic benefits relative to targeted outcomes, generalization of treatment effects, and maintenance of treatment gains (Kazantzis, Deane, Ronan, & L'Abate, 2005). The limited data from BPT studies suggests that HW is an integral aspect of BPT—

attending and being actively involved in treatment is necessary, but insufficient in producing desired outcomes following treatment (Nock & Ferriter, 2005). Unfortunately, the limited data on HW in BPT for ADHD suggests that HW is often not completed, or when completed, it is of poor quality (e.g., Chacko et al., 2009; Fabiano et al., 2009).

Empirical efforts to improve HW completion (quantity and quality) have focused on a four-phase HW process model (Kazantzis et al., 2005): (1) Designing HW; (2) Assigning HW; (3) Doing HW; and (4) Reviewing HW. The DADR model posits that specific social, cognitive, and behavioral factors related to the HW task (e.g., perceived complexity of HW), provider (e.g., ability to address obstacles to HW completion), client (e.g., perceived self-efficacy/motivation to complete HW), and environment (e.g., environmental barriers and facilitators to completing HW) impact the quantity and quality of HW completed. As an example, if the HW task is designed by the therapist in a such a way as to be too complicated (e.g., requiring a parent to utilize a BPT skill for the first time throughout the day with multiple children and a range of settings), it is likely to result in low levels of HW completion and poor HW quality. These facilitators/barriers to the DADR model have been shown to be predictive of HW compliance (Kazantzis et al., 2005).

Our own work (Chacko, Anderson, Wymbs, & Wymbs, 2013) indicates that parents often report problems throughout the DADR process, which suggests that therapists should pay close attention to how they work with parents throughout each phase. Therapist should focus on how they develop HW tasks, keeping in mind the process of shaping parent behavior relative to HW (going from small, manageable but meaningful attempts to implement HW to more complex integration of BPT skills), requesting parent input and buy-in regarding HW task, eliciting information about barriers to HW implementation, and problem- solving those barriers. Additionally, systematic discussion of HW and problem solving of barriers to HW should be conducted during the review process. Importantly, we have found in our work that according to parent report, the "doing" phase is where the most challenges occur. As such, efforts should be made to support parents in remembering to do HW (e.g., using reminders of HW tasks on smartphones), to address stressors that impede upon successful HW completion (e.g., addressing time management difficulties), and challenges that interfere with effective recall of how to implement skills taught during BPT session within the family's natural environment (e.g., providing brief sticky notes of how to implement a skill). The challenge, however, is for the therapist to provide reasonable support during the "doing" process as therapist are not with the family during the time where HW is meant to be implemented. First, effectively managing challenges during the "doing" process requires anticipating challenges and addressing these during the BPT session. Second, utilizing methods to assist in recall (e.g., reminder notes), a parent–buddy system (when group BPT is being utilized) to provide peer support to enhance HW implementation, and brief phone check-ins to parents who likely may have more challenges with HW implementation may be effective. These appear to be relatively simple methods, but even these strategies do not result in optimal homework completion (Chacko et al., 2009, 2013). Interestingly, given the advancement of technology (see Jones et al., 2013), there may in the (not-so-distant) future be increasing opportunities to improve HW implementation during the "doing" process. For instance, Jones and colleagues (2014) describe the use of mobile health applications through smartphones that video-record parents' implementing HW at home. The recorded HW allows the parent and therapist to discuss challenges in doing the HW task during the subsequent BPT session, thereby allowing the therapist to provide specific feedback to the parent on the use of the BPT skill during the "doing" process. Additionally, features such as the global positioning system, which tracks the user's location, can be programmed to prompt parents to utilize specific skills (e.g., incentive systems) when approaching an identified challenging setting (e.g., grocery store) to use for a specific child target behavior (e.g., compliance). This type of system can support parents in effectively using the right BPT skill at the right time in the right setting for the right target behavior. Collectively, applications via smartphones can serve as a personal HW support system almost anywhere and at any time to support parents' implementation of BPT skills. Importantly, studies specifically focused on systematically understanding the HW process, and methods to improve this process, including mobile health applications via smartphones, are sorely needed to better the quality and quantity of HW completed during BPT.

Moderators of Treatment Response

Over the past several decades there has been interest in identifying moderators of response to BPT (Miller & Prinz, 1990), including BPT for ADHD more specifically (Chronis et al., 2004). Data suggest that factors

at the contextual level (e.g., socioeconomic disadvantage), family level (e.g., marital conflict), parental level (e.g., maternal depression, parental ADHD); and child level (e.g., severity of child behavior) moderate response and engagement to BPT. As such, there have also been concerted efforts to address these issues (Chronis et al., 2004), with a majority of studies focused on developing "enhanced" BPT interventions and a handful on approaches to addressing parental mental health issues through pharmacological approaches.

Collectively the data on enhanced BPT interventions suggest that these interventions on engagement to treatment are beneficial, but outcomes relative to traditional BPT for ADHD are only modestly beneficial (see Evans et al., in press, for a recent review). For example, data from two recent studies of enhanced BPT for youth with ADHD (Chacko et al., 2009; Chronis-Tuscano et al., 2013; Rajwan, Chacko, Wymbs, & Wymbs, in press) found statistically significant outcome benefits at the group level; however, both studies found limited incremental, clinically significant benefits of enhanced BPT relative to traditional BPT interventions. These data suggest exercise of some caution in simply utilizing interventions that target a known moderator of response to BPT. As the data attest, traditional BPT, when done well, can often be effective for families with notable risks. Similarly, psychopharmacological approaches to treating maternal ADHD have resulted in improvements in parental ADHD symptoms but more limited effects on parenting behavior and functional outcomes for youth with ADHD (see Chronis-Tuscano & Stein, 2012, for a review).

To date, the majority of empirical investigations on enhancements to BPT for disruptive behavior disorders (broadly defined as ADHD, ODD, and CD), as well as pharmacotherapy for maternal ADHD, has taken a variable-centered approach that emphasizes the impact of a specific risk factor (maternal depression, stress, marital discord, maternal ADHD, single-parent status, etc.) on engagement in and response to BPT, and, subsequently, how to address the risk factor during treatment. However, as observed by many clinicians, high-risk parents often present with multiple risk factors for poor response to BPT, not just one. As such, consideration as to which (among many) factors to address is a question that has yet to be explored. Moreover, although it is clear that multiple factors/processes often influence response to BPT, as we described earlier, these factors are imprecise predictors of treatment engagement and response. In other words, having one or more of these factors at the start of BPT does not *guarantee* that there will be poor response and engagement to BPT. As such, participating in BPT often results in clinically significant improvements for families, and enhancement to BPT and/or medication for parental ADHD may provide incremental benefit on outcomes for only a subset of families. In other words, despite having significant risk factors, traditional BPT may be sufficient for many families.

Given that multiple risk factors (which tend to co-occur) for poor engagement to and response from BPT often present in families of youth with ADHD, particularly when comorbid ODD or CD is present, and that no single risk factor is entirely predictive of engagement and response to BPT, how should these factors be addressed, if at all, in BPT? It may be that shared processes (e.g., emotion regulation; Maliken & Katz, 2013) across these risk factors (e.g., substance use, depression, marital distress, etc.) attenuate engagement and response to BPT and, as such, implementing an "enhanced" component to BPT that can be readily applied to address shared processes will allow for a more parsimonious approach than utilizing a different adjunctive treatment component for different risk factors. Some authors have considered emotion regulation as a core process related to various parental risk factors that moderate outcomes to BPT, and a focus on supporting emotion regulation in general within the context of BPT may be warranted (Maliken & Katz, 2013). Similarly, for parents with multiple contextual factors that also impede effective parenting (unstable housing and employment, child care difficulties, etc.), training in general problem-solving (e.g., D'Zurilla & Nezu, 2007) may often address a wide range of practical challenges (Chacko et al., 2009).

A separate but related issue arises if common processes are identified. Should they always be targeted and if so, is there a benefit to targeting these processes immediately? Our perspective is that this may not always be necessary. Rather, a response-to-treatment approach may help to guide timing of implementation of an adjunctive enhanced component to address risk processes, if and when they are found to interfere with BPT. This may maximize efficiency of treatment by providing adjunctive treatment to those parents who need it, when they need it, rather than applying adjunctive treatment to all families at the start of BPT when a risk factor is identified. Collectively, answers to these questions may lead to more direct methods to address how and when it is best to target these multiple risk factors. For instance,

perhaps there is an intervention enhancement modality that can flexibly address multiple risk factors/processes (e.g., transdiagnostic intervention for emotion regulation; Ellard, Fairholme, Boisseau, Farchione, & Barlow, 2010). Or perhaps a treatment algorithm approach can be used to determine how best and when to target various parent-level processes that interfere with BPT (cf. Weisz et al., 2012, for an algorithm approach to treating comorbid child psychiatric disorders). Given these issues, the implication for clinical practice is to assess for common moderators of BPT engagement and outcomes (e.g., maternal depression) and track these moderators alongside progress monitoring of targets for treatment outcome (discussed earlier). Assessing the corelationship between these moderator(s) and target outcomes will provide for the clinician a sense of whether the moderator is attenuating treatment response. As a simple example for illustrative purposes, if depressive symptoms and a target goal of BPT (e.g., compliance) are measured systematically over the course of BPT, assessing the relationship between change in depressive symptoms and compliance may offer insights into whether depressive symptoms are interfering with response to BPT. If this is found to be the case, intervening to improve depressive symptoms may be warranted. Clearly, more empirical data are needed to compare the value of this response-to-treatment approach to always intervening to address the moderating risk factor, when present, before the start of BPT.

Maintaining Treatment Gains

BPT is a time-limited intervention (lasting at most a few months), but ADHD is a chronic condition. This suggests that families of youth with ADHD will likely need more support following BPT. Therefore, it is essential for clinicians to set appropriate expectations for parents regarding the likely longer-term outcomes associated with BPT. In general, data suggests that longer term outcomes following BPT are limited (Lee et al., 2012), particularly for high-risk families (Leitjen, Raaijmakers, de Castro, & Matthys, 2013). Our own data suggest that attenuation of effects following BPT can occur as quickly as within 3 months (Chacko et al., 2009). We believe that there are several clinical implications of this data. First, clinicians should ascertain treatment response following implementation of BPT. This is particularly true of group-based BPT, in which there is a fixed termination session. We have found that even when parents have completed a full course of BPT, some of them have not responded (well enough). As such, if treatment targets have not been met, an ongoing evidence-based, behaviorally focused intervention should be applied. Moreover, even when target outcomes have been achieved, determination of the extent to which parent(s) have gained an appreciation for and can articulate a behavioral conceptualization of modifying a child's behavior is necessary. Some parents may focus on implementing specific behavioral techniques to address a specific behavior but never fully appreciate the behavioral framework and how one generates hypotheses regarding the function of a behavior that guides development of behavioral intervention plans (which we discuss further below). Often, asking parents about what strategies or advice they would give to another parent for a particular novel challenging behavior provides the therapist an appreciation of their conceptual understanding of the BPT material. A parent's understanding of the conceptual framework of BPT and hypothesis regarding the function of a behavior suggests true learning and therefore a greater likelihood for utilizing these skills over time in novel contexts with novel behaviors. Continued monitoring and ongoing support are often necessary even for parents who have attained identified outcomes for their child and have an appreciation for the behavioral conceptualization of treatment and hypothesis generation regarding the function of a behavior. Collectively, even when time-limited BPT is effective, parents need ongoing support to maximize outcomes for their child.

CORE COMPONENTS

There are many commercial products and programs for training parents of children with disruptive behaviors (e.g., Defiant Children; PCIT; Incredible Years; Parent Management Training). Although there are differences in the style and timing with which concepts are delivered across programs, the core components are generally similar, due to a shared theoretical foundation stemming from operant and social learning theories (Garland et al., 2008). In recent years, research efforts have focused on identifying the common elements among the evidence-based treatments in an attempt to enhance ease of training and use of these procedures, particularly in community settings that serve a large number of children with diverse presenting problems (Chorpita & Daleiden, 2009; Garland et al., 2008). There are several techniques used to identify common

elements, but all methods generally involve a review of evidence-based treatment manuals and a verification process in which the final list of core elements are approved by an expert panel of established clinicians (Chorpita & Daleiden, 2009; Garland et al., 2008). The "common elements" approach eases the dissemination of evidence-based treatments because it reduces the need for clinicians to receive training from a multitude of treatment manuals; rather, clinicians may devote time to learning procedures and processes that comprise the majority of the manuals (Garland et al., 2008).

Regarding BPT, Garland and colleagues (2008) completed a common elements analysis of the core components for child disruptive behavior disorders. Their analysis identified the core components of the treatment parameters, therapeutic alliance, therapeutic content, and core techniques for BPT. Regarding treatment parameters, the majority of BPT treatment manuals comprise at least 12, 1-hour sessions that are scheduled on a weekly basis. Regarding the therapeutic alliance, common elements include rapport building and collaborative goal setting. There are also several common content elements, which we describe in greater detail below, that are taught to families in BPT, regardless of the particular treatment program (e.g., PCIT; Triple P, etc.). In fact, these common content elements have been used in our own work in BPT for ADHD (Chacko et al., 2008, 2009; Fabiano et al., 2009). Taken together, the therapeutic content and techniques provide the framework for what is covered in BPT sessions. It is important to note that one of the strengths of the common elements approach is its flexibility; clinicians may select the appropriate "dose" of BPT and individualize treatment based on the presenting problems of the family through the selection of concepts covered in BPT, by changing the order in which the content is presented to the family, and by changing the depth of coverage of the concepts in BPT (Pfiffner & Kaiser, 2010). In addition, clinicians can individualize treatment through the use of supplemental topics. We strongly recommend that traditional BPT be supplemented with information that is specific to ADHD. Potential supplemental topics that are specific to ADHD are presented at the end of this chapter. It is important to state at the outset that the information we provide below is no substitute for reading the commercially available manuals on BPT and obtaining appropriate training and supervision before conducting BPT.

COMMON SESSION TOPICS

Psychoeducation

Psychoeducation is a core, evidence-based component in psychotherapy. In BPT, it is particularly critical to educate parents because children are largely dependent on their parents for care (and, subsequently, parents play a large role in treatments such as BPT) and childhood psychopathology impacts the whole family. Information regarding the disorder, prognosis, and treatments are typically covered. Moreover, general causes of behavior, with particular emphasis on the goodness of-fit between parenting styles and child temperament, family stressors, and an overview of the behavioral conceptual model (i.e., antecedents, behaviors, and consequences [ABC] model) are reviewed. Finally, it is critical to impart to parents that the ABC model must be understood within the context of the function of a behavior. As such, BPT techniques are most effective when developed with a hypothesis about the function of a behavior. As an example of the ABC model, a behavior (aggression) that occurs every time homework is assigned (antecedent) and results in removal of the homework assignment (consequence) likely has an escape-motivated function. In other words, the ABC model would suggest that the function of the behavior (aggression) is for the child to remove (i.e., escape) an aversive activity (homework). Further assessment into understanding this behavior in context may reveal that the duration of homework is too demanding for the child given their ability to remain focused. As such, certain BPT techniques (e.g., incentive system focused on completion of a small amount of homework) may be more effective than others (e.g., assigning a time-out in response to aggression) for this specific behavior. In general, we have found that a useful way to conceptualize the function of a specific behavior is the acronym MEATS, which stands for *m*edical (e.g., behavior functions due to a medical/biological issue such as headache or sleepiness), *e*scape (behavior functions that allow one to leave or avoid a nonpreferred or aversive context), *a*ttention (behavior functions that gain attention from peers or adults in the context), *t*angible (behavior functions that obtain a tangible item such as a toy), and *s*ensory (behavior functions that obtain sensory stimulation), motivating functions for a behavior. Helping parents appreciate the importance of gathering information on a specific behavior using the ABC model and generating hypotheses regarding the function of a behavior using the MEATS acronym helps parents

and therapist to consider which BPT techniques may be most helpful in addressing behavior.

In addition, a review of the specific content of BPT sessions is often helpful. When doing so, we have taken the approach of discussing BPT as a set of tools (i.e., specific BPT techniques) in a toolbox. In line with this, it is important for parents to know which tools are best to address a problem and that not every tool is necessary or appropriate depending on the problem being addressed. Conveying to parents that using all the tools in a systematic, thoughtful manner may result in achieving the immediate goals parents have for their child but that long-term outcomes are often maximized when all the tools in the toolbox are utilized consistently over time. We have found that providing details about the BPT techniques as a set of tools in a toolkit that help with both short-term treatment goals (e.g., improving compliance) but also in developing longer-term competencies in children (e.g., developing self-control) lays the groundwork for the remainder of BPT in that it helps set appropriate expectations for what any one BPT skill can affect, helps parents appreciate the need for learning the overall set of BPT skills, and helps parents appreciate the need for full engagement in BPT to understand how skills are meant to be integrated together.

Praise, Positive Attending, and Positive Parent–Child Quality Time

Parental praise and positive attending are consequence-focused reinforcement techniques that help parents increase the frequency of desired behavior from their child and improve the quality of the parent–child relationship. Importantly, parents and therapists will have identified target outcomes prior to starting BPT (see the earlier section on progress monitoring) which are often the focus for praise. Praise when desired behavior is observed is introduced as a positive reinforcement technique; in particular, the concept of "specific labeled praise," whereby parents specify the desired behavior to praise (e.g., "Good job focusing on your math homework" vs. "Good job"), as a means of providing children with a clear understanding of what behaviors are expected of them, as well as a concrete reinforcement for these behaviors. Specific, labeled praise should also occur immediately after the identified behavior, be genuine, and include nonverbal behavior (positive affect, hugs, "high-fives", etc.). Positive attending includes skills utilized by the parent (eye contact; listening attentively; reflecting upon the child's verbal and nonverbal behavior; supporting conversations, etc.) to further develop the parent–child relationship.

It is important that the concept of praising the "positive opposites" of negative behaviors also be presented during this time, in which parents are taught to attend to and praise desired behaviors (e.g., "Great job focusing on your homework!"), rather than provide negative attention for problem behaviors (e.g., "Stop being so distracted!"). Indeed, parents often inadvertently pay more attention to problem behavior than to positive behavior. Over time, this selective attention to problem behavior increases the frequency of problem behaviors and decreases the frequency of positive behaviors because children are unlikely to exhibit behavior that goes unnoticed. It should also be noted that training parents to identify the positive opposites of problem behavior increases the likelihood that they will be successful in the identification of appropriate target behaviors during future sessions devoted to the design of incentive programs for their child.

Parents are initially asked to practice praise and positive attending skills in the context of "quality time." An important aspect of BPT is the recognition that a strong, positive relationship between the parent and child is not only important for the well-being of children and parents but it also may serve as a foundation for which some BPT techniques (e.g., time-out from positive reinforcement) will be more effective. Children are more likely to cooperate when parents' requests are made within the context of a positive relationship. Parents' reliance on coercive, controlling, and negative parenting behaviors reduces the likelihood that children will comply with parental requests. The main idea is that quality time should be free of parental judgment, correction, or direction and comprise an activity that is of primary interest to the child. Clinicians may need to assist in the identification of potential activities and times in which parents can schedule quality time with their children. It is important to emphasize to parents (who are often overwhelmed with competing demands), that in some instances, quality time may include a short conversation on the drive to school or listening to one of their child's jokes during the bedtime routine. A small amount of focused quality time can go a long way for families.

Initially, parents are asked to practice praise and positive attending skills within the context of identified, child-centered "quality" times when their child is behaving appropriately, so that praise and positive at-

tending skills are strengthened over time and parents will be ready to use these skills during more challenging situations, for example, during less structured times or times when children are exhibiting a high rate of negative behaviors and a low rate of neutral or positive behaviors.

Planned Ignoring

A child's problem behaviors may be maintained by social attention. Therefore, it is important for parents to identify frequently occurring, mildly annoying behaviors (e.g., whining, complaining, disruptive noises) that serve the function of *attention seeking* (which is best determined through a functional assessment using the ABC model—see earlier section) and to systematically remove the reinforcing consequence (i.e., increased parental attention). Parents are instructed to ignore mildly annoying attention-seeking behaviors, and over time, the frequency of these behaviors will begin to decrease as the child recognizes that the behavior is no longer being reinforced via social attention. It is important to advise parents about the potential for extinction bursts, in which negative behaviors may *increase* initially as children, surprised by the lack of familiar parental attention, increase the intensity and frequency of their negative behaviors in an effort to elicit parental attention.

It is important to note that a critical component of this session includes a focus on parental emotion management and relaxation techniques that can be performed during the planned ignoring procedure (positive coping statements, leaving the immediate area of the child, etc.). This is especially important because *consistency* in ignoring is crucial; if a parent provides attention out of frustration with the child after using planned ignoring procedures for a period of time, the problematic child behavior will have been unintentionally reinforced at an increased intensity, which will likely increase the level of difficulty of future attempts at planned ignoring. The next time a parent attempts to respond to a problem behavior with planned ignoring procedures, the child may decide to increase the intensity of the problem behavior earlier in the interaction, in an attempt to gain parental attention.

It should be noted that planned ignoring is *not* appropriate for harmful, destructive, or aggressive behaviors, which should *not* be ignored. In our clinical experience, it is helpful to have parents and children practice planned ignoring procedures in the clinic, prior to the use of these strategies at home, because planned ignoring is frequently one of the most difficult techniques for parents to practice effectively at home.

Effective Commands

This topic addresses the antecedents aspect in the ABC model, in which parents can "set the stage" for maximizing the likelihood of child compliance. In this case, *how* a parent gives a command contributes to the outcome. Some important aspects when working with parents on utilizing effective commands include the following:

1. Commands should only been given when parents have the ability to follow through with consequences for noncompliance.

2. Parents should determine whether a command is necessary for the desired outcome. At times, parents truly want to offer an option for the child (e.g., "Would you like to help me with the . . . ?") rather than require child compliance. It is important to note that the more commands a parent gives, the greater the number instances of noncompliance—as such, commands should be used conservatively.

3. Commands should be given in statement rather than question form ("It is time to take a bath now" vs. "Do you want to take a bath now?") in a firm and assertive voice with accompanying nonverbal behavior.

4. It is important to obtain the child's attention prior to issuing a command. This is especially important for youth with ADHD because poor compliance may in part be secondary to poor attention. Parents should utilize proximity control procedures that ensure they are physically close to a child and have eye contact with the child during commands. For particularly inattentive children, it may be helpful for parents to request that the child repeat the command verbally. This strategy confirms that the child is attending to the parent and that he or she understands what the parent expects. This also provides an opportunity for the parent to immediately clarify/correct any misunderstandings.

5. Commands should be simple, clear, and comprise a single step. This point is particularly important for children with ADHD, who are not only inattentive and highly distractible but often also have executive dysfunction (e.g., working memory deficits, poor planning/disorganization).

6. Transitional warnings may be helpful because it may be more difficult to comply with abrupt commands (e.g., "In 5 minutes it is time to take a bath" or "When you finish X activity, it is time to take a bath") or to transition from a preferred (e.g., watching television) to a nonpreferred activity (e.g., taking a bath).

7. The use of "when–then" commands, in which desirable activities are used as natural reinforcers for activities that are less desirable (e.g., "After you do your homework, you can watch television") typically involves rewards that children would otherwise receive "noncontingently." It is especially important to avoid the opposite technique (e.g., "You can watch one more episode if you promise to do your homework afterward") because this type of request does not provide sufficient contingencies to affect the problematic behavior (poor HW compliance).

8. Allow some time for the child to process and to act upon the command. Unless immediate compliance is requested, it is important that the child has an opportunity to comply. If there is noncompliance in response to the first command, the command should be reissued. Typically, the reissued command is accompanied by the consequence for repeated noncompliance (e.g., "John, please turn off the radio and come to the dinner table or you will lose . . .").

9. Consequences for compliance (labeled praise) and for repeated noncompliance should always be instituted soon after the behavior occurs. This allows the child to appreciate the consequences of his or her behavioral choices. Negative consequences should be done in a matter-of-fact manner, and parents should anticipate that this may result in further negative behavior by the child, which often can be managed through planned ignoring. As in the case when implementing planned ignoring, parents should utilize skills to regulate their emotions in the face of negative child behavior.

Incentive Systems

Incentive systems, such as token economies, are a major component of BPT and are used as both an antecedent (setting clear behavioral expectations) and a consequence (providing tangible reinforcement) strategy. Incentive systems are additional positive reinforcement procedures that are used whenever extra motivation may be needed, beyond that of praise or positive attending. An incentive system is a means of providing concrete, meaningful, and immediate rewards for positive behaviors. Incentive programs provide a structured means of giving rewards in a way that is more meaningful and salient for children with ADHD, secondary to motivational impairments, such as preference toward immediate rewards due to delay aversion. Before initiating incentive systems, parents are required to clearly define target behaviors (if they have not done so already or if new challenging behaviors have emerged during the course of treatment), how to set realistic goals using concepts from shaping procedures and child development, and how to select appropriate primary reinforcers (e.g., games) that are feasible for parents to provide immediately after the positive behavior, or secondary reinforcers (e.g., tokens, points) that can be exchanged for primary reinforcers.

An important aspect of implementing incentive systems that is particularly relevant for a child with ADHD is reinforced practice of a specified behavior using an incentive system. Reinforced practice is a technique that can help a child to sharpen a developing skill, one that previously has not been consistently demonstrated (Kazdin, 2005). For example, some children may have difficulties separating from their parents for sleep. For children with ADHD, this may be because they have difficulty settling in for the night due to restless behaviors, but like any other child, it may also be because they have nighttime fears. A reinforced practice procedure would encourage parents and children to practice separating for sleep several times a day. Parents are specifically encouraged to select times of day in which everyone is in a good mood and the household is relatively free of stress (Kazdin, 2005). The clinician helps to develop the positive opposites of current problem behaviors, and children earn primary or secondary reinforcers in a reward system for practicing the following behaviors when prompted by their parents: (1) going to the bedroom within 10 seconds of the parental request, (2) changing into their pajamas in a timely fashion, and (3) lying down in their bed and staying there for 2–3 minutes as parents say good night and close the door behind them. When the child consistently demonstrates the positive behaviors, the rewards for practice are gradually faded and replaced with the opportunity to earn rewards for demonstrating these behaviors during the actual nighttime routine utilizing the incentive system (Kazdin, 2005). Reinforced practice using an incentive system is particularly important for behaviors that are accompanied by emotional dysregulation in children (e.g., a child being told "no" by a parent in response to a request). In these situations, it is not unusual for children to exhibit some level of emo-

tional dysregulation (e.g., crying, whining, etc.), but for children with ADHD, their emotional dysregulation may be more severe. As such, using reinforced practice (a child practicing appropriate reactions to an undesired response from a parent) allows the child to practice demonstrating the target behavior in a "emotion-free" context where it is likely that the behavior will be exhibited and reinforcement will be provided. This also allows a child to understand the positive consequence for exhibiting the desired behavior, which will likely increase the chance of the desired behavior occurring during the targeted context.

Past experiences are also discussed, and clinicians should attempt to identify the presence of maladaptive cognitions related to incentive systems. Given that incentive systems have been discussed in many nonacademic parenting magazines in a cursory way, parents are likely to have come across and (unsuccessfully) attempted to apply an incentive system. Some incentive systems also include a response cost component for particularly serious or resistant behaviors, in which a token is removed. For children with ADHD, we have found that the response cost procedures are most effective after several weeks of positive reinforcement only (response cost procedures work in the face of effective positive reinforcement, also known as "positives before negatives"). However it is important to avoid a "punishment spiral" and debt accumulation because these function to undermine the system and reduce child interest. Importantly, instructing parents on the use of incentive systems should be very detail-oriented and include modeling by the therapist, role play, and working with the parent and child to illustrate the implementation of the incentive system.

Time-Out from Positive Reinforcement

"Time-out" from positive reinforcement is one of the most commonly used consequence-focused strategies in BPT. Time-out should be utilized when other techniques, such as praise, positive attending, planned ignoring, and incentive systems are not successful, because time-out is often very difficult for parents to implement consistently. Importantly, time-out should only be used for behavior whose function is to obtain attention or a tangible item (e.g., aggression directed at another child to obtain a toy) or, arguably, when the child is too emotionally dysregulated to engage in productive verbal communication with parents. Time-out functions as a time away from reinforcement and also provides the space and time for a child to calm his or her physiological response to anger or upset. It is important to spend a great deal of time discussing past experiences with time-out, as well as the logistics and how to troubleshoot time-out (e.g., what to do if the child refuses to stay in place), because it is very difficult to implement time-out correctly, and many parents may have had failed attempts in the past. For instance, the operationally defined behaviors that (functionally) are best addressed by time-out should be identified, length and location of time-out should be determined, and procedure for assigning and ending time-out should be detailed, as well as procedures for managing escalating behavior during time-out. These discussions must be very detailed and include modeling by the therapist, role play, and working with parent and child to discuss the use of the time-out procedures.

Problem Solving

Problem solving is a nonspecific cognitive-behavioral therapy (CBT) tool that can be adapted to address a number of different issues. Typically, parents are requested to (1) identify a single problem on which to focus, (2) define the problem in a concise manner, (3) brainstorm all potential solutions for the problem, (4) consider all potential advantages and disadvantages of the potential solutions, (5) eliminate the identified solution with low likelihood of advantages, (6) consider possibly combining remaining individual solutions for a more effective solution, (7) decide on one solution to implement, and (8) evaluate the outcome. In the context of BPT, this exercise provides opportunities for parents to work through individual problems that were not necessarily addressed in the context of other sessions and that may be outside the scope of BPT but affect the quality of response to BPT (e.g., coparenting challenges). Importantly, we have found that this simple procedure is of great benefit to parents, a finding demonstrated in studies of BPT (Webster-Stratton, 1994). For some parents who may not have addressed problems in a structured way, as detailed above, discussion of how to problem-solve in a systematic manner is often very helpful.

SUPPLEMENTAL TOPICS SPECIFIC TO ADHD

In our clinical experience, there are a number of issues that are common in the families of children with

ADHD that require attention on the part of clinicians. For example, children with ADHD have been shown to exhibit more problematic behaviors in the context of their relationships with siblings (see Chapters 7). During the session devoted to incentive programs, it may be helpful for clinicians to present the concept of a shared sibling reward plan. Kazdin (2005) suggests that dueling siblings be paired together to earn a reward that is highly motivating for both siblings. This can be a shared reward, such as an outing to a favorite restaurant, or the siblings may select separate incentives from a rewards menu. The overarching principle is that the siblings must earn the rewards together by demonstrating the target behavior (e.g., using kind words with each other; playing gently with each other; and demonstrating the ability to compromise effectively with each other). If the siblings have not yet demonstrated that they have the ability to perform any of these target behaviors, they may initially earn points on their shared sibling reward plan by practicing these behaviors. It is recommended that these programs be coupled with the use of a timer to signal the interval in which points can be earned; the intervals can gradually be lengthened over time, as the siblings demonstrate success. Parents are encouraged to check on siblings at varying intervals and provide bonus points, particularly when the shared sibling program is first introduced.

The completion of routines is another common problem for children with ADHD. The morning routine is particularly problematic, as this is often a time of great stress for parents who must get all of the children, as well as themselves, ready at the same time. Additionally, homework routines are often challenging given the sustained attention required to complete what may be difficult academic assignments for children with ADHD. Importantly, early morning and late afternoon may be particularly difficult for parents because the effects of stimulant medications may not have taken effect or they are wearing off. This may not be the case for certain nonstimulant ADHD medications (see Chapter 27). As such, effective behavioral strategies to support behavior during these times of the day are particularly important.

In our clinics, we typically recommend that parents utilize a mix of interventions to improve the morning routine. First and foremost, parents are encouraged to utilize antecedent interventions to set the stage for success: Lay out school clothes the night before, pack lunches the night before, keep the child's backpack and shoes by the front door, and so forth. Second, we recommend the use of both a visual schedule and a timer to encourage effective, efficient behavior. Next, we work with parents to design a targeted reward program in which children earn tokens or points for completing small, well-defined steps in a timely fashion during the morning routine. For example, a child may earn one point for getting out of bed within 5 minutes of his or her wake-up time, another point for getting dressed, a point for brushing teeth, and a point for getting to the kitchen for breakfast within 15 minutes of the wake-up time. These points can be turned in for immediate morning rewards, such as choice of breakfast, watching 15 minutes of cartoons before catching the bus, or the using a preferred electronic device on the car ride to school.

The homework routine is another potential source of stress for parents of a child with ADHD. Power, Karustis, and Habboushe (2001) developed a helpful family–school intervention program to address these issues; although a review of the entire program is beyond the scope of this chapter, we present a basic review of some of the most important procedures. Parents, who are concerned with their child's academic futures frequently develop a management style in which they attempt to manage all aspects of the HW routine. A parent often sits next to the child, redirects behavior, threatens to take away privileges if the HW is not completed, checks over the HW afterward for accuracy, and commands that the child redo assignments that are incorrect or sloppy. Over time, learned helplessness, increased inefficiency, and even efforts to countercontrol (increased defiance, spitefulness) may develop in children with ADHD who are managed in this manner during the HW routine. The following steps may be necessary to improve the routine.

First, parents are encouraged to schedule the routine as early in the afternoon or evening as possible. This is particularly true if the child is taking stimulant medication and the goal is to complete the homework routine while the medication is active. An earlier homework routine is also preferable because it allows a child the opportunity to earn several rewards following the completion of the HW routine; it becomes difficult or impossible to provide rewards if the HW routine is spilling over into time that should be spent on other important activities (e.g., dinner or sleeping). Second, parents are encouraged to discuss the HW problem with the child's teacher and to work with the teacher to develop an upper limit on the amount of time the child should spend on the HW routine, with the understanding that

once the child has completed his or her time for the day, the remaining work will be returned to school incomplete. Third, a reward system, coupled with positive attending and planned ignoring for problem behaviors (daydreaming, fidgeting, whining, etc.) is designed with the family. In addition, parents are encouraged to limit their own management of the HW routine and are instructed to check in quietly and award points and nonverbal praise when they catch their child doing well (i.e., writing the HW answers, thinking about their assignments, etc.). Last, when the child has completed the teacher-approved amount of time to be spent on HW, he or she should be able to immediately turn in the points earned from their HW routine for privileges that are meaningful, such as playtime or access to electronics.

Impairing behavior that occurs in the social context is another frequent presenting issue for children with ADHD and their families (see Chapter 23 for specific strategies to help parents support social skills in their child). Parents often tell us that their child is no longer able to participate in sporting activities because the ADHD symptoms are so impairing in this setting. For example, parents often state that it is common for their child to talk over the coach's instructions, to not respect the personal boundaries of other children and place their hands on teammates during inappropriate times, to exhibit overly silly behavior characteristic of much younger children, and to seem never to be in the right place or follow rules of the sport. Parents can be instructed to set up a portable reward system in which children earn check marks in a parent's notebook for exhibiting the desired target behaviors: (1) eyes on the coach, (2) participating actively during drills, and (3) keeping one's hands to oneself. Parents should inform the child about the new reward system well in advance and work with the child to identify rewards that can be given immediately after. For example, the family may stop by their favorite ice-cream shop on the way home if the child has earned a predetermined number of points. In addition, parents are instructed to err toward being overly rewarding and positive in giving the child points when starting this program, in an effort to increase the child's motivation to participate in the program. A similar program can be developed for monitoring playtime with peers.

It is also important to teach parents how to advocate for their child in the school setting. School is frequently a major area of impairment for children with ADHD, and some children require modifications to their academic structure or accommodations in order to be successful. Any BPT program for a child with ADHD should cover the academic rights of children with ADHD, and parents should receive specific instruction for requesting an evaluation to determine eligibility for 504 Plans or an Individualized Education Plan. In addition, in our experience, parents often need a great deal of instruction and support around developing an effective plan to address the child's ADHD collaboratively with the school, once their child has been determined to be eligible for accommodations or modifications of the curriculum. These sessions are also excellent times to review the procedures for an effective Daily Report Card, which is one of the most effective tools for improving a variety of problem behaviors that occur in the classroom for a child with ADHD (see Chapter 24). Helpful resources for clinicians and parents are available on the website for National Initiative for Children's Healthcare Quality (NICHQ; the first edition of the ADHD toolkit was still available and free at the time of this writing (*www.nichq.org/areas_of_focus/adhd_topic.html*) and the website for the association of Children and Adults with Attention Deficit Hyperactivity Disorder (CHADD, *chadd.org or help4adhd.org*).

CONCLUSIONS AND FUTURE DIRECTIONS

BPT, which is arguably the most well-studied psychosocial intervention for ADHD (Evans et al., in press), is clearly an important and essential component of comprehensive care, particularly for younger children with ADHD, but it also has clear benefits for school-age children. Over the past decade, advances in enhancing BPT for difficult-to-engage and -treat families (Chacko et al., 2009; Chronis-Toscano et al., 2013; Fabiano et al., 2009) suggests that certain enhancements made to BPT (addressing practical barriers, parental attributions, etc.) can have a significant impact on improving engagement in treatment. Data are less convincing for the incremental benefits of these enhanced BPT interventions on outcomes relative to traditional BPT, demonstrating the robust effect of traditional BPT. However, even these trials suggest that both enhanced BPT and more traditional BPT do not normalize functioning for most youth with ADHD and their families, and that longer term outcomes are still problematic for youth upon completion of BPT. As such, future research should continue to attend to how best to target and provide support for parents enrolled in BPT who

experience challenges engaging in and responding to BPT. Developing a parsimonious approach to providing additional targeted support to parents is likely necessary in order to utilize the often limited resources and time of parents and therapists efficiently. Moreover, greater attention must be paid to how best to manage ADHD within the context of a chronic care model. We believe that BPT in general, and supporting parents of youth with ADHD more specifically, must be part of any model that aims to improve the longer-term functioning of youth with ADHD.

KEY CLINICAL POINTS

- ✓ As earlier chapters indicate, ADHD in children is associated with high levels of parent–child conflict and associated family distress, parental psychological maladjustment, and marital or cohabitation difficulties that can stem from or be correlates of high rates of child dysregulated behavior.
- ✓ BPT is among the three most evidence-based treatments for helping parents manage children with ADHD.
- ✓ BPT is often necessary for the majority of families with children with ADHD due to (1) the need for parents to learn more systematic and effective ways of coping with ADHD symptoms and associated disruptive behavior, and (2) the comorbidity of ODD with ADHD in 65% or more of all cases.
- ✓ ADHD can contribute to ODD and defiant behavior through its component of emotional dysregulation, but an additional contributor to such defiance is disrupted parenting. The development of ODD is then a harbinger of risk for later CD and internalizing disorders.
- ✓ BPT can help parents reduce ODD symptoms and, we hope, reduce these downstream risks from unmanaged ODD.
- ✓ The inclusion of BPT with medication management of ADHD can often help to boost the impact of medications, provide parents with a better understanding of ADHD and a set of skills for its management, improve parents' self-confidence, and better meet parents' expectations regarding the acceptability of a treatment plan than can medication-only approaches.
- ✓ Numerous BPT manuals and programs exist, but they share a core set of common principles and methods that derive largely from social learning theory (behavioral models) and may therefore be relatively equally effective in assisting families of children with ADHD and disruptive behavior.
- ✓ Effective BPT can be conceptualized as occurring in two phases: preparation and implementation. Key elements in the preparation phase of BPT are psychoeducation of parents about the nature of ADHD, identifying possible obstacles to engaging in the treatment program, and helping parents to reduce or surmount such obstacles. This phase also includes identifying key issues for parents, reducing them to their core problems, prioritizing these issues, then agreeing on and selecting issues for discussion in the current session.
- ✓ In the implementation phase, key factors to consider are program format (which BPT program to choose and whether to use group, individual, or mixed delivery); age of the child (success of BPT likely declines with age); HW; and identifying moderators of treatment response, such as low SES, degree of marital conflict, and child problem severity and comorbidity, among others. Parental psychological maladjustment may require enhancement of BPT with supplementary methods that help parents to address their own problems.
- ✓ Clinicians must also address the issue of how best to ensure program generalization (from clinic to home), such as through homework, in-session practice, parental discussion of anticipated obstacles to implementing the program that week, and so forth. They must also be concerned with how to help families sustain initial treatment gains that may otherwise attenuate within 3 months posttreatment (through booster sessions, periodic maintenance checkups, support through social media and smart technologies, etc.).
- ✓ The core components of all BPT programs appear to include (1) psychoeducation about applicable disorders (ADHD, ODD) and social learning theory views of child misbehavior, (2) reactive positive strategies to implement to enhance appropriate child conduct, (3) planned ignoring, (4) proactive positive strategies to enhance the likelihood that positive behavior will occur (token systems, behavior contracts, teaching when–then strategies, altering the style and format of parental commands, implementing transition planning, etc.), (5) reactive parental disciplinary tactics (e.g., response–cost and time-out methods), and (6) training parents in behavioral problem solving.
- ✓ Parents may also need guidance in advocating for informal and formal special educational services at the child's school.

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CHAPTER 22

Training Families of Adolescents with ADHD

Arthur L. Robin

Adolescence is a challenging developmental period for families because children are undergoing exponential physiological, cognitive, behavioral, and emotional changes. The typical problems of adolescence are magnified exponentially for the individual with attention-deficit/hyperactivity disorder (ADHD) and the family because the core symptoms, executive function deficits, associated features, and neurobiological deficits of ADHD interfere with successfully mastering the developmental tasks of adolescence (Barkley, 1997, 2012a, 2012b; Robin, 1998). As a result, teens with ADHD become embroiled in many unpleasant conflicts with their parents and suffer academic failure, social isolation, depression, and low self-esteem. They experience a lower quality of life than their peers without ADHD (Schei, Jozefiak, Novik, Lydersen, & Indredavik, in press; Topolski et al., 2004).

Parents encounter a variety of home management problems with their adolescents who have ADHD, including noncompliance with rules; conflicts over issues such as chores, homework, curfew, friends, and driving; Internet, video game, and cell phone use; and the teenager's "attitude." These conflicts often reflect independence-related themes; the adolescent desires more freedom to make his or her own decisions about chores, homework, and so forth, but the parents desire to retain decision-making authority. Such conflicts take the form of arguing, shouting, yelling, name-calling, and even physical fighting. Each family member attempts to get his or her way by coercion.

Consider the typical coercive interchange. Mrs. Smith commands Mike: "Stop texting your friends and start your homework." Mike stalls: "In a minute." Mrs. Smith's demand escalates: "Get off that phone this minute or else." Mike's annoyance escalates: "Don't bother me." Mrs. Smith says, "You are in big trouble now," and Mike retorts, "You never do anything to me anyway." Mother and son continue this escalating exchange until either Mrs. Smith grabs the cell phone from Mike or he locks himself in his room and his mother is forced to back off. If Mrs. Smith grabs the phone, her behavior is negatively reinforced by removal of the aversive stimulus of her son's argumentative, resistive behavior, and her son's argumentative behavior is punished; analogously, if she backs off, her son's argumentative behavior is negatively reinforced by the removal of his mother's coercive tactics, and her behavior is punished. Such interchanges are by definition coercive in nature. All families experience coercive interchanges, but they occur much more frequently and with greater intensity and negative impact in families with adolescents diagnosed with ADHD (Edwards, Barkley, Laneri, Fletcher, & Metevia, 2001). Clinicians need both a model to understand why coercive interactions often rise to the

level of clinical significance in families of teenagers with ADHD and a method of intervention to change them.

FOUR-FACTOR AND BEHAVIORAL FAMILY SYSTEMS MODELS

Barkley (2013; Barkley & Robin, 2014) has updated his classic four-factor model that explains how coercive interchanges reach clinically significant levels in families of teens with ADHD. This model is well grounded in a long tradition of social learning and child development research (Forgatch & Patterson, 1989; Patterson, 1982; Patterson & Forgatch, 1987). The four factors include the teen's characteristics, the parents' characteristics, the family environment and stressors, and parenting practices (Barkley & Robin, 2014). First, for adolescents, pertinent characteristics include (1) genetic influences; (2) the normal developmental changes of adolescence, including striving for autonomy from their parents and the resulting conflict; (3) diagnosed conditions such as ADHD, oppositional defiant disorder (ODD), conduct disorder (CD), or mood disorders; (4) extremes of temperament traits, such as strong willfulness, high reactivity, emotionality, and rigidity; (5) deficits in cognitive ability and executive functions (e.g., behavioral inhibition; poor task initiation; difficulty sustaining attention and effort; problems with working memory, organization, time management, and follow-through), and (6) any pertinent chronic medical conditions or physical disabilities such as diabetes, asthma, seizure disorders, spina bifida, and cerebral palsy.

Second, the same characteristics may apply to the parents, except for adolescent development. Parents are also more likely than their adolescents to be suffering from depression, anxiety, substance abuse, and personality disorders. In addition, it is important to highlight that parental ADHD is clearly an important factor. As the genetic basis for ADHD is becoming more widely known and accepted, therapists are diagnosing many parents of children with ADHD as having ADHD themselves. The stress of having two or more distractible, hot-tempered, impulsive, restless family members raises the probability of clinically significant conflict exponentially and decreases positive response to treatment.

Third, a variety of negative environmental–contextual characteristics and stresses may greatly escalate the chances for coercive interchanges. These may include maternal social isolation, single-parent status, financial hardship, marital discord, unemployment, housing problems, unsafe neighborhoods, legal problems, and so forth.

Fourth, negative parenting practices include disrupted parenting characterized by poor monitoring of the teen and ineffective, inconsistent, indiscriminant, lax, and/or timid child management techniques, often carried out in an harsh, openly hostile manner, further fueling escalation of coercive interchanges.

To this model we add poor problem solving and communication skills, distorted cognitions, and family structure problems for a full understanding of the home problems of families of teens with ADHD (Barkley & Robin, 2014; Robin & Foster, 1989). Given the way a normal adolescent strives for autonomy, parents need to learn when to negotiate solutions to disagreements using the steps of problem solving and when to impose rules for issues not subject to negotiation (e.g., drugs, alcohol, violence, abuse language). Teenagers also need to learn when and how to negotiate with their parents and when to accept parental rules. The verbal and nonverbal communication style of family members during family discussions is central to their success; family members need to learn to identify and change a variety of negative communication habits. By consistently modeling effective problem solving and solution-oriented communication, parents call forth reciprocal, positive problem-solving and communication behaviors from their adolescents.

In addition, beliefs, thoughts, and cognitions that parents and adolescents have about each other greatly impact the tone of the interactions. Extreme, distorted, negative thinking spurs angry affect and interferes with effective parenting and effective communication between parents and teens with ADHD. Family members must learn to identify and correct distorted beliefs, unreasonable expectations, and malicious attributions that elicit angry affect and sidetrack solution-oriented communication. A parent, for example, may fear the ruinous consequences of giving too much freedom to an adolescent; demand unflinching loyalty or obedience; or incorrectly attribute innocent adolescent behavior to malicious, purposeful motives. An adolescent may jump to the conclusion parental rules are intrinsically unfair and likely to ruin any chance of having fun in peer relations, and that teenagers should have as much autonomy in decision making as they desire. Such unrealistic cognitive reactions mediate emotional overreactions that spur continued conflict and impede

the use of effective parenting practices. Adolescent and parental cognitions may also be viewed as part of the first two factors in the four-factor model.

"Family structure problems" can be viewed as part of the third factor, contextual or stressful conditions that contribute to coercive interchanges. All families have a hierarchy or "pecking order," and in Western civilization parents are typically in charge of children. Adolescence is a transitional period when parents are supposed to be upgrading the children's status in the hierarchy, culminating in an egalitarian relationship between adult children and their parents. It is easy for a coercive child to overwhelm the parents, and by adolescence, such a child may have too much power in the family—a situation we call "hierarchy reversal." Sometimes one parent and the adolescent may also take sides against the other parent, forming a "cross-generational coalition." Two family members may place the third in the middle of a conflict, forcing the third to take sides. This pattern, called "triangulation," often occurs in adolescents who have ADHD.

For example, the father comes home to find that the mother and son have had a major battle earlier that afternoon. Mother and son both turn to the father, presenting their sides of the argument and appealing for support, and the father is triangulated or caught in the middle. Sometimes the father sides with his wife, other times with his son. Each of these structural problems may result in a "divide and conquer" situation, which dilutes parental teamwork and promotes the escalation of coercive interchanges to clinical proportions. An effective intervention must identify, analyze, and correct such problems of family structure. Although no comprehensive research has validated this entire model, researchers have examined the problem-solving–communication skills component, demonstrating that families of teens with ADHD plus ODD exhibit more specific disputes, negative communication, and aggressive tactics and hostile affect than families with typically developing adolescents (Edwards et al., 2001).

When families of teens with ADHD present to clinicians, they are usually trapped in a downward spiral of coercive interchanges, ineffective problem solving, negative communication, extreme thinking, and problematic coalitions and triangulation. They display extreme temperament characteristics, multiple family stresses, and inconsistent and ineffective parenting. They do not understand why they are stuck or how these factors have contributed to their current state of misery. It is the job of clinicians to educate families about this model, give them hope that change can happen, and translate each of the components of the model into specific, evidence-based interventions. I turn now to that task.

INTERVENTION

Overview of the Steps of Intervention

Table 22.1 outlines the 11 steps of this intervention. Sometimes a step can be accomplished in a single session; other times, it may take several sessions. The clinician should follow the order of these steps, since later steps build on the family's successful completion of the earlier steps. During the first five steps, therapy sessions are held weekly; during the remaining steps, therapy sessions are held every other week, giving the family sufficient time to implement interventions at home. The steps include most of the elements of the *Defiant Teens* manual (Barkley & Robin, 2014; Barkley, Robin, & Benton, 2013), with some modifications based on a decade of clinical experience. Departures from the manualized approach are noted throughout the chapter. Two significant departures from the manual are noted here. First, for Steps 1 through 4, I advise the therapist to have the parents attend approximately every other session without their teenager, and for Steps 5 through 11 have the teenager join them for each session. In the manual, parents attend the first nine sessions without the teenager, and the teenager attends the next nine sessions with the parents. I have

TABLE 22.1. Steps of Intervention

Step 1.	Educating Families I: ADHD, Coercive Interchanges, Four-Factor Model
Step 2.	Educating Families II: Parenting Principles
Step 3.	Fostering Realistic Beliefs and Expectations
Step 4.	Preparing Families for Medication
Step 5.	Breaking the Negativity Cycle: One-on-One Time
Step 6.	Praise, Ignoring, Commands
Step 7.	Implementing Positive Incentive Systems
Step 8.	Implementing Punishment Systems
Step 9.	Problem Solving Negotiable Issues
Step 10.	Improving Communication
Step 11.	Putting It All Together

found that rapport with the adolescent is diminished if he or she is not included in the first part of the intervention, and that by being included the adolescent is more cooperative with the contingency management portion of the program.

When completing each step, the clinician first meets with the parents to explain the tasks they will be asked to do with their adolescent and to address their questions; then, the parents implement this step and return to the next session with their adolescent. During this session, the adolescent's reactions to the parental intervention are discussed, successful outcomes are praised, and any difficulties that the family encountered are problem-solved. Second, in this chapter, I do not cover the step on school advocacy step from the original manual because other chapters in this book address the school problems of teenagers with ADHD.

Readers need to understand that the modifications suggested here have not been subjected to empirical scrutiny. The research described later in the chapter is based strictly on the original program (Barkley & Robin, 2014).

Families Who Are Appropriate for this Intervention

This intervention was designed for 12- to 18-year-old adolescents and their parents with average intellectual and language abilities, externalizing behavior problems, ADHD, ODD, and adjustment disorder or CD, provided that defiant behavior and/or conflict between the adolescent and the parents is the major presenting problem. It is not designed for physically aggressive and assaultive adolescents and/or parents, or those with severe psychiatric problems such as schizophrenia or personality disorders (parents). Such aggressive and assaultive adolescents are better treated through in-home multisystemic therapy (Henggeler, Schoenwald, Borduin, Rowland, & Cunningham, 1998), day treatment settings, residential treatment facilities, inpatient psychiatric units, or intensive outpatient treatment programs (Sibley, Smith, Evans, Pelham, & Gnagy, 2012).

This intervention has been successful with single- and two-parent families, and families in many ethnic/cultural backgrounds and socioeconomic levels. Clinical experience suggests that the effectiveness of the program is reduced when divorcing parents who exhibit severe, open hostility toward each other are unable to work as a team in consistent administration of the strategies taught to them in the therapy sessions.

Step 1. Educating Families I: ADHD, Coercive Interchanges, Four-Factor Model

During the first half of the session, the therapist defines and explains ADHD to parents as a disorder of executive function with a genetic and neurobiological basis (Barkley, 2012a, 2012b). Then, the therapist explains the coercive interchange, the four-factor model, and the problem-solving communication model, using the material presented earlier in this chapter but applying it to examples unique to each family. Usually, this portion of the session goes smoothly and culminates in the therapist indicating that if the parents change their parenting practices, then they may see some positive changes in their adolescents, even if the changes take time and are not monumental in nature.

In the second half of the session, the therapist meets individually with the adolescent to (1) present the facts about ADHD and state the treatment options, (2) listen to the adolescent's reactions to the presentation of the facts and the treatment options, and (3) apply cognitive restructuring to correct myths and instill positive attitudes toward ADHD treatment.

Giving the Facts

The therapist begins by making a clear statement that ADHD applies to the adolescent, giving a brief definition of ADHD, discussing its neurobiological/genetic etiology, and highlighting how it impairs the quality of the adolescent's life in practical ways to which the teenager can relate. He or she uses simple sentences that the teenager can understand, incorporates information the teenager has previously provided, and pauses often to check for understanding and answer questions. If the adolescent does not spontaneously bring up the most common myths about ADHD and its treatment, then the therapist brings them up and debunks them.

The following points, briefly illustrated in the language the therapist uses with teenagers, need to be covered throughout this presentation, although not necessarily in the order given here:

1. "ADHD is a disorder that involves difficulty paying attention, organizing and following through, acting before thinking, and sometimes feeling or acting restless." For bright adolescents, the therapist includes a discussion of executive functions.
2. "You are not crazy or sick if you have ADHD. It

is an invisible disability that represents the extremes of traits or characteristics that all people exhibit to a greater or lesser degree."

3. "ADHD usually lasts a lifetime, but it changes as you mature and grow older. In particular, the restlessness changes from more physical to more mental, but the inattention, disorganization, and impulsivity remain."
4. "ADHD affects all areas of your life, not just school. It influences driving, getting along with people, romantic relationships, sleep, eating, sports, self-esteem, and your future jobs."
5. "ADHD is not your fault, your parents' fault, or anyone's fault. It is a physical disorder, usually inherited, and it is caused by a difference in brain chemistry."
6. "Chemicals called 'neurotransmitting chemicals,' which pass signals for self-control throughout the brain, aren't operating efficiently in people with ADHD. It would be like having too little brake fluid in your car; when you press the brake pedal, you can't stop. When an idea to do something pops into your mind, you can't stop and think whether it is good or bad before you do it because the chemicals that help the brain stop and think aren't working properly."
7. "Because the disorder is usually inherited, it is possible that your parents, brothers or sisters, or other relatives also have ADHD, even if they don't know it. This could make family life like a real roller coaster!"
8. "ADHD is also influenced by your environment—for example, your parents, your school, and your friends. A good family, a good school, and good friends can make life a lot easier for the person with ADHD."
9. "ADHD is a challenge, not an excuse. You are still responsible for your actions, even though you have a physical disorder that makes it harder for you to control your actions."
10. "ADHD is influenced by your physical health. It will be easier to deal with ADHD if you take proper care of yourself—for example, get enough sleep, maintain good nutrition, don't smoke or put drugs or alcohol in your body, and exercise regularly."
11. "Because ADHD is inherited and physical, we can't totally cure or eliminate it. Instead, we can help you learn to cope so that life goes well for you. There are three general methods for learning to cope: (a) medical, (b) behavioral/psychological, and (c) educational. We will talk about these in detail as time goes on."

Listening to the Adolescent's Reactions to the Presentation

After presenting the facts, the therapist listens carefully to the adolescent's reactions—using active listening to clarify how the adolescent is feeling, but not challenging or being confrontational with him or her. It is very important for the adolescent to feel that he or she has been listened to and understood, and that his or her opinions have been taken seriously, because in the past, his or her ideas may have been discounted by adults. Let us look at an example of 15-year-old Bill, voicing his concerns about the diagnosis of ADHD:

BILL: So if I have ADHD, does this mean I am dumb and have a bad brain?

DR. ROBIN: You're feeling like having ADHD means you're stupid.

BILL: All the retards on the special education bus have ADHD. The whole football team whips their butts at gym.

DR. ROBIN: You feel like a retard, and you think your friends on the football team would give you a hard time about having ADHD.

BILL: Yeah, this is the kiss of death for me. My parents are going to freak out and take me to a million doctors, tutors, and shrinks. I'll probably miss football practice and get kicked off the team! And they will make me take medicine that will make me weird.

DR. ROBIN: So ADHD is going to mess up your whole life, take away all your free time and fun, and make you into a zombie.

BILL: Yeah, and just when Jennifer was starting to like me, too. Now Mike will get her for sure.

DR. ROBIN: You will also strike out with girls. This all sounds like a nightmare.

BILL: Yeah.

As Bill voices his fears and anxieties about peer ridicule, feeling stupid, having to go to a lot of doctors, getting kicked off the football team, losing his freedom, and never having a girlfriend, I empathetically clarify them but do not yet deal with them. Many adolescents

may be thinking what Bill has verbalized, but it may take several sessions before they become comfortable confiding with the therapist about their worries,—although an advantage of their being impulsive is that they often blurt out their worries, despite their desire to hide them.

Dealing with Reactions: Application of Cognitive Restructuring

We apply cognitive restructuring (Kendall, 2011) to dealing with adolescents' negative reactions to the ADHD diagnosis. Common distorted beliefs include the following:

"ADHD is a life sentence; my life is over. I'll never amount to anything."

"This means I am really dumb, stupid, crazy, or a bad person. All the bad things my parents and teachers have said about me are really true."

"I'll never have any friends; they will all think I'm a total nerd."

"I'll never have any fun because I will have to spend all my time with tutors, doctors, and therapists."

"Medication will change my personality. I like being wild, loud, and crazy. This is me, who I am, and no one is going to change me."

"I'm different from my friends, and I'll never be normal."

"I've really messed up now. It's all my fault."

"This whole ADHD thing is bull; it's just one more way my parents are trying to control my life."

These beliefs are really variations on three underlying extreme belief themes to which adolescents commonly adhere (Robin, 1998; Robin & Foster, 1989): (1) ruination ("This ADHD diagnosis is going to ruin my life, fun, and friends"); (2) autonomy ("Having ADHD will take away or limit my freedom"); and (3) unfairness ("It is so unfair that I am different from others and have to take medicine, get help in school, or see counselors").

In cognitive restructuring with an adolescent, the therapist tactfully collaborates with the patient to (1) identify the distorted belief, (2) provide a logical challenge to it, (3) suggest a more reasonable belief, and (4) help the patient discover through collection of evidence that the reasonable belief is more valid than the unreasonable belief (Barkley & Robin, 2014; Robin & Foster, 1989). Let us see how cognitive restructuring might proceed with Bill:

DR. ROBIN: I understand how you feel that ADHD will mess up your whole life, but before we jump to any quick conclusions, let's look at the evidence.

BILL: What evidence? I'm done, finished, all washed up!

DR. ROBIN: Let's start with the idea that you are dumb. On the IQ test I just gave you, you received a score of 115, which is above average. You may feel like you are dumb, but in fact you are smart. ADHD has nothing to do with being smart or dumb.

BILL: If I'm so smart, why do I do dumb things like spray paint on the garage?

DR. ROBIN: Good question. Your brain is like an expensive sports car without any brake fluid. We all get crazy ideas popping into our minds. People without ADHD press the brake pedal and it works; they don't act on their crazy ideas. People with ADHD press the brake pedal and nothing happens. They just keep on acting. This has nothing to do with IQ. You have a high IQ, just as the sports car has a great engine. But without brake fluid, the car won't stop, no matter how good the engine is. Now let's take your worry about having to go to the office to take pills and your friends teasing you. First of all, not everyone with ADHD takes medication. You would only take medication if you agree to, after you fully understand it. But let's say you did agree. We now have medicines that you take once in the morning and they last all day, so the only way your friends will know about it is if you tell them.

BILL: Great! Those drugs would make me into a weird zombie all day, then.

DR. ROBIN: The truth is, most people don't feel any different on medicine for ADHD, except they are not as hungry while it is in their bodies. Are any of your good friends on medicine for ADHD?

BILL: You wouldn't catch me hanging out with those retards.

DR. ROBIN: Do you know a kid named Danny Jones?

BILL: Danny Jones? Sure. The whole school knows him. He is captain of the football team, Mr. Cool. Every girl in school goes nuts over him. But he is really a great guy.

DR. ROBIN: He has ADHD and takes medicine for it every morning.

BILL: No way. Not Danny. He's too cool. Doc, you're kidding, right?

DR. ROBIN: Nope. Don't take my word for it. Ask him. He is glad to talk about it privately, and he gave me permission to tell other teens with ADHD about it, but he has no reason to announce it on the overhead speaker system in school. And don't forget to ask him whether medicine makes him feel weird.

The steps of cognitive restructuring flow together in this case example. The discussion of the IQ test illustrates challenging a distorted belief with the introduction of a more reasonable alternative and clear-cut evidence to back it up. The introduction of the highly regarded, positive peer model who happens to have ADHD is the most potent type of evidence for changing beliefs about ADHD in teenagers because peers are such an important part of their lives. It behooves clinicians who work with adolescents to develop a referral list of such positive peer models with ADHD in the local areas in which they work. Of course, the peers must consent in writing to have their names released. Clinical experience strongly suggests that adolescents will be more convinced to accept and cope with ADHD by their peers than by adults. The clinician can suggest books and DVDs designed to educate adolescents about ADHD and foster coping attitudes to treatment (Dendy & Zeigler, 2007, 2011).

Step 2. Educating Families II: Parenting Principle

In Step 2, the therapist presents to the parents alone the principles for parenting an adolescent with ADHD. Step 2 typically takes place in a single session. Table 22.2 summarizes these principles. The therapist tells the parents that these principles do not always work, but they are based on sound behavioral research. It is helpful to derive a course of action from one of these principles when a parent does not know how to respond to a problem situation with an adolescent who has ADHD. The therapist presents each of these principles to parents and engages them in a discussion of how the principle might be relevant to their family.

TABLE 22.2. Principles for Parenting the Adolescent with ADHD

1. Shift your parenting style away from authoritarian control or permissiveness and more in a democratic direction.
2. Divide the world of issues into those than can be negotiated and those that cannot.
3. Give explanations for the stated rules regarding the nonnegotiable issues.
4. Give the adolescent more immediate feedback and consequences.
5. Give the adolescent more frequent feedback.
6. Use more powerful consequences.
7. Use incentives before punishments.
8. Strive for consistency.
9. Act, don't yak.
10. Plan ahead for problem situations.
11. Actively encourage and shape responsible independence-related behavior.
12. Involve the adolescent in decision making regarding negotiable issues.
13. Maintain good communication.
14. Actively monitor the adolescent's behavior outside the home.
15. Maintain structure and supervision longer than you think you should.
16. Be the adolescent's cheerleading squad.
17. Encourage the adolescent to build on his or her strengths.
18. Keep a disability perspective.
19. Don't personalize the adolescent's problem or disorders.
20. Practice forgiveness.

1. *Shift your parenting style away from authoritarian control or permissiveness, and more in a democratic direction to foster responsible independence-seeking behavior.* The extremes of authoritarian control or permissiveness are not effective with adolescents. Parents do not have the power and cannot possibly control all of their adolescent's behavior. At the other extreme, failure to exercise sufficient authority leads to the adolescent's experimenting with dangerous behavior.

2. *Divide the world of issues into those that can be negotiated and those that cannot.* There is an important

distinction between issues that can be handled democratically and those that cannot. This is our basic framework for disciplining adolescents. Each parent has a small set of bottom-line issues that relate to basic rules for living in civilized society—values, morality, and legality—that are not subject to negotiation. Such issues usually include drug and alcohol use, aspects of sexuality, school attendance, religion, and perhaps several others. These are the non-negotiable issues. The remainder of issues can be negotiated between parents and their adolescent. Each parent needs to list and present clearly to the teenager those issues that are non-negotiable.

3. *Give explanations for the stated rules regarding non-negotiable issues*. Adolescents are more likely to accept non-negotiable rules if they are legitimized with a compelling rationale, rather than presented through pure power assertion ("Do it because I'm your mother" or "Do it because I told you to"). Parents show respect for the adolescent's emerging identity as an independent being by taking the time to give him or her reasons for decisions.

4. *Give the adolescent more immediate feedback and consequences*. Adolescents with short attention spans and impaired behavioral inhibition are more likely to stay on task when given immediate positive feedback contingent upon performance of boring and tedious tasks, coupled with mild negative consequences for shifting off task. Punishments given long after the misbehavior is committed are ineffective.

5. *Give the adolescent more frequent feedback*. Adolescents with ADHD benefit from frequently hearing nice things said about their actions and appearance, as well as frequent feedback and corrections for their errors. Because so many factors in the life of the average adolescent with ADHD reduce his or her self-esteem, the adolescent desperately needs to hear frequently what he or she did right.

6. *Use more powerful consequences*. Because those with ADHD satiate easily on any one stimulus and respond best to highly salient stimuli, effective parenting involves using a wide variety of highly salient consequences, ranging from physical affection to verbal praise to material reinforcers.

7. *Use incentives before punishments*. Parents commonly load on immense punishments until they have used up all their ammunition and the adolescent has little else to lose by misbehaving. When parents wish to modify a behavior, the therapist needs to train them to ask first what positive behavior they wish to see the adolescent perform, and next how they can reinforce that positive behavior. Only after taking this step should they select a punishment for the negative behavior.

8. *Strive for consistency*. Parents of adolescents with ADHD often give up easily on behavior change interventions at the first sign of failure. These adolescents incessantly bicker with their parents, sometimes wearing them down to the point that the parents back off. The therapist needs to help parents to stick with their interventions and demands (i.e., to maintain consistency over time and resist coercion by their adolescent).

9. *Act, don't yak*. Many parents repeat themselves incessantly when their adolescents fail to comply with their requests. Adolescents quickly learn that Mom, Dad, or both are "all talk, no action." The therapist needs to help parents learn that the time to talk is during family meetings and when negotiating solutions to disagreements, but after the rules have been stated and the consequences decided, it is the time to act, not yak.

10. *Plan ahead for problem situations*. Because many conflicts between parents and adolescents are highly predictable, it behooves therapists to help parents learn to anticipate and plan in advance to handle these situations.

11. *Actively encourage and shape responsible independence-related behavior*. Because becoming independent from the family is the primary developmental task of adolescence, and because individuals with ADHD need extra guidance and learning trials to acquire new behaviors, parents need to look for opportunities to gradually give their adolescent more freedom in return for demonstrating responsibility. A parent might break the terminal independence response into small units and shape each behavior, moving on to the next step after the teenager has demonstrated responsibility on the previous step.

12. *Involve the adolescent in decision making regarding negotiable issues*. Teenagers are more likely to comply with rules and regulations they have helped to create. Furthermore, they may have novel and creative perspectives on issues because of their youth and unique position in the family. Often, their perspectives lead them to suggest novel solutions. I outline later in the chapter how parents can problem-solve negotiable issues with their adolescents.

13. *Maintain good communication*. Parents need to make themselves available to listen when their adolescents wish to talk but not expect their adolescents to confide regularly in them. Parents and adolescents need to learn effective skills for listening to each other and expressing their ideas and feelings assertively, but without putting down or hurting each other.

14. *Actively monitor an adolescent's behavior outside the home*. Parents should always know the answer to four basic questions:

Who is your adolescent with?

Where are they?

What are they doing?

When will they be home?

Research has shown that parents who cannot consistently answer these four questions have adolescents who are at risk for drifting into deviant peer groups, substance abuse, and delinquency (Patterson & Forgatch, 1987).

15. *Maintain structure and supervision longer than you think you should*. Parents often ask when they can relax the increased structure they have created to monitor their adolescent's academic performance and home behavior. Individuals with ADHD need to be more closely monitored for their entire lives, but we expect them to learn to do some of their own monitoring and/or to enlist the help of spouses or significant others in monitoring their actions by adulthood.

16. *Be the adolescent's cheerleading squad*. Adolescents with ADHD need unconditional positive regard from their parents and focused positive time with their parents. Follow-up studies (Weiss & Hechtman, 1993) indicate that successful adults with ADHD state that the single most important thing during their adolescence was having at least one parent (or, in some cases, an adult outside the family) who truly believed in their ability to succeed. Adolescents with ADHD need their parents to believe in them, to applaud their every positive achievement, and generally to be their cheerleading squad.

17. *Encourage the adolescent to build on his or her strengths*. Many adolescents with ADHD receive so much criticism they actually begin to believe that they are lazy and unmotivated. Therapists need to teach parents to help these teenagers identify those interests, hobbies, artistic pursuits, sports, and activities that are pockets of strength, and help them pursue and succeed at these pursuits to build on their strengths.

18. *Keep a disability perspective*. This principle has to do with expectations and beliefs, which I consider later. Briefly, therapists need to help parents remember that their adolescents with ADHD have a neurobiologically based disability, and that there is a "can't do" as well as a "won't do" component to their unthinking actions. Thus, parents can keep from overreacting with anger when their adolescents inevitably make mistakes.

19. *Don't personalize the adolescent's problems or disorder*. Closely aligned to the preceding principle, this principle is designed to help parents keep from blaming themselves or losing their personal sense of self-worth because of their adolescent's problems.

20. *Practice forgiveness*. Parents need to forgive themselves for the mistakes they will inevitably make raising an adolescent with ADHD, and to forgive their adolescent for his or her mistakes. Adolescents should, however, be held accountable for their actions, and consequences should be administered as planned, but parents should not "hold a grudge" afterward.

At the end of this session, the therapist explains that when applying specific interventions, he or she will reference these principles. He or she also refers parents to books that will be of assistance (Dendy, 2006).

Step 3. Fostering Realistic Beliefs and Expectations

Step 3 usually comprises two sessions: (1) parents alone, and (2) adolescent alone. At the beginning of the parental session, the therapist reviews the list of non-negotiable rules that the parents have brought in and gives them feedback. Then the therapist focuses on the beliefs, expectations, and attitudes of the parents and the adolescent. This focus continues to be interwoven with other material throughout the remainder of the therapy. The parents are given a "crash course" in the basics of adolescent development to help them foster realistic expectations. From a cognitive restructuring point of view, the crash course also represents a "normalizing," or reframing with a positive intent, of much of the negative behavior adolescents inevitably emit. By presenting this information within the context of adolescent development, the therapist makes it easier for the parents to accept it without activating any natural defensive reactions they might otherwise have.

The therapist is helping the parents learn to apply Principle 19 ("Don't personalize the adolescent's problems or disorder") by distancing from the constant barrage of strange teenage behavior, understanding it within a developmental framework, and learning to prioritize what to respond to and what to ignore. The sensitive therapist will be cognizant of this attitudinal portion of the agenda and monitor the parents' level of defensiveness and reactivity during the crash course, pacing his or her statements to shape their responses in productive directions.

The therapist reviews the five developmental tasks of adolescence: (1) individuate from parents, (2) adjust to sexual maturation, (3) develop deeper peer relationships, (4) form a self-identify, and (5) plan for a career. Then, the therapist points out that becoming a productive, happy, and personally fulfilled adult depends on successful accomplishment of these tasks. The adolescent is supposed to accomplish these tasks while getting along with the family and doing his or her schoolwork. The therapist helps the parents to realize that the adolescent has a great deal of work to do.

The nature of independence seeking or individuation from parents is explored in more depth. I find it useful to present the metaphor of a nation establishing its independence:

> "Imagine a nation establishing its independence, going from a dictatorship to a democracy. What often happens? This process does not typically go smoothly. There may be a bloody revolution with a great deal of fighting. Or if there isn't physical fighting, there are certainly a lot of power plays and verbal rhetoric. Why should you expect your family to make it through the independence seeking of your adolescent without a disturbance of the peace? A certain amount of conflict is inevitable and even healthy. I worry more about adolescents who never do anything rebellious than I do about those who do rebel. This rebellion typically happens in early adolescence, between ages 12 and 14. In order to become independent, teenagers need to push against something, and parents are the something that they push against. Typically, teenagers rebel more strongly against their mothers than their fathers. Wise parents learn how to channel their conflicts into more innocuous areas that have no ultimate impact on life. It is much better, for example, to have conflicts with your adolescent over how clean the room is rather than over sexuality and drugs."

We go on to help parents understand that it is natural for adolescents to reject established parental and other adult societal values during this process of individuation, and to be embarrassed about being seen with their parents. To begin to establish their own identity, adolescents need to experiment with a variety of alternative ideas and values, usually those of their peers, and decide what makes them comfortable. At the same time this is happening, their bodies are changing very rapidly, and their minds are maturing to the point that they now can think more abstractly. The multiple influences of rapid physical maturation, cognitive development, and emotional change are very unsettling to adolescents, leading them to have a fragile self-image. One response to this fragile self-image is to project an air of omnipotence or, put another way, to shy away from anything or anyone who suggests that they are less than perfect physically or mentally. Thus, it is natural for a developing adolescent to be less than enthusiastic about disabilities, psychiatric diagnoses, chronic physical illnesses, or any other condition that may be seen as a further insult to an already fragile self-image. Therapists help parents to understand that this is the basis for resistance to accepting the diagnosis of ADHD and its treatments.

The therapist then turns to the question of how ADHD interacts with these natural developmental tendencies during adolescence. Adolescents with ADHD undergo the same physical changes and face the same developmental challenges as other teenagers. They experience the same desires for independence and freedom as other teenagers. Yet their social and emotional maturity may lag behind that of other teenagers. They may be less ready to assume the responsibilities that accompany more independence.

Specifically, teenagers with ADHD may lag behind other teenagers in the overall development of self-control and organization (Barkley, 2012a). Because of inefficient operation of their executive functions, they may be less able to exercise hindsight, forethought, and planning, or to engage in future- and goal-oriented behavior. Given the additional difficulties with self-regulation of affect (see Chapter 3), they may remain more likely to be victims of the moment—acting on impulse, self-centered, and insensitive to the needs of others. Poor attention and follow-through make it more difficult for them to stick to discussions and carry out agreements with their parents and finish homework. Impulsivity translates into increased moodiness, hypersensitivity to criticism, emotional over reactivity, poor

judgment, and low resistance to temptations. Hyperactivity often continues more as minor motor restlessness and mental restlessness than as overt physical overractivity. Such restless behavior is easily misinterpreted as "disrespect" by parents. Repeatedly badgering parents to get their way is another manifestation of hyperactivity in some adolescents with ADHD.

The ADHD symptoms become inextricably intertwined with the developmental changes of adolescence. Many parents ask the therapist whether a particular adolescent behavior is a result of ADHD or "just adolescence." They may be wondering whether to excuse or to punish the behavior. Did Stephanie really "forget" to put away the dishes, or was she just being "oppositional"? The answer usually is that the behavior is an example of both ADHD and the developmental changes of adolescence. I usually advise the parent to hold the adolescent accountable for his or her actions and apply whatever consequence is warranted, but also to temper his or her affective response and avoid attributing the adolescent's behavior to malicious motives. I often use the example of a teenager who gets stopped by a policeman for going through a red light shortly after getting his or her driver's license. The adolescent may tell the policeman, "I didn't notice the red light because I have a disability, and I'm protected under the Americans with Disabilities Act," but the policeman is not going to care. The adolescent will be held accountable for adherence to the traffic laws, regardless of his or her ADHD.

Next, the therapist moves on to address expectations and beliefs, reminding the parents of Principles 18 ("Keep a disability perspective"), 19 ("Don't personalize the adolescent's problems or disorder"), and 20 ("Practice forgiveness"), and pointing out that they are now going to discuss the beliefs and attitudes underlying these principles in more depth. Then, he or she might ask the parents to engage in the following mental imagery exercise, which vividly teaches people the connection between extreme thinking, negative affect, and behavioral overreactions:

> "Close your eyes, and imagine you are opening the mail. You find a progress report from your son's [or daughter's] school. The progress report indicates that he is failing English and math, and has 15 late assignments in history. Suddenly you can feel your blood begin to boil and the tension mount throughout your body. Your son lied to you again! He said he was up to date on homework and passing all of his courses. This is one more example of irresponsible behavior. He is always irresponsible. You told him to keep an assignment book and get help from the teachers. He never does what he is told. He is so disobedient. If he keeps on going this way in school, he is going to fail. He will never graduate, never go to college, and never get a good job. You will be supporting him until the day you die. And the thought of confronting him is not appealing at all. He will deny it all at first, then blame it all on the teachers, showing you total disrespect. He is just doing all of this to get you mad and upset. He has no consideration for your feelings. Now open your eyes, and tell me how you feel and what you are thinking. And also tell me, how would you react if your son walked through the door at this very moment?"

Through a Socratic discussion, the therapist helps the parents to realize how the extreme thinking evokes extreme affect and how difficult it would be to deal with the adolescent rationally, as a principle-centered parent is advised to do, in such a strong state of negative affect. Afterward, the therapist suggests that the parents need to strive toward adherence to the following overall coping expectation:

> "We will encourage our adolescent with ADHD to go for the stars, to do his or her best, but we will accept that it is not a catastrophe when he or she fails to achieve perfection, and it does not mean that he or she is headed for certain ruination or that he or she is purposely trying to anger us."

After discussing this rationale and the more positive coping attitude, the therapist then gives a copy of Table 22.3 to each parent, and reviews the most common unreasonable beliefs. As they go through each unreasonable belief, the therapist asks the parents to rate their own adherence to this belief and to provide examples of particular situations that activated the belief. They look at the reasonable alternative beliefs and expectations in the right-hand column, and the therapist asks the parents whether they find them credible. If the parents do find the reasonable beliefs credible, the therapist continues; otherwise, he or she reviews the evidence for each unreasonable versus reasonable belief and suggest experiments the parents can do to test this evidence on their own after the session is over. A therapist does not usually have time to review every belief; he or she may quickly survey the table and concentrate

TABLE 22.3. Parents' Expectations and Beliefs

Unreasonable beliefs	Reasonable beliefs
I. Perfection/obedience: Teens with ADHD should behave perfectly and obey their parents all the time without question.	I. It is unrealistic to expect teens with ADHD to behave perfectly or obey all of the time; strive for high standards, but accept imperfections.
A. School	
1. He should always complete homework on time.	1. I will encourage him to complete homework all the time, but I recognize this won't always happen. That's the way it is.
2. She should study 2 hours every night, even when she has no homework.	2. If your attention span is short, you are lucky to get your basic homework done. Extra study is just unrealistic. These kids need a break after all the effort it takes to do basic homework.
3. He should always come to class prepared.	3. He will sometimes come to class unprepared, but I will help him learn good organizational techniques.
4. She should do papers for the love of learning.	4. Research shows that teens with ADHD need salient, external reinforcers to motivate their behavior.
B. Driving	
1. He should never get any speeding tickets.	1. All teens with ADHD get at least one speeding ticket. He should be responsible for paying it and take his medicine.
2. She will never have an accident.	2. Research shows that most teens with ADHD will get in at least one accident. She should take her medicine and do her best. She should drive an old car.
3. He should never text his friends while driving	3. He should avoid texting. If texting while driving is illegal in your state, he should face the legal consequences if stopped by the police.
4. She will always stop completely for stop signs.	4. I should stop completely at stop signs, to model good behavior when my teen is in my car. I should only expect my teen to do as well as I do.
C. Conduct	
1. He should be a perfect angel in church.	1. This is unrealistic. As long as there are no major disturbances, I'm satisfied. Perhaps I should find a youth group service of more interest for him anyway.
2. She will impress all the relatives with her love for family gatherings.	2. Give her space. Teens just don't want to be with their families that much. This is typical. She should attend some family functions, but that is all I can reasonably expect.
3. He should never treat us disrespectfully.	3. You can't become your own person without some rebellion. Some backtalk is natural. He shouldn't curse or ridicule severely and might be expected to apologize or face negative consequences.
4. She should get out of a bad mood when we tell her to change her attitude.	4. People with ADHD are just moody and can't stop it. She should let us know when she is in a bad mood and wants to keep to herself. We should not make a lot of demands on her at such times.
D. Chores	
1. She should put away the dishes the first time I ask.	1. It won't always happen the first time, but after several reminders, I should act, not yak (i.e., apply consequences).
2. He should always get the room spotless.	2. He should get it generally neat. Spotless isn't realistic.
3. She should not waste electricity by leaving the lights on.	3. She is just forgetful. We could work out a reminder system. But this is the least of my worries with a teen with ADHD.
4. He shouldn't be on his cell phone when I've sent him to his room to clean it up.	4. Teens with ADHD will get off task; I will redirect him back to the task, and if it happens too much, I will take away the cell phone for 24 hours.

(continued)

TABLE 22.3. *(continued)*

Unreasonable beliefs	Reasonable beliefs
II. Ruination: If I give my teen too much freedom, she will mess up, make bad judgments, get in big trouble, and ruin her life.	II. She will sometimes mess up with too much freedom, but this is how teenagers learn responsibility: a bit of freedom and a bit of responsibility. If they backslide, no big deal. I just pull back on the freedom for a while, and then give her another chance.
A. The room is incompletely cleaned: He will grow up to be a slovenly, unemployed, aimless welfare case.	A. The state of his room has little to do with how he turns out when he grows up.
B. Home late: She will have unprotected sex, get pregnant, dump the baby on us, take drugs, and drink alcohol.	B. I have no evidence that she would do all these things. She is just self-centered and focused on having fun.
C. Fighting with siblings: He will never learn to get along with others, have friends, have close relationships, or get married. He will end up a loser, and be severely depressed or commit suicide.	C. There is no scientific evidence that sibling fighting predicts later satisfaction in relationships. Siblings always fight. They will probably be closer when they grow up.
III. Malicious intent: My adolescent misbehaves on purpose to annoy me, or get even with me for restricting him.	III. Most of the time adolescents with ADHD just do things without thinking. They aren't planful enough to connive to upset parents on purpose.
A. Talking disrespectfully: She mouths off on purpose to get even with me for punishing her.	A. Impulsive teenagers just mouth off when frustrated. I'll try not to take it to heart but will issue consequences.
B. Doesn't follow directions: He doesn't finish mowing the grass on purpose to get me angry.	B. Teens with ADHD are allergic to effort. They don't take the time to plan to upset parents.
C. Restless behavior: She shuffles her feet and plays with her hair to get on my nerves.	C. Teens with ADHD just can't contain themselves. I'll try not to attach meaning to her restlessness and ignore it.
D. Spending money impulsively: She bought $100 of songs for her iPod just to waste our money.	D. She probably just saw the song listings online and had to have them. Poor delay of gratification is part of ADHD. She won't get any extra money for lunch or gas.
IV. Love/appreciation: My teen should love and appreciate all the great sacrifices I make; if she really loved me, she would confide in me more.	IV. Teens with ADHD are so self-centered that they don't easily show appreciation until they grow up and have their own children with ADHD. Only then will they realize what you did for them.
A. Money: What do you mean you want more allowance? You should be grateful for all the money I spend on you now. Some kids are not so lucky.	A. "You will have to earn more allowance. I'd appreciate a thank-you, even though I understand you don't really think about what I do for you."
B. Communication: She never tells me anything anymore; she must not love me.	B. It's natural as teens individuate to keep more to themselves. As long as I am available when she wants to talk that's all I can expect.
C. Spending time: If he really loved us, he wouldn't spend so much time alone in his room.	C. Spending time alone has nothing to do with love. It has to do with wanting privacy as he becomes more independent.

on the beliefs that seem most salient for a particular family. At the end of the session, the therapist assigns the parents the task of looking for examples of these extreme beliefs in their interactions with their adolescent and challenging them, replacing them with more realistic beliefs.

In the second session of Step 3, the therapist starts with the working hypothesis that most teenagers with ADHD feel that their parents are unfair and restrictive of their freedom, and that the restrictions are interfering with their lives. With the adolescent, the therapist's goals are (1) to assess the extent and rigidity of these beliefs, (2) to determine how the amount of freedom given to the adolescent compares to the local norms for other adolescents of a similar age in the same schools and neighborhood, and (3) to correct any wildly unrealistic expectations that the adolescent may have. The therapist gives Table 22.4 to the adolescent and uses it as a springboard for discussion. He or she should carry out the discussion in a lighthearted, tongue-in-cheek style, trying to remain animated and to keep the adolescent's attention. He or she should make liberal use of exaggerations for effect, and abbreviate the session or shift gears if the adolescent seems to be drifting; the therapist must not conduct a monologue and should not worry too much if the adolescent misses the subtleties of his or her points. The extent to which the therapist will be able to accomplish these goals varies greatly from adolescent to adolescent, depending on the adolescent's attention span, level of resistance, and general maturity.

Let us look in on Dr. Sam as he conducts a discussion of beliefs with Abe, a 15-year-old recently diagnosed as having ADHD.

DR. SAM: Look at the first thing on the list—the idea that your parents' rules are totally unfair and will mess up your life. Have you ever felt that way?

ABE: Yep. Just like the curfew one. They made me come home early from the homecoming dance. My friends probably thought I was a real nerd.

DR. SAM: If you keep thinking, "My parents are unfair, my parents are unfair, they're going to mess me up," and so on, how are you going to feel?

ABE: Pissed off at them. I do feel that way.

DR. SAM: So if you are pissed as hell at them and try to get a later curfew, are you going to have a nice, calm discussion?

ABE: We always have a yelling match. And I get grounded.

DR. SAM: So maybe you can do something to keep from getting so pissed off at them that you lose your cool and then your privileges. If I were you, I'd try thinking to myself something like this: "Yes, I don't like coming home early from the dance, but parents always worry too much about what could happen. Yes, it's unfair, but it's not the end of the world. My friends are loyal and will understand. There will be more dances, and maybe I can get a later curfew. I'm going to tell myself to stay cool and calm when I approach them to discuss this. I'm not going to blow it and get grounded again."

ABE: Do you really think I can convince them to change my curfew for the Halloween dance?

DR. SAM: I don't know, but if you stay calm and don't think the worst, you might. I'll help you and your parents to try to work it out to everyone's liking. What about the idea that you should have as much freedom as you want all the time? Do you ever feel that way?

ABE: Yes, it's like they are always bossing me around. Especially about homework. My mother keeps bugging me to start my homework.

DR. SAM: So your mom is the big bad slave driver on homework. Now I want you to be totally honest, and I will never tell, but do you really think you would get your homework done without your mother bugging you?

ABE: Well, I don't know. . . . Doc, probably you're right. Nope.

DR. SAM: People with ADHD need structure to get things done. So how can we get you the structure around homework without you feeling like she is taking away your freedom? Any ideas?

ABE: I could set an alarm on my phone to go off when it's time to do homework.

DR. SAM: Great idea. Go ahead and set it now (Sam does so). We can talk that over with your parents.

ABE: Can we talk that over next week? How much longer until we stop?

DR. SAM: You've done a great job with this discussion. Let's stop right now.

Here, Dr. Sam discusses unfairness/ruination and autonomy with Abe. The therapist uses practical

TABLE 22.4. Adolescents' Expectations and Beliefs

Unreasonable beliefs	Reasonable beliefs
I. Unfairness/ ruination: My parents' rules are totally unfair. I'll never have a good time or any friends. My parents are ruining my life with their unfair rules. They just don't understand me.	I. Yes, I don't like my parents' rules, and maybe they are sometimes unfair. But who said life is supposed to be fair? And how many other teenagers have gone through the same thing? They turned out OK. So will I. I'll just have to put up with it the best I can.
A. Curfew: Why should I have to come home earlier than my friends? They will think I'm a baby. I'll lose all my friends.	A. My friends are loyal. They will understand that my parents are creeps about curfew. I won't lose any friends.
B. Chores: Why do I get stuck doing all of the work? Sam [brother] doesn't have to do anything. That's unfair!	B. Sam has some chores too. I'll count them up, and if I have more, I'll talk nicely to my parents about it.
C. School: My teacher is unfair. She picks on me all the time. I always get stuck doing extra homework. I'll never have time for fun. Life is one big homework assignment.	C. Maybe she does pick on me. There could be a reason. I never am with the class or know the answer when she calls on me. Maybe if I kept up with the work, she wouldn't call on me so much.
II. Autonomy: I ought to have complete and total freedom. My parents shouldn't boss me around or tell me what to do. I'm old enough for freedom now.	II. No teen has complete freedom. No adult really does, either. Sometimes I need my parents, like for money, or God forbid, even to talk to in times of trouble. I want a lot of freedom, but not total freedom.
A. Chores: I don't need any reminders. I can do it totally on my own.	A. I have not been getting them done on my own. I need to stop being an idiot and accept a little help.
B. Medicine: I don't need medicine any more. I'm grown up now and can handle everything on my own.	B. Maybe I need to see whether I do better or worse on or off medicine. I'll keep an open mind about it.
C. Smoking: It's my body. I can do whatever I want with it. You have no right to tell me not to smoke.	C. It's my body. But do I really want to mess it up? My friends have gotten hooked on smoking. It costs a lot. And it tastes terrible when you kiss.
III. Love/appreciation: Getting material things is a sign that your parents love you. Getting your way is a sign that your parents really love you.	III. Material things don't tell you whether someone really cares about you. Neither does getting your way all the time. It's how you are inside that makes the difference.
A. Clothes: If my parents really loved me, they would let me buy those designer clothes.	A. I would like designer clothes, but that's not how I tell whether my parents love me. I can tell from how they act toward me and the affection they show.
B. Smartphone: If my parents really loved me, they would buy me the latest iPhone and let me use Facebook and Instagram as much as I like at any hour of the day or night.	B. Getting an iPhone is a big privilege, not a sign of love. And once I have an iPhone, it is natural that they will be concerned about what I do with it.
C. Sexuality: If I have sex with my boyfriend, then he will really love me forever and marry me.	C. Love does not equal sex. I need to judge from how my boyfriend acts and expresses his feelings to me whether he loves me. All boys want sex. So this tells me nothing about love.

motivations—for example, the possibility of a later curfew and getting Abe's parents to stop nagging him about homework—to help reinforce the utility of considering more reasonable beliefs. Teenagers respond better to such tangible contingencies than to an abstract discussion, such as why the world is intrinsically unfair or why unlimited autonomy is bad for adolescents. After a reasonable effort, when Abe indicates he is losing interest in the discussion, the therapist stops the session. Covering one or two of the expectations and beliefs may be as much as it is reasonable to expect in a session with an adolescent who has executive function challenges. The therapist devotes the first few minutes of the next session with the family to a summary of Step 3, pointing out that they will return as needed to beliefs and expectations throughout the remainder of the intervention.

Placement of cognitive restructuring early in the overall family intervention represents a significant departure from the published manual for the *Defiant Teens* intervention (Barkley & Robin, 2014). In the published version, cognitive restructuring is covered in the 15th (out of 18) session. I have made this change because (1) parents with extreme beliefs resist the early steps of family intervention, and integrating discussion of extreme beliefs into educating them about ADHD and adolescent development circumvents such resistance and sets the therapist up for success; (2) knowledge of the adolescent's beliefs permits me to judge how receptive he or she will be to contingency management and problem solving; and (3) I can easily use cognitive restructuring during any of the subsequent steps of the intervention to deal with resistance to the interventions from any family member.

Although there are not yet any comprehensive studies of the process of educating and addressing the beliefs of adolescents with ADHD, Blotnicky-Gallant, Costain, and Corkum (2013) developed and pilot-tested a demystification program for adolescents with ADHD. Twenty-seven adolescents with ADHD in grades 7–9 participated in a 2-hour workshop covering the definition of ADHD, how it impacts one's life, the brain and ADHD, evidence-based treatments, myths surrounding ADHD, creating personal strength profiles, and learning self-advocacy skills. Measures given before, after, and at 2-week follow-up from the workshop tapped knowledge of ADHD, opinions about medication, behavioral/psychological and alternative treatments for ADHD, and satisfaction with the workshop. Knowledge of treatments, positive opinions about medication treatments, and self-advocacy scores increased from pre- to postassessment. The investigators need to refine the measures, add a control group, and select larger sample sizes to further test the effectiveness of this promising program.

Step 4. Preparing Families for Medication

Medication has repeatedly been demonstrated to be an effective intervention for the core symptoms, executive function problems, and associated impairments of ADHD in children and adolescents (see Chapter 27). In Step 4 the therapist first meets with the parents, provides information regarding medication for ADHD, assesses whether they are willing to have their teenager started on medication, and if so, provides referrals to appropriate physicians. Since adolescents often have misgivings about medication, in this section, I provide the practitioner with tools to use when discussing medication with teenage patients.

Research examining children and adolescents' attitudes toward their medication paints a mixed picture of their perceptions (Bowen, Fenton, & Rappaport, 1991; Doherty, Frankenberger, Fuhrer, & Snider, 2000; Moline & Frankenberger, 2001). Although the vast majority of participants reported a positive impact of stimulant medication on their functioning, the studies using anonymous self-reports found that over one-third were ready to give up taking the medication. Not surprisingly, when parents were in the room while adolescent attitudes were assessed, most children and adolescents reported that they wanted to continue taking their medication. Researchers also found that teenagers without ADHD did not consider their peers with ADHD who took medication to be any different than peers without ADHD.

Therapists understand the reluctance of many adolescents with ADHD to take medication within a developmental context. During a time of identity exploration marked by a fierce desire to be different from their parents but carbon copies of their peers, adolescents do not want to do anything that makes them feel different from their friends. Nor do they wish to follow regimens they perceive as imposed by adults—either parents or physicians. They believe they know what is best for themselves and look on medication as a source of "external control" from which they need to "individuate." Teenagers who took stimulant medication as younger children often complain that it calms them down too much, or, as one athletic youngster said, "Medicine takes away my killer instinct." What they mean is that they enjoy being wild and impul-

sive, which medication curbs. They also often attribute a variety of extraneous somatic complaints to the medication, even though most of these may bear little relation to the actual side effects of either stimulant or nonstimulant medications, and at least one study has suggested that adolescents may have inaccurate beliefs about the effect of medication on their behavior (Pelham et al., in press).

A sensitive professional must take developmental factors into account when presenting medication to an adolescent. The traditional "doctor knows best" style of presentation often backfires. A Socratic approach that permits the adolescent to be in control and make decisions for him- or herself about the use of medication minimizes resistance (Schubiner, Robin, & Young, 2003). We have also found motivational interviewing (Naar-King & Suarez, 2010) to be a useful approach to increasing adolescents' willingness to take medication. The following excerpt from a discussion between a physician and an adolescent illustrates the socratic approach:

DR. JONES: I understand that things are pretty rotten at school. Tell me about it.

BILL: Yeah. I'm getting lousy grades. My teachers get on my case about talking out.

DR. JONES: So you are upset about low grades and teachers bugging you about talking too much.

BILL: Yeah, I'd rather get C's, B's. Then, no one will bug me.

DR. JONES: What stops you?

BILL: School's so boring. I can't make myself listen to the teacher, study, even if I want to. And all the noise in class bugs me.

DR. JONES: Concentrating on the teacher, books, and homework is tough.

BILL: Yes. Is that part of ADHD?

DR. JONES: Yes.

BILL: What can I do change it?

DR. JONES: Think of changing it like wearing glasses. You and I both wear glasses. When we take our glasses off, what happens?

BILL: Things look foggy.

DR. JONES: Right! We need glasses to see clearly. We didn't choose to need glasses. That's the way our bodies and eyes are, right?

BILL: I guess so.

DR. JONES: ADHD is similar. You didn't choose to have trouble with concentrating and thinking before acting. Your brain just does it. I don't have any glasses for concentration, but I do know of some medications that may help.

BILL: What do you mean? I'm not taking any pills! Only retards need that.

DR. JONES: You feel like I think you're a retard because I'm suggesting medication.

BILL: Yes. All my friends make fun of those special ed kids on the bus who take pills.

DR. JONES: On the one hand, you think others will mock you for being a retard who takes medication, yet Dr. Robin explained that your IQ was above average.

BILL: Yeah. He did say that.

DR. JONES: And as for kids making fun of you, how will they find out unless you tell? By the way, does wearing glasses make either of us retards?

BILL: I guess not.

DR. JONES: I prescribe medication for ADHD frequently because it helps most people. I think medication might help you, but it is totally up to you. I don't have stock in medication companies, and I don't really care if you take medication or not. But I do care if you do well in school. If you want to see if this medication can help you do better in school, I will help you find the best medicine. If not, we will stop talking about it. It is up to you.

BILL: What if this medicine makes me feel weird?

DR. JONES: I won't ask you to put up with any bad side effects. If the medicine makes you feel weird, we will change it right away—either the amount or the type of medicine.

BILL: How do I know my parents won't trick you into making me take medicine for longer than I want? Or taking so much medicine that I turn into a wimp?

DR. JONES: I give you *my word* that you have *the last word* on all decisions about medication. I will not make any decisions about medication that you do not agree with, no matter what your parents want. We will discuss it just between ourselves and tell your parents afterwards what we decided.

BILL: OK, I will give it a try, but just for 1 month.

DR. JONES: You're the boss. One month it will be, and then you can tell me what you think.

Dr. Jones first established that Bill wished to improve his school performance and that increased concentration was essential. Then he used the analogy of visual impairment to provide a rationale for stimulant medication. When Bill objected, he empathized with Bill's concerns but provided accurate information about medication. He made it clear that he did not have a personal investment in whether Bill took medicine or not, but that he cared about helping Bill do better in school. He debunked several myths Bill mentioned about the medication and reiterated that Bill would be in control of all medication decisions. He "put his money where his mouth is" by agreeing to Bill's request for a 1-month trial on medication. When physicians follow this approach, they rarely encounter adolescents refusing to try medication.

However, when using this approach, the therapist needs to be prepared to accept the decisions of those adolescents to forgo medication and to convince the parents that they also need to accept this decision. He or she explains to parents that if they try to force an adolescent to take medication now, they may ruin any chances that the adolescent will ever agree to take medication. It is better to use other interventions and see whether the adolescent comes to his or her own decision later to try medication. In such cases, the therapist helps the adolescent develop other approaches to improving school performance and behavior at home. However, the adolescent is asked to make a contract with the therapist that if school grades and behavior at home improve, medication will not be mentioned again, but if other interventions prove insufficient to improve school grades and behavior at home, the adolescent will consent to reconsider medication at the end of the next marking period.

The mental health professional plays an important role in helping the adolescent maximize the positive effects of medication. When prescribing medication for adolescents, a physician must pay particular attention to ensure an adequate dose level, careful titration of doses to particular activities (school, driving, homework, etc.), clear-cut criteria for evaluating outcome, careful attention to minimizing side effects, and adjustment of timing to provide adequate length of medication coverage throughout the day. I help my adolescent patients and their parents decide what help they want from medication, then, with their permission, I talk with their physician. This may involve advocating with physicians for the addition of short-acting stimulants to a single dose of a long-acting stimulant or obtaining feedback about the effectiveness of the medication. Adolescents with ADHD do not typically know how to judge the effectiveness of their medication. In addition to teacher feedback, I help them pinpoint medicine-sensitive behaviors to use as a yardstick to judge the effectiveness of various doses. For example, reading a boring textbook or doing math problems is often medication-sensitive. The teen is asked to read the boring book several times on each dose of medicine and report the results to the physician. The ability to resist texting while driving may also be a medication-sensitive target behavior.

Step 5. Breaking the Negativity Cycle

As noted earlier in the chapter, when many parents and adolescents present for therapy, they are stuck in a seemingly endless downward spiral of negativity. The teenager is used to criticism, correction, direction, and punishment, to the point that he or she may feel demoralized, depressed, and helpless to change things. The parents are used to arguments, back talk, having to repeat commands many times, and failure to follow instructions. After explaining coercion and the four-factor model to them and getting medication started, the therapist needs to introduce tasks that will interrupt this cycle. Step 5 presents "one-on-one time" as a strategy for breaking the negativity cycle. First, the therapist meets with the parents, explains one-on-one time, gets them to "buy" into it, and sends them home to implement it with their teen. Then, they return to the next session with the teen to discuss implementation of one-on-one time and to deal with any problems that arose. They are asked to continue to implement it at home. *The therapist does not continue to Step 6 until Step 5 has been fully implemented* because clinical experience suggests all subsequent steps will fail until the negativity cycle is broken.

The therapist introduces this task by asking the parents how they would feel about two different kinds of supervisors at work: a highly critical supervisor versus a positive, encouraging supervisor. Parents inevitably prefer the positive supervisor. Similarly, the adolescent would prefer an encouraging, noncritical parent as supervisor. The therapist explains that one-on-one time is designed to help parents come across like a positive supervisor. The parents are asked to spend 15–20 minutes of one-on-one time with the teenager, five times per week; in two-parent families, mothers and fathers take turns. During this time, the teen selects an activity that he or she enjoys, and parent and adolescent participate in the activity together. The therapist ex-

plains that these are activities that can be done at home, without traveling or spending money.

The teen needs to experience the parents as totally non-demanding, noncritical, attentive, and positive. Therefore, the parents are to refrain from giving commands, asking questions, giving directions, suggesting changes, criticizing, or organizing the activity; the parents are to be totally accepting and make only neutral to positive remarks. The teen is completely in charge of the activity. If the teen cheats or does not follow the rules of a game, the parents are to go along with the deviation from the rules during one-on-one time but indicate that the rules do apply at other times. The therapist explains that the goal is for the parent and adolescent to have fun together during one-on-one time, as perhaps they did some time in the past before conflict escalated. The therapist hopes that the teen will rediscover that parents can be fun, at least some of the time. The therapist relates this task to parenting Principle 16 ("Be the adolescent's cheerleading squad"). If the parent anticipates that the adolescent will refuse to participate in any activity with him or her, the therapist advises parent to wait until the adolescent is engaged in a fun activity, nonchalantly stand next to the teen, and ask to join in the activity.

Clinicians have found that there is for the parent a distinct advantage in carrying out the one-on-one time at the beginning of the largest chunk of time during the day that the parent and adolescent are together (Kaufman, 2003). The positive feeling created by experiencing a parent as totally accepting and noncritical often persists for several hours after the completion of one-on-one time. As a result, the adolescent is more cooperative and respectful toward the parent during those hours.

Step 6. Parental Attention: Praise, Ignoring, and Commands

At the beginning of this step, the therapist reviews the homework assignment of one-on-one time. If any problems arose, the therapist helps the parents find solutions to these difficulties; afterward, the therapist prescribes continued practice of one-on-one time five times per week.

Step 6 involves instructions for praising positive behaviors, ignoring minor negative behaviors, and giving effective commands. First, the therapist asks the parents to identify common situations in which the teenager misbehaves. The therapist inquires as to how they now handle such situations. Often the parents describe taking away privileges, grounding, or using other punishments to handle misbehavior. Then the therapist asks the parents to imagine the same scene, except that the adolescent now behaves appropriately. What would the parents do then? Often they respond, "Nothing." The therapist points out how misbehavior results in parental attention, while appropriate behavior receives no attention. Reminding parents of parenting Principle 7 ("Use incentives before punishments"), the therapist suggests that parents should praise the teen for positive behavior as often as is feasible. Minor negative behaviors should be ignored; the goal is to shift parental attention from negative to positive behavior. Parents are then assigned the homework task of identifying a minor misbehavior to ignore. They are instructed to ignore the minor misbehavior and praise all instances of the opposite, positive behavior.

Second, the therapist focuses on increasing praise for appropriate behavior. The parents are asked to consider the last 100 statements that they made to their teenager. How many were positive? Negative? Most parents realize that the majority of their statements are negative. The therapist explains that increasing praise for appropriate behavior will help break the negativity cycle. Parents are assigned the task of finding 10 new opportunities per day to praise their teenager, even if the behaviors they praise are "minor." If a parent cannot find such opportunities, the therapist asks him or her to wait until the teen is not engaging in a problem behavior for 15 seconds, then say, "I like the way you are acting now."

Third, the therapist models effective commands for the parents, following the following guidelines:

1. Make sure that you mean it when you give a command. That is, only give those commands on which you intend to follow up.
2. Do not present a command as a question or favor; state it simply, directly, and in a business-like tone.
3. Do not give more than one command at a time.
4. Make sure you have your teen's attention before giving a command.
5. Reduce all distractions (TV, computer, video games) before giving a command.
6. Ask your teen to repeat the command right after you give it.

Parents are assigned the task of practicing effective commands at home over the next week.

Step 7. Implementing Positive Incentive Systems

The creative use of positive incentive systems is a major technique that parents can use. Point systems and behavior charts are appropriate for adolescents under age 13, but behavioral contracts are appropriate for adolescents ages 13–18. I focus on the behavioral contract, which is a written agreement between a parent and an adolescent specifying an exchange of behavior for privileges. Spelling out an agreement in writing underscores each party's commitment to change and prevents later misunderstanding of the terms of the agreement. The contract can be short or long, simple or complex. The parents attend the first session of Step 7 without their adolescent and receive help in developing a contract; the teenager returns to the next session with the parents to discuss implementation of the contract.

Developing an effective behavioral contract takes six steps. First, the therapist asks the parents to make a list of the behaviors that they want the adolescent to do more often; if the parents focus on negative behaviors to be stopped, the therapist helps them refocus on the positive behaviors that are the opposites of the negative behaviors they bring up. Second, the parents are asked to rank-order the target behaviors, based on how difficult it would be for their teen to comply in terms of time and effort, and the likelihood of cooperation. Third, the therapist asks the parents to make a list of potential privileges that the teen can earn; they are asked to review this list with the adolescent at home and modify it according to the adolescent's input. Fourth, the therapist prompts the parents to select a target behavior of relatively low difficulty and a privilege of moderate value to the adolescent. Fifth, a brief contract is written up, specifying that the adolescent will only gain access to the privilege by completing the target behavior. The written contract should clearly specify the behavior to be performed, the date and time the behavior is to be performed, the consequences for compliance, and the consequences for noncompliance. Sixth, the parents are instructed to present this contract to the adolescent, ask the adolescent to sign it, and then implement it.

Here are several examples of contracts:

> I, Bill Peterson, agree to take the trash cans from the garage to the street every Tuesday night by 8:00 P.M. If I carry this out, I will earn $3.00 and an extra hour of X-Box Live.

> Jenny Jones agrees to clean up her room every Sunday by noon. We, John and Jane Jones, consider the room clean if:
>
> a. The bed is neatly made.
> b. The clothes are off the floor and in the drawers, the hamper, or hanging in the closet.
> c. All books, papers, electronic equipment, makeup, and so forth, are off the floor and in a container.
> d. The carpet has been vacuumed (i.e., we heard the vacuum cleaner running for at least 6 minutes and there is no visible dirt on the floor).
>
> One of us will inspect the room at noon at Sunday. If the room meets all of the previous criteria, Jenny can go to the mall with two friends that afternoon. We will drive Jenny and her friends and give her $30 spending money.

Completing school homework is an example of a problem area that can be handled by a more complex behavioral contract. Figure 22.1 illustrates a complex behavioral contract for homework completion.

Parents often say that there are no privileges that the teenager wants—that they "already have it all." We explain to parents that teenagers have come to regard access to smartphones, tablet PCs, television, computers, the Internet, video games, and the car (for those who are driving) as their "birthright." In many families, a teen has access to all of these privileges unless they have been taken away as a punishment for misbehavior. However, there is no written law that adolescents should be in charge of access to these activities. The therapist asks the parents to recall their own childhood and the things to which they had access. Most parents will admit that they had to work to earn access to such privileges. The therapist points out that the parents have the right to make all of the privileges contingent upon appropriate behavior.

By the end of this session, the parents should have written a behavioral contract, with the therapist's coaching. They are assigned for homework the task of explaining the contract to their adolescent and implementing it. Upon their return with their adolescent to the next session, the therapist reviews implementation of the behavioral contract. If the contract was effectively implemented, the therapist praises the parents and continues to coach them to develop a second contract. If they encountered difficulties implementing the contract, the therapist helps them plan to work around

I, Michael Adams, and my parents, my teachers, my guidance counselor, and Dr. Jones agree to carry out to the best of our ability the following homework plan:

I. Keeping track of assignments
   A. My teachers will write the assignments on the board every day. They will also post a copy of all the assignments for the week on the school website, which Mrs. Smith, my guidance counselor, will access each Monday. My parents will check the website daily and contract Mrs. Smith if they cannot find the assignments there.
   B. I will write down the assignment from the board in my planner every day before I leave each class. My teachers will also permit me to take a picture of the board with my phone. I will read over what I have written down to make sure I understand it. I will ask the teacher to explain any assignment I do not understand.
   C. At the end of school, before I leave, I will read over each assignment I have written down and make sure I understand what I am being asked to do. I will go to my locker and gather any materials that I need to take home for completing the assignments.

II. Bringing home materials
   A. I will bring home all the materials I have gathered along with my planner book.
   B. My mother agrees to ask me nicely one time without nagging to see my list of assignments in my planner. I agree to show it to her without a big hassle or an attitude. If anything looks incomplete, she will look it up on the website.

III. Schedule and setting for doing homework
   A. From Sunday through Thursday, I agree to work on homework from 6:00 to 8:00 P.M. If I finish early, I will show my completed work to a parent, and if he or she agrees that it is completed, I can do whatever I want.
   B. I will do my homework at the big desk in the den. I can listen to soft music with headphones, but no loud rock, no instagram or Facebook, no texting, and no phone calling. If I find myself getting distracted, I will take a short break, do something physical (not telephone), and start working again.
   C. My mother will remind me once without nagging to start on my homework at 6:00 P.M. I will start without an attitude.

IV. Daily plan for organizing homework completion
   A. With help from my mother, I will make an organized plan for each night's homework. This plan will guide me in what subject I will do first, second, etc. It will also divide up homework time between assignments due tomorrow and long-term assignments. My mother agrees to permit me to determine the order of doing homework.
   B. In my plan, I will estimate the time needed to complete each assignment, as well as how I will check each assignment over for accuracy, completeness, and legibility.
   C. The plan will specify how often I will take breaks during homework time, how long the breaks will be, and how large assignments will be divided into smaller units.
   D. The plan will specify where I will put the completed assignments and how I will make sure I turn the work in.

V. Medication. I agree to supplement my long-acting medicine with a dose of short-acting medicine at 5:00 P.M. on Sunday through Thursday, to help me concentrate on homework.

VI. Turning in assignments
   A. As I finish an assignment, I will put it in the section of my binder for that class.
   B. I will do my best to remember to hand in each assignment.
   C. I will back up all assignments typed on my computer to a USB memory stick.

(continued)

FIGURE 22.1. Example of a complex behavioral contract for homework completion.

VII. Feedback. My teachers agree to tell me how I did within 2 days after I hand in an assignment. They also agree to e-mail my parents feedback about how many of the last week's assignments were turned in on time when they send the next week's assignment list.

VIII. Rewards. For each week that I meet the terms of this contract, for the next week my parents grant me full use of my iPhone, including unlimited texting and phone calls and unlimited internet access. I agree to stop iPhone use between 10 P.M. and 7 A.M. Sunday through Thursday.

Signed, *Michael Adams Robert Adams Barbara Adams Bill Jones, Principal Brenda Smith, Guidance Counselor Millie Broadbent, Algebra Tom Jones, English Darla Breeze, French William Sonoma, Chemistry F. A. O. Schwartz, Gym Neiman Marcus, History*

FIGURE 22.1. *(continued)*

those difficulties and implement the contract more effectively. The therapist should coach families to write and implement several behavioral contracts before moving onto Step 8.

Step 8. Implementing Punishment Systems

In accordance with parenting Principle 7 ("Use incentives before punishments"), the therapist does not introduce the material on punishments, such as taking away privileges, work detail, and grounding, until the parents have experienced success implementing a positive incentive system in the form of a behavioral contract. Punishment techniques are typically needed when positive incentive systems only partially change a significant problem behavior, or when non-negotiable rules are broken or severe misbehavior occurs. Taking away a privilege can be a creative form of punishment; for example, parents can remove an infinite variety of privileges, such as texting and Internet access on smartphones, video game access, iPod access, television time, computer use, having a friend over, borrowing things, special foods, bicycle, skateboard, sports equipment, access to playing various sports, use of the car, various types of privacy (e.g., having a door on the teen's room), parental transportation to special events; parents may also charge monetary fines. Work detail involves unpleasant work around the house, but parents should only use it when they can get their adolescent to complete the work without a secondary power struggle. Grounding refers to being told to remain home at home when the teen wants to leave the house and socialize with friends. The therapist should ask the parents to list all of the adolescent's meaningful privileges. Then he or she should give the parents examples of how to "fit the punishment to the crime"—that is, to come up with a loss of privileges of appropriate intensity and duration for the problem behavior that it is designed to decrease. The therapist should give correct versus incorrect examples, explaining to the parents the rationale for each case. A few examples follow:

1. *Misbehavior*: Alice refuses to clean up her room. *Appropriate removal of privileges*: Alice loses all her electronics (TV, radio, computer, video games, cell phone) for one evening. *Too mild*: Alice loses 1 hour of TV. *Too severe*: Alice loses all her electronics for 1 week.
2. *Misbehavior*: Peter curses frequently at his younger sister. *Appropriate removal of privileges*: A monetary fine of 50 cents per cursing episode. *Too mild*: A monetary fine of 1 cent per cursing episode. *Too severe*: Peter loses all electronics for 1 month.
3. *Misbehavior*: Sharon lies about not having homework. *Appropriate response cost*: Sharon loses her iPhone for 4 days. *Too mild*: Sharon can't use her iPhone for 1 hour. *Too severe*: Sharon loses her iPhone until the next report card comes out. Parents should only remove those privileges that they can control. For example, it is not effective for a mother who works until 6:00 P.M. to tell her 15-year-old son, who is home alone, that he cannot play X-Box after school.
4. *Misbehavior*: Bill is caught smoking marijuana with his friends in the backyard; it is a first offense. *Appropriate removal of privileges:* Bill cannot go out with those friends or communicate with them by phone, instagram or Facebook for 1 month, and can only visit with them at school or in his home with a parent present. He also has to submit to random drug testing at his parents'

discretion and write an essay about the dangers of smoking marijuana. *Too mild*: Bill cannot see his friends for one day. *Too severe*: Bill can never see his friends again and must find a new group of friends. His parents also report him to the police and have charges brought in juvenile court.

Grounding the adolescent (i.e., confining him or her to the house for a period of time) is also a common and effective punishment, often reserved for more serious misbehavior. However, it is easy for parents to pile on long groundings, one after another, until it becomes more of a punishment for the parent who has to stay home to enforce the grounding than for the adolescent. Then the parent is also backed into a corner, with no more punishments left to give. Many adolescents decide that they have nothing more to lose by acting very disrespectful at such times; severely negative verbal behavior occurs, and the overall level of conflict escalates rapidly. Grounding the adolescent for one weekend or 2–3 days is usually as effective as grounding for a week or longer. Parents do need to be present to monitor and enforce groundings; if a parent cannot be present, the grounding should be postponed.

After discussing loss of privileges, work detail, and grounding in a meeting with the parents, the therapist should coach them to select a problem behavior that was insufficiently corrected through positive incentives and add a punishment to it. For example, Mr. and Mrs. Smith had implemented a behavioral contract to reduce fighting and teasing between their sons, ages 13 and 12, both of whom had ADHD. The contract divided the school day into three intervals: before school in the morning, after school until dinner, and after dinner until bedtime. The contract stipulated that for each interval that the boys cooperated with each other or left each other alone, they each earned a quarter. Over the first 2 weeks of the contract, fighting decreased from four episodes per day to two episodes per day. Nonetheless, two physical fights per day were still excessive, so response cost was added. For every fight, no matter who started it, both boys were deprived of all electronics for the rest of that day and the next. Over the next 3 weeks, fighting stopped completely.

Step 9. Problem Solving Negotiable Issues

In Steps 5 through 8, the parents were taught to use behavior management techniques for improving parent–adolescent relationships. I first presented each technique to the parents, sent them home to implement it, then had them return with their adolescent to discuss implementation and improve upon any problems that arose. In Steps 9 through 11, parents and adolescents attend each session together, and the therapist teaches them how to resolve negotiable issues and improve their communication. During Step 9, families are taught to follow the four-step model of problem solving in Table 22.5 when discussing parent–child disagreements over negotiable issues (Robin & Foster, 1989). First, each family member *defines the problem* by making a clear, short, nonaccusatory "I statement" that pinpoints the others' problem actions and describes why these are problems. As each person gives his or her definition, the therapist teaches the others to verify their understanding of the definition by paraphrasing it to the speaker. This phase ends with a statement by the therapist acknowledging that there may be several different "problems" defined, but that if all agreed on the same definition, there would be no disagreement.

Second, the family members are given a worksheet such as that in Figure 22.2. They take turns *generating a variety of alternative solutions* to the problem. Three rules of brainstorming are enforced by the therapist to facilitate free exchange of ideas:

1. List as many ideas as possible—quantity breeds quality.
2. Don't evaluate the ideas at this point; criticism stifles creativity.
3. Be creative, knowing that just because you say it doesn't mean you will have to do it.

The therapist has family members take turns recording the ideas on the worksheet. At first, the adolescent may be asked to record the ideas—a strategy that helps maintain a minimal level of attention to the task. Usually, parents and adolescents begin by suggesting their original positions as solutions. Gradually, however, new ideas emerge. If the atmosphere is very tense or the family runs out of ideas, the therapist may suggest ideas, too, but usually the therapist suggests outlandish ideas to lighten the atmosphere and spur creativity. When the therapist judges that there are one or two "workable" ideas (i.e., ideas that may achieve mutual acceptance), the family is asked to move to the next phase of problem solving.

Third, the family is asked to *evaluate the ideas and decide on the best one*. The members take turns evaluating each idea, projecting the consequences of imple-

TABLE 22.5. Problem-Solving Outline for Families

I. Define the problem.
   A. Tell the others what they do that bothers you and why. "I get very angry when you come home 2 hours after the 11 P.M. curfew we agreed upon."
   B. Start your definition with an "I" statement; be short, clear, and don't accuse or put down the other person.
   C. Did you get your point across? Ask the others to paraphrase your problem definition to check whether they understood you. If they understood you, go on. If not, repeat your definition.

II. Generate a variety of alternative solutions.
   A. Take turns listing solutions.
   B. Follow three rules for listing solutions:
      1. List as many ideas as possible.
      2. Don't evaluate the ideas.
      3. Be creative; anything goes since you will not have to do everything you list.
   C. One person writes down the ideas on a worksheet.

III. Evaluate the ideas and decide on the best one.
   A. Take turns evaluating each idea.
      1. Say what you think would happen if the family followed the idea.
      2. Vote "plus" or "minus" for the idea and record your vote on the worksheet next to the idea.
   B. Select the best idea.
      1. Look for ideas rated "plus" by everyone.
      2. Select one of these ideas.
      3. Combine several of these ideas.
   C. If none are rated plus by everyone, negotiate a compromise.
      1. Select an idea rated "plus" by one parent and the teen.
      2. List as many compromises as possible.
      3. Evaluate the compromises (repeat steps III.A and III.B).
      4. Reach a mutually acceptable solution.
      5. If you still cannot reach an agreement, wait for the next therapy session.

IV. Plan to implement the selected solution.
   A. Decide who will do what, where, how, and when.
   B. Decide who will monitor the solution implementation.
   C. Decide upon the consequences for compliance or noncompliance with the solution.
      1. Rewards for compliance: privileges, money, activities, praise.
      2. Punishments for noncompliance: loss of privileges, groundings, work detail.

Name _____ Date _____

Problem or topic _____

SOLUTIONS	EVALUATIONS		
	Teen	Mom	Dad

Agreement _____

_____

Implementation plan

A. Teen will do _____ by the following time _____.

B. Mom will do _____ by the following time _____.

C. Dad will do _____ by the following time _____.

D. Plan for monitoring whether this happens _____

_____

E. Any reminders that will be given. By whom? How Many? _____

_____

_____

F. Consequences for compliance and noncompliance

1. Teen _____
2. Mom _____
3. Dad _____

FIGURE 22.2. Example of a problem-solving worksheet. From Robin (1998). Copyright 1998 by The Guilford Press. Reprinted by permission.

menting it and rating it "plus" or "minus." The therapist teaches family members to clarify each other's projections of the consequences of particular ideas, but to refrain from critical cross talk, which could sidetrack the discussion. The ratings are recorded in separate columns for each member on the worksheet. Here, the therapist prompts members to consider carefully whether the ideas address their perspectives on the original problem. When the ideas have all been rated, the family reviews the worksheet to determine whether a consensus was reached (all "plus") for any ideas. Surprisingly, a consensus is reached about 80% of the time. The family then selects one of the ideas rated positively by everyone, or combines several such ideas into the solution.

If a consensus was not reached on any idea, the therapist teaches the family negotiation skills. The therapist looks for the idea on which the family came closest to a consensus, and uses it as a catalyst for generating additional alternatives and conducting further evaluations, to spur agreement to a compromise position. A great deal of emphasis is placed on analyzing the factors impeding the parents and teen from reaching agreement and addressing them. Often cognitive distortions underlie intransigence in reaching a consensus, and these factors must be addressed (following suggestions provided earlier in this chapter) before a consensus can be reached.

During the fourth phase of problem solving, the family *plans to implement the selected solution and establishes the consequences for compliance versus noncompliance*. Family members must decide who will do what, when, where, and with what monitoring, to make the solution work. For adolescents with ADHD in particular, establishing clear-cut consequences for compliance versus noncompliance is very important because we know that their performance deteriorates in the absence of regular structure and immediate consequences. It is important to provide prompts for performing behaviors related to the solution, reinforcement for successful task completion, and punishment for noncompliance. Occasionally, a home behavioral contract may be useful if reinforcement is needed for a number of solutions. Prompts must be salient and timely because the natural distractibility and forgetfulness that are part of ADHD make it difficult for the teenager to remember effortful tasks; for example, if the adolescent needs to remember to take the trash out on Tuesday and Thursday evenings, the mother might post a bright sign as a reminder earlier those afternoons and give one verbal reminder as the evening begins. Figure 22.3 illustrates a completed worksheet for a problem with chores.

Problem-solving skills are taught through the use of instructions, modeling, behavior rehearsal, and feedback. The therapist briefly introduces problem solving at the beginning of this phase of treatment and helps the family select an issue of moderate intensity for discussion. Moderate-intensity issues are better than hot issues in the early stages of training because the family can concentrate on skills acquisition without excessive anger. The therapist gives instructions and guides the family to rehearse each step of problem solving. As family members demonstrate each problem-solving behavior, the therapist gives them feedback, successively approximating criterion responses by prompting them to restate their point in an improved fashion. To facilitate completion of the discussion, negative communication is interrupted and redirected rather than corrected.

At the end of the session, the family is asked to implement the solution at home and report back to the therapist during the next session. If the solution was effectively implemented, the therapist praises the family and begins a new problem-solving discussion. Otherwise, the reasons for failure are analyzed, and the problem is again discussed to help the family members reach a more effective agreement. The generalization of problem solving is programmed by having the family establish a regular meeting time during which problem solving is applied to accumulated complaints. The therapist stays with Step 9 until the family has successfully problem-solved and implemented solutions for at least two issues.

Step 10. Improving Communication

Step 10 consists of helping the family members improve their communication. This step often takes 2-3 therapy sessions. The therapist introduces communication training by distributing a copy of Figure 22.4 and reviewing these common negative communication patterns on the left side of the handout with the family. The therapist asks the parents and the adolescent to recall recent incidents of any negative communication habits that apply to them. The review of the incidents identifies who said what to whom and the impact on the listener, as well as on the relationship between the speaker and the listener. The therapist is careful to note how negative communication not only produced bad feeling and a counterattack but also sidetracked the discussion away from effective problem solving. Thus, the hurtful

Name of family: The Joneses Date: 11/25/05

Topic: Household Chores

Definitions of the problem:

Mom: "I get upset when I have to tell Allen 10 times to take out the trash and clean up his room."

Dad: "It bothers me to come home and find the trash still in the house and Allen's CDs and books all over the family room, with my wife screaming at him."

Allen: "My parents tell me to take out the trash during my favorite TV show. They make me clean up my room when all my friends are out having fun."

Solutions and evaluations:

	Mom	Dad	Allen
1. Do chores the first time asked	+	+	−
2. Don't have any chores	−	−	+
3. Grounded for 1 month if not done	−	+	−
4. Hire a maid	+	−	+
5. Earn allowance for chores	+	+	+
6. Room cleaned once—by 8 P.M.	+	+	+
7. Parents clean the room	−	−	+
8. Close the door to room	+	−	−
9. Better timing when asking Allen	+	+	+
10. One reminder to do chores	+	+	+

Agreement: Nos. 5, 6, 9, 10

Implementation plan: By 9 P.M. each evening Allen agrees to clean up his room, meaning books and papers neatly stacked and clothes in hamper or drawers. Doesn't have to pass "white glove test." Will earn extra $1.00 per day on allowance if complies with no reminders or one reminder. By 8 P.M. on Tuesdays, Allen agrees to have trash collected and out by curb. Will earn $2.00 extra if complies. Punishment for noncompliance: grounding for the next day after school. Dad to monitor trash; Mom to monitor room.

FIGURE 22.3. Example of a completed problem-solving worksheet.

effects of negative communication are identified, and the reciprocal escalation of negative interchanges can be highlighted. Any examples that occur during the session become prime material for discussion.

Since the therapist has been working with each family for several months by the time they get to Step 10, the most salient negative communication habits are apparent. If the family does not identify these patterns, the therapist brings them up.

Next, the therapist points out the alternative, more constructive methods for communicating negative affect, disagreement, or criticism, or generally telling another person that his or her behavior is unacceptable; these are on the right-hand side of Figure 22.4. Family members are asked to rehearse specific positive communication interchanges that apply to them. The therapist is careful to emphasize that he or she is not urging family members to suppress their feelings and hide their anger, but rather to express their legitimate affect with intensity but nonhurtful specificity.

Following this overview of communication skills, the therapist pinpoints one or two negative communication patterns per session and intervenes to change them. Whenever the negative pattern occurs, the therapist directly stops the discussion, gives feedback about the occurrence of the negative communication, and asks the family to "replay the scene" using more constructive communication responses. Such corrections

Check if people in your family do this:	*More positive way to do it*:
1. ____ Call each other names.	Express anger without hurtful words.
2. ____ Put each other down.	"I am angry that you did _____."
3. ____ Interrupt each other.	Take turns; keep it short.
4. ____ Criticize all the time.	Point out the good and bad.
5. ____ Get defensive when attacked.	Listen carefully and check out what you heard—then calmly disagree.
6. ____ Give a lecture/big words.	Tell it straight and short.
7. ____ Look away, not at speaker.	Make good eye contact.
8. ____ Slouch or slide to floor.	Sit up and look attentive.
9. ____ Talk in sarcastic tone.	Talk in normal tone.
10. ____ Get off the topic.	Finish one topic, then go on.
11. ____ Think the worst.	Keep an open mind. Don't jump to conclusions.
12. ____ Dredge up the past.	Stick to the present.
13. ____ Read the others' mind.	Ask the others' opinion.
14. ____ Command, order.	Ask nicely.
15. ____ Give the silent treatment.	Say it if you feel it.
16. ____ Throw a tantrum, "lose it."	Count to 10; take a hike; do relaxation; leave room.
17. ____ Make light of something serious.	Take it seriously, even if it is minor to you.
18. ____ Deny you did it.	Admit you did it, but say you didn't like the way you were accused.
19. ____ Nag about small mistakes.	Admit no one is perfect; overlook small things.

Your "Zap Score" (total no. of checks) _____

FIGURE 22.4. Family handout on negative communication.

are frequent during this phase of intervention. To be effective, the therapist must wield a "velvet sledgehammer"—coming down consistently on each instance of the inappropriate behavior, but landing with aplomb. To program generalization, the family is assigned homework to practice positive communication skills in daily interchanges and at family meetings. Family members are taught how to correct each other's communication without spurring excessive antagonism, extending the "velvet sledgehammer" approach to the home. Over several sessions the therapist helps the family change their most salient negative communication patterns.

Experience has suggested that the use of problem-solving and communication training in families of adolescents with ADHD involves a number of special considerations. First, the therapist must maintain the adolescent's attention during crucial moments of each session—not a trivial task for many teens with ADHD. Keeping comments brief, bringing the adolescent into the discussion at crucial moments while addressing the remainder of the comments to the parents, and talking in an animated manner are three useful hints for the therapist.

Second, some younger (12- to 14-year-old) adolescents with ADHD are not able to understand the concepts of problem solving or may not be ready emotionally and/or developmentally to assume responsibility for generating and negotiating solutions. In such cases, the therapist can rely more on having the parents use the contingency management techniques taught earlier in the intervention, mainly, consulting the adolescent about the choice of reinforcers.

Third, family members with ADHD may have such "short fuses" because of their deficits in behavioral inhibition that they often explode at each other during the sessions. The therapist should see Robin and Foster

(1989, pp. 219–221) for advice on maintaining session control—interrupting "runaway chains" as soon as they start, establishing nonverbal cues for "having the floor," teaching anger control and relaxation techniques, and being as directive as necessary to control the session.

Fourth, adolescents with ADHD can be so impulsive and distractible that their parents feel the need to correct everything they do or say, creating an endless series of issues and negative communication patterns. Such adolescents are not typically aware of how their behavior "drives their parents up a tree," and they react strongly, spurring endless conflict. The therapist must build on the advice given during the earlier ADHD family education and beliefs/expectations phases of treatment: The parents must realize that the adolescent did not choose to be this way and cannot help some of the forgetful, counterintuitive behavior. Parents need to learn to "pick their issues wisely," deciding on what to take a stand and what to ignore. For example, fidgety/restless adolescent behavior during family discussions is best reframed as the result of a biological tendency and then ignored, rather than treated as "another sign of disrespect for authority." Fifth, in cases where negative communication is so severe that problem solving is impossible, the therapist may choose to do Step 10 before Step 9. First working on negative communication prepares such families for problem solving.

Step 11. Putting It All Together

Parents and adolescents attend the Step 11 sessions together. Step 11 typically lasts one to two sessions. During this step the therapist briefly reviews the principles of parenting an adolescent with ADHD and all of the contingency management, cognitive restructuring, and problem-solving–communication interventions that the family has implemented. Family members are praised for successfully implementing a variety of interventions and developing reasonable beliefs and expectations. In the case of interventions that are not working well, the therapist helps the family modify them to improve implementation. The therapist assesses the extent to which the family has integrated contingency management, problem solving, and communication training into their daily routines, making suggestions to facilitate further such generalization. The synergy of these behavioral interventions and ADHD medications is stressed. When significant obstacles, such as marital discord or comorbid psychiatric disorders in either the parent or adolescent, limit the effectiveness of family interventions, referrals are made to other professionals for marital therapy or individual cognitive-behavioral therapy. The therapist ends Step 11 on a positive note, stressing the positive changes that the family made over the course of therapy.

EFFECTIVENESS OF THIS INTERVENTION

These therapeutic interventions have been subjected to empirical scrutiny in two outcome studies. In the first study, Barkley, Guevremont, Anastopoulos, and Fletcher (1992) compared behavioral management training (BMT), problem-solving–communication training (PSCT), and structural family therapy (SFT) with sixty-one 12- to 18-year-old adolescents with ADHD and their parents receiving eight to 10 sessions of one of these treatments. All three treatments resulted in significant mean group improvements on most measures from before to after treatment, with further gains in many cases from posttreatment to follow-up. Clinical significance analyses showed that 10 to 24% of the families made reliable changes and moved into the normal range on the dependent measures; there were no systematic differences across treatment conditions. In the second study, Barkley, Edwards, Laneri, Fletcher, and Metevia (2001) attempted to increase the efficacy of the family-based treatments by doubling the length of treatment and combining two of the treatments, BMT and PSCT. Ninety-seven teens with ADHD and ODD, ages 12–18, and their parents were assigned to receive 18 sessions of either PSCT alone or nine sessions of BMT followed by nine sessions of PSCT (BMT/PSCT). The latter condition is closest to the intervention described in this chapter and published in the *Defiant Teens* manual (Barkley & Robin, 2014). Both of the treatment conditions demonstrated significant improvements on mean group ratings of parent–teen conflict, the number and anger intensity level of specific disputes, and conflict tactics, as reported by mothers, fathers, and adolescents.

Interestingly, there was a highly significant differential rate of dropout from the two treatment conditions. At midpoint, 23% of the families receiving PSCT versus 8% of those receiving BMT/PSCT had dropped out; at posttreatment assessment, 38% of those receiving PSCT versus 18% of those receiving BMT/PSCT dropped out. At follow-up, 46% of the families in PSCT condition versus 23% of those in the BMT/PSCT condition failed to attend this assessment. Clinical significance measures showed that at posttreatment assessment, a maximum of 20–24% of each treatment

group showed reliable changes, depending on the measure and source of information; there were no differences between groups on these measures. The percentages of families within the normal range were also computed. At posttreatment, as reported by mothers, 34–78% of the families were within the normal range; as reported by fathers, 25–91% of the families were within this range.

Taken together, these two studies demonstrated that BMT and PSCT, alone or in combination, can help some families with teens who have ADHD/ODD improve their relationships. However, high percentages of the families did not make clinically significant changes and/or move from the abnormal to the normal range on the dependent measures. In Study 1, 80–95% of the families did not make clinically significant changes. In Study 2, with double the length of therapy, 76–80% of the families failed to make reliable changes, and 22–64% were still in the abnormal range at the end of the study. These results are sobering for the clinician but must be understood in their proper context:

1. Medication was not standardized in these studies, and many of the adolescents were not taking medication; thus, the treatment groups in these studies might be likened to the behavioral intervention alone group in the Multimodal Treatment Study of ADHD (MTA) with younger children (MTA Cooperative Group, 1999), which produced much less positive treatment outcomes than the medication alone or the medication plus behavioral intervention group.
2. Traditional family therapy produced even lower rates of clinically significant changes than either BMT or PSCT (Barkley et al., 1992).

In addition, these two studies used strictly manualized versions of BMT and PSCT. There is no research testing the combined and expanded versions of the intervention discussed in this chapter.

CONCLUSION

We (Barkley & Robin, 2014) have enhanced the manualized version of the *Defiant Teens* intervention used in the earlier research with many of the suggestions included in this chapter. The enhancements include but are not limited to (1) including the adolescent in approximately every other session throughout the intervention, (2) contemporary psychoeducation with the adolescent at the start of the intervention, including cognitive restructuring to address the concerns that cause adolescents to drop out of treatment, (3) contemporary psychoeducation with the parents about the executive functioning model of ADHD and adolescent development, (4) providing principles for parenting an adolescent with ADHD and incorporating discussion of these principles into every stage of intervention, (5) moving cognitive restructuring designed to foster realistic expectations for parenting teens with ADHD (parents) and realistic expectations for dealing with parents (adolescents) from later to earlier in the intervention, and (6) helping teenagers benefit from medication by preparing them for it, defining target behaviors, and speaking with their physicians. Research is needed to evaluate the effectiveness of this enhanced version of the family intervention and the contribution of each component to positive outcomes.

In addition, different stages of adolescent development for the teen with ADHD may require different variations and combinations of family intervention and individual cognitive-behavioral therapy. For example, 17- to 19-year-old adolescents about to leave their families and go to college, as well as those who attempted but failed to succeed away from home at college and have now returned home, each require very specialized forms of intervention that differ from the version discussed here. Preliminary evidence suggests that a downward extension of the evidence-based cognitive-behavioral therapy programs used for adults with ADHD may benefit adolescents with ADHD (Antshel, Faraone, & Gordon, in press). Perhaps such interventions will prove useful with older adolescents and emerging adults who have ADHD. Research is sorely needed to provide evidence-based interventions for families trying to launch emerging adults with ADHD into the world of higher education and work. In conclusion, clinical practice suggests that adding the enhancements discussed in this chapter to the *Defiant Teens* program (Barkley & Robin, 2014) may improve the clinical significance of the outcomes for many adolescents with ADHD and their parents.

KEY CLINICAL POINTS

- ✓ Adolescents with ADHD can be expected to have increased family conflicts as a consequence of the weaknesses in inhibition, attention, and self-regulation

(executive functioning) that accompany the disorder, making it less likely that they can successfully meet age-appropriate standards for compliance, independence, and self-responsibilities.

- ✓ These conflicts will be heightened in cases in which ODD and/or CD may be present, given the social conflicts inherent in these comorbid disorders and their greater likelihood of arising from disrupted parenting, intrafamily conflicts, and parental psychopathology.
- ✓ The foregoing factors combine with the natural inclination of adolescents to seek individuation from their parents, greater self-determination in matters affecting them, closer identification with peers (some of whom may be deviant or antisocial), and less time under parental supervision—all of which may pose issues ripe for parent–teen conflict.
- ✓ This intervention for adolescents with ADHD therefore strives to (1) educate parents and teens on the nature of ADHD; (2) provide parents with a set of principles to guide them in coping with their adolescent who has ADHD; (3) foster realistic beliefs and expectations in parents and teenagers for change in their relationships; (4) prepare adolescents and their parents to benefit from medication; (5) break the cycle of parent–teen negativity through the use of one-on-one time, praise, and ignoring minor misbehavior; (6) teach parents the effective use of positive incentive and punishment contingencies; (7) teach parents and teenagers to negotiate disagreements following the steps of problem solving; and (8) help families improve communication by targeting specific negative communication habits.
- ✓ Research using the original version of this intervention indicates that a combination of BMT and PSCT does reduce conflict between teens with ADHD and their parents, but the effects are modest, reliably helping approximately 25% of the families. However, medication was not maximized, and many of the adolescents in those studies were not taking medication. As yet, there is no research on the effectiveness of a combination of medication and the psychosocial intervention or the modifications to the original intervention discussed in this chapter. Such research is sorely needed.

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CHAPTER 23

Social Skills Training for Youth with ADHD

Amori Yee Mikami

Although the diagnostic criteria for attention-deficit/hyperactivity disorder (ADHD) focus on the presence of inattentive and/or hyperactive–impulsive symptoms (see Chapter 2), problems in peer relationships are extremely common for many youth with this condition (Chapter 8). These peer impairments are significant, persistent, and highly impairing, underscoring the importance of finding efficacious treatments for social difficulties. In this chapter, I first review the common types of social impairments faced by youth with ADHD, then present evidence that social problems can be refractory to established interventions for ADHD symptoms (pharmacotherapy, behavioral management). Social skills training (SST) as a treatment option is next discussed, including a critical review of its efficacy to date for children with ADHD. I then present hypotheses about two potential shortcomings of traditional SST as it has historically been delivered and speculate on ways to address these shortcomings in order to make SST more useful in ADHD populations. The chapter concludes with some illustrative examples of ways SST has been modified in recent years and key future directions for SST interventions.

SOCIAL PROBLEMS IN ADHD

Social impairment in youth with ADHD, as shown in Chapter 8, can manifest itself in multiple ways, such as displays of poor social behavior and social-cognitive deficits, and/or being poorly regarded by peers (Dirks, Treat, & Weersing, 2007). Although one category of problem may be related to another category of problem (e.g., poor social behavior may lead to a child being poorly regarded by peers), these are three distinct aspects of social impairment that have not always been considered separately in existing literature (Dirks et al., 2007). However, distinctions between types of social problems are important because, as will be discussed, they may necessitate different interventions. As well, interventions may have varying efficacy depending on what type of social problem is considered to be the outcome measure.

Social Behavior

Definition and Measurement

Social impairment in youth with ADHD often takes the form of poor social behaviors in peer interactions. Social behaviors are typically assessed by asking parents and/or teachers to read a description of a behavior, then indicate whether the child engages in that behavior rarely, sometimes, or often, for example (Gresham & Elliott, 2008). Observers may also be trained to watch the child in peer interactions for predetermined time intervals, then indicate the child's display of key social behaviors during each interval (Abikoff, Martin,

& Gittelman, 1985); this method of assessing social behavior is common among researchers, but it may be less practical for clinicians.

Pertinence for Youth with ADHD

Youth with ADHD may show compromised social behaviors because of either the presence of negative behavior or the absence of positive behavior, and it is important to distinguish between these two situations. Specifically, many children with ADHD display negative social behaviors that are disruptive and offensive to peers. These disruptive/offensive behaviors may be related to the core symptoms of ADHD (Landau, Milich, & Diener, 1998). In particular, hyperactive–impulsive symptoms can lead the child to have trouble waiting in line or taking turns in a game, to intrude into peers' ongoing activities, or to say things to peers without thinking. Comorbidities common with ADHD (e.g., oppositional defiant disorder [ODD] or conduct disorder [CD]) can lead to the child displaying aggressive behaviors in social situations (Gresham, MacMillan, Bocian, Ward, & Forness, 1998).

However, the lack of positive, prosocial/socially skilled behavior in youth with ADHD may also be related to the core symptoms of ADHD (Landau et al., 1998). Inattention can lead the child with ADHD to miss social cues that a peer is bored, so the child fails to ask the peer if he or she wants to play something else. Or, because of inattention, the child may seem disengaged with peers. Again, comorbidities common with ADHD (e.g., anxiety or depressive disorders) can further contribute to the child displaying a lack of interest in, or empathy for, peers (Mikami, Ransone, & Calhoun, 2011).

A large body of research has documented the presence of disruptive/offensive behavior as well as deficiencies in prosocial/socially skilled behavior in elementary school–age children with ADHD (Chapter 8). One of the earliest observational studies on this topic paired boys in a "space flight" task, in which one boy (the mission control) had to give instructions to the other (the astronaut). Boys with ADHD, relative to comparison boys, were observed to make significantly more off-task and intrusive comments and to register disagreement with their partner more often; as well, they were less able to attend to their partner's needs (Whalen, Henker, Collins, McAuliffe, & Vaux, 1979). These behavioral deficiencies appear quickly. One study revealed that as early as the first day of meeting unfamiliar peers in a summer program, boys with ADHD were observed to show more aggressive, noncompliant, and rule-violating behaviors, as well as some differences in prosocial behaviors such as less social contact, fewer leadership qualities, and lack of conflict mediation (Erhardt & Hinshaw, 1994). In addition, a vast body of research demonstrates that both parents and teachers tend to rate children with ADHD as showing more disruptive/offensive behavior (e.g., impatience, aggression, being a sore loser) and less prosocial/socially skilled behavior (e.g., less helpfulness) than their typically developing peers (see review in Gardner & Gerdes, in press; Hoza, 2007).

However, some research suggests that differences between children with ADHD and typically developing children may occur more in the presence of disruptive/offensive social behavior and less in the absence of prosocial behavior. Interestingly, in the study by Erhardt and Hinshaw (1994), boys with ADHD were more consistently observed to display more negative behaviors but do not differ in the amount of prosocial interactions than comparison boys. This finding has been replicated in more recent work. In a study using an online chat room, children with ADHD gave an equivalent number of compliments to peers as typically developing children, but children with ADHD also made hostile and insensitive comments, whereas typically developing children did not (Mikami, Huang-Pollock, Pfiffner, McBurnett, & Hangai, 2007). As well, Ronk, Hund, and Landau (2011) found that boys with ADHD demonstrated twice as many attention-getting strategies (disrupting the ongoing activity, talking about themselves) as typically developing boys in a play situation. However, children with ADHD and typically developing children did not differ in their use of socially competent, prosocial strategies (Ronk et al., 2011).

Social Cognition

Definition and Measurement

Deficiencies in social cognition—or the thought processes we use to understand and relate to other people—constitute another type of social impairment for many youth with ADHD (Uekermann et al., 2010). Social cognition is typically assessed by asking youth to self-report on their perceptions or interpretations of interpersonal events. For instance, children might hear a hypothetical story involving an interaction with a peer, then be asked to report their interpretation of the peer's

behavior, how they might handle the situation, what their social goals would be in such an interaction, or what emotions the peer is experiencing (Sibley, Evans, & Serpell, 2010).

Pertinence for Youth with ADHD

Youth with ADHD display several types of social-cognitive deficits. One example pertains to a prominent model of social cognition known as the social information-processing model (Crick & Dodge, 1994; Dodge, 1993), which holds that in order to respond effectively in situations involving peer conflict, children must first accurately interpret peers' intentions, then generate effective solutions. Aggressive children, by contrast, tend to attribute hostile intentions to peers' ambiguous actions and to generate less effective solutions to social problems (Dodge et al., 2003). Although the social information-processing model has predominantly been applied to understanding social-cognitive deficits in aggressive children, Matthys, Cuperus, and Engeland (1999) found that children with ADHD (regardless of whether they also displayed aggressive behavior) generated fewer adaptive responses when presented with hypothetical situations involving ambiguous peer provocation, although they did not differ from typically developing peers in their interpretations of peers' intentions. Similar results were found in a female sample; girls with ADHD generated more aggressive, ineffective solutions to hypothetical peer conflicts, whereas typically developing girls generated more negotiation strategies (Thurber, Heller, & Hinshaw, 2002). Another social-cognitive deficit may concern the goals of children with ADHD in peer interactions. Melnick and Hinshaw (1996) found that boys with ADHD, relative to comparison boys, expressed having social goals prioritizing attention seeking at the expense of rules and fairness, although Thurber and colleagues (2002) did not find this result among girls with ADHD.

A third type of social-cognitive deficit pertains to the difficulty that children with ADHD have in comprehending the key aspects of stories, particularly in understanding why a character performed a certain action, and in inferring cause-and-effect relationships between story events (Lorch, Milich, Astrin, & Berthiaume, 2006; Lorch, Milich, & Sanchez, 1998). Although story comprehension deficits (and associated working memory problems) have been most commonly postulated to pertain to the academic difficulties of children with ADHD, some researchers have theorized that these same deficits may also explain the social impairment of children with ADHD. Specifically, if children with ADHD cannot comprehend the causal structure of events in a story, they presumably also have more difficulty predicting (and understanding) why a peer would act in a certain fashion (Sibley et al., 2010).

Furthermore, there is evidence that children with ADHD may have difficulty understanding peers' emotions and perspectives. Marton, Wiener, Rogers, Moore, and Tannock (2009) reported that children with ADHD were rated by their parents as having less empathy for peers than did typically developing children; however, this result was accounted for by the comorbid ODD and CD in children with ADHD. Nonetheless, on interviews coded by observers in which children were presented with hypothetical vignettes involving conflicts with peers, children with ADHD displayed poorer levels of perspective taking and accurate identification of the peer's feelings relative to typically developing children, and these group differences persisted after taking into account the role of language abilities, cognitive functioning, and ODD/CD (Marton et al., 2009). In another study, boys with ADHD who were presented with fictional stories made more errors in identifying the correct emotion in the characters than did typically developing peers (Braaten & Rosen, 2000).

A final social-cognitive problem for children with ADHD is the tendency to perceive themselves as *not* having social deficits, a phenomenon known as "positive illusory bias" (see review in Owens, Goldfine, Evangelista, Hoza, & Kaiser, 2007). For instance, one study paired children with ADHD and typically developing children together for an interactive task; subsequently children were asked to self-report how socially skilled they thought their behavior had been. An interesting pattern emerged: Self-perceptions of social skills were much higher in children with ADHD than in typically developing children, yet observers, kept unaware of children's diagnoses, reported the opposite—that children with ADHD appeared much less socially competent than their typically developing peers (Hoza, Waschbusch, Pelham, Molina, & Milich, 2000). A similar pattern has been documented in other studies. Children with ADHD perceive themselves to be equally (or better) liked by peers than do to typically developing children, yet parents and teachers rate typically developing children to be far better liked by peers than children with ADHD (Hoza, Pelham, Dobbs, Owens, & Pillow, 2002).

Peer Regard

Definition and Measurement

This category of social impairment refers to peers' affective judgments about children with ADHD. The ideal way to assess peer regard is to use a sociometric procedure whereby classroom peers nominate the children they like, dislike, and consider to be friends (Coie, Dodge, & Coppotelli, 1982). Scores calculated for the child with ADHD indicate the proportion of peers who said that the child was someone they liked (often referred to as "peer acceptance") or disliked (often referred to as "peer rejection"). If a peer nominates the child with ADHD as a friend, then it is possible to determine whether the child also independently nominated the peer as a friend; if so, the friendship is considered to be mutually reciprocated (Parker & Asher, 1993). Friends may also be asked about the quality of the friendship, or the manner in which two friends interact may be observed (Mikami, 2010). Although asking peers themselves about their affective judgments is the ideal way to measure peer regard, it can be time consuming and impractical, especially for clinicians. An alternative is to ask the teacher to estimate the proportion of classroom peers who like and dislike the child, and the number of friends the child has; such an approach has been shown to be associated with sociometric measures (Dishion & Kavanagh, 2003).

Pertinence for Youth with ADHD

Children with ADHD are extremely disliked by peers (Hoza, 2007), with some studies indicating that 60–80% of children with ADHD score one standard deviation above their classroom mean (e.g., in the top 16% of their class) in the number of peers who nominate them as someone they dislike (Hoza, Mrug, et al., 2005; Pelham & Bender, 1982). As well, children with ADHD are significantly less likely to be named by peers as someone they like (Hoza, Mrug, et al., 2005). Peers' negative judgments of children with ADHD occur very quickly—within the first few hours of meeting (Erhardt & Hinshaw, 1994; Mikami, Jack, Emeh, & Stephens, 2010), and once established, poor peer regard tends to be stable over time (Blachman & Hinshaw, 2002).

Although children who are well liked by peers also tend to have many friends, friendship and peer acceptance are distinct constructs. For instance, it is possible for a child to be generally disliked by peers yet still have a reciprocated friend (Parker & Asher, 1993). However, children with ADHD are about half as likely as their classmates to have friendships (Blachman & Hinshaw, 2002; Hoza, Mrug, et al., 2005). Some studies indicate that up to 80% of children with ADHD and comorbid conduct problems, in fact, do not have a single reciprocated friend (Gresham et al., 1998). Furthermore, any friendships that children with ADHD do have may be less stable and of lower quality (e.g., less warmth, more conflict) than the friendships of typically developing children (Blachman & Hinshaw, 2002; Normand et al., 2011).

Comorbidity, Gender, and Subtype Issues

Approximately half of children with ADHD have comorbid ODD or CD (Jensen, Martin, & Cantwell, 1997), and the presence of these co-occurring conditions is detrimental for positive social functioning (Gresham et al., 1998; Pfiffner, Calzada, & McBurnett, 2000). Children with ADHD and comorbid ODD and CD display more disruptive/offensive behaviors than do children with ADHD alone (Gresham et al., 1998). Comorbid ODD and CD may also be associated with exacerbated problems in social cognition. In the study by Matthys and colleagues (1999), children with ADHD and comorbid ODD or CD showed additional deficits in selecting aggressive responses, above and beyond the deficits in generating effective solutions shown by children with ADHD, regardless of ODD comorbidity. In the study by Melnick and Hinshaw (1996), the tendency to have maladaptive goals in social situations was present in children with ADHD alone, but it was magnified for those with comorbid aggression. Comorbid ODD or CD was also found to account for incremental decreases in peer regard (more disliking, less liking) in the study described earlier by Hoza, Mrug, and colleagues (2005), although these results were no longer significant once a correction for multiple comparisons was applied. To summarize this literature, one meta-analysis reported that the average effect size of peer problems for children with ADHD relative to typically developing youth was $d = 0.72$ if children with ADHD had no comorbidities, but it was $d = 1.25$ if they had comorbid ODD/CD symptoms (Waschbusch, 2002).

Although around one-third of children with ADHD are estimated to have a comorbid anxiety disorder, this co-occurring condition has been less studied relative to ODD or CD (Pfiffner et al., 2000). Some research suggests that children with ADHD and comorbid anxiety

tend to have fewer prosocial/socially skilled behaviors, as rated by parents and teachers, than do children with ADHD and no comorbid anxiety (Mikami et al., 2011; Pliszka, 1992). In the study described earlier by Hoza, Mrug, and colleagues (2005), children with ADHD and comorbid anxiety appeared to have slightly fewer friendships than children with ADHD and no anxiety; however, this was the only group difference found (out of 19 peer relationship variables tested) and it did not survive a correction for multiple comparisons.

Regarding gender, the literature is mixed in terms of whether boys or girls with ADHD have greater social impairment. The results of a meta-analysis reported no gender differences overall in peer problems (Gershon, 2002). However, in community samples, boys may have more peer impairment than girls, but the opposite may be true in clinical samples (Carlson, Tamm, & Gaub, 1997; Gaub & Carlson, 1997; Gershon, 2002). Patterns of comorbid aggressive behavior may account for these differences. In community samples, boys with ADHD are more likely than their female counterparts to have comorbid aggressive behavior, and the presence of aggression is strongly associated with being disliked by peers. Yet evidence suggests that when girls with ADHD present with equivalent aggression to that found in boys (e.g., in clinical samples), their peer relationships are worse than those of boys—perhaps because aggression is considered to be more socially deviant in female peer groups (Mikami & Lorenzi, 2011). A similar effect may occur regarding comorbid anxiety; the negative impact of anxiety on peer regard may be more pronounced for girls than for boys with ADHD (Becker, McBurnett, Hinshaw, & Pfiffner, 2013).

Research also indicates that children with the combined type (ADHD-C) and the hyperactive–impulsive type of ADHD (ADHD-HI) are more likely to show disruptive/offensive behavior, to be disliked by peers, and to have comorbid ODD/CD than are children with the inattentive type of ADHD (ADHD-I; Carlson & Mann, 2000). By contrast, children with ADHD-I may show specific deficits in prosocial, socially skilled behavior (Andrade & Tannock, 2013). In a recent study of children's play behaviors, researchers found that children with ADHD-C and ADHD-HI displayed more mischief, clowning, and rule breaking than did children with ADHD-I; however, children with ADHD-I found it more difficult to become engaged with peers (Cordier, Bundy, Hocking, & Einfeld, 2010). In another study, children with ADHD-I, but not those with ADHD-C, differed from typically developing children in that they spent more time alone or as onlookers of peers' activities (Hodgens, Cole, & Boldizar, 2000). Similarly, a study involving a chat room interaction task indicated that children with ADHD-I displayed more problems with passivity and missing subtle social cues than did children with ADHD-C (Mikami et al., 2007). (For the overlap of ADHD-I with the more recent concept of sluggish cognitive tempo/concentration deficit disorder [SCT/CDD] and its associated social problems, see Chapter 17.)

Social Problems across the Lifespan

ADHD in adolescence and adulthood has also been understudied relative to the childhood manifestations of the disorder (Weiss, Hechtman, & Weiss, 1999). However, all available evidence suggests that social problems persist across the lifespan (Barkley, Murphy, & Fischer, 2008). For instance, there are robust findings that parents and teachers rate adolescents with ADHD as having poorer social behaviors than do typically developing adolescents (Bagwell, Molina, Pelham, & Hoza, 2001; Hinshaw, Owens, Sami, & Fargeon, 2006), and that this remains true in young adulthood (Barkley et al., 2008).

As well, there are suggestions that adolescents with ADHD may continue to have deficits in social cognition. Similar to the results for children with ADHD (Matthys et al., 1999), Mikami, Lee, Hinshaw, and Mullin (2008) found that adolescent girls with ADHD generated more ineffective solutions to hypothetical vignettes of peer provocation but did not show impairments in the other aspects of the social information-processing model, such as attributing hostile intent to peers. Results remained the same regardless of whether comorbid ODD and CD were covaried (Mikami, Lee, et al., 2008). These findings were replicated in another adolescent sample, in which teenagers with ADHD offered poorer solutions to social problems than did typically developing peers but did not differ in their tendency to make hostile attributions for peers' ambiguous behavior (Sibley et al., 2010). Finally, similar to children, adolescents with ADHD have difficulties understanding cause-and-effect relationships in stories (Sibley et al., 2010), and present with positive illusory bias (Hoza, Murray-Close, Arnold, Hinshaw, & Hechtman, 2010).

Although in secondary school it becomes more difficult to collect sociometric measures of peer regard (because adolescents are switching classes and not with

a single peer group throughout the day), teachers rate adolescents with ADHD to be more disliked/rejected by peers and less liked/accepted by peers than are typically developing youth (Hinshaw et al., 2006). There is some evidence that adolescents with ADHD may be attracted to members of deviant peer groups who influence one another to engage in antisocial behaviors (Marshal, Molina, & Pelham, 2003). Problems with friendship quality for youth with ADHD also appear likely to persist as children with ADHD move into adolescence (Normand et al., 2013).

Romantic partnerships become an important, and new, component of peer relationships for young adults. Perhaps not surprisingly, initial evidence suggests that college students with ADHD have more conflict in their relationships with romantic partners (Canu, Tabor, Michael, Bazzini, & Elmore, in press), and that adults with ADHD are more likely to be divorced (Klein et al., 2012) than are typically developing individuals of similar age.

More longitudinal work is needed to understand better why social impairment persists across the lifespan for many individuals with ADHD. However, developmental theory suggests that children learn key social skills (e.g., empathy, perspective taking, conflict management) in their close friendships in elementary school (Pedersen, Vitaro, Barker, & Borge, 2007). In early adolescence, youth must display these social skills in order to make their close friendships progressively more intimate, and these experiences set the stage for the development of romantic relationships in adolescence and adulthood (Buhrmester, 1990). Therefore, children with ADHD who are deprived of positive peer interactions in elementary school miss out on important socialization experiences that would have predisposed them toward healthy interpersonal relationships in later years (Mikami, 2010).

Implications of Social Problems

Children with ADHD are at risk for various adverse outcomes in adolescence and adulthood (Hinshaw et al., 2006; Klein et al., 2012; Molina et al., 2009). However, if children have peer problems as well, the risk for adverse outcomes increases. Mrug and colleagues (2012) found that the extent to which children with ADHD were disliked by peers in elementary school predicted their increased depression, cigarette smoking, and overall global impairment 6 years later, after they statistically controlled childhood levels of these same problems and ADHD symptom severity. As well, teacher reports of the extent to which classroom peers disliked the participant in childhood were related to children's lack of prosocial, socially skilled behavior, which in turn exacerbated peer rejection, as well as poor adjustment in adolescence (Murray-Close et al., 2010). Other researchers similarly found that ADHD status and peer problems in childhood (as assessed by parent–teacher ratings of poor social behavior, as well as sociometric nominations of peer regard) had additive effects on increased substance abuse, delinquency, depression, academic failure, and eating pathology 5 years later (Greene, Biederman, Faraone, Sienna, & Garcia-Jetton, 1997; Mikami & Hinshaw, 2006; Mikami, Hinshaw, Patterson, & Lee, 2008).

Interestingly, there is conflicting evidence about whether the presence of a friendship buffers the negative effects of peer rejection on adjustment. Whereas some investigators have found this that it does (Bagwell, Newcomb, & Bukowski, 1998), notably, in other research, only peers' dislike of the child (and not the number of friendships) predicted adjustment problems 6 and 8 years later (Mrug et al., 2012). However, it is possible that the sheer presence of friendship may be less important than the quality of the relationship (Mikami, 2010; Normand, Schneider, & Robaey, 2007) given that we know children with ADHD are more likely to have more conflict, less closeness, and less stability in any friendships they do have (Blachman & Hinshaw, 2002; Normand et al., 2011).

Less is known about the extent to which social-cognitive deficits in childhood may contribute to adjustment problems in adolescence or adulthood. However, research with community samples (not specific to children with ADHD) suggests that hostile attributions of peers' ambiguous behavior can lead to peer rejection and, ultimately, to increases in aggressive social behavior toward peers in a vicious cycle over time (Dodge et al., 2003; Lansford et al., 2006). Other work on positive illusory bias indicates that overly inflated self-perceptions may be detrimental for children with ADHD because children who fail to accept that they have deficiencies are not motivated to engage in psychosocial treatments (Mikami, Calhoun, & Abikoff, 2010).

Again, the mechanisms by which childhood peer problems result in exacerbated maladjustment are unknown, but it is thought that children with poor peer relationships are deprived of important socialization opportunities to learn how to regulate negative emo-

tions or to deal with social stress and conflict—skills that may help to prevent delinquency or drug problems (Parker & Asher, 1987; Pedersen et al., 2007). Children with poor peer relationships may also be lonely (Boivin, Hymel, & Burkowski, 1995), which predisposes them to depression or anxiety. As well, they may disengage from school because of negative peer experiences there (Buhs, Ladd, & Herald, 2006), contributing to academic failure. Collectively, these findings underscore the importance of early identification, and treatment, of social problems in ADHD populations.

EFFICACY OF INTERVENTIONS FOR ADHD SYMPTOMS ON SOCIAL IMPAIRMENT

The first-line treatments for the core symptoms of ADHD are pharmacotherapy (see Chapter 27) and behavioral contingency management interventions, such as behavioral parent training (Chapter 21) and behavioral classroom management (Chapter 24) (Arnold et al., 1997). For instance, the Multimodal Treatment Study of Children with ADHD (MTA; MTA Cooperative Group, 1999a) is the largest clinical trial to date involving the population of children with ADHD, enrolling 579 children across six sites in the United States and Canada. In the MTA, the combination of intensively applied pharmacotherapy and behavioral management for 14 months, as well as pharmacotherapy alone, were most efficacious in reducing parent- and teacher-reported ADHD symptoms relative to treatment as usual in the community (MTA Cooperative Group, 1999a). The beneficial effects of pharmacotherapy and behavioral management persisted 10 months after treatment was discontinued (MTA Cooperative Group, 2004), although this was no longer the case at subsequent follow-up points (Jensen et al., 2007; Molina et al., 2009).

However, whereas both pharmacotherapy and behavioral management are documented to reduce children's inattention and hyperactivity–impulsivity, their efficacy for ameliorating social impairment appears to be weaker overall. Pharmacotherapy and behavioral management interventions may show the most robust effects on improving children's social behaviors—specifically, in reducing disruptive/offensive behavior (Chronis, Jones, & Raggi, 2006; Hinshaw, Henker, Whalen, Erhardt, & Dunnington, 1989; Pelham & Fabiano, 2008). These interventions may also increase prosocial, socially skilled behavior. In the MTA, for instance, the combination of pharmacotherapy and behavioral management maximally improved parent and teacher reports of social skills (MTA Cooperative Group, 1999a). It is worth noting, however, that effects on social skills did not persist at follow-up points once treatments were discontinued (MTA Cooperative Group, 2004).

Efficacy on other categories of peer impairment may be weaker. As reviewed by Uekermann and colleagues (2010), the efficacy of pharmacotherapy and behavioral management on social-cognitive deficits remains relatively unknown. Pharmacotherapy and behavioral management may have the least impact on the way peers regard children with ADHD. Few trials have assessed peers' impressions of children with ADHD as a dependent variable (as opposed to parent and teacher reports of social behaviors), in part because getting peer reports can be logistically challenging. Some studies indicate that pharmacotherapy (vs. placebo), or pharmacotherapy when added to behavioral management (vs. behavioral management alone), for children with ADHD may result in peers' increased liking (Pelham et al., 2000; Whalen et al., 1989). However, even in these trials demonstrating the success of pharmacotherapy, peer regard is far from normalized.

More disappointing are the treatment effects on peer functioning in the MTA; note that sociometric measures were obtained from 285 of the 579 children who participated in the original trial (Hoza, Gerdes, et al., 2005). None of the treatment conditions administered as part of the MTA differentially impacted peer sociometric acceptance, rejection, or reciprocated friendship once a correction for multiple comparisons was applied. Rather, children with ADHD remained quite lacking in terms of peer regard and friendships, regardless of what intervention they received. As noted by the study authors, it is surprising that first-line treatments for ADHD that were demonstrated to be efficacious for the core symptoms of this disorder and administered intensively under ideal conditions for 14 months largely failed to affect peers' regard (Hoza, Gerdes, et al., 2005).

HISTORY OF SOCIAL SKILLS TRAINING

Given that impaired peer relationships in children with ADHD appear quite resistant to established treatments for the core symptoms of ADHD (Mikami & Pfiffner, 2006), there is need for alternative interventions that target social problems. A popular option for

this purpose is SST, which has proliferated in the past 2 decades to address the needs of both children with ADHD and peer problems (Landau & Moore, 1991; Nixon, 2001) and children without ADHD who show social impairment (DuPaul & Eckert, 1994).

Rationale

The rationale for SST is predicated on the assumption that some children lack the core skills to enact positive, prosocial behaviors in interpersonal situations. The reason why pharmacotherapy and behavioral management interventions may fail to optimize social functioning is that these interventions, by contrast, focus more on suppressing children's disruptive/offensive behavior as opposed to teaching them skills to enact positive behaviors (de Boo & Prins, 2007). Furthermore, even if behavioral management contingencies are used to increase children's motivation to display positive, prosocial behaviors, the theory is that it will be difficult or impossible for children to do so if they lack the basic skills to enact these behaviors (Gresham, Cook, Crews, & Kern, 2004).

Following this rationale, SST containing direct instruction (and practice) in how to enact prosocial, socially skilled behaviors may be warranted. Of note, SST may also contain direct instruction aimed at changing the child's cognitive biases, such as training the child to label peers' emotions correctly, to make more accurate interpretations of peers' intentions, or to generate more effective solutions to social problems. While traditional SST does not attempt to affect peer regard directly, the theory (which, admittedly, may be faulty, as I discuss below) is that if children with ADHD improve their social behaviors and reduce their social-cognitive deficits, peers should naturally respond by liking and befriending them (de Boo & Prins, 2007).

Description

SST curricula that have been employed with children with ADHD contain some common factors (Landau et al., 1998; Nixon, 2001). First, in traditional SST, the clinician provides the intervention directly to the child with ADHD. Although some clinicians deliver SST to children individually (often because of practical reasons), many clinicians prefer a group model, so that children can practice the skills with one another. Second, although there is variability among SST curricula, children commonly attend 60- to 90-minute sessions once per week for a prescribed time period, such as 8–12 weeks.

Third, SST topics of instruction typically focus on providing children with knowledge about (and often supervised in-session practice with) key social skills that they are believed to lack. A different social skill may be covered each week, and common skills taught in SST for children with ADHD are sharing, making conversation, joining new groups of peers, following rules while playing games, taking turns, calming down when upset, and identifying emotions (Pfiffner, 2008). Many clinicians introduce the social skills to children in a didactic fashion but also include discussion and role plays within session, so that children can practice the skills (and the clinician can correct children's behaviors and reinforce their practice). There is variability in the extent to which clinicians encourage generalization of socially skilled behaviors outside of the therapeutic context. Many traditional, clinic-based SST programs do nothing to encourage generalization, which has been referred to as a "train and hope" strategy (DuPaul & Eckert, 1994). However, even when generalization efforts are incorporated in traditional SST, they typically play a lesser role than the predominant focus of the treatment, which is in-session instruction of the child by the clinician.

EFFICACY OF SST ON SOCIAL IMPAIRMENT IN ADHD

Efficacy of SST for Social Behavior

The evidence is strongest that traditional, clinic-based SST for children with ADHD may improve children's demonstration of prosocial, socially skilled interactions while in the treatment group setting (see review in Pfiffner et al., 2000). One example is a study in which Fenstermacher, Olympia, and Sheridan (2006) reported that after receiving SST, children with ADHD demonstrated more correct social behaviors when tested in analogue role-play situations in session.

Nonetheless, the evidence is much weaker that SST leads children with ADHD to generalize improved social behaviors outside of the treatment group setting. For instance, Abikoff and colleagues (2004) conducted a study involving 103 children with ADHD treated with pharmacotherapy, then randomized to one of three conditions: (1) a group receiving intensive SST (with weekly meetings for 1 year, then monthly meetings for the second year); (2) an attention control group

in which children played with peers but received no instruction; or (3) no additional intervention. The SST was quite comprehensive; in addition to targeting key behaviors (getting along with others, conversational skills, navigating problem interpersonal situations), it involved several components intended to foster generalization, such as (1) out-of-session homework assignments; (2) instruction to parents and teachers about the social behaviors targeted in SST, with encouragement to parents and teachers to reinforce children's displays of these behaviors; and (3) inclusion of the targeted social behaviors on the Daily Report Card that teachers completed each day to inform parents about how the child behaved at school. Still, results suggested *no* adjunctive benefit of receiving SST on parent report, teacher report, or observations of children's behaviors in social interactions—either positive (prosocial/socially skilled) or negative (disruptive/offensive) behavior (Abikoff et al., 2004). Another study involving 120 children with ADHD (all of whom were receiving pharmacotherapy provided by study personnel), randomly assigned to receive SST in eight, 90-minute group sessions or to a no-treatment control group, similarly reported extremely few positive effects for parent and child self-report ratings of social skills (Antshel & Remer, 2003). The authors note that, for the most part, there were no demonstrated benefits of SST and possibly some detrimental ones; 15% of the children with ADHD-I got worse with treatment, perhaps due to peer contagion effects whereby being in SST groups with peers who had ADHD-C encouraged children with ADHD-I to display increased disruptive behavior.

The studies reviewed previously are useful because of the large sample sizes of clinically diagnosed participants with ADHD, the careful provision of intensive SST to participants in a randomized design, and the use of multiple informants to comprehensively assess changes in children's social behaviors as a result of the SST (Abikoff et al., 2004; Antshel & Remer, 2003). However, both studies have the limitation that all of the children were medicated and the SST was adjunctive to pharmacotherapy. Although it seems clear that, at the least, medication does not seem to facilitate the uptake of social skills during SST, this nonetheless raises the question of whether SST might be more efficacious for children with ADHD who are not receiving intensive pharmacotherapy. Still, it is worth noting that pharmacotherapy does not tend to normalize social interactions for children with ADHD to such a great extent that there is a ceiling effect (Hinshaw et al., 1989). As well, other studies have tested the efficacy of SST with unmedicated participants and similarly found few benefits of SST, especially when outcome measures were collected from informants kept unaware of the provision of SST, such as observers (see reviews in Abikoff, 1985; Pelham, Wheeler, & Chronis, 1998). Overall, a recent major review of psychosocial treatments for children with ADHD concluded that SST, at least as is provided in traditional clinic-based settings, is ineffective (Evans, Owens, & Bunford, 2014).

Efficacy of SST for Social Cognition

The majority of studies evaluating SST have used measures of children's social behaviors as the dependent variable, as opposed to social cognition. Relative to the larger number of findings documenting efficacy of SST on the social cognition of aggressive children (Lavallee, Bierman, & Nix, 2005; Prinz, Blechman, & Dumas, 1994), few studies have examined effects of SST on social cognition in children with ADHD. One exception is the study by Pfiffner and McBurnett (1997), who reported that 8 weeks of a traditional clinic-based SST group for children with ADHD resulted in children's increased ability to generate appropriate ideas about how to behave in key social situations. Children's increased knowledge may have generalized to improved behavior at home based on parents' reports (who were aware of treatment provision), but largely failed to generalize to the school context based on teachers' reports (Pfiffner & McBurnett, 1997).

Although it is unknown what type of intervention (if any) would successfully reduce the social-cognitive deficit of positive illusory bias in children with ADHD, it has been suggested that positive illusory bias would be unaffected by traditional clinic-based SST because SST requires children to accept the fact that they have social deficits in order for them to be open to instruction (Hoza & Pelham, 1995; Mikami, Calhoun, et al., 2010). Rather, positive illusory bias is thought to result from a defensive reaction that is exacerbated when children receive criticism about their weaknesses (see review in Diener & Milich, 1997; Owens et al., 2007). As such, SST is unlikely to increase the accuracy of children's self-perceptions.

Efficacy of SST for Peer Regard

Measures of peer regard (particularly sociometric measures) are difficult to collect, so it is perhaps unsurpris-

ing that they are rarely included in efficacy trials of traditional, clinic-based SST for children with ADHD. Therefore, the efficacy of SST on peers' actual regard remains largely unknown in ADHD populations. However, it is important to remember that the theory behind SST is that if children with ADHD are trained to increase their prosocial/socially skilled behavior and reduce their disruptive/offensive behavior, then peers will observe these changes and respond accordingly with better peer regard (de Boo & Prins, 2007; Ladd & Mize, 1983; Mize & Ladd, 1990). Given the failure of SST to result in demonstrated changes in the social behaviors of children with ADHD in naturalistic settings (Abikoff et al., 2004), it seems extremely unlikely that SST would change peers' regard.

More concerning, however, are questions regarding the assumption that behavior change on the part of the child with ADHD will directly result in improvements in peer regard (Mikami, Lerner, & Lun, 2010); the potential fallacy of this presumption is explored in greater detail below. Given that pharmacotherapy and behavioral management in the MTA resulted in some improvements in actual social behaviors, as rated by parents and teachers (MTA Cooperative Group, 1999a), yet failed to have effects on peer regard of the children with ADHD (Hoza, Gerdes, et al., 2005), SST may be unlikely to result in improvements in peer regard even if it impacts social behaviors. In summary, given that SST (at least as it is traditionally delivered in the clinic) already faces challenges in improving the social behaviors of children with ADHD, it seems unlikely that it would result in changes in peer regard.

Possible Moderators of SST: Gender, Subtype, Comorbidity, and Age

Despite the overall conclusion that traditional, clinic-based SST has limited efficacy, there are suggestions that children with ADHD-I may be more responsive to SST than children with ADHD-C or ADHD-HI (Pfiffner, 2003). In the study Antshel and Remer (2003), SST resulted in relatively greater improvements for children with ADHD-I relative to those with ADHD-C, specifically on the parent-reported social skill of assertion; although, notably, children with ADHD-I benefited most when they were in SST groups containing only peers with ADHD-I. As described in greater detail below, it is possible that the social deficits in children with ADHD-I (especially that subset having SCT/CDD, see Chapter 17) are of a different nature than those in ADHD-C and may be more responsive to SST.

Regarding comorbidity as a moderator, Antshel and Remer (2003) also suggested that children with ADHD and comorbid ODD may benefit less from SST than do children with ADHD and no disruptive behavior comorbidities. One possible reason for this finding is that children with ODD may be resistant to taking direction from any adult (including clinicians) about the appropriate ways to behave (Pfiffner et al., 2000). Children with ADHD and comorbid ODD are also more likely to have positive illusory bias; in other words, they are more likely to deny that they have impaired peer relationships (Hoza et al., 2004), and it has been suggested that because of positive illusory bias, children with ADHD are not motivated to change and are therefore resistant to any psychosocial treatment, including SST (Mikami, Calhoun, et al., 2010). On the other hand, there are suggestions that children with ADHD and comorbid anxiety, who have been studied less then children with ADHD and disruptive behavior comorbidities (Pfiffner, et al., 2000), may be more responsive than children with ADHD alone to psychosocial treatment in general, which would potentially include SST (Jensen et al., 2001; MTA Cooperative Group, 1999b; Schatz & Rostain, 2006).

There are some theoretical reasons why gender might moderate the efficacy of SST for youth with ADHD (de Boo & Prins, 2007). ADHD is approximately three times more common in boys than in girls. As reviewed earlier, evidence is mixed regarding whether boys and girls with ADHD differ in the severity of their peer problems (Gershon, 2002). However, strong findings in the developmental psychology literature suggest that, even from early ages, boys and girls tend to segregate by sex, have different patterns of interaction with their peers, and value somewhat different characteristics in friendships (Keenan & Shaw, 1997; Maccoby, 1990). Therefore, SST curricula may be most efficacious if they are gender-specific and tailored to the types of social rules and friendship skills that are valued by each sex. Nonetheless, available evidence does not suggest that the efficacy of SST for children with ADHD is moderated by child gender (MTA Cooperative Group, 1999b; Pfiffner & McBurnett, 1997).

As well, there may also be theoretical reasons why age would moderate the efficacy of SST for youth with ADHD. Although social impairment has been documented to persist across childhood, adolescence, and young adulthood for ADHD populations (Bagwell et

al., 2001; Canu et al., in press; Hoza, 2007), the developmental psychology literature suggests that children of different ages tend to value different social skills. For example, it is normative for friendships of younger children to be based on play and shared activities, while intimacy and companionship become progressively more important in the friendships of older children and adolescents, preparing adolescents and young adults to engage in romantic partnerships with their peers (Bagwell et al., 1998; Bukowski, Newcomb, & Hoza, 1987). As such, the needs of children and adolescents in SST may differ from one another. However, the field has few empirically supported interventions for adolescents and adults with ADHD in general (e.g., Evans, Serpell, Schultz, & Pastor, 2007; Sibley et al., 2011), and the available limited evidence has not supported the idea of age as a moderator of SST efficacy (Cook et al., 2008; de Boo & Prins, 2007; Gresham et al., 2004).

Summary

Traditional SST, at least the way it has been provided historically by clinician-led instruction and in-session practice of social skills, administered directly to children with ADHD, with a predominant focus on teaching the skills in session as opposed to generalization of skills to out-of-session interactions, has quite limited efficacy with this population (de Boo & Prins, 2007; Evans et al., 2014; Mrug, Hoza, & Gerdes, 2001; Pelham & Fabiano, 2008). In fact, for this reason, researchers in the last decade have moved away from testing traditional SST and moved into speculating about reasons why SST may not be efficacious, as well as testing modifications of SST that may be more helpful for children with ADHD.

It is worth noting that SST may be more efficacious for populations of children who are socially impaired, but do not have ADHD, relative to children with ADHD (Asher & Hymel, 1986; Beelmann, Pfingsten, & Lösel, 1994). For instance, two studies have documented the usefulness of SST for community samples of children selected for peer problems (Bierman & Furman, 1984; Mize & Ladd, 1990), although, notably, both studies found positive effects of SST on parent and teacher ratings of social behavior, but this did not translate into improved peer regard when the intervention involved providing traditional SST to the child alone.

Other research has documented benefits of SST for clinical populations of children who have disorders other than ADHD, although, again, it has proven to be predominantly efficacious in terms of improving displays of social behavior and inconsistently on social-cognitive deficits and peer regard (Spence, 2003). Specifically, SST may be useful for children with social anxiety (Spence, Donovan, & Brechman-Toussaint, 2000), aggressive behavior (Prinz et al., 1994; Webster-Stratton, Reid, & Hammond, 2004), and autism spectrum disorders (Rao, Beidel, & Murray, 2008). Notably, Antshel and colleagues (2011) reported that SST resulted in improvement on parent and teacher reports of social skills for children on the autism spectrum with no comorbidities or with a comorbid anxiety disorder but there were no beneficial effects of SST for children on the autism spectrum with comorbid ADHD. In fact, several reviews of SST (not specific to ADHD) have also concluded that positive effects of SST are most likely to be found among socially withdrawn children who have peer problems, and least likely to be found among children with ADHD symptoms (Beelmann et al., 1994; DuPaul & Eckert, 1994).

In short, there are suggestions that the population of children with ADHD may be especially resistant to traditional SST approaches, for reasons that are particular to the nature of ADHD (discussed below). This behooves the field to consider ways to modify SST to address the unique challenges faced by children with ADHD.

REASONS FOR DIFFICULTIES IN TREATING SOCIAL PROBLEMS IN ADHD

In this section I present arguments for two key issues that, in my opinion, are not given sufficient attention in traditional, clinic-based SST. I hypothesize that these two issues impede the efficacy of traditional SST interventions for children with ADHD.

Knowledge versus Performance Deficits

A key theoretical issue pertains to whether children with ADHD display deficient social behavior (e.g., presence of disruptive/offensive behaviors, lack of prosocial/skilled behaviors) because of a deficit in their knowledge or a deficit in their ability to carry out this knowledge (Gresham et al., 2004). In other words, is the problem that youth with ADHD do not *know* the appropriate social behaviors to do, or is the problem instead (or also) that they cannot *do* the appropriate social behaviors that they know? A growing body of

research suggests the latter. For instance, Maegden and Carlson (2000) found that children with ADHD-C and typically developing children scored similarly on a measure of social knowledge in which children were presented with hypothetical social scenarios and asked to report the correct way that someone should respond. This result suggests that there must be other factors impeding children's performance (e.g., actual enactment) of the accurate knowledge that they have about how one should behave in social situations.

Researchers have theorized about what these performance deficits might be for ADHD populations. Barkley (1997, 2006) has proposed a model conceptualizing ADHD (in particular, ADHD-C) as a disorder resulting from executive functioning impairments in behavioral inhibition. Pertinent to the social domain, as a result of these problems in behavioral inhibition, youth with ADHD may be unable to enact their knowledge of correct, socially skilled behaviors because they cannot suppress various verbal, motoric, and affective competing impulses in the heat of the moment. In support of this idea, research has documented that children with ADHD-C are poor at self-regulating their negative emotions (thought to be an example of deficient behavioral inhibition; see Chapter 3), and these emotion regulation problems may lead them to act without thinking in peer situations, helping to explain their compromised social functioning (Maedgen & Carlson, 2000). As well, other research indicates that executive functioning deficits (particularly in planning/organization) partially mediate the relationship between ADHD and peer problems (Huang-Pollock, Mikami, Pfiffner, & McBurnett, 2009; Tseng & Gau, 2013).

As another example of a performance deficit that interferes with the ability of children with ADHD to carry out their knowledge about correct social behavior, Abikoff (2009) has presented the idea that individuals with ADHD possess a fundamental deficit in generalization. That is, whether it is because they are distracted by extraneous stimuli, become overwhelmed by negative emotions, or have broader executive functioning deficits, individuals with ADHD are globally impaired at enacting abstract knowledge in real-life situations. It is as if they may be unable to recognize easily, at least not in the heat of the moment, that a current situation specifically calls for the abstract social skill they know they should enact.

The debate about whether children with ADHD possess knowledge or performance deficits is quite pertinent to SST. Historically, SST curricula have heavily focused on instruction in social skills knowledge under the presumption that children need to learn the appropriate social behaviors to enact in given peer situations (Gresham et al., 2004). For instance, there might be instruction about how to give compliments, how to start a conversation with a new group of peers, or how to share (Landau et al., 1998; Nixon, 2001). The assumption is that, once children with ADHD learn what behaviors they should be doing, they will naturally be able to carry them out. In other words, the focus is predominantly on remediating a knowledge deficit and not a performance deficit.

Admittedly, many SST programs include some elements to address performance deficits in an attempt to increase children's likelihood of actually displaying socially skilled behaviors, such as arranging role plays in which children practice the social skills with the peers in their treatment group. Note that the SST curriculum administered by Abikoff and colleagues (2004) also involved incorporating the target social skills into children's Daily Report Cards (whereby teachers recorded the extent to which children displayed skilled behaviors at school, and parents were instructed to reinforce children's performance of these skills), in an effort to encourage children to generalize what they learned in SST to real-world peer interactions. Recall, however, that even the SST provided in the study by Abikoff and colleagues was found to be ineffective on parent, teacher, and observer ratings of social behaviors outside of session. Nonetheless, on balance, historically clinic-based SST may lean more heavily toward providing social skills knowledge as opposed to attending to the performance barriers that prevent children with ADHD from enacting said knowledge. This focus may be misguided, and the predominant (if not possibly, exclusive) focus of SST may in fact need to be on remediating performance deficits.

There are two important issues to note. The first is that the theorized problems in performance (as opposed to knowledge) may be less applicable to individuals with ADHD-I (and especially SCT/CDD [Chapter 17]) as opposed to ADHD-C or ADHD-HI (Pfiffner, 2003). In fact, some research has documented deficits in social knowledge in children with ADHD-I (Maedgen & Carlson, 2000; Wheeler & Carlson, 1994), which may also help explain suggestions that traditional SST interventions may show greater efficacy for children with ADHD-I relative to ADHD-C (Antshel & Remer,

2003; Pfiffner et al., 2007) especially when the distinct social problems related to ADHD-I are targeted (Pfiffner et al., 2007).

Second, to the extent that children with ADHD have been documented to possess social-cognitive deficits (Gardner & Gerdes, in press), these could be considered a type of social knowledge problem that may theoretically be responsive to SST. Whereas social-cognitive deficits such as incorrect interpretations in ambiguous peer situations, noncollaborative social goals with peers, failure to attend to the key components of social interactions, inability to read peers' emotions, or inaccurate assessment of one's own competence are not the same as lacking knowledge about what to do in a certain interpersonal situation per se, theoretically, these are all skills that can be taught via a clinic-based SST curriculum. In fact, existing SST for children with ADHD commonly involves training to remediate directly some, if not several, of these social-cognitive deficits (Landau et al., 1998; Mrug et al., 2001). Still, there is not clear evidence that attending to these social-cognitive deficits improves the efficacy of SST for children with ADHD (de Boo & Prins, 2007), which means that even if children learn to remedy these social-cognitive deficits, they may still possess performance deficits that prevent them from carrying out more adaptive patterns of thinking when in real-life peer situations.

Social Contextual Factors Affecting Peer Relationships

Another potential explanation for the limited efficacy of traditional SST interventions is the lack of attention to factors in the peer group social context that also influence the social competence of children with ADHD (Mikami, Lerner, & Lun, 2010). SST is typically predicated on the fundamental assumption that most (if not all) of the reasons why children with ADHD are socially incompetent are internal to the child with ADHD. Reflecting this perspective, children with ADHD have been referred to as the architects of their own difficulties in peer situations, and as negative social catalysts who lead to the disruption of any social interactions in which they become involved (Ladd, 1981; Whalen & Henker, 1985, 1992). Therefore, the theoretical assumption behind SST is that the child with ADHD, not the peer group, should be the object of intervention (crucially, this assumption is the same whether the training is presumed to focus on increasing knowledge of correct behaviors or on reducing barriers to skillful performance). Another assumption behind traditional SST is that if the child with ADHD improves his or her behavior (e.g., displays more prosocial, socially skilled behavior and less disruptive/offensive behavior), then peers will naturally respond by increasing their liking and friendship. It is important to note that pharmacotherapy and behavioral management interventions are based on a similar assumption: targeting interventions toward the child with ADHD and assuming that if his or her behavior improves, better peer regard will follow by default.

A growing body of research suggests that these assumptions may not be fully valid. Specifically, these assumptions place the full onus of any social problems on the child with ADHD, and ignore the influences of the peer group on social problems. Yet peer relationships do not occur in a vacuum, in which the behavior of only one child in the interaction is important (for review, see Mikami, Lerner, & Lun, 2010). Rather, peers also contribute to the social behaviors, social cognitions, and ultimately, peer regard of the children with ADHD.

First peers' perceptions, behaviors, and actions toward the child with ADHD will reciprocally influence the social behaviors of the child with ADHD. If peers are welcoming and receptive, this is likely to encourage the child with ADHD to display more skilled, prosocial behaviors in return; if peers are not, this raises the probability that the child with ADHD will simply respond in kind (Schwartz, McFadyen-Ketchum, Dodge, Pettit, & Bates, 1998). In a study involving 8- to10-year-old children, experimental manipulation of rejecting behaviors from peers led children to display more maladaptive social behaviors in response, a finding that was mediated by increases in children's negative emotions as a result of the rejecting behavior (Nesdale & Lambert, 2007). Admittedly, negative interactions can become a vicious cycle whereby both the child with ADHD and peers can trade off initiating disruptive/offensive behaviors in response to one another, in escalating displays of reciprocal social incompetence. However, the point is that behaviors displayed by the child with ADHD toward peers do not occur completely independently of the behaviors displayed by peers toward the child with ADHD.

In fact, some evidence suggests that because of bias, peers treat children with ADHD in a negative fashion. In a series of studies (Harris, Milich, Corbitt,

Hoover, & Brady, 1992; Harris, Milich, & McAninch, 1998), researchers paired up previously unacquainted boys for play sessions. All boys were typically developing. However, in half of the pairs (randomly selected), researchers told one of the two boys that the partner with whom he was about to interact had ADHD symptoms. The partner remained unaware that this false information had been given about him. Observers, kept unaware of study hypotheses and experimental condition, judged the boys whose partners had been told that they had ADHD to have significantly poorer social skills than the boys whose partners had been given no such expectation (Harris et al., 1992, 1998). These important findings demonstrate the power of peers' expectations in affecting the demonstrated social behaviors of the other child in the interaction, and also how peers' negative judgments about children with ADHD may actually lead peers to treat a child with ADHD in a fashion that elicits poorer social skills from him or her.

Peers' negative actions can also encourage the development (or maintenance) of social-cognitive deficits in the child with ADHD. Research indicates that children with histories of peer rejection may be primed to assume aggressive intent in ambiguous peer provocation situations because, in actuality, it is more likely that peers do have aggressive intent in their actions toward them (Dodge et al., 2003; Schwartz, Dodge, et al., 1998); although most of the research on this topic has been conducted with aggressive children, in a study of a sample of girls with ADHD, Thurber and colleagues (2002) found positive correlations between peer rejection and children's expectations that peers would react negatively to them. Furthermore, there is some evidence that positive illusory bias in children with ADHD is maintained as a defense, such that warmth and acceptance from others (presumably, the peer group) help children with ADHD to adopt more accurate self-perceptions, whereas criticism from others maintains children's overly-inflated self-perceptions (Diener & Milich, 1997; Emeh & Mikami, 2014; Hoza et al., 2000). As well, if peers rebuff or avoid children with ADHD, then children with ADHD lose opportunities to learn or practice key social-cognitive skills, such as perspective-taking, developing appropriate goals in peer interactions, or recognizing others' emotions (Pedersen et al., 2007). These examples document that social-cognitive deficits may not solely originate in the child with ADHD, calling into question the predominant theory behind SST that treatment for these social-cognitive deficits must exclusively target the child with ADHD.

Finally, the assumption may be incorrect that if children with ADHD improve their prosocial, socially skilled behavior and reduce their disruptive/offensive behavior, then improvements in peer regard will necessarily follow. Rather, literature suggests that peers have cognitive biases against children whom they dislike and furthermore, are often resistant to changing their negative impressions once these impressions are formed (see review in Hymel, Wagner, & Butler, 1990; Mrug & Hoza, 2007). Specifically, peers assume that ambiguous behaviors on the part of disliked children are hostile; tend selectively to remember the unskilled behaviors of disliked children, while forgetting their skilled behaviors; and attribute unskilled behaviors to internal, global, and stable causes, while attributing skilled behaviors to external, specific, and unstable causes (Peets, Hodges, Kikas, & Salmivalli, 2007; Peets, Hodges, & Salmivalli, 2008). This pattern of cognitive biases is reversed to favor children whom peers like. In summary, the peers' cognitive biases against children whom they dislike predispose them to maintain their negative impressions about disliked children, crucially, even in the face of disconfirming evidence such as positive behavior change in the disliked child (Mrug & Hoza, 2007). This suggests that even if children with ADHD improve their skills and behavior, it should not be assumed that peers will notice and be able to alter their impressions of the child with ADHD accordingly.

Summary

Two factors are omitted from SST as it is traditionally delivered in clinic-based settings: attention to performance deficits within children with ADHD that may block their ability to display the social skills knowledge they have, and consideration of factors in the peer group that also contribute to the unskilled social behaviors, social-cognitive deficits, and low peer regard of children with ADHD. I argue that the insufficient attention paid to these two factors is a major part of the reason why traditional SST has shown limited efficacy for the population of children with ADHD. Hence, new directions for SST in recent years have tended to involve increased attention to the first factor (performance deficits), and to a lesser extent (but no less important, in my opinion), to the second factor (social contextual influences). In the next section, I provide two examples from my laboratory to illustrate possible

ways that SST interventions may be modified to address these factors that make traditional SST particularly challenging for ADHD populations.

TRAINING PARENTS AS FRIENDSHIP COACHES FOR CHILDREN WITH ADHD

Rationale

Increasing the emphasis on children's generalization of skilled behaviors to real-world peer interactions is needed to improve the efficacy of traditional clinic-based SST (DuPaul & Eckert, 1994). Recent studies suggest that one way to do so is to heavily involve parents in the SST that their child is receiving—in other words, to go beyond simply informing parents on occasion about the social skills the child is learning in SST and instead conduct simultaneous parent training groups in which parents explicitly learn how to reinforce their child's display of good social behavior outside of session (Pfiffner, 2008). The theory is that parents, not therapists, are likely to be present during some of the child's naturalistic peer interactions (Frankel & Mintz, 2011). Therefore, parents are in an ideal position to provide the *in vivo* reminders that children with ADHD need to overcome their performance deficits.

Promising empirical work supports this idea. For example, Pfiffner and McBurnett (1997) randomly assigned families of children with ADHD to one of three intervention conditions: (1) children receiving SST; (2) children receiving SST, while their parents attend a simultaneous treatment group in which they learn to reinforce their child's display of competent social behaviors outside of session; and (3) a control group that does not receive intervention. The condition in which children and parents received simultaneous treatment showed incrementally more improvement over the other two conditions, especially in terms of generalization on teacher reports of children's social behaviors in the school setting (Pfiffner & McBurnett, 1997). In a more recent study (but one involving only children with ADHD-I, who may be more responsive to SST relative to the other ADHD DSM-IV subtypes), a program involving simultaneous child SST and parent groups to encourage the child's generalization of social skills performed better than a no-treatment control group in terms of parent and teacher reports of children's socially skilled behaviors (Pfiffner et al., 2007). As well, Frankel and colleagues found positive effects for an SST program involving concurrent child and parent groups for children with ADHD (including those with comorbid ODD), in which parents were taught explicitly to reinforce their child's display of socially competent behavior outside of session, again, based on parent and teacher ratings of social behavior (Frankel, Myatt, & Cantwell, 1995; Frankel, Myatt, Cantwell, & Feinberg, 1997).

Other empirical work has documented positive effects of SST on children's social behavior (teacher-reported and observations) when the SST is provided in the context of an immersive intervention for children with ADHD, the Summer Treatment Program (Pelham & Hoza, 1996). Children in the Summer Treatment Program attend for approximately 8 hours/day for 8 weeks. SST is a core part of the program, in which a target social skill is chosen each day and children receive instruction in the skill, discuss uses of the skill, and often engage in role plays with classmates to practice the skill; instruction in academic and sports skills also takes place during other parts of the program. The crucial aspect of the Summer Treatment Program is that the counselors engage in intensive, frequent, and consistent reinforcement and response cost throughout the day to shape children's behaviors. As such, the same counselors teach the SST curriculum and remain with the children for the entire day while they reinforce the child's successful displays of the social skills taught. Thus, the counselors are present to provide children with ADHD the repeated reminders and reinforcements *in vivo* that they likely need to overcome their performance deficits (Pelham & Bender, 1982). Results from studies suggest that, indeed, SST in the Summer Treatment Program results in both counselor-rated and observed increases in positive social behaviors, as well as reductions in negative social behaviors (see review in Pelham et al., 1998).

In summary, promising interventions in recent years suggest that actively training adults in the child's natural environment (e.g., parents, or counselors in the Summer Treatment Program) to reinforce the child's successful display of social skills helps children with ADHD overcome the performance deficits that impede the generalization of what they learn in SST to real-world peer interactions. However, one thing that is unclear from this literature is the extent to which it is necessary for a clinician to provide SST directly to the child. If children with ADHD have performance, as opposed to knowledge, deficits, then perhaps the emphasis of SST should be shifted entirely to help the child display the social skills he or she already knows,

as opposed to instructing the child in social skills. If this is true, then it should be more efficacious (or at least, more parsimonious) to train only adults in the child's environment about how to facilitate the child's display of social skills, without any child treatment component.

As well, for the most part, this literature does not attend to the social-contextual factors that may also influence peer problems in children with ADHD, the second potential reason why existing SST interventions may have limited efficacy. The primary purpose of involving parents (or counselors, in the Summer Treatment Program) is to promote generalization by having these adults tackle the performance deficits of the child with ADHD. Little attention is given to the contribution of the peer group's behaviors to the social deficits of children with ADHD.

Parental Friendship Coaching Intervention

In my laboratory, my colleagues and I have been working on an intervention that involves training parents to help remediate peer relationship problems in their children with ADHD. The intervention, titled Parental Friendship Coaching, provides instruction solely to parents about how parents can become "friendship coaches" for their elementary school-age children with ADHD. There is no child treatment component.

Parental Friendship Coaching is intended to address both the obstacles to traditional, clinic-based SST identified earlier. First, it addresses the hypothesized performance deficits in children with ADHD. Because parents are heavily involved in their children's social interactions, especially during playdates (Frankel & Mintz, 2011), Parental Friendship Coaching harnesses the ability of parents to provide their children with *in vivo* reminders during peer interactions, increasing the probability of generalization of skills. As such, it builds on the existing work reviewed earlier, which suggests that heavily instructing parents to reinforce what their child is learning in SST will increase efficacy of SST. However, Parental Friendship Coaching is distinct from the work reviewed earlier, in that there is no child treatment component. Parental Friendship Coaching is based on the assumption that performance deficits, not knowledge deficits, are the primary impediment to children's social competence, so the intervention predominantly focuses on increasing generalization as opposed to teaching the child social skills knowledge. Although Parental Friendship Coaching contains some instruction to the child in social skills knowledge, the parent (not the clinician) provides the child with that instruction.

Parental Friendship Coaching is also designed to address the second obstacle faced by traditional SST: lack of attention to the social contextual factors that influence peer relationships. Traditional SST focuses exclusively on improving social behaviors of children with ADHD, largely ignoring the influence of peers' attitudes, cognitions, and behaviors on the social problems of children with ADHD; it also works under the assumption that improving the behavior of the child with ADHD will result directly in peers' increased liking. Parental Friendship Coaching recognizes the social-contextual factors that also influence children's social behaviors, social cognition, and peer regard. Specifically, the intervention trains the parent to arrange fun, structured social opportunities, so that peers see the child with ADHD in a positive light (in some cases, this requires the peers to change their initial negative impressions of the child with ADHD)—all of which require the parents to broaden their own social networks. In Parental Friendship Coaching, parents also learn how to select the "correct" potential friends to help bring out the best social behavior in the child with ADHD.

The Parental Friendship Coaching program comprises eight, 90-minute group sessions for parents, and two 40-minute individual sessions, during which three main topics are emphasized. In the first topic, parents learn to increase warmth and positivity in the relationship with their child with ADHD, which is often needed given that parent–child relationships in families of children with ADHD tend to have excessive conflict (see Chapter 7). The rationale for including this topic is that a child will best be able to follow the parent's guidance about appropriate social behaviors if they first have a good relationship with that parent (Hinshaw, Zupan, Simmel, Nigg, & Melnick, 1997). Also, there is evidence that positive parent–child relationships may help children to have good peer relationships because children use their parents' behaviors as a model to follow in peer interactions (Mikami, Jack, et al., 2010; Simpkins & Parke, 2001).

In the second topic, parents learn to coach their children in key social skills. Of note, by instructing their children in social skills, the parents are filling a role similar to that of the clinician in traditional, clinic-based SST. Parents might use discussion and role plays to help children learn the skills. This component

of Parental Friendship Coaching differs in two key ways from traditional clinic-based SST, however. The first is that parents in Parental Friendship Coaching tend to focus more specifically on important skills to help the child develop friendships. This is because the situation in which parents most frequently tend to view their children's social interactions—the playdate—is more suited to the deepening of friendships as opposed to the improvement of broad regard in the peer group (Ladd & Hart, 1992). The second difference is that in Parental Friendship Coaching, parents learn how to introduce contingencies to improve the generalization of these skills during *in vivo* peer interactions. Specifically, parents learn to arrange antecedents before playdates to make it more likely that their child will show prosocial, socially skilled behavior; to monitor their child's enactment of these skills during the playdate; and if the child is not showing socially skilled behavior, to intervene with reminders or redirection. Parents also learn how to debrief their child after the playdate about the behavior he or she displayed, and provide any reinforcements, if needed, to encourage the child to display the social skills again—things found in previous research to encourage children's skillful behavior in peer situations (Russell & Finnie, 1990; Simpkins & Parke, 2001; Vernberg, Beery, Ewell, & Absender, 1993). In summary, Parental Friendship Coaching takes advantage of the fact that parents, not clinicians, are available to provide children with the *in vivo* reminders and contingencies they need in order to encourage prosocial, socially skilled behavior during real-world peer interactions.

In the third topic, parents are trained to address social-contextual factors in order to facilitate their child's peer relationships maximally. For example, evidence suggests that parents who are socially competent and have good social networks themselves help their children make friends because they arrange playdates for their children with children of their own friends (Prinstein & La Greca, 1999; Putallaz, 1987). Even to the extent that a child and a friend are inclined to like one another based on their interactions at school, evidence suggests that the parents will not agree to arrange playdates to deepen the friendship unless they view the parent of the other child as likeable and competent (Howes, 1996). Unfortunately, parents of children with ADHD are often struggling with their own ADHD symptoms (Griggs & Mikami, 2011b) or with stigma related to their child's behavior problems (Hinshaw & Stier, 2008; Mikami, Chong, Saporito, & Na, in press); both factors can impede parents in establishing good social networks themselves. Parental Friendship Coaching tackles these issues so that the parents can provide the best social context for their child to develop good peer relationships. As well, the parents are trained to select judiciously the correct peers as potential friends for their child. Again, with the recognition of the social-contextual factor of what the peer will contribute to the interaction, parents are taught to select peers who seem positively inclined toward their child (or at least not negatively inclined), who are relatively tolerant and open-minded about ADHD symptoms, who have similar interests as their child, and who bring out good behavior (or at least, do not bring out bad behavior) in their child and vice versa.

Preliminary Findings

An initial pilot study involved families of 62 children with ADHD, ages 6–10, randomly assigned to receive Parental Friendship Coaching or to be in a no-treatment control group. Parents who received Parental Friendship Coaching reported that their children's social behaviors had improved, and teachers (who were kept unaware of whether the family was provided the intervention) reported that the children were more accepted and less rejected by their peers, relative to children whose parents were in the control group (Mikami, Lerner, Griggs, McGrath, & Calhoun, 2010). Observations of warm, noncritical, and instructive parental coaching behaviors were more frequent in parents who had received Parental Friendship Coaching, and these parental coaching behaviors in some cases mediated the effect of the intervention on children's peer relationships, providing support for the theoretical model of change (Mikami, Lerner, Griggs, et al., 2010). Notably, however, sociometric measures of children's peer regard were not collected in this trial. As well, the trial did not assess whether intervention effects would remain after the discontinuation of treatment.

Subsequent work with the pilot study sample has suggested that those who benefited most from the intervention were children of parents in Parental Friendship Coaching who had greater alliance with the therapist (Lerner, Mikami, & McLeod, 2011), as well as more cohesion with the other group members (Lerner, McLeod, & Mikami, 2013). As well, child variables such as age, comorbidity, gender, ADHD subtype, and medication status did not appear to moderate the effects of Parental Friendship Coaching (Mikami, Lerner,

Griggs, et al., 2010). However, parents who had higher levels of ADHD symptoms themselves appeared to have children who benefited less from Parental Friendship Coaching (Griggs & Mikami, 2011a).

Future Directions

The results from the pilot study provide preliminary support for Parental Friendship Coaching as one possible way that traditional, clinic-based SST may be modified to improve efficacy. Because Parental Friendship Coaching intervenes with the parents alone, with no child treatment component, it may be useful for children with ADHD who are unwilling (e.g., because they are resistant to the implication that they have social problems) or unable (e.g., because of little free time given receipt of other interventions, or low cognitive functioning) to receive child-focused SST.

The initial positive results from the pilot study of Parental Friendship Coaching need replication. A new clinical trial to do so is now under way and includes the following advancements from the pilot study: (1) enrollment of 150 families of children with ADHD across two sites with diverse demographics; (2) follow-up assessments up to 8 months after intervention is discontinued; (3) observational and sociometric measures, in addition to parent and teacher reports, to assess treatment efficacy; and (4) comparison of Parental Friendship Coaching versus a psychoeducational support group matched for therapist time and parental expectations for improvement.

TRAINING TEACHERS TO IMPROVE PEER REGARD OF CHILDREN WITH ADHD

Rationale

Given findings that peers have negative attitudes, perceptions, or behaviors that can contribute to the social impairment of children with ADHD, and that peers' resist changing their impressions of children with ADHD even in the face of disconfirming evidence, interventions that directly target the peer group may be warranted. Again, innovative work in recent years has begun to explore this possibility. For instance, researchers have developed interventions in which typically developing peers are trained to be tolerant and inclusive toward children with autism spectrum disorders. These peer-directed efforts appear to augment the efficacy of traditional SST in increasing parent and teacher ratings of prosocial behavior in the children with autism spectrum disorders (Bauminger, 2002), as well as, most impressively, improving sociometric measures of peer regard (Kasari, Rotheram-Fuller, Locke, & Gulsrud, 2012).

Regarding ADHD populations, Hoza, Mrug, Pelham, Greiner, and Gnagy (2003) suggest that altering the perceptions of the entire peer group about a child with ADHD may be too challenging, and a more realistic approach may to change the perceptions of one peer at a time. To this end, they paired children with ADHD with "buddies" and carefully arranged collaborative and fun activities to encourage friendship between the two children, as an augmentation to the behavioral management and SST provided as part of the Summer Treatment Program (Pelham & Hoza, 1996). Results demonstrated that children and their buddies appeared to make friends, although the study lacked a no-treatment control group with which to compare the children receiving the buddy intervention. Nonetheless, the buddy intervention directly tackles the challenge of changing peers' perceptions of children with ADHD, albeit in a prescribed way involving one peer at a time, and in a highly supervised summer camp environment.

Again, adults in the child's regular environment (e.g., classroom teachers in the studies involving children with autism spectrum disorders and counselors in the Summer Treatment Program) implemented the interventions reviewed earlier, as opposed to clinicians. This likely helps to address performance deficits and improve generalization of desired behaviors. Specifically, these adults are present during children's naturalistic peer interactions and can best reinforce the good social behaviors of children with ADHD and provide *in vivo* encouragement to peers about being socially accepting of children with ADHD.

Making Socially Accepting Inclusive Classrooms Intervention

My colleagues and I have been inspired by the studies reviewed earlier to develop an intervention that is administered by classroom teachers with the intention of helping peers to be more socially inclusive of children with ADHD. The intervention is titled Making Socially Accepting Inclusive Classrooms (MOSAIC). Similar to Parental Friendship Coaching, MOSAIC is also designed to address both obstacles to traditional, clinic-based SST identified earlier. First, the interven-

tion is administered by classroom teachers (as opposed to therapists); teachers are present during a substantial amount of children's real-world interactions with their peers and can therefore provide the *in vivo* reminders to encourage generalization. Second, the predominant, and novel, focus of MOSAIC is in changing peers' negative impressions of and cognitive biases toward children with ADHD. As such, MOSAIC directly targets the social-contextual factors in the peer group that are missing from traditional SST interventions.

In the MOSAIC intervention, teachers are trained in three main principles. First, a teacher's warm versus frustrated response to a child with ADHD may provide peers with cues about whether these behaviors should be socially devalued and whether, therefore, children with ADHD should be rejected. A teacher's personal liking and acceptance of children with behavioral problems has been found to attenuate the typically strong correlation between a child's ADHD symptoms and low peer regard (Chang, 2003; Mikami, Griggs, Reuland, & Gregory, 2012). Conversely, the extent to which a teacher is observed to display frustration with or publicly criticize children for ADHD symptoms has mediated the relationship between these child behaviors and peers' disliking (McAuliffe, Hubbard, & Romano, 2009). In MOSAIC, to model for peers that children with ADHD are worthy of liking, teachers are instructed to develop positive relationships with children by having warm, one-on-one interactions to discuss the child's personal interests. These interactions may be brief (e.g., Teacher: "How did your swim meet go?" Child: "Great!" Teacher: "Happy to hear that") so long as they communicate to the child and to peers that the teacher values and enjoys interacting with the child.

Second, MOSAIC teachers train the peer group to refrain from excluding children with ADHD by setting and enforcing explicit classroom rules for social inclusion. In addition, teachers explicitly identify commonalities between children (e.g., "Both of you are on soccer teams; maybe you should talk about that during recess") to encourage social bonds. Teachers assign children to work in teams for collaborative activities, in which children must work together in order to succeed, while explicitly explaining to children that they should treat each other with kindness and patience (and reinforcing this behavior).

Third, teachers attempt to dismantle peers' negative impressions of children with ADHD by drawing attention to a child's behavior in a way that influences that child's reputation with peers. In MOSAIC, to draw peers' attention to positive (reputation disconfirming) characteristics of children with ADHD, teachers use daily awards publicly to identify children's genuine strengths that are unrelated to their behavioral deficiencies and valued by their peer group (e.g., great artist, awesome rapper, creative game maker).

Preliminary Findings

In a small randomized pilot study, 24 children with ADHD (ages 6–9) participating in a summer day camp with 113 typically developing peers were assigned to classrooms in which the teacher was trained to deliver either MOSAIC in addition to behavioral management and SST or only behavioral management and SST. The study used a repeated measures crossover design in which children with ADHD experienced both conditions counterbalanced for order, and we equated attention from the study team and teacher expectations for improvement in the two conditions. Results from this preliminary study suggested that children with ADHD were better liked and less disliked by peers, and had more reciprocated friendships, as assessed via sociometric measures, when they were in classrooms where MOSAIC was added to behavioral management and SST (Mikami et al., 2013). These results were supported by observations of peers behaving more positively toward children with ADHD in classrooms where MOSAIC was present. Similar beneficial effects of MOSAIC were suggested for the typically developing classroom peers of the children with ADHD (Mikami, Reuland, Griggs, & Jia, 2013). The efficacy of MOSAIC was not moderated by children's ADHD subtype, use of medication, comorbidity, or age; however, MOSAIC was suggested to be more beneficial for boys than for girls with ADHD (Mikami et al., 2013).

Future Directions

These initial promising results bear replication over a longer period of time and in general education classrooms during the school year. It is unknown whether general education teachers will be able to administer MOSAIC when then have academic demands. Yet improving peers' regard of children with ADHD in their regular classrooms during the school year will likely be important in terms of benefiting children's adjustment. Further research is also needed to increase the efficacy of MOSAIC for girls with ADHD.

CONCLUSION

In this chapter I have reviewed the significant impairments that children with ADHD face in their peer relationships, as evidenced in multiple domains (problematic social behavior, deficits in social cognition, and poor peer regard). These impairments appear to persist into adolescence and adulthood, and may portend (or lead to) negative adjustment outcomes. As discussed in this chapter, peer impairments have also been quite intractable to interventions. Pharmacotherapy and behavioral management are first-line treatments for the core symptoms of ADHD, but their efficacy for improving social functioning is considerably more modest. SST has intuitive appeal as an intervention that more directly targets the social deficits of children with ADHD, but traditional, clinic-based SST does not appear to be efficacious for this population.

In this chapter, I have presented two speculations about why this might be the case—specifically, lack of attention to the performance deficits present in ADHD and social-contextual factors in the peer group that might influence peer relationships. The chapter concluded with a discussion of recent interventions for social functioning that modify traditional SST to incorporate those two factors. It is my hope that this information will be helpful to both researchers and clinicians looking for novel ways to address the prominent peer relationship impairments in youth with ADHD.

KEY CLINICAL POINTS

- ✓ Children with ADHD have significant problems in their social interactions with peers, often leading to outright peer rejection or at least low likeability and regard.
- ✓ Traditional clinic-based methods of training children with ADHD in social skills (SST) largely have not had beneficial effects on improving the peer relationships or reputations of those children.
- ✓ This chapter argues that this lack of benefit stems from at least two problems inherent in traditional SST methods: (1) viewing ADHD as involving a lack of knowledge or skills; and (2) ignoring contextual factors in the peer group that can affect the social problems of children with ADHD. Instead, evidence suggests that ADHD involves more a disorder of performance than of skills; of not doing what one knows more than of not knowing what to do (ignorance). As well, peers' biases may contribute to the poor social functioning seen in children with ADHD.
- ✓ Thus, SST approaches must focus more on assisting children with implementing the knowledge they may have about appropriate social behavior in natural ecological situations. SST must also take into account the ecological contextual factors of the child's routine social environment, such as characteristics, behaviors, and attitudes of peers toward the child with ADHD.
- ✓ I have described two innovative programs for addressing the social performance problems of children with ADHD. One involves training parents to be social skills therapists (Parental Friendship Coaching) so as to help them model, prompt, encourage, and reinforce their children's use of appropriate forms of social behavior during arranged "playdates" with another child. The second, MOSAIC, involves teaching teachers ways to reduce peer bias against children with ADHD.
- ✓ Preliminary evidence from initial studies indicates that both programs result in improved parent and teacher relationships with children with ADHD as well as in the children's peer relationships and peer regard (reputations). More research using attention-placebo or alternative interventions for comparison to these innovative programs remains to be done (and is now under way). But the initial results encourage the use of the child's natural caregivers to deliver social skills interventions in the natural social ecology of the child to overcome the obstacles that seem to have precluded success in earlier research on SST for children with ADHD.

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CHAPTER 24

Treatment of ADHD in School Settings

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School-based behavioral interventions for attention-deficit/hyperactivity disorder (ADHD) have been classified as well-established ADHD treatments for over 15 years (Pelham & Fabiano, 2008). In a recent meta-analysis of 60 studies conducted between 1996 and 2010, school-based interventions, including academic, contingency management, and self-regulation interventions, were associated with moderate to large improvements in academic and behavioral functioning of students with ADHD (DuPaul, Eckert, & Vilardo, 2012). Of note, effect sizes for contingency management were relatively similar to those for pharmacotherapy, and effect sizes for academic interventions were superior to those found for pharmacotherapy. These findings strongly support these interventions as first-line treatment strategies for addressing the educational and behavioral needs of students with ADHD.

A number of innovations in school-based interventions have been made since the previous edition of this volume was published. Recent studies of strategies for improving academic skills, homework, and organizational skills training, as well as multicomponent interventions with both skills training and behavioral components, show a positive impact on children and adolescents with ADHD. Recent efforts focused on promoting effective school–home partnerships have yielded benefits for family–teacher relationships as well as student outcomes. There is also progress on increasing dissemination, as well as accessibility and implementation, of evidence-based behavioral approaches in authentic school settings. The U.S. Department of Education supplies written documents about recommended school-based interventions for meeting the needs of students with ADHD (see Office of Special Education Programs, 2004) and recently added a Web-based What Works Clearinghouse, which provides detailed information about empirically supported school-based programs.

Our intention in this chapter is to review advances that have been made since the previous edition of this volume, as well as the more "tried and true" behavioral strategies presented in earlier versions of this chapter. The overall focus is on providing information needed for clinicians working in or with schools and helping to bridge the gap between science and practical application.

CHARACTERISTICS OF TYPICAL SCHOOLS AND TEACHERS

Active and engaged teachers, as well as supportive administrators, are clearly crucial to treatment success because behavioral technologies and curriculum

modifications can only work if they are deployed consistently in classroom settings. Most children and adolescents with ADHD are placed in general education classrooms (i.e., they do not receive special education services). Furthermore, most general education teachers received very little, if any, preservice training in how to work with students with special needs, including those with ADHD. For example, a survey by Arcia, Frank, Sanchez-LaCay, and Fernandez (2000) revealed that many teachers lack basic information about the nature of ADHD and comprehensive classroom management programs geared for these students. We have found that when teachers have a poor grasp of the nature, course, outcome, and causes of this disorder, and misperceptions about appropriate interventions, there is little to be gained by attempting to establish behavior management programs in that classroom. Alternatively, a positive teacher–student relationship, based on teacher understanding of the student and the disorder, may improve academic and social functioning. Teachers should be aware of the following:

- ADHD is considered to be a biologically based, educational disability that is treatable but not curable by treatment. Interventions can have a powerful and positive impact because severity of symptoms and comorbid conditions are very sensitive to environmental variables. Alternatively, the refractory nature of ADHD symptomatology makes it likely that these students will continue to experience at least some difficulty in their academic and social endeavors.

- ADHD is not due to a lack of skill or knowledge; it is instead a problem of sustaining attention, effort, and motivation, and inhibiting behavior in a consistent manner over time, especially when consequences are delayed, weak, or absent. Thus, it is a deficit in performing what one knows, not a deficit in knowing what to do. Nevertheless, deficits in specific skills areas (e.g., academic, social, organizational) are also common among students with ADHD. These skills deficits may arise, in part, from the higher than average co-occurrence of learning disabilities with ADHD, as noted in earlier chapters, as well as from educational inopportunity in some instances (e.g., being adopted from a developing or war-torn country or residing within an impoverished neighborhood). Alternatively, these deficits can also arise from the direct interference of ADHD symptoms with the process of knowledge acquisition (availability for learning) and with executive functioning, which is necessary to acquire information more efficiently and deploy it more effectively.

- It is harder for students with ADHD to do the same academic work and exhibit the social behavior expected of other students. We consider the student to be 30% or more behind in executive, self-regulatory, social, and organizational abilities, as Barkley (2013b) has argued. The student with ADHD needs more structure (external support, guidance, or "scaffolding"); more frequent and salient positive consequences; more consistent negative consequences (when necessary); and, possibly, accommodations to classroom expectations and assigned work.

- The most effective interventions for improving school performance are those applied consistently *within* the school setting. Family therapy, individual therapy, and parent training, while possibly beneficial at home (Chapters 21 and 22), rarely improve academic and behavioral functioning of children with ADHD school (DuPaul & Stoner, 2014).

- School-based interventions should include both proactive and reactive strategies to maximize behavior change (DuPaul & Stoner, 2014). Proactive interventions involve manipulating antecedent events (e.g., modifying instruction or classroom context) to prevent challenging behaviors from occurring. Alternatively, reactive strategies are characterized by implementing consequences (e.g., positive reinforcement) following the occurrence of a target behavior.

- Teachers should consider the use of peers, parents, computer technology, or the students themselves to deliver classroom interventions (DuPaul & Stoner, 2014). The acceptability and feasibility of school-based interventions may be enhanced by going beyond an exclusive reliance on teachers to deliver interventions.

Ideally, education about ADHD and related disorders would be provided as part of teachers' preservice training. Unfortunately, this is rarely the case, especially for general education teachers. Thus, the primary way that education about ADHD is provided is through inservice presentations and/or brief reading materials or instructional websites (e.g., *www.help4adhd.org* or *www.adhdlectures.com*). General education teachers also require training to implement behavioral programs because such training is rarely provided in their preservice certification programs. They are less likely than special education teachers to use classroom

accommodations and behavioral interventions (Forness & Kavale, 2001; Zentall & Stormont-Spurgin, 1995), and they report that a lack of training is a significant barrier to effective programming for students with ADHD (Arcia et al., 2000). Even so, many general education teachers do report using some type of behavioral intervention in their classrooms (Fabiano & Pelham, 2003), although the effects are often limited. This is likely due to the fact that the typical teacher has only cursory exposure (not explicit training) to behavior modification and/or uses weak and untailored behavioral interventions. Thus, although a teacher may report using a behavioral intervention, it may not be an effective one, and the teacher may not have the training or skill to improve it. Teachers who receive training report increased confidence in implementing effective behavioral contracts, and adjusting lessons and materials for students with ADHD (Arcia et al., 2000).

What type of training is most effective? It has been our experience that one-day inservice presentations, although useful for imparting information about the disorder, are usually not sufficient for training teachers how to implement behavioral and academic interventions for students with ADHD. Such school-sponsored training can be effective, however, if followed up by ongoing consultation or technical support. For example, many schools have adopted collaborative consultation models, in which a behavioral consultant (e.g., school psychologist) works with educators in general and special education in a systematic manner to assess student needs and to plan and implement interventions (e.g., DuPaul et al., 2006; Sheridan & Kratochwill, 2008). Ideally, the consultant should conduct a functional assessment of the student (discussed later) that includes observation of the student in the classroom setting and meeting with the teacher about the student, and determining what antecedents and consequences may be related to the student's difficulties. Once an intervention is designed and implemented, the consultant should meet with the teacher daily or weekly to review progress. Behavioral programs usually require modification over time, so this ongoing evaluation and consultation is essential.

Other service delivery models have been developed that go beyond the typical "train and hope" approach to in-service training. Although not specific to ADHD, Atkins, Graczyk, Frazier, and Abdul-Adil (2003) initiated several programs for improving school-based models for mental health service delivery. Of relevance for teacher training and support, the Teacher Key Opinion Leaders (KOL) project focuses specifically on ways in which indigenous resources in urban schools can support classroom teachers' implementation of evidence-based educational strategies for students, including those with ADHD. This program is based on the idea that influential peers are more likely than outside consultants to motivate teachers to adopt novel classroom practices. Teachers who were highly regarded for their ability to assist with classroom issues were selected by other teachers as key opinion leaders. These leaders received training in 11 evidence-based practices (e.g., positive reinforcement, response cost, peer tutoring, school–home notes), then served as teacher consultants at their respective schools. Preliminary data show that KOL-supported teachers reported using significantly more of the 11 recommended strategies than teachers who did not receive such support. Consultation from other mental health providers was not associated with use of any of the strategies. Atkins and colleagues also developed a program to increase service integration and sustainability in urban settings by coordinating delivery of mental health services among schools and community social service agencies. This form of "wrap-around" program emphasizes use of evidence-based universal, targeted, and intensive interventions tailored to the needs of individual children and provided through close collaboration between school and mental health agencies. Funding for the program is offset by existing resources (e.g., Medicaid).

Another consideration for training teachers in school-based interventions is the extent to which teachers view strategies as acceptable. Teachers report that they tend to prefer positive over negative consequences, behavioral interventions with medication over medication alone, and time-efficient (e.g., home–school Daily Report Card) over time-consuming (e.g., response cost) interventions (Pisecco, Huzinec, & Curtis, 2001; Power & Hess, 1995), although, in actual practice, use of response cost also has been viewed favorably (e.g., McGoey & DuPaul, 2000). Acceptability of treatments may vary as a function of the child's gender, with medication being viewed as more acceptable for boys than for girls with ADHD (Pisecco et al., 2001). The acceptability of interventions may also differ by grade level. Middle- and secondary-level teachers reported more attempts and more success in using accommodations that involve the child in activities and allow for

alternative seating arrangements during independent work. General educators appear to show a greater resistance to making accommodations than special educators (Zentall & Stormont-Spurgin, 1995). This greater resistance may reflect a lack of understanding about the nature of ADHD, about individual student needs, or about how to use these interventions efficiently, all of which may be helped by inservice training and ongoing teacher support.

Alternatively, it seems reasonable that special education teachers with small classes would have less difficulty implementing behavioral programs for students with ADHD than would general education teachers of up to 30–40 students, who may find the record keeping, close monitoring of the student, and administration of a range of rewards and/or negative consequences to be time-consuming and challenging to implement. The following help with this common situation:

- The addition of a behavioral aide in the classroom can be invaluable, even when the aide must rotate across multiple classrooms because of budget limitations.
- Teachers should be provided with ongoing consultation to help them plan and troubleshoot behavioral programs.
- Teachers should be supported in their efforts to work with students with ADHD. Support may include verbal recognition for their efforts, financial compensation for special materials and books, and planning and development time. We have found that schools with effective practices for ADHD invariably have an administration that recognizes this disorder as a condition in need of individualized interventions and accommodations, and provides training and resources necessary to serve the special needs of these students adequately.

Unfortunately, even with adequate resources, some teachers may on theoretical grounds still be averse to working with students with ADHD or using behavior modification procedures (e.g., its dehumanizing or too mechanistic). In such cases of poor teacher motivation or knowledge, or when teacher philosophy greatly conflicts with the necessary interventions for a student with ADHD, parents are encouraged to be assertive in pressing the school administrators for either greater teacher accountability or a transfer to another classroom or school.

Although firm and current data on educational placement are not available, most students with ADHD receive support services in schools, including special education through the Individuals with Disabilities Education Act (IDEA) (Reid, Maag, Vasa, & Wright, 1994). Within general education (i.e., regular classroom settings), it is possible for students to receive educational accommodations and support (e.g., extra time on tests, preferential seating, reduced assignments) through Section 504, a civil rights regulation stipulating that individuals with disabilities receive reasonable accommodations in educational and occupational settings. In order to qualify for Section 504 accommodations, a student must not only demonstrate significant ADHD symptoms but must also exhibit limitations to major life activities (e.g., learning) as a function of symptomatic behaviors relative to the average student in the population (Zirkel, 2013). Some students may require special education services as a function of having a learning disability, emotional/behavioral disorder, other health impairment, or some other significant educational disability (U.S. Department of Education, 2008). Current best practice involves assessing the degree to which students respond to evidence-based interventions implemented in general education settings before implementing special education services. This response-to-intervention approach is often used in the context of a three-tier model that includes universal, targeted, and individualized levels of service delivery (Sugai & Horner, 2006).

Given the extent to which students with ADHD exhibit educational impairment (for review, see Chapter 6), a variety of services may be provided in the context of general education, Section 504, and special education. It is clear that school-based services are quite costly in terms of personnel and financial resources. In fact, Pelham, Foster, and Robb (2007) estimated the total annual societal costs (including services provided by mental health, health care, judicial, and educational systems) associated with ADHD to be $42.5 billion, with an average annual educational cost of $5,007 per student relative to peers without ADHD, above and beyond costs associated with general education (Robb et al., 2011). Thus, a significant proportion of costs attributed to addressing the needs of individuals with ADHD are accrued within schools, necessitating collaboration among mental health clinicians, health care professionals, educators, and families.

CLINICIAN ROLES AND CONSULTATION APPROACHES

Importance of Collaboration between Home and School

An important consideration for enhancing the effectiveness of school interventions is the relationship between home and school. In cases where both teacher and parents are knowledgeable about ADHD, have realistic goals, and are motivated to work with ADHD, effective collaborations develop easily. In other cases, home–school conflicts can be significant and ultimately compromise the student's progress. Parents may blame their child's difficulties on the school or feel that the school system fails to address their child's needs adequately. Teachers may believe that family problems are causing the child's symptoms, or that medication should be considered in lieu of interventions and/or accommodations in the classroom. Many times, conflict between home and school is due to misinformation and can be addressed through education about ADHD. Parents and teachers need to dispel notions of blame and work toward improving the fit between the student's characteristics and the environments at school and at home. A behavioral consultant/clinician with expertise in ADHD and evidence-based interventions can help mediate these problems by providing information on the nature of ADHD and its causes, as well as information on the role of behavioral and academic interventions (including both their strengths and limitations) in the treatment of ADHD.

Because ADHD symptoms impact children's functioning across home and school settings, and because parents and teachers may experience the aforementioned challenges to working together, efforts have been made to design, implement, and evaluate treatment strategies that involve collaboration between home and school. For example, the Collaborative Life Skills Program (CLS; Pfiffner, Villodas, Kaiser, Rooney, & McBurnett, 2013) uses multiple behavioral interventions in school and home for elementary school-age children with ADHD. CLS comprises three components (classroom behavioral intervention, group behavioral parent training, and child skills group) delivered simultaneously over 12 weeks by school-based mental health professionals. The classroom component includes a school–home Daily Report card, homework plan, and individualized accommodations (e.g., preferential seating). Group-based parent training includes ten 1-hour sessions in which behavior management skills (e.g., contingent reinforcement) are taught (as described in Chapter 21; Barkley, 2013a). Children also participate in ten 40-minute group sessions during the school day to learn social skills (e.g., good sportsmanship) and independence (e.g., establishing and following routines) through didactic instruction, behavior rehearsal, and practice role plays. Pfiffner and colleagues (2013) found CLS to be associated with statistically significant, moderate to large improvements in ADHD symptoms, homework problems, task engagement, achievement test scores, and report card grades. Academic improvements were partially mediated by CLS improvements in student organization skills. Although the effects of CLS need to be evaluated relative to a control condition, initial findings are quite promising in relation to improvement in a critical area of functioning for students with ADHD (i.e., educational performance).

In similar fashion, Family–School Success (FSS) was designed to improve family and educational functioning for elementary school students with ADHD (Power et al., 2012). FSS includes 12 sessions with six simultaneous, separate parent and child group sessions, four individualized family therapy sessions, and two family–school consultation sessions for parents and teachers. In these sessions, clinicians guide participants through standard behavioral parent education procedures, establishing a Daily Report Card, homework interventions, and conjoint behavior consultation (see description in the section on consultation models). The FSS is manualized, and implementation is associated with high levels of integrity. When evaluated relative to a comparison condition providing education and support to parents, FSS was found to have statistically significant, small to moderate effects on the quality of the family–school relationship, homework performance, and parenting behavior (e.g., reduction in negative/ineffective discipline), with effects on the latter maintained at 3-month follow-up (Power et al., 2012). Given these promising results, school professionals should consider partnering with community-based clinicians to implement home–school partnership interventions such as CLS and FSS to address the multiple, cross-situational needs often exhibited by students with ADHD.

Consultation Models for Working with Teachers and Parents

Clinicians can facilitate the use of evidence-based assessment and intervention for students with ADHD

through structured consultation with teachers and parents. Although there are several school-based consultation models (Erchul & Martens, 2010), the behavioral or consultative problem-solving model is the best match for meeting the educational needs of students with ADHD (DuPaul & Stoner, 2014). The consultative problem-solving model involves consultants and consultees (e.g., teachers, parents) working together through four stages: problem identification, problem analysis, treatment implementation, and treatment evaluation (Kratochwill & Bergan, 1990). After collaborating with the consultee to identify specific target behavior(s), as well as associated antecedent and consequent events, the consultant suggests possible evidence-based interventions to address the identified problem(s). The consultant also guides the consultee in selecting interventions that are based, at least in part, on the perceived function (e.g., attention, avoid/escape) of the target behavior(s). The consultant and consultee collaborate to develop a specific intervention plan that the latter views as feasible and acceptable. Once treatment is implemented, the consultant assesses the degree to which the consultee adheres to prescribed strategies and provides feedback to guide implementation. The consultant may also provide training to the consultee when the latter is unfamiliar with or lacks the skills to implement treatment. Data are collected both prior to and following intervention implementation to assess whether treatment has the desired effect and whether the consultee's goal for the target behavior is met.

The consultative problem-solving model has been used successfully to improve behavioral (e.g., Sheridan, Welch, & Ormi, 1996) and academic (e.g., DuPaul et al., 2006) functioning of students with ADHD and related disruptive behavior disorders. A variant of consultative problem-solving called "conjoint behavioral consultation" has particular relevance for addressing the difficulties exhibited by children and adolescents with ADHD because this model involves consultants working with multiple consultees (e.g., teachers, parents) simultaneously. One of the goals of conjoint behavioral consultation is to address children's problems across home and school settings in a consistent, collaborative fashion (Sheridan & Kratochwill, 2008). As such, conjoint behavioral consultation may be a particularly effective strategy for clinicians seeking to design, implement, and evaluate interventions that may impact children's behaviors consistently across settings. As mentioned previously, conjoint behavioral consultation is a core component of the successful FSS program designed by Power and colleagues (2012).

Promoting Implementation/Adherence to Behavioral and Academic Interventions

For a variety of reasons (e.g., limited acceptance, philosophical differences, feasibility concerns, time and/or resource constraints), teachers may not implement prescribed intervention procedures despite their involvement in treatment design or their apparent enthusiasm for treatment. Thus, it is critical for clinicians to determine whether the intervention has been implemented as prescribed by monitoring treatment integrity (Noell et al., 2005; Perepletchikova, Treat, & Kazdin, 2007). For example, when a parent or teacher is asked to use a specific strategy (e.g., classroom-based token reinforcement program), treatment adherence can be assessed in several ways. Ideally, direct observations of teacher behavior would occur occasionally throughout treatment to assess whether the intervention steps are being carried out as planned. Of course, there would then be no way to ensure that treatment integrity was intact during intervention sessions in which an observer was not present. Thus, an additional assessment method would be for the teacher or treatment agent to complete a checklist every time the intervention is implemented to determine what steps are being followed appropriately. Alternatively, someone other than the treatment agent (e.g., classroom aide) could complete the treatment step checklist on a regular basis. Another option is to audiotape intervention sessions for later review regarding implementation integrity. Finally, permanent products generated by treatment implementation (e.g., reward charts, Daily Report Cards) can be reviewed on a regular basis to determine whether treatment steps are being followed. The bottom line is that without at least occasional treatment integrity checks, one cannot be sure that the intervention is being applied as designed.

GENERAL BEHAVIORAL GUIDELINES

Behavioral interventions for ADHD in the classroom include a range of modifications to the classroom environment, academic tasks, in-class consequences, home-based programs, and self-management interventions. These interventions stem from the model by Barkley of ADHD (see Chapter 16) as an impairment in the self-

regulation of behavior by its consequences and by rules, most likely owing to weaknesses in inhibition, motivation, and executive functioning. They are most effective when embedded during teaching activities and should be considered critical parts of effective teaching rather than time-consuming adjuncts. In most cases, they can be administered by the lead classroom teacher; however, behavioral aides in the classroom may be necessary to implement interventions for students with more severe symptoms.

For maximally effective behavioral interventions tailored to the specific needs of the student, one must go beyond the diagnosis of ADHD and identify specific behaviors for which change is desired (e.g., deportment, academic problems, social skills), as well as the function that these behaviors serve for the student. Effective targeting of behaviors should do the following:

- *Focus on teaching children a set of skills and adaptive behaviors to replace the problems* (DuPaul & Stoner, 2014). For example, a target behavior to address organizational problems may involve teaching students to use and store materials in their desk or locker properly; aggressive children may be taught to increase good sportsmanship skills. If positive alternative behaviors are not taught and only problem behaviors are targeted for intervention, children may simply replace one problem behavior with another.

- *Include academic performance (e.g., amount of work completed accurately) rather than just on-task behavior, because improvement in classroom deportment is often not paralleled by improvement in academic functioning (e.g., children who are sitting quietly may not be any better at completing their work).* Increased attention to the development of academic skills (e.g., reading, writing, and spelling) in students with ADHD has also been stressed, to prevent the deficits in academic achievement commonly experienced by these students in their later elementary years.

- *Include common problem situations, such as transitions between classes and activities, recess, and lunch.* Teachers should consider very simple programs targeting these brief periods during the day.

FUNCTIONAL ASSESSMENT

Clinicians should use functional assessment to link selection of target behaviors with intervention for students with ADHD (DuPaul & Ervin, 1996; Ervin, DuPaul, Kern, & Friman, 1998). A functional assessment involves the following:

1. *Carefully defining the target behavior in question, so that the teacher is able to reliably monitor the behavior.*

2. *Identifying antecedents and consequences of the behavior in the natural environment through interviews with teachers, parents, and students, and through direct observation.*

3. *Generating hypotheses about the function of the problem behavior in terms of antecedent events that set the occasion for the behavior and/or consequences that maintain it.* Potential antecedents include difficult or challenging work, a teacher direction or negative consequence, or disruption from another child. Potential consequences include teacher or peer attention, or withdrawal of a task or teacher request. Antecedent events need not immediately precede the problem behavior to be important in this analysis. Distal events, or those occurring minutes to hours before the target behavior, may have some role to play in increasing the probability of disruptive behaviors. For instance, arguments or fights with other family members at home or with other children on the bus ride to school may alter certain affective states (e.g., anger, frustration), which may make the occurrence of aggressive or defiant behavior upon arrival at school more probable.

4. *Systematically manipulating antecedents and consequences (those that can be) to test hypotheses about their functional relationship to the target behavior.* The most common function may be to avoid or escape effortful or challenging tasks (e.g., repetitive paper-and-pencil tasks). Others include obtaining teacher or peer attention, gaining access to an activity that is more reinforcing or interested to the child (e.g., fiddling with toys rather than completing work), or accessing pleasant sensory experiences (e.g., daydreaming) (DuPaul & Ervin, 1996).

5. *Implementing interventions that alter the functional antecedents or consequences so that problem behavior is replaced with appropriate behavior.* For example, a child who is easily distracted by small toys or objects in his or her desk may be allowed access to those objects only after a specific amount of assigned work is completed.

Functional assessment provides a useful mechanism for tailoring interventions to individual children—one

that goes well beyond a diagnosis of ADHD. This approach should help the clinician predict which of many behavioral interventions will have the greatest impact on changing specific problematic behaviors. In fact, a recent single-subject meta-analysis of 82 studies and 168 participants with ADHD found that functionality-based interventions had significantly larger effects that non-functionality-based interventions (Miller & Lee, 2013).

This approach may also be useful for modifying existing behavioral programs. For example, Fabiano and Pelham (2003) report a case study in which a teacher had been using a behavioral intervention for a student with ADHD for several weeks, yet the boy had yet to achieve his behavior goal and earn a reward. A consultant observed the boy in the classroom and, based on a functional assessment, made a few simple suggestions: Provide rewards daily rather than weekly; provide immediate feedback to the boy when he violates classroom rules; and make clear the criteria for the target behaviors (fewer than three violations of each rule). These changes to the program resulted in improvement in on-task behavior and reductions in disruptive behavior.

This is not to say that the symptoms of ADHD arise purely as a consequence of social learning or dysfunctional contingencies in the school or home environment. It is to acknowledge that despite a strong neurogenetic contribution to the origins of ADHD, the disruptive behavior toward which it predisposes can still take on additional functional properties in the home and school environment that can benefit from a functional analysis and targeted intervention. Moreover, the fact that children with ADHD may respond to existing behavioral contingencies in atypical ways due to their executive/self-regulatory deficits also argues for the use of more systematic methods of behavioral management. Such methods serve to provide additional environmental feedback and support that can help moderate the symptoms of ADHD and especially reduce the impairments that arise and their associated deficits in self-regulation (executive functioning).

CLASSROOM STRUCTURE, TASK DEMANDS, AND ACADEMIC CURRICULA

Behavioral interventions have long emphasized consequence-based strategies (reviewed later) for ADHD; however, antecedent-based interventions are equally important for improving the school functioning of ADHD youth (DuPaul et al., 2012). Antecedent-based or proactive strategies include modifications to the structure of the classroom environment, classroom rules, and the nature of task assignments, as well as computer-assisted instruction, assistive technology, and explicit instruction in academic skills. Section 504 accommodations commonly include modifications to classroom structure, tasks, and expectations, and they are typically easy to implement, even in general education classrooms.

Changing the Classroom Environment and Actively Teaching Expectations

Common accommodations to the classroom that can facilitate closer teacher monitoring and less distraction include the following:

- Placing the student's desk away from others to an area close to the teacher.
- Utilizing individual and separated desks in physically enclosed classrooms.
- Having classrooms that are well organized, structured, and predictable, with the posting of a daily schedule, classroom rules, and other visual aids (brightly colored posters) that can reduce the need for frequent verbal repetitions of rules.

In the context of universal-level service delivery, teachers should be encouraged to teach rules and expectations actively to all students throughout the school year. Ideally, teaching and modeling of rules and expectations would occur schoolwide at the point of performance (i.e., setting and time in which behavioral expectations are relevant). Specifically, teachers should be encouraged to do the following:

- Actively teach expectations for student engagement by discussion, modeling, and praising children for following them (i.e., catch students following rules) and ensuring that academic and nonacademic routines are regularly taught and practiced by all students.
- Use active supervision practices, such as frequently scanning and circulating through the classroom while monitoring student attention and behavior.
- Remind students about expected engagement behaviors before an activity begins rather than waiting until after a rule has been broken.
- Correct behavioral errors (e.g., calling out without

permission) in a brief, clear, and consistent manner, similar to instructional strategies for correcting academic errors.
- Maintain a brisk pace of instruction and use a range of verbal, nonverbal, and visual cues to precorrect and redirect disruptive behaviors so that instruction is uninterrupted.
- Frequently communicate expectations about use of class time and task engagement in a clear manner through the use of explicitly taught routines and procedures.

Transitions from one area of the school to another often trigger problem behavior. Teaching, modeling, and practicing efficient transition behaviors, especially in new or typically problematic settings, can prevent problem behaviors that would otherwise be expected during these times. Transitions into and out of areas with high numbers of students, such as the cafeteria and hallways, may be particularly problematic and a source of discipline referrals. Active supervision by adults, including moving around the area, scanning for potential trouble spots, and interacting briefly and positively with students, can decrease problem behaviors in common areas.

Modifying Academic Assignments and Expectations

Students with ADHD frequently exhibit difficulties starting and independently completing academic assignments. Several recommendations for altering academic assignments to enhance performance are as follows:

1. *Assign academic work that is well matched to the student's abilities.* In the case of students with ADHD, increasing the novelty and interest level of the tasks through use of sensory stimulation (e.g., color, shape, texture) seems to reduce activity level, enhance attention, and improve overall performance (Zentall, 1993).

2. *Vary the presentation format and task materials (e.g., through use of different modalities) to help maintain interest and motivation.* When lowinterest or passive tasks are assigned, they should be interspersed with high-interest or active tasks in order to optimize performance. Tasks requiring an active (e.g., motoric) as opposed to passive response may also allow students with ADHD to better channel their disruptive behaviors into constructive responses (Zentall, 1993).

3. *Design academic assignments that are brief (i.e., accommodated to the child's attention span) and presented them one at a time rather than all at once in a packet or group.* Short time limits for task completion should also be specified and may be enforced with the use of external aids, such as timers. For example, a timer may be set for several minutes, during which time the student is to complete a task. The goal for the student is to complete the task before the timer goes off. Feedback regarding accuracy of assignments should be immediate (i.e., as it is completed).

4. *Deliver group lessons in an enthusiastic yet task focused style, keep it brief, and allow frequent and active child participation to enhance student attention.*

5. *Intersperse classroom lecture or academic periods with brief moments of physical exercise to diminish the fatigue and monotony of extending academic work periods.*

6. *Schedule as many academic subjects into morning hours as possible, leaving the more active, nonacademic subjects and lunch to the afternoon periods.*

7. *When necessary and deemed helpful, implement accommodations for written work, such as reducing the length of the written assignment (particularly when it is repetitious), breaking it into smaller work quotas with shorter work periods and brief breaks from work, and allotting extra time to complete work.*

8. *Provide task-related choices to increase on-task behavior and work.* Choice making typically is implemented by providing a student with a menu of potential tasks in a particular academic subject area from which to choose (Dunlap et al., 1994). For example, if the student is having difficulty completing independent math assignments, he or she would be presented with several possible math assignments from which to choose and would be expected to choose and complete one of the tasks listed on the menu during the allotted time period.

PROVIDING COMPUTER-ASSISTED INSTRUCTION

Computer-assisted instruction (CAI) programs seem well suited for engaging students with attentional/distractibility problems and motivational deficits (DuPaul & Stoner, 2014). For example, these programs typically include clear goals and objectives, highlight important material, simplify tasks, provide both immediate error

correction and feedback regarding accuracy, and many (perhaps the more effective ones) also have a game-like format. Students with ADHD would be expected to be considerably more attentive to these types of teaching methods than to lectures or individual written assignments. Several controlled case studies suggest that these methods are helpful for at least some students with ADHD (e.g., Clarfield & Stoner, 2005; Mautone, DuPaul, & Jitendra, 2005; Ota & DuPaul, 2002) and may be considered as an adjunct to other academic or behavioral interventions.

For example, Mautone and colleagues (2005) examined the on-task behavior and task accuracy during math instruction for three elementary school-age students with ADHD as a function of using computer software with a game format. Clinically significant increases in engagement and math performance, and concomitant decreases in off-task behavior were found relative to typical classroom conditions (e.g., completion of written assignments). Participating students and teachers generally reported CAI to be a highly acceptable and feasible intervention. Nevertheless, more research is needed to discern the degree to which CAI is a viable classroom intervention for most students with ADHD.

IMPROVING ACADEMIC SKILLS

For students with both ADHD and academic skills deficits or learning disabilities, remedial instruction in skills areas such as reading, writing, spelling, and math is recommended. For a review of instructional strategies for remediation, see DuPaul and Stoner (2014). Instructional programs for children and adolescents with social skills deficits are reviewed in Chapter 23. Many students with ADHD also have difficulty with organizational and study skills. Instruction in time and materials management is required. Such training may include note-taking strategies (Evans, Pelham, & Grudberg, 1994), desk checks for neatness, and filing systems for organizing completed work (DuPaul & Stoner, 2014; Pfiffner, 2011). We discuss organizational skills training in more detail later in this chapter.

Strategies to address academic performance difficulties associated with ADHD may include explicit instruction, parent tutoring, and peer tutoring. The most important way to address potential academic difficulties is for teachers to use principles of explicit instruction when working with students with ADHD. Explicit instruction is a direct approach to teaching that involves (1) providing clear information to students about what is to be learned; (2) teaching skills in small steps, using concrete, multiple examples; (3) continuously assessing student understanding; and (4) supporting active student participation that ensures success (Nelson, Benner, & Mooney, 2008). A key aspect of explicit teaching is the use of instructional momentum that involves lesson pacing (e.g., using a predictable lesson process that includes varied instructional activities) and managing instructional transitions (e.g., giving clear directions for transitions) (Rosenshine & Stevens, 1986). The five key elements of explicit instruction include daily review and prerequisite skills check, teaching of new content, guided practice, independent practice, and weekly/monthly review of skills attainment (Nelson, Benner, & Bohaty, 2014). Although the impact of explicit instruction on academic achievement has not been specifically studied in students with ADHD, an abundant literature supports this teaching approach for children and adolescents with emotional and behavioral disorders (see Nelson et al., 2014). Furthermore, the tenets underlying the explicit instruction approach have a long history of support in the behavioral analytic research literature.

Parents can support their children in practicing important academic skills. For example, Hook and DuPaul (1999) evaluated parent tutoring for four second- and third-grade students with ADHD. Parents were trained to tutor their child in reading, using the same stories covered in class. The procedure for parent tutoring involved students reading orally from a selected section of a story for 5 minutes and parents intervening with a set procedure when the child made reading errors. Children then read on their own for 5 minutes, followed by oral reading for 1 minute. Home–school communication forms were used to keep close track of homework. Results showed that students' reading performance generally improved. Parent tutoring is probably most helpful for students who are generally compliant and have a good relationship with their parent, and for parents who are able and interested in the intervention.

Peer tutoring is one way to utilize classroom peers as a part of the intervention process for students with ADHD. This strategy involves students working in pairs and helping each other practice academic skills, typically reading, math, and spelling. Peer tutoring focuses specifically on improving academic skills (a target that has been relatively unaffected by traditional contingency management programs) and provides a learning

environment well suited to the needs of students with ADHD (i.e., immediate, frequent feedback, and active responding at the student's pace) (DuPaul & Stoner, 2014). A meta-analysis of 26 single-case research design studies including over 900 students from the general school population (including those with and without disabilities) found moderate to large effects of peer tutoring on academic achievement (Bowman-Perrott, Davis, Vannest, Williams, Greenwood, & Parker, 2013). Peer tutoring effects were consistently strong across dosage (i.e., duration, intensity, and number of sessions), grade level, and disability status. Of particular relevance for the use of this strategy with students with ADHD, the strongest effects were found for youth with emotional and behavioral disorders relative to other disability groups.

The most prominent and widely studied peer tutoring program is classwide peer tutoring (CWPT; Greenwood, Maheady, & Delquadri, 2002), in which all students are paired for tutoring with a classmate. Students are first trained in the rules and procedures for tutoring their peers in an academic area (e.g., math, spelling, reading). Sitting in an adjacent seat, the tutor reads a script of problems to the tutee and awards points to the tutee for correct responses. The tutor corrects incorrect responses, and the tutee can practice the correct response for an additional point. The script (problem list) is read as many times as possible for 10 minutes, then the students switch roles, with the tutee becoming the tutor and the tutor becoming the tutee. During the tutoring periods, the teacher monitors the tutoring process and provides assistance if needed. Bonus points are awarded to pairs who follow all of the rules. At the end of the session, points are totaled and those with the most points are declared the "winners." Studies indicate that CWPT enhances the on-task behavior and academic performance of unmedicated students with ADHD in general education classrooms (DuPaul, Ervin, Hook, & McGoey, 1998; DuPaul & Henningson, 1993). Furthermore, the results of DuPaul and colleagues (1998) indicated that typically achieving students also showed improvements in attention and academic performance when participating in CWPT.

TEACHER-ADMINISTERED CONSEQUENCES

Teacher-administered consequences continue to be the most well-researched and commonly used behavioral interventions for students with ADHD. A combination of positive consequences (praise, tangible rewards, token economies) and negative consequences (reprimands, response cost, time-out) has been shown to be optimal. However, their success for students with ADHD is highly dependent upon how and when they are administered. Consequences that are immediate, brief, consistent, salient, and (in the case of positive consequences) delivered frequently seem to be most effective for behavior management.

Strategic Teacher Attention

"Strategic teacher attention" refers to the practice of purposely using attention to help students remain on task and to redirect those who are off task. Praise and other forms of positive teacher attention (smiles, nods, pats on the back) have documented positive effects on students with ADHD. Withdrawal of positive teacher attention contingent upon undesirable behavior (i.e., active ignoring) can decrease the behavior. A teacher's approval, appreciation, and respect for a child with ADHD can go a long way toward enhancing the teacher–student relationship.

Although these procedures may seem unusually simplistic, the systematic and effective use of teacher attention in this manner requires great skill. Praise appears to be most effective when it (1) specifies the appropriate behavior being reinforced, (2) is delivered in a genuine and personal fashion—with a warm tone of voice and varied content appropriate to the child's developmental level, and (3) is delivered as soon as possible following desired behavior (e.g., getting started on work, raising a hand to talk, and working quietly). It is this *strategic* timing in the application of teacher attention contingent upon appropriate child conduct, and attention to behaviors that are usually expected, that is so crucial to its effectiveness as a behavior change agent. Research also shows that praise focused on effort and not intelligence or ability is best for improving motivation and persistence on challenging tasks (see Dweck, 2006). For example, instead of saying "You are so smart," a better approach would be to say, "You worked really hard on that problem." This is because ability-focused praise encourages a fixed mind-set, in which intelligence (or whatever trait is being praised) is static and cannot be changed, whereas effort-focused praise encourages a growth mind-set, in which personal characteristics are considered malleable. Children with a growth mindset tend to take on more challenges and be higher achievers since they feel empowered and that

their effort can make a difference. This concept also applies to best strategies for helping students when they do not perform well. For example, instead of telling a student that his or her poor performance was because of an innate weakness in an area (e.g., not good at math or because one has ADHD), an approach to improve motivation and effort toward change is to help the student see that he or she needs better strategies to develop skills for that area (e.g., a tutor, organizational skills, Daily Report Card with clear goals).

"Active ignoring" requires the complete and contingent withdrawal of teacher attention—an approach most suitable for nondisruptive minor motor and nonattending behaviors intended to gain teacher attention. Because ignored behavior often increases at first, active ignoring is generally not effective in modifying problem behavior that is not maintained by teacher attention, and it should not be used for aggressive or destructive behavior. Most behavior problems exhibited by children with ADHD are not purely bids for teacher attention, so this strategy alone is unlikely to result in dramatic changes in the behavior of these children. However, the simultaneous use of praise and ignoring can be quite effective. Thus, appropriate behavior (e.g., sitting in one's seat) that is incompatible with ignored behavior (e.g., wandering around the class) should be consistently praised. In addition, one of the most powerful uses of teacher attention for modifying problem behavior capitalizes on the positive spillover effects of positive attention. In this procedure, the teacher ignores the disruptive student and praises students who are working quietly. Then the teacher praises the previously disruptive student once the latter begins working quietly. The student's problem behavior often improves as a result, presumably due to vicarious learning that has occurred through this modeling procedure and the child's desire for positive teacher attention.

To assist teachers with remembering to attend to and reinforce ongoing appropriate child conduct, several cue systems can be helpful. One such system involves placing large smiley-face stickers about the classroom in places where the teacher may frequently glance, for example, toward the clock on the wall. When these are viewed, they serve to cue the teacher to remember to check out what the student with ADHD is doing, and to attend to it, if it is at all positive. Another strategy is to have the teacher place 10 or so bingo chips in his or her left pocket that must each be moved to the right pocket whenever the teacher gives positive attention to the child with ADHD. The goal is to move all 10 chips to the right pocket by the end of that class period. A third idea is to use a small vibrating device containing a timer that can be programmed to any interval desired by the teacher (e.g., The MotivAider™, available from *www.addwarehouse.com*).Teachers can wear the device on a belt or in a pocket, and when they detect the tactile vibration, use this as a cue to monitor the class and briefly respond to both positive and negative student behaviors.

Tangible Rewards and Token Programs

Because of their decreased sensitivity to reward and their failure to sustain effort when reinforcement is inconsistent and weak, students with ADHD usually require more frequent and powerful reinforcement, often in the form of special privileges or activities, to modify classroom performance. For example, a student may earn extra free time for completing assigned classwork promptly and accurately. In other cases, a token economy may be used. In this system, students earn tokens (points, numbers, or hash marks for older children; tangibles, such as poker chips, stars, or tickets, for younger children) throughout the day, then later exchange their earnings for "backup" rewards (privileges, activities). Backup rewards are assigned a purchase value, so that rewards can be matched to the number of tokens or points earned. As we describe later, some programs also include a response cost component, in which children lose points for inappropriate behavior. Some tangible or backup rewards are distributed on a daily basis, while longer periods (e.g., weekly) of appropriate behavior or academic functioning may be required for more valuable rewards.

The identification of powerful rewards is critical for program success and may be achieved through interviews with children about the kinds of activities or other rewards they would like to earn, and by observing the high-rated activities in which they normally engage. Access to these activities can then be used as reinforcement. Monitoring the manner in which the child spends free-time activities over a week or so may suggest what privileges or activities are especially rewarding for that particular child. We have found the following to be effective reinforcers: homework passes; removing the lowest grade or making up a missing grade; a grab bag with small toys or school supplies; free time; computer or video game time; stickers/stamps; running errands; helping the teacher; earning extra recess; playing special games; and art projects.

In some cases, rewards available at school may not be sufficiently powerful to alter a child's behavior. Home-based reward programs, discussed in a subsequent section, may be considered in these cases. It is also possible to have parents provide a favored toy or piece of play equipment from home to the teacher for contingent use in the classroom as part of a classroom token or reward system. Asking parents at the start of the school year to clean out their closets and donate unused toys to the classroom can result in a rich harvest of items that may be attractive as rewards to other students. Another idea would be to ask each parent of a child in that classroom to donate a few dollars to assist with purchasing prizes that can be used as rewards. The Parents–Teachers Association (PTA) also may be a resource to fund class prizes.

Reward programs can be designed for individual children or the entire class. Individual programs or classwide programs wherein students earn rewards for their own behavior are often best for the student with ADHD. Involving the entire class may be particularly effective when peer contingencies are competing with teacher contingencies (e.g., when peers reinforce disruptive students by laughter or joining in on their off-task pursuits). Some sample programs include the following (see Pfiffner, 2011):

- *Challenge Games.* In these games, students compete with teachers for tokens (e.g., chips, pegs, points) during designated time periods. Students earn tokens by following rules; teachers earn tokens when the student does not follow the rules. Whoever has the most tokens selects an activity for the class.

- *Lotteries and Auctions.* In these popular programs, students earn stamps, stickers, or tickets for a variety of target behaviors throughout the day and exchange them for chances in the lottery or items during class auctions offered at least once a week (daily at the beginning of the program).

- *Team Contingencies.* In this variation of group programs, children are divided into competing teams and earn or lose points for their respective team, depending on their behavior. The team with the greatest number of positive points or fewest negative points earns the group privileges. For example, teams may be divided by tables or rows. Points are given to a team for behaviors of the individual members, such as getting along and keeping the area clean. Either the team with the most points or all teams who meet a specified criterion earn the reward.

- *Class Movies and Theme Parties.* To keep things interesting, we have found that posters depicting the activity to be earned and a record of class progress toward earning the activity are helpful. In one example, for every 15 minutes that class members are on task, the children in the picture are moved an inch closer to a picture of a theater. When they reach the theater, the class members earn the movie.

- *The Good Behavior Game* (Barrish, Saunders, & Wolf, 1969; *www.interventioncentral.org/index.php/behavioral-resources*). In this approach, the class is divided into two teams. Each team receives marks for rule violations of individual team members. After a specified period of time, both teams earn a reward if their marks do not exceed a certain number; otherwise, the team with the fewest marks wins. This game has effectively improved student behavior and is also well accepted by teachers (Tingstrom, 1994).

Group programs targeting all students' behavior have the advantage of not singling out the child with ADHD. Given some teachers' concerns about possible stigmatization or undue attention to children receiving treatment for behavior problems, a group procedure may be preferable. This may also be the treatment of choice when there is concern that children not involved in treatment may increase their misbehavior in order to be a part of the program and receive reinforcement. It should be noted, however, that concerns about stigmatization and escalation of problem behavior have not been substantiated in research studies. Still, when a teacher is using group contingencies, care should be taken to minimize possible peer pressure and subversion of the program by one or more children. Powerful reward-only programs may be effective in those cases.

The success of token programs in numerous studies and the utility of these programs with a wide range of problem behavior have led to their widespread use in school settings. However, appropriate and realistic treatment goals are critical for the success of the program. No matter how motivated such a child may be initially, if the criterion for a reward is set too high, the child will rarely achieve the reward and is likely to stop trying. To prevent this occurrence, rewards should initially be provided for approximations of the terminal response and should be set at a level that ensures the child's success. For instance, a child who has a long history of failing to complete work should be required to complete only a part, not all of his or her work, in order

to earn a reward. Similarly, a child who is often disruptive throughout the day may initially earn a reward for exhibiting quiet, on-task behavior for only a small segment of the day. As performance improves, more appropriate behavior can be shaped by gradually increasing the behavioral criteria for rewards.

In addition, target behaviors such as "completing assigned work" or "participating appropriately in class discussions," which encourage active, appropriate behavior rather than the simple absence of inappropriate or disruptive behavior, are most effective. In our experience, teachers like the idea of targeting academic behaviors (e.g., completion and accuracy of work) because these behaviors are incompatible with disruptive behavior, are more easily monitored than classroom deportment, and tell the child exactly what is expected.

It is important to reiterate that students with ADHD typically lose interest if the same reward is used for too long. Rewards are much more effective if they are novel and change regularly. We recommend using a "reward menu" (a list of varied activities, privileges, or objects) and having a child choose his or her own rewards. The "packaging" of the reward is especially important. We strongly recommend that teachers be enthusiastic and make the reward fun and interesting by using colorful posters, creative tokens, and special words to describe the treat (e.g., "bonus," "challenges").

Negative Consequences

Whereas positive approaches should be emphasized in working with students who have ADHD, negative consequences are usually necessary. However, the effectiveness of negative consequences, particularly for students with ADHD, is highly dependent on several of the stylistic features described below.

Reprimands/Corrective Statements

A number of studies (e.g., Pfiffner & O'Leary, 1993) indicate that reprimands or corrective statements that are immediate, unemotional (noninflammatory), brief, and consistently backed up with time-out or a loss of a privilege for repeated noncompliance are far superior to those that are delayed, long, or inconsistent. Proximity also seems to make a difference; reprimands that are issued in close proximity to a child have an edge over those yelled from across the room. Mixing positive and negative feedback for inappropriate behavior appears to be particularly deleterious. For example, children who sometimes are reprimanded for calling out, but at other times are responded to as if they had raised their hands, are apt to continue (if not increase) their calling out. In addition, children respond better to teachers who deliver consistently strong reprimands at the outset of the school year (immediate, brief, firm, and in close proximity to the children) than to teachers who gradually increase the severity of their discipline over time. Finally, the practice of using encouragement ("I know you can do it") in an attempt to coax a student into good behavior is not as effective as clear, direct reprimands (Abramowitz & O'Leary, 1991).

Response Cost

"Response cost" involves the loss of a reinforcer contingent upon inappropriate behavior. Lost reinforcers can include a wide range of privileges and activities. Response cost also can be used in the context of a token program. This procedure involves a child's losing tokens for inappropriate behavior, in addition to earning them for appropriate behavior. It is convenient, easy to use, and readily adapted to a variety of target behaviors and situations. Furthermore, response cost has been shown to be more effective than reprimands for children with ADHD and may also increase the effectiveness of reward programs.

Response cost has been used in a variety of formats. For example, color-coded response cost methods have been implemented in several programs (e.g., Barkley et al., 2000; Kotkin, 1995). In these programs, student behavior is reviewed every 30 minutes, and each student receives a color card corresponding to how well he or she did. For example, each student starts the period with a red card (the color representing optimal behavior). Following a minor infraction, the card color changes to a yellow; following a major infraction the color changes to a blue. Color strips are either attached with Velcro or inserted in paper pockets on a board with the students' names down one side and class periods listed across the top. Color earnings are totaled once or twice per day (twice for younger children, once for older children). Earnings are exchanged for graduated activities and privileges (e.g., red earns choice of most desirable activities, blue earns fewest choices). Weekly rewards based on daily earnings are also provided.

Point totals also can be tracked with a battery-operated electronic "counter" with a number display kept at the student's desk. The teacher decreases point values on the display via a remote transmitter. Such

a device, called the Attention Trainer™, designed by Mark Rapport and commercially developed and marketed by Michael Gordon (Gordon Systems, DeWitt, NY; *www.gsi-add.com*), has received strong empirical support (DuPaul, Guevremont, & Barkley, 1992; Evans, Ferre, Ford, & Green, 1995; Gordon, Thomason, Cooper, & Ivers, 1990). The device can be faded out over 4–6 weeks and replaced by a less intensive class token system or self-monitoring program, or by a home–school report card (described later).

Response cost has also been implemented in a group format. In one procedure, a self-contained class was given 30 tokens (poker chips) each day at the beginning of a 90-minute period. A token was removed contingent upon each occurrence of an interruption by any student. Tokens were counted at the end of the period; the teacher exchanged remaining tokens for 1 minute of reading time. Significant reductions in interruptions occurred, and most of the students rated the program very favorably (Sprute, Williams, & McLaughlin, 1990).

Response cost raffles also have been successful in reducing mild disruptive behavior of junior high school students (Proctor & Morgan, 1991). A response cost raffle was recently applied to reduce misbehavior in the cafeteria at lunchtime (Fabiano et al., 2008). At the beginning of each lunch period, classes were given six lottery tickets. Throughout the lunch period, lottery tickets were lost for violations of school rules. Students could also earn additional bonus lottery tickets for following rules during several random checks. Class tickets were entered into a daily school lottery. Each morning, two class winners were drawn from the previous day's remaining tickets, and those classes earned a privilege (e.g., extra recess, walk outside). This procedure greatly reduced rule violations during lunchtime.

As with other punishment procedures, response cost is most effective when it is applied immediately, unemotionally, and consistently. When delivered in this way, response cost is as effective as token reward programs. In addition, teachers' and children's attitudes about response cost programs appear to be as positive as they are for reward programs. However, special efforts should be made to continue monitoring and praising appropriate behavior when response cost programs are in effect, to avoid excessive attention to negative behavior. When rewards and response cost are used together, the opportunity to earn tokens should be greater than the possibility of losing them, to avoid negative earnings (i.e., below zero). In the case of aggressive or very coercive behavior, teachers may be reluctant to administer the procedure right away because they fear that the behavior will escalate. However, response cost needs to be implemented consistently and immediately to be effective. Escalation may be minimized by reducing the amount of the "cost" when the student does not lose control.

Time-Out

Time-out from positive reinforcement (i.e., "time-out") is often effective for children with ADHD who are particularly aggressive or disruptive. This procedure involves the withdrawal of positive reinforcement contingent upon inappropriate behavior. Several variations of time-out can be used in the classroom, including the following:

- Removal of adult or peer attention by removing the child from the area of reinforcement or the opportunity to earn reinforcement. This may involve having the child sit in a three-sided cubicle or sit facing a dull area (e.g., a blank wall) in the classroom.
- Removal of materials, as in the case of having children put their work away (which eliminates the opportunity to earn reinforcement for academic performance) and their heads down (which reduces the opportunity for reinforcing interaction with others), for brief periods of time.
- Using a "good-behavior clock" as implemented by Kubany, Weiss, and Slogett (1971). In this procedure, rewards (e.g., small trinkets, candy) are earned for a target child and the class, contingent upon the child's behaving appropriately for a specified period of time. A clock runs whenever the child is on task and behaving appropriately, but it is stopped for a short period of time when the child is disruptive or off task.
- Instituting a "Do a Task" procedure, in which the child is instructed to complete a specified number of simple worksheets while in time-out. When the teacher issues a task time-out, the child is to proceed immediately to an isolated desk at the back of the class, count out the specified number of worksheets, complete them, then put them on the teacher's desk, after which the child returns to his or her usual seat.
- The key ingredient to all variations of time-out is "swift justice." That is, the speed with which

teachers invoke time-out immediately following misbehavior is primarily what makes it effective, rather than the length of the time-out interval to be served.

Most time-out programs set specific criteria that must be fulfilled prior to release from time-out. Typically, these criteria involve the child's being quiet and cooperative for a specified period during time-out. In some cases, extremely disruptive children may fail to comply with the standard procedure, either by refusing to go to time-out or by not remaining in the time-out area for the required amount of time. To reduce noncompliance in these cases, (1) a child can earn time off for complying with the procedure (i.e., the length of the original time-out is reduced); (2) the length of time-out can be increased for each infraction; (3) the child may be removed from the class to serve the time-out elsewhere (e.g., in another class or in the principal's office); (4) a response cost procedure can be used, wherein activities, privileges, or tokens are lost because of uncooperative behavior in time-out; (5) work tasks, such as simple copying or marking tasks, can be made contingent on failure to follow time-out rules; and (6) the child can stay after school to serve time-out for not being cooperative in following time-out rules during school hours. The use of this last procedure, however, depends on the availability of personnel to supervise the child after school. We do not recommend the loss of recess time as a punishment given evidence that physical exercise benefits children with ADHD in reducing and coping with their symptoms (Pontifex, Saliba, Raine, Picchietti, & Hillman, 2013).

Overall, time-out appears to be an effective procedure for reducing aggressive and disruptive actions in the classroom, especially when maintained by peer or teacher attention and adequate levels of positive reinforcement are available in the classroom. Time-out may not be effective when the inappropriate behavior is due to a desire to avoid work or be alone, in which case the time-out may actually be reinforcing. It is important that time-out be implemented with minimal attention from teacher and peers. When a child's problem behavior consistently escalates during time-out and requires teacher intervention (e.g., restraint) to prevent harm to self, others, or property, alternative procedures to time-out may be indicated. Overall, procedural safeguards and appropriate reviews are important to ensure that time-out is used in an ethical and legal way (Gast & Nelson, 1977).

Suspension

Suspension from school is sometimes used as punishment for severe problem behavior, but it may not be effective for students with ADHD. The use of suspension violates several critical features of effective punishment: It is not immediate; it is not brief; and it may not remove rewarding activities (many children may find staying at home or full day day care more enjoyable than being in school). Also, suspension should not be used when parents either do not have the appropriate management skills needed for enforcement or they may be overly punitive or abusive. In a study of inner-city public school students, Atkins and colleagues (2002) found that detentions and suspensions were apparently ineffective for children who were aggressive, lacking in social skills, and highly hyperactive. In-school suspension programs, on the other hand, may be appropriate for particularly chronic, severe, intentional infractions (serious aggressive or destructive behavior) for which response cost, time-out, and reward programs have been ineffective. For example, in-school suspension may be effective as a backup consequence when a student fails to take a time-out or to accept a "cost" and becomes violent or seriously disruptive. If in-school suspension is used, then the suspensions should be short term (usually not more than a day or two) and have clear entry criteria and rules, and structured educational assignments for the student to do during the suspension period.

Minimizing Adverse Side Effects

Despite the overall effectiveness of negative consequences, adverse side effects may occur if they are used improperly. The guidelines presented in previous versions of this chapter are still timely and are reviewed here as well:

- *Punishment should be used sparingly.* Teachers who frequently use punishment to the exclusion of positive consequences may be less effective in managing children's behavior, due to a decrease in their own reinforcing value and/or because children are satiated or have adapted to the punishment. Excessive criticism or other forms of punishment may also cause the classroom situation to become aversive. As a result, children may begin to avoid certain academic subjects by skipping classes, or avoid school in general by becoming truant. Frequent harsh punishment may even acceler-

ate a child's overt defiance, especially when a teacher inadvertently serves as an aggressive model.

- *When teachers use negative consequences, they should teach and reinforce children for alternative appropriate behaviors incompatible with inappropriate behaviors.* This practice helps them teach appropriate skills and decreases the potential for the occurrence of other problem behaviors.
- *Punishment involving the removal of a positive reinforcer (e.g., response cost) is usually preferable to punishment involving the presentation of an aversive stimulus.* Use of the latter method, as exemplified by corporal punishment, is often limited for ethical and legal reasons.
- *Punishment should be administered privately.* Teachers who publically criticize a student may inadvertently increase the likelihood of rejection from the student's classmates and further damage the student's reputation among his or her peers.

As effective as contingency management programs are for improving children's behavior, additional procedures may be necessary to promote positive peer relationships. Mikami and colleagues (2013) developed a supplemental curriculum to a standard contingency management program which involved using a variety of strategies to increase social inclusion of children with ADHD. Strategies included developing positive student–teacher relationships by having warm, one-on-one interactions about personal interests, providing feedback privately, taking away points from children who ostracize others, promoting collaborative relationships and kindness to others, and daily awards to showcase student strengths valued by the peer group. Students in this condition, relative to those in the standard contingency management program, had similar behavior improvement but received more favorable ratings, reciprocated friendships, and positive messages from peers. These results indicate that teachers can play a crucial role in facilitating both positive behavioral gains and healthy peer relationships.

MAINTENANCE AND GENERALIZATION

The most effective approach for promoting improvement in children's behavior in all classes and periods (including recess and lunch) is to implement behavioral programs in all the settings in which behavior change is desired. To maintain gains, a number of procedures can be used. The most effective seems to be gradual withdrawal rather than abrupt removal of the classroom contingency programs. Gradual withdrawal of token programs may be accomplished by reducing the frequency of feedback (e.g., fading from daily to weekly rewards) and substituting natural reinforcers (e.g., praise, regular activities) for token rewards. One particularly effective procedure for fading management programs involves varying the range of conditions or situations in which contingencies are administered, in order to reduce a child's ability to discriminate when contingencies are in effect. The less the child is able to discern the changes in contingencies when fading a program, the more successful it appears to be. When a student is making the transition to a new class, it is wise initially to implement the same or a similar program in the new class, then fade it once the student's behavior is stable.

Self-management skills such as self-monitoring and self-reinforcement (to be described in a subsequent section) have also been taught in order to improve maintenance of gains from behavioral programs and to help prompt appropriate behavior in nontreatment settings. However, they are not effective in the absence of teacher supervision, and there is little evidence to suggest that they facilitate generalization across settings. At this time, it should be expected that specially arranged interventions for children with ADHD will be required across school settings and for extended periods of time over the course of their education, given the developmentally disabling nature of their disorder.

DAILY REPORT CARDS

Homebased contingency programs continue to be among the most commonly recommended interventions for students with ADHD. Briefly, these programs involve the provision of contingencies in the home, based on the teacher's report of the child's performance at school (Barkley, 2013a; Kelley, 1990). Teacher reports, often referred to as a "report card," list the target behavior(s) and a quantifiable rating for each behavior. Teacher reports should be sent home on a daily basis at first. As student behavior improves, the daily reports may be faded to weekly, biweekly, monthly, and in some

cases to the reporting intervals typically used in the school—although for many students with ADHD, report cards should be used throughout the year on at least a weekly basis.

The following points should be considered when designing and implementing Daily Report Cards for students.

Select important target behaviors. In-class behavior (sharing, interacting appropriately with peers, following rules) and academic performance (completing math or reading assignments) may be targeted along with homework, which is a common problem for students with ADHD who often have difficulty remembering to bring home assignments, completing the work, then returning the completed work to school the next day. We recommend including at least one or two positive behaviors that the child is currently reliably displaying, so that the student will be able to earn some points right from the beginning of the program.

The number of target behaviors may vary from one to as many as four or five. Targeting very few behaviors is suggested when first implementing a program (to maximize the child's likelihood of success), when few behaviors require modification, or when teachers have difficulty monitoring many behaviors.

The daily ratings of each target behavior should be quantifiable. Ratings may be descriptive (e.g., *poor*, *fair*, *good*), with each descriptor clearly defined (e.g., *poor* = more than three rule violations), or more specific and objective, such as frequency counts of each behavior (e.g., interrupted less than three times) or the number of points earned or lost for each behavior.

Students should be monitored and given feedback during each period, subject, or class throughout the school day. In this way, a student's difficulty early on can be modified later in the day. Note that Daily Report Cards that try to summarize the entire day in a single rating, such as with Smiley-grams (a single smiley face for an entire day), should be avoided in favor of frequent evaluations across the day. In order for students with a particularly high rate of problem behaviors to achieve success, they may initially be rated for only a portion of the day (e.g., 1 hour). As behavior improves, ratings may gradually include more periods/subjects, until the student is being monitored throughout the day. When students attend several different classes taught by different teachers, programs may involve some or all of the teachers, depending on the need for intervention in each of the classes. When more than one teacher is included in the program, a single report card may include space for all teachers to sign, or different report cards may be used for each class and organized in a notebook for students to carry between classes.

The success of the program requires a clear, consistent system for translating teacher reports into consequences at home. The student may take a new card to school each day, or the cards can be kept at school and be given to the student each morning, depending on the reliability of the parents to give the card out each day. Upon the child's return home, the parent immediately inspects the card and discusses the positive ratings first with the child. The parent may ask about any negative ratings, but the discussion should be very brief, neutral, and business-like (not angry!). The child is then asked to formulate a plan for how to continue earning positive ratings and avoid getting negative ratings the next day. (Parents are to remind the child of this plan the next morning before the child departs for school.) The parent then provides the child with a reward dependent upon his or her earnings. Some programs involve rewards alone; others incorporate both positive and negative consequences. However, when parents tend to be overly coercive or abusive, reward-only programs are preferable. At a minimum, praise and positive attention should be provided whenever a child's goals are met; however, tangible rewards or token programs are usually necessary. For example, a positive report card may translate into TV or computer time, a special snack, or a later bedtime, or into points as part of a token reinforcement system. Both daily rewards (e.g., time with parent, special dessert, TV time) and weekly rewards (e.g., movie, dinner at a restaurant, special outing) are recommended, although parents should understand that daily rewards are most important for motivating children with ADHD. Parents should be strongly encouraged to use rewards that are basic privileges and activities that the child enjoys—not elaborate or expensive items.

Parents and teachers should be involved in planning the Daily Report Card system from the outset to ensure the child's understanding and cooperation with the procedures. Older children and adolescents should be included in planning the program for the same reasons. Furthermore, goals and procedures are modified on an ongoing basis in accordance with student progress or the lack thereof. Stated differently, as the student shows progress, daily/weekly goals are changed to encourage further growth.

The following are several types of homebased reward programs that rely on daily school behavior ratings. The card shown in Figure 24.1 contains four areas of potentially problematic behavior for children with ADHD. Columns are provided for up to six different teachers to rate the child in these areas of behavior or for one teacher to rate the child multiple times across the school day. The teacher initials the bottom of the column after rating the child's performance during that class period and checking for the accuracy of the copied homework, to ensure against forgery. For particularly negative ratings, we also encourage teachers to provide for parents a brief explanation of the reason for the negative rating.

At home, at the end of the day, the parent then awards the child points for each rating on the card (0 = *no points*, 1 = *one point*, 2 = *two points*). The child may then spend these points on activities from a home reward menu.

A similar Daily Report Card system may be used when a student is having problems with peers during school recess periods. The card shown in Figure 24.2 is completed by the recess monitor during each recess period, inspected by the class teacher when the student returns to the classroom, and then is sent home for use, as in the earlier example, in a home point system. The class teacher can also instruct the student to use a "think aloud, think ahead" procedure just prior to leaving the class for recess. In this procedure, the teacher reviews the rules for proper recess behavior with the student, notes their existence on the card, and directs the student to give the card immediately to the recess monitor.

Overall, homebased reward programs can be an effective adjunct to classroombased programs for children with ADHD, and an extensive body of evidence supports their beneficial effects (Fabiano et al., 2010; Jurbergs, Palcic, & Kelley, 2008, 2010):

Classroom Challenge

Name: _____ Date: _____

Please rate child in areas below according to this scale:

2 = Very good

1 = OK

0 = Needs improvement

Class period/subject

TARGET BEHAVIOR	1	2	3	4	5	6
Participation						
Classwork						
Handed in homework						
Interaction with peers						
Teacher's initials						

Total points earned: _____

Homework for tonight (list class period by assignment:

FIGURE 24.1. A card for a home-based reward program targeting classroom behavior.

Recess/Lunch Challenge

Name: _____ Date: _____

Please rate this child in the following areas during recess and lunch. Use a rating of 3 = very good, 2 = OK, 1 = needs improvement.

	Recess or lunch		
	1	2	3
Keep hands and feet to self—donesn't fight, push, kick, wrongly touch, or take other's belonging.			
Follows rules.			
Tries to get along well with others.			
Recess/lunch monitor's initials.			

Total points earned: _____

Comments:

FIGURE 24.2. A card for a home-based reward program targeting recess/lunch behavior.

- The Daily Report Card intervention with home-based rewards offers more frequent feedback than is usually provided at school.
- Home–school communication programs afford parents more frequent feedback about their child's performance than would normally be provided and can prompt parents to reinforce a child's behavior or notify them when the child's behavior is becoming problematic and requires more intensive intervention.
- The Daily Report Card intervention with home-based rewards takes advantage of the type and quality of reinforcers available in the home and is typically far more extensive than those available in the classroom (a factor that may be critical for children and adolescents with ADHD, as reviewed earlier).
- Virtually any child behavior can be targeted for intervention with these programs.
- Daily Report Cards require somewhat less teacher time and effort than a classroombased intervention and may be particularly popular with teachers who are concerned that use of classroom rewards for only some students is unfair.
- More than 70% of students with ADHD can be expected to show behavioral improvement within the first month of a Daily Report Card program, with additional gradual improvement over the course of several months of treatment (Owens et al., 2012).
- In many, but not all, cases, when a Daily Report Card program leads to symptom reduction, children also show improvement in academic functioning (Owens, Johannes, & Karpenko, 2009).

Note, however, that effective implementation of Daily Report Cards is not a simple procedure (Abramowitz & O'Leary, 1991). All of the behavioral skills needed for developing and implementing classroom contingency programs are also needed for use of Daily Report Cards; in addition, teachers need to work effectively with parents. Both teacher and parent must understand basic behavioral strategies, how to select and rotate rewards, and the need for consistency (i.e.,

teachers need to implement the program every day and parents need to provide rewards exactly as specified). In addition, plans should be established for handling students who attempt to subvert the system by failing to bring home a report, forging a teacher signature, or failing to get certain teacher signatures. To discourage this practice, missing notes or signatures should be treated the same as a "bad" report (e.g., the child fails to earn points or is deprived of privileges or points). In cases where parents may be overly punitive or lack skills to followthrough with consequences, their implementation of appropriate consequences should be closely supervised (possibly by a therapist), or other adults (e.g., school counselors, principal) may implement the program.

HOMEWORK STRATEGIES

Given the academic and behavior difficulties experienced by students with ADHD, it is not surprising that many of these students have problems completing homework on a consistent basis (Power, Werba, Watkins, Angelucci, & Eiraldi, 2006). Typical homework-related difficulties include not writing down assignments, not bringing assignments home, not completing assignments in an accurate fashion, arguing with parents about completing homework, and failing to turn in assignments on time (Power, Karustis, & Habboushe, 2001). Problems with homework completion are critical because homework success is positively related to academic achievement (Cooper, Robinson, & Patall, 2006). Thus, various intervention programs have been developed to address homework difficulties (e.g., Olympia, Jenson, & Hepworth-Neville, 1996).

For example, Power and colleagues (2001) developed and pilot-tested a homework program specifically for students with ADHD. Their Homework Success program for parent groups involves seven, 90-minute sessions. Because the program is parent-mediated, it may be incorporated into an ongoing parent education program or a stand-alone program. The strong collaborative component of this program also includes teachers and children at appropriate points in treatment. For example, a parent–teacher conference is held at the beginning of the program to identify specific homework problems and to emphasize the importance of home–school collaboration.

The core components of the Homework Success program are goal-setting and contingency management procedures to encourage more consistent homework performance. Session topics include introducing the program, establishing a homework ritual, providing positive reinforcement, managing time and goal setting, using aversive procedures appropriately, anticipating future homework problems, and providing follow-up support. This is a skills-based program wherein parents are instructed in new strategies during each session. Parents are expected to implement prescribed strategies between sessions, and their success in doing so is reviewed at the beginning of each subsequent session. Power and colleagues (2001) emphasize the use of data to track changes in homework performance over time and provide several measures to facilitate feasible data collection. Although extensive research has yet to be conducted with this program, controlled case study data indicate that Homework Success leads to improvements in homework completion and accuracy (Resnick & Reitman, 2011).

COGNITIVE TRAINING AND SELF-REGULATION INTERVENTIONS

Self-regulation interventions, which include self-monitoring, self-reinforcement, as well as cognitive training in self-instruction and problem solving, were originally developed to treat directly the impulsive, disorganized and noneflective manner in which students with ADHD approach academic tasks and social interactions. With their emphasis on the development of selfcontrol, it was thought that these interventions would reduce the need for extrinsic rewards and result in better maintenance and generalization of gains made by children with ADHD than those achieved with contingency management programs.

Unfortunately, cognitive training (e.g., self-instruction and problem solving) has been ineffective in changing behavior or improving school functioning for students with ADHD. Reviews of the cognitive training literature using meta-analyses have typically found the effect sizes to be only about one-third of a standard deviation and, in many studies, even less than this (Baer & Nietzel, 1991; DuPaul & Eckert, 1997; Dush, Hirt, & Schroeder, 1989). Although such treatment effects may occasionally rise to the level of statistical significance, they are nonetheless of only modest clinical importance and usually are to be found mainly in relatively circumscribed laboratory measures (e.g., Brown et al., 1986) rather than more clinically impor-

tant measures of functioning in natural settings. Thus, school-based cognitive training is not recommended as a treatment for children and adolescents with ADHD.

Alternatively, self-regulation strategies have had some success with students with ADHD. In self-monitoring and self-reinforcement, students monitor and evaluate their own academic and social behavior, and reward themselves (often with tokens or points) based on those evaluations. Training typically involves teaching students how to observe, record, and evaluate their own behavior to determine whether they deserve a reward. Students may be prompted to observe their own behavior by a periodic auditory signal (tone) or visual cue (teacher's hand gesture), and they are trained to record instances of appropriate behavior. Accuracy of student ratings is usually assessed by comparing these ratings against the teacher's records.

At least two meta-analyses shed light on the effects of self-regulation interventions for students with ADHD. Reid, Trout, and Schartz (2005) examined 16 studies conducted between 1974 and 2003 that focused on four types of self-regulation strategies (self-monitoring, self-monitoring plus reinforceAvment, self-management, and self-reinforcement). Effect sizes for on-task behavior, inappropriate behavior, and academic accuracy and productivity were large, with similar results across the four types of self-regulation intervention. Interpretation of these findings is limited due to high variability in effect sizes across studies, lack of inclusion of unpublished studies, mixing of effect size calculations across within-group and single-subject designs, and small sample size, which precluded statistical comparison of effect sizes across the four self-regulation intervention categories.

DuPaul and colleagues' (2012) recent meta-analysis of school-based intervention studies for students with ADHD identified a total of eight cognitive-behavior intervention studies (out of a total of 60 outcome studies); the majority of which ($k = 6$) were single-subject design studies. Five of these six studies evaluated self-regulation (rather than cognitive) strategies. Effect sizes for behavioral and academic outcomes were large. In fact, the behavioral effect size for cognitive-behavioral interventions (i.e., self-regulation strategies) was significantly greater than that found for contingency management and academic interventions. As was the case in the Reid and colleagues (2005) meta-analysis, findings from DuPaul and colleagues are limited due to the small number of self-regulation outcome studies, the near exclusive use of single-subject designs, and the lack of larger sample, controlled trials of this intervention approach.

To illustrate the use of a self-regulation intervention for students with ADHD, Hoff and DuPaul (1998) conducted a controlled case study of a self-regulation program for three children exhibiting significant ADHD-related behaviors in general education classrooms. These children participated in several treatment phases, beginning with a teacher-managed token reinforcement program and proceeding through successive stages of self-evaluation and self-reinforcement (i.e., a modification of procedures first reported by Rhode, Morgan, & Young, 1983). Prior to the first stage of self-evaluation, each student was trained by the teacher to recognize target behaviors associated with ratings from 0 (*broke one or more rules entire interval*) to 5 (*followed classroom rules entire interval*). The teacher modeled these behaviors for the child and also role-played target behaviors, while stating the rating associated with the behavior. During the first stage of self-evaluation, student and teacher independently rated the student's performance during one academic period. Ratings were compared (1) if the student's rating was within one point of teacher's, the student kept the points he or she gave him- or herself; (2) if the student's rating matched the teacher's rating exactly, the student received the points he or she gave him- or herself plus one bonus point; and (3) if student and teacher ratings deviated by more than one point, then no points were awarded. As in the token reinforcement phase, points were exchanged for preferred activities on a daily basis.

During successive stages of the treatment, the frequency of teacher–student matches was gradually reduced to 0%. For example, during the 50% match stage, in a coin flip following each rating period, the student was required to match the teacher an average of 50% of the time. Given that the outcome was random and unpredictable, the student could not assume prior to the coin flip that he or she did not have to match the teacher's rating. On the occasions when the student's rating did not have to match that of the teacher, the student automatically kept the points he or she gave him- or herself. Generalization across school settings was programmed for and systematically evaluated. All three students were able to maintain behavioral improvements initially elicited under token reinforcement despite the fading of teacher feedback. It is important to note that by the end of the study, the students continued to provide written ratings of their performance and continued to receive backup contingencies. The

ideal outcome would be for written ratings to be faded to oral ratings, while phasing out backup contingencies.

Self-monitoring and self-reinforcement strategies are the most promising self-regulation interventions; however, complete transfer of management of a program from teacher to student is unrealistic in most cases because continued teacher monitoring of the ratings is necessary to ensure honest reporting. Most gains are achieved from self-regulation programs when the training is of sufficient duration and there is overlap between the skills taught during training and the requirements of the classroom or playground. Training is required in all settings in which selfcontrol is desirable, both for students and for the adult supervisors (e.g., teacher, recess monitors), and adults need to encourage children's application of the skills in day-to-day activities in each setting. In fact, self-regulation programs are best used in conjunction with teacher-administered consequences, such as a token reinforcement program. In this context, self-regulation programs for academic and behavioral goals are relatively simple to implement, and they may further motivate student's participation and facilitate partial or full fading of contingency programs.

ORGANIZATIONAL SKILLS TRAINING

Organizational skills deficits often underlie common academic problems in ADHD and include deficits such as misplacing or losing personal belongings, failing to record assignments, forgetting assignments, and difficulty prioritizing assignments and workload. These deficits are unlikely to be normalized by medication or behavioral treatments, which suggests that direct skills instruction may be needed. As discussed earlier, self-management strategies can be applied to improve functioning in these areas.

Formal organizational skills training programs for children and adolescents with ADHD also have been developed and evaluated over the past several years. These programs generally focus on strategies for improving school-related organizational tasks, such as managing and preparing materials, managing time, and homework. Children are taught specific skills, such as how to record assignments and due dates, organize school papers into binders, use checklists for materials needed, track time required to complete the task, and break tasks into steps and self-monitor steps completed using checklists. Rewards are provided at school and/or home for meeting organizational goals (e.g., consistently utilizing a school binder). Versions have been developed for upper elementary school (Abikoff et al., 2013) and for middle school (Langberg, Epstein, Becker, Girio-Herrera, & Vaughn, 2012), and have been embedded in several multicomponent programs (see below). The version developed for third to fifth graders, referred to as organizational skills training (Abikoff et al., 2013) includes 20 hour-long sessions, two times per week in a clinic setting. Parents and teachers are trained to prompt and reward children's use of the skills (e.g., recording assignments and due dates, organizing school papers into binders, and using checklists for materials needed). In a randomized clinical trial, this skills training approach was as effective as an approach emphasizing contingency management, which involved working mostly with parents and teachers on strategies for rewarding their children for achieving individualized organizational goals at home and at school (e.g., assignments completed on time, desk/cubby neat and organized, bringing home all materials needed to do homework, backpack packed by bedtime). Both types of treatments, skills training and contingency management, resulted in significant improvement on parent and teacher ratings of organizational skills, academic performance, homework, and family functioning; thus, both appear to be viable treatments for addressing children's organizational problems.

An organizational skills training program developed by Langberg and colleagues (2012) for middle school students is called the Homework, Organization, and Planning Skills (HOPS) intervention. It includes 16 session (20 minutes per session) over 11 weeks and also includes two parent sessions for behavior monitoring and contingency management at home. HOPS focuses on strategies for improving physical organization, followed by homework management, time management, and planning skills. Parents reported gains as a result of the intervention and children's grade point averages (GPAs) also increased. Gains were attributable more to use of the structured binder organization system than to the time management and planning training (Langberg et al., 2012). The manual for this intervention has been published (Langberg, 2011), and outcome studies suggest that the intervention can be effectively implemented with relatively little training by school-based mental health professionals.

MULTICOMPONENT SCHOOL–HOME BEHAVIORAL TREATMENTS FOR ELEMENTARY AGES

In the past several years, additional programs incorporating school and home components have been developed for the elementary school-age group. The Child Life and Attention Skills (CLAS) program targets students with the inattentive presentation of ADHD (Pfiffner et al., 2007). The program emphasizes adaptive skills, functional competence, and compensatory strategies; uses cues, prompts, and routines; and involves teachers and parents to provide necessary environmental supports at school and at home. Parents and children attend eight concurrent group meetings and up to four family meetings, and teachers attend five consultation meetings with a therapist and each child's family. Children are taught a series of modules focused on independence (homework/study skills, self-care skills, getting chores done, routines, organization, and time tools), and social skills (making friends, handling teasing, assertion, accepting, being a good sport, and problem solving). The parent group focuses on strategies to support their children's use of these skills at home and at school. Teacher consultation meetings focus on development and implementation of an individualized Daily Report Card and classroom accommodations specific to concerns of each child (e.g., additional time or "time challenges" to complete work, preferential seating, reduction in workload, use of assignment book, use of completed work folder). Target behaviors are based on the needs of each child and typically include academic work (e.g., completion of assigned work, completion and return of homework, accuracy of completed work), work behavior/study skills (e.g., following directions, having necessary materials to begin work, getting started on work, staying on task), and social interactions (e.g., entering peer groups, accepting consequences, being a good sport, using assertive behavior). Skills taught in the child group are shared with teachers, so that the children's use of these skills can be reinforced (often by including them as targets on the classroom challenge) at school. Parents are taught a set of transferable skills for working with their child's teachers in the future. Results support efficacy of the program (Pfiffner et al., 2007, in press). Based on the success of CLAS, Pfiffner and her colleagues extended this program in order to target the more general ADHD population, with the objective of establishing a sustainable implementation method in a public school setting (Pfiffner et al., 2011). The adapted intervention, the CLS program, discussed earlier in this chapter, is designed to be implemented at the child's school by mental health personnel. Initial findings reveal broad benefits relative to attention, academics, organization, social behavior, and classroom engagement (Pfiffner et al., 2011, 2013; Villodas, McBurnett, Kaiser, Rooney, & Pfiffner, 2014).

Additional studies also support a model of multicomponent school-home intervention delivery in the school setting. As discussed earlier, Power and colleagues (2012) expanded the focus of traditional parent training programs and developed the FSS program, a family-mediated educational intervention. Positive outcomes for family–school relationships, homework, and parenting occurred relative to a support and education program for parents even though about 40% of the youth were already taking medication. Owens, Murphy, Richerson, Girio, and Himawan (2008) evaluated a school-based program that included Daily Report Cards, behavioral parenting sessions, teacher consultation, and individual child sessions, and found positive effects on children's ADHD symptoms and disruptive behaviors at school, their relationships with parents and teachers, and family and classroom functioning. Although not specifically designed for children with ADHD, the First Step to Success program, another intervention in which school personnel implement a parent training component focused on skills for success and a contingency management program at school (Seeley et al., 2009), also improves a variety of social, behavioral, and academic outcomes.

The version of this chapter in the preceding edition of this volume also described several programs to serve students with ADHD and more severe problems in self-contained full-day settings. The Summer Treatment Program (STP; Pelham & Hoza, 1996; Waschbusch, Pelham, Gnagy, Greiner, & Fabiano, 2008) has been implemented with success for a number of years. Several sites around the country are currently providing this manual-based intervention; it was implemented as a part of psychosocial treatment in the Multimodal Treatment Study of ADHD (MTA), and a year-round version is operational. The program includes intensive behavioral programs (a point system with daily and weekly rewards, time-out, SST) implemented by special education teachers during computer, art, and academic learning centers, and by five counselors as children par-

ticipate in recreational activities (soccer, swimming) (Hoza, Vallano, & Pelham, 1995). The STP is associated with clinically significant gains across multiple areas of functioning (academic, social, and behavioral) for the majority of children participating in the program while it was in operation (Chronis et al., 2004). However, evidence is lacking as to whether these gains are maintained after departure from the program or generalize to untreated school settings.

TAILORING SCHOOL-BASED PROGRAMS FOR ADOLESCENTS

Many of the school-based strategies described to this point apply as much to adolescents with ADHD as to children. However, there are important developmental considerations for adolescents. As reviewed in previous chapters, impairments during the teen years include many of the same problems found during childhood (e.g., poor grades, incomplete work, peer relationship problems) in addition to some new ones, such as poor class attendance, high potential for school dropout, delinquency, substance abuse, and risky driving behaviors. Importantly, adult supervision occurs far less often for adolescents than for younger children; adolescents spend a significant amount of their time unsupervised. Middle school and high school also bring challenges due to the increased number of teachers involved in middle and high school; the short duration of the class periods; the greater emphasis on individual self-control, organization, and responsibility for completing assignments; and the frequent changes that occur in class schedules across any given week. Teachers at large secondary schools often teach more than 100 different students per day and spend relatively little time with any one student. All of this is likely to result in a dramatic drop in educational performance in many children with ADHD after the elementary grades. It is very easy for the average adolescent with ADHD to "fall through the cracks," especially since many, if not most, adolescents with ADHD are not included in special education. It is at this age level that educational performance becomes the most common reason adolescents with ADHD are referred for clinical services (Chapter 6). To add to the challenges, middle and high school teachers often expect independence from their students and may be far less eager than elementary school teachers to implement recommended intervention strategies (DuPaul & Weyandt, 2006).

To better address changes in development and school structures, a number of adaptations can be considered during the teen years. First, behavioral programs should involve teens as collaborative partners in treatment. Their buy-in is critical given that they will be on their own to implement various aspects of the intervention and many of the problematic settings are also unsupervised by adults. Self-management strategies can be used to a greater extent, and more successfully, with teens given their increased cognitive capacity for self-monitoring. As reviewed earlier, strategies that focus on teens' concrete organizational goals may effectively improve a range of classroom preparatory behaviors. Contingency management programs remain important but require greater sophistication and involvement of the teen. For example, behavioral contracts are recommended instead of stars and tokens. School-based contracts and school–home contracts (e.g., Daily Report Cards described earlier) are often useful. Contracts should specify clear expectations and incentives, and like token economies, should emphasize clear links among specific behavioral goals (e.g., target behaviors), the time frame in which these goals need to be achieved, and rewards (Wolraich & DuPaul, 2010). Rewards can take the form of privileges at school or at home (e.g., homework pass, cell phone access, screen time). Crucially, teens should be involved in the design of the program (logistics, types of rewards) so that they are invested in its success. Key also to the success of these programs is that a parent and teacher(s) who have daily contact with the teen be taught to support the delivery of behavioral programs, organizational systems, and the like, since without some adult oversight, teens are less likely to use the strategies.

Steps for Working with Adolescents and Their Middle and High Schools

Given the various complexities discussed earlier, we have listed a number of steps in the attempted to manage poor educational performance and behavioral adjustment problems in middle and high school.

1. The clinician should determine whether the adolescent has been identified for services through the IDEA or Section 504 (or, if in private school, whether the student has been identified for similar programs). If this has never been determined, or not within the past 3 years (federal law requires a reevaluation every 3 years for a child in special education), this should be con-

sidered. Special educational services will not be forthcoming until this evaluation is completed, and this can take up to 90 days or longer in some districts. Without formal identification, accommodations are often not forthcoming.

2. Adolescents with ADHD should be provided with education/counseling on the nature of their condition. Although many may have been previously told that they are "hyperactive" or have ADHD, they may not understand what this means. This counseling is not intended to discourage the adolescents by bringing up what they cannot do; rather, it is to help them become more accepting of the extra supports and strategies that will be needed.

3. It is often essential at the beginning of each academic year at a teen's school to schedule a team meeting that includes the teachers, school psychologist or guidance counselor, principal (if available), parents, *and the adolescent with ADHD.* The clinician should briefly review the nature of ADHD (with a handout) and the need for close teamwork among the school personnel, parents, and teen to improve the teen's academic performance. Each teacher should describe the teen's current strengths and problems in his or her class and suggest how they might help with current problems (e.g., being available after school a few days each week for extra assistance; reducing the length of written homework assignments; allowing the teen to provide oral means of demonstrating his or her acquired knowledge rather than relying on just written, timed test grades; developing a subtle cueing system to alert the teen when he or she is not paying attention in class, without drawing the whole class's attention to the fact). At this meeting, the teen should be asked what he or she is going to strive to do to make school performance better. By the end of the meeting, there should be clarity regarding targets for improvement. These may include academic, behavioral, or social targets. A written contract may be helpful. Once plans are made, the team should agree to meet again in 1 month to evaluate the success of the plans and troubleshoot any problem areas. Future meetings may need to be scheduled, depending on the success of the program to date. At the least, meetings twice a year are to be encouraged even when a program is successful, so as to monitor its progress and keep the school attentive to the needs of this teen. The adolescent should attend these meetings.

4. School-based accommodations based on the needs of the student should be identified. Common accommodations for teens (as for the children in the earlier discussion), include preferential seating, modified assignments, testing accommodations, and systems for tracking homework, academic, and behavior progress. Many schools are now using Web-based portals to provide class and homework assignments, grades on tests/quizzes, assignments, and feedback about behavior, if necessary. If teachers update student progress each day, then parents and teens can check the portals daily, and provide home-based reinforcement for meeting goals. However, in our experience, these portals are used inconsistently both within and across teachers. If the portals are not updated in a way that lets the parent know the assignment and the teen's performance that day, the clinician should introduce the idea of a structured binder for recording daily homework assignments verified by each teacher. A written school–home contract (e.g., the Daily Report Card described earlier) also can be introduced at the meeting as a way to track academic and behavior goals. For example, the teacher might complete ratings about predetermined target behaviors specified on the contract. In conjunction with this, a home point system might be set up to include a variety of desired privileges the teen can purchase with the points earned for meeting target goals at school (based either on teacher reports on the portals or a Daily Report Card). Such things as use of a cell phone or the family car, time-out of the home with friends, extra money, clothes, downloads, computer time, special snacks kept in the house, and so forth, can be placed on the program. Points can also be set aside in a savings book, so that the teen can work toward longer-term rewards. However, the daily, short-term, accessible privileges and not these longer-term rewards are what give the program its motivational power. Thus, the reward menu should not include too many long-term rewards. Once the adolescent is able to achieve goals for 3 weeks or so, then the contract can be faded to a once- or twice-per-week schedule of completion. After a month of satisfactory ratings, the contract can either be faded out or reduced to a monthly rating. Assignments and grades should be closely tracked by parents throughout the school year.

5. Ideally, the school will provide a second set of books to the family (even if a small deposit is required to do so), so that the teen can still do homework even if he or she forgets a book required for homework. Alternatively, parents can purchase an extra set; usually, used copies are available through various websites.

6. A mentor or coach is recommended to provide daily check-ins to keep the student on track. The mentor or coach may be one of the teen's classroom teachers, the homeroom teacher, the school guidance counselor, or a learning disabilities teacher. This person's role is to meet briefly with the teen to help keep him or her organized. The number and timing of check-ins should be tailored to the needs of the student. Check-ins should be brief but interspersed during the school day. For teens with milder difficulties, one check-in at the end of the day may be sufficient. For more severe problems, additional interventions may be warranted to provide ongoing coaching, monitoring, and feedback (see below).

7. Getting a private tutor for the teen may be beneficial. Many parents find it difficult to do homework with a teen or to provide tutoring in areas of academic weakness. The teen often resists these efforts as well, and the tension or arguments that can arise may spill over into other areas of family functioning even after the homework period has passed. When this is the case and the family can afford it, hiring a tutor to work with the teen even twice a week can be of considerable benefit in both reducing the teen's academic weakness and "decompressing" the tension and hostility that arise around homework in the family. Alternatively, online courses can be accessed. Khan Academy (*www.khanacademy.org*) is an excellent resource with a self-paced format that often works well for teens with ADHD.

8. Parents should set up a special time each week to do something alone with their teen that is mutually pleasurable, so as to provide opportunities for parent–teen interactions that are not task-oriented, school-related, and fraught with the tensions that work-oriented activities can often create with teens who have ADHD. This can often contribute to keeping parent–teen relations positive and counterbalance the conflicts that school performance demands frequently create in such families.

MULTICOMPONENT PROGRAMS FOR TEENS

The Challenging Horizons Program (CHP) developed by Evans, Schultz, DeMars, and Davis (2011) is a comprehensive school-based treatment program for adolescents with ADHD. The CHP incorporates many of the features discussed earlier. Two versions of the program exist: an afterschool model and a consulting model implemented during most of the school year (usually at least 6 months). In the afterschool version, students attend the program for 2 hours, 2-4 days per week (Evans et al., 2011). The program comprises two separate group interventions for improving social and educational skills, and 20-minute one-on-one meetings with a counselor to review progress, practice specific skills, and maintain a therapeutic relationship and connection with the program. The counselors work with each student's parents and teachers to ensure that appropriate problems are being addressed and to facilitate generalization from the afterschool program to school and home. In the consulting model, school staff members (teachers, administrators, counselors) implement interventions similar to those in the afterschool program during the school day through consultation with study staff. A school counselor serves as a mentor for each student to review progress and practice skills, just as the counselor does for students in the afterschool program. Both versions include several interventions for educational skills similar to those reviewed previously in this chapter. Every student uses an assignment notebook for homework, with school and home contingencies for its correct use. Teachers initially sign the book for accuracy, but these signatures are tapered as students become more independent. Students are also taught note-taking and study skills; they practice these first in the CHP, and are then required to show that they use the skills at school and at home. An individualized homework plan, which requires mandatory daily time for homework and use of rewards, is developed with the parents. Disruptive behavior is acknowledged within each group session; other contingencies are added, if needed. Daily or weekly report cards are implemented, if needed, to address problem behaviors at school or at home. The social skills intervention includes social problem solving, recognition of social cues, and skills development. Students view and evaluate videotapes of their own behavior during group sessions to facilitate their learning. A number of studies support the efficacy of the intervention, and the CHP is currently recognized on the Substance Abuse and Mental Health Services Administration (SAMHSA) National Registry of Evidence-Based Programs and Practices.

Few school-based programs have been developed and evaluated at the high school level. One promising approach is a modified coaching version of the previously described CHP (Evans, van der Oord, & Langberg, 2013). The program includes parent training, interpersonal skills Group, and in-school coaching. Parent

and teen groups are held in the evening; the parent group focuses on communication and problem-solving, and developing a homework management plan; the teen group focuses on establishing individual social-interaction goals, and extending these goals to actual social situations, while learning to interpret and utilize feedback of others regarding their success in reaching these goals. The in-school coaching component is provided by CHP coaches (BA level) who meet with teens regularly for 20 minutes or so over the course of the entire school year to provide ideas for academic-focused interventions for the teens to select and implement. Results show reduced inattention, better family functioning and some improved school grades from this program; crucially, gains were greatest for students who attended most sessions. A strength of this program is that it was integrated during school hours, allowing for greater accessibility to sessions.

To address more severe student problems, the STP, originally developed for children with ADHD, has been expanded for adolescents (STP-A; ages 11–16) with ADHD. Like the child version, the adolescent version is conducted over 8 weeks, Monday through Friday, from 8:00 A.M. to 5:00 P.M. The STP-A includes modules to mimic a secondary school setting. Academic core modules include various subjects (Health, Science, History, Creative Writing) during which time skills for note taking, summary writing, critical listening, studying, partner work, and quiz taking are taught, and provided activities target direction following, planning, and on-task behavior. Academic support modules focus on development of organizational skills (e.g., daily planner use, binder organization, homework). Vocational modules incorporate training in job skills, and social skills modules include skills drills and social performance. A behavior tracking system, daily feedback, and Daily Report Cards are utilized, with home and program rewards. Parents participate in weekly, group-based parent training. Individualized treatment plans are tailored to specific needs. Initial pilot studies support the value of this program (Sibley et al., 2011).

MANAGING SCHOOL-BASED NEEDS OF PRESCHOOLERS WITH ADHD

Although preschool educational programs are typically play-oriented and offer more free-choice activities than later school programs, the early reading and math tasks, social activities, and art projects that comprise schooling of young children require sustained attention and compliance with rules for short periods of time. Thus, young children who exhibit ADHD-related behaviors can significantly disrupt structured activities, transitions from one activity to another, and group interactions (e.g., circle times). Furthermore, preschoolers who are highly active and more impulsive than their peers may have difficulties sharing, waiting their turn, and controlling frustration even during less structured activities such as free play. Thus, given the typical early onset of ADHD symptoms, it is important for school personnel, particularly those working with young children, to be aware of (1) how ADHD manifests in early childhood, (2) how to identify young children at risk for ADHD, and (3) how to design programs that reduce symptomatic behaviors and enhance academic, social, and family functioning.

Although issues such as rapid developmental changes between ages 2 and 6 make diagnosis of preschool-age children somewhat tenuous (Lahey et al., 1998), research indicates that symptoms of ADHD emerge at a very young age (e.g., Egger, Kondo, & Angold, 2006; Strickland et al., 2011) and are associated with significant deviations in brain structure (Mahone et al., 2011). Furthermore, the behavioral symptoms of ADHD exhibited by preschool-age children mirror those of older children with respect to prevalence, presentation, and gender differences. As is the case with older children, ADHD in preschoolers is associated with significant impairment in behavioral, social, and early academic functioning; affected children are approximately two standard deviations below their typically developing peers in all three areas (DuPaul, McGoey, Eckert, & VanBrakle, 2001), and for many preschoolers, this impairment persists across childhood (Lahey et al., 1998). This research strongly supports the early emergence of a constellation of symptoms characteristic of ADHD that is atypical of preschool-age children, associated with significant and chronic impairment across settings, and requires early identification and intervention.

Prior comprehensive reviews of treatment for young children with or at risk for ADHD have examined studies of psychotropic medication, parent education programs, and school-based behavior modification (Charach et al., 2010, 2013; Ghuman, Arnold, & Anthony, 2008; McGoey, Eckert, & DuPaul, 2002). In general, it appears that, as is the case for older children with ADHD, classroom interventions based on behavioral principles are effective in reducing disruptive be-

haviors in preschool settings (e.g., McGoey & DuPaul, 2000). Unfortunately, definitive conclusions regarding the efficacy of behavioral and academic interventions for preschool-age children with ADHD are premature because relatively few studies have investigated the effects of these intervention strategies in preschool classrooms. Furthermore, many of the "classroom" interventions studied thus far have been examined in laboratory rather than preschool settings, limiting the generalizability of obtained results. In addition, the preschool-based ADHD intervention literature is limited by small sample sizes, lacks assessment of treatment integrity, and provides minimal treatment follow-up and generalizability data (McGoey et al., 2002). Furthermore, preschool interventions have been applied using a "one-size-fits-all" approach that is based on the assumption that all young children with ADHD will respond similarly to a particular intervention. More work is necessary along the lines of that by Boyajian, DuPaul, Wartel Handler, Eckert, and McGoey (2001), who designed preschool interventions using functional assessment data. The latter allow individualization of behavioral interventions in an attempt to optimize outcomes.

SUMMARY AND CONCLUSIONS

Advances in our knowledge of effective school interventions for ADHD continue to be made. Research studies clearly indicate that behavioral, academic, and self-regulation interventions should be considered first when designing school-based programs for students with ADHD. Like behavioral interventions, medication treatment is also considered to be a "well-established" treatment for ADHD. The combination of the two treatments is likely to be optimal in more serious cases of ADHD. However, it is important to note that when given in sufficiently intensive doses, behavioral interventions produce significant improvements in symptom management in their own right; this may lead to less need for medication, or lower doses of medication, in the school setting. Similarly, the behavioral intervention may be less intense when medication is simultaneously delivered (Fabiano et al., 2007). The following points should be considered when working with schools to improve the academic, behavioral, and social effectiveness of children with ADHD.

As we reported in the previous edition of this volume, adequate training of teachers in the implementation of evidence-based behavioral interventions still remains elusive. This also extends to school-based mental health professionals. Although progress is being made, greater training in evidence-based approaches prior to entry into teaching and mental health professions is needed. In addition, adequate resources for school personnel and funding of services continue to be concerns. Unfortunately, although mental health insurance plans often cover non-evidence-based treatments such as play therapy, they typically do not cover evidence-based behavioral interventions in schools. Educational systems face serious challenges in funding training for teachers and the needed resources for interventions. Further advances in helping students with ADHD achieve success at school and beyond depend on the continued development of cost-effective, evidence-based programs and on solving the critical problems in terms of lack of resources and training for teachers and mental health professionals, so that these programs can be widely available in schools across the country.

KEY CLINICAL POINTS

- ✓ Teachers do not always have adequate knowledge of evidence-based interventions, so school-based interventions should incorporate information to improve educators' basic knowledge about the nature, causes, course, and treatments for ADHD.
- ✓ Collaboration between home and school is critical so as to produce a more uniform, consistent, and effective plan of management that incorporates the major caregivers. Multicomponent treatments that include parents, teachers, and youth provide the most comprehensive approach and likely result in the greatest yield across all domains of difficulty for youth with ADHD.
- ✓ Core school-based interventions described in this chapter derive from the understanding that ADHD disrupts self-regulation, executive functioning, and motivational systems; thus much of the extra "structure" that children with ADHD so often require is specifically aimed at redressing these executive weaknesses and motivational deficiencies.
- ✓ Core accommodations and interventions for ADHD require (1) altering the physical classroom layout and structure, as needed; (2) modifying academic tasks and using computer-assisted instruction to match

each child's abilities and deficits; (3) improving academic readiness skills; (4) altering teacher-delivered consequences (attention, reprimands, tokens, time-outs, etc.) for appropriate and inappropriate conduct, while minimizing adverse side effects; (5) collaborating with parents to enhance in-school outcomes and promote maintenance and generalization of gains outside of school setting; (6) using peers to facilitate academic success and behavioral control; (7) developing home-based reinforcement programs (Daily Report Cards); (8) striving to enhance self-monitoring and self-management through self-regulation approaches; and (9) modifying these approaches based on the developmental level of the child or teen.

✓ Formal special educational services under the IDEA and Section 504 may also be required for a child with ADHD if prereferral accommodations are not sufficient.

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CHAPTER 25

Dietary Management of ADHD

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It has long been recognized that health depends on adequate dietary intake of all macronutrients and micronutrients uncontaminated by noxious substances. More recently this appreciation has expanded to mental health. Disorders such as myxedema madness (iodine deficiency), Wernicke's encephalopathy (B vitamin deficiency), psychosis of B_{12} deficiency, and cognitive impairment from lead poisoning have long been known to be related to dietary adequacy, but attention is now turning to subtler issues. These include genetically based differing needs for various nutrients, genetically differing sensitivity to dietary components, and mild dietary insufficiencies, complicated by changes in modern diets from those found throughout most of human evolution. An additional complication is medication, which can "waste" nutrients (as with some anticonvulsants) or interfere with appetite (as with U.S. Food and Drug Administration [FDA]–approved stimulants for attention-deficit/hyperactivity disorder (ADHD). We review in this chapter the various dietary strategies and supplements hypothesized to help ADHD, either as alternatives or adjuncts to established treatments. We do not consider herbs, which, although often classified as dietary supplements, are actually crude over-the-counter drugs.

The evidence for these complementary and alternative treatments varies by treatment from open trials to multiple placebo-controlled trials. Their difficulty, expense, and risk also vary widely. Therefore, clinicians need a way of deciding whether a given treatment makes clinical sense. One method is the SECS versus RUDE guideline (Arnold, Hurt, Mayes, & Lofthouse, 2011); a treatment that is safe, easy, cheap, and sensible does not need as much evidence to justify a trial for an individual patient as one that is risky, unrealistic, difficult, or expensive. Note that any of the latter four characteristics increases the needed quality and quantity of evidence to justify a patient trial.

DIETARY ELIMINATIONS

Elimination Diets

The treatment of ADHD symptoms with an elimination diet is based on the hypothesis proposed by Feingold (1975) that some children are sensitive to certain dietary substances, including artificial food colors, flavors, preservatives, dietary salicylates, and potentially natural food proteins, and that reactions to these substances contribute to ADHD symptoms. Elimination diets (including few-foods, defined, or oligoantigenic diets) involve avoidance of foods or substances to which the child may be sensitive for 1 to 3 weeks, then reintroducing foods/substances systematically and assessing

the child's reaction. Typical foods removed in an elimination diet include dairy products, gluten, citrus fruits, corn and corn-containing products, all processed foods, and caffeine. Two recent meta-analyses have evaluated the efficacy of restricted/elimination diets (REDs) for children with ADHD. Nigg, Lewis, Edinger, and Falk (2012) reported that the response rate in 14 open trials was 47%; however, response rates across trials varied significantly ($p < .001$). In six controlled trials, the response rate was similar (41.5%); however, one trial (Pelsser et al., 2011) was an outlier (for a comprehensive critique of the methodological flaws in this outlier study, see Barkley, 2012). When this study was appropriately excluded from analyses, the response rate was only 33%. With all six studies, there was a medium aggregate effect size ($g = 0.58$); however, with the outlier study removed, the effect size was small ($g = 0.29$). The authors concluded that benefit from elimination diets on ADHD were "potentially clinically meaningful" (Nigg et al., 2012, p. 90) and deserved further investigation. Sonuga-Barke and colleagues (2013) included seven randomized controlled trials (RCTs) of RED (one trial included results for two distinct age groups) and reported separate results from the most proximal assessments (rating/observation closest to therapeutic setting) and the probably blinded assessments (rating by the rater who was most likely to be blind to treatment assignment). With all seven trials included (with eight analyses), the standard mean difference (SMD) for the most proximal assessments was large (1.48) and significant ($p = .01$). Five studies (six analyses) included probably blinded assessments (excluding two by Pelsser and colleagues [2009 and 2011]); for these five blinded studies the SMD was medium (0.51), and only marginally significant ($p = .06$).

The following considerations and clinical recommendations appear to be applicable:

- A RED typically eliminates the following foods: dairy products, gluten, citrus fruits, corn and products that contain corn, and all processed foods, but most children are not sensitive to all of these, if any.
- For nutritional balance, substitutions are often needed for dairy products and gluten/other grains if the child is found to be sensitive. Substitutions include the following:
  - For dairy products: soy milk, soy cheese, rice milk, rice-based ice cream.
  - For gluten/other grains (wheat/barley/oat/rye): brown rice, buckwheat, spelt, millet, potatoes, sweet potatoes.
- First assess the child's current dietary patterns. If the foods to be eliminated make up the bulk of the child's current dietary pattern, parents may first want to consider increasing the child's variety of foods to include foods that are allowed in the RED.
- Parents should also assess how much control they will have over the child's dietary intake. If the child is eating the RED at home but continues to eat restricted foods at school, day care, or friends' houses, results from the RED will be inconclusive. Parents may need to plan the initial RED period for a time when they have the most control of the child's diet (e.g., summer vacation, winter break).
- Preliminary evidence suggests that RED may be most effective for motivated families that have a positive family environment prior to its initiation. Therefore, the family environment should be assessed prior to initiation of the RED. If the family has difficulty with organization and structure, these skills may need to be remediated prior to the initiation of the RED (e.g., with organization skills training) to ensure the intervention is followed systematically.
- Similarly, a child's compliance with parental rules should be considered. If mealtimes are already challenging and the child is particularly resistant to change, parents may first want to consider parent training in behavior management and/or family therapy to improve the child's compliance to rules/requests, to increase the child's flexibility/tolerance for change, and to improve parent–child communication. On the other hand, if the child's oppositional defiant behavior is due to or aggravated by a dietary sensitivity, it may improve in a week with an enforced RED; this may be particularly true for the youngest children.
- It is generally recommended that the RED be followed for a couple of weeks to allow for noticeable effect (if any occurs). Following a strict elimination diet for longer than a month without significant benefit is not recommended given time, effort, and family resources required and the risk of nutritional imbalance.
- If the child ingests caffeine regularly (tea, coffee, many "soft" drinks), withdrawal symptoms from caffeine may last a few days. If caffeine withdrawal is a concern, parents may want to plan the initial few days of the RED for a time period in which the child's sched-

ule is relatively free (i.e., a weekend, instead of a week during which the child has examinations).

- If the child's behavior improves during the initial elimination period, restricted foods should be added back one at a time (allowing a day for each) to assess behavior change. If no significant behavioral worsening occurs, that food may remain in the diet. If the child's behavior worsens, that food should be eliminated indefinitely. If the results are equivocal, rechallenge after another week.

- The long-term effects of elimination diets have not been evaluated. Only foods that definitely cause behavioral worsening should be eliminated.

- Parents should be cognizant of their children's nutritional intake and ensure they are ingesting all needed nutrients, even with limited food variety. A Recommended Dietary Allowance/Reference Daily Intake (RDA/RDI) multivitamin/mineral supplement may be prudent. Support from a physician and/or dietician during an elimination diet is recommended.

Restriction of Artificial Food Colors and Preservatives

A modified, less stringent strategy is the elimination or minimization of artificial food colors (AFCs) and preservatives from the diet. Both previously discussed meta-analyses (Nigg et al., 2012; Sonuga-Barke et al., 2013) also evaluated the specific effect of the reduction of AFCs on ADHD symptoms; in general, for these studies, children were placed on an additive-free diet, then challenged with AFC/preservatives or placebo. Sonuga-Barke and colleagues (2013) found a pooled SMD (similar to Cohen's *d*) of 0.32 (p = .02, a small effect) when using the most proximal assessments; a small to medium SMD was found (0.42; p = .004) when using probably blinded assessments. Nigg and colleagues (2012) reported a small effect size (g = 0.18) for parent-reported ADHD symptoms, although teacher-reported ADHD symptoms were nonsignificant (g = 0.07). Notably, the effect size for psychometric tests of attention was significant, though small (g = 0.27; p = .007). Furthermore, the impact of AFCs was not significantly different in children who had been preselected to be sensitive to food additives.

Although elimination diets and restriction of food additives are often discussed as a complementary/alternative treatment for children with ADHD or clinical levels of ADHD symptoms, there is emerging evidence that the negative effects of food additives are not restricted to a clinical population. Two studies (Bateman et al., 2004; McCann et al., 2007) evaluated the effect of food additives in a nonclinical population of preschool-age (age 3) and school-age children (ages 8–9). All children were given an additive-free diet for 1 week, then challenged with a mix of AFCs and sodium benzoate versus placebo. Both studies reported significantly more severe ADHD symptoms when children were challenged with the mix than when challenged with placebo. Neither the presence of clinically significant ADHD symptoms nor the presence of atopy moderated the effect of AFCs/benzoate on child behavior. Thus, the effect of AFC/preservatives may not be limited to children diagnosed with ADHD, and all children, even those without diagnosed mental health problems, may benefit from restricted intake of food dyes. In fact, the government of the United Kingdom requested that manufacturers substitute natural food colors for AFCs, and the European Union has asked companies to voluntarily eliminate AFCs from foods and beverages or to add a warning label. In 2011, the FDA Advisory Committee convened a meeting regarding the research on AFC; it concluded that there was not enough evidence to recommend banning AFC or requiring a warning label. It is noteworthy that the per capita consumption of AFCs has quadrupled in the United States in the last 50 years.

Given current evidence, the following clinical recommendations have been made:

- Although avoiding food dyes/preservatives is not as restrictive, onerous, or complex as a full elimination diet, a high level of organization, including reading all labels, and child compliance with dietary change will still be required. The recommendations about family organization mentioned earlier therefore also apply here. Because minimization of AFC intake is desirable for all children, it may be easier to avoid AFCs in the whole family food supply, so the child with ADHD does not need special rules.

- Versions of some processed foods that typically contain food dyes/preservatives may be available in AFC/preservative-free alternatives in the natural foods sections of most grocery stores. In addition, recently, Kraft Foods Group announced that it would remove Yellow No. 5 and Yellow No. 6 from certain macaroni and cheese products in the United States. If additional

food manufacturers follow this lead, there may be more options for AFC/preservative-free food in the United States, which will significantly increase the ease of this intervention for all children.

Sugar Restriction

A third dietary restriction strategy is the simple elimination of sugar from the diet. Although many parents believe that sugar ingestion leads to problematic behavior, research has not supported this claim. A well-controlled 3-week trial of a sugar-restricted diet found no effect on children's behavioral and cognitive functioning (Wolraich et al., 1994). A subsequent meta-analysis by the same research group (Wolraich, Wilson, & White, 1995) of 23 double-blind, placebo-controlled studies concluded that sugar did not significantly affect the behavioral or cognitive functioning of children. However, results from a classroom study of children not selected for ADHD symptoms found an increase in inattentiveness during the morning if the children had not eaten breakfast (Wesnes, Pincock, Richardson, Helm, & Hails, 2003). This decline in attention was exacerbated by a glucose-drink breakfast, but it was attenuated by a same-calorie breakfast of whole-grain cereal with milk. Thus, although sugar restriction cannot be recommended as a *treatment* for ADHD, an appropriate minimization of high-sugar foods and beverages is considered to have overall health benefits without risk and passes SECS criteria as general good practice. Children can be provided snacks of nuts, jerky, fruit, carrots, or celery sticks, and so forth, in place of candy, soda pop, popsicles, donuts, cookies, and so forth.

NUTRITIONAL SUPPLEMENTS

Nutritional supplementation is the opposite of elimination diets, which are based on the assumption that ADHD symptoms are caused by something in the diet that is noxious to the child and should be removed. Supplementation is based on the hypothesis that a lack or insufficiency of something in the diet is contributing to ADHD symptoms, and that the substance should be added. We discuss three categories of nutritional supplementation for ADHD: macronutrients (polyunsaturated fatty acids, glyconutritional saccharides, and amino acids), micronutrients (selected vitamins and minerals), and metabolites (L-carnitine, and dimethylaminoethanol [DMAE]).

Macronutrients

Essential Fatty Acids

Polyunsaturated fatty acids (PUFAs) with the first unsaturated bond at the third carbon from the noncarboxyl "tail" (omega-3 or *n*-3) and at the sixth carbon from the tail (omega-6 or *n*-6) are considered "essential," because mammals cannot synthesize them and they must be consumed, like vitamins and minerals. They are necessary for neuronal membrane structure, fluidity, and function, which provide a nest for receptors. They also constitute the substrate for prostaglandin and other eicosanoid production. The important omega-3 PUFAs are eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA), which can be anabolized from alpha-linolenic acid (ALA) by intact metabolism. In the omega-6 series, gamma-linolenic acid (GLA) and arachidonic acid can be anabolized from linoleic by intact metabolism. Both omega-3 and omega-6 have been reported to be lower in children with ADHD than in typically developing comparison children, and the severity of ADHD symptoms in diagnosed children correlates inversely with PUFA levels (Stevens et al., 1995).

Two meta-analyses have recently evaluated the efficacy of fatty acid supplementation for ADHD symptoms (Bloch & Qawasmi, 2011; Sonuga-Barke et al., 2013). Bloch and Qawasmi (2011) included 10 double-blind, placebo-controlled trials of omega-3 fatty acid supplementation in children diagnosed with ADHD or with clinically significant ADHD symptoms. They found a small but significant effect of omega-3 supplementation on ADHD symptoms (SMD = 0.31; $p < .0001$). Significant treatment effects were reported for both inattentive (SMD = 0.29; $p = .009$) and hyperactive symptoms (SMD = 0.23; $p = .005$). EPA showed a dose–response of increased efficacy. However, this dose–response could have been an artifact of higher EPA dosages occurring in studies that used a triple combination of EPA, DHA, and GLA (of the omega-6 series). It is not clear whether it was the higher EPA dose or the mixture or both that made the difference. Sonuga-Barke and colleagues (2013) included 11 clinical trials of omega-3 and/or omega-6 supplementation in children diagnosed with ADHD or who met clinical cutoffs on rating scales. They also found small but significant effects of PUFA supplementation on ADHD symptoms by both the most proximal assessments (SMD = 0.21; $p = .007$) and probably blinded assessments (SMD = 0.16; $p = .04$).

A concern of many alternative treatments is the lack of safety data. Manor and colleagues (2013) reported safety data for a double-blind, placebo-controlled, 15-week trial of omega-3 supplementation followed by a 15-week open-label extension. There were no significant differences in adverse events between omega-3 supplementation and placebo. In the open-label phase, only 5/102 children had adverse events that were judged "probably related" (including gastrointestinal distress, headache, insomnia, high triacylglycerides). No adverse effects on weight and growth were observed with omega-3 supplementation.

Two double-blind, placebo-controlled studies evaluated PUFA supplementation as a complementary treatment to stimulant medication (Behdani, Hebrani, Naseraee, Haghaghi, & Akhavanrezayat, 2013; Voight et al., 2001). In both studies, PUFA supplementation did not significantly enhance the therapeutic benefit of stimulants according to parent ratings, teacher ratings, and laboratory measures of inattention and impulsivity. All children in Voight and colleagues (2001) were considered medication responders; thus, there may not have been enough room to demonstrate the benefit of adding PUFA. Although children in the study by Behdani and colleagues (2013) were not medicated at enrollment, it is not clear whether they were all stimulant-naive. The study found that those in the methylphenidate (MPH) + placebo group had significant reduction in symptoms, indicating it is likely that most of the sample would be considered stimulant responders. Thus, PUFA supplementation is not likely to be a beneficial adjunct *for children who respond well to stimulants*. No study has evaluated whether PUFA supplements would allow a reduction of optimal stimulant dose.

On the other hand, Perera, Jeewandara, Senviratne, and Gurage (2012) evaluated PUFA as a complementary treatment for children who had an unsatisfactory response to MPH and behavior therapy (all children had Swanson, Nolan, and Pelham–IV (SNAP-IV) item mean scores > 1.89 after 6 months of evidence-based treatment). Children were randomized to receive PUFA versus placebo for 6 months; all participants continued MPH and behavioral intervention. At 6 months, those randomized to PUFA did significantly better than those taking placebo, based on parent report. Thus, adjunctive PUFA seems *beneficial for children whose response to evidence-based treatments is unsatisfactory*.

Based on the evidence reviewed, the following clinical recommendations seem appropriate:

- PUFAs pass the SECS criteria for treatment of ADHD.
- PUFAs are probably not necessary if the child eats three servings of oily wild ocean fish weekly.
- Between 1 and 2 grams (containing at least half a gram of EPA) appears to be effective and safe.
- Make sure there is adequate intake of antioxidant vitamins (E, C) because PUFAs are especially prone to oxidation.
- Make sure the fish oil label has USP (U.S. Pharmaceopeial Convention) on it or indicates that the fish oil has been refined to eliminate mercury.
- Caution the patient and family that it takes 3 months to get results, so they need to be patient.
- PUFA supplementation may be an appropriate complementary treatment for children who have not responded to evidence-based treatments (medication and behavior therapy).
- PUFA supplementation probably does not provide additional benefit for children who have an excellent response to traditional medication. However, it may conceivably allow maintenance of benefit at reduced dose (there are no data supporting this yet).
- If fish or krill oil alone is not satisfactory, addition of a small amount of GLA may be useful, either by switching to a commercial triple combination (EPA, DHA, and GLA) or by adding a *small amount* of evening primrose oil.

Amino Acids

Amino acid supplementation has been suggested as an ADHD treatment because children with ADHD have lower levels of amino acids (Baker, Bornstein, Rouget, Therrian, & van Muden, 1991; Bernstein, 1990) and exhibit nitrogen wasting (Stein & Sammaritano, 1984), which suggests poor use of protein. Although some short-term benefit for ADHD has been seen from tryptophan, tyrosine, and phenylananine, which are necessary for neurotransmitter production (e.g., Nemzer, Arnold, Votolato, & McConnell, 1986), symptoms return within 2 months (Wood, Reimherr, & Wender, 1985). In addition, the "supply-side metabolism" may pose risks from toxic catabolic products resulting from increased neurotransmitter turnover. Given the lack of sustained benefit and potential risks, processed/refined amino acid supplementation does not currently pass SECS criteria and is not currently recommended

for children with ADHD. Instead, they should eat a diet with adequate protein, which would compensate for any nitrogen wasting and ensure substrate for neurotransmitter production. A possible exception to the failure to meet SECS criteria may be S-adenosylmethionine (SAMe), which is not a neurotransmitter precursor like the others, but is involved in redox metabolism; it deserves further research.

Glyconutritional Supplementation

Glygonutritional supplements, derived from vegetables, contain basic saccharides, which are essential for cell communication and the formation of glycoprotein and glycolipids. In two open trials (Dykman & Dykman, 1998; Dykman & McKinley, 1997), significant improvement was reported for ADHD symptoms and oppositional behavior, and gains were maintained for 6 weeks. However, glyconutritional supplementation has not been evaluated in controlled trials. Given the current lack of efficacy and safety data, glyconutritional supplementation is not recommended at this time.

Vitamin Supplementation

There are four strategies for vitamin supplementation for ADHD symptoms in diagnosed and nondiagnosed samples: RDI/RDA multivitamins, megadoses of single vitamins, megadoses of multiple vitamins, and multiple mixtures of intermediate doses.

Although some reports suggest that there are mild deficiencies in blood levels of vitamins in children with ADHD, there has not been an open or controlled trial of RDI/RDA vitamin supplementation in a diagnosed sample. Two placebo-controlled trials in a nondiagnosed classroom sample found improvement in nonverbal cognitive skills (Benton & Cook, 1991; Benton & Roberts, 1988). Improved concentration and decreased fidgeting were reported in the second trial (Benton & Cook, 1991); however, improvements may be limited to children with poor diets (Benton & Buts, 1990). A 7-month placebo-controlled trial (Crombie et al., 1990) with 11- to 13-year-old typically developing children did not find a significant advantage of vitamins over placebo in reasoning tasks. Given some research support and lack of risk, except in the case of rare genetic disorders, RDI/RDA supplementation passes the SECS criteria. Like sugar reduction, RDI/RDA supplementation should not be considered a stand-alone *treatment* for ADHD, but complementary supplementation can be recommended, especially when stimulant-based appetite suppression threatens dietary balance and adequate micronutrients.

Despite encouraging pilot data (e.g., Brenner, 1982; Coleman et al., 1979), megadoses of single vitamins have not been adequately studied. In three placebo-controlled trials, no benefit was found for megadoses of multiple vitamins in either short (2 weeks) or longer (6 months) trials for children with ADHD and learning problems (Arnold, Christopher, Huestis, & Smeltzer, 1978; Haslam, Dalby, & Rademaker, 1984; Kershner & Hawke, 1979). Given the lack of research support and concerns regarding safety (e.g., hepatotoxicity, peripheral neuropathy) megadoses of either single or multiple vitamins cannot be recommended at this time.

A possible compromise is a mixture of multiple vitamins and minerals in amounts that are larger than RDA/RDI levels but within limits considered safe by published standards. A recent placebo-controlled RCT in adults in New Zealand (Rucklidge, Frampton, Gorman, & Boggis, 2014) indicated that relative to placebo, supplementation with micronutrients significantly improved self- and observer- rated ADHD symptoms on a standardized ADHD rating scale and on clinician's global ratings of improvement in ADHD symptoms and overall functioning. Although these results are promising, this study will require replication with a pediatric sample before specific recommendations may be made for multi-micronutrients in larger than RDA/RDI doses for children and adolescents with ADHD.

Mineral Supplementation

Iron, magnesium, and zinc have been proposed for supplementation because of reported greater deficiencies in these minerals in children with ADHD than in controls (Kozielec, Starobrat-Hermelin, & Kotkowiak, 1994).

Iron Supplementation

Two samples of children with ADHD were reported to have iron deficiencies (Cortese, Konofal, Bernadina, Mouren, & Lecendreux, 2009; Konofal et al., 2007); however, the deficiency may be limited to children with comorbid disorders (Oner & Oner, 2008). Two small pilot trials of iron supplementation have been completed. In an open trial of 17 nonanemic school-age chil-

dren, ADHD symptoms were significantly reduced after 30 days of 5mg/kg/day iron supplementation (Sever, Ashkenazi, Tyano, & Weizman, 1997). In a 12-week, randomized, placebo-controlled pilot trial, supplementation with 80 mg/day ferrous sulfate significantly decreased ADHD symptoms of 23 young children (ages 5–8) with ADHD and borderline deficient iron status (Konofal et al., 2008). Given the small samples of the two completed trials and concern regarding hemochromatosis (excess iron), iron supplementation in the treatment of ADHD requires further study. Thus, iron supplementation only passes the SECS criteria for children with ADHD who have documented insufficiency/deficiency, for which iron supplementation would be standard treatment.

Iron, the most common mineral deficiency in children, is especially likely to affect those in a rapid growth phase, such as preschoolers and adolescents. Menstruating girls are especially vulnerable. Because iron can be suboptimal without frank anemia, ferritin may need to be checked. Inflammation may artificially elevate ferritin, so if ferritin is normal, it is advisable to check C-reactive protein (CRP) and/or transferrin receptor.

Magnesium Supplementation

Magnesium deficiency can result from a variety of causes and is associated with a wide spectrum of psychiatric problems (Flink, 1981). Researchers who conducted three open-label trials in Europe reported significant decreases in ADHD symptoms following 1 to 6 months of supplementation (Mousain-Bosc et al., 2006; Mousain-Bosc, Roche, Rapin, & Bali, 2004; Nogovitsina & Levitina, 2007). In an additional European trial, Starobrat-Hermelin and Kozielec (1997) compared magnesium supplementation in children with ADHD and documented magnesium deficiency to a treatment-as-usual control group (it is unclear whether there was random assignment); similar to the open-label trials, parent-reported ADHD symptoms decreased significantly in the supplemented group relative to the control group.

In a Polish sample of 116 children with ADHD, 94% were found to be deficient in magnesium relative to laboratory norms (Kozielec & Starobrat-Hemelin, 1997). However, in an American sample of 70 children with ADHD, none of the children were found to be deficient in magnesium compared to laboratory reference ranges (unpublished data). Thus, additional information regarding magnesium deficiency in ADHD across different cultures is required. Future research should compare magnesium deficiency status to a control group without ADHD rather than to laboratory reference ranges. In order to further evaluate magnesium supplementation for ADHD, an RCT is needed. Animal studies on magnesium supplementation suggest a U-shaped behavioral response curve (Izenwasser, Garcia-Valdez, & Kantak, 1986); thus, children who are not deficient in magnesium may experience behavioral worsening with supplementation. Like iron supplementation, magnesium supplementation is currently recommended only for children with documented deficiency, for whom it would be a standard treatment.

Zinc Supplementation

Zinc is a cofactor for more than 100 enzymes. It is necessary for melatonin production and fatty acid absorption, and it regulates dopamine production. Relative to children without ADHD, children with ADHD have lower zinc levels (Bekaroglu et al., 1996), and zinc serum levels are negatively correlated with ratings of inattention (Arnold et al., 2005). In two Turkish RCTs of zinc supplementation for school-age children with ADHD (Bilici et al., 2004; Uckardes, Ozmert, Unal, & Yurdakok, 2009), zinc monotherapy was associated with improvements in ADHD symptoms relative to placebo, although the benefit of zinc supplementation in one study (Uckardes et al., 2009) was limited to children of mothers with only a primary school education.

Zinc supplementation is one of the few dietary treatments for ADHD that has been evaluated as a complementary treatment with stimulant medication. In an Iranian trial, children randomized to MPH + zinc sulfate improved significantly more on parent and teacher ratings of ADHD symptoms than did children randomized to MPH + placebo (Akhondzadeh, Mohammadi, & Khademi, 2004). However, an American trial of zinc supplementation did not confirm the benefit of zinc as an alternative (zinc monotherapy) or complementary treatment (zinc + stimulant) (Arnold, DiSilvestro, et al., 2011). In a study in which children were initially randomized to zinc monotherapy versus placebo, zinc was not significantly more effective than placebo. Following 8 weeks of zinc–placebo monotherapy, a standard dose of stimulant medication was added and optimized for 3 weeks. The addition of zinc did not augment the benefit of stimulant medication; however, the optimized dose of stimulant was 37% lower in children randomized to zinc supplementation than in

those randomized to placebo. Given these discrepant results and the risk of interference with iron/copper absorption, zinc does not pass the SECS criteria. Similar to iron and magnesium, zinc in higher than RDA/RDI amounts is recommended only for children with a documented zinc deficiency, for which zinc supplementation would be standard treatment.

Metabolites

Carnitine

Carnitine is a normal metabolite necessary for energy production, lipid transport into mitochondria, and PUFA elongation. It is considered a semiessential nutrient because most people do not make enough on their own (usually 25% of needed supply). There have been two published trials of carnitine supplementation for children with ADHD (Arnold et al., 2007; Van Oudheusden & Scholte, 2002). In a crossover study of 24 Dutch school-age boys diagnosed with ADHD, 13 boys were responders based on parent report, and 12 were classified as responders based on teacher report; however, mean scores for each treatment phase were not provided (Van Oudheusden & Scholte, 2002). Arnold and colleagues (2007) reported a randomized controlled pilot trial with 112 children diagnosed with ADHD. In the full sample, there was no significant improvement with carnitine supplementation relative to placebo; however, these results were moderated by ADHD subtype. Children with ADHD, inattentive type, improved significantly more on carnitine relative to placebo; children with ADHD, combined type, showed no significant difference with carnitine supplementation relative to placebo. Although current research support is minimal, given documented cardiac benefits, L-carnitine passes the SECS criteria for children with ADHD, inattentive type (now presented in DSM-5; American Psychiatric Association, 2013). The following clinical recommendations are offered:

- Carnitine supplementation should be tried only for ADHD, inattentive type. It is not recommended for ADHD, combined type, or ADHD, hyperactive–impulsive type.
- A 4-month trial is needed to evaluate effects.
- The dose used in the published trial was 1.5 grams twice daily, but if benefit is found, it may be possible to reduce the dose for maintenance.
- A possible side effect is fishy breath.

Dimethylaminoethanol

DMAE, also referred to as deanol and dimethylethanolamine, is a precursor to choline and may increase production of acetylcholine (Re, 1974). It was previously marketed as a drug for hyperactivity (Deaner). However, it was withdrawn after the FDA began requiring evidence of efficacy as well as safety. A recent review of open-label and placebo-controlled trials (Arnold, Hurt, et al., 2011) concluded that most trials had significant methodological flaws and outcomes were variable, with some showing superiority of DMAE, others showing superiority of placebo, and still others showing no effect. The best study is likely a three-group RCT comparing DMAE (500 mg), MPH (40 mg), and placebo (Lewis & Young, 1975). Relative to placebo, DMAE significantly reduced ADHD symptoms (d = 0.1–0.6), although not to the level of benefit found with MPH (d = 0.8–1.3). DMAE appears to pass the SECS criteria for children with mild symptoms of ADHD.

The following clinical guidelines are offered:

- DMAE is acceptable for children with mild symptoms of ADHD who have not responded well to medication or for children whose parents do not want them to use medication.
- Effects do not approach efficacy of stimulant medication; thus, children may require additional treatment, although the effectiveness of DMAE in combination with other treatments has not been established.
- It may have some advantages in terms of side effects: notably shorter sleep delay, less appetite suppression, and less of the evening rebound found with stimulants.
- An appropriate dose would be 250–500 mg twice or three times a day.

KEY CLINICAL POINTS

- ✓ Several proposed dietary treatments for ADHD target specific etiologies, which should be assessed prior to imitation of treatments.
- ✓ Families who are considering dietary management may benefit from meeting with a physician and dietician, in order for the child to receive a comprehensive physical examination and dietary history.
- ✓ Based on the medical evaluation, a complete blood

count and evaluation of specific electrolytes–minerals–vitamins may be valuable as a general screen to pick up any mineral–vitamin deficiencies. In particular, if the child is dark-skinned or spends little time in the sun, especially if presenting in late fall or winter, vitamin D is worth checking.

- ✓ Children in rapid growth phases (preschool and adolescence) may outgrow their iron stores and are particularly vulnerable to iron insufficiency, which is the most common mineral deficiency in children and adolescents.
- ✓ When indicated from the dietary history or if the child lives in areas of endemic deficiencies, a more complete mineral screening should also be completed.
- ✓ The risk of delaying evidence-based treatments should be considered if the dietary treatment is replacing a standard treatment. Many dietary treatments require several months to determine whether the intervention is effective. Therefore, parents should consider whether the child is in need of a standard treatment, which has the potential for a faster response.
- ✓ Diverting family resources (time, effort, money) to a dietary treatment that is not effective is another risk to be considered. Parents should discontinue treatments that obviously have no effect after a reasonable trial and consider a different dietary treatment, noningestible alternative treatment, or evidence-based treatment (FDA-approved medication and behavior therapy). These risks are particularly salient for elimination diets, which require a considerable amount of effort and organization.
- ✓ When trying any treatment, including dietary treatments discussed in this chapter, parents are encouraged to change one thing at a time and log the results carefully. If the treatment does not have the desired effect in the expected time, parents should try a different treatment.
- ✓ The expected time varies by treatment, for example, within a few weeks for dietary eliminations, 3 months for PUFAs, and 4 months for carnitine. None works as quickly as stimulant medication.
- ✓ Although many parents may be tempted to initiate multiple treatments at the same time to increase the likelihood that something will be effective, it is not always the case that more treatment is better. One treatment could conceivably interfere with another, and parents may not have the time, energy, and focus to carry them all out well, resulting in none having an adequate trial. Furthermore, there have been no systematic studies evaluating the combination of these dietary interventions. Therefore, we recommend that one dietary treatment be tried at a time, with the exception of reasonable restriction of sugar and RDI/RDA supplementation, which should not be considered stand-alone treatments.

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CHAPTER 26

Executive Function Training for Children with ADHD

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The growing numbers of innovative, nonpharmacological treatments developed for children with attention-deficit/hyperactivity disorder (ADHD) in recent years reflect an emerging synergy between basic and applied science, and symbolizes how they reciprocally inform and advance the field. This chapter examines executive function training, one category of these emerging treatment interventions that has garnered widespread interest among clinicians, mental health professionals, and parents of children with ADHD. We summarize the accumulating evidence from neuroimaging studies initially to provide fundamental insights into why children with ADHD continue to experience significant impairment across a wide range of functional outcomes despite receiving the most efficacious treatments available over an extended time period. The ensuing section highlights the evolving literature on *executive functions* (EFs) and the extent to which particular EFs and/or their component processes are deficient and related to core clinical features (inattention, hyperactivity–impulsivity) and functional outcomes in children with ADHD. Afterward, we describe two broad categories of EF training programs, *facilitative intervention training* (FIT) and *mindfulness*. We focus on these two particular training programs because they share two common tenets: (1) that many of these children's behavioral, cognitive, interpersonal, and learning difficulties are outcomes of underdeveloped or poorly regulated EF processes, and (2) that these EF processes can be strengthened or corrected through training and extended practice.

Recent meta-analytic reviews of FIT and mindfulness are examined in a later section to examine the extent to which training results in significant gains within and across two types of outcome measures. The first of these involves the extent to which training specific EFs and/or integral attentional processes transfers to untrained tasks that rely on nearly identical cognitive processes (i.e., *near-transfer effects*). Training children's short-term memory (STM) with an adaptive digit span task and demonstrating that training improves performance on a word list recall task is an example of a near-transfer training effect. Documenting these effects is necessary to ensure that improvement is associated with training as opposed to task-specific factors associated with practice or expectancy effects, and it also helps to validate the mechanisms responsible for potential transfer to more distal (i.e., *far-transfer ef-*

fects) cognitive and behavioral outcomes (Shipstead, Redick, & Engle, 2012). Demonstrating far-transfer effects, however, is by far the more critical training objective given that the goal of FIT and mindfulness is not to improve children's scores on laboratory-based EF tasks, but to improve specific cognitive abilities and the myriad functional outcomes dependent upon these abilities. Far-transfer effects represent post-treatment improvements in abilities or behaviors that depend on the trained cognitive abilities and involve overlapping brain regions (Unsworth & Engle, 2007) but are assessed using dissimilar and qualitatively different tasks than used during training. Training children's working memory (WM) and demonstrating that training improves academic performance and/or interpersonal functioning that relies on WM are examples of far-transfer training effects. Theoretically, the degree of improvement on far-transfer measures is limited to a considerable extent by two factors: the magnitude of documented near-transfer change and the extent to which improvement on far-transfer outcome measures rely on these newly trained abilities (Redick et al., 2012). Stated differently, if a training program results in limited or insignificant improvement on tasks that are highly *similar* to those used during training, there is no obvious theoretical reason to expect improvement on far-transfer measures that require a combination of trained and untrained cognitive processes for successful performance. The degree to which a far-transfer outcome measure requires cognitive processes similar to those being trained can be estimated by examining the magnitude of the relationship between the two tasks or outcome variables. For example, if training focuses on improving visuospatial WM, and a fluid reasoning measure such as the Raven's Progressive Matrices is selected to assess far-transfer training effects, very little improvement would be expected even if visuospatial WM training were highly effective. This is because the type of fluid reasoning necessary to perform well on the Raven's Matrices only partly reflects a child's WM ability, as evidenced by the limited relationship (r = .42, or less than 18% of shared variance) between the two constructs in past investigations.

In the final section, we summarize extant evidence supporting FIT and mindfulness interventions. We also discuss what we believe represents the most critical issues that must be resolved to enable these types of interventions to bring about fundamental and lasting changes in the cognitive abilities and associated functional outcomes in children with ADHD.

WHY CURRENT TREATMENTS FAIL: INSIGHTS FROM NEUROIMAGING STUDIES

Children with ADHD are in dire need of innovative and effective treatments in light of the disheartening results of the Multimodal Treatment of ADHD study (see Chapter 28) documenting significant and continued impairment across a wide range of clinical, educational, and interpersonal outcomes after 3–8 years despite receiving the most potent, evidence-based treatments available for the disorder for an extended time period (Jensen et al., 2007; Molina et al., 2009). The failure of these treatments (individually titrated psychostimulant medication alone, intensive parent training and classroom contingency management alone, or their combination) to significantly improve the long-term functioning of children with ADHD is not altogether unexpected. Neither treatment was based on a theoretical framework of the disorder. Psychostimulants were discovered serendipitously by an astute physician noting improved concentration and reduced motor activity in children administered Benzedrine who suffered postpneumoencephalography[1] headaches. Contemporary parent and classroom contingency management (behavioral) therapies, in contrast, were appropriated from the widespread application of operant conditioning principles for individuals with developmental/intellectual disabilities beginning in the 1960s. When administered in their most potent forms and monitored carefully, psychostimulant medication alone and combined with behavioral treatment is associated with large-magnitude acute reductions in inattention and hyperactivity–impulsivity symptoms (effect size range = 1.53 to 1.89) that may last for up to 24 months if treatment is sustained (Van der Oord, Prins, Oosterlaan, & Emmelkamp, 2008). In contrast, psychosocial intervention used alone is associated with more moderate reductions in core symptoms (effect size range = 0.31 to 0.87; Fabiano et al., 2009; Van der Oord et al., 2008). These impressive reductions in core behavioral symptoms and impairment ratings, however, are not accompanied by significant or sustained improvements in ecologically valid academic and learning outcomes such as quiz and test grades, overall grade point averages, grade retentions, high school graduation rates, and standardized achievement test scores (Molina et al., 2009; Van der Oord et al., 2008). In addition, no study has demonstrated sustained maintenance of medication or psychosocial treatment-related behavioral changes beyond 24 months of therapy (Jensen et al., 2007; Molina et al., 2009).

The relative impotence of psychostimulant and intensive behavioral treatment to provide lasting improvements in academic and learning outcomes in children with ADHD once treatment is withdrawn warrants consideration if the field is to progress in designing innovative therapies for the disorder. Psychostimulants such as methylphenidate act primarily as dopamine and norepinephrine reuptake inhibitors, and to a lesser extent, as direct agonists that stimulate the release of dopamine and norepinephrine into the synapse. The well-documented finding that both processes promote the availability of these neurotransmitters in cortical–subcortical pathways involving the frontal/prefrontal cortex, temporal lobe, and basal ganglia is of particular relevance for the treatment of ADHD (see Dickstein, Bannon, Castellanos, & Milham, 2006, for a meta-analytic review). These anatomical structures play a critical role in supporting EFs, an umbrella term for higher-order cognitive processes such as WM, set shifting, and inhibitory control that enable goal-directed behavior and novel problem solving (Garon, Bryson, & Smith, 2008; Miyake et al., 2000). EF deficits are implicated in most contemporary models of ADHD (Barkley, 1997, 2012; Rapport et al., 2008; Willcutt, Doyle, Nigg, Faraone, & Pennington, 2005) and associated with adverse educational (Jensen et al., 2007), interpersonal (Kofler et al., 2011), and occupational outcomes (Barkley & Murphy, 2010).

Although psychostimulants usually result in moderate- to large-magnitude improvements on non-EF measures such as regulation of attention and response speed, smaller magnitude and nonsignificant effects are reported for tasks with a prominent executive component (Bedard, Jain, Hogg-Johnson, & Tannock, 2007; Epstein et al., 2006; Kobel et al., 2009; Rhodes, Coghill, & Matthews, 2006). These results suggest that actuating the anatomical structures underlying those EFs typically assessed by cognitive testing improves important aspects of the attentional and motor response elements related to children's task performance but fails to improve essential component processes related to more meaningful functional outcomes such as learning and academic performance.

Empirically supported behavioral treatments, in contrast, are based on the underlying assumption that ADHD-related impairment in school performance/learning and interpersonal relationships reflects inadequate social learning histories and/or underlying volitional control deficits that can be managed through the contingent application of learning principles such as reinforcement and response cost. Treatment contingencies focus conventionally on increasing attention, compliance, and academic productivity, and decreasing excessive gross motor activity and impulsive behavior. These selected targets are based on the expectation that strengthening and weakening desirable and undesirable behaviors, respectively, will result in enduring behavioral change. Extensive evidence supports the efficacy of operant techniques for acutely improving a wide range of behaviors in children with ADHD while contingencies are actively implemented (for a review, see Pelham & Fabiano, 2008). No study to date, however, has demonstrated sustained maintenance of conditioned behavioral changes over an extended time frame after treatment is withdrawn (Jensen et al., 2007; Molina et al., 2009) or the transfer of effects to EF-related cognitive performance outcomes, even when accompanied by inordinate incentives (Dovis, Van der Oord, Wiers, & Prins, 2012). Collectively, our current and most potent evidence-based therapies provide effective, short-term reductions of externalizing symptoms and improve some areas of functional impairment while treatment is active, but they minimally affect the EF deficits and adverse learning outcomes common to ADHD, especially once treatment is withdrawn.

Accumulating evidence from neuroimaging studies provides important insights into this enigma. Widely distributed hypoactivity in frontal–prefrontal cortical regions implicated in EF is well documented in children with ADHD (see Dickstein et al., 2006, for a meta-analytic review; see Chapter 14), and the relations among central nervous system (CNS) arousal, increased activity level, and task performance are well established (for reviews, see Barry, Clarke, McCarthy, Selikowitz, & Rushby, 2005; Rapport et al., 2008). The near-normalization of attention and gross motor activity observed with psychostimulants and incentivized behavioral interventions likely reflects the impact of these treatments on arousal-regulating mechanisms needed to activate EF-supporting structures within these brain regions (Cortese et al., 2012). Repeated resonance scans acquired prospectively from children with ADHD, ages 5–15, however, reveal a nearly 3-year delay in attaining peak cortical thickness in these same prefrontal–frontal regions relative to typically developing children (Shaw et al., 2007). Activating these regions is therefore unlikely to improve children's cognitive functioning and related learning outcomes due to the ontogenetically underdeveloped structures themselves and the EFs these structures support.

EFs AND FUNCTIONAL OUTCOMES

The clinical model of psychopathology, and by extension, the WM model of ADHD (Figure 26.1), hypothesize that interventions aimed at improving suspected underlying neurological substrate(s) and core psychological/cognitive features of ADHD should produce the greatest level and breadth of therapeutic change (Rapport, Chung, Shore, & Isaacs, 2001). Conversely, those aimed at improving peripheral behaviors should show limited generalization upward to core features, and minimally affect other peripheral symptoms. Novel interventions are therefore more likely to be successful if they target aspects of EF that not only are deficient in ADHD but also related to the primary behavioral, learning, and interpersonal difficulties associated with the disorder. In the ensuing sections, we summarize the empirical basis for designing novel treatments targeting each of the three, higher-order EFs (namely, *WM*, *behavioral inhibition*, and *set shifting*) and related attentional components, evidence for and against ADHD-related deficits in each EF, and research examining the role of each EF in ADHD behavioral symptoms and functional impairments.

Working Memory

Of the 25 cognitive training studies included in the Rapport, Orban, Kofler, and Friedman (2013) meta-analysis, 68% describe WM as a primary target for remediation, a finding that is consistent with mounting evidence documenting functional relationships among WM deficits and a broad range of behavioral and functional impairments in children with ADHD. As will become apparent, however, nearly all of these protocols primarily target STM rather than WM.

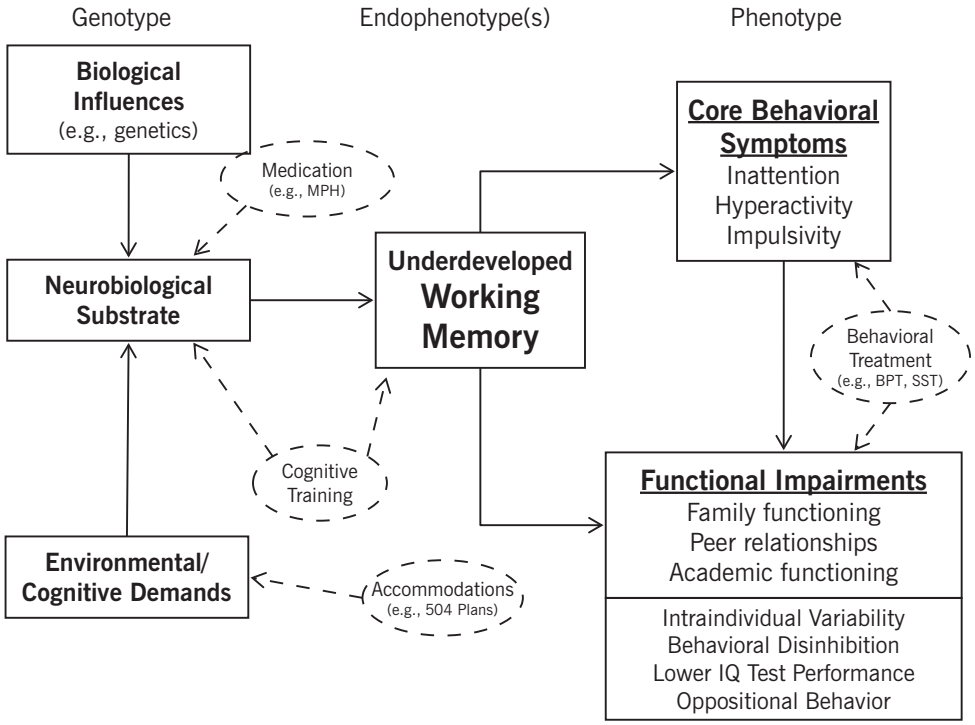

FIGURE 26.1. Visual schematic of the functional working memory (WM) model of ADHD. BPT, behavior parent training; MPH, methylphenidate; SST, social skills training. From Rapport, Chung, Shore, and Isaacs (2001). Adapted with permission from Mark D. Rapport, PhD.

WM is a limited-capacity system responsible for the temporary storage, rehearsal, processing, updating, and manipulation of information held internally. This multicomponent system plays a critical role in guiding everyday behavior and underlies the capacity to perform complex tasks such as learning, comprehension, reasoning, and planning. The *working* component of WM involves mental processing, updating, and reordering information held internally for use in guiding behavior. The terms used to describe these processes differ across neurocognitive models, and includes labels such as "central executive," "internal focus of attention," and "secondary memory." No memory/storage functions are ascribed to the *working* component of WM; instead, it functions to process or manipulate the information currently held within the two anatomically distinct short-term storage–rehearsal components: the *phonological* and *visuospatial* subsystems. These subsystems handle verbal and nonverbal (visual and spatial) information, respectively (see Figure 26.2).

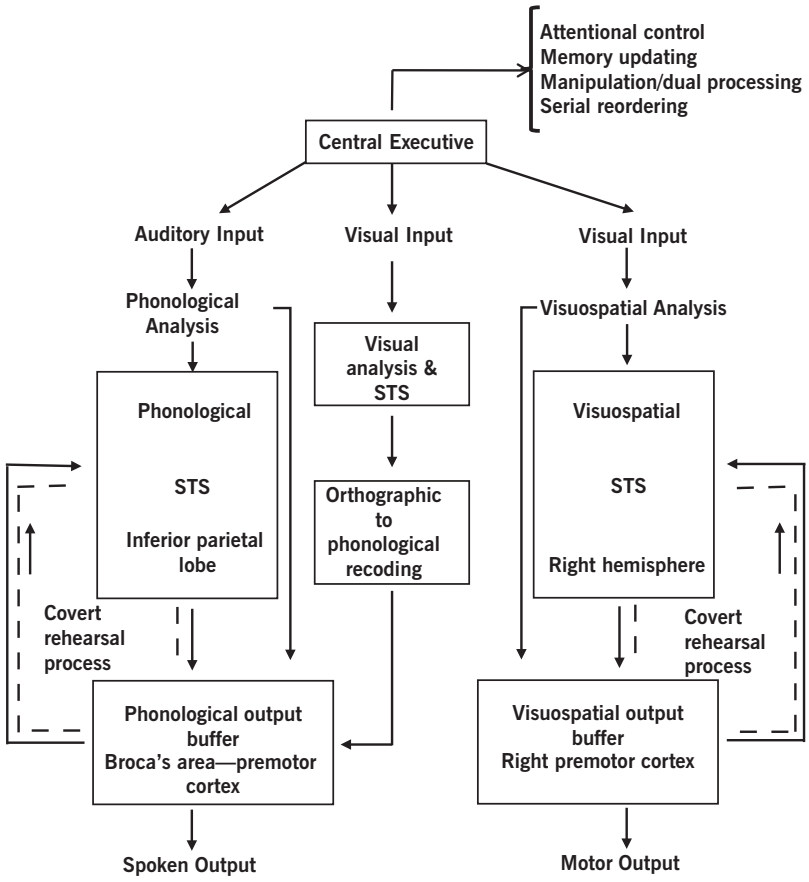

FIGURE 26.2. Visual schematic of an expanded version of Baddeley's (2007) working memory model and associated anatomical loci. Highlighted are the primary processes associated with the domain-general central executive (CE) and two (phonological, visuospatial) storage/rehearsal subsystem components. STS, short-term store. From Rapport et al. (2008). Adapted with permission from Mark D. Rapport, PhD.

Distinguishing between *working* (central executive) and *memory* (storage–rehearsal) deficits is critical for treatment development given the differential relationships among central executive processes, short-term storage processes, and ADHD-related functional impairments. For example, recent meta-analytic results indicate that children with ADHD and typically developing children differ by more than two standard deviations in core central executive abilities, or, stated differently, that at least 81% of children with ADHD have deficits in the *working* component of WM.[2] In addition, these underdeveloped central executive abilities appear to be functionally, if not causally, related to inattention (Kofler, Rapport, Bolden, Sarver, & Raiker, 2010), hyperactivity (Rapport, Bolden, et al., 2009), impulsivity (Raiker, Rapport, Kofler, & Sarver, 2012), and social problems (Kofler et al., 2011). In contrast, children with ADHD typically have medium-magnitude impairments in phonological and visuospatial storage–rehearsal (*memory*) processes, and these STM deficits are unrelated to or contribute minimally to core symptoms or important functional outcomes. The *working* (central executive) component of WM is also intricately involved in a wide range of academic and intellectual abilities, ranging from math, reading, listening comprehension, and achievement to complex learning and fluid reasoning. In contrast, the *memory* components of WM are associated with more limited roles in learning outcomes (see Sarver et al., 2012, for a review).

Inhibition

Behavioral inhibition (BI) is hypothesized as a cognitive process that subserves behavioral regulation and EF, and underlies the ability to withhold (*action restraint*) or stop (*action cancellation*) an ongoing response. BI deficits are frequently cited as a core underlying deficit responsible for ADHD (Barkley, 1997), and children with ADHD often underperform on BI tasks relative to typically developing (TD) children. The results of recent meta-analytic reviews, however, indicate that the suboptimal performance observed in children with ADHD on BI tasks is more parsimoniously explained by difficulties with basic attentional, performance variability, and/or WM processes (Alderson, Rapport, & Kofler, 2007; Lijffijt, Kenemans, Verbaten, & van Engeland, 2005).

Evidence supporting a link between BI and ADHD symptoms is similarly modest. For example, studies of community samples report a modest (r = .30) relationship between performance on an inhibition task and parent–teacher ratings of ADHD symptoms. In contrast, most ADHD clinical studies report nonsignificant relationships between inhibition and both subjective and objective measures of classroom hyperactivity, impulsivity, or inattention. In addition, experimentally manipulating demands on inhibition exerts no discernible effect on objectively measured motor activity in children with ADHD (Alderson, Rapport, Kasper, Sarver, & Kofler, 2012). Collectively, these studies suggest that previously reported BI deficits, as reflected in psychological tests and reported to exist in children with ADHD, may occur secondarily to other cognitive process deficits and are weakly or unrelated to ADHD behavioral symptoms, including those of hyperactivity–impulsivity.

Set Shifting

"Set shifting," or cognitive flexibility, refers to the ability to flexibly switch between tasks or mental sets. Tasks commonly used to assess set shifting require children to mentally hold two response sets simultaneously and switch between these response sets according to prespecified criteria (e.g., every other trial), or to monitor their performance and change response sets based on performance feedback. Meta-analytic reviews reveal moderate-magnitude set shifting deficits in children with ADHD and indicate that approximately 25–35% of children with ADHD have deficits in this aspect of EF (Frazier, Demaree, & Youngstrom, 2004; Willcutt et al., 2005). Extant evidence that set shifting deficits are related to ADHD symptoms, however, is limited. We were able to locate only two studies that examined this relationship. One reported a moderate correlation (r = .61), and the other reported a more modest (r = .17) relationship between set-shifting performance and ADHD symptoms. Collectively, few studies have examined set shifting in children with ADHD, and the limited evidence available indicates that set-shifting performance deficits, if they exist at all, are related weakly to moderately to ADHD symptoms.

Attention

Several cognitive training protocols for children with ADHD directly target one or more components of attention. The empirical rationale for targeting attention is based on compelling evidence of parent–teacher re-

ported attentional problems in children with ADHD, coupled with large-magnitude impairments in objectively observed classroom attention (Kofler, Rapport, & Alderson, 2008; Rapport, Kofler, Alderson, Timko, & DuPaul, 2009). Attention is also considered an integral component of all EFs, and attentional resource limitations are often assumed to reflect WM and other EF deficits. These perspectives suggest that targeting attentional processes in children with ADHD may result in generalized performance improvements across EFs.

Identifying the specific cognitive components of attention that are impaired in ADHD has been considerably more challenging. Among the diverse models of attention, studies of childhood ADHD frequently focus on four components of attention: *orienting/alertness* (the ability to enhance one's activation level following a stimulus of high priority), *selective/focused attention* (the ability to facilitate the processing of one source of environmental information while preventing the processing of others), *divided attention* (the ability to attend and respond to multiple tasks or multiple task demands simultaneously), and *vigilance/sustained attention* (the ability to maintain a tonic state of alertness during prolonged and sustained mental activity).

Converging evidence indicates that approximately 33 to 55% of children with ADHD demonstrate vigilance/sustained attention deficits. In contrast, orienting/alertness processes appear to be intact in ADHD. The evidence is mixed with regard to selective/focused attention and divided attention. Children with ADHD have been reported to perform worse, similar to, and better than children without ADHD on indices of these two attentional components.

The relationship between vigilance/sustained attention and ADHD behavioral and functional impairments is similarly complex. Performance on vigilance/sustained attention tasks is correlated weakly to moderately with both parent and teacher ratings of ADHD behavioral symptoms, and objectively observed classroom attention. In addition, deficient sustained attention is associated with poorer academic performance, lower grades and standardized test scores, and higher rates of special education placement and comorbid learning disabilities. In contrast, treatment-related improvements in sustained attention generally fail to result in improved learning or academic performance in approximately 50% of treated children (Rapport, Denney, DuPaul, & Gardner, 1994), highlighting the multifarious link between attention and ADHD-related functional impairments.

Summary

A substantial literature indicates that many children with ADHD have significantly underdeveloped central executive (the *working* component of WM) and vigilance/sustained attention abilities. In addition, these two cognitive functions consistently predict myriad behavioral and cognitive outcomes, rendering them highly credible targets for innovative treatments. The evidence supporting inhibition, set shifting, STM (the storage/rehearsal components of WM), and other attentional components, in contrast, is more limited.

FIT PROGRAMS

FIT programs were designed and introduced in the early 2000s to foster the development of ontogenetically underdeveloped brain structures that support EFs and related attentional processes in children by engaging them in challenging, progressively more difficult, computer-based (or automated) training exercises. A central tenet of these programs is that lasting, quantitative improvement in the development and/or efficiency of EF-related neural substrates can be accomplished by means of extensive training involving repetition, practice, and feedback. Resulting improvement in EF, in turn, is expected to generalize or transfer to other tasks, activities, and abilities to the extent that they rely on these trained neural networks. Examples include enhanced general cognitive functioning, learning, academic performance/achievement, behavioral functioning, and interpersonal relationships. As a result, the success of these programs rests to a considerable extent on the expectation of training-induced *neuroplasticity*—the brain's ability to create new pathways (neurogenesis) and rearrange and expand existing ones (synaptogenesis) for purposes of neural communication. This critical assumption of FIT programs differs in important ways from traditional CBT strategies (see Chapter 31) that rely on teaching regulatory and problem-solving strategies as compensatory change agents rather than augmenting the development of hypothesized underdeveloped neurological substrates.

Many of the FIT programs are available commercially and assert that their computer-based cognitive training exercises provide significant and lasting improvement in attention, impulse control, interpersonal functioning, academic performance, and complex reasoning skills for children with ADHD. The meta-

analytic results summarized in the ensuing sections examine the veridicality of these claims.

FIT Program Descriptions and Examples

Currently Cogmed® and BrainTrain's® Captain's Log MindPower® are two of the most widely used FIT programs for children with ADHD. The former is described as a "computer-based solution for attention problems caused by poor WM" (*www.cogmed.com/program*, May 12, 2014), and incorporates a wide range of visuospatial and verbal activities to train the two anatomically distinct, modality-specific WM subsystems described in Baddeley's model (2007; Figure 26.2).

For example, one of the tasks intended to improve visuospatial WM involves a panel of small lights arranged in a four-column by four-row format, wherein a predetermined number of lights are illuminated one at a time in a random order (Figure 26.3). The child's task is to reproduce the order immediately by clicking on the bulbs in the same order in which they were illuminated.

Children typically begin at a low set-size level (e.g., three stimuli), which requires them to hold briefly and recall the observed location of a limited number of the visuospatial stimuli. Following a preestablished number of successful recall trials, they advance to the next level, which requires them to correctly recall four stimuli.

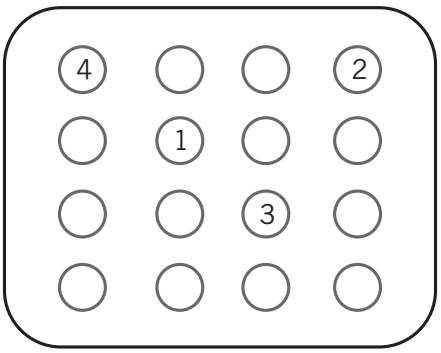

FIGURE 26.3. The figure represents a visual schematic of a hypothetical visuospatial task similar to one described by Cogmed. Numbers represent the order in which the lights are illuminated. Children are instructed to replicate the order by clicking on each of the bulbs in the same sequence in which they were illuminated.

The training exercise continues in this manner and reflects what is referred to as "adaptive training"—an instructional approach similar to Vygotsky's (1978) "zone of proximal development" concept, in which children's learning is thought to advance most efficiently when they are required to engage in activities they have not mastered fully. The training exercise, however, is more accurately described as a visuospatial STM task rather than a visuospatial WM task—children are tasked with holding the location of visuospatial stimuli temporarily in STM without any corresponding processing requirements.

A second visuospatial exercise described on the company's webpage involves watching rotating asteroids light up, one by one, on a computer monitor. Visuospatial recall is demonstrated by clicking on the asteroids in the same order in which they appeared. Similar to the previous exercise, children begin by recalling a limited number of stimuli, and they are required to recall increasingly more stimuli correctly as they proceed from lower to higher set-size levels. Based on the website description, this exercise also appears to be a relatively straightforward visuospatial short-term storage rather than a WM training exercise.

Several verbal training exercises are also included in Cogmed's training program and reflect the well-established finding that children with ADHD also experience moderate- to large-magnitude deficiencies in their ability to store/rehearse and process phonologically based information (Bolden, Rapport, Raiker, Sarver, & Kofler, 2012; Kasper, Alderson, & Hudec, 2012). For example, in one training exercise, children are shown a square-shaped keypad with nine consecutive, single-digit numbers in a 3 row × 3 column format. An audible digit string is emitted by the program's computer software (e.g., 4 9 2) and children are required to recall the digit string in reverse order using a manual keypad response. The exercise is highly similar to the Wechsler Intelligence Scale for Children–IV (WISC-IV) digit span backwards task, with one exception—children are able to view the number pad while hearing the numbers, which makes the exercise more of a mixed verbal–visuospatial STM task if children use both modalities to store–rehearse–reverse and recall the information.

Another verbal exercise designed to strengthen the phonological WM subsystem—termed *Stabilizer*—requires children to view a panel with 11 small bulbs located in a circular arrangement around an empty oval. A different bulb is illuminated momentarily and

paired with the sound of a unique letter (e.g., T, G, and E might correspond with the second, third, and fifth bulbs illuminated as illustrated in Figure 26.4), and children must remember which letter corresponds with each of the lit bulbs. Afterwards, one of the previously articulated letters appears in the center of the panel (e.g., G), and children are instructed to identify the correct bulb that was illuminated at the time the letter was heard by clicking on its location. As with the previous example, the task appears to require a mixture of visuospatial and phonological short-term storage rather than WM, depending on how children elect to store and recall the stimuli sequence.

Mental health and educational professionals undergo training to qualify as Cogmed coaches, which enables them to offer the training package and provide ongoing supervision to children whose families can afford the cost (the fee is set by individual providers and $1,500 appears to represent the current modal training cost). Children are typically required to complete approximately eight training exercises per day (i.e., about 30–45 minutes), 5 days per week, over a 5-week training period, and engage in computer games afterwards as an incentive.

An alternative set of cognitive training exercises (Captain's Log MindPower) offered by BrainTrain, allows individuals to customize cognitive training by selecting among nine modules designed to strengthen visual and verbal short-term and WM abilities, various attentional abilities, and problem-solving skills. For example, in one of the WM skills module tasks—*Code Cracker*—children are shown visual stimulus sets that comprise letters and symbols (e.g., L, *, &, R, #), and each stimulus is matched with one of nine unique digits (1, 2, 3 . . . 9). Afterwards, a row containing the letters and symbols appears directly above a 3 row × 3 column symmetrical grid that contains the numbers 1 through 9. Children are tasked with clicking on each of the previously viewed digits associated with the letters and symbols displayed in the top row to "crack the code" and open the safe (see Figure 26.5). Despite its inclusion in the WM skills module, Code Cracker might be more accurately described as a paired associate learning exercise that requires visuospatial and/or phonological STM, depending on whether children encode the stimuli phonologically or by their shapes.

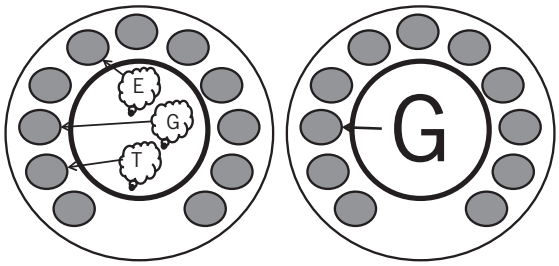

FIGURE 26.4. The figure represents a visual schematic of a hypothesized visuospatial, short-term memory training task similar to one described by Cogmed. Children hear the letters T, G, and E emitted from the computer speaker (left circle), and one of the enunciated letters appears in the middle of the screen immediately afterwards (right circle). Children are tasked with clicking on the circle that was lit up when they heard that particular letter (i.e., the circle marked G).

Turning to the Captain's Log *Attention Skills* module reveals an exercise that is described as training alternating attention and response inhibition (*Stimulus Reaction/Inhibition* or *Red Light Green Light* training exercise) to improve children's "mental processing speed" and "self-control." Children view a computer monitor and their task is to determine whether the image that appears on the screen is the same color as the screen's border, then to click the mouse button as quickly as possible following color match trials and not to click the button following color mismatch trials. Based on the website's description, the task appears to represent a relatively straightforward, simple visual matching paradigm without a clear-cut response inhibition element. For this latter element, prepotency to respond to a particular stimulus would need to be established beforehand to ensure a high degree of readiness to respond and/or difficulty withholding (action restraint) or stopping (action cancellation) a response.

A task in the Auditory Working Memory module, named Reverse Recall (*Touchdown!*), is reportedly designed to train phonological WM. In this task, children hear sequences of letters, numbers, directions, chores, sounds, and other items listed in a specific order. They are instructed to recall the items in reverse order by clicking on a visual representation of each item (see Figure 26.6). Reversing auditory stimuli in this manner, however, is similar to the WISC-IV digit span backwards task, which loads on the same dimension as digits forward (Colom, Abad, Rebollo, & Shih, 2005; Swanson & Kim, 2007) and is considered a measure of phonological short-term storage rather than WM.

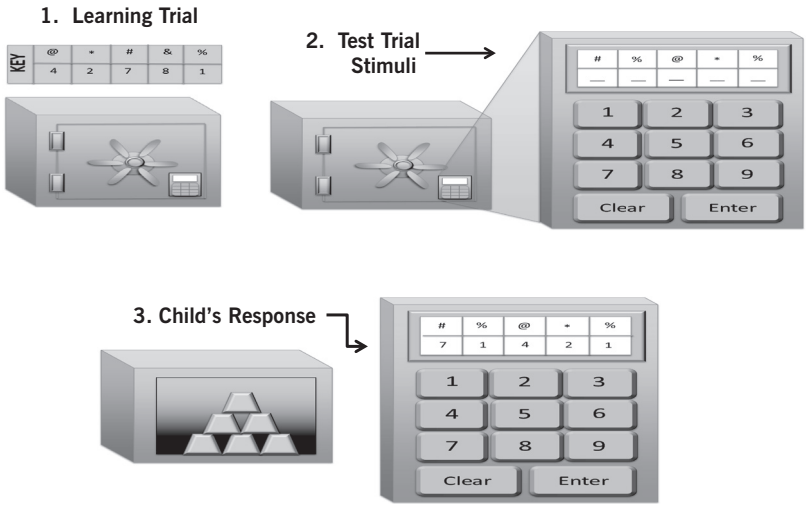

FIGURE 26.5. Visual schematic of a hypothesized combined phonological/visuospatial short-term memory training task similar to one described on the BrainTrain website. Children undergo a learning trial (1), are shown the letters and symbols of stimuli to be learned (2), and enter the correct "code" to open the safe (3).

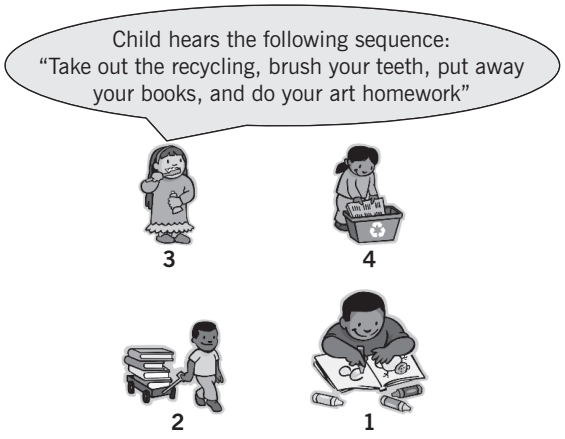

FIGURE 26.6. Visual schematic of a hypothesized phonological working memory training task similar to one described on the BrainTrain website. Children hear a sequence of chores (top of figure) and are tasked with recalling them in reverse order by clicking on the appropriate pictures. Numbers directly underneath the pictures (1, 2, 3, 4) indicate the correct reversed order in the example.

Educators and professionals can individualize treatment by selecting which modules and exercises to use during training. Similar to Cogmed, the available exercises utilize an adaptive training approach and adjust the difficulty level of the exercises to ensure that children are continually challenged by the tasks demands. Licenses for Captain's Log MindPower can be purchased through the BrainTrain website for $395 per client or $1,495 per computer station. Children are encouraged to complete 2 hours of training per week in a manner that best fits their schedules.

Key Questions

Improving the functioning of underdeveloped neurological substrates that enable goal-directed behavior is highly dependent upon the integrity of the training programs. As a result, we pay particular attention to several critical questions in the ensuing meta-analytic review of these programs (Rapport et al., 2013).

The data in Table 26.1 addresses the first of these questions reviewed earlier—namely, to what extent do extant FIT programs target the (1) empirically documented EF deficits and associated core/peripheral features and (2) functional outcomes in children with

TABLE 26.1. Executive Functions and Attention Processes Targeted for FIT

Author (year)	Training target	Working memory[a]			Short-term memory[b]		Behavioral Inhibition		Set shifting	Attention			
		Updating	Manipulation/ dual-task	Serial reordering	PH	VS	Motor	Cognitive		Orienting alertness	Vigilance sustained	Selective	Divided
Beck et al. (2010)	STM				X	X							
Dahlin (2011)	STM				X	X							
Gibson et al. (2011)	STM				X	X							
Gray (2011)	STM				X	X							
Green et al. (2012)	STM				X	X							
Holmes et al. (2010)	STM				X	X							
Klingberg et al. (2005)	STM				X	X							
Mezzacappa & Buckner (2010)	STM				X	X							
Prins et al. (2011)	STM					X							
Kerns et al. (1999)	Attention										X	X	X
Lange et al. (2012)	Attention									X	X	X	X
Semrud-Clikeman et al. (1999)	Attention										X	X	
Tamm et al. (2012)	Attention										X	X	X
Tamm et al. (2010)	Attention										X	X	X
Tucha et al. (2011)	Attention									X	X	X	X
Halperin et al. (2012)	Mixed EF				X	X	X					X	
Hoekzema et al. (2010)	Mixed EF				X						X		
Johnstone et al. (2012)	Mixed EF					X	X						
Johnstone et al. (2010)	Mixed EF					X	X						
Klingberg et al. (2002)	Mixed EF				X	X	X						
Rabiner et al. (2010)	Mixed EF					X					X		
Shalev et al. (2007)	Mixed EF							X		X	X	X	
Steiner et al. (2011)	Mixed EF				X	X				X	X	X	X
Van der Oord et al. (2012)	Mixed EF			X		X	X		X				
Kray et al. (2012)	Set Shifting								X				

Note. Studies are grouped by training target and alphabetized within grouping to permit direct comparisons of study characteristics and effect sizes in subsequent tables. PH, phonological; VS, visuospatial; STM, short-term memory; EF, executive function.

[a]All tasks require at least minimal WM/central executive resources (e.g., maintaining task instructions). Tasks are coded as targeting WM processes if these processes were targeted explicitly through adaptive training components designed specifically to increase abilities in one or more WM central executive subprocesses (updating, manipulation/dual processing, and serial reordering) by a majority of training components. All studies using Cogmed were coded as targeting STM given empirical evidence indicating that this training paradigm improves short-term but not WM processes (Gibson et al., 2011).

[b]STM refers to the storage/rehearsal components of the WM system (i.e., the *memory* components of WM).

ADHD? The significance of this question rests on the assumption that targeting nonaffected or minimally impaired substrate mechanisms/processes is unlikely to result in substantial far-transfer effects.

Table 26.1 summarizes the type and range of EFs and/or attentional processes targeted by each of the 25 FIT studies. It indicates that nine of the 25 reviewed FIT studies used Cogmed training exercises, which are intended to improve WM; however, scrutiny of these training exercises reveals that most of them require children to store/rehearse auditory and/or visuospatial stimuli for a brief time interval and do not include *working* memory processing demands such as actively updating memory, manipulating/dual processing, and serial reordering. An additional nine FIT studies focused primarily on training phonological (PH) and visuospatial (VS) STM, in addition to at least one attentional component, such as orienting, vigilance, selective attention, or divided attention. Six of the remaining FIT studies focused exclusively on training two or more attentional components, and one focused exclusively on training set shifting. Collectively, none of the reviewed FIT studies included appropriately designed tasks to rigorously train empirically established WM processing deficits in children with ADHD; however, several included components designed to train children's vigilance or ability to sustain attention over time—a well-documented deficiency in children with ADHD.

Table 26.2 provides an overall summary of the FIT interventions, including the number of children who participated in the treatment and control groups, the name of the program, the type of control group employed (e.g., waiting list, nonadaptive). It also addresses a second set of critical questions regarding the extent to which FIT programs incorporate *adaptive* training, and the relative potency or dosage effects related to training. As shown in Table 26.2, nearly all of the FIT programs incorporated adaptive training methodology, which continuously adjusts the difficulty of the training exercise based on each child's performance to ensure that the suspected underlying neurological substrate is challenged continuously. The optimal duration of sessions, number of sessions, and intensity parameters required to obtain clinically meaningful improvement in children's WM is unknown currently; however, the 25 studies reviewed include a wide range of values for each of these parameters. For example, minutes per session ranged from 15 to 60, total sessions ranged from three to 36, total training minutes ranged from 105 to 2160, and total training weeks ranged from four to 18. We examined these parameters in the meta-analysis; however, they did not prove to be significant moderators for any of the findings.

Table 26.3 addresses important methodological questions concerning the extent to which FIT programs incorporate appropriate experimental controls (e.g., inclusion of both near- and far-transfer measures; blinded raters) to ensure that any behavioral–cognitive gains realized can be attributed to the training rather than to unsystematic factors or Hawthorne effects (i.e., illusory biases). Most (68%) studies included near-transfer objective measures of cognitive performance, whereas a smaller percentage of studies (44%) included far-transfer measures of cognitive performance, and relatively few (12%) included far-transfer objective measures of academic achievement. Finally, 32% and 52% of the studies included far-transfer subjective measures of behavior change based on blinded and unblinded raters, respectively. Effect size estimates based on Cohen's *d* (corrected for sample size) are reported for near and far objective and subjective outcome variables for all 25 studies included in the meta-analysis. Cohen's *d* effect sizes are in standard deviation units, such that an effect size of 1.0 indicates a change in one standard deviation from pretreatment to posttreatment. An effect size of 0.2 is interpreted as small (detectable only through statistics), 0.5 as medium (detectable to a careful observer), and 0.8 as large (obvious to any observer; Cohen, 1988).

FIT Programs and Empirically Based Outcomes

A tiered analytic approach adopted to examine the FIT outcome studies addressed the key questions discussed in the previous section. The Tier I analysis examined 17 studies reporting posttreatment outcomes on tasks similar to the training tasks (immediate, near-transfer effects; 58 effect sizes); Tier II examined the three studies reporting long-term follow-up of near-transfer effects (long-term, near-transfer effects; 20 effect sizes); Tier III examined 21 studies (22 independent subgroups) reporting posttreatment data on outcomes dissimilar to training tasks (immediate, far-transfer effects; 233 effect sizes); and Tier IV examined the seven studies reporting long-term follow-up of far-transfer effects (long-term far-transfer effects; 125 effect sizes).

TABLE 26.2. FIT Study Characteristics

First Author (year)	T (*n*)	C (*n*)	Program	Control group	Adaptive	Computerized	Total minutes	Total sessions	Total weeks	Minutes/ session
Beck et al. (2010)	27	24	Cogmed	Waiting list	Y	Y	750	25	6	30
Dahlin (2011)	41	15	Cogmed	Waiting list	Y	Y	600	20	5	30
Gibson et al. (2011)	38	—	Cogmed	None	Y	Y	600	20	6	30
Gray (2011)	36	24	Cogmed	Adaptive	Y	Y	900	20	5	45
Green et al. (2012)	12	14	Cogmed	Nonadaptive	Y	Y	625	25	—	25
Holmes et al. (2010)	25	—	Cogmed	None	Y	Y	600	20	6	30
Klingberg et al. (2005)	20	24	Cogmed	Nonadaptive	Y	Y	1,000	25	5	40
Mezzacappa & Buckner (2010)	8	—	Cogmed	None	Y	Y	1,000	25	5	40
Prins et al. (2011)	27	24	Study developed	Adaptive	Y	Y	105	3	3	35
Kerns et al. (1999)	7	7	Pay Attention!	Nonadaptive	Y	N	480	16	8	30
Lange et al. (2012)	16	16	AixTent	Adaptive	Y	Y	480	8	4	60
Semrud-Clikeman et al. (1999)	21	12	APT	Waiting list	Y	N	2,160	36	18	60
Tamm et al. (2012)	54	51	Pay Attention!	Waiting list	Y	N	480	16	8	30
Tamm et al. (2010)	19	—	Pay Attention!	None	Y	N	480	16	8	30
Tucha et al. (2011)	16	16	AixTent	Adaptive	Y	Y	360	8	4	45
Halperin et al. (2012)	29	—	TEAMS	None	Y	N	177.5	5	5	35.5
Hoekzema et al. (2010)	10	9	Study developed	Nonadaptive	Y	N	450	10	—	45
Johnstone et al. (2012)	40	20	Study developed	Adaptive and waiting list	Y	Y	375	25	5	15
Johnstone et al. (2010)	15	14	Study developed	Nonadaptive	Y	Y	500	25	5	20
Klingberg et al. (2002)	7	7	Cogmed	Nonadaptive	Y	Y	607.5	24.3	5	25
Rabiner et al. (2010)	25	27	Captain's Log	Adaptive and waiting list	Y	Y	1,400	28	14	50
Shalev et al. (2007)	20	16	CPAT	Adaptive	Y	Y	960	16	8	60
Steiner et al. (2011)	11	9	Captain's Log	Adaptive and waiting list	Y	Y	960	32	16	30
Van der Oord et al. (2012)	18	22	Study developed	Waiting list	Y	Y	1,000	25	5	40
Kray et al. (2012)	10	10	Study developed	Nonadaptive	N	Y	120	4	4	30

Note. T, treatment group; C, control group; *n*, number of participants within each group; APT, attention process training; TEAMS, training executive, attention, and motor skills; CPAT, computerized progressive attention training. Training time data represent lower value of range reported by authors.

TABLE 26.3. FIT Program Near- and Far-Transfer Effects

				Effect Sizes					
				Near objective		Far objective		Far subjective	
Author (year)	Program	Training target	Control group	COG	ACH	COG	ACH	Blinded	Unblinded
Beck et al. (2010)	Cogmed	STM	Waiting list	—	—	—	—	0.23	0.64
Dahlin (2011)	Cogmed	STM	Waiting list	0.85	—	—	0.41	—	—
Gibson et al. (2011)	Cogmed	STM	None	0.45	—	—	—	0.27 PH 0.09 VS	0.62 PH 0.18 VS
Gray (2011)	Cogmed	STM	Adaptive	0.28	—	0.49	0.03	—	0.03
Green et al. (2012)	Cogmed	STM	Nonadaptive	0.70	—	—	—	0.16	—
Holmes et al. (2010)	Cogmed	STM	None	0.84	—	0.11	—	—	—
Klingberg et al. (2005)	Cogmed	STM	Nonadaptive	0.62	—	0.42	—	0.32	—
Mezzacappa & Buckner (2010)	Cogmed	STM	None	0.99	—	—	—	—	0.91
Prins et al. (2011)	Study developed	STM	Adaptive	0.64	—	—	—	—	—
Kerns et al. (1999)	Pay Attention!	Attention	Nonadaptive	0.0[a]	—	0.31	—	0.0	—
Lange et al. (2012)	AixTent	Attention	Adaptive	0.55	—	—	—	—	—
Semrud-Clikeman et al. (1999)	APT	Attention	Waiting list	0.90	—	—	—	—	—
Tamm et al. (2012)	Pay Attention!	Attention	Waiting list	–0.03	—	0.25	—	—	0.42
Tamm et al. (2010)	Pay Attention!	Attention	None	—	—	0.18	—	—	0.40
Tucha et al. (2011)	AixTent	Attention	Adaptive	0.38	—	—	—	—	—
Halperin et al. (2012)	TEAMS	Mixed EF	None	—	—	—	—	—	0.51
Hoekzema et al. (2010)	Study developed	Mixed EF	Nonadaptive	—	—	0.0[c]	—	—	—
Johnstone et al. (2012)	Study developed	Mixed EF	Waiting list[d]	0.00	—	0.10	—	—	0.37
Johnstone et al. (2010)	Study developed	Mixed EF	Nonadaptive	0.04	—	—	—	—	0.67[b]
Klingberg et al. (2002)	Cogmed	Mixed EF	Nonadaptive	0.86	—	1.05	—	—	—
Rabiner et al. (2010)	Captain's Log	Mixed EF	Adaptive[e]	—	—	—	0.11	—	0.25
Shalev et al. (2007)	CPAT	Mixed EF	Adaptive	—	—	—	—	0.41	—
Steiner et al. (2011)	Captain's Log	Mixed EF	Adaptive[e]	—	—	–0.07	—	0.11	0.21
Van der Oord et al. (2012)	Study developed	Mixed EF	Waiting list	—	—	—	—	—	0.46
Kray et al. (2012)	Study developed	Set shifting	Nonadaptive	0.70	—	0.44	—	—	—

Note. COG, cognitive performance; ACH, standardized achievement; STM, short-term memory; APT, attention process training; TEAMS, training executive, attention, and motor skills; CPAT, computerized progressive attentional training; PH, phonological; VS, visuospatial; EF, executive function. Effect sizes are Cohen's *d* corrected for sample size.

[a]Reflects a nonsignificant change on a continuous performance test; a measure of sustained auditory attention was considered an outlier ($d = 3.02$) and excluded from this analysis.

[b]Reflects within (adaptive treatment) group pre–post differences; insufficient data available for the nonadaptive control group.

[c]Authors reported no significant group differences in performance on three cognitive tasks, and did not respond to e-mail requests for unreported data for two additional measures.

[d]Two active treatment groups receiving identical treatment with the exception of one component were collapsed and compared to a waiting list group by the authors.

[e]The more rigorous control group was compared against the treatment group.

Immediate Near-Transfer Effects

The 17 studies reporting data on 636 individuals with ADHD were included in the analyses examining immediate near-transfer effects of ADHD FIT programs (Table 26.3). We found significant differences in effect magnitude across studies, which necessitated examination of potential moderators of these effects. Training Target was examined initially to examine the extent to which effect sizes differed systematically as a function of cognitive training target. Studies were classified into three categories: STM only (k = 8), mixed EFs (k = 3), and attention only (k = 5). Set shifting (d = 0.70, nonsignificant) was examined qualitatively but not included in the analyses due to insufficient degrees of freedom (k = 1). Our results revealed that Training Target explained significant between-study differences, such that no significant between-study residual differences remained after accounting for Training Target. As shown in Table 26.4, studies targeting STM only (d = 0.63) were associated with moderate-magnitude increases in STM. In contrast, studies targeting Attention only (d = 0.05, nonsignificant) and mixed EFs (d = 0.06, nonsignificant) failed to find significant post-treatment improvement on near-transfer measures of targeted cognitive processes.

Long-Term Near-transfer effects

Only 3 of the 17 studies reporting near-transfer effects reported data sufficient to calculate long-term follow-up effect sizes; follow-up duration ranged from 3- to 6-months across studies. Collectively, results from the three studies reporting long-term follow-up data suggest that STM gains may be maintained for up to 3–6 months; however, additional follow-up studies are needed to ensure the veracity of these findings.

Immediate Far-Transfer Effects

A total of 21 studies reporting data on 733 individuals with ADHD were included in the analyses examining immediate far-transfer effects of cognitive training for children with ADHD (Table 26.3). Despite finding that only STM training programs were associated with improvements in their training target, classifying studies by training target did not explain between-study differences in far-transfer outcomes. As a result, we examined whether the far-transfer results might depend on the type of outcome measures (i.e., objective and subjective) used in the studies. The results revealed that studies that relied on unblinded raters—and not objective outcome measures—accounted for the immediate far-transfer FIT training effects. In contrast, FIT training did not improve blinded behavior ratings or academic achievement, and it exerted a minimal impact on cognitive test scores for children with ADHD. Collectively, these results were disappointing and indicate that the significant far-transfer training benefits reported previously are in the *eye of the beholder* and consistent with an illusory bias or expectancy effect.

Long-Term Far-Transfer Effects

Seven of the 21 studies reporting far-transfer outcomes included long-term follow-up data sufficient to calculate effect sizes (total N = 231)—three trained STM, three trained mixed EFs, and one trained attention. Far-transfer gains reported at the conclusion of training were maintained at 1- to 9-month postassessment intervals; however, five of the seven studies reporting long-term follow-up data on far-transfer effects relied exclusively on unblinded behavior ratings and, as reported earlier, likely reflect uncontrolled expectancy effects.

Summary of Findings

The meta-analytic results revealed moderate magnitude improvement on near-transfer measures of children's cognitive performance for FIT programs targeting STM, and these effects remained evident at 3–6 months in the circumscribed number of studies (k = 3) that examined near-transfer maintenance. In contrast, FIT programs targeting mixed EFs (e.g., combined inhibition and STM training), set-shifting, or only attention processes were not associated with significant improvements in the trained cognitive processes. This pattern of results was consistent with expectations derived from our literature review of EF deficits in children with ADHD and their association with impaired functional outcomes with one exception: the lack of significant near-transfer effects for FIT programs targeting vigilance/sustained attention deficits. The latter finding may reflect the limited time devoted exclusively to strengthening vigilance/sustained attention abilities due to time spent training attention components that are likely not impaired in children with ADHD (i.e., inadequate potency).

The lack of a significant Training Target moderator effect led us to an examination of all FIT programs

TABLE 26.4. FIT for ADHD: Meta-Analytic Summary

	Near-transfer effects			Far-transfer effects		
	Immediate (Tier I) $k = 17$	Pre to follow-up (Tier IIa) $k = 3$	Post to follow-up (Tier IIb) $k = 3$	Immediate (Tier III) $k = 22$	Pre to follow-up (Tier IVa) $k = 7$	Post to follow-up (Tier IVb) $k = 7$
Cohen's *d* effect size:	0.46 (0.26 to 0.66)	0.73 (0.46 to 0.99)	–0.18, *ns* (–0.42 to 0.06)	0.38 (0.21 to 0.54)	—	—
Corrected for sampling error	0.45 (0.25 to 0.65)	0.71 (0.45 to 0.97)	–0.17, *ns* (–0.41 to 0.06)	0.36 (0.20 to 0.51)	—	—
Corrected for sampling error/publication bias	0.23 (0.04 to 0.42)	0.71 (0.45 to 0.97)	–0.20, *ns* (–0.42 to 0.01)	0.36 (0.20 to 0.51)	—	—
Cohen's *d* effect size corrected for sampling error/ publication bias						
Moderator analysis: Training target	0.63 (0.46 to 0.80) $k = 8$	—	—	0.39 (0.13 to 0.66) $k = 9$	—	—
STM only						
Attention	0.05, *ns* (–0.29 to 0.38) $k = 5$	—	—	0.33 (0.08 to 0.57) $k = 3$	—	—
Mixed EF	0.06, *ns* (–0.22 to 0.33) $k = 3$	—	—	0.28 (0.10 to 0.45) $k = 9$	—	—
Set shifting	0.70, *ns* (–0.17 to 1.57) $k = 1$	—	—	0.44, *ns* (–0.42 to 1.30) $k = 1$	—	—
Moderator analysis: Outcome type						
Cognitive performance	—	—	—	0.14 (0.03 to 0.25) $k = 11$	0.45 (0.17 to 0.74) $k = 2$	–0.003, *ns* (–0.41 to 0.40) $k = 2$
Academic achievement	—	—	—	0.15, *ns* (–0.15 to 0.45) $k = 3$	0.28, *ns* (–0.13 to 0.69) $k = 2$	0.11, *ns* (–0.30 to 0.52) $k = 2$
Blinded subjective ratings	—	—	—	0.12, *ns* (–0.02 to 0.25) $k = 8$	0.15, *ns* (–0.19 to 0.49) $k = 2$	–0.11, *ns* (–0.45 to 0.23) $k = 2$
Unblinded subjective ratings	—	—	—	0.48 (0.30 to 0.66) $k = 13$	0.52 (0.31 to 0.73) $k = 5$	0.07, *ns* (–0.13 to 0.28) $k = 5$

Note. Cohen's *d* effect sizes (95% confidence intervals [CIs] in parentheses) were corrected for sample size due to the upward bias of small-*N* studies. Effect sizes are considered significantly different from 0.0 (statistically significant at $p < .05$) if their 95% CIs do not include 0.0. Moderator subgroup effect sizes are corrected for sampling error and publication bias when significant. *ns*, nonsignificant (95% CI includes 0.0; $p > .05$). *k*, number of studies; STM, short-term memory; mixed EF, studies training two or more executive functions.

incorporating far-transfer measures across four mutually exclusive outcome categories. These included two categories each of objective (i.e., cognitive and standardized academic achievement subtest scores) and subjective outcome measures (i.e., blinded and unblinded ratings). The meta-analytic results revealed no evidence that FIT improves children's academic achievement or blinded ratings of their behavior; however, significant, small-magnitude far-transfer effects were evident among the 11 studies that included cognitive performance outcome measures. This enhanced performance, albeit marginal and detectable only by statistical analysis (Cohen, 1988), warrants scrutiny given that nearly three-fourths of the studies reporting far-transfer cognitive performance outcomes either failed to incorporate near-transfer measures (27%) or reported far-transfer effects (46%) that were of similar or greater magnitude than their near-transfer effects. For the former studies, the lack of demonstrated near-transfer improvements makes it impossible to determine the extent to which improved cognitive performance reflects random or systematic influences, such as task-specific practice and expectancy effects, rather than the assumed strengthening of cognitive functioning. The latter studies' findings are equally perplexing and incongruent with transfer theory predictions, which limit the magnitude of transfer to the multiplicative relation between near-transfer improvement (i.e., the near-transfer effect size estimate) and the established relation between the training target and far-transfer constructs. As an example, Klingberg, Forssberg, and Westerberg (2002) reported that children demonstrated larger magnitude far-transfer improvements (effect size = 1.05) relative to near-transfer improvements (effect size = 0.86) following visuospatial STM and inhibition/choice reaction time (CRT) training. However, the far-transfer measures used in the study—the Stroop task and Raven's Progressive Matrices—are predicted only modestly by visuospatial STM measures (beta = 0.18 and 0.28; Engle, Tuholski, Laughlin, & Conway, 1999; St Clair-Thompson & Gathercole, 2006). A somewhat higher correlation is reported between tasks with combined inhibition/CRT elements (e.g., stop-signal paradigm) and the Stroop task (i.e., beta = 0.49; St Clair-Thompson & Gathercole, 2006). Accordingly, the maximum far-transfer training effect size expected for this study is between 0.16 and 0.24 (attributable to visuospatial STM improvements) and 0.42 (attributable to inhibition/CRT improvements); transfer theory specifies that far-transfer effect sizes in excess of this hypothesized ceiling cannot be attributable entirely to neuronal-level improvements in the trained cognitive functions.[3]

Finally, unblinded parents and teachers reported moderate-magnitude improvement in children's behavior and/or EF in the absence of objective evidence for these changes (i.e., illusory effects). The finding that far-transfer gains were similar to or larger than near-transfer improvement in several of these studies (e.g., Mezzacappa & Buckner, 2010), despite the modest relationship (r = .18–.35) and limited variance (3–12%) shared between span measures and parent ratings (Naglieri, Goldstein, Delauder, & Schwebach, 2005), raises additional interpretative and methodological concerns that warrant scrutiny in future investigations.

MINDFULNESS INTERVENTION TRAINING PROGRAMS

Mindfulness is a meditative technique that focuses on "awareness that emerges through paying attention on purpose, in the present moment, and non-judgmentally to the unfolding of experience moment by moment" (Kabat-Zinn, 2003, p. 145). With origins in Buddhism, mindfulness techniques have been used in a secular context since the 1940s to reduce stress and discomfort in patients diagnosed with medical conditions such as chronic pain, fibromyalgia, cancer, heart disease, and arthritis and treatment-related side effects. The techniques have also been used to treat psychological symptoms such as anxiety, depression, and binge eating in recent years, and are associated with moderate physical (d = 0.42) and mental (d = 0.50) health benefits (Grossman, Neimann, Schmidt, & Walach, 2004).

Mindfulness-based meditation techniques—in addition to improving subjective ratings of medical and psychological distress—are often associated with changes in neurophysiological and immune functioning. For example, documented neurophysiological changes associated with the implementation of meditative practices include increased alpha and theta electroencephalographic (EEG) activity, particularly in the anterior cingulate cortex and dorsomedial prefrontal cortex (Ivanovski & Malhi, 2007; Lagopoulos et al., 2009), whereas improved immune response has been demonstrated by increased titer development to influenza vaccine (Davidson et al., 2003).

Changes in brain regions associated with attention and EFs following mindfulness training (namely, the

dorsolateral prefrontal cortex) prompted clinical researchers to modify and adapt the techniques for typically developing children, and more recently, for children with attentional problems and those diagnosed with ADHD. The child and adolescent adaptations involve training children to be fully aware of and in the present moment—a goal accomplished by helping them develop the ability to focus on the here-and-now of any activity in which they are involved—and to process this information from various unique perspectives. The *awareness* aspect of training includes attending to the multiple sensations (e.g., sights, sounds, smells, tastes, and tactile sensations) experienced while thinking about unique ways in which to approach and/or solve a task or activity, and recognizing but letting go of nonrelevant external distractors and internal thoughts.

Although not described as such, several mindfulness training exercises target EF deficits commonly observed in children with ADHD. For example, the *awareness* exercises are clearly intended to promote and strengthen children's ability to focus and sustain their attention on only those stimuli (sensory and cognitive) relevant to performing or completing an assignment, task, or activity. Other exercises are geared toward developing and strengthening cognitive functions that are critical to holding and processing information in the short-term storage/rehearsal WM subsystems, and to inhibit irrelevant external (distractions) and internal stimuli (thoughts) that are likely to interfere with processing the information (i.e., cognitive inhibitory or interference control). As reviewed earlier, sustained attention, WM, and central executive-related cognitive inhibitory control processes represent the most promising EF component targets based on their moderate to strong associations with core symptoms and functional outcomes in children with ADHD.

Mindfulness Programs and Empirically Based Outcomes

One of the first clinical demonstrations of a mindfulness training intervention modified for children was reported by Napoli, Krech, and Holley (2005). The authors examined the effectiveness of a school-based intervention (i.e., the Attention Academy Program) developed to improve attention through the practice of mindfulness. Children assigned to the mindfulness treatment group received 24 weeks of training (45 minutes twice a month) that focused on teaching them to control their breathing and body movements, to develop sensorimotor awareness as a means to improve their attention to the present, to approach without judgment, and to view experiences in a novel manner (see Napoli et al., 2005, Appendix A, for detailed training exercise descriptions). The authors reported moderate-magnitude improvement on measures of social skills (effect size = 0.46), test anxiety (effect size = 0.39), and some measures of attention (effect sizes = 0.49–0.60), but no significant changes in sustained attention. The extent to which improvement on these outcome measures reflect mindfulness training as opposed to increased involvement with adults and expectancy was unclear, however, due to the absence of near-transfer training measures, reliance on unblinded subjective ratings, and contrasts with a passive control group. The sample of children participating in the study may also have constrained the magnitude of treatment-related effects. Participants were described as typically developing children as opposed to children with documented attention-related difficulties or ADHD, perhaps placing an upper limit on the extent to which young children who already possess appropriately developed attentional abilities can improve their attention with prolonged training.

A Limited Meta-Analytic Review

As of this writing, there are only two published studies of mindfulness training for children and adolescents with ADHD. The first (van de Weijer-Bergsma, Formsma, de Bruin, & Bögels, 2012) evaluated the effectiveness of an 8-week (1.5 hour weekly sessions) mindfulness training protocol based on adaptive versions of mindfulness-based cognitive therapy (Segal, Williams, & Teasdale, 2012) and mindfulness-based stress reduction (Kabat-Zinn, 1990) in 10 adolescents with ADHD and their parents. Adolescent and parent completed Mindfulness Attention and Awareness Scale (MAAS) measures, and two computerized sustained attention tasks from the Amsterdam Neuropsychological Tasks (ANT) battery (i.e., Sustained Attention Dots and Sustained Attention Auditory tasks) were used to evaluate near-transfer effects associated with mindfulness training. Far-transfer measures included the attention, internalizing, and externalizing subscales of the Child Behavior Checklist (CBCL), the Teacher Report Form (TRF), the Youth Self-Report Form (YSR), and the Behavior Rating Inventory of Executive Function (BRIEF) Metacognition and Behavioral Regulation subscales.

The second study (van der Oord, Bögels, & Peijnenburg, 2012) investigated the potential efficacy of a nearly identical 8-week (1.5-hour sessions) mindfulness program for 18 children with ADHD and their parents, based on the same adapted versions of mindfulness training used by van de Weijer-Bergsma and colleagues (2012). No near-transfer measures were used in the study; far-transfer measures included unblinded parent and blinded teacher ratings on Disruptive Behavior Disorders Rating Scale subscales (Inattention, Hyperactivity and Impulsivity, Oppositional Defiant Disorder, Conduct Disorder).

The overall results stemming from the two mindfulness intervention studies for children and adolescents with ADHD and their parents are summarized in Table 26.5. No significant near- (d = 0.13, *ns*) or far- (d = -0.01, *ns*) transfer effects were reported by van de Weijer-Bergsma and colleagues (2012), whereas van der Oord and colleagues (2012) reported small- to moderate-magnitude improvements on their far-transfer parent ratings (d = 0.34). The parents completing the rating scales, however, were actively involved with their child's treatment and received an adult version of the same treatment themselves. Consequently, their unblinded ratings likely reflect the well-documented illusory biases that occur in lieu of appropriate controls for expectancy effects—an explanation consistent with the nonsignificant mindfulness training effects based on blinded teacher ratings.

SUMMARY AND FUTURE DIRECTIONS

Considered collectively, our initial meta-analytic review indicates that extant claims regarding the benefits associated with FIT programs, including improved academic achievement, cognitive performance, and reduced symptomatology in children with ADHD, are not supported by empirical evidence. It would be premature, however, to conclude that bringing about fundamental and lasting changes in the cognitive abilities of children with ADHD is unattainable given the significant design and methodological limitations characteristic of the field.

One of the most fundamental design issues entails the lack of correspondence between the cognitive functions targeted by FIT programs and extant empirical evidence. WM is a patent example. Each of the STM FIT studies identified in the literature search relied on a program that describes itself as an intervention for improved WM. A majority of its exercises, however, focus on training the least impaired aspects of WM in children with ADHD (namely, visuospatial and phonological short-term storage capacity), as opposed to the significantly larger magnitude central executive processing deficits associated with impaired functional outcomes identified in the ADHD literature.

The scant literature examining mindfulness as a potentially therapeutic technique for youth with ADHD suffers from nearly identical methodological design limitations that characterize the FIT literature. These include the need for (1) credible (adaptive) control groups, (2) objective (blind) ratings, and (3) multiple near- and far-transfer measures to allay extant validity concerns (including nontransfer measures that would not be expected to improve following treatment as an index of divergent validity). There is also a dearth of information concerning potentially critical treatment parameters for FIT and mindfulness intervention programs. Most of these center on dosage effects and include establishing the optimal duration and spacing of sessions for youth of different ages, and how long training needs to continue to ensure optimal treatment effects. Most children with ADHD have experienced difficulties with inattentiveness, impulsivity, excessive gross motor activity, and associated adverse functional outcomes, such as learning difficulties and poor peer relationships for their entire lives. Significant improvement in core symptoms and associated adverse outcomes is to a considerable extent likely to depend on whether and the degree to which empirically informed interventions can normalize identified EF deficits that govern behavior and contribute to successful academic functioning and interpersonal interactions. Given the 3-year delay in peak, frontal–prefrontal cortical maturation associated with ADHD, however, it is unlikely that training children for 30 minutes a day across 5 weeks or for 90 minutes a day for 8 weeks—the modal training parameters adopted in past FIT and mindfulness investigations, respectively—will normalize behavioral, academic, and neurocognitive functioning for children with ADHD. We remain optimistic, however, regarding the potential for future interventions to target empirically informed EFs successfully (e.g., central executive and sustained attention/vigilance) and alter the developmental trajectory of implicated brain systems. Supplementary training such as specialized, intensive academic tutoring will almost certainly be required to maximize far-transfer gains related to academic achievement.

TABLE 26.5. Mindfulness Study Characteristics

Author (year)	T (*n*)	C (*n*)	Program	Control group	Total minutes	Total sessions	Total weeks	Minutes/ session	Near-transfer ES (CI)	Far-transfer ES (CI)
van de Weijer-Bergsma et al. (2012)	10	—	Study Developed based on MBCT and MBSR	None	720	8	8	90	0.13, *ns* (–0.51 to 0.77)	–0.01, *ns* (–0.64 to 0.62)
van der Oord, Ponsioen, et al. (2012)	22[a]	11	Study Developed based on MBCT and MBSR	Waiting list	720	8	8	90	—	0.34 (0.16 to 0.53)
									Overall effect	
									0.13, *ns* (–0.51 to 0.77) *k* = 1	0.30 (0.09 to 0.52) *k* = 2

Notes. T, treatment group; C, control group; *n*, number of participants within each group; ES, effect size; CI, 95% confidence interval; MBCT, mindfulness-based cognitive therapy (Segal et al., 2012); MBSR, mindfulness-based stress reduction (Kabat-Zinn, 1990).

[a]Represents the larger number of children analyzed in the posttest contrasts and includes children who initially served as waiting-list controls and later participated in the active treatment condition.

KEY CLINICAL POINTS

- ✓ Current empirically supported treatments for children with ADHD produce significant acute beneficial effects during treatment, yet are unlikely to generalize to improving problems in untreated settings, and children fail to maintain these gains once treatment is withdrawn.
- ✓ Working memory (central executive) and sustained attention problems are well documented in children and teens with ADHD. These problems are associated with underdevelopment and underfunctioning of various brain regions and networks. It therefore makes sense to develop FIT programs that may improve or expand these neurological regions and networks by targeting these EF deficits. Targeting empirically identified neurocognitive functioning has the potential to improve the breadth, generalization, and maintenance of treatment gains over time relative to traditional treatments for ADHD.
- ✓ However, it is essential that FIT developers understand the nature of the EF findings and focus FIT treatments on these specific deficits. For instance, evidence indicates that the most significant WM problems in ADHD may be associated with the manipulation, updating, and reordering of information (executive) rather than with the online maintenance (storage) and rehearsal of information. FIT programs targeting maintenance and rehearsal features of WM are therefore not likely to improve outcomes for children with ADHD.
- ✓ Many FIT programs, such as those utilizing Cogmed or BrainTrain, claim to target WM but appear to primarily target storage/rehearsal rather than central executive processes such as manipulation, updating, and reordering. Other programs have focused on a mixed set of EFs and others on attentional components. These studies were combined into a meta-analysis to examine their effectiveness at (1) near-transfer improvements (improved performance on similar tasks to those used in training), (2) maintenance of such near-transfer improvements over time, and (3) immediate and maintenance of far-transfer improvements on tasks less related to the training tasks, ratings of ADHD symptoms, and EF deficits in daily life, or measures of academic achievement and school performance.
- ✓ Results of this meta-analysis indicated that none of the available programs stress training WM central executive processes and that FIT programs that targeted primarily WM storage and rehearsal produced significant near-transfer improvements. Those studies that targeted mixed EF components or attention abilities were not effective. Three studies collected follow-up measures, and these indicated that the gains on the near-transfer WM measures were sustained for 3–6 months, but such evidence is limited by so few studies examining this issue.
- ✓ Evidence for far-transfer effects of training on parent and teacher behavior ratings, academic achievement, or other functional outcomes was disappointing. Significant benefits were reported only by unblinded raters and appear to reflect illusory or Hawthorne effects rather than true changes in behavior. There was scant evidence of far-transfer effects based on objective measures of functional outcomes. These results are quite sobering and disappointing, and contradict claims made by treatment developers and FIT proponents that such programs are effective for children and teens with ADHD.
- ✓ These findings further suggest that FIT programs are not targeting the areas of greatest EF deficits—sustained attention and the manipulation, updating, and reordering aspects of WM.
- ✓ An alternative cognitive training program recently developed for children and teens with ADHD is mindfulness meditation training. As of this writing, only two studies have examined the use of mindfulness meditation training, and the results again were mixed and largely disappointing. While there is some evidence that this approach improves unblinded parent–teacher ratings, parents' active involvement in both delivering and receiving the intervention strongly suggests that the effects on their ratings are largely due to expectancy effects and not to treatment itself. No evidence was found for improvements in blinded teacher ratings or other functional outcomes.
- ✓ FIT programs have not demonstrated sufficient effectiveness for improving ADHD symptoms or the important functional outcomes related to the disorder; however, some FIT programs improve STM storage and rehearsal performance on specific verbal and nonverbal WM tasks.
- ✓ Until there is greater evidence of treatment effectiveness in ADHD, we do not recommend the adoption of these treatment approaches for routine clinical practice.

NOTES

1. Pneumoencephalography, a now obsolete medical procedure used during the early 20th century, involved draining most of the cerebrospinal fluid from around the brain and replacing it with air, oxygen, or helium to enhance X-ray imaging.
2. Estimates reflect the percentage of overlap between ADHD and non-ADHD groups (i.e., only approximately 19% of children with ADHD score within the typically developing range).
3. Multiplying the near-transfer effect size (expressed in *SD* units) by the beta-weight (which gives the *SD* change in the far-transfer outcomes associated with a 1 *SD* change in the near-transfer outcome), provides the maximum expected effect size for far-transfer that is attributable to improvements in the near-transfer (trained) construct. For example, if a 1 *SD* change in STM performance is associated with a 0.18 *SD* change in Stroop task performance, then a 0.86 *SD* change in STM performance (the near-transfer effect size) could yield a maximum of 0.16 *SD* change in Stroop performance ($0.86 \times 0.18 = 0.16$). The obtained ES could be higher, allowing for the possibility of synergistic effects, measurement unreliability, or improvements in unmeasured EF processes, but it could also be lower due to the use of all incongruent Stroop trials in the study, which nullifies its relationship with WM (Hutchison, 2011).

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*Denotes studies included in the FIT meta-analysis. **Denotes studies included in the mindfulness meta-analysis.

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CHAPTER 27

Stimulant and Nonstimulant Medications for Childhood ADHD

Daniel F. Connor

An expanding body of clinical research documents the safety and efficacy of medications to treat pediatric ADHD. A new generation of stimulants that has emerged over the last two decades improves duration of medication action through novel sustained-release formulations. Nonstimulant noradrenergic ADHD medications are now available and provide an evidence-based alternative to the stimulants. This chapter reviews stimulant and nonstimulant medications for child and adolescent ADHD.

STIMULANTS

Central nervous system (CNS) stimulant medications are the most common psychotropic medications used to treat the symptoms of attention-deficit/hyperactivity disorder (ADHD) in children, adolescents, and adults. Stimulants include compounds containing *d,l*-methylphenidate, *d*-methylphenidate, or *d*-amphetamine. Stimulants are sympathomimetic drugs that exert CNS actions similar to those of endogenous catecholamines. These compounds are known to enhance dopaminergic and noradrenergic neurotransmission in the CNS. At the level of individual neuronal neurotransmission, stimulants increase the availability of norepinephrine and dopamine at the synaptic cleft by reversibly binding to the presynaptic transporter protein with resultant inhibition of catecholamine reuptake into the presynaptic neuron, thereby increasing concentrations of catecholamines in the extraneuronal space, and enhancing postsynaptic CNS catecholaminergic neurotransmission (Volkow et al., 2002). Amphetamine also increases the release of dopamine from presynaptic cytoplasmic storage vesicles and blocks the uptake of dopamine into neuronal cytoplasmic storage vesicles, making dopamine more available in the presynaptic neuronal cytoplasm for release into the synaptic cleft. Thus, the actions of methylphenidate and amphetamine are not identical (Minzenberg, 2012).

Established Indications for Use

Established indications for stimulants include ADHD symptoms in children 6 years of age, adolescents, and adults. Specifically, stimulants are helpful in treating age-inappropriate and impairing symptoms of inattention to task, impulsive behavior, and motor hyperactivity that are not due to another cause, such as sleep apnea, depression, bipolar disorders, substance use disorders, anxiety disorders, or psychotic disorders, and are persistently severe enough to cause impaired func-

tioning at school, at work, at home, or in the community. In individuals with ADHD, stimulant treatment improves a wide variety of cognitive abilities such as sustained vigilance to nonreinforcing and repetitive tasks, increased productivity at school, and improved performance on academic testing. However, stimulants do not improve core cognitive vulnerabilities due to learning disabilities, which may require specific special educational remediation. Narcolepsy is an established indication for stimulant medications, but it will not be further discussed here.

Possible Indications for Use

Oppositional defiant disorder (ODD) and conduct disorder (CD) are psychiatric conditions frequently comorbid with ADHD. ODD is characterized by a recurring pattern of frequent angry/irritable mood and argumentative/defiance toward parents and authority figures. CD is defined as a repetitive and persistent pattern of behavior in which the basic rights of others or major age-appropriate social norms and rules are violated. Children with ADHD and either disorder may be vulnerable to the expression of impulsive aggression and/or having a "short fuse," leading to explosive episodes of irritable affect (Connor & Ford, 2012). Children with ADHD and comorbid ODD or CD are at risk for a poorer prognosis than children with ADHD alone. Children with ADHD and CD are especially at risk of developing antisocial behaviors as adults (Shaw et al., 2012). Although the standard of care emphasizes behavioral therapy for these disruptive behavior disorders, when they occur in the context of ADHD, stimulant treatment may be effective (Connor & Doerfler, 2008; Connor, Glatt, Lopez, Jackson, & Melloni, 2002).

Symptoms of hyperactivity and impulsivity, with or without sustained attentional deficits, are common in children with developmental disorders. ADHD occurs in children with intellectual disability at prevalence rates of 18–40% and in children with autism spectrum disorders at rates of 41–78% compared to ADHD rates of 4-7% in the general population (Murray, 2010). Indeed, the most recent edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-5; American Psychiatric Association, 2013), allows separate diagnoses of ADHD and autism spectrum disorder to be given in the same individual. Stimulants may be effective for ADHD symptoms in developmentally disabled children. However, response rates are lower, and adverse events are more frequent in these children. For example, one controlled study of methylphenidate in children with autism with impulsivity–hyperactivity found a 49% response rate and an 18% rate of treatment discontinuation associated with medication side effects (Research Units on Pediatric Psychopharmacology, 2005). This compares with the large Multimodal Treatment of ADHD Study (MTA; MTA Study Group, 1999) of 7- to 9-year-old children with ADHD, which found a 69% response rate with only a 1.4% discontinuation rate because of treatment emergent side effects. Nevertheless, the American Academy of Pediatrics now recommends stimulants as a medication choice in children with ADHD symptoms in the context of autism spectrum disorders (Mahajan et al., 2012). Treatment response may decrease and side effects may increase when the child's IQ is less than 55 (Research Units on Pediatric Psychopharmacology, 2005).

Traumatic brain injury (TBI) may result from infectious disease in the CNS (encephalitis), accident, violence, or vascular disease. Depending on the location of the CNS insult, TBI may cause hyperactivity, distractibility, and/or impulsivity, especially if the prefrontal cortex sustains insult. For example, in one study conducted over 2 years after childhood TBI, new-onset ADHD following injury ranged from 14.5% at 12 months to 18.3% at 24 months (Levin et al., 2007). Although further research is needed, researchers find modest support for the efficacy of stimulants in the treatment of TBI-associated ADHD symptoms in children and adults (Jin & Schachar, 2004).

Symptoms such as inattention and impulsivity–hyperactivity may frequently occur in children, adolescents, and adults with epilepsy or other seizure disorders. ADHD symptoms are found in 12–39% of children with epilepsy (Torres, Whitney, & Gonzalez-Heydrich, 2008). It is unclear whether the symptoms of ADHD are caused by the epilepsy, exacerbated by anticonvulsant medications, and/or constitute a separate comorbid disorder. A number of mechanisms may contribute to the higher rate of ADHD in children with epilepsy than in the general pediatric population. These may include the effects of CNS neuropathology underlying both ADHD and epilepsy, the effects of certain anticonvulsant medications (e.g., phenobarbital is known to exacerbate ADHD symptoms), the cognitive effects of chronic epilepsy on CNS mechanisms subserving attentional, emotional, and behavioral control, and/or the possible effects of electroencephalography (EEG)–ascertained nonconvulsive epileptiform discharges on

vigilance, working memory, and processing speed (Torres et al., 2008). Although the research base to date is not extensive, methylphenidate formulations are studied more than amphetamine preparations in children with epilepsy and ADHD symptoms. In general, these studies report significant improvement in ADHD symptoms in patients with epilepsy and ADHD, without an exacerbation of seizures or an adverse effect on anticonvulsant drug serum levels (Baptista-Neto et al., 2008). What remains unclear is the effect of stimulants on seizure thresholds in patients whose epilepsy is untreated or poorly controlled by anticonvulsive therapy. Although preclinical studies indicate concern that stimulants may lower the seizure threshold in uncontrolled epilepsy, open-label clinical studies in children with uncontrolled epilepsy and ADHD suggest that methylphenidate is effective and does not increase seizure frequency in the majority of children studied (Koneski, Casella, Agertt, & Ferreira, 2011).

ADHD may present early in life with impairing symptoms in preschool children. The diagnosis appears relatively stable over a 6-year period (Riddle et al., 2013). The Preschool ADHD Treatment Study (PATS) is a six-site study funded by the National Institutes of Mental Health (NIMH) to determine the efficacy and safety of immediate-release methylphenidate (MPH-IR) given three times daily to children ages 3.0 to 5.5 years with ADHD who did not respond to behavioral therapy. In 165 preschoolers with ADHD randomized into the medication trial, significant improvement in ADHD symptoms was found for MPH-IR at 2.5-mg, 5-mg, and 7.5-mg doses given three times daily (Greenhill et al., 2006). However, compared with older children with ADHD on the same medication, effect sizes were smaller in preschoolers with ADHD who also had more medication side effects, such as irritability, feeling crabby, and more proneness to crying, than in school-age ADHD children. While the PATS study demonstrates that preschoolers with ADHD may respond to MPH-IR, given the higher rate of treatment emergent side effects, smaller doses of stimulants, slower titration schedules, and close clinical monitoring of these preschoolers on stimulant medication is advised (Vaughan & Kratochvil, 2012).

Stimulant Effects

Numerous studies have indicated that stimulants enhance performance on measures of vigilance, impulse control, fine motor coordination, and reaction time. Higher stimulant doses tend to be associated with more robust responses, and clinicians should beware of underdosing. Positive stimulant effects have been obtained on measures of short-term memory and learning performance on both simple and complex learning paradigms. Stimulants facilitate the speed of symbolic and verbal information retrieval. Reduced risk-taking behaviors and improvements in interpersonal and social relationships are noted when ADHD children and adolescents are treated with stimulants. Increases in self-esteem may be observed (DeVito et al., 2008). There is now research that focuses on elucidating the neurobiological mechanisms of action of stimulants in enhancing cognition (Arnsten & Rubia, 2012; Swanson et al., 2013).

Pharmacoepidemiology of Stimulant Use

Rates of stimulant use continue to grow among children and adolescents in the United States. Studies documenting this trend utilize a variety of methodologies and time frames, yet conclusions are generally consistent. For example, Winterstein and colleagues (2008) conducted a 10-year longitudinal analysis of claims data for all Medicaid beneficiaries younger than 20 years of age with 6 months or more of continuous insurance, identifying 2,131,953 covered lives, to determine longitudinal changes in ADHD drug utilization. Between 1995 and 1996 and 2003 and 2004, ADHD prevalence increased 1.70-fold and ADHD drug use increased 1.84-fold. In 2003 and 2004, 20% of 10- to 14-year-old white males received an ADHD medication (mostly stimulants). While the most common age for starting an ADHD medication in this claims-based study was 5–9 years, large increases were observed for adolescents starting an ADHD medication for the first time (Winterstein et al., 2008). In another study, data from the U.S. National Ambulatory Medical Care Survey (NAMCS) for the years 1991 through 2008 were used to identify office-based visits (OBVs) in which medications for ADHD were prescribed (Sclar et al., 2012). Over the 18-year time frame examined, they discerned a 4.1-fold increase in the diagnosis of ADHD and a 4.9-fold increase in the rate of OBVs in which medications for ADHD were prescribed. In a third study, the 1996–2008 database of the Medical Expenditure Panel Survey, a nationally representative annual survey of U.S. households, documented an annual growth rate of stimulant use of 3.4% in children 18 years and younger (Zuvekas & Vitiello, 2012). These studies document a

steadily upward trend in ADHD drug utilization rates for children and adolescents. Part of this trend is due to increasing rates of ADHD diagnosis in the population. For example, an ecological study of trends in ADHD diagnosis in a large managed care network found a 24% increase in rates of ADHD diagnosis between 2001 and 2010 (Getahun et al., 2013). These studies also document gender and ethnic differences in prescribing, such that white males are more likely diagnosed and treated than Hispanic or African American males, and that males are more likely to be treated than females. While gender and ethnic differences persist, the age distribution of ADHD medication treatment appears to have shifted toward older children and adolescents, suggesting that treatment of middle school and adolescent children is now driving ADHD medication treatment rates (Winterstein et al., 2008).

In addition to rising rates of ADHD monotherapy use in children and adolescents, rising rates of ADHD medication coprescription are documented. This refers to the simultaneous prescription of two or more medications of different classes to the child with ADHD. Examining patterns and trends in multiclass psychotropic treatment among youth OBVs to physicians in the United States, Comer, Olfson, and Mojtabai (2010) documented an increase in the percentage of child visits in which two or more psychotropics were prescribed from 14.3% in 1996 to 20.2% in 2007. Stimulants were combined with antidepressants, sedatives–hypnotics, and mood stabilizers. There was a significant increase in the coprescription of ADHD medications and antipsychotic medications (Comer et al., 2010). It is presently unclear whether coprescription is driven by frequent comorbid psychiatric conditions requiring additional treatment in children with ADHD and/or by suboptimal effectiveness of ADHD monotherapy, and/or some other factor(s). What is clear is that although there is scant research on the safety and efficacy of combined pharmacotherapy for ADHD, coprescription is becoming increasingly common in clinical practice.

Rising rates of stimulant prescription have led to widely publicized concerns over the possibility of ADHD overdiagnosis and widespread ADHD overmedication in U.S. children. This concern needs to be placed in perspective and tempered with the available scientific evidence. While stimulant prescription rates are rising and vary by geographic location, and some studies have found specific geographic areas of stimulant overprescribing (Angold, Erkanli, Egger, & Costello, 2000), most evidence suggests either appropriate prescribing or undertreatment of ADHD. For example, in a representative sample of 10,123 adolescents ages 13–18 years who participated in the National Comorbidity Survey Adolescent Supplement, there was little evidence to support concerns of widespread overmedication use or practitioner overprescribing. Only one-third of adolescents with ADHD received stimulants (Merikangas, He, Rapoport, Vitiello, & Olfson, 2013). Recent data from the National Health and Nutrition Examination Survey, a nationally representative probability sample of children ages 8–15 years residing in the community, indicate an ADHD prevalence rate of 7.8%, but only 48% of the ADHD sample had received any treatment over the past 12 months (Merikangas et al., 2010). Thus, despite much public concern about the possibility of ADHD overdiagnosis and overprescribing, there is little scientific evidence to support such claims (Connor, 2011).

Stimulants for ADHD in Children and Adolescents

Stimulants include both methylphenidate (MPH) and amphetamine (AMP) compounds. As a treatment for ADHD, the stimulants have decades of efficacy and safety data from hundreds of randomized controlled trials, beginning in the 1970s. Stimulant efficacy and safety data have expanded from an initial focus on school-age children with ADHD to encompass preschoolers, adolescents, and adults with ADHD. A more limited stimulant literature exists for females and ethnic minorities (Adler, 2009; Biederman & Spencer, 2008; Kratochvil, Greenhill, March, Burke, & Vaughan, 2004; Vaughan & Kratochvil, 2012).

The MTA, funded by the NIMH, is the basis for current childhood ADHD standards of practice recommended by both the American Academy of Child and Adolescent Psychiatry and the American Academy of Pediatrics. The MTA randomized 579 children with ADHD age 7.0 to 9.9 to one of four treatment arms: (1) manualized and standardized pharmacotherapy with MPH, (2) intensive behavioral therapy, (3) combined therapy with the two treatments, and (4) community care (MTA Study Group, 1999). At 14-month outcome, both the stimulant arm and combined treatment groups showed significant improvement in ADHD symptoms, and both were superior to behavioral therapy alone and community treatment as usual. Because the combined treatment group did not have a significantly better outcome than the stimulant-alone

group, the MTA authors concluded that stimulant medication alone appeared to have the most impact on ADHD symptoms. This provides the scientific basis for the current clinical practice of stimulant monotherapy as the first-line intervention for ADHD in school-age children (Greenhill et al., 2002). Combination treatment did lead to greater parenting satisfaction, a reduction in comorbid anxiety symptoms, and use of lower doses of MPH (Swanson et al., 2008). Given that most community as-usual treatment involved stimulant medications, the MTA demonstrated the importance of systematic dosing and titration schedules and regular monthly follow-up in improving outcomes for children receiving ADHD medications (MTA Study Group, 1999).

Children in the MTA study have now been followed for up to 8 years since the original study ended. Longitudinal assessment has facilitated a greater understanding of how ADHD unfolds across development in treated children. Consistent with results found in many other studies, children with ADHD are more at risk for the development of delinquency and substance abuse than their peers without ADHD; however, treatment with stimulants does not appear to affect vulnerability to the development of substance use disorders (Molina et al., 2013). In other words, stimulant medication for ADHD does not protect adolescents from or contribute to their risk of substance misuse. Higher risk for substance use disorder in ADHD youth appears to be conferred by the diagnosis of ADHD rather that by treatment for ADHD.

Studies indicate that between 65 and 75% of children with ADHD initially treated with a stimulant are described as improved. Between 25 and 30% of such children do not respond or do not tolerate initial stimulant medication. If a second stimulant is clinically tried, response rates may increase to 80–90% (Greenhill et al., 2002; Pliszka, 2007). Thus, the clinician should consider use of both MPH and AMP formulations before other classes of agents are considered. Research has identified one group of children with ADHD that responds preferentially to MPH, another that responds to AMP, and still another that responds to both types of stimulant (Greenhill et al., 1996). ADHD placebo responses are low, generally ranging from 2 to 39% (Greenhill et al., 2002). For example, in the MTA, placebo response rates of 13% were reported (MTA Study Group, 1999).

Recent ADHD stimulant drug development has focused on novel stimulant delivery systems designed to extend the daily duration of effective drug response and overcome acute daily drug tolerance (i.e., tachyphylaxis), which may diminish stimulant treatment effects after multiple daily dosing. As seen in Tables 27.1 and 27.2 MPH and AMP now come in a multitude of drug delivery systems that extend the duration of action of these medications and, with intermediate and long-acting preparations, allow once-daily dosing in the morning before school.

The current standard of care in the treatment of childhood ADHD now favors use of longer-acting formulations as a first-line intervention both for ease of use and more consistent ADHD treatment coverage, without the acute plasma peaks and troughs associated with multiple daily-dose immediate-release stimulant preparations. Intermediate-acting formulations are designed to cover the school hours with a once-daily dose preparation. Long-acting compounds are designed to cover both the school day and the afterschool hours with a single dose, given in the morning before school. Longer-acting formulations can then be complemented ("sculpting the dose") with immediate-release preparations for additional ADHD coverage in the early evening hours.

Different delivery systems facilitate a more personalized, patient-specific approach to treatment. For children who have difficulty swallowing pills and capsules, there is now an MPH-IR solution formulation (Methylin) and a long-acting methylphenidate extended-release solution preparation (Quillivant XR). Beaded capsule delivery systems contain differing percentages of immediate-release beads designed for rapid onset of action, and enteric-coated delayed-release beads designed to release medication approximately 4 hours later. The beaded system thus allows two doses of medication to be delivered in a single capsule. In general, a higher percentage of immediate-release beads in the delivery system will facilitate a more rapid onset of stimulant action (Connor & Steingard, 2004). For example, Metadate contains 30% MPH-IR beads and 70% delayed-release beads to provide stimulant availability over 8 to 9 hours. Ritalin LA and Focalin XR both contain a 50:50 ratio of immediate- to delayed-release MPH beads that allow for extended ADHD daily coverage. Adderall XR is a 50:50 ratio of immediate- to delayed-onset mixed AMP salt beads that allows for a pharmacokinetic profile similar to that of immediate-release Adderall given twice daily. For children who have difficulty swallowing capsules, these formulations may be opened and the beads sprinkled

TABLE 27.1. Methylphenidate Formulations FDA-Approved for Children with ADHD

Medication	Dose range (mg/day)	Delivery system	Duration of effect (hours)	FDA-approved age	Comments
Short-acting formulations					
Methylphenidate (Ritalin)	2.5–60	Tablet	3–4	Children ≥ 6 years	Give in multiple daily doses
Methylphenidate (Methylin)	5–lesser of 2.0 mg/kg/day or 60 mg	Solution[a]/ chewable tablet	4	Children ≥ 6 years	Give in multiple daily doses
d-Methylphenidate (Focalin)	2.5–lesser of 1.0 mg/kg/day or 20 mg	Tablet	4	Children ≥ 6 years	Give in multiple daily doses
Intermediate-acting formulations					
Methylphenidate (Ritalin SR)	20–60	Slow-release tablet	5–6	Children ≥ 6 years	Give in two daily doses
Methylphenidate (Metadate ER)	10–lesser of 2.0 mg/kg/day or 60 mg	Beaded capsule	6–8	Children ≥ 6 years	Single-dose coverage for school hours
Methylphenidate (Methylin ER)	10–lesser of 2.0 mg/kg/day or 60 mg	Beaded capsule	6–8	Children ≥ 6 years	Single-dose coverage for school hours
Methylphenidate (Ritalin LA)	10–60	Beaded capsule	7–9	Children ≥ 6 years	Single-dose coverage for school hours; may open capsule and sprinkle beads on applesauce
Methylphenidate (Metadate CD)	10–lesser of 2.0 mg/kg/day or 60 mg	Beaded capsule	8–9	Children ≥ 6 years	Single-dose coverage for school hours
Long-acting formulations					
Methylphenidate (Concerta)	18–lesser of 2.0 mg/kg/day or 72	OROS capsule	9–12	Children ≥ 6 years	Single-dose coverage for school and after school hours
d-Methylphenidate (Focalin XR)	5–lesser of 1.0 mg/kg/day or 30 mg)	Beaded capsule	9–12	Children ≥ 6 years	Single-dose coverage for school and after school hours; may open capsule and sprinkle beads on applesauce
Methylphenidate (Quillivant XR)	20–60	Solution[b]	9–12	Children ≥ 6 years	Shake bottle vigorously for at least 10 seconds before each use.
Methylphenidate (Daytrana Patch)	10-mg patch strength–lesser of 1.0 mg/kg/day or 30-mg patch strength	Multipolymeric adhesive patch system	9–12	Children ≥ 6 years	Wear patch up to 9 hours for 12-hour effectiveness; switch patch placement on hip daily

[a]Methylin Solution: 5 mg/5 ml and 10 mg/5ml.

[b]Quillivant XR: 25 mg/5ml.

TABLE 27.2. Amphetamine Formulations FDA-Approved for Children with ADHD

Medication	Dose range (mg/day)	Delivery system	Duration of effect (hours)	FDA-approved age	Comments
Short-acting formulations					
Mixed amphetamine salts (Adderall)	2.5–lesser of 1.0 mg/kg/day or 40 mg	Tablet	5–6	Children ≥ 3 years	Give once or twice daily
Amphetamine (Dexedrine)	2.5–40	Tablet	4	Children ≥ 3 years	Give in multiple daily doses
Amphetamine (Dextrostat)	2.5–40	Tablet	4	Children ≥ 6 years	Give in multiple daily doses
Long-acting formulations					
Mixed amphetamine salts (Adderall XR)	5–lesser of 1.0 mg/kg/day or 30 mg	Beaded capsule	10	Children ≥ 6 years	Single-dose coverage for school hours; may open capsule and sprinkle beads on applesauce
Amphetamine (Dexedrine Spansule)	5–lesser of 1.0 mg/kg/day or 40 mg	Capsule	10	Children ≥ 6 years	Single-dose coverage for school hours
Lisdexamfetamine (Vyvanse; prodrug)	20–lesser of 1.0 mg/kg/day or 70 mg	Capsule	10	Children ≥ 6 years	Single-dose coverage for school hours

on food without loss of their delayed-release profile. Concerta uses an osmotic pump mechanism to release MPH that provides stimulant availability that mimics MPH-IR given three times daily. An outer coat on the medication delivers 18% of the total stimulant dose as MPH-IR. The Concerta capsule must be swallowed whole. Lisdexamfetamine is a novel formulation in which *d*-amphetamine is covalently bound to the amino acid L-lysine. In the bound state the prodrug is inactive. Active drug is released in a rate-controlled manner after oral administration and rate-limited enzymatic hydrolysis of the covalent bond, allowing for long-acting *d*-amphetamine delivery. A MPH transdermal delivery system utilizes a multipolymeric adhesive layer attached to a backing. MPH is contained within the adhesive layer, and active drug is delivered through the skin in a consistent manner while the patch is worn on the hip. Differing MPH daily doses are contained in patches of different sizes. Duration of stimulant drug action is dependent on wear time. Generally, the patch is worn for 9 hours, which allows 12 hours of stimulant coverage as the drug is slowly absorbed from the skin upon patch removal. The patch system is useful for children with ADHD who have difficulty swallowing pills or who need a flexible duration of daily stimulant effect. The patch location must be switched from one hip to the other every day because the patch typically causes skin irritation.

Side Effects

Common, Short-Term, and Acute Side Effects

Stimulant medications are generally well tolerated. MPH and AMP preparations have similar side effects. Side effects do occur, but they are generally mild and can usually be managed by dose adjustment or changing the timing of medication intake. Common treatment-emergent stimulant side effects include insomnia, decreased appetite, weight loss, headache, stomachache, and small increases in heart rate and blood pressure. Clinically, it is important to ascertain reported medication side effects at baseline, before the child is on stimulant medication, then again at full dose. Many of the child–parent-reported medication side effects may actually be aspects of the disease and improve with treatment. Short-term acute stimulant side effects are

generally related to dose, with higher doses associated with more reported side effects. In special populations, there may be a higher incidence of stimulant-related side effects. Preschool children with ADHD who are treated with stimulants may experience a higher rate of adverse effects than older children, particularly crying, irritability, and emotional outbursts (Greenhill et al., 2006). Children with developmental delays such as autism or mental retardation (IQ ≤ 55) may also experience elevated rates of stimulant side effects (Research Units on Pediatric Psychopharmacology, 2005). These populations require increased clinical attention to monitor stimulant-related side effects.

Because stimulant therapy may exacerbate motor and vocal tics in some children with ADHD who have a personal or family history of tics or Tourette disorder, tics were previously considered to be a relative contraindication to stimulant therapy. However, more recent research has challenged this view and suggests that stimulants may be used in children with moderate to severe ADHD and mild tic disorders (Gadow, Sverd, Sprafkin, Nolan, & Ezor, 1995). Given the risk of stimulants transiently worsening preexisting tics in such children, the risks and benefits of stimulants, as well as alternative ADHD treatment, should be discussed with the family prior to treatment. Careful clinical monitoring of tic severity and frequency, as well as ADHD symptom response, is important in children with ADHD + tics who are treated with stimulants. Except for exposure to very high or toxic doses of stimulants, there is little evidence to suggest that routine clinical stimulant treatment for ADHD can cause new tics in children who were not already predisposed or vulnerable to tic disorders (Law & Schachar, 1999).

Stimulants can exacerbate psychosis in individuals with a preexisting psychotic disorder, such as schizophrenia or mania, or who possess a vulnerability to psychotic symptoms, such as a personal history of hallucinations. Psychosis may be an acute manifestation of stimulant toxicity, such as that occurring upon overdose of stimulant medications or a rapid escalation to a high daily dose of stimulants. Individuals with a psychotic reaction to stimulants should be clinically monitored for a recurrence or development of a psychotic illness (McKetin, Lubman, Baker, Dawe, & Ali, 2013).

Stimulants are sympathomimetic drugs and may raise blood pressure and pulse rate. These cardiovascular effects appear to be of little clinical significance in healthy children (although they may be of more clinical significance in stimulant-treated adults with ADHD; Vitiello et al., 2012). Recent concerns about cardiovascular safety and rare risk for sudden death in stimulant-treated children and adolescents with ADHD have not been supported by the extant scientific literature (Martinez-Raga, Knecht, Szerman, & Martinez, 2013; Olfson et al., 2012). However, monitoring of pulse and blood pressure in treated children at risk for hypertension, such as those with obesity or renal disease, is recommended. Risk for stimulant-induced adverse cardiovascular events is elevated in children with ADHD and a family history significant for early cardiac death, arrhythmia, cardiogenic syncope, or a personal history of structural cardiac abnormalities, exercise-induced syncope, chest pain, or palpitations. In such cases, consultation with a pediatric cardiologist prior to stimulant treatment is recommended.

In treated children with ADHD, deterioration in behavior and ADHD symptom control may occur in the afternoon and evening following earlier administration of stimulant medication. Deterioration in behavior may exceed that expected from baseline ADHD symptoms. This phenomenon is referred to as "rebound," and its cause and prevalence are unclear. Should rebound occur, the use of longer-acting stimulant preparations, or the addition of a small dose of immediate-release stimulant 1 hour before the onset of symptom exacerbation, may reduce rebound symptoms late in the day.

Long-Term Side Effects

Long-term stimulant use continues to elicit concern about growth trajectories across development in treated children with ADHD. Stimulants routinely produce mild anorexia and appetite suppression, and weight deficits are generally greater than height deficits. For most patients treated with stimulants into adolescence or adulthood, deficits in growth are modest. For example, in a meta-analysis of 22 studies of stimulant effects on growth, statistically significant effects on height and weight were seen in stimulant-treated youth, with more significant weight deficits than height deficits, and equally associated with either long-acting or MPH-IR or AMP formulations. The effects on growth appeared greater for taller and heavier children, and for children ages 6–12 years compared with adolescents (Faraone, Biederman, Morley, & Spencer, 2008). Discontinuing stimulant medication appears to attenuate deficits in weight but not height (Swanson et al., 2007). Some studies suggest that children with ADHD may display different growth trajectories than do children with-

out ADHD, and that the disorder itself, not stimulant treatment, is the cause for growth dysregulation (Spencer et al., 1996). ADHD standards of practice mandate monitoring of height, weight, and body mass index for gender and age in stimulant-treated children and adolescents. Results should be serially plotted on standardized growth curves. A change in weight or height crossing two percentile lines on a standardized growth curve should suggest a deficit in growth and prompt a clinical response. Responses might include stimulant drug holiday, nutritional supplementation, dose reduction, or change to a nonstimulant ADHD medication (Pliszka, 2007). Stimulant effects on child growth should be part of a risk–benefit discussion with the child and family as part of the informed consent for treatment process prior to stimulant initiation.

There is considerable evidence to support increased risk for substance use disorders in individuals with ADHD, with or without associated CD (Wilens et al., 2011). The available evidence suggests that risk is conferred by the ADHD disorder itself, not by treatment for the disorder. For example, the MTA found no association between stimulant treatment and risk for substance abuse in adolescents at 8-year follow-up (Molina et al., 2013). Moreover, stimulant treatment for childhood ADHD does not appear to increase risk for later adult substance use disorder (Biederman, Monuteaux, et al., 2008; Mannuzza et al., 2008). "Diversion" is the misuse of stimulants in persons without a diagnosis of ADHD or narcolepsy. The evidence to date suggests that the prevalence of diversion and misuse of stimulants is especially a concern in adolescent and young adult student populations and in users of other illicit drugs (Kaye & Darke, 2012). Prior to treatment with stimulants, the clinician should obtain a careful personal and family history of substance abuse, and should educate the high school or college student and develop a strategy about recognizing and managing the potential risk of stimulant diversion.

Contraindications to Stimulant Use

Known hypersensitivity to stimulants is a contraindication to their use. Patients with structural cardiac defects should not be treated with stimulants. Stimulants can exacerbate narrow-angle glaucoma and should not be used by persons with this condition. In vulnerable individuals or in overdose (toxicity), stimulants can cause psychotic symptoms. Stimulants are relatively contraindicated in children and adolescents with schizophrenia or other psychotic disorders because they may worsen these conditions in some cases. A severe tic or Tourette syndrome remains a relative contraindication to the use of stimulants. However, as noted earlier, stimulants may be used in milder cases of tics when these are accompanied by impairing symptoms of ADHD. Patients with unstable hypertension should not receive stimulants for ADHD until their high blood pressure is treated and controlled. Because stimulants have the potential to be abused, they should not be prescribed when patients exhibit active and moderate to severe substance abuse or when there is likelihood that family members or friends will abuse the medication. Finally, stimulants have the potential to precipitate hypertensive crises when used with monoamine oxidase inhibitors (MAOIs). They should not be prescribed concurrently with a MAOI or within 14 days after an MAOI has been discontinued.

NONSTIMULANT MEDICATIONS FOR CHILDHOOD ADHD

Stimulants are the first line of medication treatment and the most widely prescribed medications for pediatric ADHD. However, up to 30% of children and adolescents may have an inadequate or only partial symptom response to stimulants, or cannot tolerate stimulants because of side effects (Pliszka, 2007). In addition, because stimulants are controlled substances, some families express concern over their use based on lingering fears about the abuse potential of these medications. Nonstimulant drugs approved by the U.S. Food and Drug Administration (FDA) for use in childhood ADHD (see Table 27.3) include atomoxetine (Strattera™), a long-acting formulation of clonidine (Kapvay™), and a long-acting guanfacine preparation (Intuniv™).

Atomoxetine (trade name: Strattera)

Atomoxetine selectively blocks norepinephrine reuptake at the noradrenergic neuron and is known as a selective norepinephrine reuptake inhibitor (SNRI). Atomoxetine was the first FDA-approved nonstimulant medication for ADHD in children, adolescents, and adults. More than 15 randomized controlled trials establish the effectiveness of atomoxetine for ADHD in children and adolescents (Tanaka, Rohde, Jin, Feldman, & Upadhyaya, 2013). Atomoxetine is significant-

TABLE 27.3. Nonstimulant Formulations FDA-Approved for Children with ADHD

Medication	Dose range (mg/day)	Delivery system	Duration of effect (hours)	FDA-approved age	Approved for coadministration with stimulants?	Comments
Atomoxetine (Strattera)	0.5 mg/kg/day–lesser of 1.4 mg/kg/day or 100 mg	Capsule	24	Children ≥ 6 years and adolescents	No	Give once-daily or in two evenly divided daily doses
Guanfacine XR (Intuniv)	1–4	Extended-release tablet	24	Children ≥ 6 years and adolescents	Yes	May give dose in morning or evening
Clonidine ER (Kapvay)	0.1–0.4	Extended-release tablet	12	Children ≥ 6 years and adolescents	Yes	Must give twice daily in two evenly divided doses; if unequal doses, give the higher dose at bedtime

ly more effective than placebo, and its efficacy does not differ from MPH-IR (Garnock-Jones & Keating, 2009). However, atomoxetine is less effective in head-to-head comparator trials than long-acting oral osmotically released (OROS) MPH (Newcorn et al., 2008) or mixed-amphetamine salts extended-release formulations (Wigal et al., 2005).

Atomoxetine is FDA-approved in children 6 years or older, adolescents, and adults. It is administered as a capsule that can be given once daily or in two divided doses. In 6- to 12-year-old children atomoxetine is dosed by weight. Dosing is generally initiated at 0.5 mg/kg/day and gradually titrated over 2–3 weeks to a target dose of up to 1.4 mg/kg/day (Biederman & Spencer, 2008). In adolescents, the target dose is up to a maximum of 100 mg/day. Atomoxetine has a graded dose–response such that higher doses result in greater ADHD symptom reduction and improvement in psychosocial functioning up to 1.8 mg/kg/day, without any significant increase over lower doses in the frequency of treatment emergent adverse events (Michelson et al., 2001). Patients should be informed that it may take up to 12 weeks on atomoxetine for a full treatment effect to emerge (Bushe & Savill, 2014). In patients who are only partially responsive to atomoxetine monotherapy, combination with stimulants may sometimes improve treatment outcomes. Although combination therapy is not FDA-approved, in a recent review, Treuer and colleagues (2013) found the combination to be generally well tolerated by patients, without serious adverse events.

Atomoxetine undergoes extensive metabolism and biotransformation. Cytochrome P4502D6 is the principal oxidative enzyme for atomoxetine. Genetic polymorphisms of cytochrome P4502D6 exist, such that 5–10% of individuals of European ancestry and up to 1% of Japanese individuals are poor metabolizers of atomoxetine. The rate of clearance of atomoxetine is only 10% that of extensive metabolizers, and these individuals are at risk of exposure to high drug concentrations and run a risk of greater drug side effects (Sauer et al., 2003). Care should be taken with the coadministration of atomoxetine with medications known to inhibit the cytochrome P4502D6 enzyme system, such as fluoxetine and paroxetine.

Common adverse events include headache, abdominal pain, decreased appetite, nausea, vomiting, and somnolence. The majority of adverse events are mild, and atomoxetine is generally well tolerated. Atomoxetine is associated with clinically insignificant increases in heart rate and blood pressure. It may be less likely than stimulants to exacerbate disordered sleep in pediatric patients with ADHD (Garnock-Jones & Keating, 2009). Both preclinical and clinical data suggest that atomoxetine has a low abuse potential. Thus, it may be an effective ADHD treatment in patients at risk for substance use disorders (Heil et al., 2002). There appear to be fewer height and weight growth deficits on atomoxetine than on stimulants (Spencer et al., 2005). Rare cases of severe liver injury have been reported with atomoxetine, and the medication should be discontinued in patients who develop pruritus, jaundice,

dark urine, and/or right upper-quadrant abdominal pain, symptoms suggestive of liver injury. Although no deaths from suicide have been reported, the FDA mandates that a black-box warning for suicidal ideation be included in prescribing information for atomoxetine. This is based on meta-analytic data showing that atomoxetine is associated with a statistically significant elevation in the incidence of suicidal ideation compared with placebo. However, more recent studies have questioned this. A meta-analysis of comparative suicide-related events between atomoxetine and MPH published in five randomized controlled comparator trials (n = 1,024), found a low overall suicide risk, and no significant difference between medication classes in suicide risk (Bushe & Savill, 2014).

Clonidine Extended Release (trade name: Kapvay)

Clonidine extended release (Clon-ER) is an alpha$_2$-receptor agonist that facilitates noradrenergic neurotransmission in the primate prefrontal cortex, although its exact mechanism of action in human CNS is unknown (Arnsten, Scahill, & Findling, 2007). Based on the results of two independent randomized controlled trials, Clon-ER was approved by the FDA for the treatment of ADHD in children and adolescents ages 6–17 years (Jain, Segal, Kollins, & Khayrallah, 2011; Kollins et al., 2011). Clon-ER is also FDA-approved as an adjunctive therapy in combination with stimulants in children and adolescents with ADHD and an inadequate symptom response to stimulant monotherapy (Kollins et al., 2011).

Clon-ER is a modified oral formulation of clonidine that reduces and delays peak plasma drug concentration compared to the unmodified clonidine immediate release preparation. This results in diminished daily drug concentration peaks and troughs that result from multiple daily dosings of the clonidine immediate release compound and are associated with adverse events such as sedation. The pharmacokinetic profile of Clon-ER provides for a 12-hour pharmacokinetic half-life, resulting in a twice-daily dosing schedule for children and adolescents. Bioavailability is only 89% that of clonidine immediate release, so the two medications are not identical.

Clon-ER is an extended-release tablet and must be swallowed whole and not cut, crushed, or chewed. It may be taken with or without food. Due to the different pharmacokinetic profile of Clon-ER compared with immediate-release clonidine, substitution on a milligram-for-milligram basis with clonidine is not recommended. Dosing is initiated at 0.1 mg at bedtime. The daily dose is adjusted weekly on a 0.1-mg basis until the desired response is achieved. Doses should be taken twice daily, with the equal or higher dose administered at bedtime. The maximum daily Clon-ER dose is 0.2 mg twice daily (0.4 mg/day). This same dosing schedule is used when Clon-ER is administered as an add-on to stimulants. When discontinuing Clon-ER, the dose must be gradually tapered to prevent rebound hypertension. The total daily dose should be tapered in decrements of no more than 0.1 mg every 3–7 days. Patients should be educated about the need for gradual dose discontinuation and advised of the possible side effects of abruptly stopping Clon-ER.

The side effects of Clon-ER are related to the alpha-adrenergic blocking effects of clonidine. Side effects are dose-related and typically diminish in intensity over time. Common side effects include sedation, somnolence, and fatigue, especially in the first weeks of treatment as the dose is titrated upwards. In the registration trial for Clon-ER, the frequency of sedation, somnolence, and fatigue approached 40% and was the major reason for treatment discontinuation (Jain et al., 2011). Alpha-agonists are centrally active antihypertensive agents, and Clon-ER typically produces small decrements in pulse and blood pressure. Exercise-related side effects such as feeling faint, dizzy, or lightheaded, should prompt consultation with the physician and consideration of dose reduction. In children and adolescents with a personal history of syncope, bradycardia, a previous history of cardiac abnormalities, or cardiac surgical repair, medical clearance from a pediatric cardiologist should be sought before initiating Clon-ER. Because dehydration may exacerbate risk for orthostatic hypotension, patients should be advised to avoid dehydration and becoming overheated. When used in combination with stimulants, the side effects of Clon-ER and stimulants appear to be independent of one another, and studies have not supported the presence of harmful interactions (Kollins et al., 2011).

Guanfacine Extended Release (trade name: Intuniv)

Guanfacine extended release (GXR) is an FDA-approved alpha$_2$-receptor agonist for the once-daily treatment of ADHD in 6- to 17-year-old children and adolescents. The efficacy of GXR as monotherapy for ADHD

is supported by two short-term, pivotal 8- and 9-week trials (Biederman, Melmed, et al., 2008b; Sallee, McGough, et al., 2009). The safety profile of GXR for pediatric ADHD is supported by two long-term (24 month) open-label studies (Biederman, Melmed, et al., 2008a; Sallee, Lyne, Wigal, & McGough, 2009). GXR is also FDA-approved for adjunctive use with stimulants for ADHD symptoms only partially responsive to stimulant monotherapy (Wilens et al., 2012). Oppositional defiant disorder (ODD) is a frequently occurring comorbid condition in ADHD. Although not FDA approved for ODD, one randomized controlled study demonstrated the efficacy of GXR on ODD symptoms in 6- to 12-year-old children with ADHD (Connor et al., 2010).

GXR is a modified extended-release tablet containing guanfacine as the active drug. It is formulated for once-daily dosing either in the morning or at bedtime. The tablet should not be cut, crushed, or chewed because this will defeat the extended-release tablet modification and allow an increased rate of guanfacine release. GXR should not be substituted on a milligram-for-milligram basis with immediate-release guanfacine. For example, GXR has only 57% of the bioavailability of immediate-release guanfacine; thus, the two drug formulations are not identical. Unlike Clon-ER, there is a food effect with GXR. Administration in the fasting state results in increased maximum GXR plasma concentration compared to administration in the fed state (Sallee, Connor, & Newcorn, 2013). GXR is initiated at 1 mg/day and titrated every 3–7 days to a maximum daily dose of 4 mg/day, or until the desired response is achieved. This same dosing schedule is used when GXR is administered as an add-on to stimulants. When discontinuing GXR, the dose must be gradually tapered to prevent rebound hypertension. The total daily dose should be tapered in decrements of no more than 1 mg every 3–7 days. Patients should be educated about the need for gradual dose discontinuation and advised of the possible side effects of abruptly stopping GXR.

Similar to Clon-ER, the most frequent side effects of GXR are sedation, somnolence, and fatigue. These are the GXR side effects that most frequently lead to medication discontinuation. Syncope can occur with GXR; syncopal events possibly related to the drug were reported in two 24-month, long-term, open-label GXR trials (Biederman, Melmed, et al., 2008b; Sallee, McGough, et al., 2009). No unique adverse events suggestive of drug–drug interaction are reported for adjunctive use of add-on GXR with stimulants compared with those reported with either drug administered as monotherapy (Wilens et al., 2012). Similar patient cautions related to exercise events, prior personal cardiac history, and dehydration are as relevant for GXR as for Clon-ER (noted earlier).

NON-FDA-APPROVED PHARMACOTHERAPIES FOR CHILDHOOD ADHD

The evidence-base for non-FDA-approved pediatric ADHD pharmacotherapies is much weaker than that for the FDA-approved medications discussed earlier and limits enthusiasm for their clinical use. However, modafinil, tricyclic antidepressants, bupropion, clonidine, and guanfacine are occasionally used for ADHD children and adolescents with an inadequate response or difficulties tolerating the FDA-approved ADHD medications, or for patients with ADHD and selected comorbid conditions.

Modafinil

Modafinil (trade name: Provigil) and armodafinil (trade name: Nuvigil) are nonamphetamine wake-promoting agents indicated in patients with excessive daytime sleepiness associated with narcolepsy, obstructive sleep apnea–hypopnea syndrome, and shift work sleep disorder. Armodafinil is the R- and longer acting isomer of racemic modafinil. The precise mechanisms by which modafinil promotes wakefulness are not known. It appears to act in specific areas of the hypothalamus involved in maintaining typical wakefulness and sleep. Modafinil also appears to inhibit the actions of the dopamine transporter, thus facilitating dopamine neurotransmission in the CNS (Schwartz, Roth, & Drake, 2010). Modafinil does not activate areas in the CNS that mediate reward and it has a low potential for drug abuse (Myrick, Malcolm, Taylor, & LaRowe, 2004).

Two controlled studies support the use of modafinil and its once-daily formulation in the treatment of pediatric ADHD (Biederman et al., 2006; Swanson et al., 2006). These studies used doses between 340 and 425 mg/day. ADHD symptoms improved in a dose-dependent manner. Modafinil doses are commonly split, with one-half the dose given in the morning and the other half at noon. Common reported side effects included insomnia, headache, and decreased appetite. However, modafinil and its modified formulations were not FDA-approved for use in pediatric ADHD because of concerns about a rare and infrequent but poten-

tially serious rash, which characteristically is similar to Stevens–Johnson syndrome. Thus, modafinil should be used cautiously in patients with ADHD, with clinical monitoring for the development of rash. Patients with ADHD and serious substance use disorders who do not respond to atomoxetine, Clon-ER, or GXR might benefit from an off-label and carefully monitored clinical trial of modafinil.

Tricyclic Antidepressants

The most studied tricyclic antidepressants (TCAs) for childhood ADHD are imipramine and desipramine. Controlled studies generally report improvement in ADHD symptoms with imipramine (3–5 mg/kg/day), desipramine (3–5 mg/kg/day), and nortriptyline (2 mg/kg/day) administered in two or three divided daily doses (Biederman & Spencer, 2008). However, the potential benefits of TCAs for pediatric ADHD are overshadowed by safety concerns, including cardiovascular risks (Vaughan & Kratochvil, 2012). TCAs are not considered a first-line ADHD treatment. Although they are used infrequently, studies of TCAs report robust response rates in patients with ADHD and comorbid anxiety, depression, and tic disorders; therefore, they might work for patients with ADHD and these comorbid disorders (Biederman & Spencer, 2008). TCAs are not generally abused, so they may be of some use in the treatment of severe ADHD with comorbid substance use disorders. If used off-label for the treatment of ADHD in children and adolescents with selected comorbid conditions, careful clinical monitoring of potential adverse events is recommended. TCAs are metabolized by the cytochrome P450 2D6 enzyme oxidative enzyme system. Genetic polymorphisms of cytochrome P450 exist, such that 5–10% of individuals of European ancestry and up to 1% of Japanese individuals are poor metabolizers of TCAs. Such individuals may experience high plasma concentration levels on standard dosing, which may increase vulnerability to TCA-mediated adverse cardiovascular side effects. Predose and full-dose electrocardiographic (EKG) monitoring, therapeutic drug-level monitoring, and blood pressure monitoring are recommended.

Bupropion

Bupropion (trade name: Wellbutrin), an antidepressant in the aminoketone class, appears to have effects on dopamine and norepinephrine neurotransmission, although its precise mechanism of action is unknown. Bupropion's use in pediatric ADHD is supported by a single controlled multisite study (Conners et al., 1996). In this study, bupropion was given twice daily (between 150 mg to 250 mg/day). Common side effects include irritability, insomnia, and diminished appetite. Because of its effects on CNS dopamine, bupropion can exacerbate tic symptoms and Tourette disorder. High doses and high peak plasma concentrations are associated with increased risk of seizure, and bupropion should not be administered to patients at risk for seizure disorder or electrolyte imbalance secondary to eating disorders. The immediate release formulation must be taken two to three times daily. A twice-daily formulation (SR, sustained release) and a once-daily formulation (XL, extended release) are available.

Clonidine

Clonidine (trade name: Catapres) has been FDA-approved for use in adult hypertension since the early 1970s. Clonidine stimulates presynaptic alpha$_2$-adrenergic autoreceptors in the brain stem locus ceruleus, resulting in a reduction in sympathetic outflow from the CNS. Decrease in plasma norepinephrine is directly related to clonidine's hypotensive action. In the prefrontal cortex, clonidine influences postsynaptic alpha$_2$-receptors. Three subtypes of alpha$_2$-adrenergic receptors have been cloned in humans: alpha$_{2A}$, alpha$_{2B}$, and alpha$_{2C}$. Clonidine has affinity for all three postsynaptic alpha$_2$-receptors. Postsynaptic alpha$_{2A}$-receptors mediate norepinephrine neurotransmission in the prefrontal cortex to enhance inhibition over lower CNS structures and enhance working memory under distracting conditions (Arnsten & Rubia, 2012). The principal uses of clonidine in child psychiatry are for the off-label treatment of tic symptoms and Tourette disorder, ADHD, aggression and oppositional disorders associated with ADHD, and sleep disturbances associated with ADHD. Use in pediatric ADHD is supported by a moderate effect size found in a meta-analysis of 39 studies (Connor, Fletcher, & Swanson, 1999). Efficacy for ADHD and tic symptoms in children with both disorders is supported by a controlled study (Tourette's Syndrome Study Group, 2002).

Oral clonidine is a tablet with a short pharmacokinetic half-life, ranging from 5 hours in children to 8.5 hours in adults. Because of its short half-life, oral

clonidine must be administered in three or four divided daily doses. Treatment is generally initiated with a full or half tablet of the lowest available dose (0.1 mg) and given at bedtime because sedation is a prominent side effect. Clonidine is titrated upwards by 0.05 mg to 0.1 mg every 3–7 days depending on individual tolerability within a total daily dose range of 0.05 mg to 0.4 mg/day. Common side effects include a small but consistent decrement in pulse and blood pressure, sedation, somnolence, fatigue, dry mouth, and depression. Abrupt cessation of clonidine is not recommended because of risk for rebound hypertension. Clonidine needs to be slowly tapered down by 0.1 mg every 3–5 days upon discontinuation. Monitoring of pulse and blood pressure is recommended while on clonidine. Children with a personal history of cardiovascular disease or symptoms, depression, or exercise-related cardiovascular symptoms should not be given clonidine unless cleared by a pediatric cardiologist. Clonidine is available as a transdermal patch, FDA-approved for adult hypertension. Use of the patch system for pediatric ADHD treatment has largely been supplanted by the availability of Clon-ER (Kapvay).

Guanfacine

Guanfacine (trade name: Tenex) is an orally administered antihypertensive agent. Guanfacine is an alpha-agonist with more selective action at the postsynaptic alpha$_{2A}$-receptor, resulting in a longer duration of effect, less sedation, less hypotension, and a profile of clinical benefits similar to clonidine. Effectiveness in ADHD is suggested by open-label studies (Chappell et al., 1995) and one small, controlled trial of guanfacine in the treatment of children with tic disorders and ADHD (Scahill et al., 2001). The dose range for guanfacine is 0.5 mg/day to 4.0 mg/day. Therapeutic administration at maximum dose is most often divided into two (for adolescents) to four (for young children) daily dosings. Guanfacine is initiated at 0.5 mg at bedtime and titrated upward by 0.5 to 1.0 mg every 3–7 days. Common side effects include sedation, somnolence, fatigue, irritability, and depression. Children with preexisting cardiovascular symptoms or disease should be medically cleared prior to use. Monitoring of blood pressure and pulse is recommended because guanfacine is a hypotensive drug. Guanfacine use in children with ADHD has largely been replaced by the use of GXR (see Intuniv, previously discussed).

CLINICAL USE OF MEDICATIONS FOR CHILDHOOD ADHD

Treatment should always be preceded by a careful evaluation of the individual with ADHD within the context of his or her family. Evaluation should include attention to psychiatric, physical health, social, cognitive, and educational/occupational aspects of the child's development (Pliszka, 2007). Parental and child attitudes about pharmacotherapy must be explored. Some parents may be overly optimistic about medication effects on their child with ADHD, whereas others may simply not be supportive of drug therapy for ADHD. With older children and adolescents, it is important to discuss the use of medication and explain its rationale in the treatment of ADHD. In evaluating the family of a child with ADHD, a clinician must pay attention to the possibility that a parent or sibling also has ADHD. ADHD is a highly heritable disorder (heritability rate is approximately 70%), and not infrequently, first-degree biological relatives of the identified patient may have ADHD themselves (Faraone & Mick, 2010). The presence of a parent or sibling with ADHD must be taken into account during treatment planning.

A recent screening physical examination should be available to rule out medical illness or sensory impairments (e.g., hearing loss) that may contribute to symptoms or influence treatment decision making (American Academy of Child and Adolescent Psychiatry, 2009). Special attention should be paid to issues of ADHD and comorbid learning disorders, which may also contribute to educational or occupational underperformance. Comorbid learning disabilities are important to identify, because they do not respond to stimulant medications and require supplemental educational remediation. It is important to evaluate possible comorbid psychiatric disorders that frequently occur in children with ADHD and may influence symptom presentation, treatment response, and prognosis. In children with ADHD, psychiatric comorbidity may include CD and ODD, anxiety disorders, depression, or bipolar disorder. In adolescents with ADHD, additional attention should be paid to possible alcohol, tobacco, and other substance use/misuse, together with other risk-taking behaviors (Pliszka, 2003).

Medication treatment should always be part of an overall psychoeducational treatment plan for the child or adolescent with ADHD (Pliszka, 2007). Not all children and adolescents with ADHD require medication.

For example, children with mild ADHD symptoms and only minimal impairment in daily functioning may respond to educational supports, psychoeducation about the disorder, and evidence-based psychotherapy approaches, without the use of adjunctive medications.

The large number of medications available for ADHD treatment come in varied formulations, including solution, beaded capsules, tablets, and adhesive patches. Stimulant medications are formulated for different durations of action including immediate-release, intermediate-, and long-acting preparations. Nonstimulant ADHD medications are formulated for once- or twice-daily administration and generally have a longer duration of action than stimulant compounds. With so many available options, it is important that the child and parents be actively involved in the decision and that individual choices about medication selection be made for each patient.

In making the choice of which medication is optimal for a child or adolescent with ADHD, the clinician should consider several factors. First, stimulant medications have larger effect sizes than nonstimulant medications (Faraone, 2009). Second, for children who have difficulty swallowing medication, MPH is available as an adhesive patch system and as a liquid formulation in both immediate-release and long-acting formulations. Furthermore, both long-acting AMP and MPH formulations are available as beaded capsules that may be split and the beads sprinkled on yogurt or applesauce. Third, if substance misuse is a concern, atomoxetine, GXR, and Clon-XR have minimal abuse potential. Fourth, if tics are present along with ADHD, atomoxetine, GXR, and Clon-XR may be useful in treating ADHD without exacerbating tics. Fifth, if a strong family preference is stated for treatment with a nonstimulant, these same three medications have evidence for efficacy in ADHD. Finally, the choice of medication preparation will depend on the profile of action required over time to achieve agreed-upon ADHD treatment goals.

A systematic method of initiating and titrating stimulant medication to an effective dose should be used by the prescriber. Use of a validated ADHD symptom severity scale is recommended, in addition to clinical interview data, in ascertaining treatment outcomes. Monitoring of ADHD medication treatment should include regularly scheduled doctor appointments in order for the prescriber to assess effectiveness, safety, and tolerability of the medication over time. Weight, height, and body mass index should be obtained and plotted on a standardized growth curve every 3–5 months while in treatment. If the medication is ineffective or not tolerated, reevaluation and development of a new treatment plan is indicated. Consistent communication with the child's primary care provider is recommended.

The goals of medication treatment for youth with ADHD are becoming increasingly well defined and standardized. Given the robust effect size of stimulants and the moderate effect size of adrenergic agents on the symptoms of ADHD, a minimum goal for the clinician to achieve in the treatment of ADHD is at least a 50% reduction over baseline in ADHD symptom severity, with a concomitant improvement in daily functioning (Rostain, Jensen, Connor, Miesle, & Faraone, in press). A more ambitious goal is "ADHD syndromal remission," defined as loss of diagnostic criteria for meeting the ADHD diagnosis while in treatment (Biederman, Mick, & Faraone, 2000). The prescribing clinician should have in place a systematic and quantitative method for measuring outcome in ADHD treatment in order to ascertain whether treatment goals are achieved.

Longitudinal epidemiological data indicate that the prevalence of ADHD diminishes over development from childhood to adulthood. Between 3 and 8% of school-age children meet diagnostic criteria for ADHD, and prevalence diminishes in adulthood to between 4 and 5% (Angold et al., 2000; Kessler et al., 2006). That some children with ADHD appear to outgrow the diagnosis has led to the recommendation that clinicians periodically reevaluate the need for ongoing pharmacotherapy (Pliszka, 2007). A convenient time to accomplish this may be at the end of each school year and the start of summer vacation.

SUMMARY

ADHD is a prevalent and heterogeneous neurodevelopmental disorder with a strong neurobiological etiology. Careful evaluation, treatment planning, and clinical follow-up remain the mainstays of treatment. While stimulant medication remains the first-line treatment for youth with ADHD who require pharmacotherapy, nonstimulant medications affecting noradrenergic neurotransmission provide an important and evidence-based alternative. The wide variety of available formulations facilitates a personalized approach to medication choice. A growing body of research, including the MTA and PATS studies, supports the efficacy and safety of medications for pediatric ADHD.

KEY CLINICAL POINTS

- ✓ A variety of medications, as well as drug-delivery systems, now exist for the management of childhood adolescent, and adult ADHD.
- ✓ The stimulants (MPH, AMP) have been the longest used and studied medical interventions for ADHD; hundreds of studies attest to their safety and efficacy.
- ✓ Nonstimulant medications for ADHD have also been developed, tested, and approved by the FDA during the past 15 years, including atomoxetine, GXR, and Clon-ER.
- ✓ Besides the original immediate-release tablets, new technologies developed over the past 15 years as delivery systems have broadened the convenient use of the medications (liquids) and provide longer medication effects across the day. These include liquid preparations, extended-release osmotic pumps, extended-release pellet technologies, skin patches, and prodrugs.
- ✓ The availability of different delivery systems permits clinicians to tailor better treatment to the individual child than was previously the case.
- ✓ Long-acting, extended-release medications are now the standard of care in the United States as first-line (and first-choice) interventions.
- ✓ While nearly than half of children with ADHD in the United States are eventually diagnosed and treated with medications, more than half go undiagnosed and untreated, contradicting frequent claims in the trade media that childhood ADHD is overdiagnosed and overtreated.
- ✓ Between 65 and 75% of children with ADHD respond to any single medication; trying a second medication for those who have not initially responded may increase positive response rates to 80–90%.
- ✓ Medications should be used as part of a larger, comprehensive psychoeducational plan of interventions for children with ADHD (parent education, parent training, school consultation, etc.).

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CHAPTER 28

Combined Treatments for ADHD

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Our aim in this chapter is to provide interesting and practically meaningful updates to our chapter in the previous edition of this volume on combined treatments for attention-deficit/hyperactivity disorder (ADHD), in which we reviewed the extant literature and concluded that combined pharmacological (primarily stimulant medication) and behavioral treatments provided by parents and teachers are superior *in terms of efficacy* to either of those interventions in isolation (Smith, Barkley, & Shapiro, 2006). However, we expressed reservations about combined treatment owing to limitations in the research literature and questions about the acceptability and sustainability of the interventions tested. Fortunately, there have been major advances in research on combined treatments, and updates in this chapter have substantial implications for parents, teachers, and treatment providers working with children and adolescents with ADHD.

We begin by describing additional evidence-based treatments for ADHD that should be considered as potential elements of multimodal interventions that can be used in combination with other evidence-based treatments. Next, we provide an update on research from the National Institute of Mental Health (NIMH) Multimodal Treatment of ADHD Study (MTA), which is probably the best known, most controversial, and most influential study on combining pharmacological and psychosocial treatments (Swanson et al., 2008). Then we summarize some meta-analytic reviews on combined pharmacological and behavioral treatments, and compare these review findings with the MTA findings. Taken together, these studies have important implications for the range of outcomes positively impacted by combining stimulant and behavioral treatment for ADHD.

The developments in the research literature are enlightening; however, the novel and most clinically relevant contribution of this chapter is recent documentation from a high-quality study showing that there is an interaction between stimulant medication and behavioral treatments. This interaction created by combining stimulant medication and behavior modification has major implications for practice that have not yet been implemented widely. We propose that the proper coordination and strategic changes of dose across the day, week, and year can have large impacts on the efficiency, acceptability, and sustainability of treatment for ADHD. Given the chronic nature of ADHD, sustainability of treatment over time is critically important. The dosing considerations proposed in this chapter

could make treatment more efficient and tolerable, and therefore more sustainable, as children with ADHD grow into adolescents with ADHD.

The chapter begins with a brief statement of the need to improve treatments for ADHD, examples of efforts to improve treatments, and the hypothesis that combining treatments can lead to robust improvements in therapeutic outcomes relative to monotherapy for ADHD. The paramount question to be addressed by this chapter and future research is for whom and when is combining treatments safe, feasible, and effective? There are different ways of combining treatments, and exploring these different approaches could be the key to unlocking the potential of combined treatments.

THE NEED FOR BETTER TREATMENTS FOR ADHD

Although there are many controversies surrounding treatments for children with ADHD, there is widespread consensus that no single treatment is universally and completely effective for treating ADHD. This point is so firmly established in the literature that it needs no further elaboration here. The interested reader is referred to thoughtful treatments of this topic provided elsewhere (Chronis, Jones, & Raggi, 2006; Pelham, Wheeler, & Chronis, 1998; see all other chapters in Part III).

Given the limitations of each of the evidence-based treatments for ADHD, we need better treatments for this prevalent and highly impairing disorder. One approach is to try to improve monomodal treatments. For example, the MTA showed that intensive medication management was superior to community care in which most (55% at 14 months) of the children were receiving stimulant medication (Swanson et al., 2008). However, this superiority was short-lived and faded when intensive treatment was withdrawn deliberately as part of the MTA study design. Moreover, even when treatment was at its peak effectiveness, the positive response rate was only 67%, and only a small minority of children in the study were "normalized" by the most intensive drug treatment protocol studied to date. Thus, this monotherapy approach to improving treatment (e.g., strengthening drug treatment) worked somewhat but did leave considerable room for improvement.

One of the aims of the MTA was to see whether combining treatments across modalities could lead to substantial improvements in response to treatment for ADHD. Combining medication and behavioral treatments is a logical and widely recommended approach forwarded by many reviewers and experts on the treatment of ADHD (see all chapters in Parts III and IV). As we presented in the previous edition of this volume, based on the MTA data, a good case can be made for the superiority of combining intensive medication management treatment with intensive psychosocial treatments for children with ADHD (Smith et al., 2006). However, two major caveats must be stated. First, the intensity of the combined treatment approaches taken in the historic MTA are unlikely to be feasibly delivered in real-world community settings. Second, the extent to which combined treatments are superior to medication alone is a controversial issue, especially given the relatively high cost and limited availability of many psychosocial interventions.

Since we wrote the previous chapter, there have been some important developments in the research literature that are pertinent to combined treatments for ADHD. First and foremost is the addition of some new treatments to the list of evidence-based treatments for ADHD. We cannot emphasize strongly enough that it is hardly worth studying combinations of treatments in which one of the components is not firmly established as an evidence-based treatment in its own right. Thus, we review the treatments that appear to have robust empirical support, then limit our review of combined treatments to those from the evidence-based list. Of note, in the previous edition of this volume, we reviewed some combinations of treatments, such as social skills or anger management, that were not considered evidence-based therapies for ADHD at the time (Pelham et al., 1998). Such therapies as traditionally delivered have still not reached the level of evidence-based treatment (Fabiano et al., 2009; see Chapter 23) and are not reviewed as combined treatments in this chapter. Second, compared to a nearly a decade ago, there has been growth in the number of studies of combined treatments, with enough studies to support some meta-analytic reviews, which are reviewed later in this chapter.

Although several studies have advanced the knowledge base on combined treatments, in our opinion, the foremost advance in combined treatment research is a landmark study of the various strengths of stimulant medications and behavioral treatments (Fabiano et al., 2009). This study and previous case studies of varying doses of behavioral and stimulant treatment in combination with each other suggest that there are practical-

ly meaningful interactions between these treatments. These findings have some potentially paradigm-changing implications for research and practice that were not addressed in the previous chapter.

REASONS FOR COMBINING TREATMENTS FOR ADHD

To begin, we define *combined treatment approaches* as those that couple psychosocial interventions with pharmacological interventions. *Multimodal approaches* are defined here as those that combine multiple psychosocial approaches, such as the Summer Treatment Program, which provides intensive group behavioral support, peer interventions, academic interventions, and parent training (Pelham & Hoza, 1996). There is also the notion of *polypharmacy*, in which multiple medications are provided during the course of the day. Studies such as the MTA compared multimodal psychosocial treatments separately and in combination with pharmacological treatment that was usually, but not always, limited to one medication at a time. However, pharmacotherapy is not simply giving a pill, so even a monomedication therapy is a treatment package that includes a variety of factors, such as patient education, skillful engagement, social support, and positive expectations. In summary, our chapter carves nature at the joint between evidence-based pharmacological and nonpharmacological treatments even though these treatments are complex and should be regarded as treatment packages that combine a variety of effective ingredients—any one of which could interact with another treatment element—and make it impossible to separate treatments into neatly defined monotherapies.

In addition to carefully considering the underlying nature of so-called "monotherapy" treatment approaches, we must also consider why combining treatments is important or necessary. The major reasons for using combined treatments, as clearly articulated by many researchers (Chronis et al., 2006; Pelham, 1999; Pelham et al., 1998) rest with the following well-described and often-noted limitations of monotherapies:

- *Limitations of coverage.* Stimulant medication, the primary pharmacological approach for treating ADHD symptoms, cannot cover a full 24-hour day. This is particularly true given that side effects, such as insomnia and appetite suppression, require breaks in medication coverage over the course of a full day. Early morning and evenings are two high-risk times for potential misbehavior, and these are the times least likely to be impacted by active stimulant treatment. Likewise, parents or teachers who may be effective in managing ADHD-related problems cannot be present throughout the day, so there are gaps in psychosocial treatment. Strategic timing of psychosocial and pharmacological treatments could fill the gaps inherent in monotherapies.

- *Treatment effects are temporary.* The efficacy of treatments for ADHD, most notably stimulant medication and behavioral treatments, can be verified using reversal designs that alternate days (or times) when treatment is provided with days (or times) it is not provided. This shows that treatments not only work but also they have little or no carryover effects from one day to the next. Currently available treatments do not cure ADHD, are needed at the point of performance, and must be continually provided in order to have impact. Again, strategic timing of psychosocial and pharmacological treatments could fill the gaps inherent to monotherapies.

- *The scope of monotherapy is limited.* Medication alone does not teach youth the skills necessary for more competent functioning. Likewise, behavioral treatment may not encourage sufficiently focused attention to benefit from instruction or interventions. One of the appealing concepts of combined treatments is that medication may very well set the stage for effective learning to occur in the context of behavioral supports and skills training. Thus, there is the potential of simultaneous provision of complementary effects of pharmacological and psychosocial treatments.

- *There are a large number of nonresponders to any particular treatment for ADHD.* A sizable proportion of youth (by most estimates, up to one-third) do not respond to stimulant or first-line medication. The rate of nonresponse to psychosocial treatment is not as well established, but is certainly unacceptably high. One of the objectives of combined treatments is to increase the response rate relative to monotherapies, and there is research to support this objective (reviewed later in this chapter).

- *Normalization is rare with any monomodal treatment.* Even among those who improve in response to treatment for ADHD, they rarely recover fully. Even with highly intensive treatment, symptoms and/or impairments remain in the dysfunctional range for the

majority of persons with ADHD and have both immediate and long-term consequences for adjustment. Thus, treatments that increase normalization rates are highly desirable, and there are some indications that combined treatments can improve normalization rates.

- *Acceptability and feasibility issues.* For a variety of reasons, some scientifically based and others not, there are families that question the acceptability of their children taking medication, and they either refuse medication or overly limit its use. Although some say that they prefer nonpharmacological treatments, most parents obtain nonpharmacological treatments at a much lower rate than medication. Parents face multiple barriers with the availability, cost, and demands of psychosocial treatments. Nevertheless, a promising finding is that combined treatments get higher consumer satisfaction ratings. We explore some potential reasons for this finding later in this chapter.

- *Youth with ADHD have a tendency to have problems across multiple domains of functioning that are differentially impacted by monotherapies.* Examples include evidence of a strong stimulant treatment impact on symptoms of ADHD and related disorders, such as oppositional defiant disorder (ODD), yet little impact on academic or peer functioning. Psychosocial treatments have more impact on family relationships and academic functioning but less relative impact on actual symptoms of ADHD. Thus, combining treatments can address a wider range of pertinent outcomes than monotherapy.

To summarize, there are at least seven good reasons for combining pharmacological and nonpharmacological treatments. Moreover, there is preliminary empirical support for certain combinations of treatments. Overall, the goal is to provide the right kind of treatment at the point of performance (the place in the natural setting where the symptoms are distressing and/or the impairment exists; see Chapter 16). The right kind of treatment has high response and normalization rates, addresses the full range of symptoms or impairment, and is acceptable and feasible to deliver. In some cases, the right treatment might require the simultaneous active ingredients of psychosocial and pharmacological treatments. In other cases, delivering the interventions sequentially may provide the appropriate support. Importantly, these statements about combing treatments are limited to treatments that have been shown to work through rigorous and replicated clinical studies.

EVIDENCE-BASED TREATMENTS FOR ADHD

The list of evidence-based treatments for ADHD continues to expand. These interventions can be categorized primarily as either biological or behavioral. The wealth of research on treatments for this disorder supports the ability to use meta-analytic approaches to combine findings from multiple studies. "Meta-analysis" is a quantitative approach that combines data across a group of similar studies in order to provide better estimates of the impact of a given intervention than might be possible with a single study. Meta-analysis can only be conducted when a number of studies use interventions that are judged to be similar enough to warrant pooling results in a manner that should improve the confidence with which we draw conclusions about an entire pool of studies. For example, because a sizable number of studies is needed for this approach, studies are likely to come from multiple researchers, which may reduce bias.

An interesting new development is a resurgence of attention to the impact of diet on ADHD symptoms; the number of separate studies has grown to the point that meta-analyses are appearing in the research literature. Examples include recent meta-analytic reviews of the impact of synthetic food color additives, restriction diets, and omega-3 fatty acid supplementation on behavior (Nigg, Lewis, Edinger, & Falk, 2012; Sonuga-Barke et al., 2013).

Because of the power of this approach, we first briefly consider recent meta-analyses of psychosocial and behavioral treatments, medication treatments, and other interventions (i.e., dietary), then turn our attention to recent meta-analyses of combined treatment approaches.

Psychosocial, Behavioral, and Nonpharmacological Interventions

It has been widely accepted for decades that behavioral parent training and behavioral classroom management are robust, evidence-based treatments for ADHD (Pelham et al., 1998; see Chapters 21 and 24). Several behavioral interventions meet stringent criteria for efficacy, effectiveness, and readiness for dissemination (Flay et al., 2005). Behavioral interventions cover the settings in which difficulties arise, namely, at home, at school, and in community program settings. Additionally, research in specialty settings supports the

therapeutic benefits of such treatments, for example, intensive behavioral interventions delivered in summer program settings (Pelham & Fabiano, 2008). Given known challenges for youth with ADHD across multiple settings, it is necessary to have interventions developed and tested specifically where impairments are likely to be seen.

In a thoughtful and thorough meta-analysis of behavioral interventions for youth with ADHD, Fabiano and colleagues (2009) included 174 studies of behavioral interventions including between-group, pre–post, within-subject, and single subject designs. Fabiano and colleagues argue that inclusion of this range of studies afforded the opportunity to quantitatively summarize and evaluate the impact of behavioral interventions; disregarding within- and single-subject designs would exclude a significant proportion of the extant behavioral treatment literature. Indeed, exclusion of within-subject and single-subject designs has resulted in some reviews reaching conclusions that are at odds with widespread research consensus about best practices for treating ADHD. On the other hand, in the comprehensive review by Fabiano and colleagues, effect sizes indicate a very strong impact of behavioral treatments, providing clear and convincing evidence that behavioral interventions implemented in home, school, and peer settings can work to improve functioning of youth with ADHD. Inclusion of studies with a wide range of participants (i.e., females and nonwhites), in both school and clinic settings, increases confidence that these results are generalizable to the sizable group of youth with ADHD (Fabiano et al., 2009).

In contrast, in a controversial review, Sonuga-Barke and colleagues (2013) conducted a meta-analysis of a wide range of treatments, including dietary and broadly categorized psychological treatments. However, this meta-analysis included only group randomized studies (excluding studies using single-subject designs) and examined studies using a restricted set of outcome measures. In the Sonuga-Barke and colleagues review, dietary approaches included elimination diets, artificial food color exclusions, and free fatty acid supplementation; psychological treatments included cognitive training, neurofeedback, and behavioral interventions. A total of 54 studies were included, and the authors concluded that both dietary and psychological treatment approaches resulted in effect sizes suggesting a meaningful impact on ADHD assessment measures. However, when results were reanalyzed using more strict criteria of "best probably blinded assessment," a small but significant effect size remained for free fatty acid supplementation and a moderate and significant effect size remained for artificial food color exclusion. Elimination diets and psychological intervention effects, when held to this standard, disappeared.

The idiosyncratic selection criteria of studies in the Sonuga-Barke and colleagues (2013) meta-analysis help to explain why their results are disparate from those of several other reviews. Nevertheless, as indicated by Sonuga-Barke and colleagues, dietary interventions are starting to gain some support for efficacy. In a meta-analysis of the impact of dietary approaches on symptoms of ADHD, Nigg and colleagues (2012) found evidence of a statistically significant impact of restriction diets and synthetic food color additives on parent ratings of ADHD symptoms. The effect size for restriction diets was moderate, while the effect size for food colors was small. Importantly, the synthetic food color additive effect was largely found outside of the United States in countries using non-FDA-approved synthetic food colors (Nigg et al., 2012). The impact is big enough that the long-dormant issues in the U.S. research literature on the impact of dietary restrictions on ADHD symptoms may be reawakened after a decades-long hiatus. Research on the benefits of omega-3 fatty acid supplementations seems promising, but effect sizes of improvements in a meta-analysis have been small (Bloch & Qawasmi, 2011). Perhaps this treatment should be looked at further in combination with other evidence-based treatments for ADHD.

Pharmacological Interventions

Two recent meta-analyses have examined the impact of pharmacological interventions for youth with ADHD. In one meta-analysis of 23 double-blind, placebo-controlled studies of stimulant medication for youth and adolescents with ADHD, Faraone and Buitelaar (2010) compared methylphenidate and amphetamine on core symptoms of ADHD (hyperactivity, impulsivity, and inattention). They concluded that effect sizes for amphetamine were slightly higher than those for methylphenidate. However, the mean differences in effect sizes were small (i.e., less than 0.3 and very close to being within the 95% confidence interval). It may also be noteworthy that this review was sponsored by makers of amphetamine treatments for ADHD (Faraone & Buitelaar, 2010).

A meta-analysis of the nonstimulant atomoxetine likewise found significant benefits from the drug in the

reduction of ADHD symptoms (Cheng, Chen, Ko, & Ng, 2007). Significant benefits were also found relative to people with ADHD and comorbid ODD symptoms and possibly anxiety. A later meta-analysis (Hanwella, Senanayake, & Silva, 2011) comparing atomoxetine and methylphenidate found them to be equally efficacious in the reduction of ADHD symptoms and in treatment acceptability to families. However, oral osmotic release (OROS) methylphenidate appeared to be somewhat more effective than atomoxetine in symptom reduction.

A second meta-analysis focused exclusively on the question of medication treatment impact on school behavior and academic achievement. Prasad and colleagues (2013) analyzed the results of 43 randomized controlled trials of medication (methyphenidate, amphetamines, and atomoxetine) on the academic achievement and classroom behavior related to academics of children ages 4–6. An overall significant impact was found for medication treatment with methylphenidate and amphetamine formulations, but not atomoxetine, relative to seatwork completion and increased time on task. Moreover, no impact was found for any of these drugs on the accuracy of performance. Thus, stimulant medications can make small but measurable improvements (approximately 15%) in academic achievement, including grades, and classroom behavior. However, quality of classroom work in these studies was not impacted.

META-ANALYSES OF MONOTHERAPIES

Meta-analytic studies have indicated that there is robust support for the efficacy of behavior modification, stimulant medication, and nonstimulant medication (atomoxetine) for the treatment of ADHD. This has been known for a long time, but it is worth noting that studies and support for delivering these interventions is plentiful enough to say that they are ready for widespread dissemination. Other treatments, such as fatty acid supplementation, seem to be emerging as possible viable treatments for ADHD, but this research should still be regarded as preliminary for a variety of reasons, such as uncertainty about the optimal dose or type of fatty acid supplementation. Some other treatments that have some popular appeal, such as cognitive training, electroencephalographic (EEG) biofeedback, and self-control or cognitive-behavioral training for children, do not have robust enough empirical support to be considered viable treatments for ADHD (Fabiano et al., 2009). Therefore, we are restricting our focus on combined treatments to the two major classes of evidence-based approaches: behavioral and pharmacological treatments.

EARLY RESEARCH ON COMBINED TREATMENTS

Some early research studies examined the utility of combining psychosocial and pharmacological treatment packages, with interesting results. In some of these studies the combination of contingency management training of parents or teachers and stimulant drug therapies was generally little better than either treatment alone for the management of ADHD symptoms (Firestone, Kelly, Goodman, & Davey, 1981; Gadow, 1985; Pollard, Ward, & Barkley, 1983; Wolraich, Drummond, Salomon, O'Brien, & Sivage, 1978). Several other studies found impressive results for classroom behavior management methods (Carlson, Pelham, Milich, & Dixon, 1992; DuPaul & Eckert, 1997; Pelham et al., 1988) but that the addition of medication provided further improvements beyond those achieved by behavior management alone. Some authors suggested that the combination might have resulted in the need for less intense behavioral interventions or lower doses of medication than might be the case if either intervention were used alone. When there was an advantage to behavioral interventions, it appeared to be related to functioning rather than to symptom relief, such as reliably increasing rates of academic productivity and accuracy (see DuPaul & Stoner, 2003).

Most of the studies reviewed in the previous paragraph were short-term studies. In contrast, Satterfield, Satterfield, and Cantwell (1980) attempted to evaluate the effects of individualized multimodality intervention provided over extensive time periods (up to several years) on the outcome of boys with ADHD. Interventions included medication, behavioral parent training, individual counseling, special education, family therapy, and other programs as needed by particular individuals. Results suggested that such an individualized program of combined treatments continued over longer time intervals could produce improvements in social adjustment at home and school, as well as in rates of antisocial behavior, substance abuse, and academic achievement. These results seem to have been sustained across at least a 3-year follow-up period (Sat-

terfield, Cantwell, & Satterfield, 1979; Satterfield, Satterfield, & Cantwell, 1980; Satterfield, Satterfield, & Cantwell, 1981, 1987). Although this research suggests great promise for the possible efficacy of multimodality treatment extended over years for children with ADHD, the lack of random assignment and more adequate control procedures in the Satterfield and colleagues studies limits the ability to attribute improvements obtained in these studies directly to the treatments employed. And these limitations certainly preclude establishing which of the treatment components was most effective. Still, studies such as these and others (Carlson et al., 1992; Pelham et al., 1988) raised hopes that intensive multimodality treatment might be effective for ADHD if extended over long intervals of time.

THE NIMH MTA

As mentioned throughout this chapter, the NIMH collaborative MTA has been a major influence on our thinking about combined treatment (Jensen et al., 1999a, 1999b). Part of the rationale for the MTA was that even though much research has documented the short-term effectiveness of medication and behavioral interventions to treat ADHD, significant questions have remained unanswered about the long-term effects of these interventions, alone or in combination, on the multiple functional outcome areas impaired by ADHD. The ambitious and groundbreaking MTA was designed to help answer some of these major questions by randomly assigning children to four treatment groups: medication alone (MedMgt), behavior modification alone (Beh), the combination of medication and behavior modification (Comb), and community comparison (CC). In order to obtain a sufficiently large and diverse sample of youth with ADHD to begin to address these questions (N=571), a multisite study was initiated in 1992 by NIMH, along with funding from the U.S. Department of Education.

Study Design/Methodology

In order to be eligible for the study, children had to be between ages 7.0 and 9.9 years; to be in grades 1–4; to meet *Diagnostic and Statistical Manual of Mental Disorders*, fourth edition (DSM-IV) diagnostic criteria for ADHD, combined type, based on the Parent version of the Diagnostic Interview Schedule for Children (supplemented by teacher-reported symptoms, if a case was near the diagnostic threshold); and to have been living with the same caretakers for at least the previous 6 months. Youth with comorbid internalizing or externalizing psychiatric disorders were included, as long as these conditions did not require treatment incompatible with study treatments. The schools the children attended also had to express cooperation with both the treatment and assessment protocols. Other exclusionary criteria included situations that would prevent full participation in the study, such as not having a phone, intellectual and adaptive functioning in the borderline range or below, or major medical illness (for complete information on the screening and selection procedures, see Jensen et al., 1999a).

Important characteristics of the sample selected for the MTA included variables identified a priori as potential moderators of treatment: gender (20% female); prior medication status (31%); ODD or CD diagnoses (40% and 14%, respectively); DSM-III-R anxiety disorders (34% with simple phobia alone not included); and families receiving welfare, public assistance, or Supplemental Security Income (19%). It is noteworthy that the 579 children selected for the MTA represented only 13% of those initially contacting the project, 25% of those passing an initial rating scale screening, and 62% of those completing the diagnostic interview and evaluation of school cooperation.

Once selected, participants were randomly assigned to one of the four conditions noted previously. Treatments were delivered over a 14-month period; comprehensive assessments of functioning in multiple domains were conducted at baseline prior to randomization as well as at 3, 9, and 14 months (with the 14-month assessment constituting the treatment endpoint assessment). The MTA Cooperative Group also published results of 24-month and 36-month follow-ups, with numerous commentaries based on exploratory or secondary analysis (Swanson et al., 2008).

Behavioral treatments (in both the Beh and Comb conditions) encompassed parent, child, and school domains. Behavioral parent training was provided by experienced training consultants and based on models by Barkley (1997) and Forehand and McMahon (1981). This intervention comprised 27 group sessions and eight individual sessions. Child behavioral treatment consisted of intensive summer treatment (based on the Pelham Summer Treatment Program [STP] model), as well as school consultation services (similar to those in the University of California at Irvine/Orange County Department of Education [UCI/OCDE] model). The

MTA's version of the STP was an intensive 8-week, 9-hour a day program; study training consultants supervised staff members working with the children and continued to provide parent interventions during the summer. The same training consultants provided school consultation services (10–16 sessions of teacher consultation and establishment of a Daily Report Card), and the staff members working with the children in the STP worked in the schools in the fall as paraprofessional aides (12 weeks at half-time under supervision of the training consultants and the children's teachers). Families attended an average of 77.8% of parent training sessions, 36.2 of 40 possible STP days (90.2%), 10.7 teacher consultation visits out of 16 (62.5%), and 47.6 (out of 60) possible days with a classroom aide (80%). Delivery of behavioral treatments was faded over the course of treatment, so that by the endpoint assessment at 14 months, therapist contact with parents had ended or was reduced to once per month.

Like the intensive behavioral interventions, the medication treatments (in both MedMgt and Comb conditions) in the MTA were provided in a much more rigorous and intensive way than is typical in clinical practice. All medication treatment provided by the MTA included an initial 28-day double-blind, placebo-controlled titration consisting of placebo plus four different doses of methylphenidate (MPH; 5, 10, 15, and 20 mg) randomly given over the titration period. Dosing three times per day was used in the titration (and typically during treatment), in which the full dose was given in the morning and at lunch, as well as a half-dose in the midafternoon. Parent and teacher daily ratings were collected during the titration; graphs portraying the results were rated by a cross-site panel of experienced clinicians. A "best dose" was chosen, and the double blind was then broken; that dose became the initial dose for treatment. If the dose chosen was placebo, alternative medications were openly titrated until a satisfactory medication was chosen (or, in the case of a robust placebo response, the child was not medicated). Approximately 89% of youth assigned to MedMgt or Comb successfully completed titration; of these, 68.5% were assigned to initial doses of MPH averaging 30.5 mg/day, given three times per day. Of the remaining group of youth who completed titration but were not started on MPH, 26 received an unblinded titration of dextroamphetamine because of unsatisfactory MPH response, and 32 were given no medication because of a robust placebo response. Of note is that of the 289 subjects assigned to MedMgt or Comb, 17 families refused titration; another 15 subjects did not complete titration; (11 because of side effects or problems with titration); and inadequate amounts of titration data were gathered for a further four subjects (Jensen et al., 1999a).

Youth assigned to the CC condition received no intervention by the MTA staff but sought "treatment as usual" provided in the community. Referrals to non-MTA providers were made as necessary for these families; all of the youth and families returned for assessments at the same time as youth in the other three conditions of the study. Initially, it was thought that the CC group would provide a minimal- or no-treatment comparison group. However, as we describe later in this section, about two-thirds of the children in the CC group actually received medication for ADHD.

Outcomes in the MTA study were assessed with a large number of measures in multiple domains, including verbal report information (via interview and paper-and-pencil measures) by parents, teachers, and children; direct observation in the clinic and school; and computerized assessments of attention. Given the large number of measures, settings, and informants used in the study, data reduction methods were conducted to condense measures into outcome domains. The major outcome domains that have received attention in the literature are as follows: ADHD symptoms, oppositional/aggressive symptoms, social skills, internalizing symptoms, parent–child relations, parental discipline, and academic achievement.

Major Findings on ADHD Symptoms

All four MTA groups showed symptom reduction over time (Jensen et al., 1999a). In our opinion, the trends in the data favored the Comb treatment over the other three conditions, but this conclusion may depend on how one looks at the data—and the MTA data have been examined a lot of different ways in the peer-reviewed literature. When an idiographic approach that examines individual outcomes is used, there is a clear advantage for the Comb condition. Swanson and colleagues (2001) created a categorical measure of treatment outcome based on composite Swanson, Nolan, and Pelham Questionnaire–IV (SNAP-IV) ADHD and ODD symptom scores from teachers and parents. Successful treatment was identified as scoring, on average, 1 or below on a composite SNAP-IV score at the end of treatment (representing symptoms falling in the *not at all* or *just a little* range of categories at treat-

ment endpoint). Success rates for the four conditions were as follows: 68% for Comb, 56% for MedMgt, 34% for Beh, and 25% for CC. A similar, but less robust, pattern of results was observed at the 24-month follow-up. Specifically, the normalization rates (as defined earlier) were 48, 37, 32, and 28% for Comb, MedMgt, Beh, and CC, respectively (Jensen et al., 2007).

Another way to look at the MTA data is in terms of statistical significance of the group means, which is the type of analysis that has received the most attention in the published literature. When using this approach on the 14- and 24-month follow-up data, the MTA Collaborative Group reached the conclusion that treatments involving intensive medication management (i.e., MedMgt and Comb) were superior to those that did not include it (i.e., Beh and CC). Based on significance tests of means, the Beh and CC conditions were statistically equivalent. Likewise, the MedMgt and Comb groups were comparable, thus indicating no advantage of Comb relative to intensive MedMgt (Swanson et al., 2008). A few comments on these findings are warranted.

Some effects on ADHD symptoms were apparently mediated by medication effects (Swanson et al., 2008). Therefore, it is important to note that 67% of the children in the CC group were taking medication; thus, the CC group was an active treatment group rather than a no-treatment control. In other words, the group that received only behavior modification (Beh) was being compared to a group that received medication in the community. It is also important to consider the implications of some substantial differences in the doses of medication across the treatment groups. For instance, at the 14-month follow-up, the average daily dose (MPH equivalent) for Comb was 31.2 mg, while the average daily dose for MedMgt was 37.7 mg (Jensen et al., 1999a). Given that the Comb and MedMgt groups had identical medication titration procedures, the difference in dose at 14 months suggests that the intensive behavioral intervention allowed individuals to take lower doses of medication. Lower doses are a considerable therapeutic advantage because most stimulant side effects, including the mild growth suppression observed in the MTA, are dose-dependent (i.e., lower doses lessen the risk and severity of side effects).

When the group data are examined, it is tempting to conclude that the MedMgt condition was superior to CC, even though most of the participants in the CC group were medicated. Such a conclusion implies that the package of procedures in the MedMgt protocol, which includes monthly supportive contact and decisions supported by high-quality data, is superior to routine community care. Indeed, this has been one of the major messages from the MTA Cooperative Group.

Another consideration in comparing the Beh and Comb conditions with MedMgt and CC is that intensive behavioral treatments were faded by the study's endpoint (Pelham, 1999). Due to this unequal treatment activity, it is plausible that the comparison of Beh and Comb to MedMgt at the 14-month follow-up may have been biased in favor of the MedMgt. This issue has been argued on theoretical grounds (Pelham, 1999) and is consistent with the observation that the therapeutic effect size of intensive MedMgt diminished by 50% from the intensive phase to the follow-up phase (Swanson et al., 2008). At the 36-month follow-up, intensive treatment effects had faded to the point that there were no statistically significant differences between the randomly assigned groups in the MTA (Swanson et al., 2008). Effect sizes faded from about 0.6 at the 14-month follow-up (medium to large effect), 0.3 at 24 months (a small effect), and 0.1 at 36 months (a negligible effect) (Swanson et al., 2008).

It cannot be stressed strongly enough that *the fading of significant treatment effects in the MTA does not mean that the treatments were ineffective*. Rather, it shows that if provision of intensive treatment is stopped, after about 2 years the incremental benefits of intensive treatment on reducing symptoms of ADHD are not distinguishable from community care. This should not be a surprising finding given the widespread knowledge that ADHD is a chronic disorder and the main treatments (stimulant medication and behavior modification) have acute but temporary effects. Nevertheless, there were some surprising and important findings pertinent to combined treatments at the first MTA follow-up:

- At the 14-month MTA follow-up, children in the Comb group were taking 20% less medication than those in the MedMgt group. This was interpreted to mean that "compared to MedMgt, the addition of behavioral treatment in the Comb treatment contributed to the same or better results with a lower dose" (Swanson et al., 2008, p. 7).

- The relative superiority of combined treatment was highlighted by Conners and colleagues (2001), who conducted a post hoc analysis using a composite outcome measure. This was done in an effort to examine further the relative impact of the MedMgt versus

Comb conditions, which did not differ statistically due to the presence of multiple outcome measures in the primary analyses. When the composite measure was used, however, a statistically significant difference was detected: Comb outperformed MedMgt, with an effect size of 0.28 (low to moderate). In addition, use of the composite resulted in reduced effect sizes for comparisons of MedMgt versus Beh alone (0.26), and a moderate effect size of 0.35 for MedMgt versus CC. Use of the composite measure therefore places combined treatment in the lead, albeit by only about one-fourth of a standard deviation.

- When measures of other symptoms are considered, most of the trends favor the Comb condition. For instance, when the MTA Cooperative Group ordered treatments based on the number of times each group placed first compared with all others on 19 outcome measures, the results were as follows: Comb (12), MedMgt (4), Beh (2), and CC (1). The four times that MedMgt was superior were for parent ratings of symptoms of inattention and hyperactivity, and classroom observations of hyperactivity and impulsivity (Jensen et al., 1999a). Although such data appear strongly to favor combined treatment over unimodal or community interventions, this analysis does not take into account the relative importance of the outcome measures. We submit that the areas tapped, including oppositional/aggressive symptoms, internalizing symptoms, social skills, parent–child relations, and academic achievement, are critically important.

- Satisfaction scores given by parents for the Comb and Beh conditions were equal to each other and significantly better than parent satisfaction scores for the MedMgt condition (Jensen et al., 1999a). Given the emphasis placed on consumer satisfaction in terms of third-party payments, this is not a trivial matter. Indeed, the highest attrition rates were for the MedMgt condition.

- Analyses by Molina and colleagues (2013) on outcomes that became relevant in adolescence found only one significant effect using the MTA intent-to-treat analysis: There was a benefit of the intensive psychosocial treatment in preventing early substance use. This effect was evident in the comparison of the Beh and Comb treatments relative to the MedMgt and CC groups. Although this effect is not specific to Comb, it is noteworthy given that a number of uncontrolled studies supposedly show that medication treatment can prevent or reduce substance use (Swanson et al., 2008). In contrast, the relatively well-controlled MTA study found that substance use, which is a major concern for adolescents and adults with ADHD, may be prevented by the MTA psychosocial treatment.

Moderator Effects in the MTA

The terms "mediators" and "moderators" are often confused; therefore, we begin this section with a brief review (see Holmbeck, 1997, for an excellent discussion of the mediator–moderator distinction). Briefly, *moderators* include participant characteristics that could affect outcome, either positively or negatively. *Mediators* are intervening variables operating during treatment that could have an impact on outcome. Knowledge of moderators helps in making decisions about who benefits from what treatment. Knowledge of mediators can help identify causal pathways from intervention to outcomes. The MTA Cooperative Group (Swanson et al., 2008) has been careful to note that the mediator- and moderator-defined subgroup analyses are exploratory because they are affected by sample size/power limitations and also suffer from the effects of repeated analyses.

In the MTA the initial set of moderators was selected a priori and included gender, prior medication status, ODD or CD diagnoses, DSM-III-R anxiety disorder, and receipt of public assistance. Study outcomes did not vary as a function of gender, prior history of medication, or comorbid disruptive disorders. However, some advantages were found for combined treatment in the moderator analyses:

- When considering ADHD symptoms, Comb treatment was more effective than MedMgt for families on public assistance, but not for families with greater resources (Jensen et al., 1999b).
- When considering ADHD symptoms, Comb treatment was superior to MedMgt for children with comorbid anxiety disorders, but not for those who did not have comorbid anxiety (Jensen et al., 1999b).

Mediator Effects in the MTA

One of the mediator analyses in the MTA focused on treatment acceptance/attendance (Jensen et al., 1999b). In this analysis, mediators were defined as acceptance of treatment and attendance at treatment sessions, specifically either as "as intended" or "below

intended." Operational definitions included accepting the treatment assignment, as well as percentage of treatment sessions attended: for MedMgt, 80% medical visits attended with prescriptions written/delivered during the sessions; for Beh, 75% attendance at group parent training sessions and STP days, as well as the presence of a child and a paraprofessional together in the classroom for 75% of the possible days of this aspect of the intervention. Comb families needed to meet both sets of unimodal criteria in order to be placed in the "as intended" category. Interestingly, neither individual parent training session attendance nor teacher–therapist consultation visits—both vital portions of intensive behavioral intervention—were counted.

In the "as intended" subgroup, the main intent-to-treat analyses held (MedMgt = Comb, and both better than CC and Beh). However, in the "below intended" subgroup, Comb was superior in terms of ADHD symptom reduction, with MedMgt = Beh (Jensen et al., 1999b). Thus, there was an effect of compliance with treatment outcome, and the Comb condition was apparently more robust to noncompliance.

Another mediator analysis examined the effects of naturally forming subgroups around medication use: consistent use of medication, no use of medication, stopping medication, or starting medication (Swanson et al, 2008). These subgroups were examined to evaluate the mediating effect of pharmacological treatment on ADHD symptoms. This resulted in some important revelations:

- Most of the decline in treatment efficacy for the Comb and MedMgt groups relative to Beh and CC groups was mediated by a lack of maintenance of taking medication (Swanson et al., 2008).
- Height and weight gains were less in the consistent use of medication group than the no use of medication group (Swanson et al., 2008), showing a clear link to stimulant-mediated growth suppression of about 1 cm per year in height gain (0.4 of an inch) and 1.2 kg per year in weight gain (2.6 pounds). There was no evidence of growth rebound. Importantly, growth suppression appears to be dose dependent, so the 20% lower dose in the Comb treatment has some important implications for managing growth suppression side effects.
- Analysis of change from the 24- to 36-month follow-up revealed that continued use of medication was a marker for deterioration rather than benefit (Swanson et al., 2008). Further analysis of medication subtypes (using a statistical grouping of response over time) found that about 66% of initial responders to medication displayed dissipating effects of medication, with only about 34% of the medication responders showing long-term positive benefit from medication.

Summary of MTA Results

The initial press releases of the MTA suggested the MedMgt was the best treatment. In retrospect, many involved with the MTA regard this as a mistake and feel that combined treatment was superior to medication alone (Schwartz, 2013). At best, the advantage of medication was true only in the short run and only for ADHD symptoms. When outcomes other than ADHD symptoms are considered, the MTA found numerous advantages of Comb treatment. Taken together, the MTA findings firmly support the superiority of the MTA Comb intervention over the MTA MedMgt or Beh conditions.

META-ANALYSES OF COMBINED TREATMENTS

Despite the richness in scope and size of the MTA study, it is only one study that included a single combined treatment arm. Results of a number of other studies since the 1970s on combined treatment have been summarized in two relatively recent meta-analyses published since the previous edition of this volume. An important consideration when examining the results of these meta-analyses, as illustrated by the disparate results of meta-analyses on psychosocial interventions, is to consider carefully what kinds of interventions and studies are included.

In a meta-analysis of combined interventions by Majewicz-Hefley and Carlson (2007), selected studies included a combination of pharmacological and psychosocial treatments, and examined outcomes including symptoms of ADHD (i.e., inattention, hyperactivity, impulsivity), social skills, and/or academic skills. Eight studies met the inclusion criteria. Participants in these studies were primarily elementary school-age children, ages 5–12 years (mostly males ages 7–8 years of age). A large number of studies of combined treatments were excluded (18 of 26) because data needed to calculate effect sizes were missing.

According to Majewicz-Hefley and Carlson (2007), combined treatment approaches appeared to have the

largest and most significant impact on core symptoms of ADHD and in the domain of social skills. However, impact on academic functioning was small. Nevertheless, there were school effects because effect sizes from teacher ratings tended to be higher than ratings made by parents. A potential anomaly in this review was that the effect sizes obtained for medication treatment alone were of greater magnitude than those seen with other studies, thus potentially obscuring some of the combined treatment effects. Of note Majewicz-Hefley and Carlson commented on the limitations of comparing effect sizes from one meta-analysis to those of another, so this unusually large effect of medication may not be a salient issue.

Regardless of the issue of effects sizes relative to other meta-analyses, when these findings are considered alongside results of meta-analyses of monotherapies, it appears that combined treatment approaches can address core symptoms as well as at least one key domain of functioning (peer relationships). However, in this meta-analysis, academic performance was not appreciably impacted.

A meta-analysis by Van der Oord, Prins, Oosterlaan, and Emmelkamp (2008) included 26 studies that specifically examined fixed doses of short-acting MPH as the pharmacological agent, psychosocial treatments that were only behavioral or cognitive-behavioral in orientation, and their combination in school-age children (6–12). Outcomes included core symptoms of ADHD, symptoms of ODD/CD, social behaviors, and academic functioning. Only group-based studies conducted in outpatient settings were selected for use; case studies and single case designs were excluded (Van der Oord et al., 2008).

A total of 24 studies assessed ADHD symptom outcomes. Moderate to large effect sizes were found for medication, psychosocial, and combined treatments; effect sizes reported by teachers were greater in the medication condition. In the 17 studies that examined symptoms of ODD and CD, medium to large effect sizes were reported for all three interventions (although teacher effects sizes were small for psychosocial interventions). With regard to social behavior, medium effect sizes were found for all three interventions, with combined treatment resulting in moderate to large effects.

A smaller number of studies measured academic outcomes (seven studies). Behavioral effect sizes were small (0.19), and, with the exception of one study, medication effect sizes were negative to small. Combined treatments had small effect sizes. Behavioral treatments appeared to result in somewhat stronger average impacts compared to interventions categorized as cognitive-behavioral.

Based on their review, Van der Oord and colleagues (2008) concluded that for ADHD symptoms, both MPH and combined interventions were equally effective (with large effect sizes) and behavioral/cognitive-behavioral interventions were somewhat less effective (moderate to large effect sizes). All three treatments resulted in moderate to large improvements in ODD/CD symptoms and social behavior. Medication had no appreciable impact on academic functioning, and behavioral monotherapy had relatively small effects, at least by typical standards of effect sizes seen for other variables.

In areas other than core ADHD symptoms and academic performance, comparable amounts of improvement were found for all three treatments. Of note, mean weighted effect sizes were largest for combined treatment approaches in all domains, but owing to the limited number of studies, this difference in effect size was not significantly different than that for medication. Based on this potentially flawed statistical reasoning (i.e., likely a type II error), which was also seen in some MTA publications, Van der Oord and colleagues (2008) concluded that in school-age children there is no advantage to combined treatments over MPH on ADHD, ODD/CD, social behavior and academic functioning. They also concluded that psychosocial treatment was just as good for improving social behavior and ODD/CD symptoms as MPH.

An important consideration raised by Van der Oord and colleagues (2008) as well as others (e.g., Pelham, 1999) is the relative timing of interventions. If treatment with a medium to large dose of stimulant such as MPH comes first, there may be no room for improvement if psychosocial treatments are added (these conditions appeared equal in this meta-analysis). Given the advantages of low doses of medication relative to moderate to high doses of medication, it is worthwhile to examine what happens if psychosocial treatment is added to a low dose of medication.

EFFECTS OF VARYING DOSES OF TREATMENT

In a landmark study, Fabiano and colleagues (2007) tested the hypothesis that combining low doses of stimulant medication with low doses of behavior mod-

ification could achieve the same effect as high doses of either of these interventions delivered individually. This hypothesis was based on the results of a number of small, school-based studies reporting that the combination of behavior modification and stimulant medication resulted in a treatment synergy that showed up at the lower doses (many of these are reviewed in the section on early research). Briefly, the presence of behavioral treatment seems to change the dose–response curve of stimulants, such that most of the benefit is seen at low doses, with diminishing therapeutic returns at higher doses. Likewise, low doses of stimulants alter the dose–response curve of behavioral intervention, such that low-level behavior modification plus low-level stimulants lead to similar benefits as found in high-level behavioral intervention.

Prior to the Fabiano and colleagues (2007) study there was lack of agreement in the extant literature about the intensity of interventions. Furthermore, a restricted range of intensity and a wide variety of measures and interventions limited inferences from these studies. To address the ambiguity regarding the strength of the behavioral interventions, Fabiano and colleagues operationalized three ecologically valid levels of school behavior support. The lowest level was designed to resemble the typical behavior management in schools that *do not* use state-of-the-art positive behavior support (Sugai & Horner, 2009). Thus, there was verbal feedback about rule violations but no systematic consequences, no time-outs, low rates of verbal reinforcement, noncontingent access to most activities and privileges (e.g., recess), and no systematic tracking or reporting of behavior.

To provide a somewhat stronger intervention, Fabiano and colleagues (2007) implemented a low-intensity behavior management condition that resembled what might be implemented in a school that has adopted Positive Behavior Support (PBS). Thus, there were rules and daily prompts about the rules, the opportunity to earn points for completing tasks but no point loss for breaking rules, time-outs for serious rule violations, social reinforcement and social honors, and Daily Report Cards. In a PBS framework, this level of intervention would be regarded as more than a basic or Tier 1 intervention; we think Fabiano and colleagues' "low-intensity" intervention is consistent with a Tier 2 intervention (Sugai & Horner, 2009) and this might therefore be considered a moderate-intensity level of behavioral support.

The highest level of behavior support provided by Fabiano and colleagues (2007) included a daily review of rules, reminders of rules at the start of each activity, earning points for completing tasks and for task accuracy, losing points for breaking rules, time-outs for seriously inappropriate behavior, recess contingent on points, high rates of social reinforcement, social honors, Daily Report Cards, and individualized behavior plans. There is strong support for the acute effectiveness of this level of behavioral support for children and adolescents with ADHD. In the context of PBS, this would be regarded as a Tier 3 intervention (Sugai & Horner, 2009) and is probably not feasible in the typical classroom. However, the level of intensity should resemble what might be implemented in a very sophisticated special education classroom. Thus, unlike the heroic level of psychosocial intervention in the MTA study, this level of intensive contingency management should be available to a large number of children and adolescents with ADHD.

In addition to standardizing the dose of behavioral intervention, Fabiano and colleagues (2007) extended the range of doses of the stimulant medication MPH that was studied. In previous studies, the so-called low dose typically was 0.3 mg/kg of MPH. However, this is a pretty typical dose of MPH that might be regarded as a moderate dose. Based on the early studies, there are some indications that in the context of strong behavioral treatments lower doses of MPH than 0.3 mg/kg could result in sufficient therapeutic gain. Therefore, Fabiano and colleagues introduced a lower dose than in seen in most stimulant research (0.15 mg/kg MPH), which was compared with the more typically studied 0.3 and 0.6 mg/kg doses of MPH. This resulted in average doses of about 5 mg, about 11 mg, and about 21 mg of MPH given in three doses a day. There was also a placebo condition.

Doses of medication and behavioral support were randomized in the context of a counterbalanced 3×4 within-subject factorial design (three levels of behavioral support and four levels of medication). Data were collected on 48 children ages 6–12 who were diagnosed with ADHD and participated in the 9-week Summer Treatment Program (Fabiano et al., 2007). Subjects attended the program 9 hours a day, 5 days a week. Data for this study were collected during 2 hours of daily classroom time in which behavioral support and medication were systematically varied. Major outcome measures include rule violations in the classroom, academic

productivity, and teacher ratings of behavior, impairment, and stimulant side effects.

The overall pattern of results in the Fabiano and colleagues (2007) study supported the idea that there is a moderating effect of combining treatments that changes the dose–response curve of stimulant or behavioral treatment for ADHD. Specifically, in the presence of behavioral treatment, lower doses of stimulant than usually prescribed have therapeutic benefits. Furthermore, in the presence of Tier 2 behavioral intervention, the dose–response curve of stimulants shifts, such that most of the benefit is seen at low doses of MPH, with very little additional benefit of higher MPH doses. Likewise, low doses of stimulants alter the dose–response curve of the behavioral intervention, such that Tier 2 behavior modification plus low-level stimulants lead to similar benefits as those found in high-level (Tier 3) behavioral interventions.

The finding that combining treatments has a moderating effect on dose–response curves has major implications for the practicality and sustainability of treatment. To begin with, most negative stimulant medication side effects are dose-dependent, so using lower doses should result in a safer, more tolerable, and sustainable treatment. Furthermore, behavioral treatment at lower intensities may allow a student to remain in a regular classroom setting using procedures that are acceptable, feasible, and sustainable for parents and teachers to deliver together, with minimal support from mental health professionals. Taken together, compared to high-dose treatments, the lower-dose treatments could result in more students with ADHD receiving effective treatment for longer periods of time, which is badly needed given that ADHD is a chronic disorder that is not cured by either of these treatments.

We should stress that these findings about the moderating effects of combining treatments on dosage effects have been studied primarily in classroom settings. Thus, these findings may not necessarily generalize to social behavior, and the moderating effects of combined treatments still need to be systematically tested. Nevertheless, there is a parallel literature to the classroom studies that seems to show a similar dose–response effect on the behavior of children and adolescents with ADHD. For instance, Evans and colleagues (2001) reported that there were diminishing returns of higher doses of stimulants, such that 10 mg of MPH three times a day produced effects similar effects compared to those for 20 or 30 mg on classroom performance in the context of a strong behavioral intervention. Likewise, the same sample of adolescents showed a very similar dose–response pattern of stimulants on social behavior in the context of a strong behavioral intervention (Smith et al., 1998). The pattern of results showed that almost all of the benefit at the group level was at the lowest dose (10 mg), with very little incremental benefit at 20 or 30 mg.

Another consideration in studies of the moderating effect of combining treatments on dosage curves is that these results are for group trends. In the sample studied by Smith and colleagues (1998), about 75% of the adolescents showed minimally sufficient positive response to stimulants. Of those who had positive response, two-thirds reached the therapeutic threshold at 10 mg, another one-sixth reached the threshold at 20 mg, and another one-sixth reached it at 30 mg. This finding indicates that there is a strong tendency in this ideographic analysis toward benefit of a low dose but, because one-third of the adolescents needed higher doses, this finding also reiterates the need to conduct individualized dose–response studies. In future studies, following the example of Fabiano and colleagues (2007), these individualized assessments should check for different dosing amounts (e.g., .15, .3, and .6 mg/kg of MPH) across settings with specified levels of behavioral support (e.g., none, Tier 1, Tier 2, or Tier 3).

CONCLUSION

In our chapter in the previous edition of this volume, we graded stimulant, behavioral, and combined treatments. Based on advances in the science of combined treatments, those grades should be revised. Owing to a better appreciation of the limitations of monotherapy for ADHD, highlighted by the MTA and other studies, stimulant medication and behavior modification, which we formerly gave a grade of A, have been downgraded to grade B. These monotherapies have strong support for efficacy but limitations on sustainability and scope of intervention (i.e., not all children benefit, those who do benefit are not all normalized, compliance over time fades, and effects are temporary). We previously gave combined intensive medication management and psychosocial treatment a grade of C, owing to robust efficacy but concerns about the acceptability, feasibility, and sustainability of the heroic levels of intervention used in the MTA Study. However, the finding by Fa-

biano and colleagues (2007) and some historical studies suggest that combinations of low doses can be provided to achieve similar efficacy to monotherapy with high doses. This suggests that the question clinicians should pose regarding combined treatment is not *whether* combined stimulant should be used, but *when* they should be used. Accordingly, we have revised our grade for combined treatments upward (from C) to a B+ or A–, pending future research. Long-term approaches to treating ADHD, such as those described by Satterfield and colleagues (1979, 1980, 1981) that thoughtfully deliver a variety of types and intensities of evidence-based treatments have the potential to make major changes in the development of children and adolescents with ADHD, possibly developing a grade A treatment for ADHD, which is badly needed for this chronic, impairing disorder.

KEY CLINICAL POINTS

- ✓ The current research base provides considerable support for combining behavioral and stimulant treatments for children and adolescents with ADHD. Therefore, children and adolescents who are not normalized by treatment with either stimulant or behavior therapy alone should be given a trial of the combination of these treatments.
- ✓ When behavioral and stimulant treatments are both active, lower doses of both treatments may be indicated. Providing lower doses has many practical and therapeutic advantages. For example, compared to higher doses, lower doses can decrease the cost of providing interventions, increase the availability of intervention (e.g., Tier 2 behavioral interventions are more likely to be accessible than Tier 3 behavioral interventions), and decrease the side effects of interventions (e.g., most stimulant side effects are dose dependent).
- ✓ The dose of intervention of combined treatments can be varied during the day to achieve maximum therapeutic benefit in the most efficient way possible. For example, the strongest behavioral interventions (Tier 2 or 3) should be set up for the morning and evening when stimulant medication is not an option. During the middle of the day in combination with low doses of medication, lower doses of behavior modification (Tier 1 or 2) can be offered, which should be feasible for most students with ADHD during the school day.
- ✓ Combing treatments can increase the range of outcomes that are positively impacted. Expanding the range of positive outcomes may be critically important to address the broad range of impairments typically exhibited by children and adolescents with ADHD. The MTA and the meta-analyses of combined treatments both reached the conclusion that pharmacological treatment had the strongest effects on ADHD symptoms, but adding psychosocial interventions resulted in improvements in oppositional aggressive behaviors, internalizing symptoms, social skills, parent–child relations, and academic performance.
- ✓ In addition to variations during the day, combined treatments should be adjusted to match fluctuating intervention needs during the week, month, or year. For example, during the weekend there may be no need for treatment during fun and engaging activities, but a high need during other activities (e.g., sports practice, studying, or doing chores). The intervention plan may be set to use the treatments that address the particular need for an activity (e.g., focus during an academic activity or appropriate social behavior during sports practice). During weekend or summer peer activities when appropriate behavioral modification is not available (e.g., birthday parties or summer camp) higher doses of stimulant mediation might be taken. Parents who want to lower doses of medication, can provide more behavioral support when possible.
- ✓ Successful delivery of combined treatments requires teamwork and communication among providers. Teachers, parents, and physicians will need to work together to plan, implement, modify, and sustain combined treatments. Initially this coordinated effort may be facilitated by a mental health professional who can establish treatment priorities, select appropriate outcome measures, and provide evidence-based progress monitoring (e.g., individual case study methods) to evaluate the effectiveness of the various treatments. An important goal of the team may be to find the minimally sufficient dose of each intervention, and to determine when during the day, week, or year doses should be raised or lowered.
- ✓ The combined treatment team might consider combinations of treatments other than stimulant medication and behavioral interventions; however, this should be done very carefully. First, the team should focus on psychosocial and pharmacological approaches

that have an evidence base. Combining untested or weakly supported monotherapies is unlikely to offer a significant advantage, could replace or interfere with an effective treatment, and could expose the child or adolescents to unexpected or unnecessary negative side effects. Second, all of the evidence-based treatments to date are active at the point of performance, so various treatments that are not active at the point of performance (e.g., cognitive therapies and EEG biofeedback) should be regarded with skepticism. Third, when dealing with combinations of interventions other than stimulants and behavior therapy there may be unknown moderating effects on doses. For instance, combining a dietary supplement with medication could alter the potency of the medication. Thus, novel combinations of treatments should be very carefully monitored for efficacy, efficiency, and a wide range of outcomes, some of which may be unwanted side effects.

- ✓ Previous research has focused mostly on that additive or incremental effects of combining treatments. The new science of combining treatments should also examine interactions or dose response curves such as were found in Fabiano and colleagues (2007). This will require systematically varying doses of the different treatments used in combination with each other (either simultaneously or sequentially) and examining synergies between treatments and complimentary effects.
- ✓ Given that the apparent synergy between stimulants and behavior modification allows for lower doses of these treatments (when they are delivered simultaneously) to have equal the effects of high doses of monotherapy, researchers should probe for the minimally sufficient dose of each treatment. This may involve testing much lower doses than previously thought to be viable. In addition to finding a threshold for practical significance, these studies should also examine cost effectiveness. This public health perspective in psychosocial treatments is well articulated in multitiered approaches to behavioral supports provided by parents (Sanders, 2008) and schools (Sugai & Horner, 2009). From the perspective of treating ADHD, low- to moderate-intensity behavioral supports that are implemented throughout the day and in all settings could eliminate the need for secondary treatment for many youth, and have a big effect in lowering the intensity of treatment for those who need secondary or tertiary intensive intervention. Using the lowest dose possible should improve the cost–benefit ratios, and otherwise improve acceptability, tolerability, and sustainability of the intervention.
- ✓ If doses are going to be systematically varied across the day, week, or year, then researchers are going to need to take a more fine-grained approach to measuring outcomes. Data collection will need to be tied to when doses of treatment vary during the day, week, or year. For instance, morning might be defined as the time until the morning dose of medication takes effect, weekdays might be defined as the school day (or more precisely by variations in behavior support and dose of medication during the school day), and so on. This is very different measurement strategy than the "one dose fits all" approach of the past when data were collected over much longer periods of time, such as parent ratings that summarized behavior over an entire day (or some sometimes an entire month) without considering variations in treatment during that time period.
- ✓ Studies of combined treatments for adolescents and adults are lacking, and will probably be very different compared to those for children. Behavioral interventions, typically administered by others (i.e., a responsible adult), are not going to available to some adolescents and most adults with ADHD. Thus, other types of behavioral interventions, possibly self-monitoring and self-reward programs with the help of a coach, can be provided and tested to see whether a low dose of behavioral modification appropriate for adolescents or adults can develop a synergy with medication, similar to that seen with children and adolescents, that can expand the efficiency, range, availability, and sustainability of treatment for ADHD.
- ✓ Consistent with the lack of research on adults, there is a general lack of research that approaches ADHD as a lifelong, chronic disorder. Although most of the lifespan studies of ADHD indicate that at least half of individuals diagnosed with ADHD in childhood grow into adults with ADHD, almost all of the treatment studies of ADHD examine short-term effects of interventions that are known to have temporary effects. This is certainly the case with behavioral and stimulant treatment, which can be studied with reversal designs because there is minimal carryover when the interventions are stopped. Even heroic treatment efforts, such as those delivered in the MTA, failed to show any long-term lasting benefit relative to usual care. Thus, for combined

treatments to have better long-term outcomes than the current standard of care, they need to be sustained throughout the lifespan and adapted to the varying developmental needs and environmental supports across the lifespan.

- ✓ Researchers and clinicians should examine the effects of treatment of ADHD on the behavior of parents and other adult caregivers. Children with ADHD are much more likely to be the target of negative adult behavior and harsh discipline; youth with disabilities, including behavioral disorders, appear to be an increased risk for maltreatment, although disability and maltreatment definitions vary by study (Sullivan, 2009; Sullivan & Knutson, 2000). Using Add Health data, an association between childhood ADHD and maltreatment have been identified in self-reports of adolescents (Ouyang, Fang, Mercy, Perou, & Grosse, 2008). Harsh discipline and maltreatment are very serious and preventable outcomes that have not received sufficient attention in the ADHD treatment literature. We are optimistic that combined treatments may reduce rates of maltreatment of children with ADHD. Studies have shown that, compared to parents of children with ADHD who are not medicated, parents of medicated children are less controlling and negative in their interactions with the child with ADHD (Barkley, Strzelecki, Karlsson, & Murphy, 1984). Likewise, parent training programs have produced major and clinically meaningful increases in positive behavior and decreases in negative behavior when parents of children with ADHD are interacting (Herbert, Harvey, Roberts, Wichowski, & Lugo-Candelas, 2013). Based on these studies, we believe that researchers should attend to parent–child interaction variables and test the hypothesis that combining medication and parent management training in low doses may reduce maltreatment of youth with ADHD a manner that is more feasible and sustainable than either approach in isolation.
- ✓ In addition to stimulant therapy and behavior management, researchers should consider the addition of omega-3 and/or omega-6 fatty acid supplementation. Other therapies do not seem to have robust enough support at this time to justify investigation as a combined treatment. This could change over time, but we are skeptical that treatments not active at the point of performance (e.g., cognitive therapy, social skills training, memory training, and EEG biofeedback) will be effective unless there is some documented carryover effect that is active at the point of performance.

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CHAPTER 29

Driving Risk Interventions for Teens with ADHD

Gregory A. Fabiano and Nicole K. Schatz

The transition to licensed driving is one of the final milestones in a long line of developmental transitions experienced by families as children move toward becoming independent adults. The act of handing the keys over to a licensed, teenage driver is a concrete representation of the increased independence and autonomy long-considered to be an important characteristic of healthy adolescent development. Yet it is also a transition with considerably higher stakes relative to other, comparable ones such as learning to ride a bike, starting kindergarten, or working at a first job. The intersection between increased autonomy for the teen, decreased control of teen behavior for the parent, and the greater consequences of errors during this activity make this a developmental transition that can trigger concern for parents and others, creating tension between the desire for autonomy on the part of the teen and the desire for safety on the part of the parent.

Data on teenage drivers indicate that this concern is warranted. Americans drivers between ages 16 and 20 years have by far the greatest injury and fatality rate compared to other age groups (National Highway Traffic and Safety Commission, 2005), and adolescent drivers are a prominent public health concern (American Academy of Pediatrics, 2006; *www.cdc.gov/injury/index.html*). Teens are involved in more fatal car crashes, and they are more likely than drivers with more experience to be the cause of serious car crashes (Compton & Ellison-Potter, 2008; Curry, Hafetz, Kallan, Winston, & Durbin, 2011). Insurance companies have long been aware of the pronounced risk that teenage drivers present, as anyone who has opened a policy invoice for a teenager knows well.

Outcomes are even worse when one considers the driving records of adolescents diagnosed with attention-deficit/hyperactivity disorder (ADHD). ADHD is associated with significant impairment for adolescent drivers (see Chapter 11; Barkley & Cox, 2007; Fischer, Barkley, Smallish, & Fletcher, 2007; Jerome, Segal, & Habinski, 2006; Thompson, Molina, Pelham, & Gnagy, 2007). There is now clear, converging evidence from multiple prospective studies comparing well-diagnosed adolescents with ADHD and those without ADHD that teen drivers with ADHD have more accidents (Fischer et al., 2007; Nada-Raja et al., 1997; Thompson et al., 2007; Woodward, Fergusson, & Horwood, 2000) that result in greater costs (Fischer et al., 2007), injuries (Woodward et al., 2000), and more fatalities (Lambert, 1995). Adolescent drivers with ADHD are also more likely to receive a traffic citation for reckless driving, driving without a license, having hit-and-run accidents, and having a revoked or

suspended license (see Barkley & Cox, 2007, for a review). Young drivers with ADHD demonstrate poorer performance in driving simulators than drivers without ADHD, and self-report and other-report ratings indicate worse driving practices among young adults with ADHD (Barkley, Guevremont, Anastopoulos, DuPaul, & Shelton, 1993; Biederman et al., 2007; Fischer et al., 2007). Thus, across a number of studies, adolescents with ADHD, *who are already at-risk due to their age*, fare significantly worse than other teenagers on measures of car accidents, moving violations, and other driving-related negative behaviors such as "road rage" (Barkley & Cox, 2007; Jerome et al., 2006). Adolescents with attention problems are also significantly more likely to drive under the influence of alcohol and to have been arrested for this behavior (Woodward et al., 2000).

Along with these driving-related difficulties, parents of adolescents find that parenting becomes more difficult and complex, with increased conflict during this developmental transition (Steinberg, 2000; Steinberg & Morris, 2001). It can be presumed that parents of youth with ADHD may experience at least the same amount, if not greater conflict, than parents of typically developing teens. Notably, teenagers with disruptive behavior disorders, who have the greatest incidence of negative outcomes, also drive the most often once licensed (Woodward et al., 2000). Thus, due to a concentration of multiple risk factors around this developmental transition, teenagers with ADHD are a group that warrants intensive support as they enter the roadway.

The published 8-year follow-up of the Multimodal Treatment Study of ADHD (MTA; Molina et al., 2009) suggested some findings related to teen drivers with ADHD that shed light on the initiation to driving for teenagers with and without ADHD. In this follow-up of youth with ADHD, combined type, relative to comparison teens, those with ADHD fared worse on 91% of outcome measures assessed. Interestingly, driving outcomes (accidents and/or citations) were one of the few measures on which teens with ADHD did not fare worse than the comparison group. However, this is perhaps not reason for optimism: The study analyzed these driving outcomes for the youth if they were eligible to drive, regardless of whether they were licensed drivers. Youth with ADHD were less likely to have a license (40.43%) relative to the comparison teens (57.14%), and this difference was significant (Molina et al., 2009). Yet rates of accidents/citations were similar, 22% and 28%, respectively, for youth with and without ADHD. It is possible that the most severely impaired youth did not even have licenses, due to parents' decision to prolong the prelicensure period or to teens' difficulty completing prelicensure requirements (e.g., attending a 5-hour prelicensing course). It is also quite possible that the driving careers of these young teens were quite brief at this follow-up, yielding inadequate time for the driving risks to emerge. One may speculate that as these teens with ADHD become older, and more of them obtain licenses and have more opportunities to drive, differences between the groups will become apparent, similar to the other measures of outcome. Also of interest in this study is the impact of childhood treatment for ADHD on driving outcome. In this follow-up report, no childhood treatment (i.e., medication, behavior modification, combined treatment) positively or differentially impacted driving outcomes for youth with ADHD. This finding indicates the need for an intervention at the onset of driving for teens with ADHD because it does not appear that childhood interventions reliably support this teenage behavior.

WHY DO YOUTH WITH ADHD FARE WORSE AS DRIVERS?

The leading theory of teenage driving in ADHD includes a conceptual model pioneered by Michon (1978) that is still commonly used today (Barkley & Cox, 2007). In Michon's model, there are three hierarchical levels of tasks that need to be completed while driving. Besides the predriving level (basic abilities such as vision, motor coordination, etc.), the first driving level is the operational level. The practical, concrete skills that are related to driving are at the operational level (e.g., braking, steering, visually scanning the roadway). Immediately above the operational level is the tactical level, which includes the decisions and choices made at the operational level relative to the context of other drivers on the roadway (e.g., speeding up to pass another car). Finally, guiding the entire enterprise of driving is the strategic level, which includes the planning of the driving trip and its goals, choice of routes, and other decisions made about the trip (e.g., whether to drive by a peer's house to pick him or her up on the way). New drivers are likely to have difficulty in the operational level, given their novice status as drivers, and in the tactical and strategic levels due to lack of experience, as well as underdeveloped judgment. New drivers with ADHD may also have difficulties at the basic psychological level given their symptoms of in-

attention, distractibility, impulsivity, and poor motor control. The model currently conceptualized for teen drivers with ADHD is an adaptation and expansion of the one proposed by Michon (1978) and enhanced by Barkley and Cox (2007). Many of the actual driving-related domains at each level of the conceptual model are potential targets of intervention in an ADHD population. For instance, an intervention might focus on modifying attention and concentration at the basic or preoperational level and therefore affect the operational level. Presumably, this is one reason why stimulant medication has shown promise as an intervention for individuals with ADHD (e.g., Barkley, Murphy, O'Connell, & Connor, 2005; Cox, Merkel, Penberthy, Kovatchev, & Hankin, 2004).

In light of the model and associated skills embedded within it (described earlier), if one wanted to create a context that would be maximally impairing for the neurocognitive deficits experienced by youth with ADHD, the U.S. roadway might be the answer. Driving requires sustained vigilance, motor coordination, good judgment, behavioral inhibition, rule following, planning, and accurate self-evaluation skills. These constructs are all impaired in youth with ADHD relative to typically developing youth (see Chapter 2), and this may be one explanation for the increased negative outcomes within ADHD samples. We briefly describe each of these behaviors which are potential targets for intervention efforts.

Sustained Vigilance

Driving, especially for protracted periods of time, is a real-world continuous performance task. In a typical driving situation, long periods of unremarkable information are punctuated by abrupt attentional demands (e.g., children playing near the curb; a garbage can that the wind blew into the street) that must be observed, processed, and negotiated. Teenagers with ADHD may have more difficulty sustaining attention, and this may become more problematic during longer trips. According to Biederman and colleagues (2007), young adults with ADHD were more likely to collide with a stimulus that appeared suddenly on a driving simulation task (i.e., a dog running across the street) relative to young adults without ADHD, and this was exacerbated in a monotonous driving condition that was similar to highway driving.

In addition to failure to notice hazards that appear in the roadway, teen drivers may fail to notice road signs (stop signs, speed limit signs, signs indicating lane closures, etc.), traffic signals, signals from other vehicles (e.g., turn signals and brake lights), as well as other information critical to safe driving. Teen drivers who fail to observe this key roadway information due to difficulties with sustained vigilance therefore are a greater risk to their own safety, as well as to the safety of others.

Judgment

Judgment is a critical aspect of a driver's behavior. Past the operational stage, driving includes a social dimension wherein a driver must anticipate and interpret the behavior of others on the roadway. Although there are cues that help with this task during driving (e.g., left turn signal, brake lights), as well as explicit rules of the road, there are also less formal cues that might be used on the roadway (e.g., another driver might wave a car ahead at a four-way stop). Thus, in addition to responding to the demands of operating the car, a driver must also operate the car effectively within the context of other drivers operating their own cars, as well as other factors within the driving environment.

Intersections are perhaps the most taxing context for judgment while driving due to the multiple potential interactions with other drivers. It is estimated that more than 95% of the blame for accidents at intersections resides with the drivers of vehicles; only a small percent of blame is related to the environment or to vehicle malfunction (National Highway Traffic Safety Administration, 2010). In fact, "false assumptions about others' actions" and "misjudgment of a gap or another's speed" are factors that contribute to crashes.

Although exercising appropriate judgment to navigate intersections and other driving situations safely and effectively can pose a challenge for all drivers, it is reasonable to speculate that this task may be particularly difficult for drivers with ADHD. Difficulties with social judgment may be attributable to multiple factors. As described in the previous section, poor vigilance may lead to a failure to attend to and encode important information and driving cues. Thus, teen drivers with ADHD may fail to notice social cues of other drivers and may therefore also fail to react appropriately to such cues. Difficulties with social judgment for teen drivers with ADHD, however, may extend beyond difficulties with vigilance and attention. Teen drivers with ADHD may struggle to respond appropriately even when they observe cues from other drivers. There is considerable evidence that youth with ADHD are less competent in

social interactions (Landau & Milich, 1988; Pelham & Bender, 1982; see also Chapter 8). These problems are likely to continue throughout development and impact the driving judgment of those with ADHD. Defensive driving, for example, is predicated on anticipating the behavior of other drivers (e.g., anticipating that a driver is going to speed through a yellow light even though it is about to turn red), and this defensive driving skill may be difficult for youth with ADHD to attain early in their driving experience. Perhaps verifying these difficulties, Reimer and colleagues (2005) reported that individuals with ADHD who completed a self-report questionnaire about driving performance were significantly more likely to report a history of driving errors, and this difference between drivers with and without ADHD was greatest at the youngest age range (18–25 years).

Substance Use

Alcohol and drug use while driving contribute to higher rates of traffic fatalities, and drunk driving appears to be associated with especially high risk for teen drivers (Kelly, Darke, & Ross, 2004; Williams, 2003). The driving risk associated with substance use may be particularly salient for teenage drivers with ADHD given that they are more likely to use alcohol or other drugs at a high rate per using episode (Molina & Pelham, 2003; see Chapter 11). Furthermore, youth with ADHD are also more likely to drive while intoxicated (Barkley & Cox, 2007), which, again, suggests poor judgment. This combination of behaviors suggests the presence of heightened risk, and experimental research indicates that youth with ADHD who are driving while their ability is impaired by alcohol may be among the most high-risk drivers on the road. Experimental research suggests that a sober adult with ADHD shows driving behavior that is comparable to a typical adult with a 0.08 blood alcohol level (Weafer, Camarillo, Fillmore, Milich, & Marczinski, 2008), and that alcohol has a severe impact on the driving skills of individuals with ADHD (Barkley & Cox, 2007; Weafer et al., 2008). Thus, for young drivers with ADHD who choose to drink and drive, the level of impairment associated with ADHD is compounded by the deleterious effects of alcohol. Although drunk driving is appropriately viewed as a serious public health concern for the general population, it is an even more serious concern for individuals with ADHD given their greater impairment in driving skills and behaviors.

Behavioral Inhibition and Impulse Control

Behavioral inhibition and impulse control are important processes in driving. In some cases, inhibition involves stopping an ongoing behavior or preventing oneself from initiating a behavior. In a driving scenario, this might involve taking one's foot off the accelerator when a light turns red and moving it to the brake, or stopping the car when an object or pedestrian enters the roadway. But it also involves social inhibition in situations where one must subordinate one's immediate goals or interests to those of others, as is often required by official driving rules (e.g., turn taking at intersections). Behavioral inhibition has been identified as a neurocognitive process that is dysfunctional in youth with ADHD (Nigg, 2000, 2001). Multiple studies have demonstrated impairments in behavioral inhibition in youth with ADHD relative to typically developing controls, and this may be one explanation for the more frequent collisions observed in ADHD samples. Furthermore, although inattentive and distracted driving appears to play a large role in traffic accidents in the general population (Lam, 2002), Thompson and colleagues (2007) found that severity of inattentive symptoms was not significantly related to adverse driving outcomes. Instead, hyperactivity–impulsivity mediated the relationships between childhood ADHD and both number of tickets and number of automobile accidents during adolescence and young adulthood. Thus, impulsivity appears to be a strong contributor to the most serious driving outcomes experienced by youth with ADHD.

Another aspect of behavior than can be influenced by behavioral inhibition and/or impulsivity relates to the control of aggressive or angry impulses. As any driver knows, other drivers can exhibit behaviors that are at times aggravating (running a stop sign, holding up traffic by driving very slowly, etc.). The term "road rage" has made it into the lexicon to describe the strong anger and commensurate aggressive use of a motor vehicle by some drivers when another driver commits an error, exhibits a driving behavior that puts other drivers at risk or that frustrates the former driver. Experimental research involving youth with ADHD and comorbid disruptive behavior disorders has indicated that individuals with ADHD are more easily provoked and react with anger (Waschbusch, et al., 2002). Barkley and Cox (2007), in their systematic review of the literature, reported that drivers with ADHD are significantly more likely to experience and exhibit road-rage related

behaviors. Anger and frustration are possible antecedents to subsequent poor driving behaviors, and youth with ADHD (especially those with comorbid oppositional or conduct problems) may be at heighted risk for these maladaptive behaviors (see Chapter 3).

Rule Following

One of the challenges faced by individuals with ADHD is following rules. In childhood, there is clear evidence that among many other rules in organized settings, complying with adult requests, remaining seated when expected to, or raising a hand to speak in the classroom are difficult for youth with ADHD to follow consistently (Fabiano et al., 2007; see also Chapter 2). Similar to a classroom or home setting, the roadway is a rule-governed setting, and many driver behaviors are predicated on the presumption that other drivers are complying with rules, which increases the accuracy of behavioral predictions. This system works in general: Note that over 95% of collisions at intersections are due to driver errors, which typically include noncompliance with rules (e.g., failure to comply with right-of-way rules or to obey traffic signals; National Highway Traffic Safety Administration, 2010).

When the driving behavior of individuals with ADHD is considered, there is evidence of noncompliance with rules. Reimer and colleagues (2005) found that young adults with ADHD were significantly more likely to endorse behaviors consistent with violations of driving rules (e.g., ignore speed limits very late at night or very early in the morning; become impatient with a slow driver in the left lane and pass on the right). These violations increase the risk for negative driving outcomes because they result in driving behaviors that are less predictable and may result in greater propensity for errors on the part of the driver or other drivers. Barkley and Cox (2007) and Jerome and colleagues (2006) provide additional data in their systematic reviews that emphasize how driving rule violations occur at a greater rate among drivers with ADHD.

Poor Insight

Children with ADHD have poor insight into their symptoms and associated impairments, which makes them poor reporters of diagnosis or treatment effects. This has been termed in the larger literature the "positive illusory bias" (Owens, Goldfine, Evangelista, Hoza, & Kaiser, 2007): Individuals with ADHD overestimate their contributions to personal success and underestimate or do not acknowledge their contributions to personal failure. This finding from the larger ADHD literature is also documented within the functional domain of driving. Individuals with ADHD appraise their own driving skills as being better than parents or objective records report, suggesting a serious lack of insight into their own driving limitations (Fischer et al., 2007). This cognitive style characterizes ADHD in childhood and continues into adolescence (e.g., Hoza, Pelham, Dobbs, Owens, & Pillow, 2002). It represents a general attributional style that cannot be explained by deficits in driving knowledge (Barkley, Murphy, & Kwasnik, 1996) or maturation to adulthood (Knouse, Bagwell, Barkley, & Murphy, 2005). This characteristic of individuals with ADHD, along with an overestimation of abilities, is problematic because there is evidence that individuals with ADHD are less knowledgeable about driving situations (Barkley, Murphy, DuPaul, & Bush, 2002). Of concern, teens with ADHD are not likely to be motivated to work to improve driving knowledge and skills if they do not view themselves as being impaired in that area.

Sensitivity to Peer Influence

It is well established that teenage passengers negatively impact the driving outcomes of novice, teenage drivers. The presence of additional passengers is associated with teenage drivers' increased risk for crashes (Preusser, Ferguson, & Williams, 1998). Interestingly, the risk associated with passengers appears to vary depending on driver gender and the age and gender of the passengers. Risk associated with passengers is greater for male teen drivers than for female teen drivers, and risk appears to be especially high when the passengers are young males rather than young females (Chen, Baker, Braver, & Guohua, 2000; Simons-Morton, Lerner, & Singer, 2005). In other words, individuals who were found to be the riskiest drivers were also found to be the riskiest passengers. This is of particular concern for youth with ADHD given that teens with ADHD are both more distractible and more likely than other teens to affiliate with deviant peers who may themselves be risky drivers. Cardoos, Loya, and Hinshaw (2013) found that perceived deviant peer affiliation mediated the relationship between inattention and negative driving outcomes for teenage girls. Although the precise reason for these findings is not known, it may be that youth with ADHD are more easily influenced by peer atten-

tion and/or behaviors while driving, at the expense of attention to driving-related activities. Alternatively, teens with ADHD may drive in more attention-seeking ways to obtain the attention of teen passengers. Certainly, this is an area in need of additional inquiry and research.

Comorbidity

In addition to driving difficulties related to symptoms and impairment associated with ADHD, youth with ADHD may experience additional difficulties due to comorbid conditions that may also interfere with safe driving. Compared to other youth, those with ADHD are more likely to be diagnosed with a variety of comorbid conditions (see Chapter 5). In particular, higher rates of oppositional defiant disorder (ODD) and conduct disorder (CD) are found in youth with ADHD (Jensen, Martin, & Cantwell, 1997). Aggressive and defiant behaviors are associated with these behavioral disorders and may contribute to additional rule breaking and angry outbursts among teenage drivers with ADHD. Although to a lesser degree than ODD and CD, anxiety and other internalizing disorders also appear to be more common in youth with ADHD than in typically developing youth (Jensen et al., 2001). These conditions may also interfere with driving because anxious youth may attempt to avoid anxiety-provoking driving situations and therefore engage in less driving practice. Similarly, depression may contribute to lack of motivation to practice driving or it may serve to increase irritability during driving practice. As noted earlier, substance abuse/dependence in drivers with ADHD is an additional comorbidity that needs to receive more careful research attention (e.g., Weafer et al., 2008). As clinicians work with youth with ADHD, comorbidities should be assessed and treated as appropriate because these conditions may also influence driving outcomes.

EFFECTIVE INTERVENTIONS FOR ALL TEEN DRIVERS

Fortunately, there are a number of common interventions for novice drivers. A brief review of these interventions, and their effectiveness, informs intervention efforts directed at youth with ADHD. They include public policies, rules and regulations, and interventions for emerging drivers (e.g., those learning to drive) and for youth who are licensed and independent drivers. We briefly review each of these types of interventions.

Graduated Driver's License

A graduated driver's license (GDL) is typically a statewide policy that introduces independent driving to teens through successive approximations. Teens generally move from supervised driving (i.e., a learner permit) to restricted independent driving (i.e., only daytime driving), then to unrestricted licensure. In this way new drivers are able to gain road experience in supervised and potentially less challenging settings before experiencing the challenges present across settings. Most states have moved to a GDL program in which novice teen drivers are only permitted to drive during periods of relatively lower risk (e.g., during the day). Nearly every U.S. state has a GDL policy, with 46 states requiring a minimum period of supervised driving, 48 states enforcing nighttime driving restrictions, and 47 states setting restrictions for nonfamily passenger (Governor's Highway Safety Association, 2013). Notably, the GDL approach has resulted in reductions in car crashes where implemented (e.g., Shope & Molnar, 2003; Shope, Molnar, & Elliott, 2001).

Although the GDL approach reduces accidents and injuries when employed on a statewide level, the approach has limitations. Practically, the GDL limits are largely enforced through parental monitoring and contingency management, with involvement of law enforcement likely only as a consequence of a negative driving event (e.g., after a license is checked during a traffic stop for speeding). This puts considerable pressure on parents not only to know the GDL rules but also to monitor and enforce them consistently. This may be why studies indicate that there is considerable variability in parents' oversight of the GDL limits, which lessens the impact of the potentially effective program (e.g., Goodwin, Waller, Foss, & Margolis, 2006). When educational or informational interventions are used alone, results are nonsignificant (e.g., Chaudary, Ferguson, & Herbel, 2004). When asked directly during the entry to licensure, parents in general also have nonspecific plans for monitoring teenage drivers, and they are sometimes ambivalent about monitoring teen driving behaviors, in deference to the teen's privacy (McCartt, Hellinga, & Haire, 2007). This difficulty with monitoring and contingency management may be more pronounced given the long history of difficulties in parenting often experienced in families of a child with

ADHD. An additional limitation of GDL approaches is that they are typically age-rather than experience-informed. In New York State, for example, GDL limits do not apply if a driver is over 18 (or over 17 and has taken an approved driver education course). Thus, if youth with ADHD wait longer to receive their licenses than typically developing youth (Molina et al., 2009), they experience less time under the protective restrictions of the GDL approach. This suggests a potential "perfect storm" in which the most at-risk drivers receive the least amount of supervised practice/independent driving yet enter the roadway with fewer policy-enforced safeguards. Furthermore, if their parents also have ADHD, then this may result in both poor modeling of appropriate driving behavior and less supervision of the teen's driving, given that adults with ADHD also have impaired driving (see Chapter 11) and parental ADHD is associated with reduced monitoring of their children's activities (see Chapter 7).

GDL limits are among a number of contingencies that likely support safe driving in teenagers. Notably, GDL approaches are largely preventative and help the teen avoid challenging driving situations until he or she has sufficient on-road experience. Antecedent control strategies such as GDL are likely to be preferable to consequence control strategies, which may include a number of negative results for the teen and his or her family. These include obvious consequences such as traffic citations and fines, insurance increases following a negative driving event, cost of automobile repairs following a collision, and inability to drive while repairs are being made.

Driver Education Classes

Preventive and training approaches such as driver education classes in the community or in high schools are also commonly employed for novice drivers. Typical driver education programs include classroom instruction to introduce content related to driving rules, strategies, and risks, coupled with in-car driving practice on the roadway. Although routinely employed, the outcome data on driver education classes are modest. For instance, in a review of the results of a number of systematic, quantitative reviews of driver education outcome, Lonero and Mayhew (2010) concluded that there is little evidence to support the efficacy of driver education as a prevention program for future negative driving outcomes such as collisions. In fact, a few studies have yielded iatrogenic effects of driver education, wherein youth who complete driver education programming are at *greater* risk for collisions. Speculation for these unexpected findings relates to teens being overconfident following driver education, parents' reduced monitoring and oversight once the teen has complete the training class, and "time discounts" that reduce the GDL limits for those who receive training and put teens in risky situations sooner (e.g., nighttime driving restrictions are lifted if the teen completes a driver education class).

Checkpoints

Recognizing that an active approach may be needed to support families' adherence to GDL policy and driver education efforts, a novel, innovative approach to reducing the risk of novice drivers is the Checkpoints program, which is an intervention for novice drivers that includes the delivery of persuasive videos and newsletters aimed at parents and teens, and the establishment of the Checkpoints Parent–Teen Driving Agreement. The Checkpoints program assists parents in implementing a GDL approach for their teen driver, and the driving agreement strictly limits teen driving during high-risk situations (e.g., at night); parents are encouraged to maintain this agreement during the first year or more of the adolescent's licensure. The Checkpoints program increases parental limit setting related to adolescent driving through the provision of clear information and concrete behavioral tools (e.g., Simons-Morton, Hartos, & Leaf, 2002), and it has established efficacy in a statewide clinical trial on traffic violation and accident outcomes (Simons-Morton, Hartos, Leaf, & Preusser, 2006, 2007). Furthermore, parents who constructed a driving contract with their teen to set limits, rules, and expectations surrounding driving reported sustained parent–teen communication and better teen compliance with limit setting at initial follow-up, though these effects waned over time (Simons-Morton, Hartos, & Beck, 2003; Simons-Morton, Hartos, Leaf, & Preusser, 2005). Thus, there is clear consensus that parents of adolescents should be an integral part of the process of teaching and monitoring teen drivers (*www.cdc.gov/ncipc/factsheets/teenmvhactivities.htm*; *www.nichd.nih.gov/about/org/despr/studies/driving/checkpoints.cfm*), and that limit setting and monitoring are key ingredients of parental involvement (American Academy of Pediatrics, 2006). Although effective for teens in general, this type of video- and newsletter-based intervention may not be sufficiently intensive for teen drivers with ADHD.

But, as we discuss below, interventions for youth with ADHD can build upon programs such as Checkpoints.

Limitations of Current Interventions

Driving risk interventions for youth with ADHD can build upon the successful interventions for typical novice drivers. However, these interventions may also be insufficient for youth with ADHD. As previously reviewed, impaired attention and impulse control, poor insight into mistakes and misbehavior, and susceptibility to peer and other negative influences may be too severe to permit community-level interventions to work. Furthermore, parents of teenagers with ADHD may find management and supervision of GDL limits to be difficult if the teen is frequently noncompliant with limits or exhibits a high rate of behaviors that require intervention. A third concern is that teens with ADHD may fail to benefit from GDL limits if they are delaying licensure (Molina et al., 2009). Lonero and Mayhew (2010) suggest that "time discounts" that release teenagers from GDL limits early given participation in driver education classes are potentially unwise. Teens with ADHD may be inadvertently obtaining "time discounts" on the front end of driving if they are delaying supervised driving practice and GDL-mediated independent driving due to delayed licensure. Although it is unwise to push a teen into licensure, it is possible that it may be equally unwise, given the current structure of the steps used for initiating unencumbered driving in youth, to delay driving as well, and this appears to be occurring in samples of youth with ADHD.

Given the heightened driving risk for youth with ADHD, effective assessments and interventions are needed. Below, examples of such assessments and interventions are introduced and reviewed. The discussion is intended to provide an overview for practitioners working with youth with ADHD who are beginning to drive, as well as licensed youth/young adults with ADHD.

PRACTICAL ASSESSMENTS FOR DRIVERS WITH ADHD

Practitioners working with teenage drivers need to start with an assessment of driving behavior, contexts, and consequences. Any clinician working with a family of a teen with ADHD must carefully consider the purposes of assessment and use assessment tools that meet the expressed purposes. Mash and Hunsley (2005) describe the purposes of psychological assessment as including diagnosis and case formulation, screening, prognosis, intervention planning, intervention monitoring, and evaluating outcomes. Over the past decade, a number of driving-related measures specific to youth with ADHD have been developed, and though it is not an exhaustive review, a sample of available measures is described below. As we demonstrate, measures can be useful for some of the purposes of assessment outlined by Mash and Hunsley, and it is likely that a multimethod, multirater approach will be necessary to address multiple purposes, and new measures may need to be developed to inform the identification and monitoring of driving behavior in ADHD samples.

Measures of driving behavior in individuals with ADHD typically aim to identify whether these driving-related skills, strategies, and behaviors are impaired, and in what contexts. The Driver Behavior Questionnaire (DBQ; Donovan, 1993; Reimer et al., 2005) is one example used in the broader driving literature, but it is also validated in samples of individuals with ADHD. The DBQ is a 24-item self-report rating scale that asks the respondent to rate the frequency with which he or she engages in driving behaviors on a 6-point Likert scale. This measure assesses driving problems related to driving errors, attention lapses, and traffic law violations. It has good reliability and validity for adolescents with ADHD (Reimer et al., 2005). A measure that addresses a similar goal is the Driving Performance Rating Scale (DPRS) developed by DuPaul in collaboration with Barkley (Barkley et al., 1993; see also Barkley & Murphy, 2006) and used in a number of driving studies (Barkley & Cox, 2007). The 20-item DPRS is rated on a Likert scale and can be completed as a self-report or the report of a collateral. It may be useful for screening or case formulation purposes. The Jerome Driving Questionnaire (JDQ; Jerome et al., 2006) is another measure specifically created to assess driving competencies and potential risk in drivers with ADHD. The JDQ includes a series of questions about driving history (i.e., citations, collisions), then uses a visual analogue scale to assess driving behaviors in both city and highway conditions. A preliminary analysis yielded a four-factor structure for the tool, including Attention, Impulsivity, Alertness, and Emotional Lability factors. Interestingly, this measure may have merit as a screener. Jerome and colleagues (2006) reported that higher levels of self-reported impulsive and inattentive behaviors predicted collisions and citations at a 3-year follow-up in a normative sample of young drivers.

Rating scale data are efficient, and there is support for these measures in research settings. Practitioners may find it useful to include assessments of impairment as well. The reasoning behind this is multifold. First, in adolescent samples, impairment may be a better indicator of ADHD-related functioning than the DSM symptoms of ADHD themselves because in spite of impaired functioning, not all teens receive elevated symptom ratings (Sibley et al., 2011). This means that practitioners would do well to collect information on the teen's current functioning in important domains (family, driving) rather than just ADHD symptom-based ratings to screen for individuals in need of treatment. Second, impaired functioning identifies the socially meaningful targets of treatment. Furthermore, assessments of impairment also assist in case formulation and treatment planning, and they are the areas that treatment outcome evaluations should also address. Fabiano and colleagues (2011) utilized weekly ratings of impairment on the Impairment Rating Scale (Fabiano et al., 2006) completed by parents and teens to evaluate improvement in driving for licensed teens with ADHD, and this assessment approach was sensitive to driving-targeted treatment effects for some youth with ADHD.

Although self- and other-reported rating scale information may be useful, there are limitations to this approach. Parents may be unaware of the extent to which the teen is exhibiting risky behaviors while driving, and the teen may not be a veridical reporter of driving-related impairments. Therefore, objective indicators of driving performance may also be helpful to practitioners. The downside to objective measures of driving performance is that it can be difficult to obtain useful objective data. One potential objective measure of youth driving outcomes is the record from the Department of Motor Vehicles (DMV), which typically includes car accidents and traffic violations. This index is limited, however, because the official driving record may not include all incidents (e.g., a car accident that was settled individually by the family and did not go through insurance or have a police report filed; a hit-and-run accident in which the teen was responsible), and some incidents logged on the driving record may not reflect the actual incident (e.g., a more serious offense plea-bargained to a lesser offense). Additionally, driving records may not be consistently available to parents and practitioners. To supplement whatever information may be available from the official DMV driving record, it will be necessary to ask adolescents about the number of accidents, moving violations, and other violations that occurred, as well as specific details (cost of repairs, increase in insurance premiums, extent of injuries, etc.) about the event in order to obtain a comprehensive picture of more serious citations or collisions.

Fortunately, the development of new technologies may address many of the difficulties with obtaining objective driving data in teenagers with ADHD. There are a number of mechanisms through which technology has enhanced driving assessment; some are readily available for consumer use, while others are limited to research settings. These range from driving simulators to in-vehicle camera systems that record driving behaviors, to simple computer chips that monitor speed and engine performance. Of these options, the engine-performance monitors and some car cameras are the least expensive and most easily accessed by the general public. They are available for order online, and some products are less than $500 (a common collision insurance deductible). When viewed in light of the deductible plus rising insurance rates postcollision, purchasing and using these monitors may be cost-effective. Driving simulators, on the other hand, are generally less practical because families and practitioners typically do not have access to a simulator outside the context of a research study. Engine performance monitors, video monitors, and driving simulators provide access to clinically rich and accurate information about driving performance. As the field moves forward, they may become more prominent in ADHD assessments. For this reason, we briefly discuss the benefits and limitations of each in turn.

Driving Simulators

Driving simulators have been utilized in multiple fields as assessment tools (e.g., pilot training). Simulators confer advantages in that they can allow a researcher or practitioner to observe behavior that would be impractical or unsafe to observe in real-life settings. Simulators can be used as assessments, as well as mechanisms for rehabilitation or intervention. Notably, simulator use in ADHD research settings has increased. A number of studies of drivers with ADHD have been conducted with simulators. Barkley and Cox (2007) suggested that they are a viable and acceptable way to obtain information about the driving performance of individuals with ADHD. For instance, Narad and colleagues (2013) investigated whether distractions (conversation, text messaging) impaired driving in groups of adolescents with

and without ADHD. All teenagers performed worse on a simulated driving exercise while simultaneously texting. Those with ADHD exhibited more variability in their driving than did teens without ADHD. Simulator studies have also demonstrated the effectiveness of stimulant medication in youth with ADHD while driving and indicate that the simulated driving scenarios are sensitive to medication manipulations (Cox et al., 2006). Fabiano and colleagues (2011) used simulators as a laboratory-based setting for parents to practice parenting strategies directed toward the teen within the driving context, illustrating a different and valuable use of this tool for new drivers with ADHD. Although simulators are useful for laboratory assessments, they have limitations. As previously mentioned, one limitation is that they are quite expensive, costing from $100,000 to several million dollars. A second is that high-fidelity simulators are typically not accessible to community practitioners. Furthermore, although simulated environments can be very realistic, they are still only a proxy for the authentic driving environment and correlate only modestly with ratings of driving behavior and actual driving outcomes (Barkley & Cox, 2007; Barkley, Edwards, Laneri, Fletcher, & Metevia, 2001).

Engine Performance Monitors

Due to these limitations of simulators, in-vehicle measurements are becoming increasingly widespread. These include engine performance monitors, global positioning systems, and cameras that record cabin and roadway events. Engine performance monitors access the internal car computer system. Since 1996, automobile manufacturers have been required to include an on-board diagnostics (OBD) port to access the car computer in all models of cars (typically found under the dashboard). By connecting a commercially available engine performance monitor to the OBD port, parents and practitioners can access a wealth of information related not only to vehicle diagnostics but also driver behavior. Fabiano and colleagues (2011) reported on instances of speeding, hard braking, and hard acceleration in a sample of licensed drivers with ADHD, collected via weekly review of an engine performance monitor. Assessments conducted each week were sensitive to intervention targeting these behaviors. For example, speeding decreased for a teen whose weekly top speed was reviewed with parents and a clinician. Engine performance monitors are relatively cheap, easy to use and install, and they provide useful information on driving performance. Although useful, these monitors have limitations. For example, they do not allow identification of the driver, nor do they provide contextual information such as road or cabin conditions. Thus, parents or practitioners reviewing the data generated by the monitor will not know whether a hard brake was the result of good defensive driving on the part of the teen (e.g., braked quickly to avoid hitting a child who appeared suddenly from between two parked cars) or a driving error (e.g., not paying attention to the road and had to quickly brake to avoid running a stop sign). Also, these monitors record vehicle speed but provide no information regarding the appropriateness of the speed given the speed limit, road conditions, and traffic. Additionally, these devices only record information when they are connected to the vehicle. If teens forget or choose not to connect the device while driving, parents will have incomplete information about teens' driving performance.

On-Board Camera Monitors

To address some of these limitations, on-board camera monitoring equipment has been used in research settings. The typical system includes both forward-facing and rear-facing cameras to record the roadway and cabin, respectively. Many camera systems are continuously recording, and deleting, on a video loop. Within the camera systems is an accelerometer that is sensitive to changes in g-force (side to side; back and forth; up and down). When triggered, this saves a short clip of video immediately prior to the event onto a hard drive for later review. In this way, the behaviors that occurred immediately before the risky event can be reviewed, and potential antecedents of risky driving events, if present, can be identified. Multiple studies illustrate that on-board monitoring is feasible, reliable, and provides clinically meaningful information on teen driving (Carney, McGehee, Lee, Reyes, & Raby, 2010; Farmer, Kirley, & McCartt, 2010; McGehee Raby, Carney, Lee, & Reyes, 2007; Merkel et al., in press). Merkel and colleagues (in press) found that young adult drivers with ADHD, relative to those without ADHD, exhibit more frequent driving violations (no seat belt, speeding) as recorded by the event-triggered recorder. This assessment approach can potentially yield much more information on teenage drivers with ADHD, the contextual factors that promote positive and negative driving behaviors, and associated behaviors or targets of treatment, such as parenting (Schatz et al., 2014).

The merit of an in-vehicle monitoring system for novice drivers was cleverly illustrated by Farmer and colleagues (2010). In this study, cars were equipped with an engine performance monitor; there were four groups in the study: (1) an in-vehicle alert that was triggered if the teen exhibited a risky driving event (speeding, sudden acceleration/braking, unfastened seat belt) and an immediate notification of the event to a website accessible to the teen's parent; (2) immediate notification (no alert); (3) a conditional notification that only occurred if the teen did not modify behavior following the alert (e.g., fastened seat belt; modified speed to within the speed limit); and (4) a control group that received no alerts or notifications. Results were interesting; alerts alone were generally insufficient to change behavior. Combinations of alerts, time to modify the behavior, and contingent reports to parents if the teen persisted with the risky behavior appeared to work best. Farmer and colleagues stated that "teenagers may learn to tune out the alerts over time, so reinforcement from parents appears necessary to sustain good behavior" (p. 45). This approach is consistent with contingency management approaches long used successfully in youth with ADHD (Pelham & Fabiano, 2008) and with licensed drivers with ADHD (Fabiano et al., 2011). Contingency management approaches coupled with in-car monitoring appear to be a promising area for continued study.

EFFECTIVE INTERVENTIONS FOR YOUTH WITH ADHD

Policy

Although policies at the state or national level do not directly target youth with ADHD, it is reasonable to presume that these policies are created to address risky driving outcomes, which, as reviewed earlier, are disproportionately concentrated within ADHD samples. Thus, a brief review of policies within the context of ADHD intervention is warranted. Earlier we outlined the limitations of GDL approaches, and for population-based samples, programs such as Checkpoints are necessary to realize the promise of these policies due to the need to educate and empower parents and teens to follow the guidance provided. For families with a new driver, who also has ADHD, even more intensive approaches than Checkpoints may be warranted. Given the clear impairments of drivers with ADHD (Barkley & Cox, 2007), it is possible that longer training or restricted driving periods might help to attenuate some of these strong risk factors. Certainly youth with ADHD are entitled to a license, and earning a license is a clear way for the driver to demonstrate adaptive, functional behavior. But given the high stakes associated with driving, procedures to promote safer passage may be warranted in the form of additional restrictions placed on the teen. Perhaps this could be done informally through cooperation between the parent, pediatrician, psychologist, or other professional(s) working together in the treatment of the teen. Certainly, such approaches are worthy of additional empirical study.

Pharmacological Interventions

There is evidence that well-managed treatment with stimulant medications is associated with significant reductions in the core symptoms of ADHD for many adolescents, such that stimulant medications are recommended in combination with behavior therapy as the preferred treatment for adolescents with ADHD by the American Academy of Pediatrics (Subcommittee on Attention-Deficit/Hyperactivity Disorder, 2011). These medications are associated with not only symptoms reduction but also reduction in functional impairment across a variety of domains, such as school and social settings (Evans et al., 2001). Although much attention has been paid to the efficacy of medications in academic and other settings, there has been relatively more modest research on the efficacy of ADHD medications in improving driving performance for adolescents with ADHD.

It follows logically that an intervention that contributes to reduction in the core deficits associated with ADHD (e.g., inattention and distractibility) would also contribute to improvement in driving performance linked to those core deficits. In general, existing research suggests that stimulant medications lead to improved performance during simulated driving and real-world driving conditions among young drivers with ADHD (Cox, Merkel, Kovatchev, & Seward, 2000; Cox et al., 2004, 2006; Cox, Mikami, et al., 2008). The most striking evidence for potential benefits of stimulant medication for young drivers with ADHD comes from a recent study by Cox and colleagues (2012), in which video monitors were used to capture driving behaviors over a 3-month period when drivers were taking stimulant medication (long-acting transdermal preparation) and over a 3-month period when drivers were not taking medication. None of the 17 young

adults who participated in this study had an automobile collision while medicated. In contrast, six participants had a collision, and one participant had two collisions while driving unmedicated. Furthermore, all but one of these collisions was deemed to be the fault of the driver as a result of inattentive driving (e.g., while using a cell phone). It should be noted that this study employed a relatively small sample; however, the findings do suggest that stimulant medications may contribute to the safety of young drivers with ADHD.

Although not yet tested in youth with ADHD, the nonstimulant atomoxetine has been evaluated for its effects on driving in a small sample of young adults with ADHD (ages 19–25) (Kay, Michaels, & Pakull, 2009) and in two older cohorts (Barkley, Anderson, & Kruesi, 2007; Sobanski et al., 2013). The study of young adults revealed no improvement in simulator performance from this medication, whereas a stimulant medication did result in improvement (Kay et al., 2009). In contrast, the small pilot study by Barkley and colleagues (2007) found improvements in self-rated ADHD symptoms, impairment, and safe driving behavior but not in simulator performance or others' ratings of safe driving behavior. The Sobanski and colleagues (2013) study evaluated a much larger sample and found that the drug improved both the actual, real-world driving performance and neuropsychological measures related to driving. It also found that those taking medication were three times more likely to pass the German official driving requirements criteria than those in the waiting-list control group. These results warrant replication of these findings with teens, but it is likely that given the smaller reductions in ADHD symptoms associated with atomoxetine relative to stimulants, one might expect the improvements in driving to be somewhat less.

Practitioners who consider prescribing a stimulant medication for a teen driver with ADHD should consider the timing of doses, which appears to play a role in driving ability. Not surprisingly, driving improvements associated with a short-acting preparation of methylphenidate appear to wear off several hours sooner than do driving improvements associated with long-acting preparations (Cox et al., 2004). Practitioners should consider the time of day teens will be driving and consider choosing a medication that allows extended symptom relief for teens driving in the evening and at night. Also related to medication schedules, some adolescents have a history of taking medication only on school days and not on weekends or during the summer. This type of medication schedule no longer may be appropriate as teens transition to driving given that they are likely to be driving in the evenings and on weekends and will therefore need extended medication coverage.

It is important for practitioners to keep in mind that there have been relatively few studies of the impact of stimulant medication on young adult drivers, and even fewer have focused specifically on teen drivers (for exceptions, see Cox et al., 2004, 2006). Additionally, the majority of existing studies employed small samples. More research is needed regarding potential stimulant medication benefits for younger adolescent drivers with ADHD who are new to driving and may have the greatest need for effective intervention to aid driving. Furthermore, efficacy of medication for teen drivers is but one important question. Additional questions that need empirical investigation deal with compliance with medication and procedures to promote adherence to medication regimens, appropriate dosing, and combination therapies and their efficacy (Smith, Waschbusch, Willoughby, & Evans, 2000). Thus, naturalistic studies that move beyond efficacy to questions of effectiveness and maintenance are needed to better understand the supports conferred for drivers with ADHD through pharmacological intervention.

Technology

As we discussed earlier, parents of teens with ADHD at times may utilize ineffective or maladaptive parenting skills, including low levels of behavioral monitoring (Robin & Foster, 2002). They would therefore be expected to be among the parents who exert poor or inconsistent oversight even in states with GDL limits. It is also not enough for a parent of a teen with ADHD to be a good monitor (i.e., know where the teen is going, who the teen is with, use on-board monitors of driving behavior); the information gained through monitoring must be linked to clear contingencies (i.e., positive and negative consequences). Thus, alternative approaches that move beyond the nonintensive provision of information or tools are needed to help parents of teens with ADHD implement and monitor GDL approaches. Furthermore, parents may need tools such as on-board monitoring technology to facilitate their supervision and contingency management of teen driving because parental impressions or teen report alone are insufficient (e.g., Knouse et al., 2005).

Psychosocial Intervention to Promote Safe Driving for Teenagers with ADHD

ADHD is now widely conceptualized as a relatively chronic condition for many, arising early in childhood and lasting throughout development. This means that by the time parents and teens are ready to initiate the transition to independent driving, it occurs within the context of the historical parent–teen relationship that has developed over close to two decades. This is important to acknowledge because both good and bad habits formed over time in other contexts may be present in the teaching, monitoring, and managing that is necessary for teen drivers, as well as in the response to these behaviors on the part of the teen. It is unlikely that psychoeducational or low-intensity interventions will provide sufficient support to families of teens with ADHD given this context. Thus, intensive, sustained, family-focused interventions are necessary to promote the change and maintenance of gains needed to support teens as they embark on this critical and high-risk developmental transition. The Supporting a Teen's Effective Entry to the Roadway (STEER) program is one such intervention that includes multiple intervention components aimed at the teen, the parents, and the family unit. The STEER intervention integrates best-practice interventions for teenagers with ADHD into a treatment package that is implemented over the course of the initiation to independent driving. Importantly, maintenance procedures that lean heavily on the use of innovative technologies are also included in the program because the early months to the first year of independent driving are widely acknowledged to be the most risky time for new drivers (American Academy of Pediatrics, 2006; Curry et al., 2011; National Highway Traffic Safety Administration [NHTSA], 2005). An outline of the STEER intervention is provided to guide practitioners working with teenagers with ADHD and their families. Although the STEER program is early in development, it is offered as a heuristic approach to how risky driving interventions might be integrated into a multimodal treatment for young drivers with ADHD.

The STEER program is an 8-week, parent–teen intervention focused on improving outcomes for adolescent drivers with ADHD. During each week of the STEER program, sessions are divided into two 45-minute meetings, with the first half including individual parent and teen meetings that occur in parallel and the second half including a joint activity. In an effort to address the driving impairments of teens with ADHD, the STEER program was developed. The STEER program acknowledges that the risk associated with teenage drivers with ADHD goes beyond the driver and incorporates the family. Furthermore, it is clear that the initiation to driving begins when the teen receives his or her driving permit (or perhaps even earlier, if parental modeling of driving behavior is considered) and extends to the point of licensure and postlicensure, when the teen begins to drive independently. Thus, program components include training teens in operational driving skills, training parents in parenting strategies that support positive driving outcomes, and training families in contingency management and effective communication. Technological innovations that support operational and parenting strategy practice and parental monitoring of teenage driving behavior are also infused into the program. It is important to underscore that few of the interventions included within the STEER intervention are novel; many have been around for decades and used to intervene with teens and families with and without ADHD. The STEER program includes these interventions and integrates them into a comprehensive treatment package, with the express goal of supporting the safe driving of teenagers with ADHD. The specific components of the STEER program are reviewed below.

Motivational Enhancement

The handful of studies of teenagers with ADHD within psychosocial interventions suggests high rates of dropout from treatment (Barkley et al., 2001), and this indicates that clinicians must attend to the motivation of the teens and their families. Teens may be unenthusiastic about engaging in therapeutic interventions. Lack of enthusiasm for intervention is not limited to the teen; parents of teens with ADHD may be exhausted from years of parenting a child with disruptive behavior difficulties, which may result in a potential for parental lack of engagement as well (Dishion, Nelson, & Bullock, 2004). To combat this potential lack of engagement with intervention, motivational enhancement procedures are built into the initial STEER session to help the parents and teen identify goals and think through the benefits of changing behavior to meet those goals. Using procedures related to a motivational interviewing approach (Baer & Peterson, 2002; Miller & Rollnick, 2002; Stormshak & Dishion, 2002), the parents

and the adolescent are asked to identify the benefits of participating in the program and potential costs of nonparticipation using a decisional balance worksheet. Data on the common outcomes for adolescent drivers with ADHD are also presented and discussed to help participants understand how the driving behavior of teens with ADHD is riskier relative to the outcomes for teens in general.

Motivational interviewing strategies, even brief ones, may have real merit for teen samples and for driving-focused outcomes. Monti and colleagues (1999) reported improved driving outcomes for adolescents who participated in a motivational interview after a hospital visit due to an alcohol-related issue. Combined across 3- and 6-month follow-ups, adolescents who received the motivational interview, compared to standard care, were significantly less likely to have an instance of drinking and driving or a moving violation as measured by DMV records. Importantly, Monti and colleagues focused on adolescent problem-drinkers; these driving-related outcomes may be even larger when driving is explicitly addressed.

Teen Portion of Sessions

Problem-solving communication training is an approach that may have merit for teenagers with ADHD (see Chapter 22; Barkley et al., 2001; Barkley & Robin, 2014; Pelham & Fabiano, 2008). As children with ADHD grow into adolescence, communication, negotiation, and problem solving need to occur on an ongoing basis, and about topics that may be more serious or conflictual. Conversations about driving are likely to be in this realm. Parents participating in our intervention program report multiple issues with their teen about driving: wanting to drive too soon, not being motivated to learn how to drive, forgetting medication before driving, driving poorly, being unresponsive to feedback from parents when learning to drive, and breaking rules related use of the car, among many other potential situations that cause conflict. Likewise, teens also report having issues with parents: being irritated with feedback the parents provide when the teen is learning to drive, not getting enough practice, feeling pressured to practice more, and being upset about parental restrictions regarding the car, among many other concerns. These issues are unlikely to be transient and will fester or increase in negativity, and if left unresolved, the parents and teen may reduce and avoid needed interactions. In addition to the problem of ignoring these issues, it is also possible that the teen may need to build competencies in communication-related skills and negotiation strategies in order to partner with parents effectively to discuss and solve problems related to driving. Thus, a portion of the STEER sessions involves the teen meeting with a paraprofessional counselor to support the development of problem-solving and communication strategies that can be utilized when discussing driving-related (and potentially other) issues with the parents.

During the first portion of the meeting, the teen meets individually with the paraprofessional counselor to review safe driving behaviors and learn about effective communication and social skills (Barkley & Robin, 2014; Robin & Foster, 2002; Smith, Molina, & Eggers, 1997). The content of the teen sessions includes (1) a discussion of house rules and rules of the road; (2) expressing feelings and knowing the feelings of others; (3) making an appropriate complaint; (4) appropriately answering a complaint; (5) accepting limits set by others; (6) introduction to communication skills; (7) managing and challenging unreasonable thoughts and beliefs; and (8) review and planning for maintenance (see Table 29.1). These strategies were identified by the scholars cited earlier as being important for skills building in teens with disruptive behavior disorders; in the STEER program, these skills are reviewed through the lens of learning to drive and driving independently.

Parent Portion of Sessions

The cornerstone of most effective interventions for a child with ADHD is a component to teach and reinforce parenting skills. Parenting programs typically employ well-manualized approaches and teach the fundamentals of behavior change through rewards, punishments, and modeling, as well as basic information about ADHD. Parents are given assigned readings and are taught standard behavioral techniques (Barkley, 2013; Cunningham, Bremner, & Secord, 1998; Eyberg & Boggs, 1998; Forehand & McMahon, 1981; Patterson, 1975; Webster-Stratton, 1997). The typical skills training program for parents include a series of eight to 16 weekly sessions for the initial training, and the intervention is continued as long as necessary, with built-in programs for maintenance. Parent training is accomplished individually or in groups; weekly assignments are given to parents to track behavior and practice techniques with their children between sessions.

TABLE 29.1. Overview and Description of the Parent, Teen, and Joint Sessions in the STEER Program

Session	Teen content (first 45 minutes)	Parent content (first 45 minutes)	Joint session (second 45 minutes)
Session 1	Motivational enhancement; house rules and rules of the road discussion	Motivational enhancement; house rules and rules of the road discussion; introduction to parent–teen negotiation; psychoeducational introduction	Establishment of house rules and rules of the road; review of objective monitoring of adolescent driving measure
Session 2	Expressing feelings and knowing the feelings of others	Noticing and attending to positive driving behaviors	Establishment of monitoring behaviors; simulation exercise
Session 3	Making a complaint	Using instructions that work while teaching driving skills	Review of house rules/monitoring; review of objective monitoring of adolescent driving measure
Session 4	Answering a complaint	Planned ignoring/parental inhibition of nagging or prompting to promote independent driving	Parent–teen negotiation; simulation exercise
Session 5	Accepting limits set by others	Punishment (e.g., privilege removal) for violations of road rules	Parent–teen negotiation; review of objective monitoring of adolescent driving measure
Session 6	Introduction to communication skills	Introduction to communication skills and how to discuss driving related limits and issues with the teen	Parent–teen negotiation; simulation exercise
Session 7	Unreasonable beliefs	Unreasonable beliefs about adolescent driving behavior and their impact on parenting	Parent–teen negotiation; review of objective monitoring of adolescent driving measure
Session 8	Negotiation strategies review	Negotiation strategies review programming for maintenance/ monitoring of independent teen driving	Parent–teen negotiation; establishing rules of the road for independent driving; ongoing monitoring using CarChip

General reviews of behavioral parent training (BPT) for ADHD support the use of BPT for children described as antisocial or disruptive (e.g., Barkley, 2013; Eyberg, Nelson, & Boggs, 2008; Lundahl, Risser, & Lovejoy, 2006; Pelham & Fabiano, 2008; Serketich & Dumas, 1996), the majority of whom also meet criteria for ADHD. Meta-analyses also yield positive effects for BPT (Corcoran & Dattalo, 2006; Fabiano et al., 2009; Lundahl et al., 2006; Purdie, Hattie, & Carroll, 2002; Serketich & Dumas, 1996). Thus, this treatment approach results in moderate to substantial improvement for children with a variety of disruptive behavior problems. There are currently a number of well-validated, manualized parent training programs available for clinicians to implement. These programs are typically based on the work of Patterson and Gullion (1968), Hanf (1969), and the work of K. D. and S. G. O'Leary and their associates at Stony Brook University (e.g., O'Leary & Pelham, 1978; O'Leary, Pelham, Rosenbaum, & Price, 1976). Notably, however, the majority of programs and intervention studies were aimed at elementary school-age children with ADHD. In fact, in the Fabiano and colleagues (2009) meta-analysis, only a handful of studies included teenage youth with ADHD.

Barkley has conducted some of the seminal work in the area of parenting interventions for adolescents with ADHD (Barkley et al., 1992, 2001). In an initial

trial, Barkley and colleagues (1992) compared behavior management training, problem-solving communication training, and structural family therapy interventions within families of children with ADHD, ages 12–18. In this study, all interventions, in general, resulted in improvements at posttreatment and at follow-up assessments 3 months later. In this study, clinically significant change (e.g., normalization of functioning) occurred in only a minority of cases, however. Thus, there was evidence of improvement in all groups following the intervention, but the magnitude of change was not sufficient to bring parent–teen interactions and youth behavior into a normative range of function. Barkley and colleagues (2001) replicated and extended the earlier study by comparing behavior management training with problem-solving communication training, and also combining the two interventions (and investigating the sequencing of the combination). In this study, both treatments again resulted in improvements within families. Interestingly, dropout was greater when problem-solving communications training was offered first, relative to behavior management, perhaps indicating that families were able to implement the problem-solving communication strategies only when equipped with contingency management and parenting strategies first. Taken together, these studies suggest that these intervention approaches are promising, but they also require further refinement and investigation. At the present time, BPT, problem-solving communication training, and the combination of these two interventions show promise for parents of teens with ADHD (Pelham & Fabiano, 2008). Interestingly, there were notable rates of dropout from the clinical parent–teen interventions. It may be that tying these clinical interventions to a developmental transition that is meaningful for both parents *and* teens will yield greater engagement and therefore improved outcomes.

In the STEER program, these interventions are integrated to promote parents and teens working together to achieve safe driving. While the teen meets with a paraprofessional to receive intervention components, the parent meets with a clinician to review effective parental monitoring, contingency management, and communication skills (Barkley & Robin, 2014; Barkley et al., 1992, 2001; Forgatch & Patterson, 1989; Patterson & Forgatch, 1989; Robin & Foster, 2002); this emphasis on behavior management was found to be effective and engaging for parents of teens with ADHD (Barkley et al., 2001). As noted earlier, parents may arrive at an intervention for teens with ADHD exhausted and burnt out from years of parenting a child with ADHD, and it may be difficult to reengage the parent for a driving-focused intervention. It is important to acknowledge this given that parents of teens with ADHD have likely engaged in many teacher calls and meetings, reminders to complete household chores and homework, prompts to follow house rules, and arguments over multiple issues. It is possible that the parent of a teen with ADHD has engaged in thousands, if not hundreds of thousands, more of these interactions than has the parent of a typical teen. It is no wonder that parents are exhausted and perhaps unenthusiastic about another treatment effort. There is also a risk of alienating the parent by introducing content or strategies that are familiar to them (e.g., privilege removal, praise for good behavior) as if these are novel suggestions. Thus, the initial STEER session also includes a decisional balance exercise that is consistent with motivational interviewing procedures to help parents identify goals, the benefits of changing behavior to work toward those goals, and the costs of not changing behaviors. Clinicians also explicitly state to parents that it is unlikely that the strategies introduced will be unfamiliar, and that the goal of the intervention is to partner with them to think about how to implement the strategies within the context of learning to drive and overseeing independent driving. As content is reviewed, care is made to check with parents about prior use of strategies, including how the approaches were successful or unsuccessful, and this information is then used to plan for implementation within the context of driving. Table 29.1 lists parent session content.

Parent and Teen Combined Session Portion

Behavioral contracting is a key component of the current best-practice approach to promoting safe driving in teenagers (Simons-Morton et al., 2002), and it is a component of efficacious interventions for teenagers with ADHD (Barkley et al., 1992, 2001; Robin & Foster, 2002). Following these individual meetings, the family participates for the second 45 minutes in a joint activity. In clinical practice, this could be a parent–teen negotiation and contracting session each week. During all weeks, families create or review a behavioral contract that targets issues related to driving or other areas of impairment, and the contract is typically linked to specific contingences for meeting–not meeting targeted goals. Prior work has also implemented driving practice on a driving simulator (Fabiano et al., 2011). Further-

more, parents and teens can spend time reviewing objective driving data from on-board engine performance monitors or video cameras. The timing and content of each of these joint sessions is listed in Table 29.1.

Contingency contracts created each week link objective driving behaviors to rewards and punishments (Smith et al., 1997), and this is an approach with a long history in ADHD-related treatment for teenagers (Robin & Foster, 2002). Parents and teens in the STEER program are encouraged to generate issues related to driving, to define the issue operationally and establish criteria for it, and to create a reward for facing the issue or a punishment for missing the goal. Parents and teens may also include issues and criteria peripheral to driving. For instance, for one of the cases, the teen was often noncompliant with taking stimulant medication. As part of the family's contract, the teen had to take the morning medication dose in order to use the car. Parents and the teen then agreed on how the contract would be monitored and set a date for evaluating and modifying the contract. A sample contract is presented in Figure 29.1.

During the driving simulation exercises, the teen drives and the parent rides as a passenger. The purpose of these simulations are twofold: (1) These simulations provide additional practice and experience in a safe environment for a novice driver; and (2) it is well documented that adolescents, and those with ADHD in particular, are poor evaluators of their own behavioral weaknesses (Fischer et al., 2007; Hoza et al., 2002; Knouse et al., 2005; Owens et al., 2007). Therefore, for adolescents, the simulations provide concrete behavioral evidence of driving strengths and limitations. To this end, there a number of built-in hazards to challenge the teen driver; to promote discussion between the parent, teen, and clinician; and to permit the teen to practice safe and effective driving behaviors. Following the participation in the simulator exercise, parents and the teen discuss the experience and problem-solve issues that occurred, and the parents are instructed to comment positively on safe driving behaviors exhibited by the teen.

Teens also bring the engine performance monitor to the session each week, and the therapist facilitates a re-

WRITTEN CONTRACT

Date: January 19, 2014

Issue: speeding on the highway. This is defined as a registered speed greater than 65 miles per hour at any time during the week.

Agreement: There will be no instance of recorded speed that exceeds the speed limit.

Positive consequence(s) for following agreement: If the goal is met, the car can be used to drive to a friend's house on Saturday afternoon.

Negative consequence(s) for violating agreement: If the goal is not met, driving privileges will be suspended for Saturday.

Reevaluation date: January 26, 2014

Julia Jones
Teen signature

Eileen Jones
Parent signature(s)

FIGURE 29.1. Sample teen driving contract.

view of the data generated. This review helps to model the approach parents should use to identify strengths and weaknesses of new driver's behavioral outcomes. Furthermore, it supports the teen and parents in gaining familiarity with what might be a novel technology. The review of the objective data also helps to generate ideas about potential targets for parent–teen contracts (e.g., speeding, hard braking). Finally, by integrating use of the monitoring device and the data review into the session, the clinician helps families build a habit over the eight sessions that may help to promote maintenance of this procedure over time, which is likely needed for a long-term driving intervention.

As outlined, the STEER program is intensive. It is likely that intensive and sustained procedures will be needed to best support teens and their families as they embark on an important developmental transition. Additional research is needed to investigate the STEER package, and component parts, to better inform the field on best practice for ADHD driving risk interventions.

FUTURE DIRECTIONS

Additional Study of Technological Support

As discussed earlier, technology may be a useful addition to the collection of interventions available for teenage drivers with ADHD. More work is needed on how technology might support learning in new drivers, prevent the development of poor driving habits, and perhaps prohibit dangerous behaviors (e.g., cell-phone jamming technologies to prevent texting while the car is in motion). Furthermore, technologies such as simulators may effectively promote training of both teen drivers and the parents/instructors who will be teaching them. Additional technology-supported activities, such as the review of driving data (video or graphical), may also be useful for supporting drivers with ADHD. Additional research to explore these novel and largely untested approaches for individuals with ADHD is an important area of future research study.

Additional Study of Parent Support

There are a number of venues through which parents influence teens' development as drivers. Parents may play a role in deciding when teens may initiate learning to drive. They are often involved in some aspects of teaching their teens to drive and deciding when teens are ready to obtain drivers licenses. Once teens are able to drive independently, parents may continue to be involved by monitoring teen driving and setting limits and expectations for driving. Unfortunately, there is currently little information available to parents regarding how they might most effectively promote safe driving habits in their teens with ADHD, particularly with regard to specific things parents should do when teaching their teens to drive.

As illustrated by Barkley and Cox (2007), driving is a complex task involving multiple dimensions of competency (e.g., operational, tactical, and strategic), and the task of teaching a teen with ADHD to drive is likely even more complicated. During parent-supervised driving practice, parents are typically responsible for choosing when, where, and how driving practice should occur. They are also tasked with establishing an appropriate progression of driving practice and instruction, beginning with safe, basic situations (e.g., driving in an empty parking lot), then transitioning to more challenging situations (e.g., driving in traffic) as they deem their teen to be ready. Across all families of teen drivers, there is likely considerable individual variability in the quality of parents' driving instruction, with some parents doing a very good job of teaching their teens to drive and others using less effective or even detrimental strategies.

Given that parents of children with disruptive behavior may grow weary of parenting over time and engage in less effective monitoring and supervision as children enter adolescence (Dishion et al., 2004), they may be less effective when teaching their teens to drive than other parents. Recent findings from a study involving observations of parent-supervised driving practice for teens with ADHD suggests that parents may not use effective strategies during driving practice. In this study, on-board video monitors were used to record observations of parents and teens during supervised driving practice. The monitors sampled 40-second periods of drives, yielding multiple videos for each family. According to these video observations, parents rarely provided positive feedback to teens and provided absolutely no driving-related instruction during approximately half of the videos. Of note, this study employed a relatively small sample and did not include a comparison group; however, these data begin to identify potential targets for intervention in families of teens with ADHD. More work is needed to identify which specific parenting behaviors during supervised driving practice contribute to safer driving outcomes for teens with ADHD.

Additional Study of Policy Support

As GDL approaches are used more widely, the study of how they work for special groups, such as youth with ADHD, need additional investigation. Furthermore, the interface between psychosocial and medication interventions for ADHD and policy need to be explored further. For example, as driver education appears insufficient as currently constructed in improving functioning for many youth with ADHD, it may not be reasonable or in the teen's best interest to provide "time discounts" in GDL frameworks for driver education completion. The Molina and colleagues (2009) study illustrating delays in licensure also may suggest that youth with ADHD are not maximally benefiting from GDL, an observation that warrants further empirical study. Currently, policy does not address ADHD specifically, and professional organizations (e.g., psychology or pediatric guilds) may help the field by creating practice guidelines for promoting the effective negotiation of this developmental transition.

CONCLUSION

ADHD is a chronic, developmental disorder that begins in early in childhood and often persists through adolescence and into adulthood. The past decade has generated an increase in novel research related to ADHD driving risks and potential promising interventions. This foundation of research needs to be leveraged to spur the next generation of research that will evaluate and disseminate innovative approaches for promoting safe driving in teens with ADHD. Given the high rates of morbidity and mortality associated with risky driving, this is an area of profound public health significance. It is hoped that federal funders, policymakers, treatment providers, and clinicians will attend to this important domain of functional impairment in youth with ADHD, with an eye toward making the roadways safer for everyone.

KEY CLINICAL POINTS

- ✓ Driving is an important domain of activity in which most U.S. teens and adults engage. It can also be a life-threatening one for both the driver and others.
- ✓ Adolescence is the time of highest risk for driving. Evidence clearly demonstrates that ADHD in a teenager raises these normal risks for adverse outcomes considerably (see Chapter 11).
- ✓ Adolescents with ADHD are more likely to drive before getting a license, to employ less safe driving behavior habits, to engage in greater risk-taking and other impulsive behavior, to be more inattentive and distractible while driving and less likely to obey rules, and to manifest higher levels of road rage or the aggressive use of a motor vehicle. Teens with ADHD may also be more likely to engage in substance use, especially if they have comorbid CD. As a consequence, they are more likely to experience adverse outcomes, such as more citations, especially for speeding; to have more accidents and worse accidents, as reflected in dollar damage and bodily injury; and are more likely to have their license suspended or revoked.
- ✓ For these reasons, there is a clear need to develop interventions that target this domain of impairment.
- ✓ Typical interventions to reduce driving risks for youth include official GDL programs in most states, driver education classes, and the Checkpoints program for teens and their parents. Yet these interventions are unlikely to target the specific driving-related problems posed by ADHD, and they may be less effective in reducing the driving risks for teens with ADHD.
- ✓ Assessing the driving behavior of teens with ADHD may include the use of driving behavior rating scales completed by teens and parents, review of driving history, review of existing DMV records, on-road driving examinations, and, where available, use of driving simulators. New technologies for monitoring actual driving, such as on-board vehicle performance monitors and solid-state cameras may also prove informative.
- ✓ Interventions for driving-risk reduction for teens with ADHD include using ADHD medications, technology (performance monitors, cameras, simulators) to provide feedback to teens and parents, implementation of contingency management methods targeting safe driving behavior, behavior contracting, and greater parental supervision of vehicle use, among others.
- ✓ The chapter also presents a combination of these methods in a parent–teen treatment program (STEER) that offers considerable promise for improving the driving behavior and related risks for adverse outcomes in teens with ADHD.

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CHAPTER 30

Complementary and Alternative Medicine for ADHD

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The term *complementary and alternative medicine* (CAM) encompasses a wide variety of interventions to treat various illnesses or improve health, distinct from the conventional medicines and standard care practices used by medical professionals. *Complementary medicine* refers to interventions used in tandem with standard medical care, while *alternative medicine* describes interventions used in place of standard medical care. However, despite the inclusion of the word *medicine* in CAM, it is important to note that most approaches defined under this umbrella term do not involve medicine of any sort.

In recent years, with the influence of the popular media and the Internet, the proliferation of information about CAM treatments has increased exponentially, despite the lack of a strong evidence base to recommend their use. It was reported in 2007 that 38% of U.S. adults age 18 years and over and 12% of U.S. children use some form of CAM (Barnes, Bloom, & Nahin, 2008). CAM treatments have been used for many conditions—most widely for headaches, asthma, eczema, colic, chemotherapy-induced nausea and vomiting, pain, autism, and attention-deficit/hyperactivity disorder (ADHD) (Snyder & Brown, 2012).

Although stimulant medication and behavioral therapy are the most widely recommended evidence-based treatments for ADHD, many factors may lead individuals with ADHD or affected families to seek complementary and alternative medicine in order to treat and/or alleviate symptoms. In some cases, psychopharmacological treatment is associated with a social stigma. In other cases, lack of efficacy or unrealistic fears about side effects may induce some individuals to pursue CAM therapies in place of medication. In terms of behavior therapy, logistical obstacles (including limitations with respect to time, cost, and availability of professional resources) make it difficult for many families to pursue psychological counseling. Although some CAM interventions may be quite expensive, with limited availability, other CAM approaches may be more affordable and more accessible to families. Of course, in many cases, CAM therapies appeal to consumers because they are presented as being more natural or safer.

Surveys suggest that 12 to 64% of children and adults diagnosed with ADHD use CAM alone or adjunctively with medication and/or behavioral therapy (Russing, Zima, Gary, & Garvan, 2002). In addition, over half of parents of children with ADHD treat their children's symptoms with one or more CAM treatment, yet only 10% of parents disclose this to their pediatrician (Chan, Rappaport, & Kemper, 2003). Huang, Seshadri, Matthews, and Ostfeld (2013) also found that parents

of children with ADHD and autism spectrum disorder do not believe their primary care physicians are knowledgeable and/or interested in CAM therapies.

While some of the most widely used CAM treatments have already been outlined in this book, adults with ADHD and parents of children with ADHD may be interested in pursuing less common CAM interventions. These include mind–body therapies, chiropractic techniques, physical activity, acupuncture, occupational therapy, caffeine, and noninvasive brain stimulation. Unfortunately, few of these CAM therapies have been subjected to rigorous experimental investigations; therefore, therapeutic efficacy for these treatments cannot be adequately evaluated.

MIND–BODY THERAPIES

Yoga

As a CAM therapy, yoga focuses on redirecting attention to physical sensations during each posture to improve attention, self-awareness, and physical self-control (Brown & Gerbarg, 2012; Searight, Robertson, Smith, Perkins, & Searight, 2012). The respiratory training component attempts to reduce sympathetic nervous system activity while enhancing focus. Yoga may also include postural exercise and mental visualization practices, in addition to the breathing exercises. Yoga is said to impact oxygen consumption, brain function, and cognition, all of which may potentially be impaired in people with ADHD (Jensen & Kenny, 2004). Jensen and Kenny (2004) suggest there may be some added reduction of ADHD symptoms when yoga is practiced in tandem with taking medication. Haffner, Roos, Goldstein, Parzer, and Resch (2006) found that yoga is more effective in reducing ADHD symptoms than aerobic exercise, which may be due to the enhanced attentiveness required for yoga. Other researchers have even looked at tai chi, which involves slow, flowing movements similar to yoga, and found that it can help reduce hyperactivity and anxiety in adolescents with ADHD. However, these limited studies lack large sample size and adequate control groups, and have only been performed with child and adolescent ADHD populations (Field, 2012; Hernandez-Reif, Field, & Thimas, 2001). Of interest, Harrison, Manocha, and Rubia (2004) found that parents who participated in yoga with their child reported a reduction in stress and an enhanced ability to manage their child with ADHD.

Massage

Massage therapy, like yoga, has also been poorly studied as an alternative treatment option. However, it has been theorized that both massage and vestibular stimulation (which targets sensors in the inner ear) work to influence electroencephalographic (EEG) patterns associated with attention and vagus nerve input (Brown, Gerbarg, & Muskin, 2009; Field, Pickens, Fox, Nawrocki, & Gonzalez, 1995; Field, Quintino, Hernandez-Reif, & Koslovsky, 1998; Khilnani, Field, Hernandez-Reif, & Schanberg, 2003; Suess, Porges, Plude, 1994). They may also help to increase motor inhibition as a result of increased vagal control. Most studies involving massage therapy have used adolescent ADHD populations and found improved mood, teacher-reported classroom behavior, and attention to task soon after the intervention was performed. However, these effects were not predicted to persist in the long term, and the studies were, again, limited in sample size and population representation.

More recently, there has been discussion surrounding another form of massage called craniosacral therapy (CST). The craniosacral system of the body comprises membranes and cerebrospinal fluid that surround and protect the brain and spinal cord. According to Jäkel and von Hauenschild (2012), the craniosacral system must remain mobile in order to function properly because it is connected to many other systems throughout the body. One such type of mobility is the primary respiratory mechanism, which is the "palpable motion" of the cranial bones, central nervous system, and cerebrospinal fluid. Therefore, when mobility is restricted, trained practitioners use CST to correct these abnormal restrictions and restore mobility.

CST involves the release of tension via soft, therapeutic touch of the face, spine, skull, and pelvis in order to improve the function of the craniosacral system and, subsequently, the central nervous system. As a result of these therapeutic palpations, CST is believed to affect sensory, motor, cognitive, and emotional processes of the nervous system. Its proponents therefore claim it can address and alleviate various neurological impairments, including attention deficit disorder (ADD) and ADHD. Although no studies have specifically addressed CST as a treatment for ADHD, some systematic reviews have looked at the validity and health outcomes of CST as an intervention for other disorders. One such review (Green, Martin, Bassett, & Kazanjian, 1999) stated that research studies involving CST

was inadequate and therefore provided insufficient evidence to support its use. However, a more recent review from 2012 concluded that although research in this area is limited, randomized controlled trials and observational studies have found CST is effective in reducing pain and improving the well-being of both children and adults with different clinical disorders (Jakel & von Hauenschild, 2012). However, until rigorous research in CST addresses the treatment of ADHD and other neurological disorders, this approach cannot be recommended for use in children and adults.

Meditation

Meditation involves emptying the mind of all thoughts, focusing on single images or words, and shifting one's attention from one visual figure to the next. It is believed not only to calm, balance, and strengthen the nervous system, but it may also affect EEG rhythms, similar to biofeedback training (Brown & Gerbarg, 2012; Brown et al., 2009). However, meditative practices may be quite difficult for those suffering from ADHD because most of them find it difficult to relax and concentrate, especially children. Researchers recommend that children and adults with ADHD practice quieting their mind through yoga breathing before attempting true meditative exercises. Alternatively, as a substitute, meditation exercises for adults and children with ADHD may involve the display of images in sequence, presented by a teacher or compact disc (or the parent of a child with ADHD), which allows those with ADHD to develop the ability to shift their focus of attention in a more dynamic and kid-friendly fashion. Another form of meditation called open-focus meditation uses a series of mental exercises to help both adults and children develop the ability to shift between narrow and wide foci of attention (Fehmi & Robbins, 2007).

A Cochrane review published in 2010 (Krisanaprakornkit, Ngamjarus, Witoonchart, & Piyavhatkul, 2010) looked at four studies involving both mind-based and physical-based meditation as treatment options for ADHD. They defined *mind-based meditation* as that which emphasizes focused attention, whereas *physical-based meditation* emphasizes behavioral control. Only one of these trials found that meditation was as effective as drug or standard therapy based on teacher ratings of 17 school-age subjects. Despite this finding, the Cochrane review concluded that given the limited research and the inconsistent methodology, flawed rating scales, and small sample sizes, meditation does not show any effect on core ADHD symptoms. In addition, meditation research has mainly focused its efforts toward children and adolescents with ADHD; hence, this warrants further study in adult ADHD populations (Brown & Gerbarg, 2012; Krisanaprakornkit et al., 2010; Searight et al., 2012; Skokauskas, McNicholas, Masaud, & Frodl, 2011).

Homeopathy

Homeopathy is an alternative medicine based on the concept that "like cures like." This means that treatment of a specific illness should be done with a substance that provides similar symptoms in a reduced or diluted form. Homeopathic medicine is considered a holistic approach to medicine, in which treatment is often individualized and based on a patient's specific experience with the disease and personality characteristics. A Cochrane article considered four studies looking closely at homeopathic treatment in children with ADHD (Heirs & Dean, 2007). While three of the studies were looking at the change in symptoms during the homeopathic regimen, the fourth study examined the maintenance of previously achieved improvements due to homeopathic intervention. This meta-analysis found no evidence that homeopathy was effective in reducing symptoms such as inattention, hyperactivity, and impulsivity. Furthermore, the selected studies demonstrated use of both individualized and standardized homeopathic formula, yet neither approach showed improvement in the various ADHD scales/measures (Heirs & Dean, 2007). Researchers in this field have also acknowledged that studies of this nature are inherently inadequate, stating that an average of 6.5 months of treatment is required in order to see any effect. However, most research trials of homeopathy have only lasted 4.5 months or less, which is not suitable for these types of studies (Frei et al., 2005; Frei, Thurneysen, & Von Ammon, 2006).

In 2011, a Cochrane review was published regarding the efficacy of yoga, meditation, and homeopathy as CAM therapies for children and adolescents with ADHD (Foisy & Williams, 2011). The authors stated that research in these areas is of poor quality and lack adequately randomized controlled trials. Additionally, the different rating scales of outcomes provided inconsistent results within the same intervention. The authors concluded that research involving these treatment options is insufficient and cannot be used as evidence to support any reported benefits of these in-

terventions; thus, they could not recommend their use for individuals with ADHD (Foisy & Williams, 2011).

CHIROPRACTIC INTERVENTIONS

Chiropractic intervention is described as joint adjustment and manipulation of the neuromusculoskeletal system (World Health Organization, 2005). This is another type of mind–body therapy; therefore, parents should be wary of this intervention because there have been few studies to support its benefits to children with ADHD. One such study that reported benefits of spinal manipulation therapy (Alcantara & Davis, 2010) was limited by sample size (four patients) and sample population (18 years or younger). In fact, most studies concerning chiropractic interventions are subject to bias because they are conducted by chiropractors themselves (Alcantara & Davis, 2010).

Applied kinesiology, which is a chiropractic technique that is thought to treat various illnesses by evaluating muscle strength, has also been considered as an alternative therapy for children with ADHD. One case series reported enhanced cognitive performance on various psychometric tests in children with development delay after the administration of chiropractic sessions (Galicia-Connolly, Shamseer, & Vohra, 2011). Nevertheless, the various experimental design flaws hinder any legitimacy of the results and conclusions (Galicia-Connolly et al., 2011).

A placebo-controlled, double-blind, randomized clinical trial in Australia studied the use of the Neuro Emotional Technique (NET), a type of chiropractic approach, in children with ADHD (Karpouzis, Pollard, & Bonello, 2009). NET looks to address the "biopsychosocial aspects of acute and chronic conditions" by using "manual muscle testing to evaluate structural, emotional and chemical aspects of a patient's health" (pp. 2, 4). In this parallel-design clinical trial investigating NET as an adjunct to standard therapy (medication or psychotherapy), subjects were assigned to one of three treatment groups: standard therapy alone, standard therapy with NET, and standard therapy with a sham NET regimen. Rating scales and DSM-IV symptom checklists were used to measure data throughout the course of the 7-month study. Significant improvement was noted on Conners' Parent Rating Scales; however, no improvement was noted by teachers on these scales. The authors attribute these negative teacher findings to the fact that, given the length of the intervention and other factors, the teacher making the assessment at 7 months was not the baseline teacher in more than 50% of cases. In addition to lack of stability of teacher rating, other major limitations of this study included a high subject attrition rate (39% of enrolled subjects) and nonblinding of the therapists. For these reasons, the authors conclude that the results are "suggestive of efficacy" but that "definitive recommendations cannot be made about the intervention at this time until all aspects of NET (i.e., emotional, chemical, and physical/structural) have been trialled in order to test the safety and effectiveness of NET therapy as a whole" (Karpouzis, Bonello, Pollard, & Graham, 2011, p. 36).

EXERCISE AND GREEN SPACE

Exercise, in general, has a wide range of health benefits and should be incorporated into both children's and adults' everyday routines because it improves cardiovascular health, boosts energy, combats obesity, improves mood, promotes better sleep patterns, and so forth. Aside from these reasons, recent literature suggests that physical activity may also affect brain function and structure, promoting neural growth and development, favorably affecting cognitive and inhibitory control in children, and perhaps improving ADHD symptoms (Berwid & Halperin, 2012). A review of studies from the 1980s to 2010 found modest beneficial effects of exercise on ADHD symptoms and recommends it as a supplement to medication (Gapin, Labban, & Etnier, 2011). In addition, five subsequently published studies, either correlational or experimental pilot studies, have examined the effects of exercise on ADHD in school-age children. Taken together, these studies demonstrate some support for the improvement of both behavioral and neuropsychological functions in children with ADHD. However, each was limited by small sample size, nonblinded status of the researchers, and poorly designed control conditions (Berwid & Halperin, 2012; Chang, Liu, Yu, & Lee, 2012; Gapin & Etnier, 2010; Medina et al., 2010; Smith et al., 2013; Verret, Guay, Berthiaume, Gardiner, & Beliveau, 2012).

Recently, research regarding the benefits of exercise has had policy implications relative to school systems looking to limit physical education programs due to budgetary and standardized testing concerns. These actions are being widely contested by researchers in this field, who understand the importance of exercise in school-age children. In addition, they argue that the

increased incidence of ADHD diagnoses in the United States may be a secondary consequence of the elimination of gym classes and recess. In fact, in December 2013, the American Academy of Pediatrics (AAP) released a statement about the crucial role of recess for school-age children. Not only does physical activity promote cognitive, social, emotional, and physical benefits to a child, but recess also provides a much-needed break from the rigors of academics. Pontifex, Saliba, Raine, Picchietti, and Hillman (2013) recently published a study in which 40 elementary school-age children, half of whom had ADHD, spent 20 minutes a day either running on the treadmill or quietly reading. The children who exercised performed better on math and reading comprehension tests, whether or not they had ADHD. In light of this study and pending more research in this area, there is an increasing empirical basis to suggest the benefit of having mandated physical education classes for all children during the school day.

Green space, a term used to describe exposure to natural green settings, such as trees and grass, is based on a theory of attention restoration. It is thought that different environments may have different effects on attention, and that outdoor environments with green space may have the power to restore voluntary attention by removing individuals from places that require more effortful forms of attention, such as a classroom. Because this is a fairly new approach, there are very few studies to assess its effectiveness. However, one single-blind controlled trial found that children with ADHD performed better on a verbal task that measured concentration after taking a walk in the park as opposed to a city setting, reporting an effect size equivalent to that of a pharmacological intervention. Nevertheless, one study is an inadequate recommendation of such an intervention, and further research is necessary (Faber Taylor & Kuo, 2009; Kuo & Faber Taylor, 2004).

Researchers have also looked at the relationship between climate and ADHD. Arns, van der Heijden, Arnold, and Kenemans (2013) compared the prevalence of ADHD across the United States to the intensity of sunlight those regions receive. They found that the sunnier U.S. regions had much lower incidence of ADHD (6–8%) compared to darker regions (10–14%). The authors suggested a few theories to explain this relationship, the most compelling of which proposing that sunlight can correct the damage to circadian rhythms and melatonin levels in children due to modern technology and media. The authors hypothesize that the use of iPads, mobile phones, and TV late at night may delay sleep onset, suppress melatonin levels, and shorten sleep duration, which would increase prevalence of ADHD. However, intense natural light can counteract these harmful effects; therefore, those children in sunnier areas are able to combat the negative effects of nighttime technology.

ACUPUNCTURE

"Acupuncture," which involves pricking the skin with needles and is thought to alleviate pain or treat various physical mental and emotional conditions, has also been considered as an alternative treatment option for ADHD. However, like most other mind–body therapies for ADHD, research surrounding acupuncture and electroacupuncture is limited. A systematic review and meta-analysis (Lee, Choi, Kim, Kim, & Ernst, 2011) looked at acupuncture as a CAM therapy for ADHD. Of the 114 articles reviewed, only three randomized controlled trials met the authors' inclusion criteria. All three studies used either child or adolescent ADHD populations, one of which looked specifically at electroacupuncture and had both a large sample size and adequate blinding. Due to the varying reports of benefit, the authors of this meta-analysis concluded that there is limited evidence to support the use of acupuncture in people with ADHD. Moreover, the review article concludes that because the risk of bias in the included studies was high, firm conclusions cannot be drawn. It should also be noted that children may be resistant to cooperating in acupuncture treatments; therefore, this intervention may be better suited for adults with ADHD, although no such studies have been performed (Lee et al., 2011; Skokauskas et al., 2011).

OCCUPATIONAL THERAPY INTERVENTIONS

According to the American Occupational Therapy Association (AOTA), the goal of occupational therapy is to foster the development or recovery of certain living skills of individuals with physical, mental, or developmental conditions.

Various occupational therapy (OT) interventions have also been considered for alternative treatment of ADHD, including sensory integration therapy and utilization of the interactive metronome. Sensory integration therapy, which looks to improve the body's ability to process sensory input provided by visual and auditory

sources, encompasses different techniques and products that may be used to organize and strengthen sensory systems. OT interventions, in general, are mainly used for treatment of ADHD in children, specifically those believed to have difficulty tolerating or processing sensory information due to immature or damaged sensory systems that cause overstimulation of the brain. On the other hand, sensory deficits may also be indicative of sensory processing disorder (SPD), which may or may not be associated with ADHD. However, there has yet to be a uniform consensus on how SPDs are defined; therefore, sensory integration therapies have been the subject of considerable controversy and criticism. In 2012, the AAP released a policy statement addressing the use of these techniques and recommending caution: "Parents should be informed that the amount of research regarding sensory integration therapy is limited and inconclusive" (p. 1188). It stated that OT interventions can be used as a component of a comprehensive treatment plan but should not be the sole form of therapy for children with developmental and behavioral disorders.

Two popular sensory integration therapy techniques have been evaluated in children with ADHD. These two products, the weighted vest and stability balls, work to enhance attention and focus, and decrease hyperactivity. The weighted vest is thought to improve attention and behavior by providing deep pressure stimulation throughout the body, which in turn facilitates the production of neurotransmitters that work to improve function and performance by changing the activity of the central nervous system. A small study (VandenBerg, 2001) found positive results with the use of weighted vests in children with ADHD, but only four subjects were recruited, and the intervention was brief, with no follow-up. More recently, Collins and Dworkin (2011) conducted a pilot study with a larger sample size and sham control group; they found no benefit with this intervention in school-age children. It is remarkable that such an intervention can have national "legitimacy" in the eyes of occupational therapists, with such scant research to support its use. "Stability balls," which are large, rubber inflated balls used to replace regular chairs in classrooms, have also been studied (albeit scarcely) and were found to have positive effects on children's in-class behavior. A study by Fedewa and Erwin (2011) indicated that the use of stability balls helped improve attention, participation, and behavior in a classroom setting for children with and without ADHD. However, there were limitations of this study, many of which were noted by the authors.

The interactive metronome, on the other hand, a computer-based version of the traditional music metronome, is theorized to improve deficits of neural timing between regions of the brain. Research in this area is mixed. Shaffer and colleagues (2012) found that boys who received the interactive metronome intervention improved in every measure (e.g., attention, language processing, and ability to regulate aggression) compared to boys receiving video game treatment or no treatment at all. On the other hand, Cosper, Lee, Peters, and Bishop (2009) concluded that after a 15-week intervention with 12 children, the interactive metronome training did not affect sustained attention or motor inhibition. In general, like sensory integration therapies, the research dedicated to this technique (including these two studies) suffers from bias, small sample size, and unrepresentative population. Thus, there is insufficient research to recommend use of an interactive metronome for children with ADHD.

CAFFEINE

Not quite a dietary supplement, caffeine has also been considered as a CAM treatment for children with ADHD. Caffeine is derived from methylxanthine, which functions as a mild central nervous system stimulant. Barry and colleagues (2005, 2009) have conducted multiple studies concerning caffeine's effects on arousal in both children and adults, concluding that in healthy individuals, caffeine does promote arousal, as seen through the use of EKG and skin conductance levels. In addition, in 2012, Barry and colleagues hypothesized that children with ADHD experience more "hypoarousal" than do gender-matched controls; therefore, caffeine may help to increase their arousal levels. While they found no difference in arousal level increased between children with ADHD and their matched controls, preliminary analyses revealed that caffeine-induced arousal increases in children with ADHD were positively related to the severity of their symptoms (Barry et al., 2012). Most other research concerning caffeine as a CAM treatment for ADHD is dated and sparse. Leon (2000) reviewed studies using children with ADHD and concluded that caffeine was more effective than no treatment in reducing ADHD symptoms, but it was not nearly as effective as stimulant medications. Little research has been performed to measure the effectiveness of caffeine as a treatment in adults with ADHD. Yet Liu, Liang, and Kuang (2010)

hypothesized that due to poor treatment compliance, many adults with ADHD still experience residual symptoms; thus, perhaps caffeinated tea could be a viable and effective treatment for ADHD.

Although consumed frequently by adults and increasingly by youth, caffeine is also associated with some adverse effects, especially when it is consumed in increased amounts. Coupled with its stimulating properties, caffeine also works to oppose the actions of sleep-inducing adenosine in order to increase alertness. Sleep is important for cognitive performance and learning; thus, sleep deprivation can negatively impact cognitive, emotional, and physical functioning. Therefore, sleep disturbances due to caffeine treatment in both children and adults with ADHD could essentially intensify hyperactivity and inattention symptoms.

Nonetheless, the relationship between ADHD and sleep, in general, is quite complicated because it is both multifaceted and multidirectional. It has been reported that 35–50% of children and more than 50% of adults with ADHD suffer from sleep problems (Dobson & Zhang, 1999; Hvolby, Jorgensen, & Bilenberg, 2008). Researchers in this field have proposed and studied different theories regarding ADHD and sleep problems in children. Some believe that sleep deficiencies due to sleep disorders/disturbances likely result in the appearance of ADHD-like symptoms during daytime hours. Others think that certain ADHD symptoms contribute to sleeplessness and, as a result, exacerbate ADHD behaviors of the children. The origin of these sleep disorders/disturbance is also widely debated, as researchers are unsure whether they are comorbid with ADHD or completely independent. It is also possible that these sleep problems in children with ADHD are caused by intrinsic properties of ADHD, such as altered sleep patterns and architecture. This ambiguity lends itself to many problems, such as misdiagnosis of ADHD, when the problem is actually a sleep disorder. Conversely, the complexity of this relationship has also led to a vast amount of research in this field, providing a variety of findings; however, despite this, the topic remains obscure and research should continue (Yoon, Jain, & Shapiro, 2011).

Although studies have repeatedly indicated that caffeine is not as effective as psychostimulant medication in treating ADHD symptoms, some investigators have speculated that caffeine may be a helpful adjunct to stimulant treatment. There is no convincing research to recommend this to patients. To the extent that stimulants and caffeine have similar side effect profiles, it is likely most prudent for adults with ADHD and parents of children/adolescents with ADHD to moderate caffeine consumption if stimulants are being prescribed. Vigilance regarding caffeine consumption is even more important in recent years; energy drinks containing alarming amounts of caffeine have become readily available to the public and are marketed to youth.

NONINVASIVE BRAIN STIMULATION

Noninvasive brain stimulation is a relatively new branch of neuroscience that focuses solely on the external (surface) stimulation of the brain using electromagnetic means. The few different forms of noninvasive stimulation vary in how they manipulate the electromagnetic current and the size of their target area of stimulation. So far, only two types of stimulation—transcranial magnetic stimulation (TMS) and cranial electrotherapy stimulation (CES)—have been considered for the treatment of ADHD. TMS uses a weak, small-pulsed electric current to stimulate specific regions of the brain in order to increase or repair their functioning. Researchers speculate that TMS may be able to relieve certain ADHD symptoms by inducing neurotransmitter release in deficit areas of the brain. TMS is a fairly new technique; thus, only a few pilot studies have looked at it relative to treatment of ADHD. Bloch and colleagues (2010) concluded that while TMS is a safe procedure, no treatment-specific benefits were noted in children with ADHD. Therefore, until there is more research, TMS cannot be recommended as an alternative treatment for ADHD in children (Bloch et al., 2010; Weaver et al., 2008).

CES also uses low, small-pulsed currents, but unlike TMS, it has the ability to stimulate multiple parts of the brain simultaneously. CES is hypothesized to improve attention, reduce anxiety, and relieve insomnia by increasing levels of neurotransmitters that are crucial for arousal, attention, learning, and memory. The mechanism by which this occurs is unknown, and CES research remains sparse. Although some clinical reports of benefit regarding CES as a treatment for ADHD are starting to surface, no controlled studies have been published to date (Brown & Gerbarg, 2012).

ANTHROPOSOPHICAL THERAPY

Anthroposophical medicine focuses on the maintenance of internal equilibrium between the "nerve

sense system" and the "metabolic–limb system." Anthroposophical therapy views ADHD as an imbalance of this equilibrium and works to correct it via various therapeutic techniques and alternative medications. These therapies—including forms of exercise, art, and massage—concentrate on the rhythm, movement, and synchronization of the body as means to obtain equilibrium. Natural anthroposophical medications may also be used; these are prepared from plants, minerals, and/or animals in diluted or concentrated forms. Anthroposophical professionals provide personal consultations for their patients in order to individualize and optimize the treatment course (Soldner & Stellmann, 2007). Like CST, anthroposophical medicine is widely critiqued and characterized by many as a pseudoscience.

There are no randomized controlled trials of anthroposophical medicine. One recent prospective open-label study investigated the effectiveness of anthroposophical medications and therapies in children with ADHD (Hamre et al., 2010). Subjects engaged in "eurhythmy" (movement) therapy and artistic therapies, and/or used anthroposophical medications over a 2-year time period. All data was collected via questionnaires that asked parents to report quantity and severity of ADHD symptoms after all therapies and medications were completed. Results showed an overall reduction in ADHD symptoms of participants in the months following treatment. However, lack of a comparison group, blinding, and random assignment are just some of the methodological limitations that preclude acceptance of anthroposophical medicine as an evidence-based intervention.

CONCLUSION

Mainstream clinicians who care for children, adolescents, and/or adults with ADHD strive to provide evidence-based treatment. Although there is research to suggest that some discreet CAM treatments (e.g., omega-3 fatty acids) may play a modest role as part of a multimodal integrative treatment approach, the reality is that most CAM treatments have not been adequately investigated in a methodologically rigorous manner. Instead, CAM proponents generally rely on anecdotal reports, small case series, or research studies with design flaws that impugn, or at least undermine, the generalizability of their findings. Although some might suggest that the lack of scientifically sound experimental evidence for a CAM treatment approach does not preclude the possibility that it is clinically effective, this argument would suggest clinicians take a *laissez-faire* approach in counseling patients about treatment options and no longer limit their recommendations to evidence-based strategies.

One of the great clinical challenges in caring for individuals with ADHD centers around CAM treatments. Professionals who are patently dismissive of CAM treatments will likely alienate those individuals most in need of counseling about these treatments. Since most research relating to CAM treatments is not published in the professional journals that are commonly read by mainstream providers, many clinicians may not feel comfortable advising patients or clients about CAM treatments. Realistically, it takes a concerted effort and focused initiative to be knowledgeable about and remain up to date on CAM research. (Even we were surprised at how many studies and review articles have been published since we coauthored a review article on this same topic [Bader & Adesman, 2012].)

These challenges and limitations notwithstanding, clinicians must "familiarize themselves with research regarding all available treatment options in order to make educated and informed recommendations" (Bader & Adesman, 2012, p. 768) to those who seek their advice. Just as we noted in 2012, when it comes to CAM therapies for ADHD, clinicians "must maintain the proper balance of compassion and respect for their patients while educating themselves about the most recent research in this area so that they can provide their patients with the wise counsel and clinical perspective that they deserve" (p. 768).

KEY CLINICAL POINTS

- ✓ CAMs are distinct from standard medical treatments for ADHD and may either be used alongside them or as alternatives to those standard remedies. CAM treatments have been quite popular among parents of children with ADHD and adult patients with the disorder, despite the lack of a strong evidence base for their effectiveness. Up to 64% or more individuals with ADHD have tried one form of CAM or another for management of the disorder.
- ✓ Mind–body therapies involve treatments such as yoga, tai chi, massage, chiropractic manipulations, meditation, and homeopathy. Very few studies have exam-

ined the effectiveness of any of these treatments, and the quality of study designs is often low and lacks the crucial ingredients of randomization to treatment groups, inclusion of adequate placebo, sham, or alternative treatments, and blinded assessment of the impact on symptoms. Others, such as CST, have not been studied at all. Some positive results for some of these approaches have been reported in a few publications, yet just as many report no significant results. More rigorous studies are needed to fully evaluate the effectiveness of these treatments.

- ✓ Exercise and "green space" therapy (exposure to natural green grass and tree settings or activity out of doors in park-like environments) have recently found favor among trade writers. There is some support for the value of routine exercise in reducing symptoms of the disorder but here, again, study designs are typically of low quality. Just one study to date has examined "green space" therapy with promising results, but it was neither fully blinded nor did it evaluate the impact on ADHD symptoms, so much more research on this intervention is needed.
- ✓ Acupuncture for treatment of ADHD has been evaluated in over 100 different articles but only three of these involved randomized trials. A meta-analysis concluded that the poor quality of most reports on this treatment limits any conclusions from the evidence available and means that much of it is likely biased.
- ✓ Several forms of OT treatments have been recommended for treating ADHD, such as sensory integration training or the interactive metronome. The evidence based for sensory integration training is of poor quality and the findings are mixed, so use of these approaches cannot be recommended at this time despite their considerable popularity among occupational therapists who may view these as yet unproven treatments as standard therapies. Only a few studies of the interactive metronome treatment have been undertaken, and these involve small samples, poor quality designs, unblinded or incompletely blinded assessments of treatment effects, and other study design flaws. Results of the two studies are contradictory.
- ✓ Caffeine has been suggested as a CAM for ADHD but the evidence base is limited, dated, of poor quality, and therefore too inadequate to merit recommendation.
- ✓ TMS and CES, two recently developed approaches for implementing noninvasive brain stimulation, are conjectured to possibly benefit ADHD. Only small pilot studies or clinical reports of these approaches exist, and they do not suggest much, if any, benefit for management of the disorder.
- ✓ Anthroposophical therapy involves using natural plant preparations and special movements that serve to improve and maintain "internal equilibrium." Evidence for the effectiveness of this treatment for ADHD is lacking, and it is widely critiqued as being pseudoscientific.
- ✓ Despite limited, weak, contradictory, and even nonexistent evidence for these CAMs, clinicians must maintain the proper balance of compassion and respect for their patients, while educating themselves about the most recent research in this area, so that they can provide their patients with the wise counsel and clinical perspective they deserve.

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PART IV

Treatment of Adults with ADHD

CHAPTER 31

Psychological Counseling of Adults with ADHD

Kevin R. Murphy

Although medication (especially stimulant medication) is considered to be the "gold standard" and cornerstone of treatment for ADHD, it has become increasingly evident that most adults with ADHD need at least some counseling and additional nonmedication interventions to manage the many challenges that ADHD presents over the lifespan and to achieve and maintain an optimal long-term outcome. Sure, there are some people who may do very well with only medication treatment, but they represent only a relatively small subset of the overall ADHD population that likely has a milder variation of the disorder with fewer and milder symptoms, less or no comorbidity, and fewer impairments in major life activities than those with more severe forms of the disorder. The reality is that most adults with moderate to severe ADHD need both medication and nonmedication treatments to gain optimal control of their symptoms and their lives.

Why is this? Since the previous edition of this book, we have learned a great deal about the adverse impact that ADHD can have in a wide range of adult life domains (Barkley, Murphy, & Fischer, 2008; see Chapters 11–13). Adult ADHD was once thought to be somewhat of a benign disorder that was relatively easy to treat and not particularly impairing to most adults. In fact, only relatively recently, over the last 30 or 40 years, has adult ADHD even been considered a bona fide disorder because the prevailing belief for many decades was that children grew out of having ADHD with the onset of adolescence and puberty, and that the disorder did not even exist in adults. We now know differently. As a result of new research on adult ADHD, we are now beginning to have a much greater appreciation and respect for just how impairing and even devastating this disorder can be—especially if untreated. ADHD affects people over time and across situations in multiple areas of functioning. And as children with ADHD get older and enter adulthood, the stakes (and impairments) get much higher and more disruptive.

Before discussing the topic of counseling adults with ADHD, I want to highlight some of our research findings that support why we need counseling and other nonmedication approaches to treat adults with ADHD. Here is a brief overview of some of those findings from two large-scale research studies our research group conducted on adult ADHD (Barkley et al., 2008). More details of these studies and their findings can be found in Chapters 11–13. One of these studies (the UMASS

Study) compared large samples of clinic-referred adults diagnosed with ADHD to a large control group of adults having other disorders seen at the same clinic, and to a large community control group. The second study (the Milwaukee Longitudinal Study) followed children diagnosed with ADHD to adulthood (mean age of 27) and compared them to a community control group. We also looked at differences in outcomes between clinic-referred adults diagnosed with ADHD during adulthood versus children with the disorder who were followed until young adulthood.

IMPACT OF ADHD ON ADULT FUNCTIONING

Education

Almost one-third (32%) of the Milwaukee cohort dropped out of high school and only 5% had graduated from college at the final follow-up (mean age of 27). More adults with ADHD reported having been held back a grade, receiving special education services, being suspended or expelled from school, being diagnosed with other learning disorders, and having poorer grades and class rankings. Although the educational outcomes of the adults with ADHD in the UMASS Study were generally better (higher high school and college graduation rates) than those in the Milwaukee Study, the adults with ADHD in the UMASS study still rated themselves as being more impaired in educational settings than adults in either of the other two control groups.

Occupational Functioning

Both clinic-referred adults with ADHD and children growing up with the disorder experienced significant problems in their work histories. In the UMASS Study, adults with ADHD reported significantly greater problems than the two control groups in getting along with coworkers, being fired more frequently, exhibiting behavior problems, impulsively quitting jobs out of boredom, and being formally disciplined by supervisors. Independent ratings by supervisors were also significantly worse for the ADHD group, in that supervisors rated them as more inattentive, more impaired in performing assigned work to completion, and more frequently tardy; as having more problems with time management and performance of daily responsibilities; and as being more emotionally overreactive with anger and frustration and making impulsive/offending comments to others. The Milwaukee Study had similar results except that children growing up with ADHD had lower job status and even greater rates of being fired and formally disciplined than the clinic-referred adults in the UMASS Study.

Money Management

Adults with ADHD in both the UMASS Study and the Milwaukee Study reported significantly greater problems with managing money than either of the control groups. More specifically, adults with ADHD reported more problems with saving money, more impulsive buying, missing loan payments, exceeding credit card limits, having poor credit ratings, not saving for retirement, not paying bills on time, and having utilities shut off due to nonpayment of bills.

Driving

Clinic-referred adults from the UMASS Study compared to the community control group were more likely to have had their licenses suspended, to have crashed while driving, to have been at fault in crashes, to have driven without a valid license, and to have received citations for speeding and reckless driving. The ADHD group also had a greater number of license suspensions/revocations, more crashes and more at-fault crashes, and more speeding citations than either the clinic-referred or community control adults. Similarly, the ADHD group in the Milwaukee Study had a higher risk for frequent crashes, license suspensions/revocations, and citations for reckless driving than the control group, but the differences were less robust, most likely due to the fact that they were younger and had less driving experience than those in the UMASS Study.

Health/Lifestyle Risks

The adults with ADHD in the UMASS Study (and for the most part in the Milwaukee Study as well) had a higher percentage of individuals reporting problems with sleep, social relationships, family interactions, tobacco use, drug use, seeking medical and dental care, motor vehicle safety, work, and emotional health than adults in the community control group. Furthermore, the ADHD group in the UMASS Study also reported more problems than the clinical control group with illicit drug use, driving, and emotional health.

Sexual Behavior

The Milwaukee Study found higher levels of risky sexual behavior in the ADHD group compared to the control group. The ADHD group became sexually active (intercourse) earlier, were more likely to become pregnant (if female), or to impregnate others (if male), had more teen pregnancies, were more likely to get a sexually transmitted disease by age 21, were less likely to use contraception, and were more likely to be parents by ages 21 and 27 than members of the community control group followed over the same time period.

Comorbidity

Prior research has shown that both children and adults with ADHD are at greater risk for developing comorbid psychiatric disorders than those without ADHD, and our findings further corroborate this fact. More specifically, more than 80% of our ADHD groups had at least one other disorder, more than 50% had two other disorders, and more than 33% had at least three additional disorders—much higher percentages than in our control groups in both studies. The most common comorbidities were major depression, dysthymia, anxiety disorders, and substance abuse disorders.

With all of these potential and very real risks for those with ADHD, it seems quite clear that medication alone is not enough to counteract the many challenges and obstacles that most adults with ADHD likely encounter as they move through life. Most need treatment for other problems, in addition to ADHD, such as marriage and family counseling, vocational and financial counseling, substance abuse counseling, counseling related to lifestyle changes, and self-management/organizational skills training. Our results strongly suggest that ADHD is not just a seriously impairing disorder, it is even more impairing than other psychiatric disorders, such as depression, anxiety, and substance abuse disorders. The chronic, ongoing, and pervasive impact of ADHD over the lifespan does not usually lend itself to quick fixes or simple solutions. Not only this, but some people cannot tolerate stimulant medications, have problematic side effects, do not respond well and cannot take stimulant medications because of medical or psychiatric contraindications, or are just plain philosophically opposed to taking medication due to personal or cultural beliefs. For all of these reasons, we need counseling and other nonmedication treatments as a part of our treatment toolbox.

COUNSELING THE YOUNG ADULT WITH ADHD

Now that we know why we need counseling and psychosocial treatment approaches, let us turn to some specific ways that therapists who work with adults with ADHD can be most effective. However, before presenting an overview of nonmedication treatments including individual and group counseling, vocational counseling, cognitive-behavioral therapy (CBT), coaching, use of technology, and advocacy, I want to discuss some common issues and roadblocks that can sometimes undermine both initial and long-term treatment success. This comes more from clinical wisdom I have acquired from counseling adults with ADHD for almost 25 years than from scientific study. I believe it is important to start here because addressing these fundamental and special challenges at least somewhat successfully will likely have huge implications in the long-term outcomes of these patients. Early in my career, I thought it was fairly easy to treat adult ADHD and achieve good to excellent long-term outcomes for a large percentage of patients. I no longer believe this. Although ADHD remains a highly treatable disorder, and many who are properly treated and have milder forms of the disorder do very well, most of the patients I see are on the more severe end of the spectrum and struggle in many aspects of their lives. This is the group I am describing here. ADHD is a complex disorder that in many ways is difficult to treat, and there are many barriers and obstacles that need to be acknowledged and overcome to achieve a sustained positive long-term outcome.

Common Barriers to Successful Treatment

One of the most common obstacles that I see, especially in adolescents and young adults, is a basic lack of knowledge and understanding of the nature of ADHD. Most newly diagnosed patients do not understand the neurological underpinnings of the disorder, do not understand why consistent use of medication is so crucial in treating the symptoms of ADHD, and do not understand or accept that ADHD is a chronic disability they will likely need to manage for the rest of their lives. Instead, they often seem to minimize the disorder, mistakenly believe they just need to try a little harder, and fully believe they can make a few adjustments and treat it their way, on their own terms, and everything will be fine. They may not be interested or ready to learn about ADHD and often do not buy into the idea they need

to take medication on a daily or ongoing basis. Even if the patient is not overtly expressing these sorts of sentiments and appears on the face of it to be accepting the diagnosis, his or her denial or lack of understanding about ADHD can sometimes be seen in the statements or messages he or she conveys either directly or indirectly. For example, comments such as the following signify a fundamental misunderstanding of ADHD and lack of awareness of how difficult it is to manage it:

"I don't need any help for my ADHD."

"I don't want to be dependent on medication to succeed; I want to be able to do it on my own."

"My old coping strategies I used in high school to get by will still work in college."

"I don't need to see a counselor, coach, or academic support person because I know what I need to do and will be able to do it without help."

"I'll do my homework when I get to college."

"I'll just try harder."

The implication here is that the patient believes that ADHD is something that is entirely within his or her control, and that he or she can control the symptoms and behavior if he or she really wants to. This, of course, is not true.

Clinically significant ADHD is bigger than the person's ability to control it. Indeed, this is what makes it a disorder. Despite one's best efforts at compensating for the symptoms, it overwhelms one's efforts, disrupts functioning, and produces impairment in multiple life domains. The young or newly diagnosed patient often does not understand or accept this, and it takes time to finally realize this. People essentially don't know what they don't know. Their life course seems to be like taking one step forward then two steps back, two steps forward and three steps back. It can be extremely difficult to sustain any consistent positive momentum, and the frequent setbacks are frustrating and demoralizing. Unfortunately, it often takes multiple failures or setbacks before patients really "get it" and truly understand what they need to do to manage their disorder. These failures or setbacks may take the form of struggling in school, being placed on academic probation or being dismissed from college, losing jobs or relationships, and generally feeling ineffective, frustrated, or "stuck" in their attempts to get ahead in life.

Acceptance

On the other hand, the adults who do well in managing their disorder are generally those who have *accepted* their ADHD and the hand they were dealt. They have finally recognized that their way of managing ADHD was not working, and they ultimately make a conscious decision to do what they need to do to manage it, and get busy fighting back effectively instead of avoiding or sweeping their ADHD under the rug. In many ways, successful treatment of ADHD begins with true *acceptance* of the disorder. From acceptance springs action and a willingness to embrace treatment and accept help from others. Without acceptance and accurate understanding of ADHD, it is an even more difficult disorder to treat. Mental health professionals can attempt to employ all kinds of psychosocial techniques and the strategies that are discussed below, but if the patient does not understand and *accept* that ADHD is a chronic neurobiological disability that he or she needs to manage aggressively on an ongoing basis, then treatment efforts will likely be ineffective. This is in part why ADHD can be so hard to treat effectively. The need to manage their disorder and maintain treatments consistently over a long time period is not a strength of most people who struggle with ADHD. Hence, in my view, treatment needs to start with helping the patient understand what ADHD is, how it is affecting his or her life, and *accepting it* as a serious issue that demands their serious attention. Without understanding and acceptance, patients are more likely to give lip service to their ADHD, minimize it, and not approach treatment as seriously as they need to.

How do mental health professionals help these patients accept their ADHD and "get it" sooner rather than later? There is no easy answer to this question. I sometimes lament to my patients (and their parents) that I wish I could simply get a syringe and inject them with a solution containing equal parts of maturity, wisdom, ADHD knowledge and acceptance, perseverance, organizational/executive functioning skills, and life experience—and be done with it. Unfortunately, it is not that simple. However, providing sound education and accurate information about the disorder is a good start, and I discuss this and other strategies that help to accomplish this below. Regardless, we need to acknowledge that the disorder has the potential to seriously disrupt lives and help our patients understand this, so they can avoid the potential pitfalls that lie ahead. This takes time, dialogue, sufficient developmental maturity,

and counseling, and it does not happen overnight or by magic. This represents a crucial opportunity for therapists to engage patients and activate them to get involved and take charge of their treatment. We need to do anything we can to foster acceptance of the disorder in our patients because true acceptance is a catalyst for action and change, and a necessary component for optimal long-term success. Stated another way, I believe acceptance needs to occur in order for patients to have a realistic chance to gain optimal control of their disorder and their lives.

One very interesting finding in the Milwaukee Study further illuminates the importance of acknowledging and accepting one's ADHD. It also suggests how common and easy it is for adolescents and young adults to deny their ADHD, and how difficult it can be to accept their ADHD at such an early age. When we conducted the first 10-year follow-up when participants with ADHD were roughly between ages 16 and 22, we found that, as a group, they tended to deny the existence of ADHD symptoms and impairments in their lives, and generally were not embracing treatment for their ADHD, and they tended to externalize blame for any difficulties they acknowledged on others. If we were to accept their reports at face value, we would have to conclude that ADHD largely disappears during later adolescence because only a small percentage of the subjects still met full DSM criteria based on their self-reports. However, when we collected data from their parents at the same 10-year follow-up, their reports were very different from those of their children. The parents reported the continuation of many ADHD symptoms, and life impairments and painted a much more severe (and accurate) picture than their children's reports. This suggested that the young adults with ADHD may have had a good deal of denial going on by the time of the first 10-year follow-up.

Interestingly, at the next follow-up, when most of the participants were between ages 27 and 32, many were no longer denying their symptoms and life impairments as they had done previously. We hypothesized that a major reason for this reversal was that they had now lived long enough and experienced enough life problems that they could no longer deny the impact that ADHD was having on their lives. More were also living away from home and could no longer blame their parents for their ongoing problems. And, of course, they had all undergone another 7 years or more of neurological maturation, especially of their executive networks, so they may have been more capable of self-reflection. For whatever reason, as a group, they were now more willing to accept their ADHD and embrace treatment for it—including medication. It is unfortunate that it took so long for this cohort to "get it." It seemed that, as a group, they were not developmentally ready to understand ADHD or accept it during adolescence or early adulthood, but they were able to later on in their late 20s or early 30s. In my experience, this sort of delayed acceptance only after experiencing a number of setbacks or failures is a common occurrence for young people with ADHD and underscores the importance of using early counseling efforts to help them to understand the disorder as soon as possible. Of course, counselors and all treatment providers also need to understand ADHD so that they do not overreact to the inevitable setbacks, failures, mishaps, or repeated mistakes that these patients experience in their daily lives. Indeed, professionals who treat adult ADHD need patience, tolerance, a sense of humor, realistic expectations, an ability and willingness to help their patients through crises, and a capacity to hang in there with them, no matter what may be around the next corner.

Parental Acceptance

Sometimes it is not just the young adult patient who needs to work on acceptance of the disorder; the parents may need to do so as well. Parents typically have their own goals, aspirations, ideas, visions, and dreams that they desperately want their children to attain, and that they feel their children should be able to attain. In some cases, ADHD may ultimately prevent their children from reaching these parental aspirations. Coming to grips with this and letting go of these goals and dreams can be extraordinarily difficult for parents. There are times when parents need to accept that their children have this disorder and adjust their personal hopes and expectations for their children to something that is more realistic and attainable, or that may be a better fit for their child. This is not easy and it usually comes only after much conflict, heartache, anger, and failure. The ability for parents finally to let go of what they may have wanted and to begin focusing on what is best for their child is perhaps an indication that they have begun to accept their child's ADHD. Counseling can help parents deal with this process and assist them in supporting their children in whatever their future endeavors may be.

PSYCHOSOCIAL TREATMENTS FOR ADULT ADHD

Although there has been an increase in the scientific study of psychosocial treatments for adult ADHD since the previous edition of this book, this research is still in its early stages and much work still needs to be done. Therefore, for the most part, we are still unable to draw firm scientific conclusions about the efficacy of psychosocial treatments. However, a growing body of empirical evidence does support structured, skills-based psychosocial treatment (both individual and group) for adults with ADHD as a valuable adjunctive treatment to stimulant medications (Knouse et al., 2008; see Chapter 32). Recent studies have also provided some evidence that cognitive behavioral skill building approaches can have statistically and clinically significant effects for adults with ADHD (Hesslinger et al., 2002; Knouse, Cooper-Vince, Sprich, & Safren, 2008; Ramsay, 2010; Ramsay & Rostain, 2007;Safren et al., 2005; Solanto, Marks, Mitchell, Wasserstein, & Kofman, 2008; Virta et al., 2008). Critical components of these interventions appear to be psychoeducation, skills training in concrete strategies (e.g., organization and planning skills), and emphasis on practice and maintenance of these strategies in daily life (Knouse et al., 2008). Even though the empirical base for psychosocial treatments for adult ADHD has improved substantially, there is still a way to go before we can rely on established conclusions from scientific research to inform our psychosocial interventions. Nevertheless, I briefly discuss a range of common nonpharmacological ADHD treatment approaches that now have at least some empirical support and should be reviewed with adults during counseling. For a more comprehensive discussion of psychosocial treatments for adult ADHD and the recent research that supports them, see Vidal-Estrada and colleagues (2012).

Although part of this chapter is a description of research findings (the science), much of it is also based on personal and collective, "in the trenches" clinical experience (the art) about what this population needs and which psychosocial methods appear to help which patients. Most practitioners would agree that pragmatic behavioral skills building, organizational and planning skills, and self-management strategies are more useful for the types of issues adults with ADHD encounter than are more traditional, nondirective, insight-oriented, psychodynamic approaches—and the research supports this as well.

Adult Consequences of Growing Up with ADHD

Most adults with ADHD have suffered years of feeling demoralized, discouraged, and ineffective because of a long-standing history of frustrations and failures in school, work, family, social, and daily adaptive domains. Many report a chronic and deep-seated sense of underachievement and intense frustration over squandered opportunities, and are at a loss to explain why they cannot seem to translate their obvious assets into more positive outcomes. Furthermore, many report having heard consistent complaints about themselves from parents, teachers, spouses/partners, friends, or employers. Such complaints focus on their behavioral, academic, interpersonal, or productivity shortcomings. The cumulative effect of such a history can sometimes lead to feelings of intense frustration and demoralization, and to a sense of anticipating failure as the predictable outcome of their efforts. Sadly, some appear so wedded to this belief system that they eventually give up believing life can be different for them. Many are completely unaware that their condition is a highly treatable one.

Therefore, instilling hope, optimism, and motivation during the counseling of adults with ADHD is critical, so that patients can better understand and accept their condition and be more inclined to engage in and follow through with a multimodal treatment plan. An important ingredient of this counseling is to help patients view their disorder from a perspective that empowers them to believe their lives can be different, and that encourages their active (and hopefully enthusiastic) involvement in treatment. The principles and treatments described here are not new and are in many ways generic to psychosocial counseling with any psychiatric population. However, interventions such as education about the disorder, cognitive restructuring, reframing the past, and instilling hope seem to lend themselves particularly well to the treatment of ADHD in adults.

Some of the more common correlates associated with ADHD in adults are low self-esteem, avoidance/anxiety, depression, school and job performance problems, marital/couple discord, poor driving outcomes, and substance abuse. Many adults with ADHD report low self-esteem as a result of years of frustration with their academic, work, social, and day-to-day family lives. They often report a long-standing and nagging sense of knowing something is wrong, but never knowing exactly what it is. In many cases they have sought help from

multiple mental health professionals who overlooked the possibility of ADHD and instead conceptualized their problems as related solely to mood, anxiety, or character disorders. Treatment for the underlying neurobiological condition (ADHD) that may be driving at least some of their behaviors/symptoms may never have even been considered, which may explain why many adult patients report that past counseling experiences were not especially helpful. Consequently, some end up attributing their problems to characterological or moral defects in themselves and pay a heavy emotional price as a result. This underscores the importance of reframing the disorder as neurobiological and not characterological, of rebuilding self-esteem and self-confidence, and of instilling hope for the future.

Other common consequences of having ADHD are anxiety about and avoidance of situations that have historically been unsuccessful or troublesome for the patient. One example of this avoidance is the idea of returning to school after having failed and dropped out in the past. Some adults who may express a desire to return to school are understandably hesitant because of their prior record of school struggles. They fear that they will fail again, and they wish to avoid another setback. They report that if they had reason to believe that their school experience might be different this time, they would be more willing to attempt it. But for many, it is safer not to try, so they avoid school, even though deep down they have a strong desire to go. This is indeed unfortunate because proper diagnosis, treatment, and motivation can open new possibilities and potentially make the difference between success and failure in school.

Another example concerns social/interpersonal relationships. In part because of their impulsivity, interrupting, forgetfulness, inattentiveness, hyperactivity, difficulty reading social cues, temper, and/or mood swings, adults with ADHD frequently report having difficulty with interpersonal functioning and maintaining friendships. Others may view their behavior as rude, insensitive, irresponsible, or obnoxious, and their peers may sometimes ostracize them. Some associate social interaction with embarrassment, disappointment, criticism, or failure. When confronted with future opportunities for social interaction, these adults with ADHD sometimes withdraw or avoid others to protect themselves. Again, treatment can sometimes improve their verbal and behavioral impulsivity, disinhibition, and focusing/listening ability, and as a result may improve their overall social functioning.

Depression is another relatively common consequence associated with adult ADHD. Some studies have shown that depression is a commonly occurring comorbidity with ADHD (see Chapter 13; Barkley et al., 2008; Murphy, Barkley, & Bush, 2002). Some adults with ADHD have become so demoralized over their past failures, and being misunderstood and mistreated by others, that they require concurrent treatment for a mood disorder.

A substantial minority of adults with ADHD gravitate toward substance abuse—possibly as a way of relaxing or calming the mental restlessness they often experience. Some studies suggest that those with ADHD are at increased risk for developing substance use problems (Mannuzza, Gittelman-Klein, Bessler, Malloy, & LaPadula, 1993; Murphy & Barkley, 1996; Murphy et al., 2002; Weiss & Hechtman, 1993; see also Chapters 11 and 13). Approximately one-third of the patients our group studied at the UMASS Adult ADHD Clinic met criteria for substance abuse or dependence at some point in their lives. Many appeared to be self-medicating in an attempt to cope with their underlying ADHD symptoms. Most reported using alcohol and/or marijuana as their primary drugs of choice. After these patients were treated with stimulant medication, a fair number of them reported improvement in not only their ADHD symptoms but also their substance abuse. Others have also found this to be true (Schubiner et al., 1995). One possible hypothesis is that the stimulant medication may lessen the desire to self-medicate. Hence, it may in some cases be a mistake routinely to disqualify patients with ADHD and substance abuse from medication treatment. To do so may be depriving these patients of a potentially important and needed treatment. This is different in the case of active substance dependence. Patients should be referred for treatment of the substance dependence before undergoing any medication treatment for ADHD. In most cases, it is suggested that patients with substance dependence achieve at least 1–2 months of stable sobriety before medication for ADHD is introduced. Those with comorbid substance abuse/dependence and ADHD require close follow-up to monitor progress and safety. Clearly, the relationship between ADHD and substance use disorders warrants further scientific investigation.

An important goal for professionals who treat adults with ADHD is to respond to these and any other negative sequelae of living with ADHD in a way that instills hope; fosters personal potency; and encourages patients to believe that with a combination of treatment, accep-

tance, support, perseverance, and hard work, their lives *can* be improved.

Explaining the ADHD Diagnosis

Treatment for adults with ADHD begins at the time the patient is diagnosed. How clinicians communicate the diagnosis to the patient is, I believe, critical to both understanding and perhaps acceptance of the disorder and the willingness to engage in and persist with treatment. If clinicians can help patients understand the disorder, offer a plausible rationale for how it causes their symptoms, frame it as something that is treatable, and instill hope and optimism for their future, patients are more likely to feel motivated to work at and follow through with treatment. Increased knowledge and understanding of the disorder, and continuing involvement in treatment, is likely to increase the chances of more positive outcomes. Conversely, if patients are left with only a vague notion of what ADHD is, are confused or unsure of how they might be helped, and are not activated to feel hope, they are far less likely to embrace treatment, persevere, and achieve a positive outcome. I have worked with many adult patients who had prior ADHD evaluations in their lives and reported having very little understanding of ADHD and being unaware of the impact it had on their lives. Either the disorder was apparently never explained to them in a way that they could receive and understand it, or perhaps they may not have been developmentally ready to hear, accept, and integrate it. Clinicians can have substantial control over the feedback process and have an opportunity to influence whether patients become actively engaged or disengaged from treatment. The framework described next may assist clinicians in developing strategies and skills to explain the diagnosis more effectively to adults.

Education about the Disorder

Perhaps the most important nonpharmacological strategy for adults with ADHD is to educate themselves as much as possible about the disorder. Most have little knowledge of ADHD or the pervasive impact it can have on their day-to-day lives. Having an accurate knowledge base can help adults make sense of what has been troubling them, help them set realistic and attainable goals, and ease their frustration. Just knowing that there is a neurobiological reason for many of their struggles, and that this reason has a name, can be therapeutic in itself. The realization that somebody finally "gets it" and truly understands their lifelong difficulties can also be extraordinarily therapeutic. Once these adults are accurately diagnosed by a professional who understands ADHD, there is often a sense of tremendous relief at finally having an explanation for their long-standing difficulties. The clinician can begin by explaining the rationale for arriving at the diagnosis of ADHD and any comorbid conditions. Providing such an explanation can help demystify the diagnosis and put it in the context of each patient's own unique life experience. For example, explaining all of the following can help a patient begin to understand ADHD: (1) The patient and a spouse/partner or parent have endorsed a sufficient number of the symptoms of ADHD, according to the DSM-5 (American Psychiatric Association, 2013); (2) the onset of symptoms occurred during childhood or, at the latest, early adolescence; (3) the symptoms have caused chronic and pervasive impairment in academic, social, vocational, and/or daily adaptive functioning; (4) the patient has no other psychiatric or medical condition or situational stressor that better explains the ADHD symptoms; and (5) he or she has a long-standing behavioral, school, and/or work history reflecting typical impairments associated with the diagnosis.

Reframing the Past

An important next step is to continue educating the patient about what ADHD is and how it affects his or her life. Learning about ADHD is especially important at the beginning of treatment, but it should be viewed as a lifelong endeavor because the disorder plays out over time and across situations. Patients need to have at least a general understanding that they have a neurological condition, not a character defect or moral weakness. The realization that many of the problems they have experienced stem from neurological causes rather than from laziness, low intelligence, or lack of effort can begin the process of repairing self-esteem. Often patients have internalized negative messages over the years from parents, teachers, spouses/partners, and employers, who have concluded that they are stupid, lazy, incompetent, immature, or unmotivated. It should be explained that the likely reason for many of the problems they experienced in school, work, and/or social relationships was a subtle neurobiological deficit in the brain over which they had little control. Their problems were not the result of deliberate misbehavior, low intel-

ligence, or lack of effort. These misguided and damaging perceptions should be recast in a more positive and hopeful light, so that patients can begin to rebuild their self-confidence and believe that successful treatment is possible. As a consequence, patients will ideally be in a better position to break out of the shackles of feeling stuck, demoralized, and chronically frustrated.

Patients also need to understand that they themselves are a potent force in their treatment, and that what they do from this point forward really matters in their final outcome. Treatment for ADHD is very much a collaborative process. Patients need to accept their disorder and do their part by actively engaging in treatment, practicing new skills, communicating honestly about obstacles they are encountering, dealing with inevitable setbacks, taking medication consistently, and making a genuine and persistent effort at making changes in their lives. Educating spouses/partners, family members, and friends is also important, so that those others can understand and be better able to help. A common knowledge base can help patients, spouses/partners, and family members cope more effectively, establish realistic goals and expectations, and reduce conflict.

Instilling Hope

Another important aspect of setting the stage for successful treatment is *instilling hope*. Without hope for a better future, there is little chance that patients will engage in or persist in treatment long enough to accomplish significant gains. To achieve an optimal outcome, patients need to feel that their clinicians are partners with them and sincerely believe they can be helped. If clinicians are genuine in their desire to become involved in helping, and this is clearly evident to patients, it can go a long way toward instilling hope and motivation in the patients. Conversely, if clinicians are perceived by patients as merely technicians performing their routine in a relatively uninvolved manner, the opposite is true. Caring, support, compassion, and encouragement are crucial ingredients, and their importance should never be underestimated. The pressures of the managed care environment and the reality of doing more in less time with fewer resources can make this a real challenge in today's health care environment. Nevertheless, the message that should come through loud and clear is that with proper treatment—including education, counseling, medication, behavioral strategies, hard work, advocacy, and the support of family and friends—adults with ADHD can significantly and sometimes dramatically improve their lives.

As an additional educational resource, providing a packet of educational literature to patients at the end of an evaluation may be helpful. This may include a fact sheet about ADHD; a list of relevant books (see Barkley, 2011), magazines, or newsletters; websites of advocacy organizations such as Children and Adults with Attention-Deficit/Hyperactivity Disorder (CHADD; *www.chadd.org*) or the Attention Deficit Disorder Association (ADDA; *www.add.org*); brief articles on relevant topics, such as ADHD in college or in the workplace; and/or information sheets on medication (copies of fact sheets on a wide range of medications can be found in Dulcan & Lizarralde, 2003). For those who may not be inclined to read educational literature, there are DVDs (Barkley, 1994) and free online resources (e.g., *www.adhdsharedfocus.com*) or the lectures by Barkley online (*www.adhdlectures.com*) or on YouTube that provide access to educational videos on various topics relating to ADHD. Although there is no scientific proof that such educational media are useful (or even actually read or viewed), one would hope that providing this type of immediately relevant educational material can promote better understanding and help motivate some patients to engage in ongoing treatment.

It can also be helpful to provide some specific examples of treatment strategies that are relevant to the problems that patients are currently experiencing. For example, patients who are disorganized and forgetful may benefit from training in prioritizing and list making, how to use an appointment calendar, posting visual reminders in strategic locations, blocking out time in schedules for priority tasks, breaking large tasks down into smaller units, building minirewards into projects, and the like. For patients who are college students, it may be useful to describe some specific types of classroom modifications, lifestyle or class schedule adjustments, study skills, or other accommodations that are appropriate and justified given the nature and history of their functional impairment.

Providing education to patients about medication also seems important. Explaining how medication may help patients improve the quality of their lives by enhancing their ability to focus and concentrate, and to curb their impulsivity may provide further hope and motivation. Explaining how their lives may be different if they respond well to medication by using actual examples from their personal histories may be useful.

Taking the time to answer questions about side effects, and providing enough factual information for patients to make informed decisions regarding medication, also appears useful. Patients often have mistaken notions and unrealistic fears/myths about medication that need to be addressed before they agree to try it. Providing fact sheets (as mentioned earlier) in addition to these verbal explanations can give them further information to share with family or friends.

It is important to understand that treatment should not be approached with the idea that ADHD can be "cured," because there is no treatment or combination of treatments that can cure the disorder. Instead, it is more accurate to approach treatment in terms of symptomatic relief, or learning how to manage symptoms and cope with the challenges that the disorder presents across the lifespan. A central tenet of treatment is to assist patients in becoming "the best that they can be" by helping them to focus and build on their strengths, and learn to compensate better for their weaknesses (Murphy, 1995).

Instilling hope for the future, balanced with the reality that changing habits and behavioral patterns requires hard work and sustained effort, can foster a proper and realistic attitude toward treatment. Clinicians can exert a strong influence in constructing a therapeutic atmosphere of hope and optimism, to counter the demoralization and pessimism so often experienced by adults with ADHD. Equipped with this combination of hope, knowledge, acceptance, and awareness of ADHD, adults with ADHD should be in a much better position to benefit from treatment and learn to adapt better to current tasks and responsibilities, and to lead more fulfilling lives than had previously been the case.

Psychosocial Treatments

A combination of treatments is usually recommended for adults with ADHD. Again, treatment of the individual with ADHD does not produce a cure for the underlying cause of the disorder. Treatment is aimed at symptom reduction and minimizing the negative effects of the disorder to improve one's overall quality of life. Despite the fact that early researchers suggested that clinic-based treatments focused on skills training, such as social skills, self-control, or cognitive-behavioral training, were not of much benefit to children with ADHD (Abikoff, 1985, 1987; Diaz & Berk, 1995), and that short-term psychosocial treatment effects did not generalize outside the context in which they are applied (Abikoff & Gittelman, 1984; Barkley, 1997b; Barkley, Copeland, & Sivage, 1980), new research suggests these conclusions may not be entirely accurate (see Chapter 32; Knouse et al., 2008; MTA Cooperative Group, 1999; Ramsay & Rostain, 2007; Solanto, 2011; Vidal-Estrada et al., 2012). In fact, the National Institute of Mental Health (NIMH) Multimodal Treatment Study of ADHD (MTA; MTA Cooperative Group, 1999), the largest randomized treatment study ever undertaken, found that psychosocial treatments in combination with medication resulted in the best outcomes in some circumstances (see Chapter 28). Psychosocial treatment may not "cure" the underlying brain dysfunction that gives rise to core ADHD symptoms, but it may well help to improve the side effects, emotional sequelae, and/or comorbid conditions that often go along with ADHD.

The most common types of psychosocial treatments for adults with ADHD include individual counseling, group counseling, family and marital/couple counseling, vocational counseling, coaching, use of technological aids, and advocacy. For an excellent summary of the research that supports these approaches see Knouse and colleagues (2008) and Vidal-Estrada and colleagues (2012).

Individual Counseling

The initial stage of individual counseling usually includes information/education about ADHD, outlining goals, developing strategies to meet those goals, and dealing with any acute conflicts or crises that may be present. Follow-up meetings monitor progress, discuss medication issues, add or alter treatment approaches, and work on improving specific areas of difficulty. Examples may be problem solving about a specific work, school, or relationship situation; assisting with life transitions, such as a career change or a divorce; dealing with comorbid mood or anxiety disorders; or working on organizational and time management skills. Individual counseling can bring adults with ADHD increased awareness of how the disorder affects their lives, and can thereby help to identify appropriate behavioral/self-management strategies to manage symptoms better. Awareness of the impact of the disorder can also influence future life decisions. For example, knowledge of ADHD can influence one's job choice, choice of spouse/partner, choice of major in school, or decisions about whether to return to school and where (preferably one with an established program for assisting students with ADHD and/or learning disabilities). Acquiring this kind of self-knowledge can assist adults

with ADHD in making more appropriate choices and goodness-of-fit decisions.

Adults with ADHD may also benefit from individual counseling on behavior modification principles and strategies. Treatment for ADHD appears to respond best to an active and pragmatic approach on the part of both therapist and patient. In general, the more structure and routine that can be incorporated into a patient's life, the better. Most often, the goals of treatment are to change disruptive or maladaptive behavior and thought patterns that consistently interfere in day-to-day functioning. Patients usually prefer utilizing strategies that help them function more effectively right now, as opposed to a long-term, insight-oriented approach. Stated another way, they would rather implement a behavior plan today to prevent daily loss of their car keys than explore or attempt to interpret the underlying meaning of this behavior.

CBT is a form of individual counseling that seems particularly useful to adults with ADHD and that has some research support behind it (see Chapter 32; McDermott, 1999; Ramsay, 2007; Ramsay & Rostain, 2008; Safren et al., 2005; Stevenson et al., 2002; Virta et al., 2008). Specifically, training in methods of time management, organizational skills, communication skills, anger control, use of a daily planner, self-monitoring, chunking large tasks into a series of smaller steps, and changing faulty cognitions are thought to help adults with ADHD more efficiently meet the demands of daily work, family, and social life. Such training helps patients to develop explicitly stated goals, specific methods to accomplish goals, and established time frames for meeting goals. In essence, the same sorts of suggestions that may prove useful to children with ADHD in school may also be of value to adults with ADHD when upgraded to their performance contexts. Implementing behavioral strategies to target the most impairing problems can help patients gain greater control over their lives, reduce anxiety and frustration, and improve productivity. Providing patients with the following suggestions, and helping them to develop or improve proficiency in these areas, may be beneficial:

- Practice proactive planning by setting aside time every evening to plan for the next day. Get needed materials ready (e.g., books, clothes, keys, phone numbers, medication, important papers), pack the car the night before, or do whatever else that will prevent frantic chaos the next day.
- Learn how to make an effective and reasonable "to do" list of important tasks and priorities, and keep it with you at all times. Make additional copies in case it gets lost or misplaced.
- Remind yourself by keeping important tasks visually in sight by posting appointments, "to do" lists, or schedules in strategic areas at home and at work.
- Practice using an appointment book, a smartphone, or other technology device, or a daily planning calendar, and learn to write down appointments and commitments immediately.
- Keep notepads in strategic locations (car, bathroom, bedroom, etc.), or have a portable audio recorder handy to capture important ideas and thoughts that cross your mind and that you wish to remember.
- Learn and practice time management skills. Purchase a programmable alarm watch or set an alarm on your smartphone, so that you do not lose track of time.
- Use a color-coded file system, desk and closet organizers, storage boxes, or other organizational devices to reduce clutter and improve efficiency and structure in your life. Consider hiring a professional organizer to assist in creating a workable system for you; this may include ensuring that bills are paid on time, balancing the checkbook, and decluttering your living space.
- Make multiple sets of keys, so that losing one set is not a disaster.

Preparing patients for the expected and inevitable feelings of disappointment and frustration when setbacks occur can also be helpful. Rather than viewing setbacks as catastrophic failures or evidence of incompetence, patients can be helped to conceptualize them as "normal," expected, and even desirable because they represent opportunities for learning and personal growth. For example, adults with ADHD may conclude that making lists or using an appointment book is fruitless because they frequently lose them. Explaining that changing habits and learning new strategies requires ongoing practice and are not one-trial learning affairs may help adults with ADHD keep trying. The goal is to continue practicing each skill until it becomes an automatic and natural part of a daily routine. Ultimately, patients must make a conscious commitment to work on behavioral change, to view it as a crucial investment in their future, and to elevate mastering these skills to a priority in their lives. Individual counseling aimed at erasing long-standing negative messages from teachers, parents, spouses/partners, and employers, and replacing these with more rational and

optimistic messages, is another area of potential benefit to adults with ADHD.

It is also important to emphasize and make explicit the strengths and positive traits that patients possess. For example, informing patients that their testing results indicated average , above-average, or superior native intelligence can sometimes be a powerful revelation. Explaining that their lower-than-expected grades throughout their school history had nothing to do with low intelligence can provide a strong measure of relief to adults who may well have lived their lives believing the opposite. Another example is to point out patients' positive character traits, such as tenacity, willingness to keep trying despite many setbacks, boundless energy and drive, assertiveness, sense of humor, or whatever else is appropriate. This may serve to counterbalance negative self-perceptions, to reinforce strengths, and to promote self-acceptance.

In summary, individual counseling may help adults with ADHD to cope with a variety of coexisting problems, including depression, anxiety, low self-esteem, interpersonal problems, and disorganization.

Group Treatment

There is now preliminary research evidence of the usefulness of various group therapy approaches to treatment (Solanto, 2011; Solanto et al., 2008; Virta et al., 2008; Zylowska et al., 2008). Group therapy has the potential to be a useful method of support, education, and validation for those with ADHD. Patients can learn a great deal from each other, feel accepted, and feel less isolated and alone. One of my patients who participated in a support group had previously refused to take medication; he ended up changing his mind after discussing the issue with fellow group members and receiving their input. Clearly, the group influenced him to try the medication, whereas I, as his individual therapist, had been unsuccessful. In addition to the support and validation offered by the group, hearing how others cope and manage their symptoms, realizing that there are others with similar problems, and having a "laboratory" for learning and trying out new social and interpersonal skills can all be helpful to group members.

The studies mentioned earlier suggest that what works best is time-limited groups (8–10 weeks in duration) that include a structured format, components of psychoeducation, weekly homework assignments, training in concrete skills (e.g., organization and planning strategies), and practice of these strategies in daily life. In contrast, ongoing, open-ended, "here-and-now" types of groups can rapidly become chaotic and disorganized, and can be difficult to lead and manage. Topics studied in the group interventions include time management, organization, planning, neurobiology and medication, motivation and initiation of activities, emotion regulation, memory, impulsivity, self-esteem, communication, mindfulness training, information for significant others, and comorbidity (see Chapter 32). Although ongoing research on group interventions is needed, this promising modality can be a useful adjunct to other forms of treatment. Participating in local support group organizations such as CHADD is another avenue for support and education.

Family and Marital/Couple Counseling

Family and marital/couple therapy may also be potentially useful for resolving difficulties that affect relationships in family members and spouses (see Chapter 34). ADHD can wreak havoc on marital/couple and family functioning, in part because it can be so disruptive to the routine tasks of daily living. We found a greater incidence of marital dissatisfaction in our ADHD groups in both the UMASS and Milwaukee studies, as well as poorer quality of dating relationships in those adults with ADHD who were not married (Barkley et al., 2008). An earlier study at the UMASS Adult ADHD Clinic reported more severe marital dissatisfaction in the ADHD group versus the control group as measured by their Locke–Wallace Marital Adjustment Inventory scores (Murphy & Barkley, 1996). Spouses and partners of adults with ADHD who do not have ADHD themselves often report feeling confused, angry, and frustrated. They may complain that the adults with ADHD are poor listeners, unreliable, forgetful, self-centered or insensitive, messy, and often seem distant or preoccupied, do not finish household projects, or behave irresponsibly. Gaining a greater understanding of the disorder, and realizing that many of these problems may not necessarily stem from "willful misconduct," may enable the partners to take a fresh look at their problems from an ADHD perspective, stop blaming each other, and begin to align together as a team to reduce conflict. For this to be successful, however, a patient's spouse or partner must perceive the patient to be making a sincere and legitimate effort at behavioral change. If the patient uses the ADHD as an excuse to justify continued behavioral problems without demonstrating an observable commitment to behavioral change, there

will be little chance for improvement in the relationship. Framing the situation as a family problem instead of pointing the finger at the "identified patient" may help to reduce defensiveness. If both spouses/partners have a mutual understanding of how ADHD affects their relationship, understand what each needs from the other, and work together as a team in improving the family situation, the chances for a positive outcome are greatly enhanced.

Working together as a unified and cohesive team is especially important in families in which both a parent and a child have the disorder. When multiple family members have ADHD, this adds another layer of complexity and challenge to effective family functioning. The potential for conflict, stress, lack of follow through, miscommunication, and family chaos is much higher when both parents and children have the disorder. Ideally, each will seek his or her own individual treatment to manage the symptoms. They will also likely benefit from family counseling to explore ways to manage conflict, improve communication and follow through, and increase family harmony. Key ingredients are incorporating a structured daily routine to aid in staying organized and reducing forgetfulness, and maintaining a sense of humor—especially when inevitable setbacks occur.

Vocational Counseling

As the summary of our findings on vocational functioning at the beginning of this chapter suggests, workplace problems can be particularly troublesome to many adults with ADHD. Impulsivity, inattention, careless mistakes, disorganization, poor time management, tardiness, short temper, missing deadlines, and inconsistency are just some of the things that can interfere in job performance. Most adults with ADHD who experience workplace problems do so not because of incompetence or lack of effort, but because their jobs are ill suited to their strengths. They frequently leave jobs because of boredom or inability to tolerate what they perceive as a boring and tedious daily routine. Vocational counseling aimed at identifying strengths and limitations and matching patients to jobs that "fit" for them is of critical importance for many adults with ADHD. This may involve vocational testing to identify interests and aptitudes, job coaching and training, or advocacy with potential employers. Unfortunately, the need for such services greatly outweighs the availability of skilled resources. Nevertheless, successful vocational adjustment is not only central to individual well-being and self-esteem, but it can also have a positive effect on family and marital/couple functioning, as well as family financial health.

Coaching

Another potentially helpful area of intervention for adults with ADHD is personal coaching. Although there has been very little scientific study of coaching since the previous version of this book was published, coaching is still a popular adjunctive treatment for adults. The small number of published studies on coaching for adults with ADHD have focused mostly on the college setting (Kubik, 2010; Stevenson et al., 2002; Stevenson, Stevenson, & Whitmont, 2003; Swaetz, Prevatt, & Proctor, 2005; Zwart & Kallemyn, 2001). Although these studies suffered from some methodological shortcomings, they did suggest that coaching was beneficial.

The International Coach Federation defines coaching as "partnering with clients in a thought-provoking and creative process that inspires them to maximize their personal and professional potential" (Sleeper-Triplett 2010, p. 2). Coaching is a supportive, pragmatic, and collaborative process in which a coach and an adult with ADHD work together (usually via daily 10- to 15-minute telephone conversations) to identify goals and strategies to meet those goals. Because most adults with ADHD have difficulty persisting in effort over long periods and often cannot sustain ongoing motivation to complete tasks, coaches can assist them in staying on task by offering encouragement, support, structure, accountability, and at times gentle confrontation. There is no standard methodology. The coaching relationship is tied to the needs and desires of each patient and can be structured in any way that is acceptable to the coach and the person being coached. Some may talk with their coaches on a daily basis and others may do so far less frequently. Some may correspond via e-mail. The intended outcome is to assist adults with ADHD to take charge of and better manage their lives by learning to set realistic goals and stay on task to reach those goals, in an atmosphere of encouragement and supportive understanding. Although we await future results of scientific inquiry into the effectiveness of coaching, it is likely to continue to be a frequent treatment recommendation for the adult population with ADHD. For a more detailed discussion of coaching, see Ratey (2002, 2008) and Sleeper-Triplett (2010).

Technology

Professionals who work with adults with ADHD should be aware of technological advances that offer valuable and much needed assistance to people struggling with ADHD. A variety of tools and devices can help greatly in communication, writing, spelling, keeping track of time, and the like. Word processors and programs with spell-check and grammar-check options can aid in writing and spelling more quickly, legibly, and effectively. A personal digital assistant (PDA) offers a wide range of components, including an electronic address book, a planner/calendar, a "to-do" list, and notepad. Smartphones and text messaging make communication easier, more spontaneous, and faster. Many software programs are available to assist with personal finances and taxes. Websites devoted to organizational skills, time management, and just about any other relevant topic are immediately available on the Internet. Electronic banking offers online bill paying that includes setting up automatic payments at regular intervals to protect against delinquent payments and late fees. Books on tape and voice-activated word-processing programs can assist in learning and writing. Live Scribe Pens can greatly assist students in note taking and recording classroom lectures. These sorts of devices and interventions should be used whenever appropriate, but they require time, practice, and persistence to master.

Advocacy

Self-advocacy, an important and sometimes overlooked skill, can be a key to success on the job, in an academic environment, or in other life situations. It is crucial that individuals create a strong foundation for self-advocacy by developing both an understanding of their own ADHD and the ability to explain their strengths and weaknesses to others (Roffman, 2000). Rehearsing or role playing with a counselor a succinct explanation of what ADHD is, how it interferes with functioning, and what is needed to accommodate it can be helpful in achieving the necessary confidence and skill.

A key ingredient to successful self-advocacy is thorough, professional documentation. When patients are armed with high-quality documentation, their chances of having others understand their challenges and view their situation as credible are much higher. The value of developing self-advocacy skills should never be underestimated.

Regardless of how good a person's self-advocacy skills are, however, there are times when a professional advocate will be beneficial. In high-stakes situations such as eligibility for test accommodations or workplace accommodations (see Chapter 33), especially when supervisors or professors refuse to believe or accommodate a diagnosis, professional advocacy may be necessary. Examples of situations in which professional advocacy can make a significant difference include attending special education or individualized education plan meetings, writing letters of recommendation for college or job applications, writing comprehensive reports for test accommodation eligibility, meeting with supervisors or professors to explain the diagnosis and reasons for accommodation, communicating with probation officers, participating in disciplinary meetings, and participating in workplace discussions about appropriate job modifications or placements. A qualified professional who fully understands a patient's situation can enhance the patient's self-advocacy by adding explanatory power and additional credibility.

FINAL COMMENTS

A subgroup of the adult population with ADHD may need additional treatment for specific problems that may coexist with ADHD, such as substance abuse/dependence, credit /money management problems, eating disorders, or anxiety and mood disorders. Because those with ADHD are at greater risk for developing comorbid problems, treatment efforts need to take into account the totality of each patient's problems. Whatever combination of treatments is used for a given patient, it is likely that intervention will need to be extended over long time intervals, much like the management of a chronic medical illness such as diabetes (Barkley, 1997a; see Chapter 16). In general, treatments and lifestyle habits need to be maintained consistently over long periods of time to sustain optimal benefit. If treatments are removed or discontinued, the symptoms and associated impairment are likely to resurface within a short time. This is why a major goal of treatment is to work toward instilling lifelong habits and permanent lifestyle changes rather than short-term, transient, or quick-fix strategies. For example, when a clinician is counseling a college student on developing time management and organizational skills, these should not be viewed as short-term tools for merely achieving a grade, passing a test, or getting to a class on time. Rather, they

should be taught in the context of life skills training for the future as well. These are examples of skills and habits that, when put into practice as part of a daily routine, will have a positive ripple effect in all aspects of life, including work, social, marital/couple, and daily adaptive functioning. Periodic follow-up for support, adjustment to treatment, academic or workplace advocacy, or new interventions as life circumstances change will probably be necessary for most adults with ADHD in the ongoing management of their disorder.

KEY CLINICAL POINTS

- ✓ ADHD is a disorder that can be effectively treated with both medication and psychosocial approaches.
- ✓ Although the research on psychosocial treatments for adult ADHD is still in its early stages, a growing body of scientific evidence suggests that counseling in general and CBT approaches in particular may be useful, including structured, skills-based training with ongoing practice.
- ✓ Comorbidity is common with ADHD, so clinicians need to incorporate treatments for both ADHD and the range of coexisting diagnoses that often accompany it, including mood and anxiety disorders and substance abuse.
- ✓ Explaining the ADHD diagnosis in an understandable way that instills hope and activates patients to accept their ADHD and be active participants in their treatment is important and improves chances for more positive outcomes.
- ✓ Multimodal treatment that combines medication, education, behavioral/self-management skills, a variety of counseling approaches, coaching, and either academic or workplace accommodations is likely to result in the best outcomes.

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CHAPTER 32

Cognitive-Behavioral Therapies for ADHD

Laura E. Knouse

Cognitive and behavioral therapies (CBTs) are based on a scientific understanding of the complex interplay of thoughts, feelings, and behavior in the etiology and maintenance of a wide range of psychological disorders. Behavior therapies based on principles of classical and operant conditioning have merged with cognitive approaches emphasizing the influence of beliefs, appraisals, and their modification in the mediation of new learning (Craske, 2010). Although empirically supported cognitive-behavioral approaches have been developed for a wide array of disorders—including behavioral interventions to help manage attention-deficit/hyperactivity disorder (ADHD) in childhood—CBT for ADHD in adulthood is a much more recent development. This is scarcely surprising given the delayed recognition of the disorder's persistence into and impact on adulthood. Fortunately, the last 15 years have seen a steady and growing stream of interest in and—most importantly—empirical studies of CBT for adult ADHD. Manualized approaches are now available for both group and individual application, and larger randomized trials with active treatment control conditions support the efficacy of CBT (Safren et al., 2010; Solanto et al., 2010).

Despite its short history, the psychosocial treatment landscape for adult ADHD is growing increasingly crowded. Several research groups have published outcome data from trials of various approaches: group psychoeducation (Wiggins, Singh, Getz, & Hutchins, 1999), dialectical behavior therapy skills training (Fleming, 2013; Hesslinger et al., 2002; Hirvikoski et al., 2011; Philipsen et al., 2007), group cognitive remediation (Stevenson, Whitmont, Bornholt, Livesey, & Stevenson, 2002), CBT for medication-treated adults with residual symptoms (Safren, Otto, et al., 2005; Safren et al., 2010), problem-focused therapy (Weiss & Hechtman, 2006; Weiss et al., 2012), CBT with stimulant medication (Ramsay & Rostain, 2011; Rostain & Ramsay, 2006); cognitive-behaviorally oriented group rehabilitation (Salakari et al., 2009; Virta et al., 2008, 2010), mindfulness meditation training (Mitchell et al., in press; Zylowska et al., 2008), cognitive-behavioral group therapy (Solanto, Marks, Mitchell, Wasserstein, & Kofman, 2008; Solanto et al., 2010), a CBT workshop series (Bramham, Young, Spain, McCartan, & Xenitidis, 2009), and Reasoning and Rehabilitation 2 for ADHD (Emilsson et al., 2011). Many (but not all) of these treatment programs are described as using cognitive-behavioral strategies or adhering to CBT principles, although there appears to be considerable variability in program content and in the extent to which programs focus on providing psychoeduca-

tion versus teaching clients to implement specific skills (Knouse & Safren, 2010, 2013a). Because of the variety of approaches currently available, one aim of this chapter is to provide information on the principles and hypothesized mechanisms of change in CBT, so that readers have a clear understanding of the application of these principles to the treatment of ADHD in adults.

This chapter provides an introduction to the science and practice of CBT for adult ADHD and next-step resources for those wishing to learn more. As such, the chapter does not review every psychosocial approach for adult ADHD listed earlier; instead, it focuses on CBT treatment principles and the most rigorously tested approaches. The information presented draws heavily on the growing research literature on CBT in general and specifically for adult ADHD. In the latter part of the chapter, I also provide some general recommendations to clinicians implementing skills-based treatment with this population, drawing on the clinical literature and my experience as a clinician conducting CBT with clients in the context of research trials and general outpatient work. Because of its relatively short history, CBT for adult ADHD is an exciting and dynamic area of research and practice despite the many questions that remain to be answered.

In sum, this chapter will:

- Describe essential elements of cognitive-behavioral therapy and their application to adult ADHD.
- Provide a theoretical and empirical rationale for using CBT with this population.
- Familiarize readers with two major empirically supported approaches.
- Provide key recommendations for conducting skills-based ADHD treatment for adults.
- Describe next-step directions for the field.
- Suggest resources for readers wishing to learn more in an annotated references section.

ESSENTIAL FEATURES OF CBT

Before describing specific CBT treatment programs for adult ADHD, I first discuss key cognitive-behavioral principles and give examples of how they may be applied to adult ADHD treatment. For readers new to CBT, this is important background information. For readers already familiar with CBT for other disorders, this section links prior knowledge with applications in adult ADHD. In addition, for all readers, this section provides a basis on which to evaluate treatment approaches and treatments as they are actually delivered. Because of the research supporting many CBTs for major disorders, and perhaps because of the appeal of short-term, evidence-based practice to managed care payors, CBT appears to be more popular than ever. But what elements are necessary to qualify a treatment as CBT? This question is not a trivial one. When clinicians in the community report that they are using CBT, the specific techniques they employ may not include key "active ingredients" of empirically supported treatments (Freiheit, Vye, Swan, & Cady, 2004; Stobie, Taylor, Quigley, Ewing, & Salkovskis, 2007). Thus, understanding key principles is foundational to learning CBT techniques and evaluating the research literature on this topic.

As a point of reference, let us consider key elements in the description of CBT provided by the Association for Behavioral and Cognitive Therapies (2013), the major professional organization for CBT clinicians and researchers. Four major characteristics contained in that description are (1) CBT is based on scientific evidence; (2) CBT is short term; (3) CBT takes into account interactions among thoughts, emotions, and behaviors; and (4) CBT teaches clients specific skills.

Based on Scientific Evidence

A crucial feature of CBT is that it is based on scientific evidence. This means that the techniques a therapist uses are based on a scientific understanding of how human thoughts, emotions, and behavior interact with one another and the environment to produce and maintain clinical problems (e.g., basic principles of operant conditioning; effects of emotion on attention and memory). A CBT clinician also takes an empirical approach with each individual client, collecting data throughout treatment and modifying working hypotheses and treatment strategies as needed based on those data (Craske, 2010).

Valuing scientific evidence also means that CBT scientists and practitioners are willing to subject their treatments to rigorous empirical tests. Because clinicians are not exempt from cognitive biases that affect humans in general, they may attend to and remember only the shining clinical success stories and selectively attribute client lack of progress to factors other than the treatment itself. As a result, in the absence of objective data, therapists can easily continue to use less-than-optimal strategies. This is why many CBT prac-

titioners value research evidence so highly. Using a treatment that has been shown to work for a significant proportion of clients provides a scientific foundation upon which to base one's practice and also provides a systematic way of improving the treatment over time. Ideally, systematic research isolates the most important "active ingredients" of a treatment (e.g., inhibitory learning during exposure for anxiety disorders; Craske et al., 2008), so that clinicians can better understand when and how to adapt CBT for an individual client while maintaining the core processes associated with treatment-related change.

Given the CBT focus on scientific evidence for efficacy, I later describe two approaches that have been tested using the most rigorous methods thus far—randomized controlled trials and active treatment control conditions. This does not mean that other approaches will not be found to be as effective, or more effective, in the future. It means that these two approaches currently represent the best that we have from the perspective of scientific evidence.

Short-Term

One appealing feature of CBT for some clients, therapists, and managed care organizations is that it is designed to be relatively short-term. CBT, however, is not short-term only for the sake of convenience. Instead, its time-limited nature is an outgrowth of other features more relevant to efficacy—specifically, CBT is goal-directed, structured, and client-empowering. First, CBT is ideally goal-directed (not open-ended), with therapist and client agreeing on what will be the target outcomes of the treatment. Research from many areas of psychology supports the importance of goal setting in enhancing regulation of behavior (Carver & Scheier, 2011; Locke & Latham, 1990). It stands to reason that a goal-directed treatment will be more efficient. In addition, clear treatment goals and ongoing assessment of progress toward those goals allows the therapist to be flexible in altering strategies if progress is not being made.

Second, CBT is structured in a way that directs client and therapist efforts toward the goal. The understanding that treatment will be short term provides an incentive for therapist and client to work efficiently. As such, CBT sessions are usually guided by an agenda, so that time and effort can be focused on the most important treatment targets. Finally, CBT is short term because the overarching goal is to train clients to use skills in the absence of direct support from the therapist (i.e., to "be their own therapist"). Thus, CBT is not short term simply for the sake of making it appealing to stakeholders—rather, it is goal-directed, structured, and ideally designed to render the therapist obsolete as clients gain skills.

Emphasizes Interaction among Thoughts, Emotions, and Behaviors

CBT recognizes the complex interplay of a client's thoughts, emotions, and behavior in the treatment of any clinical problem. Consideration of thought–emotion–behavior interactions is an integral part of the assessment process because it gives the therapist a full picture of how the clinical problem plays out and where interventions should be used. Although CBT approaches are based on general models of how these elements reinforce one another, the therapist must apply the model to each client's specific problem.

In CBT, it is also imperative that the client have a clear understanding of the way the model applies to his or her problem, in order to understand the rationale for specific treatment strategies and recognize when and where to use these strategies in everyday life. Thus, effective psychoeducation is necessary (but not sufficient) for effective cognitive-behavioral treatment. When providing psychoeducation about the CBT model, it is important to use examples collected from the client's actual experience as opposed to simply engaging in a theoretical discussion of how thoughts, feelings, and behaviors might interact. CBT often involves training clients in the metaskill of stepping back and "seeing" these interactions as they occur, so that they can use skills when and where they are needed. Thus, a crucial element is that CBT addresses the role of thoughts, emotions, and behavior in the conceptualization and treatment of clinical problems and includes skills that address these relationships.

Teaches Specific Skills

The key mechanism of change in CBT is that clients learn specific cognitive and behavioral skills that they use to change the way they interact with the environment. In CBT, clients must use skills in their daily lives, not merely discuss them in session. This explains the CBT therapist's emphasis on practice of skills outside of session (i.e., homework assignments). What matters most is whether the client learns to use skills in daily

life. It is not enough for clients to learn *about* skills—they must be able to *demonstrate* skills and apply them flexibly in new situations. Just talking about thoughts and behaviors with a client, or even talking about specific skills, is not necessarily doing CBT. For example, I could have a perfectly lovely conversation with a client about what his or her ideal calendar and task list system would look like. However, as a cognitive-behavioral therapist, I must also focus on how, when, and where the client will implement this system during the next week, assess and target barriers to use of skills, and use other techniques to maximize the likelihood that this skill happens in the "real world." Thus, CBT therapists focus their efforts on helping clients *learn and use* specific skills in their daily life to address their problems.

This point cannot be overemphasized with respect to CBT for adult ADHD. ADHD has been described not as a disorder of "knowing what to do" but as a disorder of "doing what you know"; it is a disorder of performance more than one of knowledge (Barkley, 1997; see Chapter 16). Put a bit differently, ADHD is less a disorder of *attention* and more a disorder of *intention*. Thus, for a skills-based treatment to be successful, it cannot simply educate the client or try to convince him or her that skills might be, in theory, a good idea. Clients with ADHD may wholeheartedly agree that a skill is valuable, completely understand how it would help them, and fully comprehend intellectually the steps to implement the skill, yet still be unable to use it successfully in daily life. Thus, CBT therapists for adult ADHD must constantly incorporate strategies designed to help clients implement skills (Ramsay, 2010). As mentioned earlier, homework assignments in CBT are a good example of a key feature that focuses on implementation. Therapist and client set a specific goal for the week in terms of the client trying out a new skill, including where, when, and how the skill will be implemented and what barriers he or she can anticipate. Of course, following up on the homework each week and troubleshooting are critical to maximizing results.

Consistent with the importance of skill use as a mechanism of action in CBT, in past reviews, Steven Safren and I have hypothesized that the active ingredient in CBT for adult ADHD is the extent to which clients *implement* new skills to compensate for their ADHD-related deficits (Knouse & Safren, 2010, 2013a). Psychosocial approaches for adult ADHD, including those described as CBT, seem to vary quite a bit in terms of the extent to which they focus on implementation of specific skills versus psychoeducation on a broader range of topics. For example, some treatments include one or two sessions devoted to topics not directly related to compensatory skills, including substance abuse, depression, anger management, communication, and relationships. While it may be important for clients to better understand the ways that their ADHD impacts these areas and to reduce the self-blame associated with those problems, it seems unlikely that clients will learn and implement specific skills in these areas based on a few topical discussions—especially given that these problems are complex and likely tied up in the functional impairment that clients are experiencing as a result of ADHD symptoms. Again, this is not to say that psychoeducation is not important or potentially therapeutic, but psychoeducation without skills use and behavior change does not reflect the principles of CBT.

Importantly, empirical evidence thus far appears to support the importance of a focus on skills in the treatment of adult ADHD. CBT approaches for adult ADHD with the strongest empirical support focus on teaching clients to use specific compensatory skills consistently to ameliorate symptom-related deficits (Knouse & Safren, 2013a), and treatments with these characteristics also appear to demonstrate the largest effect sizes (Knouse & Safren, 2010). Relatedly, a few studies show a positive relationship between skills practice outside of session and symptom improvement. Both homework completion and a stronger relationship between weekly homework completion and symptom reduction have been shown to predict better treatment outcome (Solanto et al., 2010; Yovel & Safren, 2007). Skills include both behavioral strategies to improve self-regulation and cognitive reappraisal to increase the likelihood of effective coping in the presence of negative emotions.

Assessing Treatment According to CBT Principles

Based on this discussion of CBT treatment principles, below is a list of questions that clinicians may use to assess treatment approaches and to engage in self-assessment regarding their own practices. Even experienced CBT therapists have sessions that go off track or have particular clients with whom it is especially difficult to stay focused on skills implementation. Asking the following questions may help both new and experienced clinicians to focus their work toward the goals of CBT:

- Has the treatment I am using been tested empirically? How rigorous were the tests? How large were the effects?
- Does my treatment train clients in specific skills to address their ADHD-related deficits, as well as provide the means by which to implement those skills in daily life?
- Is my treatment focused on helping the client reach specific goals that are meaningful to him or her?
- Am I collecting data throughout therapy to evaluate progress?
- Is the structure of the treatment—including format, session structure, and homework assignments—consistent with the goal of helping the client learn skills?
- Does treatment incorporate the interplay of thoughts, feelings, and behaviors in the client's difficulties?
- At the end of treatment, what will the client be *doing differently*?
- How likely is it that my treatment methods will lead to that behavior change?
- Am I teaching my clients how to "be their own therapist?"

WHY CBT?

Addressing Common Concerns

This section focuses on building the case for CBT as applied to the problems of adults with ADHD. It begins by addressing three common objections that might be raised in response to the idea of using this approach to address the core symptoms of the disorder.

1. *ADHD is a neurobiological disorder, so a nonmedication treatment won't work.* First, it is important to establish that just because a condition is "biological" or largely dependent on heredity and underlying neurobiology does not preclude environmental or behavioral intervention. A good counterexample makes this point obvious. Children with phenylketonuria (PKU) have a genetic disorder in which their bodies do not produce a functioning version of the enzyme that breaks down the substance phenylalanine. If they ingest this substance, it accumulates in their bodies and causes untoward neurological outcomes, including mental retardation. The key intervention for PKU is purely environmental and behavioral in nature: Eliminate foods containing phenylalanine from the child's diet (hence, the warnings printed on cans of diet soda.) Similarly, because the functional impairment associated with ADHD arises from the *interaction* of the client's neurobiology with his or her environment, modifications to the environment and the client's behavior may be helpful in ameliorating negative consequences experienced by the adult with ADHD, as they have been for children with the disorder (Fabiano et al., 2009).

2. *Medications for ADHD are already effective for adults.* Although medications are a crucial treatment for many adults with ADHD, they may not always be enough. Some adults are unwilling or unable to take medications. Some clients are medication nonresponders, and many others may experience significant residual symptoms even with efficacious medication treatment. In many studies, a 30% reduction in ADHD symptoms categorizes a patient as a "responder" (Steele, Jensen, & Quinn, 2006), so adults with high levels of baseline symptoms before treatment may continue to be quite impaired even with medication. Furthermore, even if a client does experience significant symptom reduction from medications in adulthood, growing up with ADHD may have impeded the development of self-management skills necessary to meet his or her current goals (Safren, Sprich, Chulvick, & Otto, 2004).

3. *Didn't people try CBT on kids with ADHD in the 1980s and it didn't work very well?* Yes, but response to CBT may be age-related, with adults having sufficient neuropsychological development, especially of their executive functions, to benefit from CBT. More importantly, CBT for adult ADHD is meant to train clients in compensatory skills that help to ameliorate—not cure—ADHD-related deficits. This is an extremely important distinction to make given that these earlier attempts at "cognitive training" for children with ADHD turned out to be largely unsuccessful (Abikoff, 1991). Those approaches (e.g., self-instructional training) were predicated on the idea that children with ADHD could be trained to use self-instructional statements that would allow them to engage in reflective problem solving, and that this nonspecific skill would modify cognitive processes, generalize to new settings, and reduce impulsive responding. In contrast, current CBT approaches for adults with ADHD do not purport to change the underlying processes that produce symptoms; instead, they help clients learn specific strategies

to work around their inattentive symptoms. In other words, the effects of the treatment are not assumed to extend beyond the boundaries of clients' use of specific compensatory skills. Notably, this is consistent with CBT for other conditions. The skills clients learn do not directly "get rid of" anxiety or depression; rather, they disrupt the cognitive and behavioral chains of events that maintain those conditions and lead to functional impairment. Furthermore, the most efficacious CBTs for adult ADHD include procedures specifically designed to enhance skill use in the face of ADHD-related deficits such as repetition to automaticity and placing behavioral cues for skills at critical points in the environment and in time.

The CBT Model of ADHD

Having addressed some common objections, the following sections more fully outline the theoretical basis and empirical evidence for CBT in adult ADHD. The CBT model of adult ADHD is based on the fact that ADHD is a neurobiological disorder with core symptoms that impact executive functioning (Chapters 10 and 16; also see Ramsay, 2010; Safren et al., 2010; Solanto et al., 2010) and sensitivity to reinforcement (Solanto et al., 2010). Over time, the interaction of these core symptoms with the environment results in functional impairment in multiple domains. For example, Safren and colleagues (2004) emphasize the secondary effects of core ADHD symptoms over time, including deficits in self-management skills (behavioral) and patterns of dysfunctional depressogenic or anxiolytic thinking (cognitive) that get in the way of using skills. Chronic perceived failure experiences contribute to patterns of avoidance and demoralization that reinforce functional impairment.

Skills-based CBT is intended to disrupt this cycle. Clients learn basic compensatory strategies to organize their lives, manage their time, and motivate themselves. They also learn to recognize and counteract the thought patterns that lead to avoidance of skill use. Efficacious approaches emphasize that learning skills will be harder for adults with ADHD than for people without the disorder because core symptoms can interfere with skills acquisition. For example, forgetfulness associated with ADHD can often get in the way of key skills, such as using a daily task list. The client may forget to look at the list or forget where he or she put it. Thus, CBT approaches emphasize implementation strategies to cue skill use—for example, tying list use to other events that occur during the day or changing the location and format of the task list to reduce the likelihood of it getting lost. As such, the importance of altering the environment to reduce cognitive workload and support use of skills is also emphasized (Ramsay, 2010). On the cognitive side of the model, CBT approaches also include intervention components that identify and target the client's demotivating assumptions and interpretations. In addition, as clients experience small successes in working around their ADHD symptoms, this motivates further skill use and disproves their negative assumptions about their inability to meet important goals, providing motivation for further adoption of skills.

Empirical Evidence

Empirical evidence has been accumulating at an encouraging rate across the short history of CBT for adult ADHD since the first retrospective chart review study by Wilens and colleagues (1999). One way to describe the magnitude of effects in treatment outcome studies is to calculate, compare, and summarize effect sizes (e.g., Cohen's *d*, the standardized mean difference). Published open trials (i.e., with no control group) of CBT and other psychosocial approaches for adult ADHD show, on average, medium to large effect sizes from pre- to posttreatment (Knouse & Safren, 2010). However, there has been wide variability in the size of outcomes among the various treatment approaches with pre- to posttreatment effect sizes ranging from small to very large.

Although positive results from open trials are important in beginning to establish evidence for efficacy, randomized controlled trials (RCTs) are a more scientifically rigorous method to test whether specific treatment techniques account for observed improvements in symptoms. In its policy statement on evidence-based practice in psychology (APA Presidential Task Force on Evidence-Based Practice, 2006), the American Psychological Association supports systematic review of RCTs as the highest-quality evidence of clinical efficacy. Among RCTs, waiting-list or treatment-as-usual control groups are considered less rigorous than comparisons with active, attention-matched controls groups (Chambless, 1998).

How do CBT approaches fare in RCTs? Compared to waiting-list or treatment-as-usual controls, between-groups effect sizes for ADHD symptoms have ranged from medium to very large (0.76–1.72; Emilsson et al.,

2011; Safren, Otto, et al., 2005; Stevenson et al., 2002). In RCTs that compare CBT to an active, time- and attention-matched control condition, it is expected that effect sizes will be smaller, since nonspecific effects of therapy in the control group are likely to have some impact on symptoms. In three published RCTs comparing CBT for ADHD to either group supportive therapy or applied relaxation training (Hirvikoski et al., 2011; Safren et al., 2010; Solanto et al., 2010), effect sizes of medium magnitude compared to control are reported (0.53–0.57). For the sake of comparison, a meta-analysis of CBT for anxiety disorders summarizing placebo-controlled RCTs yielded an average effect size of 0.73 (Hedge's *g*; Hofmann & Smits, 2008). Thus, the effects of CBT for adult ADHD observed so far compare favorably with more established CBTs for other disorders.

The American Psychiatric Association (APA) Division 12 Task Force on Empirically Supported Treatments (Chambless, 1998) developed criteria to evaluate quality of evidence for psychosocial treatment approaches. Considering CBT approaches together, the APA Division 12 website (2013) for empirically supported treatments labels CBT for adult ADHD as having "strong research support" and therefore meeting criteria as an empirically supported treatment. If distinguishing among CBT treatment approaches, two currently have higher levels of evidence for their efficacy than others: Safren, Perlman, and colleagues' (2005) CBT for medication-treated adults with ADHD and residual symptoms and Solanto and colleagues' (2010) group CBT for adult ADHD. Because these manualized treatments have shown positive results in RCTs compared to active treatment control conditions, they each appear to meet the criteria for "probably efficacious" treatments[1] (Chambless, 1998; Knouse & Safren, 2013a). A second successful RCT compared to active control of either treatment conducted by an independent research group would be necessary to raise the individual classification to "empirically supported."

Research evaluating CBT for adult ADHD has become increasingly rigorous with time and hopefully this trend will continue. As findings from psychosocial treatment studies are published, it is important to critically evaluate the rigor of the study design in understanding the effects that are reported. As mentioned earlier, average effect sizes mask heterogeneity among the effects of various approaches. When reading treatment outcome research, the presence of certain study characteristics can increase one's confidence that the treatment was responsible for the effects observed in the study. Readers may place their highest levels of confidence in studies with the following characteristics: randomization to groups; comparison to active, attention-matched controls; clear a priori hypotheses about which effects will be observed on what measures; reporting of intent-to-treat analysis (i.e., results from all participants randomized to treatment, not just those who completed the study); use of reliable and valid assessment tools for ADHD symptoms; assessment of symptoms using a method other than self-report (e.g., assessor blinded to treatment status); and reporting of results at follow-up intervals after treatment has concluded (to gauge durability of effects). Of course, this does not mean that results of studies without these characteristics do not "count," just that those with these features may be considered particularly robust.

The next section provides a more detailed description of the structure and content of the two CBT approaches that currently have the most empirical support.

TWO APPROACHES TO CBT

Although a detailed review of all cognitive-behavioral approaches for adult ADHD is well beyond the scope of this chapter, this section provides descriptions of two treatment programs—one individual and one group. As described earlier, each has demonstrated efficacy in a larger RCT when compared to an active treatment control group. Readers who are interested in learning more about or implementing these approaches or any CBT approach should also obtain and study the treatment manuals, as well as seek out additional training and supervision.

Individual CBT for Medication-Treated Adults with Residual Symptoms

Recognizing that many adults who take medications for ADHD continue to experience difficulties, Safren and colleagues (Safren, Otto, et al., 2005; Safren, Perlman, Sprich, & Otto, 2005) developed a cognitive-behavioral approach specifically designed to meet the needs of this population. The treatment comprises three core skills modules: organization and planning skills, distractibility reduction skills, and skills to address dysfunctional thought patterns. Two optional modules include a session designed to rally the support of the client's signifi-

cant other and a session on applying previously learned skills to the problem of procrastination. An earlier version of the treatment also included optional modules on communication skills and anger management; however, Safren, Otto, and colleagues (2005) reported that these modules were only selected by a minority of clients.

Individual sessions follow a typical CBT session structure: setting an agenda, reviewing self-reported ADHD symptoms from the prior week, reviewing the results of previous skills practice (homework), introducing new skills material, troubleshooting possible barriers, and setting the next assignment. Importantly, medication adherence is also tracked weekly, and barriers to medication adherence are addressed using skills learned in the program. An accompanying client workbook contains psychoeducational information, notes, and homework assignments for each session of the treatment (Safren, Sprich, et al., 2005).

The treatment begins with psychoeducation using a CBT model of adult ADHD, setting concrete and reasonable goals for treatment, and addressing motivation for change. Clients are taught that repetition and troubleshooting of skills will be necessary so that they can fully integrate the new behaviors into their lives and see the benefits. The first core skill[2] is introduced at the end of the first session—a calendar and task list system, which serves as a foundation for the skills to follow. The organization and planning skills module proceeds with strategies to prioritize, break down large and daunting tasks, use problem-solving skills, and organize papers and mail. The second core module of distractibility reduction begins by having clients collect data about their attention span and structure tasks accordingly. Reducing distractions in the environment and using visual and auditory reminders are other key skills in this module. The third core module, adaptive thinking, uses traditional cognitive restructuring strategies (Beck, 1995) to help clients begin to recognize when overly negative thoughts and assumptions might be blocking their use of skills and increasing avoidance. Clients are then taught to question these automatic thoughts and develop more realistic and motivating ways of thinking. Clients have the option of completing a session that applies previously learned skills to the problem of procrastination. In addition, they have the option of a session involving a significant other or family member, so that this person can learn about the treatment and discuss how they might help to support the client's efforts at behavior change.

Clinical efficacy of this approach is supported by two RCTs. The first was a trial of 31 medication-treated adults with ADHD and continued symptoms, randomized to either CBT or continued medication only (Safren, Otto, et al., 2005). CBT was associated with significant ADHD symptom reduction, as rated by blinded investigator and self-report with very large effect sizes compared to continued medication alone (Cohen's d = 1.2–1.7). The second RCT of 86 patients taking medication compared CBT to an attention-matched control group that received sessions of relaxation training applied to ADHD symptoms (Safren et al., 2010). This study was a more rigorous test of the specific efficacy of the skills taught during CBT. CBT was associated with significantly greater reductions in blinded investigator-rated and self-reported ADHD symptoms (Cohen's d = 0.52 and 0.77 at posttreatment compared to control), and there were more treatment responders in the CBT group (e.g., 67 vs. 33% by self-report). Importantly, responders and partial responders to CBT maintained their gains at 6- and 12-month follow-up, showing durability of effects.

Group CBT for Adult ADHD

Mary Solanto and her colleagues (Solanto, 2011; Solanto et al., 2008) developed a group CBT[3] designed to help clients develop executive self-management skills to compensate for core neuropsychological deficits that underlie the inattentive symptoms of ADHD. Weekly 2-hour group sessions train clients in time management, organization, and planning of a longer-term project. This order of skills presentation allows clients to work first on skills that apply to basic daily tasks, then master skills relevant to more complex self-management. Clients also learn to address motivation by using self-reward and by learning skills to recognize, challenge, and restructure depressive or anxious cognitions that block skill use.

Importantly, the treatment incorporates several elements designed to help clients implement skills in daily life (Solanto, 2011). First, a full hour of each 2-hour group session is devoted to reviewing the at-home practice assignment from the previous week. This allows for extensive troubleshooting and provides clients with an opportunity to receive feedback and positive reinforcement from other group members for their behavior change efforts. Second, each take-home practice activity is guided by a structured worksheet that includes session notes, instructions, and prompts for

clients to use in evaluating the results of the activity. Third, the treatment makes use of maxims or mantras that are invoked repeatedly throughout the treatment to help clients remember to use skills in key situations (e.g., "If you're having trouble getting started, then the first step is too big"). Finally, clients are encouraged to actively visualize the longer-term positive outcomes of using their skills in order to increase motivation. Each of these elements is designed to increase the likelihood that clients will successfully implement skills in daily life and generalize them to new situations.

Clinical efficacy of this approach is supported by an open trial (Solanto et al., 2008) and an RCT (Solanto et al., 2010). In the open trial, 38 participants with ADHD who varied in medication status completed an 8- or 12-week version of the group CBT. Self-reported DSM-IV inattentive symptoms decreased significantly from pre- to posttreatment, with a very large effect size (Cohen's d = 1.22), and self-reported organization and planning skills increased by a similar magnitude (Cohen's d = 1.11). Outcomes did not depend on medication status. Solanto and colleagues (2010) followed up this open trial with a larger RCT (N = 88) comparing group CBT to a support group control condition. CBT was associated with significantly greater reductions in blind investigator-rated DSM-IV inattentive symptoms (Cohen's d = 0.55 at posttreatment compared to control) and inattention and memory problems as reported by a significant other (Cohen's d = 0.57). For self-reported inattention and memory problems, level of pretreatment symptoms interacted with treatment group such that participants with the highest levels of symptoms at baseline experienced the greatest benefit from CBT over and above supportive therapy. Again, in this study medication status did not moderate any of the observed effects.

As illustrated by these treatment approaches, CBT is designed to help adults with ADHD acquire and implement skills in their daily lives that help to compensate for their symptom-related deficits and maintain their motivation to apply skills over time. The next section provides a few clinical recommendations for helping adults with ADHD learn and use skills in the context of CBT.

HELPING ADULTS WITH ADHD USE SKILLS

The preceding sections of this chapter have focused on the "what" of CBT for adult ADHD—the theoretical rationale for its use, the empirical evidence thus far, and a description of two key approaches. This section offers some recommendations on the "how" of CBT for adults with ADHD designed to increase the likelihood that clients acquire and implement skills to compensate for their ADHD symptoms, improve their functioning, and achieve their goals. These recommendations are based on elements from empirically supported approaches described earlier, from the clinical literature on CBT for adult ADHD, and from clinical experience in this emerging area of practice.

Before addressing specific recommendations, it must be emphasized that the therapist's interpersonal skills and the quality of the therapeutic relationship are exceptionally important. CBT therapists are sometimes wrongly characterized as rigidly adhering to protocols and not valuing a collaborative working relationship to the same extent as therapists trained in other orientations. Nothing could be further from the truth. CBT therapists invite their clients into the risky process of learning new ways of thinking and behaving. They ask their clients to trust them as they work together to figure out the most effective tools. For this process to work, it is imperative that the therapist have a flexible, nonjudgmental problem-solving orientation, viewing client difficulties in treatment as a normal part of the behavior change process and framing each successive approximation of a new skill as a sign of progress. Skilled CBT clinicians must bring all of their interpersonal skills and capacity for empathy to bear on their work with adults with ADHD, and the best CBT therapists are expert teacher–motivators who can flexibly apply the cognitive-behavioral model to each individual client.

The recommendations offered here do not constitute a comprehensive discussion of how to conduct CBT for adult ADHD. For more extensive clinically oriented guidance, readers should access the aforementioned treatment manuals and the excellent book on CBT for adults with ADHD coauthored by J. Russell Ramsay and Anthony Rostain (2008). Two other recent practice-oriented chapters on the subject may also be of interest (Knouse & Safren, 2011, 2013b). In addition, the reference section at the end of this chapter has been annotated to provide even more guidance on particularly helpful resources for this area of practice.

Readers are also encouraged to seek out training opportunities and supervision to develop their skills. Adults with ADHD, because of their deficits in executive functioning and self-regulation (Chapters 10 and

16), can pose a significant challenge to one's skills as a CBT clinician—particularly in keeping treatment structured, goal-directed, and on track. However, working with adults with ADHD who are ready for change can be extremely rewarding—particularly when clients experience success via an approach tailored to their needs after years of struggling to manage their symptoms on their own.

Three recommendations are offered here for helping clients acquire and—most importantly—*use* CBT skills in their daily lives.

Do More with Less (Content, That Is)

Asking an adult with ADHD to learn self-regulation skills is no small request, and a skilled CBT therapist understands that a client with ADHD will have to work much harder than a person without ADHD to acquire and consistently use compensatory skills. This is a clinical population for whom the "spaghetti at the wall" approach (i.e., throwing out a lot of content and "seeing what sticks") is particularly ill-advised. Fully integrating a particular skill into the behavioral repertoire may take several iterations and clients (and therapists!) can become overwhelmed if too much information and too many skills are presented too quickly. As mentioned earlier, the most successful CBT treatments thus far maintain a laser-focus on skills implementation rather than including a wider range of psychoeducational content.

Clinicians may want to consider covering fewer skills but spending more time in treatment "locking them in." The choice of which skills to include must be based on a detailed assessment of the client's most problematic symptoms and functional impairment. For example, in her treatment manual, Solanto (2011) includes instructions for adapting group CBT for adult ADHD to an individual format, including assessment tools to aid therapists in choosing the most relevant skills to target. Fewer well-learned and consistently implemented skills effect more positive change in the client's life than lots of content that never leaves the therapist's office.

Be Intentional about Implementation

To help clients with ADHD successfully apply skills in daily life, the CBT therapist must constantly be questioning when, where, and how the client will implement the to-be-learned skill and building in intervention components that cue and support skills application. Discussions about skills are of no use in CBT if they do not actually lead to use of skills. When planning homework assignments for skills practice, the therapist should help the client identify the specific situations and times where the skill will need to be used. This includes having a specific location for any tools that are needed (e.g., daily planner, task list), preferably at the "point of performance" (Chapter 16; Barkley, 1997, 2012; Barkley, Murphy, & Fischer, 2008) or close to the location where the client needs to perform the skill. If possible, the client should practice using the skill during the session as much as possible before he or she is required to use it in the "real world." Finally, when presenting each homework assignment, the therapist should ask the client what is likely to get in the way of successfully using the skill. If barriers are identified in advance, then strategies can be included to address them before they derail the client's change efforts.

It is equally important that the therapist diligently follow up on each skills practice assignment the following session. When assigning homework, it may be helpful for therapists to keep in mind a maxim adapted from behavioral parent training: Don't give a homework assignment that you don't intend to follow up on. Therapist and client should take the time to thoroughly assess and troubleshoot skills attempted the previous week, modifying strategies and reassigning practice as necessary. Detailed follow-up is especially important when the client does not complete the assignment, as it can provide important information on motivational barriers to skill use.

As far as specific strategies to aid implementation and generalization, some elements of current manualized treatments that have been discussed earlier include maxims or mantras, workbooks or structured homework sheets for skills practice, and use of self-reward and cognitive restructuring to address emotional and motivational barriers. Another important strategy is to place cues for skills use at key points in the client's physical environment or at critical points in time (Safren, Perlman, et al., 2005). For example, a client might set a cell phone alarm to go off daily at a specific time when he or she is likely to get off task, and use it as a cue to look at the task list. Finally, and perhaps most importantly, the client is most likely to experience lasting behavior change if he or she is supported in practicing new skills repeatedly, until they become habitual and an integral part of his or her daily life.

Assess Avoidance

Avoidance is at the behavioral core of disorders such as anxiety and depression[4] (Ferster, 1973; McNally, 2007), and it is addressed directly by CBT for those disorders (e.g., exposure; behavioral activation). The role of avoidance in the problems experienced by adults with ADHD has been discussed in prior clinical writings (Mitchell, Nelson-Gray, & Anastopoulos, 2008; Ramsay, 2002) and when manuals address the behavioral effects of negative automatic thoughts (i.e., reducing the likelihood that clients will use skills). Nonetheless, the role of cognitive and behavioral avoidance of aversive emotional states in adult ADHD may be underappreciated. Difficulty tolerating unpleasant thoughts and feelings ("experiential avoidance" in the language of acceptance and commitment therapy, Hayes, Strosahl, & Wilson, 2012) may motivate adults with ADHD rapidly shift attention and intention away from the provoking stimulus. Unfortunately, in daily life, important tasks often provoke unpleasant feelings, at least initially. Aversive states triggered by everyday experiences include anxiety, self-doubt, boredom, impatience, frustration, helplessness, and feeling overwhelmed. Due to either their learning history of past failure experiences (Ramsay, 2010; Safren et al., 2004) or symptom-related problems with delay aversion and distress tolerance (Sonuga-Barke, 2003), adults with ADHD may be more likely than other adults to respond to fleeting aversive states by moving attention elsewhere, engaging in alternative activities, or thinking overly optimistic thoughts (i.e., cognitive and behavioral avoidance) (Knouse & Mitchell, in press; Knouse, Zvorsky, & Safren, 2013; Sprich, Knouse, Cooper-Vince, Burbridge, & Safren, 2010). As a result, behaviors that might otherwise be described as distractibility, forgetfulness, or even impulsivity may be the result of attempts to avoid unpleasant feelings (Knouse & Safren, 2013b).

Clinicians should consider assessing for the potential role of avoidance when their clients experience difficulty implementing CBT skills in daily life and instead engage in off-task behaviors. Key questions to ask include the following:

"What feelings come up when you think about doing that task?"

"What runs through your mind when it comes time to use that skill?"

"When you do it the old way, what do you *not* have to deal with?"

"What feelings do you find it the hardest to just sit with and not push away from?"

The best way to get accurate answers to these questions is probably to have clients self-monitor their reactions in daily life. Tools from the cognitive restructuring modules of manualized treatments are especially useful in facilitating self-monitoring. If it becomes clear that avoidance of negative private experiences is derailing a client from using skills, therapist and client can work together to increase awareness of these vulnerable situations and develop techniques to promote active coping (Knouse & Mitchell, in press). For example, the client could formulate an implementation intention that states what the client will do when he or she notices the aversive feeling or thought (i.e., engage a particular skill) (Gawrilow, Gollwitzer, & Oettingen, 2011; Ramsay, 2010). Or consider the maxim used in Solanto's (2011) manual: "If I feel overwhelmed, then the first step is too big"—an excellent example of a specific skill linked to an aversive emotion. Depending on the client and the situation, other versions might include "If I feel like I'm drifting, then I need to look at my task list," "If I feel like I want to escape, then I may need a short (timed!) break," or, "If I feel deprived, then I should choose a reward for myself when I complete the task at hand."

Assessing the role of avoidance and folding it into the treatment strategy is another way to promote the client's implementation of skills in daily life.

ON THE CBT HORIZON

CBT for adult ADHD is still in the early stages of development compared to approaches for other disorders. This section highlights CBT research directions that are taking shape and provides recommendations for future directions that would improve CBT outcomes and access to care.

Mindfulness

Mindfulness-based approaches to psychotherapy have shown promising results in people with anxiety and depression (Hofmann, Sawyer, Witt, & Oh, 2010), and

mindfulness techniques are being applied to a growing variety of clinical problems. For emotional disorders, it is hypothesized that the ability to be nonjudgmentally aware of experiences in the present moment facilitates emotion regulation by giving clients the opportunity to reappraise automatic maladaptive thoughts and divert emotion-driven behaviors (Barlow et al., 2011). Zylowska and colleagues (2008) conducted an open feasibility study of mindfulness meditation training for adolescents and adults with ADHD, hypothesizing that mindfulness practice might improve attention and executive functioning skills, and emotion regulation. Study completers self-reported significant reductions in ADHD symptoms at posttreatment (d = 0.80). A recent pilot RCT compared group mindfulness meditation training to a waiting-list control group and found very large effect sizes for self-reported and clinician-rated ADHD inattentive and hyperactive–impulsive symptoms and emotion dysregulation (Mitchell et al., in press). Furthermore, adaptations of DBT skills training (Linehan, 1993) for adult ADHD also incorporate mindfulness skills, along with other, more traditional CBT skills.

The recent increase in research attention to mindfulness for adult ADHD is encouraging and establishes a stronger empirical basis on which to recommend these skills as part of CBT for adult ADHD. In addition, future studies should focus on better understanding the mechanism of action of mindfulness skills for adult ADHD. For example, these skills may exert their effects via improved emotion regulation (see Chapter 3) or greater awareness of avoidance-motivated behavior. Importantly, although there may not yet be enough evidence to support mindfulness meditation as a monotherapy for adult ADHD, there is ample reason to integrate mindfulness practice into a comprehensive treatment program for clients with ADHD who also experience difficulties with stress management or comorbid internalizing symptoms.

Dissemination and Effectiveness Research

Although additional controlled trials may be needed, research on CBT for adult ADHD must also begin to focus on testing the effectiveness (i.e., external validity; ecological validity) of manualized approaches in "real-world" clinical practice settings (Knouse & Safren, 2013a) and with samples of clients that are more representative of the entire population of adults with ADHD (Knouse & Safren, 2011). Researchers should also evaluate what level of training and supervision therapists in the community need to deliver CBT for adult ADHD in a way that results in optimal response rates. Is studying the therapy manual or attending a workshop enough, or do clinicians need to be supervised by an expert clinician to be effective? Research could also investigate whether lower-intensity versions of the treatment delivered in alternative formats (e.g., online) might be a cost-effective treatment for less impaired clients. As with CBT for other disorders (McHugh & Barlow, 2010), improving access to treatment and optimizing it for the "real world" are important next steps in refining CBT for adult ADHD.

Adapting CBT for Comorbidity

Because a large proportion of adults with ADHD also meet criteria for other psychological disorders, including mood, anxiety, and substance use disorders (Kessler et al., 2006; Miller, Nigg, & Faraone, 2007), clinicians need additional guidance on how best to address comorbidity in therapy. CBT for adult ADHD may be combined with CBT approaches for other disorders and, in some cases, there is overlap in the skills recommended for each disorder (e.g., as in CBT for depression; Knouse et al., 2013). For example, van Emmerik-van Oortmerssen and colleagues (2013) are conducting an RCT of CBT for adults with comorbid ADHD and substance use disorders. They are comparing the efficacy of an empirically supported CBT for substance use disorders by itself versus combining substance abuse treatment with Safren, Perlman, and colleagues' (2005) approach for adult ADHD. Hypothesizing that ADHD symptoms contribute directly to problems with substance use, the researchers are particularly interested in whether adding treatment of ADHD enhances the impact of CBT on substance use. This intriguing study is one example of how CBT for ADHD might be integrated with treatments for other disorders and potentially enhance their effects.

Adapting CBT for Specific Settings

Current CBT approaches for adult ADHD were developed for the general adult population, but skills could also be tailored to the unique needs of adults with ADHD in specific settings. For example, Fleming, McMahon, Moran, Peterson, and Dreesen (in press) recently completed a pilot RCT (N = 33) of group DBT skills training adapted for the unique developmental

needs of college students with ADHD (Fleming & McMahon, 2012). Compared to students who received self-guided skills handouts, participants in the 8-week group showed significantly greater improvements in executive functioning on the Brown Attention-Deficit Disorder Scales, greater improvements in quality of life, and a trend toward significantly greater reductions in self-reported DSM-IV inattentive symptoms. In addition to these promising findings, other treatment studies being conducted at the time of this writing are evaluating adaptations of CBT for adult ADHD for college students, and these results should be available in the next few years.

In addition to targeting higher-functioning young adults with ADHD who have been able to enroll in college, CBT researchers should also investigate whether their treatments can be adapted to better meet the needs of young adults who do not attend college (the majority) and who better represent the general population of adults with ADHD in the community. Furthermore, because adults with ADHD are overrepresented in the prison population (Rösler et al., 2004), CBT should be adapted to that setting. Additional settings that might be appropriate for specialized CBT for adult ADHD include vocational rehabilitation programs and child psychopathology clinics where adults with ADHD may be identified via their child receiving an ADHD diagnosis, and where child-focused ADHD interventions may need to be adapted for parents who themselves have ADHD (Chronis-Tuscano et al., 2011).

Developing New Funding Sources

Compared to federally funded research studies of psychosocial treatment for other disorders, funding for adult ADHD has been surprisingly sparse considering the prevalence rate of the disorder in the U.S. population (4.4%; Kessler et al., 2006) and its strong association with multidomain functional impairment. Only two R01 research grants investigating CBT for adult ADHD have been funded, and a recent perusal of *clinicaltrials.gov* did not identify any additional studies in progress. Given the general tightening of federal resources for scientific and medical research, it makes sense to seek creative ways to fund the next phase of innovations in CBT for adult ADHD. For instance, private sources, such as foundation grants, may also fund an increasing proportion of studies in the future and even "crowdsourced" funding from many small donors may not be outside the realm of possibility.

Proliferation, Refinement, Collaboration

An informal count identifies at least 12 distinct psychosocial treatment programs for adult ADHD described in the research literature, with additional approaches in the clinical literature. In the early stages of a new treatment paradigm, proliferation of different approaches is expected as clinicians and clinical researchers develop their own strategies for filling the gap. As CBT for adult ADHD enters the next phase of development, the benefits of increasing diversity of approaches (e.g., additional innovations in treatment content) can be balanced with refinements to existing programs and adaptation to new populations and settings. Importantly, studies that isolate core "active ingredients" of CBT for adult ADHD could make treatments more efficient and exportable. Finally, more frequent consultation and collaboration among research groups would help to coordinate limited resources in the most efficient way and might speed progress in the development of the best possible CBT for adult ADHD. More collaboration between clinical researchers and clinicians who focus on adults with ADHD in their outpatient practice would aid in the design and execution of dissemination and implementation studies. At this early point in the development of CBT for adult ADHD, our collective next steps could make an immense difference in the pace of future progress.

KEY CLINICAL POINTS

- ✓ CBTs rely on a scientific understanding of the interaction of thoughts, emotions, and behaviors with the environment in the maintenance of psychological disorders. These treatments are frequently subjected to empirical tests to evaluate their efficacy.
- ✓ These structured, goal-directed treatments are designed to train clients to become their own therapists by learning to use specific cognitive and behavioral skills in daily life.
- ✓ It is hypothesized that the "active ingredient" in CBT for adult ADHD is clients' implementation of compensatory skills that ameliorate their symptom-related deficits.
- ✓ Focusing on CBT strategies that enhance implementation of skills is especially important for adults with ADHD because even when they understand what to do, they have difficulty doing it.
- ✓ Cognitive-behavioral models of ADHD emphasize the

deleterious effects of core neurobiological symptoms on acquisition of self-management skills and on clients' automatic appraisals, which themselves have a negative impact on motivation to use skills.

- ✓ Empirical evidence for psychosocial treatments with cognitive-behavioral elements is promising, averaging large effect sizes in open trials and medium to very large effects in comparisons with treatment as usual. However, there is considerable variability in the magnitude of effects observed across trials.
- ✓ More recently, larger RCTs comparing CBT with active treatment controls have yielded significant effects of medium magnitude (Safren et al., 2010; Solanto et al., 2010). These studies appear to qualify CBT for adult ADHD as an empirically supported treatment (APA Division 12, 2013).
- ✓ Clinical recommendations for helping clients with ADHD use CBT skills include focusing more time and effort on fewer skills, a consistent focus on strategies to aid implementation, and consideration of the role of avoidance in failure to use skills.
- ✓ New frontiers in CBT for adults with ADHD include mindfulness skills, tailored approaches for comorbidity and specialized settings, and enhancing dissemination and effectiveness through clinical research collaborations.
- ✓ Clinicians are encouraged to learn more by reading published treatment manuals, accessing the resources described in the annotated references section, and seeking out additional training opportunities.

NOTES

1. The study of group dialectical behavior therapy (DBT) for adult ADHD by Hirvikoski et al. (2011) may also qualify it as a "probably efficacious" treatment; however, the use of only self-report outcome data and lack of significant findings using intent-to-treat analysis suggest some caution in interpreting the results. Forthcoming data from a large RCT of DBT for adult ADHD alone and combined with medication compared to medication alone (Philipsen et al., 2010) may provide further evidence for DBT for adult ADHD as an empirically supported treatment.
2. For several of the skills described in this section, video clips of role-play demonstrations are available online by accessing the article by Sprich, Knouse, Cooper-Vince, Burbridge, and Safren (2010).
3. This treatment was originally named "metacognitive therapy," as it is called in the cited treatment outcome studies, but in the published manual it is now simply referred to as CBT for adult ADHD.
4. Anxious clients engage in behaviors designed to escape anxiety-provoking stimuli in the short-term, which paradoxically maintains anxiety in the long-term. Depressed clients become locked in a cycle in which escape-motivated behaviors replace reward-motivated behaviors, reducing access to reinforcers and further narrowing the behavioral repertoire.

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Annotations are included for some entries (in **bold**).

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CHAPTER 33

Assessment and Management of ADHD in Educational and Workplace Settings in the Context of ADA Accommodations

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Evaluating individuals for attention-deficit/hyperactivity disorder (ADHD) poses challenges for clinicians under any circumstances. The absence of biological and behavioral markers, the logistical burdens inherent in gathering information from external sources, symptoms that are common to many disorders as well as human nature, and the sheer vagueness of diagnostic criteria all make for a daunting process. These challenges have become all the more formidable now that ADHD represents a potential basis for seeking accommodations under the Americans with Disabilities Act of 1990 (ADA). Indeed, conducting ADA-related assessments demands specialized knowledge about the legal definition of disability, the nuances of documentation requirements, and the complexities involved in establishing what constitutes a reasonable accommodation.

Beyond these technical issues, the proper evaluation of ADHD in this quasi-forensic context pushes clinicians to adopt a different mindset than the one that typically guides their diagnostic process. Understanding this shifting of roles when the goal is a plausible disability determination represents an important step toward producing a credible evaluation.

Our aim in this chapter is to provide clinicians with the core concepts underlying ADHD evaluations conducted for the purpose of determining disability and reasonable accommodations. Understanding these principles from the outset helps diminish the confusion that can emerge whenever a practitioner ventures out of the clinical world into the legal one. We also present detailed information about the practicalities of evaluating a client seeking an ADHD diagnosis to secure accommodations.

Beyond diagnostic issues, we discuss what accommodations make sense given the nature of the disorder and the settings for which they are intended. We also cover the (limited) research available relevant to accommodations and ADHD.

AN ADA PRIMER

Clinicians who understand Congress's intent when it passed and amended the ADA are far more likely to better serve clients seeking accommodations. Toward that end, we present here the fundamental principles

that underlie the ADA as it relates to educational and workplace accommodations.

1. ***The ADA is, first and foremost, an anti-discrimination law.*** The ADA evolved from the Rehabilitation Act of 1973 (RA), a law that made it illegal to discriminate against individuals with disabilities who worked in certain government-funded settings or participated in activities funded by government monies. Since most educational institutions receive at least a small amount of federal funding, the RA has applied to them as well. In fact, many K–12 students still receive accommodations under Section 504 of the RA (Yell, 2012).

The ADA extended the protections of the RA to nongovernmental entities. They include private business, state and local governments (including public schools), and public accommodations. The latter refer to any privately owned properties that the public uses—from concert halls and baseball stadiums to stores and private colleges. However, while the ADA extends the reach of anti-discrimination coverage, it uses concepts and definitions that are identical to the RA, especially when it comes to what qualifies as a disability. Throughout this chapter, we refer primarily to the ADA, but in certain federal government settings, the RA would be the controlling authority.

In 2008, the ADA was amended under the ADA Amendments Act (ADAAA). It altered certain definitions and generally broadened the coverage of the law. At times, the ADAAA explicitly refuted interpretations of the ADA that had been made by federal courts, including the U.S. Supreme Court. However, many core features of the original ADA were left intact. We continue to refer to the ADA (since it still exists, as the ADA, in its amended form), unless we are discussing particular alterations included in the ADAAA.

That the ADA was intended as an anti-discrimination law informs all the regulations and practice guidelines that have emerged over the years. The intent of the law has always been to guarantee that individuals with disabilities have equal access to opportunities. It was never designed to guarantee success. Accommodations are not meant to ensure that an individual with a disability *achieves a high score* on an admissions test or *does well* on the job. It only assures that the individual has equal access to that test or job. Clinicians who write in reports, "This student requires accommodations to make sure he can perform his best on the exam," miss the point of the legislation. The focus is not on maximizing success but on ensuring that a disability irrelevant to performing a task does not unfairly interfere with the opportunity to engage in that task. For example, a thoroughly competent accountant should not be kept from working because he cannot guide his wheelchair through the office doors.

Some clinicians err in assuming that the ADA is an adult extension of special education laws, such as the Individuals with Disabilities Education Act (IDEA) (Gordon, Lewandowski, Murphy, & Dempsey, 2002). That law was designed to maximize the educational outcome of children with special needs by providing support in the least restrictive environment. The intent of special education laws, then, has been to ensure that children with disabilities make the most of their talents. Optimizing outcome was never the goal of either the ADA or the ADAAA.

The issue of access versus success is important because accommodations that directly guarantee success are often unfair to other individuals. For instance, giving an examinee with ADHD as much extended time as is necessary to pass a certification test for a profession is unfair to other examinees who would also have benefited from such a success-focused accommodation. Success is not assumed to be anyone's "right," regardless of disability status; it is something that some, but not all, students, examinees, and employees earn. Access, in contrast, is a universal right. Because we assume that nondisabled individuals have sufficient access, only individuals with disabilities require accommodations designed to enable access.

2. ***A clinical diagnosis is not necessarily the same as a legal determination of disability.*** The most common misperception among clinicians unfamiliar with the ADA is that a clinical diagnosis is the same as a legal determination of disability. Too many practitioners assume that asserting a diagnosis is sufficient to justify the requested accommodations. That misconception usually leads to evaluations that offer little benefit to clients seeking accommodations.

The main reason a diagnosis is not tantamount to a disability is that the ADA sets a different standard for what qualifies someone as disabled. In the ADA realm, individuals are qualified as disabled if they are impaired in a major life activity as compared to "most people in the population" (Equal Employment Opportunity Commission [EEOC] Final Regulations published in the Federal Register on March 25, 2011). This metric, often referred to as the "average person standard," can

differ substantially from the one that clinicians use in their non-ADA evaluations. Clinicians frequently see clients who perform well relative to the average person, but not as well as they would like given their educational aspirations or level of intelligence. For instance, a law student may consider himself impaired because he has struggled to finish exams as quickly as other law students. Or perhaps a medical student is distressed because she is slower than others in her class when taking a patient's history.

While clinicians may choose to regard these complaints (and the distress they engender) as sufficient to warrant a diagnosis, the ADA would not deem them to be disabling conditions. Most individuals in the general population, of course, do not have the requisite abilities even to consider admission to postgraduate training. As a consequence, only a small percentage of the population has the skills to tackle law or medical school. It is therefore often challenging to make the case that someone is disabled under the ADA, when he or she has achieved far better than most people. It is especially formidable when the applicant has managed that level of accomplishment without any history of formal accommodations (for an extensive consideration of this issue, applied to an actual case example, see Gordon, 2009).

Clinicians have to accept that the ADA is agnostic to the idea that a disability can be justified by failure to achieve relative to some presumption of a person's potential. A student who does well, but perhaps could do better, is not a candidate for ADA accommodations in many settings. The law also looks dimly upon the notion that people can qualify as disabled simply because they have had to work hard to achieve goals that are inherently hard to achieve (like graduating from medical school). In a strict interpretation of the ADA, people qualify as disabled if, in some major life activity, they function abnormally relative to the average person.

Judging disability based on the "average person standard" often runs counter to most clinicians' approach to diagnosis. In other clinical circumstances, the goal is to help people who are distressed because they have trouble functioning as well as they would like. The focus is on providing relief, not so much on judging the extent of impairment. In general clinical practice, the diagnosis serves as the first step in developing a targeted treatment plan.

Conducting evaluations for ADA accommodations requires clinicians to shed the role of helper/advocate in favor of one that is geared more toward objective evaluation of overall functioning. In this context, the clinician approaches the case more as dispassionate judge than caring professional. Of course, that stance may require explaining to some clients that it is impossible to make the case for disability. Most clinicians understandably find these kinds of conversations uncomfortable. Nonetheless, it seems more problematic to encourage a client to pursue accommodations when the justification for them is undeniably weak.

The DSM series has unambiguously acknowledged the likely distinction between clinical diagnosis and disability determination. The most recent iteration, the DSM-5, states the following:

> In most situations, the clinical diagnosis of a DSM-5 mental disorder . . . does not imply that an individual with such a condition meets the legal criteria for the presence of a mental disorder or a specified legal standard (e.g., for competence, criminal responsibility, or disability). For the latter, additional information is usually required beyond that contained in the DSM-5 diagnosis, which might include information about the individual's functional impairments and how these impairments affect the particular abilities in question. It is precisely because impairments, abilities, and disabilities vary widely within each diagnostic category that assignment of a particular diagnosis does not imply a specific level of impairment or disability. (American Psychiatric Association, 2013, p. 25)

A clinical diagnosis, then, is generally necessary but not sufficient to show that the ADA threshold of a disability has been met.

Clinicians who conduct ADA-related evaluations may perhaps be confused by what appears to be inconsistent interpretation of the law by different academic institutions, testing organizations, and businesses. An assessment that is effective in justifying accommodations in one setting may well be dismissed out of hand in another. What evaluators should realize is that institutions can decide for themselves, based on their own particular goals, how strictly they choose to interpret the ADA standards. For example, a community college may elect to set a low threshold for what constitutes a learning disability because it is primarily interested in amassing whatever resources would be necessary to help a student to graduate. An organization charged with certifying medical competence has a different agenda. It is primarily concerned that it administers tests that protect the public against practitioners who may not meet standards for minimal competence. These sorts

of organizations are likely to hold students to a higher standard when it comes to determinations of disability, out of concern that they prevent weak students from gaining licensure through an unfair advantage.

3. ***It is all about providing evidence of real-life functional impairment over time.*** A company or academic enterprise bound by the ADA would have no reason to deny accommodations to individuals who presented evidence of ADHD-type symptoms that caused impairment in actual functioning over years and in most situations. A thick packet of prior assessments, report cards, teacher comments, special education records, and job performance evaluations that documented long-standing disability would likely sail through the disability review process. Conversely, applications that are short on hard evidence of chronic impairment due to ADHD symptoms may encounter stronger resistance. Administrators and consultants are apt to deny accommodations when data supporting the case for impairment are scant.

We want to stress the importance of providing as much evidence as possible from the client's actual functioning. Credible evaluations, careful clinical and educational histories, teacher reports, transcripts, and the like are the nuggets that reviewers prospect for when considering applications. The more contemporaneous and convincing evidence the documentation can offer, the better.

Less compelling to reviewers are diagnoses that are based almost entirely on self-reported symptoms and scores from psychological testing. It is hard for them to give much weight to either source of information because of ample evidence pointing to their unreliability, especially in the context of a disability determination for ADHD.

As for self-reports, two major problems commonly arise: First, individuals applying for accommodations often assume that having symptoms of a disorder is tantamount to having that disorder. They may not realize that individuals in the general population commonly have reported ADHD-type symptoms both in their current functioning (e.g., Lewandowski, Lovett, Codding, & Gordon, 2008; Murphy & Barkley, 1996) and during childhood (Suhr, Zimak, Buelow, & Fox, 2009). They may even report instances of actual impairment (e.g., losing keys, being late for meetings) without being aware that these kinds of events do not necessarily reflect abnormality. In fact, self-reports of impairment appear to relate more to subjective distress than to actual neurocognitive dysfunction (Miller, Haws, et al., 2013).

The second major problem with self-reports is that they are easy to exaggerate or even falsify. In recent years, a sizable body of work on the malingering of ADHD has emerged (for reviews of relevant literature, see Harrison, 2006; Jasinski & Ranseen, 2011; Musso & Gouvier, 2014) Researchers have confirmed that it is relatively easy for motivated laypersons to score in the clinical range on ADHD rating scales when instructed do so (e.g., Fisher & Watkins, 2008). Given all the information about ADHD that is broadcast through cultural media and social conversations, few individuals would be unfamiliar with its common features. Similarly, research has demonstrated that a substantial minority of those individuals evaluated for ADHD exaggerate their symptoms, as indicated by their failing scores on tests of effort and motivation (Sullivan, May, & Galbally, 2007). Meanwhile, individuals with genuine ADHD may actually tend to minimize their problems due to unrealistically positive self-views (Prevatt et al., 2012).

On first glance, psychological testing would seem to represent a firmer foundation than self-report for making ADHD diagnoses. Unfortunately, no standardized psychological test has been shown to be sufficiently sensitive and specific to ADHD to be used on its own to support a diagnosis (for a review of the relevant literature, see Gordon, Barkley, & Lovett, 2006). While testing can be useful to confirm a diagnosis (and, even more so, to rule out alternative factors such as those related to IQ and academic abilities), it is not a sturdy foundation for a unilateral disability determination. Clinicians would be hard-pressed to justify a diagnosis on psychological testing alone, especially in an individual who has a lifelong record of being generally competent. Therefore, in the vast majority of cases, evidence from reality trumps psychological test scores in determining the level of functioning.

Part of the problem with relying on psychological tests is that they often present tasks that may have limited relevance to the actual demands of daily life. Consider neuropsychological tests of executive functioning, which are often used in an ADHD neuropsychological test battery. While deficits in executive functioning can certainly lead to real-world problems, many commonly used neuropsychological measures have limited ecological validity (Burgess et al., 2006; Toplak, West, & Stanovich, 2013). For example, one common task asks the individual to move colored balls from one place to another while following a certain set of rules.

Another task instructs the client to say the color of ink that words are printed in while ignoring the meaning of the words themselves. These tasks do not simulate most real-world demands. The same can be said of most tests of processing speed. While clinicians often tout them as meaningful indicators of how efficiently an individual processes information, the actual data supporting their utility in ADHD determinations is scant, perhaps again because of the gap between test demands and reality. The most common tests of processing speed are simple, repetitive perceptual–motor tasks (as in the subtests making up the Processing Speed Index on the Wechsler IQ tests) rather than tasks that resemble real-world, time-limited activities.

Evaluators often wonder whether someone can still be considered disabled even if treatments or adaptations (known in ADA parlance as "mitigating measures") allow the person to function normally. Guidance on this issue has changed over the years. Under the ADA, the Supreme Court found that a person is not "substantially limited" if measures such as medication allowed for typical functioning. The ADAAA revision now requires that "the determination of whether an impairment substantially limits a major life activity shall be made without regard to the ameliorative effects of mitigating measures" that include medication and assistive technology. Therefore, if an individual with ADHD performs in the average range when taking medication, he or she may still be substantially limited under (the amended) ADA. However, this does *not* mean that such a person would be entitled to accommodations, since accommodations may not be needed if the medication is sufficiently effective.

4. ***Accommodations must be reasonable and targeted to the disability.*** The central intent of the ADA is to ensure that individuals with disabilities are not denied access to activities and settings due to those disabilities. It compels institutions and companies to offer reasonable accommodations that promote that access. The most common (and far-reaching) example concerns wheelchair cuts. Every crosswalk in America has been modified so that individuals in wheelchairs can cross the street unhindered. Most buildings have also been required to offer handicapped-accessible restrooms to enable people with disabilities to enter the premises knowing they can use the bathrooms.

Not all accommodations, of course, are architectural. A company might provide a special desk for any employee who is unable to sit for long time periods. Organizations might also have to produce important application forms in multiple formats (large print, Braille, etc.) for individuals with visual disabilities. As for individuals with ADHD, the list of potential accommodations might include extra time on tests, distraction-free rooms, additional secretarial support, and additional break time (see below).

While institutions must provide accommodations to individuals who qualify as disabled, they are not on the hook to offer whatever the person requests. The accommodations have to meet certain standards:

- *Associated with the disability.* Accommodations must address the impact of the functional impairment on the task at hand. Unlimited time on a timed exam may not be justified given the level of the examinee's impairment or the logistics involved in allowing for unlimited time. Someone might also have a legitimate disability that would not affect the ability to take a particular exam. For example, an examinee with a bona fide math disability might not be entitled to accommodations on a test that has no items requiring mathematical computation.
- *Cannot pose an undue burden.* Accommodations are not required when they pose an "undue burden" for the entity providing the accommodation. For instance, a small business might not be able to afford a personal assistant to help an employee with ADHD to function on the job. Even large testing organizations may not be able to manage the logistics inherent in administering a test without time constraints.
- *No fundamental alterations.* An accommodation cannot change the nature of the activity to such a degree that the individual is no longer performing the core features of the task. A common example: An individual with a reading disability might ask for someone to read the items on a test designed to measure reading ability. That accommodation would fundamentally alter what the test was intended to measure. Consider also an anesthesiologist who claims that his ADHD requires that the hospital provide an assistant to help him attend to the instrumentation. The employer could argue that the accommodations fundamentally altered the essential requirements of the job. Therefore, the entire purpose of an accommodation is to remove the impact of factors that are not relevant to what a test measures or a job requires.

PREPARING DOCUMENTATION FOR AN ADA ACCOMMODATIONS REQUEST

Most institutions publish guidelines for what they require in documentation submitted by individuals seeking accommodations. Clinicians should ask clients to bring those guidelines (or provide a website link) for review prior to conducting the evaluation. Chances are that the individual will also bring clinicians one or more attestation forms to complete. These are statements institutions or businesses provide for certifying the disability.

While documentation guidelines can differ from one group to another, they generally share the following common elements.

Currency

Most institutions require that the evaluation be no more than 3 years old. The idea is that current functioning is impossible to assess if the most recent evaluation is dated. This rationale is especially applicable to ADHD given the extent to which impairment caused by the symptoms can wax and wane over time and depending on circumstances. It would be hard to make a case that someone diagnosed as having ADHD at the age of 7 was necessarily still impaired at 17. The disorder persists for many over the course of a lifetime, but certainly not for all (Antshel & Barkley, 2011).

Because the determination of disability rests on the level of impairment at the time of the request for accommodations, practitioners should avoid completing the institution's verification forms based on information gathered from evaluations conducted long ago. It is in the client's best interest to submit fresh data that include the most recent evidence of ongoing impairment.

Professional Qualifications

While institutions are surely more interested in the quality of the documentation than the qualifications of the clinician who prepared it, they do require the evaluator to have a certain level of disorder-relevant expertise. Administrators and reviewers are more apt to trust the conclusions of someone with comprehensive training and extensive clinical experience than a practitioner with little background in that area. The opinions of an expert in adult ADHD will carry far more weight compared to what an internist without specific training in ADHD might offer. Clinicians who prepare ADA documentation will therefore want to establish their professional credentials, the extent to which they have expertise in the area of the diagnosis (or diagnoses), and any other information that might establish their qualification.

Diagnostic Criteria

Clinicians who establish at the outset that they understand what it takes to justify an ADHD diagnosis and qualify an individual as disabled will be more successful in preparing compelling documentation. Our experience is that administrators and reviewers appreciate knowing that the clinician "gets" the process and is not simply advocating for the applicant based on idiosyncratic notions or a strained construal of clinical history or test scores. Simply stating that you understand the imperative of showing impairment relative to most people (and not a particular educational cohort) can go a long way in providing that level of comfort. It can also be helpful to acknowledge the importance of establishing a childhood onset and evidence of impairment that extends beyond self-report and relative discrepancies in test scores.

In addition to stating the specific criteria you used to arrive at the diagnosis, you will want to explain how the procedures you employed were intended to address those criteria. It boosts credibility when a clinician describes a methodology that cannot help but provide information relevant to the disability determination. Reviewers would be impressed if a clinician were to write something to this effect: "This evaluation was designed to establish evidence of a childhood impairment because of ADHD symptoms. I have also gathered contemporaneous information from my client's academic and occupational history that speaks to the extent to which that impairment has been chronic, pervasive, and substantial. Finally, I conducted a full differential diagnosis to rule out nonpsychiatric problems that could account for the symptoms my client presented."

Functional Limitations

The sine qua non of ADA-related evaluations is a full description of those abnormalities that would impact a person's access to the academic or occupational activity in question. The institution looks for a reasonable answer to this fundamental question: "What evidence

can you present that any ADHD-type problems substantially limit this individual's ability to access our test, educational program, or employment?" Beyond test scores, presenting complaints, or elements of the diagnostic formulation, the case for accommodations rises or falls on the degree to which the clinician submits sound information verifying that, relative to the general population, the applicant has been and continues to be disabled. A listing of symptoms is no match (and correlates only weakly) with reports of impairment (Gordon, Antshel, et al., 2006).

Many entities that are required to provide accommodations under the ADA have developed guidelines for the kinds of evidence they would like clinicians to submit when substantiating a claim based on ADHD. While we offer specific examples in the next section, the categories of information they request are predictable given the nature of the disorder. Fortunately, adults who truly meet criteria for ADHD should have no trouble providing corroboration of impairment. By definition, the impact of their symptoms should have left a paper trail that extends over a lifetime. It would be wise to submit copies of that paperwork (i.e., prior evaluations, school records, transcripts, and performance reviews) rather than only summarizing it in your report.

Finally, evaluators should not approach ADA-based reports as an exercise in dismissing evidence of stellar functioning, while trumpeting minor setbacks as the manifestation of significant deficits. That effort to construe success as failure is often transparent and unconvincing. It also brings the evaluator's objectivity into question.

Accommodations

Institutions that are covered under the ADA want clinicians to tell them exactly what accommodations the client needs and how those accommodations will reduce the impact of the identified functional limitations. The alterations also have to be relevant to the activity for which the individual is seeking accommodations. For example, it would make no sense to recommend extended time on an untimed test. You also would not recommend a distraction-reduced environment for a test that is normally administered by computer in a sound-reduced carrel. Formulating an accommodation request is all about fitting the alteration to the impairment in a way that is reasonable given the situations that will confront the individual.

EVALUATING INDIVIDUALS FOR ADA ACCOMMODATIONS: THE DETAILS

Evaluating individuals who seek ADHD-based accommodations follows the same general procedures a clinician would use for assessing any client for this disorder (see Chapter 19). As such, it should generate evidence that ADHD-type symptoms started causing meaningful impairment across settings during childhood, have persisted over time and in most situations, and could not be better accounted for by other factors. While clinicians may vary in the means they use to collect that data, the goals of the evaluation remain the same.

What can differ between ADA and general clinical assessments is the degree of rigor institutions might expect of you in applying diagnostic criteria. Organizations have the right to require strict adherence to professional diagnostic guidelines. As we indicated earlier, they also operate within a world that follows the "average person standard" for gauging impairment. Therefore, while evaluations for ADHD under the ADA are by no means idiosyncratic affairs, they require a clinician to be especially mindful of certain key requirements:

1. ***Early onset means early onset.*** Whereas the DSM-5 has extended the age at which symptoms must become impairing, it remains the case that ADHD is a developmental disorder that, by definition, must have meaningful impact on functioning by the end of childhood. It is therefore essential to offer verification that the client was significantly impaired by ADHD symptoms no later than around 12–14 years of age. In the absence of such evidence, applications for ADA accommodations might well encounter justified skepticism from reviewers.

The logic underlying this criterion is as follows. Extensive research has established that the ability to pay attention and exert self-control is central to a child's healthy growth and adjustment. In fact, much of what children need to learn—whether at home, in the classroom, or on the playground—hinges on the capacity to stop their behavior long enough to allow for a considered response. Without an adequate ability to inhibit behavior, children will inevitably encounter trouble learning rules, getting along with others, controlling emotions, acquiring academic skills, benefiting from past experience, and anticipating future events. The capacity to exert self-control and concentrate is so nec-

essary for normal development that any deficits in these areas would necessarily have an early and observable impact on adjustment in the same way that a reading disability would be obvious during the years children typically learn to read. Positing a late-onset variant of ADHD would seem about as justifiable as establishing a late-onset type of autism or school phobia.

When confronted with the reality that a particular client was never impaired during childhood (and, indeed, functioned well), some clinicians try to explain away the discrepancy by pointing to mitigating factors. Chief among them is the notion that superior intellectual levels can forestall the impact of ADHD symptoms into young adulthood. However, DSM-5's "by childhood" rule stands for everyone, regardless of IQ. In someone who truly meets the criteria for ADHD, no amount of cognitive firepower can prevent the disruptive impact of significant deficits in attention, self-control, and executive functioning, especially in nonacademic environments. The DSM does not establish IQ-based exceptions to that early-onset rule because no evidence exists to warrant them.

A second category of reasons some clinicians submit to explain late-onset revolves around the idea that ADHD symptoms can lie dormant until adulthood (perhaps because of high IQ). According to this argument, some individuals can compensate for their symptoms until they encounter high demands for paying attention and exerting self-control. From an ADA perspective, the law was never intended to protect individuals who have functioned well, but not as well as they might have preferred in a highly competitive setting. What defines a disorder is the inability to handle routine life tasks, *despite* reasonable attempts at compensation.

2. ***There's no substitute for evidence of impairment based on the client's actual functioning.*** At the risk of belaboring the point, we state again how important it is to make a case for impairment that relies on more than self-reported symptoms or the interpretation of psychological test scores. ADA-covered entities do have the right to expect hard evidence that the individual, compared to most people in the population, is substantially limited in performing major life activities. Therefore, clinicians may find themselves expending more effort than usual gathering documentation from the client, schools, and employers. Of course, problems amassing much evidence of this sort may signal that factors other than ADHD are at play.

Clients who have consistently received formal accommodations over many years will find it easier than most to gain ADA protections, especially if the evaluations justifying those alterations were credible. Because a record of having received legally mandated services can often corroborate the impact of chronic deficits, clinicians should make sure that the client provides verification regarding the nature and timing of those accommodations. The practitioner should look to document a history of legally granted accommodations, not commonplace classroom or work compensations. A stack of individualized education plans (IEPs) is far more compelling than accounts of joining study groups, participating in a study skills class, or having had received occasional tutoring.

The rigor associated with ADA-relevant evaluations extends to documenting impairment across settings. As a neurodevelopmental disorder associated with core deficits in the ability to attend and exert self-control, ADHD will necessarily cause pervasive problems in adjustment. The widespread impairment experienced by individuals with ADHD is a testament to how much of normal functioning depends on the capacity to be focused, organized, and controlled (see, e.g., Dawson & Guare, 2004; Goldberg, 2009). Disability reviewers are therefore often unconvinced when clinicians claim that the only impairment the client experiences centers on taking high-stakes tests or achieving well in a rarified academic setting.

In advocating for a laser focus on cross-situational impairment, we do not intend to dismiss the role of personal accounts, rating scales, or psychological testing. Each can contribute to the case for accommodations. Our point has been that, relative to contemporaneous data, they stand as more vulnerable to bias. For example, no amount of norming can overcome the reality that a self-report rating scale is based on self-report and is therefore subject to the warping influences that can arise when a client perceives a desired outcome. The same point can be made for psychological testing given that no psychological test has been established as sufficiently sensitive or specific to be considered solely determinative of an ADHD diagnosis.

Clinicians often wonder whether it is best to evaluate individuals with ADHD while they are on or off medications. For the purposes on justifying accommodations, it is generally advisable to test the client *off* medication. In this way, the clinician can document what might represent the highest level of impairment

the client might experience. This strategy is also perfectly defensible given the ADAAA's stance on mitigating measures.

3. ***Absolute impairment, not relative intraindividual weaknesses.*** The interpretation of psychological testing has often pointed to relative discrepancies between scores to identify problem areas. In some cases, they have actually defined the concept of a disorder. For instance, clinicians long considered an individual to be learning disabled if a significant discrepancy existed between measured IQ and achievement, even if academic functioning was average. While the discrepancy model of learning disabilities (LD) has fallen by the wayside in the face of disconfirming research (e.g., Lovett & Gordon, 2005; Sternberg & Grigorenko, 2002), only with the advent of DSM-5 have the underpinnings of the LD diagnosis shifted to actual impairment. In the world of ADA determinations, of course, relative discrepancies never held sway in light of the "average person standard."

Even though diagnostic nosologies have largely abandoned relative discrepancies as a basis for identification, the idea still persists among some clinicians that an individual can be considered disabled (and disordered) based on a profile of relative strengths and weaknesses. They argue that normal functioning is actually abnormal in the context of high intellectual abilities. According to this position, anyone with superior cognitive abilities should, by nature, be able to achieve at a superior level. Any fall off from excellence, in this view, reflects the impact of a disorder.

The core problem with this reliance on discrepancies is that, while measured IQ represents an important predictor of real-world academic and occupational functioning, it is far from perfect (Hunt, 2011). Even if IQ accounts for as much as 25% of the variance in important outcomes, discrepancies between IQ and real-world performance would still be common. And even if the prediction were even stronger, high IQ does not represent a birthright to high academic, occupational, or social advantage.

The ADA's insistence on gauging impairment against a comparison group of typical individuals has obvious implications for the norms clinicians should use in interpreting data. For example, it would be unwise to employ standard scores derived from the test performance of college graduates. They, of course, do not constitute a sample of average people, given that only about 25% of the general population earns a 4-year degree (National Center for Education Statistics, 2011). By extension, grade-level norms are problematic whenever someone is at a grade-level higher than the general population. Therefore, whenever possible, age-based norms should be the basis for comparison.

Another pitfall in establishing a case for absolute impairment is pointing to improvement in psychological scores when time limits are extended. That a client does better with extra time does not alone justify extended time on high-stakes examinations or job functions. Many nondisabled individuals perform better if they have more time (for reviews of the literature on this point, see Lovett, 2010; Sireci, Scarpati, & Li, 2005). Also, most psychological tests do not publish norms for performance under the condition of extended time. It is therefore impossible to know whether the extent to which a particular individual improved with more time is out of the ordinary.

The same caveat is in order when invoking medication-induced improvements in test scores as supportive of an ADHD diagnosis. It has been well established that such improvements are nondiagnostic (see Chapter 35). The fact is that most individuals perform better when administered such medications, particularly stimulants.

One more point about establishing impairment: Because these evaluations are conducted to support legal accommodations, clinicians are expected to vouch for the credibility of the data, perhaps more than they would in other circumstances. The presence of a desired outcome heightens the possibility that motivational factors may bias the results, consciously or otherwise. It is also the case that the prospect of malingering is hard to assess through observation or clinical judgment alone (cf. Faust, Hart, Guilmette, & Arkes, 1988). Therefore, it has become de rigeur for practitioners to administer a test of effort (for examples of effort tests used in ADHD evaluations, see studies by Jasinski & Ranseen, 2011; Sullivan et al., 2007).

4. ***A differential diagnosis makes a difference.*** Making the case that ADHD causes impairment sufficient to warrant accommodations requires special attention to these questions: "Are you certain whatever problems the individual claims to experience are not due to nonpsychiatric factors such as test anxiety, subclinical anxiety/depression, English as a second language (ESL) issues, or motivation? And what makes

you sure that the person's struggles are not related to having intellectual skills and academic abilities that, while average, are nonetheless insufficient to meet the academic or job demands?" The intent of the ADA was to help individuals with substantial physical or psychiatric disabilities that accommodations can reasonably mitigate. It was not passed to protect high-functioning individuals who might struggle to master complex demands easily.

In their zeal to advocate for an ADHD diagnosis, some clinicians avoid entertaining alternate explanations for presenting symptoms. It is fair for documentation reviewers to wonder, for example, why a clinician failed to acknowledge that the medical student's 92 Full Scale IQ might factor into his academic woes far more than any psychiatric condition. It is better to address these kinds of concerns systematically than to ignore them.

Other professionals are quick to explain away obvious evidence of test anxiety (a nonpsychiatric condition) or subclinical mood problems by asserting that they are necessarily the emotional consequence of having ADHD. However, those mood-related phenomena can often seem more compelling as sole explanations for an individual's problems taking tests, especially in the absence of a longstanding history of global impairment. In other words, the mood problems or test anxiety may be more the cause of any inattention rather than the result of deficits in attention. Therefore, the clinician should offer a convincing argument for why mood problems may indeed be secondary issues.

Institutions are only required to consider accommodations for the disability or disabilities the individual identifies in the application. A person who is severely depressed might not be granted accommodations if the evaluations only address criteria for ADHD. A proper diagnosis leads to documentation more apt to support it. Therefore, even if clients, because of concerns about stigma, are loath to claim disability based on a psychiatric condition other than ADHD, it would be wise to help them overcome that objection.

In the case of comorbid conditions, evaluators should fully explain how the client meets criteria for each disorder. However, it is most important to account for how the various conditions, individually or in combination, lead to significant impairment. The relative contributions of each diagnosis are far less important than the evidence that they substantially impede normal adjustment.

PRESCRIBING ACCOMMODATIONS IN AN ADA CONTEXT: THE DETAILS

The major challenge for most individuals seeking accommodations is verifying that they qualify as disabled under the ADA. The height of that hurdle can be high, depending on the extent of impairment and the nature of the context in which accommodations would be implemented. Once someone meets the standards for disability, institutions and companies are likely to grant them the accommodations they request. Those determinations are much easier, especially if the proposed accommodations are not out of line with the nature and extent of the disability or likely to place an undue burden on the organization.

Most of the accommodations that individuals with ADHD request are intended to reduce distractions and extend the time allotted for task completion. These kinds of alterations have a certain intuitive appeal given the problems individuals with ADHD have fending off distractions and completing tasks. For example, an individual with this disorder might conceivably perform better on a high-stakes exam if it were administered in a quiet room. The opportunity to take frequent breaks along the way might also make it easier to work on the test.

Two fundamental questions arise in considering accommodations for students or employees with ADHD. First, given the current understanding of the disorder, what kinds of accommodations are most likely to provide benefit? Second, what does the research literature have to say about what actually does tend to provide benefit?

Fundamental Deficits Associated with ADHD

Although ADHD is regarded as a heterogeneous disorder with a presentation that can vary across the lifespan, certain features and characteristics are held as generally universal. Many investigators (e.g., Barkley, 1997; Welsh & Pennington, 1988; Willcutt, Doyle, Nigg, Faraone, & Pennington, 2005) have described the deficits associated with ADHD as impairments in executive functioning (e.g., inhibition, planning, working memory, self-motivation, and emotion regulation). A closer look at some of the core features associated with this profile can point the way to selecting effective ADHD-relevant accommodations.

Problems of Self-Control

Barkley (1997) and others have noted that one of the cardinal problems associated with ADHD is a reduced ability to control motor activity, attention, behavior, and emotion. Consequently, individuals with this disorder, particularly those who manifest both inattention and hyperactive–impulsive symptoms, tend to be overactive, impulsive, disinhibited, restless, and unable to stay on a task for a sustained period of time. As students, they are often restless, bothersome to classmates, off task, and prone to blurt out whatever may come to mind. As adults, they make high rates of commission errors on continuous-performance tests (Boonstra, Kooij, Oosterlaan, Sergeant, & Buitellar, 2005), as well as various stop-and-go tasks that require inhibition (e.g., Chhabildas, Pennington, & Wilcutt, 2001). Some have recognized impulsive responding and off-task behavior as a reaction to boredom or the inability to wait for a reward (Barkley, Edwards, Laneri, Fletcher, & Metevia, 2001). It is believed that problems with impulsivity, poor self-control and disinhibition are at least partially responsible for higher than normal incidents of work injuries, conflicts, firings, as well as traffic accidents, substance abuse, and criminality (Küpper et al., 2012).

Problems of Motivation

Another feature associated with ADHD concerns deficits in self-motivation and self-direction (Barkley & Murphy, 2011; Volkow et al., 2010). In fact, there is a motivation-based theory of ADHD that hypothesizes impairment in the reward system consistent with a frontal–striatal dopamine brain circuit (Desjardins, Scherzer, Braun, Godbout, & Poissant, 2010). It appears that the motivational impairment may be present for both complex and menial tasks that are deemed to be unmotivating by the individual (Sonuga-Barke, 2005). A consequence of limited impairment in this system is that behavior tends to be more externally than internally driven. Individuals with ADHD react to environmental stimuli more strongly than peers, particularly if those cues are associated with immediate gratification. They often live in the present, rather than relying on past experience or anticipating future events. Long-range plans are seldom brought to completion because following through on intentions represents a major stumbling block, whether the task is to finish a discrete homework assignment or maintain a consistent study effort throughout an entire course or college career (Weyandt & DuPaul, 2008).

Because of the problems in self-motivation and self-direction, individuals with ADHD rely on external motivation and direction. At first, parents provide the direction and reinforce desirable behaviors, then teachers, and ultimately employers. Spouses, friends, and coaches also learn that they may have to take the lead in planning, goal setting, supporting, coaching, reinforcing, cajoling, and so on, to help individuals with ADHD be productive. The need for external support seems to be present in daily life management, school, and occupational domains (Barkley, 2013; Nadeau, 2005; Tuckman, 2007).

Problems with Time Management

Various investigators (e.g., Barkley, Edwards, et al., 2001; Smith, Warner Rogers, Newman, & Rubia, 2002) have noted that persons with ADHD tend to have difficulties regulating themselves relative to time. Studies show that they often misperceive the time it takes to do something or miscalculate how much time has elapsed (e.g., Barkley & Murphy, 2011; Barkley, Murphy, & Bush, 2001). The classic problem is underestimating how long it will take to complete a project or get from one place to another. Consequently, individuals with ADHD are often late in social, academic and work situations, and their work is often hurried, incomplete, or missing (DuPaul, 2007). As with self-direction, individuals with ADHD need help managing time. Therefore, they tend to perform better in structured settings that offer clear expectations within relatively short time frames (Rief, 1993).

Implications for Designing Accommodations

If these core deficits are truly associated with ADHD, their impact on functioning within the major life domains that are amenable to ADA-type accommodations should be verifiable. Of course, voluminous research has documented that individuals with ADHD are indeed prone to poor functioning in most settings (see Chapters 11 and 12 for detailed reviews). Substantial problems with impulse control, motivation, emotion regulation, and time management have inevitable consequences for functioning at home, school, and the workplace (Chapters 2, 3, 4, and 10; Barkley & Murphy, 2011; Barkley, Murphy, & Fischer, 2010).

Given the nature of the disorder and its impact on normal adjustment, how should clinicians determine appropriate accommodations for any particular client? This answer is inherently complicated because it depends on interactions among multiple dimensions and considerations, including the nature of the setting (classroom, high stakes testing, and workplace), the array of accommodations that are feasible within that setting, and the extent to which research has actually demonstrated that particular forms of accommodation are evidence-based.

To simplify a complex set of interactions, we have constructed what we refer to as the Accommodations Matrix (see Figure 33.1). It provides examples of accommodations that seem to be relevant in various academic and work situations. This matrix is based on distinctions that Thurlow, Elliott, and Ysseldyke (2003) provided as an organizational framework. Their work established the following classes of accommodations:

- *Setting accommodations*: alterations in the space in which task takes place. These might include, for example, administering a high-stakes test in a separate location, either with fewer students or in a private setting with few distractions. They can also involve special adaptations to the room, furnishings, or technology. A setting accommodation could include preferred seating in a classroom or a high-stakes test situation.
- *Presentation accommodations*: specialized formats for presenting learning, testing, or work materials. Examples include providing test or work materials

	Educational/Classroom	**High-Stakes Tests**	**Workplace**
Setting	• Preferred seating • Reduced distractions • Sound control • Small-group setting • Earplugs	• Semiprivate or private room • Earplugs	• Private office or cubicle • Reduced distractions • Organized setting, files, system • Technology for efficiency • Secretarial and administrative support
Presentation	• Read aloud • Computer-assisted • Break into parts	• Clear instructions • Computer-assisted	• Clear instructions • Oral and written orders • Computer-based (paperless)
Timing	• Additional time • Extra breaks • Multiple days • Short tasks	• Additional time • Extra breaks • Multiple days	• Additional time • Flexible schedule • Shorter tasks • Structured breaks
Response	• Dictation • Voice recognition • Spell and grammar check • Calculator • Scantron-free tests	• Scribe • Word processor • Calculator • Scantron-free	• Computer-assisted • Calculator • Dictation systems • Tests with movement
Comfort	• Food and drink • Headphones • Special chair	• Food and drink	• Office furnishings • Snack/break area
Other	• Frequent feedback and reinforcement • Frequent reminders, to-do lists, prompts, support • Keep things moving, novel, interesting		• Goodness of job fit • Weekly supervision meetings • Personal digital assistant • Frequent feedback • Incentives • Work with buddy/peer

FIGURE 33.1. ADHD Accommodations Matrix.

in Braille or enlarged print for visually impaired individuals.
- *Scheduling accommodations*: modifications in the individual's work schedule, assignment or task completion dates. They might also apply to the scheduling of exams and the period given to complete the exam.
- *Timing accommodations*: often involve extended time to take a test or complete an assignment, or being permitted to take additional breaks during a test or task.
- *Response accommodations*: alternative means of responding to school assignments, test items, and work tasks. They might involve dictating answers to a scribe or via a voice recognition computing system, or having access to special equipment (e.g., a dictionary, calculator).

We added a category called "comfort measures" because many organization offer them. Comfort measures are not designed to mitigate impairment per se. They can reduce discomfort that might accrue from being in the testing or working situation for a long duration. For example, a person with irritable bowel syndrome might need frequent and ready access to a restroom, while a person with ADHD might need access to water, a snack, or medication.

Practical Application of the Accommodations Matrix

In this section we elaborate on how accommodations for individuals with ADHD can work in the classroom, for tests, and in the workplace. We focus on two questions:

1. What are the most common accommodations that clinicians suggest for each setting?
2. Is there scientific evidence that these accommodations specifically alleviate the impact of the symptoms associated with ADHD?

First a word about research in the accommodations area: It is extremely limited in size and scope, although somewhat less so in the area of test accommodations. The fact that the research literature is so small is unfortunate given the substantial impact that disability decisions can have on the life of an individual and the resources of an institution. One reason for the scarcity of research is the difficulty of designing studies about accommodations that are at all applicable across settings. For example, evaluating the impact of using a note taker for a small class might not be relevant for lectures in an auditorium or business meetings within a large company. Each circumstance is unique, as is the individual who operates within that setting. It is also daunting to conduct an analogue study that simulates motivational factors, particularly when it comes to high-stakes exams. Consequently, there is little research on specific accommodations for ADHD individuals with certain impairments who are engaged in a given task (e.g., test, class assignment, or work activity).

Another challenge in conducting research is that to verify an intervention as legitimate, the researcher has to demonstrate that it does indeed specifically minimize the impact of the disability. An accommodation that represents a general benefit to anyone, disabled or not, is not truly an accommodation. For example, if extra time helps everyone, it is no longer an intervention, such as cuts in the sidewalk or Braille versions of examinations that only help individuals who have physical or visual disabilities (see Phillips, 1994; Sireci et al., 2005; Zuriff, 2000).

Educational/Classroom Accommodations

Educational or classroom accommodations for students with ADHD often focus on setting accommodations, that is, engineering the environment to enhance learning. Examples are seating at the front of the class near the teacher, reducing distractions in the classroom, keeping stimulating peers at a distance, and sometimes using a carrel or cubicle for quiet seatwork. Some have found that headphones or earplugs can reduce distractions from noise. These modifications of the setting, in theory, might help the student with ADHD to attend, focus, and learn better. The ultimate setting adjustment is to provide a lower student–teacher ratio and more individualized instruction. Private rooms are often used for testing purposes so as to minimize distractions and allow a proctor to keep the student on task.

Presentation accommodations are less commonly applied to the classroom. However, because students with ADHD often report that they must read material several times before they can comprehend it, it makes sense for instructors or proctors to read instructions or test questions aloud. Another strategy is to break

a large test or assignment into sections to ensure that the students understand the task at hand. Rather than overwhelm the students, or have them rush through an exam just to finish, this type of presentation can mitigate problems with time management, perseverance, and motivation. Of course, these kinds of accommodations are harder to apply to higher levels of schooling.

Another presentation accommodation is the use of computer technology in both instruction and test taking. Students with ADHD may find that computers make the material more interesting and the process of responding to test items or completing assignments easier. Use of a laptop for taking an exam, writing lecture notes, or completing assignments can therefore represent reasonable accommodations.

Timing accommodations are the most widely requested intervention for students with ADHD, especially as they progress to high school and encounter more challenging exams and assignments. Conventional thinking holds that extra testing time can overcome a variety of weaknesses due to ADHD, such as slow processing speed, poor working memory, loss of focus and concentration, distractibility, disorganized thinking, rereading material, and so on. Most Section 504 Plans for students with ADHD recommend 50–100% extended time for exams. On occasion, extra breaks are justifiable for certain examinations. Although it is rare, some lengthy tests (e.g., 8 hours for a professional licensure) have been administered over multiple days. All of these timing accommodations conceivably could help students with ADHD better access exams, although extended time is the most favored of these accommodations. Extra time also can be used in the classroom for completing assignments and homework. Teachers ostensibly provide a time accommodation when they reduce the amount of work required (e.g., complete 10 multiplication problems rather than 15).

Response accommodations are not particularly common because students with ADHD generally have good speaking and motor abilities. Some situations, however, can be amendable to such interventions. For example, students with ADHD who are impulsive and prone to making simple mistakes may make transcription errors when using a bubble sheet (Scantron sheet) on a multiple-choice test. Instructors may opt to have these students circle answers on the test rather than use a bubble sheet.

Because many students with ADHD have poor handwriting (see review by Racine, Majnemer, Shevell, & Snider, 2008), it might benefit them to dictate responses to a recorder or computer with speech recognition. The use of spelling and grammar checking on a computer also may offer an accommodation that removes some of the careless mistakes made by these students. Others have observed that students with ADHD tend to perform less well in mathematics than do peers (Zentall, 2007). An appropriate response accommodation for this impairment might be the use of a calculator. Both computers for writing and calculators for math are becoming such standard issue in classrooms and for exams that they are hardly considered to be accommodations any more. In fact, because more and more tests are computer-based, it should be possible to design tests that allow for a variety of presentation and response accommodations, which in turn can be adapted to the needs of students with disabilities, if not all students.

As for comfort measures in the classroom, some students with ADHD may need to take liquids or a small snack during long classes or exams. One student we know carried a handheld fan to keep him cool throughout the day because his effort, attention, and self-control diminished if he became overheated. Another comfort measure might be a heavy, immovable chair with arms that limit fidgeting and overall mobility.

Despite the many classroom accommodations we have reviewed, we are hard-pressed to find research support for any of them. Recommending classroom accommodations for a particular student therefore relies more on intuition than on science.

HIGH-STAKES TESTS

The accommodations setting that has drawn the most attention from students, clinicians, testing organizations, and lawyers involves high-stakes tests, such as the Scholastic Aptitude Test (SAT), American College Testing (ACT), and various examinations for medicine, law, and other professions. The reason for the focus on accommodations is because performance on these exams may be very important.

The vast majority of requests for accommodations are for extended time, followed in popularity by a quiet test setting (private or semiprivate). Other applicants may ask for extra rest breaks, a computer with screen-reading software, and/or permission to use a computer, headphones, earplugs, calculator, or a paper-and-pencil version of an exam typically administered by computer.

As always, two fundamental questions arise when considering test accommodations for individuals with ADHD in higher education:

1. What evidence is there that such students actually demonstrate deficits in test taking?
2. Does research support the notion that extended time on a test alleviates any test-taking limitations for only those with the disorder?

The answer to the first question, as best as we can surmise from the small literature, is counterintuitive. What research there is suggests that individuals in higher education who have been diagnosed with ADHD are *not* likely to have substantially more problems taking tests than most students. Research by Lewandowski, Hendricks, and Gordon (in press) indicates that students with ADHD tend to access as many test items as their peers in a standard amount of time, although they may make more errors on certain tasks. For example, they found that high school students with and without ADHD performed similarly on a test of reading speed and completed essentially the same number of test items on a time-constrained reading comprehension test, suggesting that they were not really impaired on this task relative to peers. The researchers did find that students with ADHD tended to make more errors on measures of reading decoding, vocabulary, and comprehension, yet they were able to access the same amount of each test in a given time period. In other words, ADHD did not restrict their ability to access the test because they were able to answer the same number of items as nondisabled peers.

Lewandowski, Gathje, Lovett, and Gordon (2013) found similar results on the same reading measures in a sample of college students with and without ADHD. In their study, the groups did not differ in accuracy or speed on any of the reading tests. Interestingly, the ADHD group had higher scores on an index of test anxiety and lower scores on an index of test confidence. College students with ADHD performed as well as their peers, yet they were more worried and less sure about their performance.

In a recent investigation, Miller, Lewandowski, and Antshel (in press) found that college students with an ADHD diagnosis who were receiving test accommodations at school performed just as accurately and as fast as peers on the timed Nelson–Denny Reading Comprehension Test, administered under standard time conditions. The students with ADHD significantly outperformed their peers when they alone were provided extended time. Again, nothing in these data pointed to a disadvantage for students with ADHD, even though they often believed that they had difficulty completing tasks (including exams) in a timely manner, including exams.

How is it that postsecondary students with ADHD fail to show significant deficits in test taking? After all, the symptoms associated with the disorder would seem to directly impair test performance. The answer may relate to the level of impairment that is typical of individuals with ADHD who nonetheless function well enough to gain admission to college. According to all studies that have followed children diagnosed by research criteria as having ADHD during childhood, only a small percentage function well enough to make it to college (approximately 20%; see Chapter 12). Perhaps those students with ADHD who make it to college function well enough that test-taking deficits are undetectable. It also might reflect the low threshold that some clinicians use to judge what constitutes sufficient impairment to warrant a diagnosis.

It is telling that many students with disabilities who are granted extended time may not take full advantage of that benefit. For instance, Cahalan-Laitusis, King, Cline, and Bridgeman (2006) found that students with LD and ADHD who were granted extended time accommodations on the SAT generally used very little of the extended time they were granted. Of course, even the relatively small amount of additional time taken may have been used because it was available, without providing any benefit; that is, we do not know whether the students with disabilities could have worked faster if they needed to do so. Pariseau, Fabiano, Massetti, Hart, and Pelham (2010) examined this possibility in a sample of children with ADHD; students given less time to complete worksheets worked at a faster rate and answered more items correctly per unit of time. Their results suggested that at least some students with ADHD can adjust their work pace and adjust to standard time limits when asked to do so.

As for the question about the efficacy and legitimacy of extended time for individuals with ADHD, the literature again is generally unsupportive. Miller and colleagues (in press), mentioned earlier, systematically compared extended and standard time performances in students with and without ADHD. They also were interested in what would happen when the

ADHD students alone were given an accommodation of 50% more time. In this condition, students with ADHD performed significantly better than peers who were given no extra time; when given double the time (100% extra), the ADHD group answered 103% more items than their peers who were given only standard time. Therefore, although it may seem that students with ADHD process some information more slowly on some tasks (e.g., reaction time tasks), they may not perform significantly slower on timed academic tests (e.g., reading comprehension), and may not need extended time, especially double time. In fact, these recent findings beg the question of whether extended time is even a valid test accommodation for college students with ADHD. Of course, test accommodation decisions must be made on an individual basis and may still be appropriate for some students with ADHD.

It appears that even the research on test accommodation preferences has yielded mixed findings. For example, in a study of 15 high school students with ADHD in the United Kingdom, most students were ambivalent about receiving accommodations, expressing concern that they would be held to higher standards of performance because of the accommodations, and that their peers would perceive the accommodations as unfair (Taylor & Houghton, 2008). Perhaps this is one reason why students with ADHD may not even disclose their disability in college or the workplace. More recently, Lewandowski, Lambert, Lovett, Panahon, and Systma (2014) conducted a survey study of college students (n = 137) with and (n = 475) without disabilities (ADHD and LD primarily), and found that a majority of all students (approximately 87%) thought that receiving 50% extra time on exams would help their performance, and over 50% of both groups would want a separate exam room as well. Despite the differences in these two studies, the volume of accommodation requests in postsecondary schools and testing agencies seems to align with the finding that accommodations are generally desirable.

In addition to extended time, students with ADHD often request a private testing room to minimize distractions. However, once again, we have no empirical support suggesting that this accommodation is effective and specific only to persons with ADHD. In fact, we could only find one study on private room testing, and it did not involve students with ADHD. Lewandowski, Wood, and Lambert (in press) examined timed reading comprehension performance of typical college students taking tests in a group or in a private room. Somewhat surprisingly, they found better performance in the *group* testing situation (d = 0.53). It seems plausible that there is a social facilitation effect on test performance in a group that is not present in a private test situation (Guerin & Innes, 2009). Also, it should be noted that examinees taking tests in a private room rather than the usual classroom do not have the benefit of a class instructor to answer questions and clarify directions. For these reasons, one might want to think twice about the utility of a private room for testing.

Another test accommodation that makes logical sense for test takers with ADHD is extra breaks. Based on the behavioral and executive function characteristics of this group, as noted earlier, one might predict that sustained attention, vigilant concentration, and ongoing use of working memory would present challenges to individuals with ADHD. They describe sustained mental activities as very effortful and tiring. To break this cycle and allow those with ADHD time to "recharge their batteries," extra breaks are sometimes requested and given during lengthy tests. No research has examined the validity and efficacy of extra breaks on the test-taking performance of students with and without ADHD, or any disability for that matter. Some limited research on testing over multiple days suggests no benefit for students with or without disabilities (Walz, Albus, Thompson, & Thurlow, 2000). In fact, Burns (1998) cautioned that breaks within a test may distract and disrupt the natural problem-solving rhythm a student developed.

With regard to one's rhythm in taking a test, Lee, Osborne, Hayes, and Simoes (2008) studied the effects of self-pacing versus computer pacing on the test performance of college students with ADHD. They tested all students on a computer, with half of the students randomly assigned to a self-paced condition and half to a condition in which the computer presented items at a fixed pace. They found no difference between the conditions but noted that the computer pacing made some students more anxious. In general, students liked the computer testing environment, and they preferred an isolated and quiet setting. It should be noted that this is one small study and the preference information was based on qualitative responses to open-ended questions.

The trend in the limited available research is that just about nothing shows a differential and specific testing benefit solely for examinees with ADHD. This con-

clusion is somewhat discouraging given what we think we know about their various behavioral symptoms and neurobehavioral problems. Despite these limitations, students with ADHD do not perform all that differently on a variety of timed tests, and they do not seem to need or use extra time in most cases.

Workplace Accommodations

Research has indicated that more people with ADHD are unemployed than peers, and that they have reduced and poorer quality of work performance, more sickness and absence, more quitting and firing from jobs, and generally lower salaries (see Chapter 12; Barkley et al., 2010; Gjervan, Torgersen, Nordahl, & Rasmussen, 2012; Halbesleben, Wheeler, & Shanine, 2013; Küpper et al., 2012). Even college students with ADHD have been shown to have more work-related problems, poorer work performance, and more firings than peers (Shifrin, Proctor, & Prevatt, 2010).

Despite these findings, hardly any scientific research is available to guide the design of workplace accommodations for individuals with ADHD. Therefore, the accommodations we present here are based on common sense rather than empirical direction. Given the lack of scientific research, we are not in a position to endorse particular accommodations at work, but we do share some recommendations that have been generated by authors in the ADHD field (Barkley, 2013; Nadeau, 2005; Ramsay, 2010).

Unlike educational and testing institutions that are well acquainted with individuals with disabilities, including ADHD, employers may have limited experience and knowledge in dealing with an employee with ADHD (not to mention that each person with ADHD can be quite different). Furthermore, not all employers are obligated to provide accommodations to employees with ADHD, in that ADA law only applies to private businesses with more than 15 employees. In addition, persons with ADHD, and other hidden disabilities, often do not disclose their disability to an employer (Newman et al., 2011). Consequently, there are probably a small number of adults with ADHD who receive formal accommodations in the workplace.

A number of authors have noted the importance of assessing the person with ADHD for strengths and weaknesses, making the employer aware of the condition and important characteristics, fitting the job to the person with ADHD to the extent possible, and making adjustments (and accommodations) that can make the person a productive employee (Nadeau, 2005; Ramsay, 2010). These authors offer advice to employers that are noted in the matrix shown in Figure 33.1. For example, they mention a more flexible work schedule, shorter work tasks with more breaks and incentives, more systems in place to keep the individual organized and on task, weekly supervision meetings, oral and written instructions, computer and other technologies that promote efficiency and minimize paperwork, as well as an environment that provides physical structure, reduces distractions, and allows for movement. What seems to be most important is an employer/supervisor attitude that shows understanding, care, and support, through thick and thin.

While some of these recommendations go well beyond what are typically considered accommodations, they all are alterations and adaptations used in a workplace to mitigate the impairment of ADHD, so that the employee can access the job and utilize his or her skills. In this way, the workplace alterations can be viewed as parallel to accommodations we see in schools and on exams. Speaking of exams, employees at times do take tests in order to maintain a certification or gain a promotion. Whether one works as a mechanic, postal carrier, Private in the Army, or truck driver, there may be paper-and-pencil tests necessary to attain or maintain a job. In these cases, the accommodations noted for the classroom and high-stakes exams would seem to apply. It should be noted that the accommodations noted earlier may be applicable to those individuals that are "otherwise qualified" to perform a particular job. If a person's disability interferes with performing the fundamental aspects of a job, an employer is not obligated to find a way to place the individual in the job. Someone whose ADHD causes symptoms of distractibility, inattention, impulsivity, and time misperception may not make a good air-traffic controller, even when using medication.

CONCLUSIONS

Notwithstanding the many requirements and caveats we have detailed in this chapter, we do not intend to portray the documentation of disability as an impossibly laborious or futile process. Most individuals who truly meet criteria for ADHD will have no trouble providing solid evidence of impairment over a lifetime. They likely will have been diagnosed already because of consistent struggles at school, at home, and in the

community. Herein lies the key advantage of evaluating adults for ADHD as opposed to youngsters: Individuals seeking ADA accommodations are known quantities, with a lifetime of experiences they can bring to the diagnostic table.

What may be somewhat more challenging for clinicians is coming to terms with the level of impairment the ADA requires in qualifying an individual as disabled. While some have argued for applying the "average person standard" across the board (Gordon, Lewandowski, & Keiser, 1999), it still is not the metric used by DSM-5 or many clinicians. It can be challenging to explain to clients that their problems, although distressing to them, may not rise to the level of a legal disability. That conversation is not common for practitioners who are trained to provide comfort.

Perhaps it can be reassuring to remember that accommodations are not necessarily a key to success on a test, in school or on the job. In the case of extra time on a test, it may not help someone who fails to use the normally allotted time wisely to have even more time to mismanage. Other individuals, especially those with problems that do not qualify as disabling, might better spend their time, money, and energy on studying or problem solving than on pursuing formal accommodations.

It is also important to keep in mind that the process around determining disability is not designed to be adversarial in nature. Our experience is that most institutions and businesses are committed to accommodating individuals with handicapping conditions. They are not intent on precluding people with bona fide disabilities from having the opportunity to take an exam, graduate from college, or work at a job they would otherwise manage. Similarly, the professionals that educational institutions and businesses might hire to review documentation, more often than not, are clinicians themselves, with solid records regarding patient advocacy. Therefore, the individuals who review a clinician's evaluation are not out to find reasons to deny the request. If anything, they tend to err on the side of granting accommodations.

Having portrayed the accommodations process as reasonably benevolent, we also acknowledge that administrators constantly wrestle with competing agendas. While they understand the responsibility to protect the civil rights of individuals with disabilities, they also have an obligation to uphold institutional standards and maintain a level of fairness for all. Granting accommodations requests too liberally could result in exams that are no longer predictive, lower academic standards, and professionals who are licensed to practice despite having had an unfair advantage on a credentialing exam. This might also create an overall sense that too many well-functioning individuals can "game the system" by taking advantage of a law designed to protect the truly disabled. Summarily granting accommodations to anyone who applies can therefore create a cascade of adverse consequences.

In our view, the most unfortunate circumstances are those that involve clients who are struggling in a training program or on the job, not because of a formal disability, but because they have chosen a career that does not match their particular talents. While they look to accommodations as a way of promoting their success, they might do better if they explored educational or vocational options more suited to their abilities. Clinicians who conduct ADA evaluations are well positioned to offer that decision-making support. They are also able to recommend strategies that are more geared to remediating weaknesses than necessarily compensating for them via accommodations. For example, some students might be better off improving study stills, learning test-taking strategies, and improving reading speed than pursuing formal accommodations for extra time or breaks.

KEY CLINICAL POINTS

- ✓ Conducting ADA-related assessments demands specialized knowledge about the legal definition of disability, the nuances of documentation requirements, and the complexities involved in establishing what constitutes a reasonable accommodation. The role requires shifting from that of a clinician advocate to that of an impartial individual in a quasi-forensic process, who seeks to objectively document the criteria necessary to gain such accommodations.
- ✓ Important for clinicians to appreciate is that the ADA (1) is not designed to grant entitlements but to protect individuals with disabilities from discrimination; (2) that a clinical diagnosis alone is not synonymous with a legal determination of a disability, as even DSM-5 acknowledges; and (3) that, in the case of ADHD, evidence of substantial real-life impairment over time must be obtained. The standards for an ADA determination of disability set a higher bar than do those for clinical diagnosis and therefore require a greater degree of

evidence or documentation that both symptoms and impairment exist to a reasonable degree.

- ✓ The determination of impairment and disability for the ADA is based on the "average person standard" and not on some intrapersonal discrepancy, such as that between IQ and some other measured cognitive or academic ability, and therefore requires evidence that the individual functions ineffectively and substantially less well than the average person in the population.
- ✓ No standardized psychological test is sufficient for documenting the diagnosis of ADHD or of impairment arising from that disorder. Evidence must also be derived from the real-life functioning of the individual, as may often be reflected in the archival record of functioning in major domains of life activity (education, work), and previous accommodations granted to the individual for his or her disability.
- ✓ Documentation of a disability for ADA purposes requires relative currency of the evaluation (typically within 3 years), demonstrated professional qualifications of the examiner to make such a determination, evidence that the diagnostic criteria for the disorder have been utilized in the evaluation, and evidence that the functional impairment that exists is a result of the disorder.
- ✓ Requested accommodations must be targeted to both the disorder and the specific needs of the individual related to that disorder, must be justified, and must be reasonable for the context or setting in which the accommodations are to be made.
- ✓ Because ADHD most often involves not only symptoms of inattention, impulsivity, and excessive activity but also poor self-regulation, self-motivation, and time management, accommodations that target these difficulties may be justifiable.
- ✓ Clinicians will find it helpful to think of accommodations as falling within a matrix (see Figure 33.1) that comprises setting, presentation, scheduling, timing, and response accommodations.
- ✓ Many of the popular accommodations requested for individuals with ADHD have little, if any, research documenting their differential effectiveness for persons with the disorder compared to those without it, such as extended time on tests or taking exams in distraction-free environments. Others remain at the level of commonsense accommodations that remain to be studied but have some reasonable basis given what is known about the disorder.

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CHAPTER 34

Counseling Couples Affected by Adult ADHD

Gina Pera

"Brad, we've got to talk," says the woman at one end of the sofa to the man at the other end, who casually looks up from his newspaper. What makes this scenario noteworthy is that the sofa floats midriver—on the precipice of a towering waterfall.

Literally, this describes a cartoon appearing in *The New Yorker* (Mankoff, 1991). Figuratively, this describes the predicament of many couples upon finally learning—or at long last *accepting*—that attention-deficit/hyperactivity disorder (ADHD) might lie at the core of their inexplicable discord, chaotic domestic lives, or frustrating and even infuriating "miscommunications." By the time they discover that one or even both may have ADHD, they teeter precipitously on the edge—of divorce, of bankruptcy, of job loss, of the last shred of goodwill between them. Or they are already crashing on the rocks below and trying to keep their heads above water.

Typically, they have already tried therapy (individual, couple, and various permutations in between, often multiple times) and plowed through self-help books. What little success they might have achieved often proves transitory: When therapy stops, so does accountability. When their situation devolves, such couples are unlikely to reengage in therapy—it is simply too time-consuming and costly, with no lasting change—until they have nothing left to lose. If they have seen only therapists who remain unaware of the effects of ADHD on these couples' lives or who lack specific techniques for addressing it, therapy itself can perpetuate pessimism. Standard couple therapy is not enough and can even cause harm because it often replicates, perpetuates, or exacerbates the self-defeating cycles of these couples (Pera, 2008).

It is possible that ADHD was mentioned long before the couple reached the edge. Perhaps there was a childhood diagnosis or an observant friend who knowingly gave the nod. But it was often dismissed due to misinformation ("What could a hyperactive little kid's disorder have to do with our grown-up problems?"), scary headlines about Big Pharma creating the myth of adult ADHD, or flat-out denial and minimization by either partner as well as their therapists or physicians. Meanwhile, the damage swells. When the couple finally declares readiness to accept that ADHD might form the foundation of their problems, they grab the diagnosis as they would a lifeline. Then their multitudinous needs can lunge at the therapist like some giant B-movie oc-

topus, each tentacle demanding immediate attention and leaving both therapist and clients vulnerable to the same sense of feeling overwhelmed.

Naturally, other couples affected by ADHD float further upstream, gently gliding along in calmer waters, never knowing that just around this bend or maybe the next—when they load their precarious little boat with children, for example—their unsustainable accommodations around ADHD-related patterns can suddenly spring a leak, threatening to sink the whole enterprise. With early education and interventions, they can avert these disasters. In fact, some seek assistance proactively, feeling they are venturing too close to the rocks or are stuck in a swirling eddy. Others simply paddle faster.

It is easy to stereotype the couples who finally find their way to ADHD-informed couple therapy: the Nagged-Upon and the Nagger; the Fun One and the Serious One; the Creative One and the Plodder; or the Sparkly Helium Balloon and the Little Bag of Rocks that keeps the balloon from soaring. But these are easy platitudes formed on superficial appearances, typically describing the end result but not how these individuals started out in their life together. In other words, apparent "opposites attract" pairings might instead be the end product of ADHD-infused relationship dysfunction, not the cause of it. Turning the tables, the pair might be characterized as the Reliable One and the Screw-Up, the Responsible Parent and the Child, the Steady One and the Flake, or even, it must be acknowledged, the Martyr and the Narcissist. These characterizations also reflect a certain reality but leave no room for the "unreliable" partner to gain tools and strategies to "grow up" and assume equal footing in the relationship. Beyond reducing complex people to caricatures, such characterizations also preclude the possibility of dual-ADHD couples. Their respective symptoms typically manifest so differently that one partner goes undiagnosed for years until the other, often after medication treatment, gains enough clarity to start piecing together the puzzle.

Yes, ADHD can create common problematic patterns in adult relationships (see Chapter 12), and this chapter explores them. But fundamentally, these millions of couples have only one thing in common: They are all dealing with highly variable aspects of ADHD and its coexisting conditions, along with all the other variable qualities and resources that humans bring to their intimate relationships and domestic lives. They need much more than template procedures or cookie-cutter bromides. It takes a considered clinical approach to sleuth out each couple's specific challenges, viewed most obviously and pervasively through the multifaceted ADHD lens but also widening that lens to include all the other issues that traditionally bring couples to therapy.

Most couples have disagreements about household chores, coparenting, and the like. Yet just as ADHD symptoms are human traits writ large—everyone forgets or gets distracted at some time or another but not to the degree suffered by adults with ADHD—standard "couple troubles" prove much more intense and debilitating when adult ADHD is involved. As Patrick Kilcarr writes, "Symptoms attending the presence of ADHD in a partnership can 'suck the air' right out of even the most promising relationship" (2002, p. 220). Compounding the fallout from the forgetting, the chaos, the poor communications, and the disorganization are the misattributions—that is, the faulty assumptions that the behaviors spring not from neurobiology but from lack of love or caring.

Effective couple treatment for ADHD should blend general evidence-based marital therapy with strategies targeting the neurobiologically based problems of ADHD (Pera, 2008, 2011; Pera & Robin, in press). To guide the clinician, this chapter first summarizes research examining the effect of adult ADHD on relationships, including the impact on both partners of documented challenges around driving, money, sex, health habits, and sleep; next, this chapter presents ADHD-informed interventions based on five principles of empirically supported couple therapy. Although research findings that pertain to these topics are touched upon in prior chapters (Chapters 11 and 12), here they are discussed relative to their impact on the cohabiting relationship.

This chapter's naming convention is "ADHD partner" and "partner." The problems with the more traditional "ADHD partner and non-ADHD partner" polarity are several, most prominently, (1) it precludes the possibility that both partners have ADHD, with each feeling the effects of the other's ADHD-related challenges in the same way that "non-ADHD" partners can, in addition to dealing with their own ADHD-related challenges; and (2) it tends to encourage overdependence on one partner to compensate for the other's ADHD-related challenges. This naming convention is used except when I cite research results that use other terminology.

RESEARCH ON ADHD AND ADULT RELATIONSHIPS

"Little is known about the family relationships of adults with Attention-Deficit/Hyperactivity Disorder," reads the introduction to one of the rare studies examining the subject (Eakin et al., 2004, p. 1). More than a decade later, formal research still lags behind other adult ADHD topics. Yet relationship problems constitute one of the most common complaints of adults with ADHD seeking treatment, many verging on divorce or breakup (Murphy, 1998). Popular consumer books detail the challenges to and suggestions for improving romantic intimacy (Halverstadt, 1998) and also examine the gamut of domestic issues, from driving safety to denial of the disorder, along with adult ADHD psychoeducation and evidence-based treatment strategies (Pera, 2008). Moreover, clinical reports over the past two decades describe the various problematic effects of ADHD on relationships (Betchen, 2003; Brooks, 2002; Dixon, 1995; Kilcarr, 2002; Pera, 2011; Pera & Robin, in press; Ramsay & Rostain, 2007; Ratey, Hallowell, & Miller, 1995; Murphy & LeVert, 1995; Robin, 2002; Quinn & Nadeau, 2002; Weiss, Hechtman, & Weiss, 1999; Wender, 2001), including the following:

- Disorganization; forgetfulness; poor follow-through
- Combative communications; poor listening
- Problematic parenting style
- Poor decision making and cooperation
- Self-centeredness and insatiability (hard to please)
- "Learned helplessness"
- Impulsive spending; acting without thinking of consequences
- Low intimacy
- Moodiness and temper; low frustration tolerance
- Reactive aggression toward intimate partners
- Lack of interpersonal sensitivity and impaired empathy
- Mental rigidity; difficulty with transitions, compromise, and cooperation
- Poor coping strategies developed over a lifetime of unrecognized or unaddressed ADHD neurobehaviors, such as avoiding, quitting, controlling, being aggressive, rushing through tasks, overgeneralizing, procrastinating, denying, minimizing, and externalizing (blaming other people or external events for unsatisfactory outcomes).

Unlike adults with ADHD, their partners can be viewed as a group only in one sense: They share varied experiences of being in relationship with a person who has a highly variable syndrome associated with various comorbidities. Even when the partners themselves have ADHD, however, the range of experiences of and reactions to an ADHD partner's behavior are predictable. Psychologist Herbert Gravitz (2004), an expert on the impact of disorders such as alcoholism and obsessive–compulsive disorder on family members, groups the stress responses of living with a partner's unrecognized, unaddressed ADHD symptoms into three stages, each accompanied and exacerbated by an intensified effort to cope (see interview in Pera, 2008):

1. *Explaining the inexplicable.* Faced with an ADHD partner's confusing actions and excuses, the partner attempts to make sense of them. It might start with denying or minimizing the behaviors ("Not everyone is good with money" or "All new relationships have their problems"). Failing to elicit improved behavior and not knowing what else to do, the partner may gradually start to compensate, taking over an increasingly unsustainable and inequitable share of responsibilities—sometimes without complaint, sometimes complaining vociferously.

2. *Managing the unmanageable.* Prolonged attempts to cope can result in destructive patterns that worsen over time, such as becoming hypervigilant to preventing disasters and hypercritical of the ADHD partner's every misstep and failing ("Can't you do anything right?" and "Why won't you listen?"). Clarity can be further obscured and anxiety is exacerbated by the ADHD partner's low insight or poor coping strategies around denial, minimization, avoidance, and blame. Lacking validation for their perceptions from their ADHD partners or outsiders who are not privy to the depths of domestic dysfunction, the partners lose touch with their own feelings. Some grieve for the reality of their relationship, compared to their initial hopes, especially when the "hyperfocus courtship" days end and their ADHD partners' focused interest flits from them to the next stimulating interest or hobby. "I wanted a partner, not a child" is the common lament. They miss that person they fell in love with; if they blame themselves for being unable to inspire a reappearance (or if they accept their ADHD partners' misplaced blame of them), they keep trying.

3. *Breaking down.* Ongoing stress responses can result in increasingly angry or anxious reactions ("Why did I ever marry you?"; "You're the most selfish person I ever met!"; "You don't care that you're killing me!"; and "You're crazy!") and eventually physical and mental illness. At that point, superficial appearances threaten to cast the partner as the "identified patient" (or at least anger-management class candidate), with the ADHD partner's comparably calmer demeanor reflecting not stability so much as cluelessness.

Published research does associate adult ADHD with a higher risk of relationship and marital distress, separation, and divorce (see Chapter 12; Barkley, Murphy, & Fischer, 2008; Biederman et al., 2006; Klein et al., 2012). Yet only a few published studies, and mostly on a small scale, have examined the day-to-day particulars:

- Greater severity of ADHD symptoms in university students has been associated with greater stress and, in their romantic relationships, a greater tendency to use maladaptive coping strategies such as disengaging from or denying their problems and resorting to wishful thinking or using substances (Overbey, Snell, & Callis, 2011).

- Eakin and colleagues (2004) found poorer marital adjustment and family functioning in ADHD couples than in comparison adults and more negative ratings in affective involvement, roles, communication, and problem solving. Interestingly, the adults with ADHD held more negative perceptions of the health of their marriages and families than did their spouses. The authors offer two possible explanations: (1) ADHD partners' greater negative affect in general may have negatively influenced their perceptions (see Chapter 3), and (2) spouses may have perceived their efforts to compensate for their ADHD partners as benefiting their marriage and families, which causes them to evaluate their marriages more positively than did their ADHD partners. Still, 96% of spouses reported that their ADHD partners' behavior interfered with functioning in one or more domains, primarily in general household organization/time management, childrearing, and communication and/or marital relationship. And 92% of the spouses reported compensating for their ADHD partners' difficulties in these areas, as well as in financial management, although non-ADHD wives compensated more for their ADHD husbands than did non-ADHD husbands for their ADHD wives. Compared to controls, the spouses of those with ADHD showed no differences in psychiatric health.

- Minde and colleagues (2003) found that family and marital functions were impaired in ADHD-affected families. The authors emphasized careful assessment of the non-ADHD parents because they seem to influence significantly the well-being of the children.

- Robin and Payson (2002), in a pilot study, developed a Marital Impact Checklist to assess the impact of common behaviors of adults with ADHD on their marriage. They identified 10 top trouble spots:

1. Doesn't remember being told things
2. Says things without thinking
3. Zones out in conversations
4. Has trouble dealing with frustrations
5. Has trouble getting started on a task
6. Underestimates time needed to complete a task
7. Leaves a mess
8. Doesn't finish household projects
9. Doesn't respond when spoken to
10. Doesn't plan ahead

Of particular interest: ADHD and non-ADHD spouses consistently concurred in their ranking of the most problematic behaviors, namely, items that lead the non-ADHD spouse to feel unloved, unimportant, or ignored. Echoing the earlier finding from Eakin and colleagues (2004), spouses with ADHD endorsed a significantly higher number of issues and reported significantly higher "unloved and negative impact ratings" than the spouses who did not have ADHD.

To date, the largest, most comprehensive survey of adult ADHD relationships is the ADHD Partner Survey, which I conducted in 2004–2005 (Pera, 2008). It comprises 173 questions covering wide-ranging topics. The 111 respondents were recruited through support groups and included only the partners of adults diagnosed with ADHD; all knew their ADHD partners before medication treatment. Its results in many areas, such as Interpersonal Violence (IPV) and parenting, approximate those of subsequent studies.

To give the clinician a sense of symptom prevalence in these relationships, Figure 34.1 lists the DSM-IV-TR ADHD symptoms, ranked according to respondents' selection of any traits that describe their ADHD part-

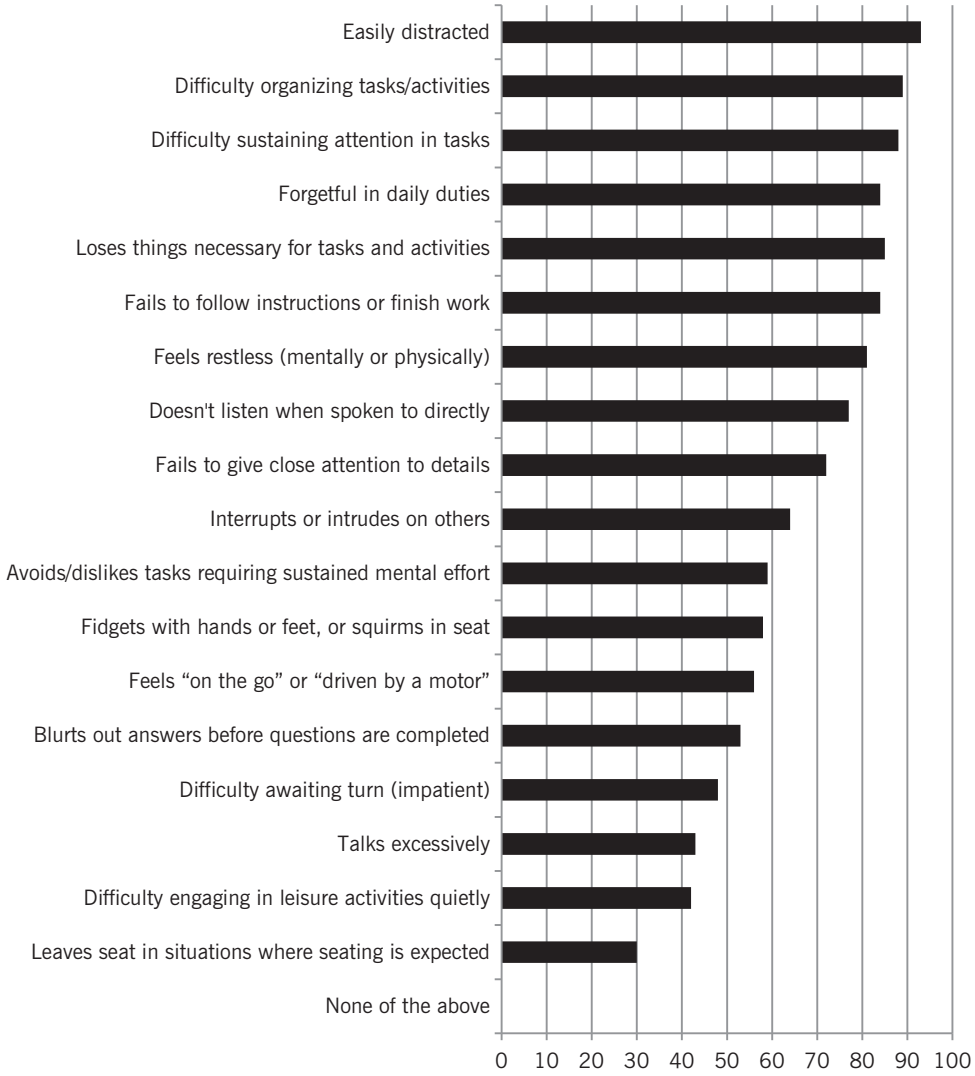

FIGURE 34.1. ADHD symptoms. Survey respondents selected each symptom (adapted from DSM-IV-TR diagnostic criteria) that described their ADHD partner's behaviors. From ADHD Partner Survey. Copyright by Gina Pera. Reprinted by permission.

ners' behaviors. Because symptom prevalence reveals only a part of the picture, Figure 34.2 depicts respondents' rating of commonly reported problems. This figure emphasizes a fundamental step in working with couples to curb emotional chaos: guiding them to put symptom terminology aside and identify their specific problems so they can focus on solutions. For example, if organization is the biggest problem (as it often is), it will do little good to work on "communications techniques" or "intimacy" until organization—of time and matter—is improved.

THE ELEPHANTS IN THE ROOM

Decades of well-documented research tells us that ADHD-related impairments extend well beyond the "interpersonal" into every domain of life, from education to occupation to financial management. Yet therapists working with these couples too often set this aside to focus primarily on dyadic interactions, often treating each partner as equally responsible for the couple's troubles. This, like other family systems-type approaches, is a common mistake, in that it fails to recognize the

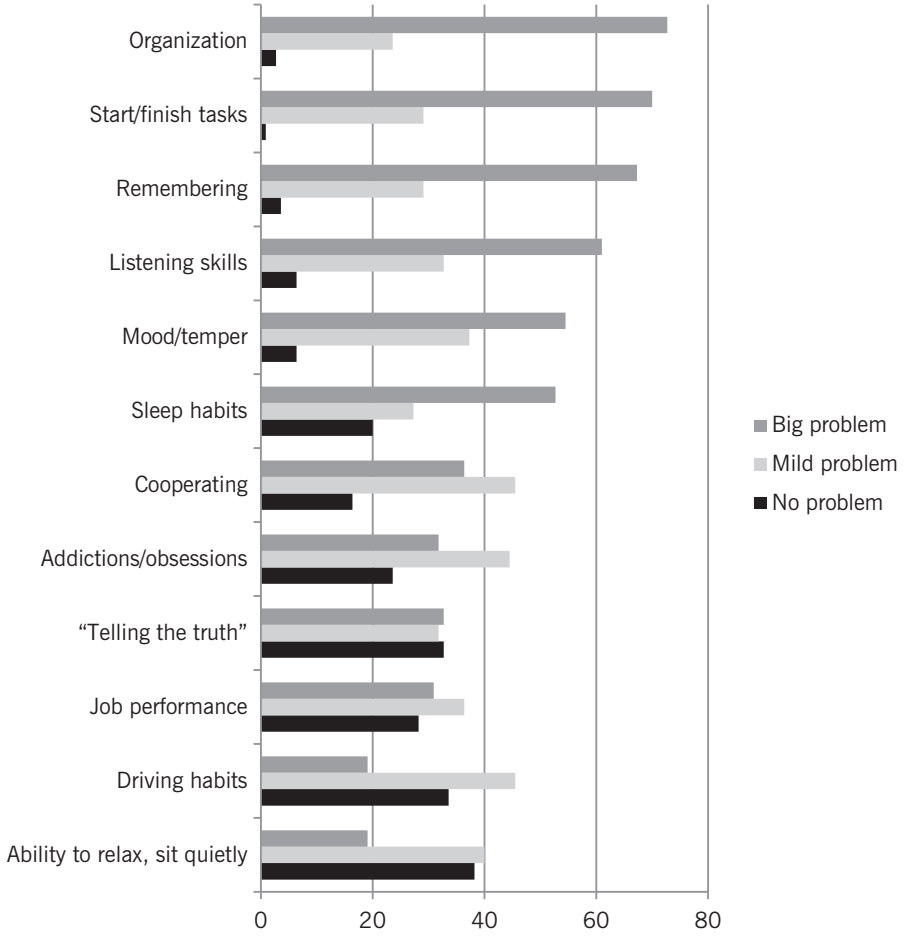

FIGURE 34.2. Big problem, little problem, or no problem? Survey respondents rated their ADHD partner's behavior or ability in each area as "no problem, a mild problem, or a big problem to the partner or the relationship." From ADHD Partner Survey. Copyright by Gina Pera. Reprinted by permission.

neurobiological, medical problem causing the turmoil. The culprit generally is not one partner or the other, or even both. The culprit is poorly managed ADHD, and the therapist can help the couple unite to counter the effects of ADHD together. In reality, couples frequently go on to solve their own interactions once the elephants in the room—a veritable circus parade of executive function deficits—quit trampling on the dyad. The clinician who focuses solely on interpersonal dynamics risks missing the foundational, ADHD-related issues that etch like acid at the couple's mutual trust, goodwill, and sense of "being in this together."

The potential adverse outcomes of untreated ADHD are detailed elsewhere in this book. In this chapter, the following section highlights key areas, followed by first-person comments, to underscore the importance of reviewing all ADHD-related risk areas in any couple-based intervention. Typically, the couples have no idea that these foundational obstacles are related to ADHD; gaining the knowledge that they are not alone in grappling with these issues, that many of their problems stem from neurological rather than psychological or volitional causes, and that solutions might be at hand, all serve to motivate them throughout the treatment process and foster optimism. Even though some topics, such as sex and money, are traditionally viewed as therapeutic distractions from underlying psychological issues, they present a particularly legitimate line of inquiry for these couples (Pera, 2008; Pera & Robin, in press).

Interpersonal Challenges

The Research

"It's only in hindsight that you realize what indeed your childhood was really like," observes architect Maya Lin. Late-diagnosis adults with ADHD can look back at their formative years with newfound understanding. For years, often under therapeutic guidance, many misattributed their adult ADHD symptoms to troubled families of origin. With diagnosis and knowledge about ADHD's high heritability, they realize that family influence formed only part of the equation. In other words, interpersonal challenges likely started early, compounded by the other family members' ADHD. Nature *and* nurture. In this way, patterns of frustration, blaming self or others, criticism, and lack of understanding or feeling understood can begin in childhood, impair critical emotional development, and continue through adulthood.

In studying 1,001 adults with self-reports of diagnosed ADHD, Biederman and colleagues (2006) found that they were far less likely than controls to perceive that they

- Had fit in with their peers (27 vs. 60% controls)
- Were popular in school (19 vs. 36%)
- Got along with their teachers (44 vs. 63%)
- Were liked by adults (46 vs. 67%)
- Had a good relationship with their parents (35 vs. 64%)

The Personal Experience

Seeking to flesh out these documented early experiences with first-person remembrances, I conducted an informal survey of 44 adults diagnosed with ADHD primarily later in life (88% diagnosed after age 30). Asked if their ADHD symptoms adversely affected family relationships while growing up, only 7% said "no." The majority (66%) said, "Yes, very much so" and the balance, "Yes, somewhat." On the upside, a similar majority (71%) said, "Learning about ADHD and perhaps pursuing treatment has improved my relationships." Here is a sampling of perspectives of how their own ADHD affected interactions with family and peers:

- "I had problems keeping up in school; not hyper, just wandered into my mind to dwell on what I wanted to think about. No conflict. Nonconfrontational due to self-consciousness from my being 'spaced-out.' Hung out with a 'friend' who put me down throughout high school. Have hated myself ever since."
- "I was hyperactive and always getting into trouble in school, at home, and in the neighborhood, so this caused lots of conflict with my parents. I also would get obsessed about things, such as forcing my sister to play 4-day-long Monopoly sessions. My sister remembers that driving her crazy. Funny, at the time I thought she was enjoying it!"

Respondents provided vivid commentary on the seldom-discussed but genetically inevitable multiplier effect of ADHD in the family; not all respondents felt their family relationships were worse than average, of course, and some felt they were better, but it is easy to understand why some lacked familial role models for harmonious adult relationships:

- "It's hard to say if it was my ADHD or the fact that my father sexually molested me from ages 9 to 17."
- "I see clearly now that we were all poor communicators who did not take the time to actually figure out the differing viewpoints in a conflict. So that was bad. The good part about my sibling and me having ADHD is that we were quick to get over negative feelings. We didn't have the attention to stick with it! Ha!"
- "We fought. Verbally and physically. Uncontrolled emotion ran riot, with our mother leading the pack. There was no guidance or understanding of what was happening and why."
- "Brother got his 'stimulant medication' from bugging everybody."
- "Looking back, my entire family lived in an alternate reality. The widespread prevalence of ADHD made for a great deal of argumentation and lack of respect for others. The high degree of criticism I received while growing up led to low self-esteem."

Asked how learning about their own ADHD has improved past and current relationships, respondents agreed with statements that emphasized the importance of accurate diagnosis and psychoeducation:

- "I can now put my past problematic behaviors in context, taking past criticisms less personally." (73%)
- "I have explained ADHD to the important people in my life and gained their understanding." (62%)
- "I am better able to 'explain how my brain works' and work on mutually agreeable strategies with others." (69%)

One write-in response is a reminder that despite mutual effort within families or couples, past hurts sometimes elude healing:

> "It's absolutely true that ADHD education has helped me and those with whom I am in relationship to understand my challenges and try to find strategies to avert problems, but that doesn't tell the whole story. Despite dramatic improvement in many ways, so much damage had already been done that, despite best intentions, some relationships will likely never fully heal. It's important that we acknowledge as an ADHD community that the very real harms incurred do not simply evaporate when the 'Oh, it was ADHD' scales fall from our eyes. Information will not magically heal all. ADHD education may be always necessary. But it is not always sufficient."

Physical Intimacy

The Research

The study of ADHD relative to sexuality is still in the preliminary stages (Barkley et al., 2008). But it is safe to say that ADHD symptoms do not stop at the bedroom door (Halverstadt, 1998; Pera, 2008, in press). Longitudinal research on children diagnosed with hyperactivity suggests that their sexual activity starts at about age 15, compared to age 16 for controls (Barkley, Fischer, Smallish, & Fletcher, 2006). Upon sexual maturity, children diagnosed with hyperactivity are less likely to use contraception, and they experience higher rates of teen pregnancy and sexually transmitted diseases. Similar patterns were found in a study of adult males diagnosed with ADHD in childhood (Flory, Molina, Pelham, Gnagy, & Smith, 2006).

With adults, much of what we know comes from retrospective studies of adults with other disorders. Kafka and Prentky (1998) found a significant correlation between ADHD and paraphilias among sex offenders. In fact, childhood ADHD was the only Axis I disorder statistically significantly associated with *paraphilias* and aggressive forms of sexual impulsivity; Kafka and Hennen (2002) found that these individuals were likely to have the combined subtype of ADHD. By contrast, men with "sexual addiction," or hypersexual disorder (paraphilia-related disorders, or PRDs) and ADHD were more likely to have the inattentive ADHD subtype. Reid, Carpenter, Gilliland, and Karim (2011) found similar results among men seeking clinical help for hypersexuality. In research by Barkley and colleagues (2006), adults with the hyperactive subtype were twice as likely as those with inattentive subtype or controls to report having a lack of sexual desire at least sometimes or more often; they were also more likely to identify themselves as bisexual (7 vs. 1% of inattentive subtype group vs. 1% of controls). Clearly relevant for the couple experiencing stress regarding PRD issues (most often reported in support groups as compulsive masturbation and dependence on pornography or online "cybersex"): Kafka and Hennen (2000) found that stimulant medication along with selective serotonin

reuptake inhibitors (SSRIs) appeared to effectively mitigate the paraphilias and PRD in men.

Other effects of ADHD on sexual intimacy can be identified, from symptoms that directly interfere with maintaining focus during sex, managing sensory sensitivity, or summoning the motivation and organization to initiate sex (especially once the stimulating days of courtship end) to ADHD-related behaviors that create conflict or alienation in the rest of life. In my experience, adult ADHD support-group discussion tends not to venture into the topic of sex (perhaps due to embarrassment, because these individuals enthusiastically attend presentations on the topic). By contrast, the partners' group readily embraces the news: "You mean ADHD can affect sex, too?" Generally, partners' reports fall into two extremes: The adult with ADHD wants sex "all the time" or "never;" that is, the ADHD partner never initiates sex or rejects a partner's overtures (Pera, 2008). Yet almost one in five ADHD Partner Survey respondents report having a satisfied to exciting sex life, including many in long-term relationships, and almost half of respondents overall agree that their ADHD partner is "a skilled and considerate lover." Respondents dissatisfied with their sex lives selected from among the following various reasons:

- "I would feel sexier if my partner helped out more at home." (56%)
- "My partner blames our lack of sex on me, even though his or her reasons don't make sense." (40%)
- "The lack of sex is one of my biggest challenges in this relationship." (38%)
- "There is no 'connection' when we have sex; it doesn't even seem like my partner is 'there.'" (30%)
- "My partner never initiates sex with me. If we have sex, it's only because I initiate." (29%)

Notably, respondents who experienced an improved sex life say that it came about after their ADHD partners began taking stimulant medication. They credit the improvement primarily to the ADHD partner's increased initiative/motivation, in addition to heightened cooperation relative to domestic responsibilities rather than direct physical effects on symptoms in the bedroom—all of which suggest that certain couples need sex therapy less than laundry and bill-paying therapy, that is, the logistical guidance and habit development I describe later in this chapter.

The Personal Experience

ADULTS WITH ADHD

- "When you enter into a long-term relationship, sex becomes a regular, required event. How many ADHDers relish routine and requirements? I love my wife, but I don't know how to fix this."
- "My fiancé is a great lover, but spending an hour in foreplay, intercourse, and snuggling after? Just the thought leaves me feeling cornered and panicky. A quickie is my limit."
- "I find sex much more interesting to talk about, to think about, than to actually do with another person. That requires too much effort, coordination, and planning—and always the risk of criticism for not doing it well."
- "Before my ADHD diagnosis, I thought my high libido made me distractible, always thinking about sex with every woman in visual range. I love my wife and want to be satisfied with her alone. When I tried a stimulant, my libido dropped to what seems a more average range. So, it seems that my ADHD was making me distracted with sexual thoughts, not vice versa. The thing is, I got the diagnosis due to forgetting, not listening well, and so forth. So this sexual improvement is a huge bonus."

PARTNERS

- "That parent–child pattern is a real impediment to intimacy. After years of trying to save her from herself, I'm too exhausted to do it anymore. But that smacks of withdrawal to her and doesn't help intimacy, either."
- "Like the joke, we have married doggy-style sex: I sit up and beg, he rolls over and plays dead. He sure was interested before we got married! Most of the time now I just work on getting it over as fast as possible because I feel so bad for putting him through it. Then I get resentful."
- "I have noticed a huge difference in overall energy and motivation since my wife started the stimulant. She used to nap constantly and slept 12 hours straight. When she got home from work, she'd plant her butt in front of the TV until bed. Now, she works in the yard and does yoga. Oh, did I mention we're having more sex?"
- "The only time I got close to talking about sex in therapy, my wife screamed at me the whole way

home for 'embarrassing' her. I wish our therapist had asked about it so I didn't have to bring it up. The truth is, she cannot even stay focused for foreplay. It's easier for her to masturbate."

- "He has some 'oppositional' traits, and I can't help wondering if a loving sexual union feels to him like surrender."
- "My man hyperfocused on me for the first 6 months. But he said he feared getting involved because he is always gung-ho at first. Once the newness fades, his mind goes in a million directions. At first, our romantic times lasted hours. But you can't do that and make a living. It was the extremes that really hurt: the burning hot, then the ice cold."

Driving

The Research

ADHD-related driving risks are well documented (see Chapters 11 and 31), as are the positive effects of medication on driving performance (for a review, see Barkley & Cox, 2007). Challenges include vigilance and reaction time, operational skills, angry reactions, and poor strategic application of driving knowledge. Imagine being in the passenger seat, perhaps with a child in the back. One study indicated that drivers with ADHD are more likely to report driving anger and aggressive expression through the use of their vehicle than non-ADHD peers (Richards, Deffenbacher, Rosén, Barkley, & Rodricks, 2006). Barkley and colleagues (2008) consider it crucial for clinicians to recognize the increased driving risks and the dangers posed by the adult with ADHD to passengers in their vehicle or other drivers; such risky behaviors likely respond to medication, *if* it is in effect during drive times.

The Personal Experience

ADULT WITH ADHD

"When I'm driving, I'm truly not paying attention to where I'm going. I don't notice landmarks or street signs. For the places I visit often, I can get from home to there, or office to there. But add a stop in-between? Forget it. Plus, it's no good to ask for verbal directions—in one ear and out the other. My GPS navigator is a lifesaver. Of course, it drives my wife nuts when I start hyperfocusing on the 'shiny' GPS screen and forget I'm driving. Oh, and God help me if I lose satellite transmission; I must keep driving until I find it again."

PARTNERS

- "My partner is generally a good driver. He had a serious car accident before we met, plus a few bad motorcycle spills—one that caused a broken clavicle. Those accidents seemed to have made an impression. My biggest issue is deciding whether to speak up when he isn't paying attention and misses a turn or starts out going in the wrong direction. When we first started dating, I spoke up: 'Hey, turn here.' He reacted defensively and resentfully."
- "I have known my husband to stop at a junction, look one way, and then go off into a kind of dream for several seconds, then look the other way and pull out in front of something coming from the other direction. Then he swears the driver came up very fast and refuses to believe what I saw him do."
- "When we were dating, my husband denied being a bad driver. He called me 'overly sensitive,' but past boyfriends' driving never bothered me. Still, you need to consider the other person's point of view, right? And he was so sure of himself! Then another couple drove with us once. A few minutes in, the guy shouted, 'Whoa there, Mario Andretti!' My husband was insulted, but I was validated and felt like a knucklehead that I'd let his overconfidence brainwash me. Things got better only after he started medication. In the meantime, it created such confusion in my mind, that someone who was kind in other ways would be so reckless with my physical well-being."

Money

The Research

Adult ADHD can affect both income and outgo, jeopardizing a couple's or family's financial stability. Higher education typically means greater income and opportunity for full-time employment, and adults with ADHD are less likely than non-ADHD control groups to have finished high school or to pursue further education (Barkley et al., 2008). Even ADHD subjects matched for educational levels, however, earn less than those who do not have ADHD; household incomes are about $10,791 lower for those with ADHD who are high school graduates and $4,334 lower for college graduates

(Biederman & Faraone, 2006). Additionally, only 34% of subjects with ADHD had full-time employment, versus 59% of controls. There are also higher medical costs associated with untreated ADHD, including higher rates of accident and illness (see Chapters 11 and 12).

When it comes to spending and saving patterns, both prospective, longitudinal follow-up studies and cross-sectional clinic studies (Barkley et al., 2008) find that compared to adults without ADHD and adults with other psychiatric conditions, adults with ADHD:

- Made more impulse purchases
- Had higher credit-card balance; were more likely to exceed credit limits; and had lower credit ratings
- Paid bills late or not at all; bounced checks more often
- Were more likely to have no savings
- Often failed to save receipts for income tax returns

Of course, many people with ADHD do well in both earning and managing money. In fact, 22% of ADHD Partner Survey respondents credit their ADHD mates' actions for a "comfortable lifestyle and money in the bank." Yet others report debt from compulsive or impulsive spending (55%), bad credit rating (39%), and secret credit cards and debts (27%)—and report feeling powerless to stop the river of red ink.

The Personal Experience

ADULTS WITH ADHD

- "With the help of a professional organizer, I've been clearing clutter. I avoided it for so long because it 'hurt' to see so much money squandered on items never used—crafts materials, self-help books, and even gizmos to help my ADHD. Disposing of the items felt like throwing money away, and we have none to spare. But the clutter was so extreme we stopped inviting friends over, and that wasn't fair to my partner."
- "Money is an abstraction. It means nothing to me. I like things alright. But money? That's a very boring topic."

PARTNERS

- "They say there are two kinds of time for folks with ADHD: now and not now. With my wife there are two kinds of money: none and endless. The amount is irrelevant and in her mind very elastic. Since I was laid off, we've been experiencing 'none' and she has taken the new austerity measures as a stimulating personal challenge. With my new job, I'm worried that she will shift into 'endless.' Missing between these extremes: the gray area of prioritizing, strategizing, and managing her emotions around money."
- "I worry. We're in our 40s with no retirement savings. My husband tries to make himself happy by buying things, never even using what he buys. I can't talk to him about it, though. He shuts me down and accuses me of acting like his mother."

Sleep

The Research

"Generally, the parents of hyperkinetic children are so desperate over the night problems that the daytime ones pale in significance," reads an early description of ADHD-related sleep disturbances (Laufer & Denhoff, 1957, p. 463). In fact, "restless sleep" was originally part of the 1980s-era diagnostic criteria for ADHD, but it was dropped due to lack of empirical evidence (American Psychiatric Association, 1980). Recent research, however, has refocused on the likely bidirectional adverse effect between ADHD symptoms and sleep. From 25 to 50% of children and more than 50% of adults with ADHD reportedly experience sleep concerns (Chapter 11; Yoon, Jain, & Shapiro, 2012). A review on ADHD and sleep focusing primarily on children (Konofal, Lecendreux, & Cortese, 2010) cites factors such as restless legs syndrome (RLS), periodic limb movements in sleep (PLMS), sleep-onset insomnia and dim light melatonin onset delay, and sleep-disordered breathing (e.g., sleep apnea).

The topic sparks energetic discussion among adults with ADHD and their partners, in face-to-face groups and online (Pera, 2009a). "But we went to a sleep clinic and the doctor never mentioned ADHD!" goes an often-heard complaint. Certainly, disordered sleep not only exacerbates daytime symptoms but also causes conflict when couples are never in bed together (thus impeding physical intimacy) or when the adults with ADHD cannot be roused from the "Sleep of the Dead" to get to work on time.

The Personal Experience

ADULTS WITH ADHD

- "ADHD affects sleep? Wow. Who knew? I will tell myself this is the last computer game, only to find myself clicking 'play new game' and then 'just one more'! It's too boring to go to bed; I'll just lie there while my thoughts play pinball. The sun comes up and my work isn't done because I didn't know where to start and decided to play 'a' game to clear my head. My parents tell me I fought sleep since I was a baby. My husband insists I'm using ADHD as an excuse to be irresponsible."
- "Since I've been on a stimulant my sleep problems have disappeared. The chronic shoulder pain that I couldn't sleep with or ease without Valium is gone. My wife likes that I'm in bed at bedtime and no longer rely on her to make me get up in the morning. When she failed to rouse me, I accused her of not loving me enough to try harder."

PARTNERS

- "For 15 years, I've wondered why my wife resists going to bed at a 'normal' hour, always insisting 'I'll be right in' but getting caught up in some TV show. I never suspected it was related to her ADHD. I assumed that she was avoiding sex with me."
- "My husband would be sleeping and just start kicking me—his legs moving a mile a minute. If I weren't a big girl, he would have launched me across the room. We just bought twin beds and formed a 'split king' to protect me from the kicking. Getting him to talk to a doctor about this? Not so easy."
- "My husband denies having ADHD the same way he denies having sleep apnea, despite his doctor telling him he had to use CPAP [continuous positive airway pressure] or risk cardiovascular trouble. Sometimes it sounds like he's choking to death, but he won't believe me. Finally, having separate bedrooms was my only option. He acts the martyr but I had to get sleep or get sick."

Health

The Research

ADHD has been linked to substance use and adverse health outcomes such as asthma, injuries, cardiovascular disease, various chronic diseases, obesity, and negative self-perceptions of health (Chapter 11; Brook, Brook, Zhang, Seltzer, & Finch, 2013; Fasmer, Halmøy, Eagan, Oedegaard, & Haavik, 2011; Fuemmeler, Østbye, Yang, McClernon, & Kollins, 2010; Hodgkins, Montejano, Sasané, & Huse, 2011; Semeijn et al., 2013; Wilens & Upadhyaya, 2007). For many adults with ADHD, their disregard for the future consequences of current actions translates into insufficient concern for acting in health-conscious ways, such as getting exercise, eating prudently, and moderating substance use (Barkley et al., 2008). Many partners' anecdotal reports confirm research findings; they cite the following behaviors as evidence that their ADHD partners disregard future consequences not only for themselves but also for the couple and the family, not to mention setting poor examples for the children:

- Poor eating habits ("self-medicating" with highly gratifying sugary or fatty foods and caffeinated beverages; going without breakfast or forgetting to eat for many hours, then ravenously eating whatever's handy)
- Irregular or no exercise (or over-exercising sporadically or to the point of injury)
- Chronic allergies and headaches
- High cholesterol and cardiovascular disease (which complicates issues around stimulant medication when the ADHD diagnosis is finally made)
- Sensory issues that constrain diet choices (often to fried or crunchy foods rather than soft or "slippery")
- Poor dental health (from lack of regular brushing and refusal to see a dentist)
- Dehydration (failing to hydrate while working or playing outside and becoming weak and dizzy)

The Personal Experience

ADULTS WITH ADHD

- "When I'm hyperfocusing, I don't stop for anything, including eating or using the bathroom. I will delay both as long as possible, sometimes with disastrous consequences."
- "My appetite fluctuates with my moods, and then the stuff I eat creates mood swings. It's a self-perpetuating cycle. Sometimes I eat nothing. Sometimes I am insatiable—rummaging through the kitchen and snarfing down sugar or carbohydrates. It's not so much that I like it, it's that I need it."

- "ADHD can be a real health hazard! Since starting medication, I have been able to stick with a class at the gym for over a year. It has really improved my mood and my self-esteem. For 40 years before that, I truly did not have the 'willpower' to stick with anything health-related. If I started (rarely), I quickly grew bored and, frankly, just didn't care if I got fat and sluggish. I wasn't really connected to my body. It's hard to explain."

PARTNERS

- "My wife is on the go all the time, drinking caffeinated Mountain Dew all day. She claims she has no time to take care of herself, but she manages her time poorly. At 37, she's the heaviest she's ever been and suffers headaches and backaches. We have no sex life. I realize that the way she feels adds to her stress, and I dare not say a word about the effect on me."
- "It's hard not to fall into 'codependency' when your husband is slowly making himself an invalid or even killing himself by ignoring his diabetes. I worry about his health at the same time I'm angered that he's making himself a liability to me."
- "My husband was a walking demonstration for the ways ADHD can be dangerous to one's health. The need for constant stimulation and instant gratification is hard on a body. His poor planning always created stressful situations. He never took his high blood pressure or cholesterol seriously. He was 'oppositional' to medication of any kind. It took his heart arresting, a quadruple by-pass, a diagnosis of diabetes, and spinal stenosis to bring him to the point where he'd watch his diet, exercise, and take medication. He died too young, and his quality of life would have been so much better if he had taken care of himself. He realized that at the end. And that broke my heart."

CLINICAL INTERVENTIONS

Where then to begin and how to progress through therapy for these couples? Naturally, the answer is: It depends. As with all couples, these clients defy categorizations, representing as they do all walks of life, ages, income groups, educational levels, and ethnic backgrounds. They also bring variable ADHD-related issues along with variable capacities in empathy and intellect, both of which are amply required in order to comprehend a condition as complex as ADHD and to follow through on evidence-based treatments. Accordingly, no procedure exists for guiding each couple, only informed and flexible guidelines that can be adapted to suit. And there is no clinical research on effective methods for counseling couples in which one (or both) partner's ADHD is the foundational issue that seems to be driving most other dissatisfactions.

Factors Affecting Couples' Ability to Manage ADHD

One major component in the clinical decision-making process is the status of the ADHD partner's diagnosis and treatment, but there are many others. Psychologist Arthur Robin points to five factors that affect a couple's ability to manage conflict and sustain positive interactions when one or both partners have ADHD (see interview in Pera, 2008):

1. *Skills deficits.* Poor ability to problem-solve or manage behavior impedes conflict resolution, leading to more criticism, put-downs, and blaming.
2. *Cognitive distortions.* Unrecognized ADHD can lead to misattributions and distorted explanations and accusations on both sides. Distortions can also be unrelated to ADHD, such as unrealistic expectations of any relationship or strict ideas about gender roles.
3. *Degree of impairment.* Severity of ADHD deficits and the number of family members (including children) with ADHD influence functionality.
4. *Coexisting conditions.* Additional diagnoses are often present, which add more complications and require targeted treatment (see Chapter 13).
5. *Family-of-origin issues.* Everyone brings some degree of "family baggage" to a relationship. Given ADHD's high heritability, dysfunctional patterns can start early and become entrenched. For example, the child with unrecognized ADHD bears constant criticism from a hot-tempered parent for being disorganized or unruly. When that child becomes an adult, a partner's criticism for the same issues compounds old wounds and sparks defenses. Given ADHD's societal prevalence, some partners will have grown up with familial ADHD and perhaps developed patterns of overaccommodation.

What about those couples who score fairly well in all five factors, who are relatively well adjusted and whose relationship is a mutual source of strength and support? Indeed, such couples appear to be more common in recent years; greater ADHD awareness is resulting in earlier identification, before mutual misattributions and recriminations take root and fester. For these clients, finding immediate ways to involve the partner in the ADHD partner's individual treatment might be more appropriate than more traditional couple therapy adapted for ADHD.

It is worth emphasizing at this point: *Clinicians who train specifically to treat individuals with ADHD are urged be vigilant for any inclination to unconsciously align themselves with the ADHD client, causing the partner's needs to be overlooked or even pathologized out of context.* For example, the clinician may deem the partner as "controlling" without taking into account the ADHD partner's chronic lack of control and the historical need for someone to "put on the brakes." Another example is judging the partner as "negative" because he or she fails immediately to embrace all the promises of ADHD treatment—with the clinician failing to comprehend that the ADHD partner's longtime pattern of "overpromising and underdelivering" and constantly moving on to the Next Big Thing has trained the partner to be self-protective and cautious, if not outright skeptical.

Similarly, there might exist the temptation for the therapist to recruit the partners immediately as adjunct "helpers" in their ADHD partners' treatment and, if they do not spring into action with a cooperative attitude, view them as stubborn saboteurs to their ADHD partner's progress. It has been said of Fred Astaire's dancing partner: "Ginger Rogers did everything Fred Astaire did, except backwards and in high heels." And in a very real sense, some partners of adults with ADHD experience the fallout of ADHD as much as—and in some cases more than—their ADHD partners. After all, they are also living with the behaviors' direct adverse impact yet they are still expected to keep the unmanageable ship afloat and far from the waterfall's edge—while also safeguarding the children.

Clearly, the stress, alienation, neglect, and even trauma experienced by some partners mean they will need individual attention before various contributing factors can be teased apart, and certainly before they can think about summoning aid to their ADHD partners. Peer support is generally very helpful (e.g., *http://groups.yahoo.com/neo/groups/adhd_partner/info*). In some cases individual therapy and medication will be required to alleviate entrenched depression, anxiety, sleep deficits, and trauma.

A Model for Couples in Which One Partner Has a Psychiatric Diagnosis

Baucom, Whisman, and Paprocki (2012) present a flexible meta-model for helping couples in which one partner has a psychiatric condition. This model, like a road map, provides an important overall orientation for such couples. Because working with ADHD couples, however, can feel like navigating an urban cloverleaf at rush hour—after all, more than 50% of adults with ADHD have at least one comorbid disorder—more detailed guidance follows this explanation of Baucom's model.

1. *Psychoeducation.* To inform the couple about the condition and its treatment, including how it will progress and how to engage in the process.
2. *Communication and problem-solving training.* To help the couple share thoughts and feelings, and to improve decision-making skills and cooperative efforts, particularly in disorder-specific interventions.
3. *Three options for partner involvement.* From here, the model veers into one of three directions (or all three, eventually, depending on the couple); the first two assume little relationship discord:
   a. *Partner-assisted.* Rather than changing the relationship, this option focuses on the changes the patient needs to make and how the partner can help. For example, even with three young children and Alex's newly diagnosed ADHD, he and wife Jan enjoy a loving, fun rapport. His "flex-time" job, however, has left Alex vulnerable to losing all sense of time, working too late, and oversleeping the next morning, missing out on time with his children. While he works on individual ADHD treatment, Jan helps Alex to schedule his working hours.
   b. *Disorder-specific.* The emphasis here is on creating long-term changes in the relationship but only those specific to the patient's disorder. The couple may revise roles and responsibilities according to strengths to avoid stressing the patient's weak points. As the patient gains newfound skills and develops stronger habits, the therapist can gradually help the couple to redistribute roles and re-

sponsibilities. Consider Bill and Tricia, happily married 11 years. It was only when Bill's job changed that Tricia's long-ignored diagnosis of ADHD created a profound impact. With Bill focused on earning more income so they could buy a house, Tricia assumed responsibility for bill paying and investments—a typical *bête noire* for adults with ADHD—and by the third month, utilities were shut off for lack of payment. Bill agreed to take over financial duties, including setting up an easy-to-follow system, and gradually shift these to Tricia when she developed better time-management skills.

c. *Couple therapy.* It is difficult to implement teamwork, which is key to the first two types of interventions, when couples are disengaged or even hostile. Therapy to address partner interaction can at any time include one or all three types of partner involvement.

In this context, couple therapy acknowledges the bidirectional nature of psychopathology and relationship discord. That is, relationship distress can both cause psychiatric disorders and hamper their treatment. Adults with ADHD often report being depressed and anxious about the state of their relationships. By the same token, when years ago I asked one long-respected ADHD researcher about the impact of ADHD on the partner, she whispered to me emphatically, "Depression!" Before finding their way to an ADHD diagnosis, couples commonly will have seen therapists who miss one partner's ADHD and instead zero in on the other partner's anxiety and depression, identifying that as the core of the couple's troubles instead of its side effect (Pera, 2008). No doubt, some of the partners have longstanding, perhaps neurogenetic, issues with depression and anxiety. Yet studies indicate that relationship distress predates onset of psychiatric disorders among married adults who did not meet criteria for the disorder at baseline, including the major depressive episode (MDE; Whisman & Bruce, 1999) and alcohol abuse (Whisman, Uebelacker, & Bruce, 2006).

In the meantime, however, the adult ADHD client may be less likely to respond to individual treatments if relationship conflicts go unaddressed. Relationship discord is associated with poorer outcome for individual treatments for a variety of psychiatric conditions, including anxiety disorders (for a review, see Dewey & Hunsley, 1990) and depression (Denton et al., 2010; Whisman, 2001); posttreatment, poorer marital adjustment also predicts higher relapse rates.

Which Comes First: Individual or Couple Therapy—or Both?

No evidence-based guidelines exist for determining the sequencing of therapy for ADHD couples; assessment must be made on a case-by-case basis. Ramsay (in press) suggests that the therapist who has CBT individual and marital therapy skills can provide both individual and couple therapies; otherwise, the couple should be referred to separate qualified therapists.

It is worth remembering that by the time some long-term couples find their way to an ADHD specialist, each partner can be too mentally and physically exhausted, too mutually entrenched in knee-jerk, hypersensitive, survival-mode defenses, and too overwhelmed to learn and implement much in the way of supportive strategies toward each other. In these instances, individual therapy following joint psychoeducational sessions and, where indicated, medication titration, can be most helpful initially, with the established goal of couple therapy once both partners find their footing.

The idea is to declare a temporary truce, in some cases, with the partners living separately until hot emotions cool and distance is achieved. (It is also a sad fact that some ADHD partners will not take seriously a partner's pleas for change until he or she initiates a separation.) Even if the couple still cohabitates, separate sessions serve to avoid devolving into highly emotional finger pointing, giving both partners emotional space to gain validation, make sense of all they have gone through, and catch their breath. The ADHD adults can explore their reactions to the diagnosis, as well as their experiences within the relationship, and nurture optimism about change without being shut down by their partners quipping, "You think *that's* bad? What do you think *I* was living with?" or "Oh right, like *that's* going to happen. I've heard your big promises before." Likewise, the partner can vent resentments without enduring more of the ADHD partner's denial and minimization that has long fueled the anger, anxiety, and sense of isolation and impotence. To some degree, these potential difficulties can be anticipated and addressed ahead of time in conjoint sessions.

When the couple seems prepared to reengage, it is important that any conjoint therapy meet three requirements, according to Baucom and colleagues (2012):

1. The intervention should be empirically grounded, based on the types of changes recommended for an individual with a specific disorder.
2. It should consider how the relationship might be a stressor or resource for the individual with a specific disorder.
3. It should employ couple interventions that specifically assist in this change-process for the well-being of both individuals as well as their relationship.

With these factors in mind, the next section summarizes five principles of empirically supported generic couple therapy and presents special adaptations and clinical interventions as they might be applied in cases of adult ADHD. The authors who conducted the meta-analysis that identifies these five principles (Benson, McGinn, & Christensen, 2012), gleaned from 40 years of couple therapy research, emphasize that the principles are best viewed not so much sequentially as building upon each other over time. The five principles are as follows:

1. Changing views of the relationship
2. Modifying dysfunctional interactional behavior
3. Decreasing emotional avoidance
4. Improving communication
5. Promoting relationship strengths

The fundamental couple therapy principles and ADHD-focused guidelines that follow are not intended to be a complete guide to couple therapy; rather, they are designed to inform and augment therapeutic approaches to counseling ADHD-challenged couples until research on such therapy is done to further guide this enterprise.

FIVE PRINCIPLES OF COUPLE THERAPY

Changing Views of the Relationship (Psychoeducation)

Research Summary

The therapist guides the couple in viewing their relationship difficulties more objectively and within context. Couples learn to cease the "blame game" and to consider each other's perspectives, as well as how each partner's own behavior might augment relational conflict. This does *not* mean holding each partner equally responsible for their problems as a couple, especially when one partner's behaviors are clearly more highly problematic. It does mean, however, emphasizing that each partner's actions have an impact. For each partner to recognize this fact marks an important step in couple therapy.

Adapted for ADHD

For the individual, ADHD treatment begins with diagnosis. The way in which the clinician communicates the diagnosis is critical to patients' understanding of ADHD and their inclination to follow through with treatment. Patients who remain confused or uncertain about the diagnosis or its treatment strategies are less likely to feel optimistic about positive outcomes and are therefore less likely to pursue them (Murphy, 1995). The same can be said for couples. Providing psychoeducation about ADHD as a neurobiological condition—that is, explaining how clinical-sounding symptoms specifically translate into the couple's specific challenges—forms a critical step in helping couples to contextualize their troubles, reframe the past, refrain from blaming, and start implementing new strategies (Robin, in press-b). For example, instead of seeing the ADHD partner's erratic follow-through on agreements as solid proof of disregard or selfishness, the partner learns about how ADHD symptoms can interfere. It is extremely helpful for the therapist to explain ADHD in the framework of executive function (EF) (Pera & Robin, in press). Vague terms such as "hyperactivity" and "distractibility" prove to be limited in explaining everyday challenges. By contrast, the EF model provides a more precise structure for comprehending the complex manifestations and identifying treatment targets. In this way, it becomes clear to the couple that learning about the causes of ADHD-related patterns does not mean simply overlooking or rationalizing away problematic behaviors (Nadeau, 1995). The ADHD partner who shirks responsibility for making the most of treatment risks renewing power struggles when the frustrated partner again reacts to feeling powerless by ratcheting up the criticism and anger. Similarly, the partner who continues to misattribute the behaviors jeopardizes any forward momentum in couple therapy.

Detailing the evidence-based strategies for ADHD, including medication and the importance of structural supports along with strategic teamwork, can inspire optimism in trying new strategies. Even if the primary problem in the relationship has been one partner's un-

addressed ADHD challenges, the other partner learns that his or her responses can engender the ADHD partner's trust—or alienation. In this way, the couple moves from the ADHD partner "being a problem" to the couple "having a problem" and, with the therapist's help, identifying ways to address problematic issues.

GUIDING REACTIONS TO THE DIAGNOSIS

Late-diagnosis adults and even many adults diagnosed in childhood tend to have feelings of low self-esteem, hopelessness, and underachievement. Their partners' prediagnosis experiences often run along parallel lines, harboring as they do feelings of personal failure and powerlessness in being able to turn the tide of dysfunction. Against this backdrop, the ADHD diagnosis can bring massive relief to both partners: Finally, there is an explanation for their previously inexplicable challenges. Of course some individuals meet the diagnosis with resistance, and some couples have mixed reactions, with one individual embracing and the other resisting. Based on clinical experience, Murphy (1995; see also Chapter 31) details the six stages typically experienced by newly diagnosed adults with ADHD, suggesting that these stages can be used in the psychoeducation process to guide clients through this adjustment and normalize the need for occasionally revisiting previous steps:

1. *Relief and optimism.* Finally, there is a name—and solutions—for the "invisible force" that has been creating so many problems.
2. *Denial.* There is no magic bullet, no cure. Second thoughts about the diagnosis or even the validity of ADHD are entertained. In a sense, these lingering doubts can protect individuals from feeling overwhelmed, as long as these feelings are transitory.
3. *Anger and resentment.* Why was ADHD missed for so many years? What were the parents thinking of? What about the teachers? What good was all that previous therapy if it did not identify the core challenge, ADHD—instead seeing only depression or anxiety—and why should they once again trust the "head shrinkers"?
4. *Grief and sadness.* What about those lost years? What about the missed opportunities and damaged relationships? Suddenly, the familiar targets of blame (e.g., dysfunctional family, critical partner, incompetent bosses, society at large, and, too often, their own "poor character") must be reassessed and redirected to . . . ADHD? What does that even mean? How can the future possibly be different from the past? What if it's too late? This is a "life sentence"!
5. *Mobilization.* With negative emotions felt and validated, the original optimism is free to develop deeper roots, this time with intensified motivation to pursue treatment and learn new coping strategies and skills.
6. *Accommodation and acceptance.* Accepting that ADHD can pose certain limitations is balanced by a clearer understanding of the individual's strengths and weaknesses and what it takes to successfully move forward.

A small qualitative study found support for this model, noting that it provides an important tool at the point of diagnosis, especially when the clinician teaches skills to anticipate future setbacks and implement appropriate coping strategies (Young, Bramham, Gray, & Rose, 2008). A follow-up study that similarly explored the experience of partners of adults diagnosed with ADHD (Young, Gray, & Bramham, 2009) described emotional reactions similar to Murphy's (1995) earlier model, including these in particular:

1. *Confusion.* How does one support the ADHD partner? Is he or she equipped to deal emotionally deal with this situation? Does he or she want to continue a relationship with someone who has a disorder?
2. *Relief.* At last, there is a framework for understanding the difficulties; there is optimism that a "solution" is possible; medication is begun and improvements seen.
3. *Acceptance.* More tolerance is shown toward behaviors that are clearly ADHD-related, with fewer misattributions.
4. *Judging limitations of medication.* The initial perceptions of medication as a solution quickly fade as it becomes clear that deeply entrenched problems and mind-sets remain. Soon the partners wonder how additional treatment might be helpful.

Regarding the second point: Some partners' relief can come in the form of anger if they have been unduly blamed for relationship woes. They are shocked to learn they unwittingly accepted primary responsibility for solving problems that were not theirs to solve,

resulting in self-blame and lowered self-confidence. At long last to have the tables turned in this way is akin to lighting a powder keg. Clinicians should expect some explosions and realize that the partners can first require an adjustment period, support, and perhaps individualized treatment before progressing to more positive reactions:

> "It's so sad it took us so long to discover ADHD. And still, so many men and women in relationships are out there tormenting themselves as I did with feelings of guilt and asking themselves, 'What else can I do?' I just want other people in my situation to know that it is not their 'fault' that their relationship is challenged; in fact, it is no one's 'fault' . . . it is just a question of raising awareness about ADHD and opening the eyes to reality."

Regarding the fourth point: As important as it can be to instill hope during the psychoeducation process (and it is *extremely* important), it is equally important to manage expectations about which problems are more likely to respond to medication (and how soon this might happen), and which require other interventions. It is helpful to caution couples that finding the most effective medication regimen takes patience and methodical effort and that they should view even adverse side effects as important data in refining the medication selection, timing, and dosage (see interview with psychiatrist Margaret Weiss in Pera, 2008). Researchers in one study found that adult patients with ADHD who received drug treatment for more than 2 years had fewer symptoms and less psychological distress compared to those treated for 2 years or less (Lensing, Zeiner, Sandvik, & Opjordsmoen, 2013).

ENCOURAGING TEAMWORK

There is a strong cultural message that goes: "Adults need to take care of themselves." This makes it a tough sell to convince certain couples that they need to work as a team on ADHD-focused strategies, especially in the beginning. Some partners balk at the seeming "codependence" of helping to monitor the ADHD partner's medication efficacy, at sitting down together each night to review the next day's calendar of activities, or at helping to break down into doable chunks an ADHD partner's household projects. Nonetheless, these pragmatic, logistics-based activities can form the foundation of effective couple therapy for ADHD (Pera & Robin, in press). The therapist can coach the reluctant partner into seeing the benefits of providing more short-term help in exchange for greater long-term functionality.

The two previously discussed studies from Young and colleagues (2008, 2009) also offered interesting nuggets of information that underscore the importance of teamwork throughout treatment. Notably, the adults with ADHD reported that, prior to medication, they had little hope and direction for the future, feeling that it would simply repeat the failings of the past. After trying medication, however, they reported a positive effect. The partners, however, identified an even wider range of improvements with medication than those self-reported. As experienced ADHD clinicians know, sometimes the initial changes with medication are subtle and not always noticed by the adult with ADHD; loved ones often possess a more accurate perspective of functional improvements, especially with respect to those affecting interpersonal relationships.

REFRAMING THE PAST AND MODIFYING COGNITIONS

As mentioned earlier in this chapter, late-diagnosis adults with ADHD and their partners have typically developed, prior to diagnosis and psychoeducation, alternative ways of making sense of their differences. These clashing "belief systems" are often negative, leading to cognitive distortions and poor compensatory strategies that, together with ADHD symptoms themselves, can keep individuals and couples stuck in dysfunctional patterns (Pera, 2008; Pera & Robin, in press; Ramsay, in press; Ramsay & Rostain, 2007). Psychoeducation can help to correct misattributions. The more entrenched patterns around belief systems and cognitive distortions provide good targets for fostering behavior change: Slowing down the process—from event to feeling to thought to action—and examining what led to poor compensatory strategies lies at the heart of individual cognitive-behavioral therapy (CBT) for ADHD.

EMPHASIZING OUTSIDE SUPPORT

Both partners can feel extremely isolated. Friends do not understand their problems ("Why do you put up with this crazy stuff?") or are misled by the disparities between public and private personae ("But Suzy is so much fun! Why are you such a grump?"). Family

members can throw a monkey wrench into the works with their own denial systems ("Well, our son certainly didn't have ADHD when he lived in *our* house!"). Even for less embattled couples, support groups for each partner can be especially powerful: Hearing from many other people experiences that are similar to their own (not just taking the word of a therapist or a book) and receiving individualized validation can be immensely affirming, stress-relieving, and hope-inspiring.It is important, though, that the support group moderator be knowledgeable about ADHD, lest neophytes be scared away by misinformation or unrelenting "Woe is me" negativity. Waves of pain-filled litanies, unbalanced by the sharing of progress or actual teaching of strategies, can douse burgeoning hopes. One woman describes her husband's experience in attending adult ADHD support groups:

> "He went the first time and hated it so much he went into a panic, shut down, couldn't even talk about it, and week after week, he made up one excuse after another to avoid attending. After a few months, I made a huge deal of it, and he agreed to try going again.
>
> "The first times he went back to the group, he would lie in bed afterward doing nothing for the rest of the day. It turns out that what he hates about it is that everyone else in his group has ADHD. Yes. Really. He says they are very annoying to be around. They don't listen. They interrupt! They talk too loud. They think their own concerns are all that matter. They lose the thread of discussions and change the subject. But once he got past those problems, he began to find it very useful. It helped that the group invited a psychologist with a specialty in ADHD to attend for several weeks as a guest speaker to focus on practical matters, and he loved that. He now comes home from group very excited and full of insights, realizations, and often good will toward me. He tells me all about it. He practices what they learn. It's amazing."

CHANGING MISATTRIBUTIONS AND MIND-SETS

To move forward, the couple must come to grips with the presence of ADHD in the relationship. For the ADHD partner, this means acknowledging how ADHD has affected the relationship and accepting responsibility for addressing challenges. For the other partner, this means acknowledging the neurobiological basis of certain behaviors instead of attributing them to lack of love or caring, or to a moral failing. For the couple, it is important to cultivate empathy, compassion, and forgiveness for each other and, at least in the beginning, to nurture a "two steps forward, one step back" expectation about progress. Typically, long before ADHD was diagnosed, couples enjoyed fleeting improvements only to devolve to baseline within weeks as the novelty dulled; with each bit of new progress, they typically still "wait for the other shoe to drop," easily spiraling down into pessimism when forward momentum reverses, such as with intolerable side effects from the first medication or the fading of the ADHD partner's enthusiasm over time. As with most psychotherapy, preparing clients for slipups and backtracking can help inoculate clients against losing hope entirely and help them stay focused on their goals.

Modifying Dysfunctional Interactional Behavior

Research Summary

This principle emphasizes careful assessment to determine whether either partner is at risk because of the other's behaviors in terms of suffering physical, psychological, emotional, or financial harm. Some individual problems, such as substance abuse, or other disruptive behaviors, such as constantly interrupting during therapy sessions, can be destructive to the point of interfering with treatment and so require immediate attention. Relationship distress is associated with emotional dysregulation (Snyder, Simpson, & Hughes, 2006), especially when sensitivities are heightened around rejection, unpredictability, and invalidating behaviors. In some cases, it is necessary to separate the partners and speak to each individually or to allow each partner to speak only to the therapist and not to each other.

Safety must always be a paramount concern. Despite published guidelines for assessment of domestic violence, one study indicated that fewer than 4% of couple therapists consistently follow these guidelines (Schacht, Dimidjian, George, & Berns, 2009). This second principle does not address all negative behaviors, only those that involve severe harm to the individuals or that interfere with therapy. The third and fourth principles address less extreme negative behaviors, such as patterns of avoidance.

Adapted for ADHD

Ramsay and Rostain (2007) cite as the first concerns in adult ADHD treatment those issues of patient safety and others that immediately affect well-being. The clinician is well justified in asking about well-known harmful behaviors relative to finances, sexual activity, addictive patterns around substances and electronic usage (e.g., video games), and coparenting, and in paying close attention to signs of child neglect or maltreatment (Easterlin, in press). The astute clinician probes the topics sensitively and, remembering that adults with ADHD can possess low insight into their behaviors, often queries partners separately. Arguments or revelations begun in session can erupt in violence afterward (O'Leary, 2008), particularly in those areas in which the ADHD partner remains in denial. The type and severity of these dysfunctional, interfering behaviors comprise one factor in assessing the need for individual treatment before couple therapy is attempted or perhaps referral to outside specialists. Two behaviors in particular are worth addressing in some depth: denial of symptoms or denial of ADHD as a valid condition and intimate partner violence (IPV).

DEALING WITH DENIAL

It is not uncommon for adults with ADHD to deny or minimize the presence or impact of ADHD symptoms on themselves and others, even after a thorough psychoeducation process has taken place. What might be enthusiastically grasped in the moment can be absolutely forgotten the next day. Obviously, the backtracking can create anxiety in the partner ("Just when I thought life was getting better!"), cause a loss of faith in treatment, and exacerbate conflicts. No one takes kindly to being told that his or her reality is not real, however, so this issue requires a thoughtful approach.

So-called "denial" can have roots in not only the *psychological* realm, such as around stigma and fear of the unknown, but also the *physiological* (anosognosia, when symptoms themselves can limit insight and objectivity). Refusing to participate in evaluations or treatment—or merely giving them lip service—usually stems from denial of either type. Partners of adults with ADHD widely express a strong desire for therapists who can help to "reach through" their ADHD partners' denial systems. To be sure, adults with ADHD also report that their partners can be "in denial" of ADHD's realities, with outright refusal to accept it as anything but an "excuse" for bad behavior or the latest fad, sometimes being more against medication or "labeling" than are their ADHD partners. Then again, some remain in denial of the nature of their ADHD partner's denial, lamenting, "He knows he has ADHD but he's still disorganized"—as if the diagnosis itself were a cure.

Before a couple can benefit fully from treatment, both partners need to accept the basis and need for it. If psychoeducation fails to budge the resistance and the couple continues to "lock horns," it is wise to consider an entirely different tack. For example, clinicians can consider eschewing the label of ADHD and instead focus on problem solving, for example, with a method called listen–empathize–agree–partner (LEAP), briefly adapted for ADHD by anosognosia researcher and clinician Xavier Amador (see Pera, 2008). Amador maintains that one does not win on the strength of an argument but on the strength of the relationship. This method emphasizes conveying respect and withholding judgment of the other person's point of view (although not necessarily agreeing with it) instead of trying to convince him or her of being wrong. Once genuine understanding and empathy are expressed, tension and defensiveness tend to decrease, leaving room for partnering to find common ground for problem solving. Another technique is motivational interviewing (MI), which can be employed to identify and strengthen motivations for change. Because confronting denial and resistance often strengthens it, the MI technique of "rolling with the client's resistance" is often highly effective. (For an overview of MI, see *www.motivationalinterviewing.org.*)

UNDERSTANDING FACTORS IN IPV

Is it any great surprise that neurocognitive challenges in managing frustration and negotiating compromise (see Chapter 3), not to mention a vulnerability to substance abuse (see Chapters 11 and 13), can lead some adults with ADHD to lash out verbally or physically (see Chapter 12)? It is equally predictable that partners who are feeling besieged might counterreact in similar ways. Clinicians should keep in mind the risk of IPV in these couples and inquire about incidents of verbal or physical violence from and toward either partner.

Indeed, studies point to elevated impulsivity and poor anger control, as well as ADHD explicitly, as increasing the risk of IPV, especially male-to-female IPV (González, Kallis, & Coid, 2013; Schumacher, Feldbau-Kohn, Smith Slep, & Heyman, 2001; Stuart &

Holtzworth-Munroe, 2005; Wymbs et al., 2012). ADHD accompanied by conduct disorder (CD), antisocial behavior, and substance abuse problems in childhood and adolescence predict aggression toward a partner for both young men and young women (Meichenbaum, 2007). Yet even controlling for CD, preliminary research suggests an association between ADHD and IPV. Wymbs and colleagues (2012) report the elevated risk of IPV among young adults with ADHD as far exceeding controls (10–14% vs. 2–3%); these results might actually *underestimate* the problem, given their reliance on self-reports. Males with childhood ADHD, especially those with conduct problems persisting from childhood, were more likely to be verbally aggressive and violent with romantic partners than males without histories of ADHD or conduct problems. Another research team found that while IPV was highest for ADHD with CD, symptoms of inattention contributed to IPV *without injury*, whereas symptoms of hyperactivity and impulsivity significantly predicted IPV resulting *in injury* (Fang, Massetti, Ouyang, Grosse, & Mercy, 2010).

As discussed in Chapter 12, in a more recent study in England (using 7,369 households), González and colleagues (2013) obtained ratings of adult ADHD symptoms and reports of violence. Adult ADHD was moderately associated with violence (odd ratio = 1.75) after they adjusted for demographic factors and known clinical predictors of violence. It was principally the hyperactive–impulsive dimension of the disorder and not inattention that was linked to such violence, which was primarily with intimate partners. Mild to moderate levels of ADHD were linked to such IPV, whereas severe ADHD symptoms were linked to violence primarily through comorbidity (antisocial personality, substance abuse, etc.).

From the partners' perspective, the ADHD Partner Survey asked several questions about IPV that can be summarized as follows:

- The majority of respondents (66%) reported their ADHD partner had threatened them physically and/or been verbally abusive.
- Roughly one-third reported being physically threatened ("hit, pushed, or shoved by your partner in anger or irritation") and another one-third were verbally abused. One in four said "both."

In a separate question, respondents were asked to review the 15 characteristic behaviors from *The Verbally Abusive Relationship* (Evans, 2009) and indicate whether they had experienced each behavior (e.g., *monthly*, *daily*, *weekly*, or *never*). This list was chosen for two reasons: It closely (but unintentionally) parallels a list of ADHD-related neurobehaviors and poor coping strategies (e.g., forgetting, trivializing, blaming, and denial), and it is a consumer book that partners of adults with ADHD often turn to in seeking guidance and explanations for their experiences. This begs the question: If untreated ADHD could explain much of perceived "abusive" behavior (which is typically considered willful, volitional behavior), could psychoeducation and treatment make a positive difference in reframing and/or mitigating behaviors considered abusive? In fact, learning about the ADHD connection to these behaviors typically comes as a revelation to both partners. This is key because each time the clinician can cut through the emotional chaos and focus issues through a more neutral, practical lens, it paves the way to more clear-headed, pragmatic problem solving. Some results follow:

- Figure 34.3 combines those respondents who, for each category, said the behavior happened on a "daily" and "weekly" basis.
- Overall, 78% of respondents said the ADHD partner displayed these behaviors in sufficient strength or frequency to contribute significantly to dissatisfaction in the partnership.

Perhaps most notably, prior to learning about the connection between ADHD symptoms and these behaviors, 74% of respondents felt that their partner was abusive toward them. Yet after learning of the ADHD connection, the figure dropped by half, with only 37% agreeing: "The behavior feels abusive, no matter what causes it," and 33% with mixed feelings: "Yes and no. Perhaps some of the behaviors were part of an untreated disorder, but many seemed very consciously done. And, those felt abusive." Said one respondent: "It's a tough situation for both parties when action and intention don't line up."

Of the respondents whose ADHD partners had started taking medication, 14% reported that the behaviors "improved a great deal," and 47% said they "improved somewhat." (Anecdotally, these improvements tend to expand and strengthen over time with continued treatment.) One woman with ADHD, age 36, and married for 1 year, told about her slow acceptance in realizing she had to change her often-combative interpersonal pattern:

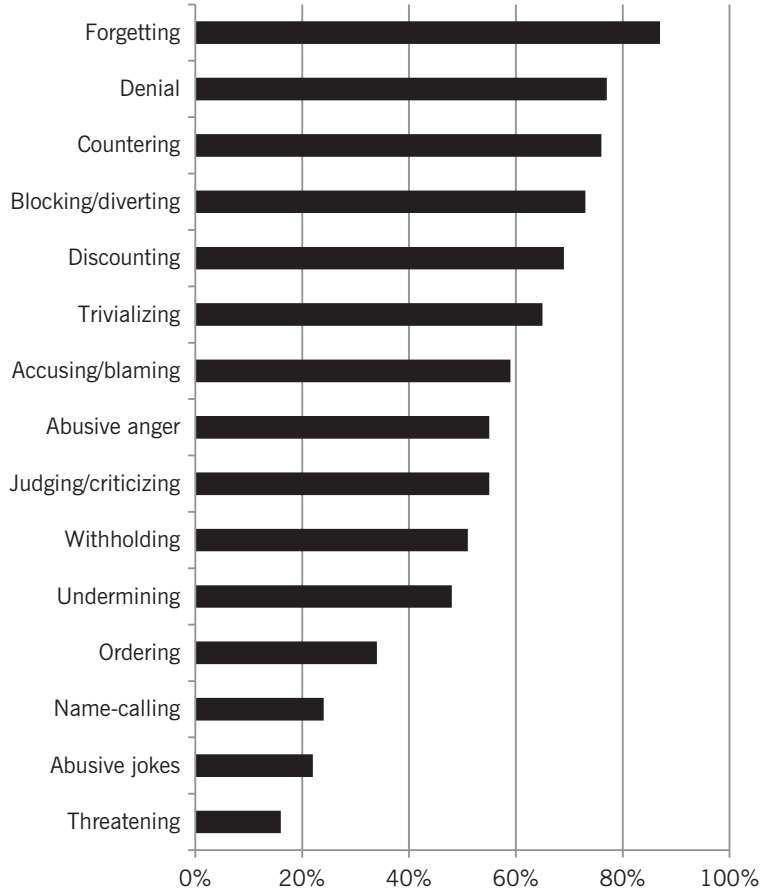

FIGURE 34.3. ADHD and verbally abusive behaviors. Survey respondents selected behaviors that their partner displayed toward them daily or weekly. From ADHD Partner Survey. Copyright by Gina Pera. Reprinted by permission.

"An important part of my learning new skills was being given extremely clear guidelines about what was not OK. First, though, our therapist helped me to understand that my behavior was leading in a very narcissistic direction if I did not change. What she described reminded me of my mother, who likely has ADHD; she doesn't respect the boundaries of people around her and is very adept with blame shifting. It does not feel good; in fact, it feels terrible, to realize that you are behaving in a way that veers toward, or is, abusive. I wasn't a bad person and didn't intend to be 'abusive,' so I think it's important to separate the behavior from the person. But I was making poor choices. I was capable of doing better; I just didn't know it yet, and I did need guidance and practice."

Explored only informally are instances of IPV *toward* the ADHD partner. (Of course, it could be argued that labeling someone an "abuser" for exhibiting unrecognized ADHD neurobehaviors is itself a type of verbal abuse.) In lectures, various clinicians have reported that the ADHD partners also suffer from their partners' abusive behaviors, such as being yelled at or demeaned, including publicly. No doubt this is a serious issue, especially because an ADHD partner, once provoked, may be more likely to contribute to escalation. Yet it is

impossible to know whether, as with the ADHD partners, these behaviors are personality-driven or poor coping strategies from living with a mate's frustrating behaviors. In fact, a contributing factor can be some ADHD partners' habit of seeking stimulation or eliminating boredom by provoking argument (Pera, 2008). Knowing that people tend to feel shame about extremely losing their temper, especially those for whom this is not characteristic, I regularly question the partners along the lines of "Have you ever gotten so frustrated that you've thrown or broken something?" While not condoning temper displays, I air this possibility as a not uncommon experience because it tends to reduce reticence to talk about it and it validates how difficult it can be to cope with an ADHD partner's behaviors.

In truth, partners who tend to respond with anger also seem more likely to persevere in seeking answers, including discovering the possibility of ADHD; they know something is very wrong and unacceptable. They provide clear contrast to those partners who internalize the hurt or blame and sometimes resort to addictive substances or "walking on eggshells." The energy behind the anger, of course, is best channeled toward more productive ends.

Decreasing Emotional Avoidance

Research Summary

When troubled couples fear expressing their private feelings to each other, they risk further emotional distance, diminished ability to solve problems, and reinforcement of dysfunctional patterns, including *mutual avoidance* of topics that both partners fear broaching, or a *demand–withdraw* pattern, wherein one partner meets the other's requests for change with withdrawal or avoidance; this phenomenon tends to intensify with each iteration (Christensen, 1988). To address dysfunctional interaction patterns and related cognitions, interventions focus on bringing the partners closer together by eliciting emotions, thoughts, and vulnerabilities that each partner fears expressing to the other and by encouraging appropriate partner responsiveness to those expressions.

Adapted to ADHD

"Private feelings" aplenty can run amok amid the dizzying confusion when ADHD has long gone unrecognized or unaddressed. For example, take one couple's long-simmering but unexpressed feelings around their sexual intimacy. First consider the partner's private fears: "Is my wife not coming to bed because she no longer finds me sexually attractive? She is on the computer so much; I think she is having an online affair." Perhaps neither is true; his ADHD partner might simply become engrossed in ever-deepening layers of Wikipedia, too distracted to nurture any reflection at all about what she *or* her partner is feeling—privately, fearfully, or otherwise. Perhaps she truly is avoiding sex with her husband, but it could be due to her own fears about telling him that lovemaking is more irritating than satisfying (because she remains unaware that her own ADHD-related sensory issues are involved and solutions could exist).

Couples long stymied by behaviors that beg explanation have either experienced avoidance or they continue to beat their heads against the wall, with tearful pleas met by angry denials (or, worse, passive nonresponse), finger pointing and blame shifting, or recriminations and counterrecriminations. It follows then that the aforementioned *demand–withdraw* phenomenon perfectly encapsulates these troubles: *Demanders* turn up the volume of criticizing and blaming; *withdrawers* turn down the volume on receptivity or response, shutting down faster and further. Avoidance is a major coping strategy for late-diagnosed adults with ADHD (and, eventually, many of their partners) for good reason; much of it reflexively stems from a chronic inability to solve problems and sort out issues. Adaptively, it stops the escalation of conflicts, if not the insidious emotional damage. Eliciting thoughts, emotions, and vulnerabilities, as recommended in this principle, can be unwise if done prematurely, before other fundamental problem-solving measures are in place.

Fortunately, psychoeducation and medical treatment can go a long way toward normalizing transmission and reception of these messages. The capacities of the ADHD partner improve, and each partner learns a new paradigm through which to view previously inexplicable challenges. Thus empowered, some couples go on to solve problems on their own, even around long-running bones of contention. Communication strategies, such as with those discussed in the fourth principle, help to get couples talking again in the safe space of the clinical session. But once emotional avoidance has built up and ossified, some individuals can become so numb to their own feelings—and so pessimistic that their partners will listen and understand or follow

through with new behaviors—that they cannot fathom breaking the logjam.

It is difficult, even in these cases, to overemphasize the extent to which "emotional baggage" has its roots in unresolved practical issues or undeveloped logistical habits: the erratic sleep patterns, the inequitable chore sharing, the absence of schedules or reliable follow-through, the clutter, and the chronic disregard on all these points, and more. Even after the misattributions abate, these problems in daily living remain, inseparable from clients' psychological and even physical distress. Although it is often assumed that the logistical task training can be outsourced to ADHD coaches, it still requires a therapist's skill to work through the various obstacles to implementing practical strategies. According to psychologist Kathleen Nadeau, ADHD therapy requires a clinician who is "aware that ADHD is a neurobiological condition and uses concrete, practical methods to treat it"; psychological issues are addressed "but in an integrated fashion, moving back and forth between the practical and the emotional, between the present and the past, and between ADHD and related or coexisting disorders" (quoted in Pera, 2008, p. 230). As explained previously, ADHD Partner survey respondents who reported an improved sex life attributed it to improved cooperation in the rest of life. This is not the case for all, of course; myriad factors affect a couple's sexual intimacy.

The point is, in the triage process of ADHD couple therapy, focusing first on tangible, logistical improvements can help to dissolve mental walls and pessimistic expectations, opening minds and hearts to trusting each other again with emotions and feelings. Moreover, as the therapist works with couples in session actively to improve cooperative efforts, ample opportunities arise to observe how problematic cognitions are interfering, and to deal with them. For example, some couples need prompting to *plan* intimate times together. Initially, they might meet such a suggestion with an automatic reaction: "Physical intimacy should be spontaneous. If you have to plan it, it is not genuine." Ramsay (2015) advises offering an adaptive view: pointing out that we schedule appointments for activities not because they are unimportant to us but because they are *so* important, worthy of making sure we devote our time to it. Planning these times need not preclude additional spontaneity within the relationship.

In modified form, the CBT models developed for adult ADHD and the strategies designed to support EF (Barkley, 2011; Ramsay & Rostain, 2007; Solanto, 2011; Young & Bramham, 2012) can help decrease emotional avoidance on a wide range of issues by improving cooperation in domestic responsibilities, unlocking resentments that keep couples stuck, and encouraging positive, caring behaviors. Ramsay has developed a CBT model for couples, and Robin has developed a procedure for ADHD couple interventions (in Pera & Robin, 2015) that fosters behavior change and new-habit formation. Highlighted below are some key components of their approaches.

TARGETING GOALS IN BEHAVIORAL TERMS

Identifying achievable objectives to implement between sessions helps to create realistic, positive outcomes. Rather than expressing vague hopes around "spending less money," the clinician works with the couple to examine on paper the monthly outgo, noting where cutbacks can be implemented. Instead of asking the ADHD partner to "be a more engaged coparent," the goals are specified: "Read with the children at bedtime three times per week."

TAKE-HOME EXERCISES

Between-session assignments (or "homework") provide an important tool for creating momentum, structure, and accountability. Ideally, each session begins by reviewing the previous assignment. If the couple achieved the tasks, congratulations are in order; remembering to celebrate their successes is important. If the exercises were not completed or they caused conflict, the obstacles and cause of dissention can be explored with a focus on troubleshooting.

GUIDING PROBLEM SOLVING

Many issues faced by these couples defy specific, individual interventions. The clinician can greatly empower these couples by helping them develop problem-solving skills. Guiding couples in problem solving, including selecting goals and examining reasons for failure to do homework, demonstrates the process and gives the therapist a sense of each partner's stumbling blocks. For example, an ADHD partner fatalistically decrees in session that the problem in question is "impossible to solve," or the opposite: gets carried away with increasingly baroque options that often involve costly expen-

ditures on gadgets or outside services. Meanwhile, the partner who is accustomed to being the "efficient one who gets things done" resorts to eye rolling at the more improbable suggestions, causing the ADHD partner to withdraw. Throughout, the clinician redirects the focus away from unhelpful behavior and toward teamwork.

PAYING ATTENTION TO TIME, ORGANIZATION, AND COLLABORATION

Everyone knows the old joke about there being two kinds of time with ADHD: Now and Not Now. But for couples and families whose harmony depends on coordinating schedules and logistics, it is no joke. As Weiss, Hechtman, and Weiss (1999) point out, these couples affected by ADHD often share deep love, affection, generosity, and the ability to communicate well; what they lack is a focus on making time for it. EF deficits in time, prioritization, and organization can hit hard. Especially once the highly stimulating courtship days have ended—or the baby arrives or the job calls for more travel—the segments of free time couples enjoy together arise more from rare serendipity than regular commitment.

The clinician guides the couple in making their environments and agreements more "ADHD friendly." A large part of laying the foundation for behavior change involves helping the couple develop new collaborative skills around actively managing to-do lists and incorporating rewards for successfully completing objectives, such as praise, affectionate gestures, or enjoyable activities. Couples are instructed to set aside 10 minutes several times weekly to talk with each other. These "check-in" times establish a platform for following through on new behavioral objectives. Overall, the goal is to improve time awareness and decrease procrastination, which includes coordinating the use of calendars and reminder systems.

MODIFYING COGNITIONS TO IMPROVE INTERACTIONS

Building on the psychoeducational measures in the first principle to rectify cognitive distortions and misattributions, the therapist further guides the couple in learning to stop and question the misinterpretations that have clouded their interactions, helping them to reframe situations more accurately. For example, the biggest point of conflict for Janet and Lorraine is money. Janet interprets Lorraine's impulsive spending as proof that she does not love or care for her. For Lorraine, who has ADHD, Janet's criticisms feel like further proof that she is "always bad with money" or, depending on her mood, a victim of her overly controlling tightwad partner. With help, Janet comes to view Lorraine's spending patterns less emotionally and more neutrally, understanding that ADHD can fuel impulsive purchases with no thought of consequences. As Lorraine develops practical measures so they can live within their means, she comes to see how her spending helped push Janet into "tightwad" positions.

Once the couple establishes success in joint problem solving on these tangible objectives, they can build on their newfound sense of *esprit de corps* in addressing more emotionally loaded issues of discord.

Improving Communication

Research Summary

All empirically supported couple therapies specifically focus on improving communication. Compared to the second principle's more basic focus on addressing IPV or other destructive behaviors, this is a higher-order process. When intimate partners discuss difficult issues, they commonly fail to hear and respond positively to each other's perspective (see meta-analysis by Sevier, Eldridge, Jones, Doss, & Christensen, 2008). Therefore, they often need coaching in how to speak to each other in a more supportive, empathic way—typically beginning with techniques such as "active listening" and "mirroring"—and in learning to avoid communications patterns that tend to create conflict or cause the other person to feel dismissed rather than heard, especially when sharing vulnerabilities. Regarding the third principle's emotional self-disclosure, couples often need guidance in how to respond positively.

Adapted for ADHD

A husband reads an article to his wife about how many words women use each day—"30,000 to a man's 15,000!" The wife replies, "That's because we have to repeat everything to men." And the husband says, "What?"

It is easy for outside observers to dismiss poor communication in ADHD couples as "Mars–Venus" issues. But when it comes to ADHD, the same phenomenon happens in same-sex relationships of both genders. Beyond gender stereotypes, our culture seems conditioned

to accept that poor communication lies at the heart of most "couple troubles," with the obvious solution being training. Pioneering ADHD researcher and clinician Paul Wender (2001, p. 171) points out the difficulties with this approach:

> Communication is difficult. The ADHD person does not attend to the other's conversation. He may tune out and drift off following his own train of thought. Not having listened, he may interrupt his spouse in response to his own thoughts. So the spouse has not been heard and is receiving a reply to a question not asked. Communication breaks down. Many ADHD couples have been treated for communication problems when the communication problem was one symptom of the underlying ADHD problems and not the sole cause of the current ones.

Many partners join support groups seeking assistance in "communicating better" with their ADHD partners, failing to grasp (or being reluctant to admit) that "miscommunication" describes only the tip of the iceberg. In actuality, many ADHD partners fail to heed agreements not because their partners communicated poorly but because they were only halfway paying attention or forgot that an agreement was made—to the point of insisting that it never was discussed! Some partners have even resorted to purchasing earwax removal kits, to no avail. Beyond a certain point, they start looking into the "verbally abusive" behaviors described in the second principle. Yet, in some cases, desperate attempts to insist it is a communications issue conceal powerful defenses against acknowledging the possibility of a "brain disorder."

Now, it is absolutely true that most of us could stand to fine-tune our ability to listen and speak clearly and thoughtfully. One has only to ask directions of the average person on the street to see the broad spectrum of styles, from helpfully succinct to overburdened with superfluous details. Individuals bestowed with strong working memory often fail to realize that other people might have trouble keeping up with their barrage of facts and data, much less tracking the most salient points. Naturally, the partners of adults with ADHD seem to fall everywhere along the "clear communicator" spectrum. Nonetheless, it is critical not to take at face value the clients' interpretation of their "communications" problems and to clearly distinguish with them the difference between ADHD's direct effect on interpersonal exchanges and, for example, its effect on the ability to remember and abide by verbal agreements. The former can be addressed with training, as explained below, and often medication; the latter involves other logistical support, as covered in the third principle.

RECOGNIZING ADHD-RELATED CHALLENGES TO VERBAL EXCHANGES

The poor verbal interaction patterns associated with adult ADHD spring both from neurobiology and poor coping strategies. For example, inattention can result in hearing only "bits and pieces" of what is being said, with the imagination filling in the rest—yet being absolutely certain of accurate recall, no matter how erroneous it actually is. Impulsivity and impatience can mean speaking over people or completing their sentences. "I was expert in compartmentalizing and shifting focus," says one adult with ADHD. "For me, it was a necessary evil so I could avoid getting 'caught' slipping away." Some individuals might be firmly convinced that they have communicated important information, when really they have simply *intended* to do so.

Brain-based emotional dysregulation heightened by years of harsh judgment can lead to misinterpreting simple questions or comments as hurtful criticism; even a nonchalantly asked, "Did you already bring in today's mail?" can pour salt into the ADHD partner's festering self-loathing about chronically misplacing important letters and bills. Some late-diagnosis adults with ADHD have learned to cope with feeling hapless or understimulated by making everything a joke or making each minor conversational point a life-or-death "debate." They must always have the last word and will follow a mate who is tired of the argument around the house in order to have it. There can be a fine line between IPV, as described in the second principle, and ADHD-related neurobehaviors jamming the lines.

Complicating communications further is the frequently reported *tone* that is not there. Dana's husband and teen daughter, both of whom have ADHD, argue constantly about *tone*:

> "When asked nicely to do something, our daughter says, 'Stop yelling at me!' My husband insists that she 'yells' at him even when she is being polite. I always thought this was an ego thing or a way out of listening. But I've noticed my daughter stopped doing this when she started taking medication."

Consider the timeworn axiom, "It's not what you say but how you say it," and add this corollary: "It's not how something is said but how you hear it." The bottom line is this: On a foundational level, good communications between two individuals depends on "good communications" within each individual's neural networks. Like many other ADHD-related issues, the comprehension deficits (secondary to poor working memory) or even auditory processing deficits (APDs) so often diagnosed these days in children with ADHD do not seem to go away once children mature. Some literature on the topic suggests that APD largely might not even exist except as secondary to ADHD—a case of "discipline bias," in which the diagnosis will depend on whether a psychiatrist or an audiologist is consulted first (Keller, 1992); several studies indicate that methylphenidate improves APD (e.g., Cook et al., 1993; Keith & Engineer, 1991).

Clearly, for some individuals, communications training of any type will go only so far without stimulant medication to address auditory processing issues as well as other interfering symptoms. As one responder to a blog post on the topic (Pera, 2009) wrote:

> When I read "it can cause a person to misinterpret content and even tone of voice," I said "Thank you, thank you God!" I have been questioning how I talk to my husband, thinking I must be unconsciously using a tone of voice that is opposite to my feelings. He won't take medication for ADHD because he says that it is bad for his high blood pressure. He used to take it, and our communication was so much better.

Preliminary study also suggests that medication can improve "theory of mind" and empathic functions in children with ADHD (Maoz et al., 2014), functions that can be crucial to truly hearing the other person and responding appropriately to requests and vulnerabilities. Psychologist Robert Brooks points to a poor sense of empathy, a lack of cooperation, and being difficult to please (insatiability) as three common ADHD-related patterns that seriously damage relationships (quoted in Pera, 2008).

Along with medication, the first two principles' emphasis on psychoeducation and reducing relationship-damaging behaviors can result in smoother interactions. When wading through the tricky waters of emotional self-disclosure (third principle), however, couples often benefit from a conceptual framework for responding in a supportive way. With this goal in mind, empirically supported protocols involve training the couple in communication and problem-solving strategies, guiding them in session and providing take-home exercises.

PROVIDING A STRUCTURE TO FACILITATE CONVERSATIONS

The goal in helping these couples is to provide a structure that promotes active listening, problem solving, and validation of the other's point of view, while simultaneously reducing reactivity, interruption, and argument. One model that ADHD clinicians find particularly helpful in meeting these objectives is Imago Relationship Therapy (IRT). Robbins (2005) explains the rationale for IRT being useful in the ADHD context, and creates a procedure for using IRT with these couples, with an emphasis on addressing EF deficits and emotional dysregulation (Robbins, in press). Briefly, the core of IRT is a procedure called the *couple's dialogue*, which comprises three parts: mirroring, validating, and empathizing. Although simple in concept, the dialogue requires active monitoring to help each partner stay within the guidelines that serve to limit argument, interruption, digression, and avoidance. Variations of the dialogue serve specific purposes, such as when partners state their appreciation of each other, express caring behaviors, or broach a sensitive topic that ordinarily results in prickly responses or avoidant shutdown.

Promoting Relationship Strengths

Research Summary

By necessity, a clinician's efforts predominantly focus on clients' challenges. This fifth common principle for effective couple therapy promotes balance by helping clients to "tell a new story" about their relationship, to identify their areas of strength and resilience along with the gains made in therapy, especially as therapy nears an end. Frequently, strengths have been present all along but were buried beneath negative patterns. Emphasizing the positive aspects of the relationship via activities in session and between sessions creates higher salience, enhancing partners' enjoyment of each other and their relationship. Highlighting positive behaviors also reinforces the chances for their reoccurrence. The clinician is cautioned against superimposing his or her views of what constitutes a couple's reasons for celebration. Only those strengths and successes that carry meaning for the couple will continue to influence their future course.

Adapted for ADHD

It bears repeating: Most of these couples seeking treatment carry long-engrained negative perspectives about the relationship and about each other, which serve only to exacerbate counterproductive behavior patterns such as nagging, misattributions, and withdrawal of affection; these corrosive patterns amplify distorted views of each other and the relationship, thus creating a vicious cycle (Robin, in press-b; Ramsay, in press).

The practice of expressing appreciation and caring behaviors, as mentioned in the fourth principle as part of the IRT model adapted for ADHD, begins to steer interactions in a positive direction. Such ongoing "codification" of caring behaviors might strike some clients as false or stultifying, but they are encouraged to understand that effective ADHD management often requires precisely this: physical, external supports in following through on desired behavior toward stated goals (Barkley, 2011), whether those supports include a list of instructions for dealing with laundry or "nice things to do for your spouse when sick with a cold or flu." Gradually, and with conscious effort, partners are coached to remember the qualities that attracted them to each other.

Eventually, they are ready to conceptualize a joint ideal of their relationship. With the clinician's guidance, this involves writing down descriptors that cover the range of relationship goals, from problem solving to providing mutual supports, and that can be keyed to specific goals (e.g., "We learn new problem-solving techniques together" and "We make time for fun each month, reserving three date nights or weekend days to do something pleasurable together and enjoy each other's companionship"). Once the pair agrees on the relationship ideals, they can be typed and printed in an attractive format (even illustrated with photos depicting the "reward," however they envision it, to keep their "eyes on the prize"), posted in a prominent place, and revisited regularly.

CONCLUSION

Working with these highly diverse couples to resolve previously intractable problems and counterproductive mind-sets—or at least to part ways on more amicable terms—is not the easiest endeavor in the world, but it can be among the most satisfying. Moreover, because such work identifies and seeks to address the foundational role that unrecognized ADHD plays in millions of couples' discordant domestic lives, it stands to revolutionize the effectiveness of couple therapy in general.

KEY CLINICAL POINTS

- ✓ ADHD in adults is associated with significant dissatisfaction in intimate partner and cohabiting (marital) relationships from the perspective of both parties. The inherent symptoms and associated EF deficits (especially emotional dysregulation) in ADHD can contribute substantially to greater conflict between couples, greater risk for intimate partner violence, and divorce.
- ✓ Extant research and my large ADHD Partner Survey provide more specific details on the nature of intimate relationship problems in adults with ADHD. These appear to center on (1) impaired interpersonal behavior, (2) physical intimacy, and (3) impaired functioning in various domains of adult major life activities (driving, financial management, childrearing, sleep, and health maintenance, among others).
- ✓ To date, no research has examined specifically how best to treat the relationship problems evident in couples in which one (or both) partners have ADHD. However, there is substantial research examining the key ingredients that make for successful couples' counseling more generally. These can be tailored to adults with ADHD, given what is known to date about the disorder and the problems they may experience in their relationships that stem from ADHD.
- ✓ Five factors appear to influence the ability of ADHD couples to manage conflict and sustain their relationships: (1) skills deficits, (2) cognitive distortions, (3) degree of impairment, (4) comorbidity, and (5) family-of-origin issues.
- ✓ Models for helping couples cope with any psychiatric disorder seem to include psychoeducation, training in communication and problem solving, and three options for partner involvement: partner-assisted, disorder-specific, and couple therapy.
- ✓ Meta-analyses and clinical experience indicate that effective couple counseling generally involves five key principles: (1) changing views of the relationship, (2) modifying dysfunctional behavior, (3) decreasing emotional avoidance, (4) improving communication, and (5) promoting relationship strengths.
- ✓ Useful counseling approaches for ADHD couples specifically have taken these and other key principles and

adapted them for the specific types of problems these couples often encounter. These approaches, recently developed by Pera and Robin (2015) and Ramsay (2015) offer some guidance on clinical intervention with couples in which ADHD is a major problem. Until such time that empirical research examines the effectiveness of these (and other) approaches to couple counseling for adult ADHD, these programs provide clinicians with an excellent starting point for providing such interventions.

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CHAPTER 35

Pharmacotherapy of ADHD in Adults

Jefferson B. Prince, Timothy E. Wilens, Thomas J. Spencer, and Joseph Biederman

Despite increased recognition that children with attention-deficit/hyperactivity disorder (ADHD) commonly grow up to be adults with the same disorder, evidence-based guidelines on the treatment of adults with ADHD are lacking. Support groups (e.g., *www.chadd.org* and *www.add.org*) assist the newly diagnosed adult by providing education, an overview of treatment options, available resources, and peer support. Recently, the World Health Organization supported the development of an easy-to-use screening tool for ADHD in adults. This tool has been validated (Kessler, Adler, et al., 2007) and is easy to access and use. Fortunately, over the past several years, several organizations, including the American Academy of Child and Adolescent Psychiatry, the Center for ADHD Advocacy/Canada, and the European Network Adult ADHD, have published clinical guidelines that provide health care providers with a thorough description of the the clinical features of ADHD across the lifespan and outline principles for the assessment, diagnosis, recognition of common comorbid disorders, and treatment of ADHD (Haavik, Halmoy, et al., 2010; Kooij, Bejerot, et al., 2010; Pliszka, 2007; Volkow & Swanson, 2013). Once a reliable and valid diagnosis is established, effective treatment of ADHD in adults blends psychoeducation, pharmacotherapy, and psychosocial treatments.

Over the course of the past decade, the database on the safety, tolerability, and efficacy of medications to treat adults with ADHD has significantly expanded. Recent reviews (Fredriksen, Halmoy, et al., 2013; Stevens, Wilens, et al., 2013; T. Wilens, Morrison, et al., 2011) support the safety, tolerability, and efficacy of medication as a cornerstone of treatment for adults with ADHD. Currently formualtions of two types of extended-delivery stimulant (i.e., amphetamine and methylphenidate) and one nonstimulant (atomoxetine [ATMX]) are specifically approved by the U.S. Food and Drug Administration (FDA) for the treatment of ADHD in adults. Prior to pharmacotherapy for ADHD, it is important for patients to undergo a thorough psychiatric evaluation assessing neurodevelopmental, psychological, medical, social, environmental, and cognitive aspects of current symptoms, their longitudinal course, as well as their impact on daily functioning. Treatment with medication for ADHD in adults should be a collaborative investigation between patient and health care provider to set clear, realistic treatment goals, as well as to target specific symptoms

and problematic areas of functioning. Since many adults with ADHD suffer comorbid psychiatric disorders, it is necessary to prioritize treatment if clinically significant psychiatric comorbidities are present, typically sequencing initial treatment for the more severe disorder. In the following sections, guidelines for pharmacotherapy are delineated, available information on the use of medications for adult ADHD is reviewed, and pharmacological strategies are suggested for the management of ADHD symptoms with accompanying comorbid conditions.

STIMULANTS

Stimulants remain the best-studied and most frequently used treatment for ADHD in children, adolescents, and adults. Over 300 controlled studies of pediatric ADHD have shown stimulants to be safe, well tolerated, and efficacious in reducing ADHD symptoms in the short term, as well as improving self-esteem, cognition, and social/family functioning (Greenhill, Pliszka, et al., 2002). Although the data in adults with ADHD are less extensive, adults appear to tolerate stimulant medication similarly to children. In order to prescribe and monitor stimulants effectively, it is important to maintain a through knowledge of the pharmacodynamic and pharmacokinetic profile of the various preparations of methylphenidate (MPH) and amphetamine (AMP) currently available (see Table 35.1 for a list of these medications).

Stimulants increase intrasynaptic concentrations of dopamine (DA) and norepinephrine (NE; Kuczenski & Segal, 1997; Solanto, 1998; Volkow, Wang, et al., 2001; Wilens & Spencer, 1998). MPH primarily binds to the DA transporter protein (DAT), blocking the reuptake of DA, increasing intrasynaptic DA (Volkow, Wang, et al., 2001). While AMPs diminish presynaptic reuptake of DA by binding to DAT, these compounds also travel into the DA neuron, promoting release of DA from reserpine-sensitive vesicles in the presynaptic neuron (Solanto, 1998). In addition, stimulants (AMP, MPH) increase levels of NE and serotonin (5-HT) in the interneuronal space (Kuczenski & Segal, 1997). Although group studies comparing MPH and AMPs generally demonstrate similar efficacy (Greenhill, Pliszka, et al., 2002; Pliszka, 2007) their pharmacodynamic differences may explain why a particular patient may respond to, or tolerate, one stimulant preferentially over another. It is necessary to appreciate that while the efficacy of AMP and MPH is similar, their potency differs, such that 5 mg of AMP is approximately as potent as 10 mg of MPH.

As originally formulated, MPH was produced as an equal mixture of *d,l*-threo-MPH and *d,l*-erythro-MPH. The erythro isomers of MPH appear to produce side effects; thus, MPH is now manufactured as an equal racemic mixture of *d,l*-threo-MPH (*Physicians' Desk Reference*, 2013). Behavioral effects of immediate-release MPH peak 1 to 2 hours after administration, and tend to dissipate within 3 to 5 hours. After oral administration, immediate-release MPH (MPH-IR) is readily absorbed, reaching peak plasma concentration in 1.5 to 2.5 hours, and has an elimination half-life of 2.5–3.5 hours. After oral administration, but prior to reaching the plasma, the enzyme carboxylesterase (CES-1), which is located in the walls of the stomach and liver, extensively metabolized MPH via hydrolysis and deesterification, with little oxidation (Markowitz & Patrick, 2008; Patrick & Markowitz, 1997). Individual differences in CES-1's hydrolyzing activity may result in variable metabolism and serum MPH levels (Zhu, Patrick, et al., 2008). While generic MPH has a similar pharmacokinetic profile to Ritalin, it is more rapidly absorbed and peaks sooner (Greenhill & Osman, 1999). Due to its wax-matrix preparation, the absorption of the sustained-release MPH preparation (MPH-SR, Ritalin-SR) is variable (Patrick, Straughn, et al., 1989), with peak MPH plasma levels in 1 to 4 hours, a half-life of 2 to 6 hours, and behavioral effects that may last up to 8 hours (Birmaher, Greenhill, et al., 1989). The availability of the various new extended-delivery stimulant formulations has greatly curtailed use of MPH-SR.

Concerta (OROS-MPH) uses the oral osmotic release system (OROS) technology to deliver a 50:50 racemic mixture of *d,l*-threo-MPH (Swanson, Gupta, et al., 2003). OROS-MPH for the treatment of ADHD in children, adolescents, and adults is available in 18-, 27-, 36-, and 54-mg doses and is indicated in doses up to 72 mg daily. The 18-mg caplet of OROS-MPH provides an initial bolus of 4 mg of MPH, delivering the remaining MPH in an ascending pattern, such that peak concentrations are generally reached around 8 hours after dosing; it is labeled for 12 hours of coverage (*Physicians' Desk Reference*, 2013; Spencer, Biederman, et al., 2006). A single morning dose of 18, 27, 36, 54, or 72 mg of OROS-MPH is approximately bioequivalent to 5, 7.5, 10, 15, or 20 mg, respectively, of MPH-IR administered three times daily.

TABLE 35.1. Available FDA-Approved Treatments for ADHD

Medication	Formulation and mechanism	Duration of activity	How supplied	Usual absolute (and weight-based) dosing range	FDA-approved maximum dose for ADHD
MPH (Ritalin)[a]	Tablet of 50:50 racemic mixture *d,l*-threo-MPH	3–4 hours	5-, 10-, and 20- mg tablets	(0.3–2 mg/kg/day)	60 mg/day
Dex-MPH (Focalin)[a]	Tablet of *d*-threo-MPH	3–5 hours	2.5, 5, and 10 mg tablets (2.5 mg Focalin equivalent to 5 mg Ritalin)	(0.15–1 mg/kg/day)	20 mg/day
MPH (Methylin)[a]	Tablet of 50:50 racemic mixture *d,l*-threo-MPH	3–4 hours	5-, 10-, and 20-mg tablets	(0.3–2 mg/kg/day)	60 mg/day
MPH-SR (Ritalin-SR)[a]	Wax-based matrix tablet of 50:50 racemic mixture *d,l*-threo-MPH	3–8 hours; variable	20-mg tablets (amount absorbed appears to vary)	(0.3–2 mg/kg/day)	60 mg/day
MPH (Metadate ER)[a]	Wax-based matrix tablet of 50:50 racemic mixture *d,l*-threo-MPH	3–8 hours; variable	10- and 20-mg tablets (amount absorbed appears to vary)	(0.3–2 mg/kg/day)	60 mg/day
MPH (Methylin ER)[a]	Hydroxypropyl methylcellulose base tablet of 50:50 racemic mixture *d,l*-threo-MPH; no preservatives	8 hours	10- and 20-mg tablets 2.5-, 5-, and 10-mg chewable tablets 5 mg/5 ml and 10 mg/5 ml oral solution	(0.3–2 mg/kg/day)	60 mg/day
MPH (Ritalin LA)[a]	Two types of beads give bimodal delivery (50% immediate-release and 50% delayed-release) of 50:50 racemic mixture *d,l*-threo-MPH	8 hours	20-, 30-, and 40-mg capsules; can be sprinkled	(0.3–2 mg/kg/day)	60 mg/day
D-MPH (Focalin XR)[b]	Two types of beads give bimodal delivery (50% immediate-release and 50% delayed-release) of *d*-threo-MPH	12 hours	5-, 10-, 15-, 20-, 25-, 30-, 35-, and 40-mg capsules	0.15–1 mg/kg/day	30 mg/day in youth; 40 mg/day in adults
MPH (Metadate CD)[a]	Two types of beads give bimodal delivery (30% immediate-release and 70% delayed-release) of 50:50 racemic mixture *d,l*-threo-MPH	8 hours	20-mg capsule; can be sprinkled	(0.3–2 mg/kg/day)	60 mg/day
MPH (Daytrana)[a]	MPH transdermal system	12 hours (patch worn for 9 hours)	10-, 15-, 20-, and 30-mg patches	0.3–2 mg/kg/day	30 mg/day
MPH (Concerta)[a, b]	Osmotic pressure system delivers 50:50 racemic mixture *d,l*-threo-MPH	12 hours	18-, 27-, 36-, and 54-mg caplets	(0.3–2 mg/kg/day)	72 mg/day

(continued)

TABLE 35.1. *(continued)*

Medication	Formulation and mechanism	Duration of activity	How supplied	Usual absolute (and weight-based) dosing range	FDA-approved maximum dose for ADHD
MPH (Quillivant XR)	Extended-release liquid	12 hours	25 mg/5 ml	(0.3–2 mg/kg/day)	60 mg/day
AMPH (Dexedrine Tablets)[c]	*d*-AMPH tablet	4–5 hours	5 mg tablets	(0.15–1 mg/kg/day)	40 mg/day
AMPH (Dextrostat)[c]	*d*-AMPH tablet	4–5 hours	5- and 10-mg tablets	(0.15–1 mg/kg/day)	40 mg/day
AMPH (Dexedrine Spansules)[c]	Two types of beads in a 50:50 mixture short and delayed-absorption of d-AMPH	8 hours	5-, 10-, and 15-mg capsules	(0.15–1 mg/kg/day)	40 mg/day
Mixed salts of AMPH (Adderall)[c]	Tablet of *d*,*l*-AMPH isomers (75% *d*-AMPH and 25% *l*-AMPH)	4–6 hours	5-, 7.5-, 10-, 12.5-, 15-, 20-, and 30-mg tablets	(0.15–1 mg/kg/day)	40 mg/day
Mixed salts of AMPH (Adderall-XR)[a, b]	Two types of beads give bimodal delivery (50% immediate-release and 50% delayed-release) of 75:25 racemic mixture *d*,*l*-AMPH	At least 8 hours (but appears to last much longer in certain patients)	5-, 10-, 15-, 20-, 25-, and 30-mg capsules; can be sprinkled	(0.15–1 mg/kg/day)	30 mg/day in children Recommended dose is 20 mg/day in adults
Lisdexamfetamine (Vyvanase)[a, b]	Tablets of dextroamphetamine and L-lysine	12 hours	30-, 50-, and 70-mg tablets		70 mg/day
Atomoxetine (Strattera)[a, b]	Capsule of atomoxetine	5-hour plasma half-life but CNS effects appear to last much longer	10-, 18-, 25-, 40-, 60-, and 80-mg capsules	1.2 mg/kg/day	1.4 mg/kg/day or 100 mg
Guanfacine ER (Intuniv)[d]	Extended-release tablet of guanfacine	Labeled for once-daily dosing	1-, 2- ,3-, and 4-mg tablets	Up to 4 mg per day	Up to 4 mg per day
Clonidine ER(Kapvay)[d]	Extended-release tablet of clonidine	Labeled for twice-daily dosing	0.1-mg tablet	0.1–0.2 mg twice daily	Up to 0.4 mg daily

Note. Trade names of medications are in parentheses.
[a]Approved to treat ADHD in youth ages 6 years and older.
[b]Specifically approved for treatment of ADHD in adults.
[c]Approved to treat ADHD in youth ages 3 years and older.
[d]Approved to treat ADHD in youth ages 6–17 years as monotherapy or as adjunctive treatment with stimulant.

Metadate CD (modified-release MPH [MPH-MR]), the first available extended-delivery stimulant preparation to employ beaded technology, is available in capsules of 10, 20, 30, 40, 50, and 60 mg, which may be sprinkled on food. Using Eurand's Diffucaps technology, MPH-MR contains two types of coated beads, MPH-IR and extended-release MPH (MPH-ER). Metadate delivers 30% of *d,l*-threo-MPH initially, and 70% of *d,l*-threo-MPH several hours later. MPH-MR is designed to simulate twice-daily (bid) dosing of IR MPH providing approximately 8 hours of coverage. The efficacy of MPH-MR capsules has been demonstrated (Greenhill, Findling, et al., 2002), and it is approved for treatment in youth with ADHD in doses of up to 60 mg/day (*Physicians' Desk Reference*, 2013). An extended-delivery tablet form of Metadate (Metadate ER) is also available in doses of 10 and 20 mg.

Ritalin-LA (MPH extended-release capsules [MPH-ERC]), another beaded-stimulant preparation that may be sprinkled on food (*Physicians' Desk Reference*, 2013), is available in capsules of 10, 20, 30, and 40 mg, essentially equivalent to 5, 10, 15 and 20 mg of MPH-IR delivered twice daily. MPH-ERC uses the beaded spheroidal oral drug absorption system (SODAS) technology to achieve a bimodal release profile that delivers 50% of its *d,l*-threo-MPH initially and another bolus approximately 3 to 4 hours later, providing around 8 hours of coverage.

The primarily active form of MPH appears to be the *d*-threo isomer (Ding, Fowler, et al., 1997; Ding, Gatley, et al., 2004; Patrick, Caldwell, et al., 1987), which is available in both IR tablets (Focalin 2.5, 5, and 10 mg) and, employing the SODAS technology, extended-delivery capsules (Focalin XR 5, 10, 15, and 20 mg). *d*-MPH is approved to treat ADHD in children, adolescents, and adults in doses of up to 20 mg per day and has been labeled to provide a 12-hour duration of coverage (*Physicians' Desk Reference*, 2013). Although not definitive, 10 mg of MPH appears to be approximately equivalent to 5 mg of *d*-MPH, and clinicians can reasonably use this estimate in clinical practice (Quinn, Wigal, et al., 2004).

The MPH transdermal system (MTS; Daytrana) delivers MPH through the skin via the DOT Matrix transdermal system. The patches are applied once daily and intended to be worn for 9 hours, although in clinical practice they can be worn for shorter and longer periods of time. The MTS usually takes effect within 2 hours and provides coverage for 3 hours after removal. MTS is available in 10-, 15-, 20-, and 30-mg patches (McGough, Wigal, et al., 2006; Pelham, Burrows-MacLean, et al., 2005; Pelham, Manos, et al., 2005). Since the MPH is absorbed through the skin, it does not undergo first-pass metabolism by CES-1 in the liver, resulting in higher plasma MPH levels (Zhu, Wang, et al., 2008). Therefore, patients may require lower doses with MTS compared to oral preparations (10 mg of MTS = 15 mg of oral MPH-ER). MTS may be a particularly useful treatment option for patients who have difficulty swallowing or tolerating oral stimulant formulations, or for those who need flexibility in the duration of medication effect.

Recently an extended-delivery MPH oral suspension formulation became available (MEROS or Quillivant XR 25 mg/5 mL). Although head-to-head trials have not been published and clinical experience to date is limited, this formulation appears to provide similar efficacy and duration of effect as other extended-delivery MPH preparations (Childress & Sallee, 2013; Wigal, Childress, et al., 2013). This preparation may be particularly helpful for patients who prefer a liquid preparation or who experience skin reactions to the transdermal patch. Prior to dosing, the manufacturer recommends shaking the bottle to ensure even distribution of medication.

AMP is available in three forms (*Physicians' Desk Reference*, 2013): dextroamphetamine (DEX; trade name: Dexedrine), mixed amphetamine salts (MAS; trade name: Adderall), and lisdexamfetamine dimesylate (LDX; trade name: Vyvanse). DEX tablets achieve peak plasma levels 2–3 hours after oral administration and have a half-life of 4–6 hours. Behavioral effects of DEX tablets peak 1–2 hours after administration and last 4–5 hours. For DEX spansules, these values are somewhat longer. MAS comprises equal portions of *d*-AMP saccharate, *d,l*-AMP asparate, *d*-AMP sulfate, and *d,l*-AMP sulfate, and a single dose results in a ratio of approximately 3:1 *d*- to *l*-AMP (*Physicians' Desk Reference*, 2013). The two isomers have different pharmacodynamic properties, and some patients with ADHD may preferentially respond to one isomer over another. An extended-delivery preparation of MAS is available as a capsule containing two types of Micotrol beads (MAS XR; Adderall XR). The beads are present in a 50:50 ratio, with immediate-release beads designed to release MAS in a fashion similar to MAS tablets, and delayed-release beads designed to release MAS 4 hours after dosing. LDX is FDA-approved for treatment of ADHD in children, adolescents, and adults. LDX (2013) is an AMP prodrug in which L-lysine, a

naturally occurring amino acid, is covalently linked to *d*-amphetamine. After oral administration, the prodrug is metabolically hydrolyzed in the body to release *d*-amphetamine. LDX appears to reduce abuse liability (e.g., misuse, abuse, and overdose); intravenously and intranasally administered LDX results in similar effects as oral administration (Jasinski & Krishnan 2009a, 2009b). It is available in capsules of 20, 30, 40, 50, 60 and 70 mg that appear to be comparable to MAS XR doses of 10, 15, 20, 25, 30 and 35 mg, respectively.

Efficacy of Stimulants in Adults with ADHD

To date we are aware of at least 23 short-term controlled studies and at least 18 longer term studies of adults receiving either MPH, *d*-MPH, MAS or LDX (for recent reviews, see Fredriksen, Halmoy, et al., 2013; Wilens, Morrison, et al., 2011). Of these, 32 controlled studies were with MPH and 9 were with AMP.

In contrast to consistent robust responses to stimulants in children and adolescents of approximately 70% (Greenhill, Pliszka, et al., 2002; Spencer, 2004; Wilens & Spencer, 2000) controlled studies in adults (see Table 35.2) have shown more equivocal responses to stimulants, ranging from 25% (Mattes, Boswell, et al., 1984) to 88% (Ginsberg, Katic, et al., 2011). Variability in the response rate appears to be related to several factors, including the diagnostic criteria utilized to determine ADHD, varying stimulant doses, comorbidity, and differing methods of assessing overall response. In clinical practice successful pharmacotherapy is usually based on a clear understanding of the "medication trial" process and the ongoing answer to two fundamental questions: (1) How well is the medication tolerated? and (2) How much does it help? As Golomb, McGraw, and colleagues (2007) noted, "Adverse drug reaction (ADR) rates in clinical trials often understate those in practice." Fortunately there are a number of ways for the vigilant clinician to assess tolerability: spontaneous patient–family report, symptom-specific inquiry, medication-specific inquiry, and a structured report form or interview. In clnical practice, it is wise to inquire about specific expected, common side effects and to familiarize the patient–family with uncommon and potentially dangerous ones (Greenhill, Vitiello, et al., 2004). In order to understand the effect of the medication, it is important for the patient and clinican to agree on how to collect information and measure response. In clincial practice this usually means collecting information from observation, rating scales, anchor points (i.e., targeted areas of impaired function), and possibly observations from the patient's family/friends. Dosing of the stimulants appears important in outcome: (1) Controlled investigations using higher stimulant dosing (>1.0 mg/kg/day of MPH or >0.5 mg/kg/day of AMP) generally resulted in more robust outcomes than those using lower stimulant dosing (< 0.7 mg/kg/day); and (2) several studies utilizing a dose-ranging paradigm found a dose-dependent response to stimulants in adults with ADHD. For instance, Spencer, Adler, and colleagues (2007) found that 40 mg of *d*-MPH-XR resulted in a larger response rate than the 20-mg dose. Similarly, cohorts described by Medori, Ramos-Quiroga, and colleagues (2008) and Casas, Rosler, and colleagues (2013) showed a similar pattern of response to OROS-MPH. Casas, Rosler, and colleagues assessed the effect of OROS-MPH in a double-blind, placebo-controlled study of 279 participants receiving either placebo, or 54 or 72 mg/day of medication for a 13-week period. In this study, improvements in the Conners' Adult ADHD Rating Scales—Observer (CAARS-O) were significantly greater with OROS-MPH 72 mg versus placebo, but not with OROS-MPH 54 mg versus placebo, suggesting the need for relatively higher doses in adults for efficacy. Along the same lines, an older meta-analysis of six double-blind, placebo-controlled studies comparing treatment of adults with ADHD with MPH (n =140) to placebo (n = 113) found a mean effect size of 0.9 that was nearly double (1.3) in those studies using higher dosing of MPH (mean dose 70 mg/day or 1.05 mg/kg/day) compared to studies using lower doses (effect size 0.7; mean dose 44 mg/day or 0.63 mg/kg/day) (Faraone, Spencer, et al., 2004).

Evidence-based dosing parameters for stimulant medications in adults with ADHD are limited. FDA guidelines for dosing reflect general cautiousness and should not be the only guide for clinical practice. The individually titrated dose should be based on therapeutic efficacy and side effects. Similar to treatment of youth with ADHD, treatment of adults with ADHD may be started with either immediate-release (IR) or extended-delivery (ED) preparations at the lowest possible dose (Greenhill, Pliszka, et al., 2002). The stimulants have an immediate onset of action and may last from 3 to 12 hours based on the formulation of the agent (IR, ED). Initiation of treatment with once-daily dosing in the morning is advisable until an acceptable response is noted. Treatment with IR preparations generally starts at 5 mg of MPH or AMP once daily and is titrated upward every 3–5 days until an effect is noted or adverse effects

TABLE 35.2. Representative Short-Term Controlled Clinical Studies of Stimulants in Adults with ADHD

Study (year)	*N*	Medication	Design	Duration	Total dose (mean and/or range)	Outcome	Comments
Wood, Reimherr, et al. (1976)	15	MPH	Double-blind, placebo crossover	4 weeks	27 mg/day	73% response rate	Diagnostic criteria not well defined; mild side effects.
Mattes, Boswell, et al. (1984)	61	MPH	Double-blind, placebo crossover	6 weeks	48 mg/day	25% response rate	Moderate rate of comorbidity; mild side effects.
Gualtieri & Hicks (1985)	22	MPH	Double-blind, placebo crossover	2 weeks	42 mg/day	Mild–moderate response	No plasma level–response associations
Wender, Reimherr, et al. (1985)	37	MPH	Double-blind, placebo crossover	5 weeks	43 mg/day	57% response rate (11% placebo)	68% dysthymia; 22% cyclothymia; mild side effects.
Spencer, Wilens, et al. (1995)	23	MPH	Double-blind, placebo crossover	7 weeks	1.0 mg/kg/day	78% response rate, dose relationship (4% placebo)	No plasma level associations; no effect of gender or comorbidity.
Iaboni, Bouffard, et al. (1996)	30	MPH	Double-blind, placebo crossover	4 weeks	30–45 mg/day	Moderate response	Improvement in neuropsych and anxiety.
Paterson, Douglas, et al. (1999)	45	*d*-AMPH	Double-blind, parallel	6 weeks	23 mg/day	58% response rate	Weight loss only major adverse effect.
Taylor (2000a, 2000b)	39	*d*-AMPH	Double-blind, placebo crossover	7 weeks	22 mg/day	48% response rate	Used as comparator in two studies of nonstimulants; response > 30% reduction in scales.
Spencer, Biederman, et al. (2001)	27	AMPH salts	Double-blind, placebo crossover	7 weeks	54 mg/day 20–60 mg/day	70% response rate, dose relationship (7% placebo)	No effect of comorbidity or gender on response; well tolerated.
Kooij, Burger, et al. (2004)	45	MPH	Double-blind, randomized crossover	3 weeks	0.5–1.0 mg/kg/day	38–51% response rate (7–18% placebo)	Higher rate of side effects for MPH and placebo.
Carpentier, de Jong, et al. (2005)	25	MPH	Double-blind, placebo crossover	8 weeks	15–45 mg/day	58% response rate on CGI (32% placebo)	Substance use disorder study; positive response to Tx not significantly higher than placebo.

Spencer, Biederman, et al. (2005)	146	MPH	Double-blind, placebo parallel	6 weeks	1.1 mg/kg/day	76% response rate (19% placebo)	Tx well tolerated despite higher dose.
Biederman, Mick, et al. (2006b)	141	OROS MPH	Double-blind, placebo parallel	6 weeks	81 mg/day	66% response rate (39% placebo)	Slight vital sign increases.
Weisler, Biederman, et al. (2006)	255	MAS XR	Double-blind, placebo parallel	4 weeks	20, 40, or 60 mg/day	55% response rate on CGI (27% placebo)	MAS XR 60-mg group had greatest improvement on ADHD RS.
Jain, Hechtman, et al. (2007)	39	MLR MPH	Double-blind, placebo crossover	5–11 weeks	58 mg/day (mean) MLR MPH; 65mg/day (mean) placebo	49% response rate on CGI (23% placebo)	MLR MPH minimal side effects; short trial.
Reimherr, Williams, et al. (2007)	45	OROS MPH	Double-blind, placebo crossover	8 weeks	57 mg/day (Tx responder mean); 75mg/day (Tx nonresponder mean)	54% response rate on CGI (22% placebo)	Total ADHD RS score decrease of 41% (vs. 14%).
Spencer, Adler, et al. (2007)	221	*d*-MPH-ER	Double-blind, fixed dose, placebo parallel	5 weeks	20, 30, or 40 mg/day	54–61% response rate on ADHD RS (34% placebo)	Inconsistent dose–response.
Adler, Goodman, et al. (2008); Weber & Siddiqui (2009)	420	LDX	Double-blind, placebo (2:2:2:1) parallel	4 weeks	30, 50, or 70 mg/day	Response rate on CGI: 57, 62, and 61%, respectively (29% placebo)	Incidence of AEs highest in first week of LDX treatment.
Medori, Ramos-Quiroga, et al. (2008)	401	Prolonged release OROS MPH	Double-blind, placebo parallel	5 weeks	18, 36, or 72 mg/day	Responders were 51, 49, and 60, respectively (27% placebo)	AE rates 75, 76, and 82%, respectively versus 66% in placebo; most commonly decreased appetite and headache.
Chronis-Tuscano, Seymour, et al. (2008)	23	OROS MPH	Double-blind, placebo controlled	7 weeks	36, 54, 72, or 90 mg/day (mean 84 mg/day)	Significant reduction in CGI scores at all doses	Few AEs.
Adler, Zimmerman, et al. (2009)	226	OROS MPH	Double-blind, placebo parallel	7 weeks	68 mg/day OROS MPH (mean); 87mg/day placebo (mean)	37% response rate on CGI and AISRS (21% placebo)	Mild to moderate AE rate, 85% MPH versus 64% placebo; OROS MPH overall effective and well tolerated in dose escalation.

(continued)

TABLE 35.2. *(continued)*

Study (year)	*N*	Medication	Design	Duration	Total dose (mean and/or range)	Outcome	Comments
Spencer, Mick, et al. (2011)	53	OROS MPH or IR MPH	Single-blind, parallel	6 weeks	77 mg/day IR MPH (mean); 80 mg/ dayOROS MPH (mean)	OROS once a day equal to IR MPH TID	OROS well tolerated and similar safety indices as IR; increased adherence with OROS.
Wender, Reimherr, et al. (2011)	105	MPH	Double-blind, placebo crossover	2 weeks	45 mg/day (10–60 mg/day)	74% experienced at least a 50% reduction on WRAADDS sx score (22% placebo)	Participants who improved on MPH IR entered a 12-month, open-label trial.
Wigal, Brams, et al. (2010)	105	LDX	Double-blind, crossover	2 weeks	30, 50, or 70 mg/day	77% response on CGI (23% placebo)	After open-label dose optimization (4 weeks), subjects entered 2-week crossover phase.
Winhusen, Somoza, et al. (2010)	255	OROS MPH	Double-blind, placebo parallel	11 weeks	18–72 mg/day	71% response rate on CGI (44% placebo)	Cigarette cessation study; cigarette smoking abstinence not significantly different between groups.
Babcock, Dirks, et al. (2012)	420	LDX	Double-blind, placebo controlled	4 weeks	30, 50, and 70 mg/day	Adults with significant baseline ADHD sx and prior AMPH treatment showed improvements in symptoms	71 participants were discontinued and 41 were receiving AMPH at screening.
Total (*N* = 26)	*N* = 3,224 15–420 (range)	MPH: 19 AMPH: 4 LDX: 3	Single-blind: 1 Double-blind: 25	2 weeks–11 weeks	MPH: 10–90 mg/day AMPH: 20–60 mg/day LDX: 30–70 mg/day	MPH, AMPH, and LDX improved ADHD Sxs	AEs mild–moderate in severity.

Note. Duration up to 11 weeks. Response rate refers to subject reported *much improved* to *very much improved* (i.e., by clinical global improvement) or with clinically significant reduction in symptoms on ADHD rating scales. AE, adverse event; AISRS, ADHD Investigator Symptom Report Scale; AMPH, amphetamine; CGI, Clinical Global Impression; *d*-AMPH, dexamphetamine; *d*-MPH, dexmethylphenidate; Dx, diagnosis; ER, extended release; IR, immediate release; LDX, lisdexamfetamine; MAS, mixed amphetamine salts; MAS XR, mixed amphetamine salts extended release; MLR, multilayer release; MPH, methylphenidate; OROS MPH, osmotic release oral system methylphenidate; RS, rating scale; SR, sustained Release; Sx, symptom;, Tx, treatment; WRAADDS, Wender–Reimherr Adult Attention Deficit Disorder Scale.

emerge. Repeat dosing of IR stimulants throughout the day is dependent on tolerability (e.g., side effects and onset–offset experiences), intensity and duration of effectiveness. Typically, the behavioral half-life of the IR stimulants necessitates at least twice-daily dosing, with the addition of similar or reduced afternoon doses dependent on breakthrough symptoms. In a typical adult, dosing of MPH-IR is generally up to 30 mg, three to four times a day, or AMP 15–20 mg, three to four times a day. Stimulants are generally dosed in an absolute manner (e.g., milligrams per day). However, absolute dose limits (in milligrams) may not adequately consider a patient's height, weight, and use in refractory cases. Furthermore, it appears that for stimulants to be most effective, doses of 0.5–1.0 mg/kg/day of MPH (lower for *d*-MPH) or up to 0.5 mg/kg/day of AMP seem necessary for efficacy (Biederman, Mick, et al., 2010). Currently, most adults with ADHD who are treated with a stimulant are initially prescribed an FDA-approved ED formulation such as MAS-XR, LDX, OROS-MPH or *d*-MPH-XR. It is notable that comparable findings between response rates and adverse effects have been reported between ER and IR stimulants. For instance, Spencer, Mick, and colleagues (2011) reported similar response rates and adverse effects using similar dosing of MPH-IR three times daily and OROS-MPH-ER once daily.

Since there is not a "holy grail" of pharmacotherapy in adults with ADHD, a useful strategy in clinical practice is to initiate and titrate patients with one type of stimulant (MPH or AMP), then, unless they have a vigorous response to the initial treatment, to try them on the other type of stimulant (AMP or MPH). Recently Ramtvedt, Roinas, and colleagues (2013) improved both the response rate (an increase from 72 to 92%) and strength of response in a group of 9- to 14-year-old youth by utilizing a straightforward crossover design in which each subject received, in a blinded manner, 2 weeks of treatment with comparable doses of MPH, DEX and placebo. Moreover, in clinical practice, it is common and usually appropriate to combine IR and ED formulations of stimulants. These combinations allow the patient–clinician team to adapt dosing to provide coverage for the patient in various context. Psychoeducation is an essential aspect of successful pharmacotherapy; therefore, it is helpful for the patient to understand several additional points about common side effects: that excessive (and sometimes ordinary) amounts of caffeine and/or nicotine, as well as decongestants, may result in physical sensations of "overstimulation"; that stimulants may alter their usual response to alcohol; and that regular physical activity, sleep, and meals are essential. In addition, clinicans may improve patient adherence to the prescribed medication regimen by more frequent follow-up appointments (espcially in early in treatment), familiarize patients with the process of obtaining the medication (e.g., written prescriptions necessary for stimulants), simplify dosing (using ED medications when possible), encourage questions, and repeat information (Adler & Nierenberg, 2010; Kripalani, Yao, et al., 2007).

There is a paucity of longer-term data related to stimulants for ADHD. Recently Fredriksen, Halmoy, and colleagues (2013) reviewed the literature on longer-term (> 24 weeks up to 4 years) efficacy of stimulants and ATMX in adults with ADHD. Although clinical studies demonstrate continued efficacy of ADHD medications when used for longer periods of time, many treated adults do not adhere to their prescribed regimen. Moreover, these investigators highlight significant limitations in our current database and advocate for additional investigations. We located 10 open (N = 1,524 subjects) and 9 controlled (N = 1,691 subjects) studies of at least 12 weeks' duration (see Table 35.3). The majority of longer-term studies are the continuation of controlled trials in which subjects are followed openly. In one of the largest controlled long-term study, Weiss and Hechtman (2006) demonstrated continued improvement with *d*-AMP alone or in combination with paroxetine (64 and 44% response rates, respectively, vs. 16% placebo) over 20 weeks. Rösler, Fischer, and colleagues (2009) showed that MPH-ER significantly improved ADHD (61 vs. 42% placebo) and related symptoms over 24 weeks. Wender, Reimherr, and colleagues (2011) studied 78 subjects who were part of a controlled trial for 12 months and found that those who responded to MPH in the short term responded to longer-term treatment, with a reductionin ADHD symptoms. These data seem to suggest that response to stimulants is sustained at the 24- to 72-week follow-up endpoints. Furthermore, Kooij, Rösler, and colleagues (2013) assessed the adherence of 276 patients in a double-blind, placebo-controlled study of OROS-MPH over a 13-week period. They found that patients on medication had reduced ADHD symptoms and that adherence was 92.6 and 93.3% (OROS-MPH, 54 and 72 mg/day, respectively) versus 97.5% (placebo). Although limited by the higher rates of adherence in this clinical trial, factors signifcantly associated with nonadherence were found to be female gender, shorter time since ADHD diganosis, and higher education level.

TABLE 35.3. Representative Longer-Term Studies of Stimulants in Adults with ADHD

Study (year)	*N*	Medication	Design	Duration	Total dose (mean and/or range)	Outcome	Comments
Levin, Evans, et al. (1998)	12	MPH SR	Open	12 weeks	68 mg/day (40–80 mg/day)	Improved ADHD and cocaine use	Cocaine abusers, 8/12 completed; no abuse of MPH.
Horrigan & Barnhill (2000)	24	AMPH salts	Open	16 weeks	11 mg/day	54% response rate on CGI	Low doses used; retrospectively analyzed.
Schubiner, Saules, et al. (2002)	48	MPH	Double-blind, placebo parallel	12 weeks	79 mg/day (30–90 mg/day)	77% response rate on global improvement scale (21% placebo)	Comorbid cocaine dependence; CBT for both arms.
Biederman, Spencer, et al. (2005); Weisler, Biederman, et al. (2005)	223	MAS XR	Open	≤ 24 months	20, 40, and 60 mg/day	ADHD RS improved for all ($p < .001$)	AEs were mild to moderate, minimal cardiac effects; extension of controlled study.
Weiss & Hechtman (2006)	98	Paroxetine and/or *d*-AMPH	Double-blind, placebo factorial	20 weeks	Paroxetine: 10, 20, 30, and 40 mg/day *d*-AMPH: 5,10, 15, and 20 mg/day	64% response rate to *d*-AMPH, 44% to paroxetine/d-AMPH, 17% to paroxetine, and 16% to placebo	Patients who received both *d*-AMPH and paroxetine had more severe AEs, but did not show greater improvement than patients treated with monotherapy.
Levin, Evans, et al. (2007)	106	MPH	Double-blind, placebo parallel	14 weeks	40 mg/day (10–60 mg/day)	47% response rate on AARS (55% placebo)	Substance use disorder study. Lower probability of cocaine in urine for MPH versus placebo ($p = .001$).
Spencer, Landgraf, et al. (2008)	274	Triple bead AMPH salts (MAS)	Double-blind, placebo parallel	5 weeks (phase 1); 2 weeks (phase 2); 7 weeks (phase 3)	13, 25, 38, 50, 63, or 75 mg/day after dose optimization	52% response rate on CGI (21% placebo)	Mild to moderate AEs of insomnia, dry mouth, decreased appetite, headache, weight loss; improved quality of life >12-hour duration.
Adler (2009)[a]	170	*d*-MPH-ER	Open	6 months	20–40 mg/day	95% response rate on CGI	Open-label extension of Spencer, Adler, et al. (2007).

Rösler, Fischer, et al. (2009)	359	MPH ER	Double-blind, placebo parallel	6 months	41 mg/day	61% response rate (42% placebo)	Relatively low doses used; increased heart rate among MPH ER group.
Weisler, Young, et al. (2009); Ginsberg, Katic, et al. (2011)	349	LDX	Open	12 months	30, 50, or 70 mg/day	84% improvement on CGI	Most AEs were mild to moderate in severity.
Bejerot, Ryden, et al. (2010)	133	MPH *d*-AMPH	Open	6–9 month follow-up	49 mg/day (18–90 mg/day) 28 mg/day (15–70 mg/day)	80% response rate	66 of 133 discontinued (38% before the 6–9 month time point).
Biederman, Mick, et al. (2010)	227	OROS MPH	Double-blind, placebo parallel	6 weeks (phase 1); 24 weeks (phase 2); 4 weeks (phase 3)	78 mg/day OROS MPH at phase 1 endpoint (mean)	62% response rate on CGI and AISRS (37% placebo)	Results include phase 1 endpoint response rates only.
Konstenius, Jayaram-Lindstrom, et al. (2010)	24	OROS MPH	Double-blind, placebo parallel	13 weeks	18–72 mg/day	84% retention in treatment completers (59% placebo)	Study in amphetamine abusers; both groups reduced ADHD Sxs; no difference between groups in craving for amphetamine.
Marchant, Reimherr, et al. (2010)	34	OROS MPH	Open	6 months	60 mg/day	85% response rate on CGI	Followed double-blind crossover phase; all 34 included for safety phase.
Wender, Reimherr, et al. (2011)	78	MPH	Open	12 months	60 mg/day (30–100 mg/day)	94% response rate on CGI	Participants who improved on MPH IR double-blind phase entered the 12-month, open-label trial.
Buitelaar, Trott, et al. (2011)	156	OROS MPH	Open	52 weeks	18, 36, 54, 72, and 90 mg/day	Mean CAARS scores declined from baseline	Subjects completing 52 weeks were eligible for 4 week randomized placebo-controlled withdraw phase.
Casas, Rösler, et al. (2013)	279	OROS MPH	Double-blind, placebo	13 weeks parallel group	54 or 72 mg/day	CAARS total score significanly different for patients on 72 mg versus placebo	CAARS total score was not significant for 54 mg versus placebo.

(continued)

TABLE 35.3. *(continued)*

Study (year)	*N*	Medication	Design	Duration	Total dose (mean and/or range)	Outcome	Comments
Kooij, Rösler, et al. (2013)	276	OROS MPH	Double-blind, placebo	13 weeks	54 and 72 mg/day receiving 2 capsules per day	Both MPH groups showed greater improvement in ADHD compared to placebo	Adherence was 92.6 and 93.3% (OROS MPH 54 and 72 mg/day, respectively), versus 97.5% (placebo). Factors significantly associated with nonadherence included female sex, shorter time since ADHD diagnosis, and higher education level.
Mattingly, Weisler, et al. (2013)	345	LDX	Open	11 months	30, 50, and 70 mg/day	95.7% response rate and 85.0% remission rate for all dosage	Followed double-blind, 4-week forced-dose trial.
Total (*N* = 19)	*N* = 3,215 12–359 (range)	MPH: 13 AMPH: 5 LDX: 2	Double-blind: 9 Open: 10	12 weeks–12 months	MPH: 10–100 mg/day AMPH: 5–75 mg/day LDX: 30–70 mg/day	Long-term effectiveness of MPH and AMPH documented	AEs mild–moderate in severity.

Note. Duration up to 12 months. Response rate refers to subject reported much to very much improved (i.e., by clinical global improvement) or with clinically significant reduction in symptoms on ADHD rating scales. AARS, Adult ADHD Rating Scale; AE, adverse event; AISRS, ADHD Investigator Symptom Report Scale; AMPH, amphetamine; CGI, Clinical Global Impression; *d*-AMPH, dexamphetamine; *d*-MPH, dexmethylphenidate; Dx, diagnosis; ER, extended release; IR, immediate release; LDX, lisdexamfetamine dimesylate; MAS, mixed amphetamine salts; MAS XR, mixed amphetamine salts extended release; MPH, methylphenidate; OROS MPH, osmotic release oral system methylphenidate; RS, rating scale; SR, sustained release.

[a]Subjects not included in overall *N*.

Open-label studies have also shown the effectiveness of long-term stimulants in adults with ADHD. Weisler, Young, and colleagues (2009), in a 12-month study following a double-blind, placebo-controlled trial of initially 349 subjects receiving 30–70 mg/day of LDX, reported 84% improvement at endpoint, and most adverse events were mild to moderate in severity. In a similar 6-month, open-label study following a randomized, placebo-controlled trial of OROS-MPH, Marchant, Reimherr, and colleagues (2010) found a similar response rate, with 85% of the 34 enrolled subjects demonstrating symptom reduction. These aggregate data seem to support the longer-term effectiveness and tolerability of stimulants in adults.

Side Effects of Stimulants

Although generally well tolerated, stimulants can cause clinically significant side effects (including anorexia, nausea, difficulty falling asleep, obsessiveness, headaches, dry mouth, rebound phenomena, anxiety, nightmares, dizziness, irritability, dysphoria, and weight loss (Aagaard & Hansen, 2013; Barkley, McMurray, et al., 1990; Efron, Jarman, et al., 1997; Graham, Banaschewski, et al., 2011; Pliszka, 2007; Wolraich, Brown, et al., 2011). Rates and types of stimulant side effects appear to be similar in patients with ADHD, regardless of age. In patients with a current comorbid mood/anxiety disorder, clinicians should consider whether an adverse effect reflects the comorbid disorder, a side effect of the treatment, or an exacerbation of the comorbidity. Moreover, while stimulants can cause these side effects, many patients with ADHD experience these problems before treatment; therefore, it is important for clinicians to document these symptoms at baseline (Efron, Jarman, et al., 1997).

Stimulants also are associated with small increases in heart rate and blood pressure that are weakly correlated with dose. In part related to changes in vital signs and their biological plausibility, there has been controversy as to cardiovascular (CV) risk in subjects receiving stimulants (Nissen, 2006). However, recent data have further illuminated CV risk in adults taking stimulants. To examine the incidence of potential healthy-user bias, Habel, Cooper, and colleagues (2011), in a large retrospective study of 443,198 adults (ages 25–64 years) from four study sites, examined serious CV events in current/new users and remote users. The authors reported on 806,182 person-years of follow-up (median, 1.3 years per person), and found no relationship between past or current ADHD medication use and serious CV or stroke outcomes. As highlighted by these authors, among young and middle-aged adults, current or new use of ADHD medications, compared with nonuse or remote use, was not associated with an increased risk of serious cardiovascular events. These data mirror the findings of a similary designed study of youth with ADHD (Cooper, Habel, et al., 2011) and a recent review of the cardiovascular literature related to stimulant exposure in ADHD (Hammerness, Perrin, et al., 2011), and seem to suggest that the vital signs changes seen acutely and chronically in adults are usually not clinically significant.

These studies, along with more general guidelines on the use of stimulants (Gutgesell, Atkins, et al., 1999; Perrin, Friedman, et al., 2008), suggest checking vital signs at premedication baseline and periodically thereafter, especially in patients at elevated risk of hypertension (Wilens, Hammerness et al., 2005). These guidelines also recommend monitoring patients for clinical symptoms relevant to underlying CV disturbance and/or deleterious CV interactions with the medication, including palipitations, chest discomfort/pain, syncopal episodes, and shortness of breath (Hammerness, Perrin, et al., 2011). For subjcts with preexisting hypertension, one small study indicated stability in blood pressure when stimulants were used concomitantly with antihypertensives (Wilens, Zusman, et al., 2006), and their use in patients with prexisting conditions such as hypertension does not appear to increase the risk for serious CV outcomes (Cooper, Habel, et al., 2011; Habel, Cooper, et al., 2011). One way to inquire about CV symptoms in the clincial mangement of adults with ADHD is found in Figure 35.1.

Adults with ADHD, with or without medication treatment, often experience sleep difficulties including longer sleep-onset latency and lower sleep efficiency (Barrett, Tracy, et al., 2013; Surman & Roth, 2011; Van Veen, Kooij, et al., 2010). Various strategies (improving sleep hygiene, making behavioral modifications, adjusting timing or type of stimulant, and switching to an alternative ADHD treatment) have been suggested to help make it easier for patients with ADHD to fall asleep (Cortese, Brown, et al., 2013; Kratochvil, Lake, et al., 2005). Complementary pharmacological treatments to consider include the following: melatonin (1–3 mg), clonidine (0.1–0.3 mg; Prince, Wilens, et al., 1996), diphenhydramine (25–50 mg), trazodone (25–50 mg), and mirtazapine (3.75–15 mg). Recently, there has been growing interest in the use of melato-

CARDIOVASCULAR HISTORY	Yes	No	COMMENT
Personal history			
Congenital or acquired cardiac disease?			
Coronary artery disease?			
Chest pain?			
Palpitations?			
Shortness of breath?			
Dizziness?			
Syncope?			
Change in exercise tolerance or tolerance to usual physical activities?			
Family history (< 30 years of age)			
Early myocardial infarction?			
Cardiac death?			
Significant arrhythmia(s)?			
Long QT syndrome?			
Objective			
Baseline (off medication) blood pressure and heart rate within normal limits			

FIGURE 35.1. A strategy to screen for cardiovascular symptoms in adults with ADHD. This tool may be useful for screening at initial assessment and prior to initiation of medication(s) used to treat ADHD. As a part of follow-up visits, this tool may be used as one way to monitor ongoing treatment as well as prior to changing medication dose(s). During ongoing treatment, we encourage clinicians to inquire about current cardiovascular symptoms, measure pulse and blood pressure, as well as changes to family history. If positive on an item, recommend referral to primary care physician or cardiology for further assessment prior to initiating medications. From Massachusetts General Hospital Cardiovascular Screen. Copyright by Timothy E. Wilens. Adapted by permission.

nin, a hormone secreted by the pineal gland that helps regulate circadian rhythms (Macchi & Bruce, 2004) to address sleep problems in children (Smits, van Stel, et al., 2003). Melatonin used alone (Tjon Pian Gi, Broeren, et al., 2003) and in conjunction with sleep hygiene techniques (Weiss, Wasdell, et al., 2006) appears to improve sleep in youth with ADHD. In these two well-designed but small studies, the most concerning adverse events included migraine (n = 1), nightmares (n = 1), and aggression (n = 1). Although not yet studied, another consideration is ramelteon, a synthetic melatonin receptor agonist (Zlotos, 2005).

Medication Interactions with Stimulants

The interactions of stimulants with other prescription and nonprescription medications are generally mild and not a major source of concern (Markowitz, Morrison, et al., 1999; Markowitz & Patrick, 2001). Concomitant use of sympathomimetic agents (e.g., pseudoephedrine) may potentiate the effects of both medications. Concurrent use of antihistamines may diminish the effects of stimulants. Likewise, excessive intake of caffeine may potentially compromise the effectiveness of the stimulants and exacerbate sleep difficulties. Although ad-

ministering stimulants with ATMX is common clinical practice, and appears well tolerated and effective based on open pediatric samples (Hammerness, Georgiopoulos, et al., 2009; Wilens, Hammerness, et al., 2009), this combination has not been tested systematically in adults. Although data on the coadministration of stimulants with tricyclic antidepressants (TCAs) suggest little interaction between these compounds (Cohen, Prince, et al., 1999), careful monitoring is warranted when prescribing stimulants with either TCAs or anticonvulsants. The stimulants and serotonin reuptake inhibitors can be coadminstered. For instance, Weiss and Hechtman (2006) studied AMP alone and in combination with paroxetine, and the combination was well tolerated. Coadministration of monoamine oxidase inhibitors (MAOIs) and stimulants may result in a hypertensive crisis and be potentially life-threatening. In fact, coadministration of stimulants with MAOIs is the only true contraindication, although this combination has been cautiously used in adults with treatment-resistent depression (Feinberg, 2004).

Despite the increasing use of stimulants for adults with ADHD, up to 50% may not respond, have untoward side effects, or manifest comorbidity that stimulants may exacerbate or ineffectively treat (Biederman, Faraone, et al., 1993; Shekim, Asarnow, et al., 1990). To date, a reported 51 studies of nonstimulant medications (N = 5,488 subjects) have included antidepressants, alpha agonists, amino acids, wake-promoting agents, and experimental agents for the treatment of ADHD in adults.

ATOMOXETINE

ATMX was the first medication approved by the FDA specifically to treat ADHD in adults (see Table 35.4). Unlike the stimulants, ATMX (Strattera) is unscheduled; therefore, clinicians can prescribe refills. ATMX acts by blocking the NE reuptake pump on the presynaptic membrane, thus increasing the availability of intrasynaptic NE, with little affinity for other monoamine transporters or neurotransmitter receptors. (Arnsten & Pliszka, 2011) In addition to prominent effects of ATMX on NE reuptake inhibition, preclinical data also show that the noradrenergic presynaptic reuptake protein regulates DA in the frontal lobes, and that by blocking this protein, ATMX increases DA in the frontal lobes (Bymaster, Katner, et al., 2002).

ATMX has been studied in at least eight controlled and seven open studies of 3,525 adult subjects. Initial 10-week studies of ATMX in 536 subjects resulted in reductions from baseline in CAARS scores of approximately 30% (vs. 20% for placebo), with similar reductions in symptoms of inattention and hyperactivity–impulsivity. More recently, ATMX was studied in two large, short-term trials, demonstrating continued efficacy for ADHD in adults (Sutherland, Adler, et al., 2012; Young, Sarkis, et al., 2011).

Longer-term data also suggest ongoing effectiveness of ATMX in adults with ADHD. In a large (384 adults), controlled, 6-month study of ATMX, significant findings compared to placebo were noted acutely (6 weeks) and at the 6-month endpoint (Adler, Spencer, et al., 2009). In this study, outcome in ADHD improved in more than 30% of symptoms compared to baseline at up to 221 weeks and no new long-term adverse effects emerged. Similarly, Marchant, Reimherr, and colleagues (2011) reported a study of 384 adults treated openly for up to 156 weeks, in which responders had significant reductions in ADHD symptoms and emotionality. Interestingly, 39% of ATMX subjects enrolled in a double-blind nonresponder study became responders during the open-label treatment (Marchant, Reimherr, et al., 2011).

ATMX is rapidly absorbed following oral administration, and food does not appear to affect absorption. ATMX's C_{max} (maximal concentration) is 1–2 hours after dosing. ATMX is primarily metabolized via the hepatic cytochrome P450 system through the 2D6 enzyme to 4-hydroxyatomoxetine (Ring, Gillespie, et al., 2002). There are a number of alternative metabolic pathways including the 2C19 enzyme. Although ATMX is metabolized by 2D6, it does not appear to either induce or inhibit 2D6 activity.

It is recommended that ATMX be initiated slowly at 0.5 mg/kg/day for 2 weeks and increased over a month to target dose of 1.2 mg/kg/day. Current dosing guidelines for ATMX recommend maximum dosage of 1.4 mg/kg/day or 100 mg/day, though increases up to 1.8 mg/kg/day may be necessary in refractory cases. Youth with ADHD treated with doses of ATMX up to 3 mg/kg/day appeared to tolerate the increased dose much as they did "regular dosing" and experienced similar reductions in ADHD symptoms; Kratochvil, Michelson, and colleagues (2007) concluded that while higher doses appeared to be well tolerated, they did not appear to confer a clinical advantage. Moreover, Dunn, Turgay, and colleagues (2005) found that while subjects with a plasma level of ATMX greater than 800 ng/ml experienced more robust responses, they also had more

TABLE 35.4. Representative Clinical Studies of Nonstimulants in Adults with ADHD

Study (year)	*N*	Medication	Design	Duration	Total dose (mean and/or range)	Outcome	Comments
Wood, Reimherr, et al. (1982)	8	L-DOPA (+ carbidopa)	Open	3 weeks	625 mg/day (62.5 mg/day)	No benefit	Side effects: nausea, sedation; low doses.
Wender, Wood, et al. (1983)	22	Pargyline	Open	6 weeks	30 mg/day (10–50 mg/day)	68% response rate	Delayed onset; brief behavioral action.
Wender, Wood, et al. (1985)	11	Deprenyl	Open	6 weeks	30 mg/day	66% response rate	Amphetamine metabolite; 2 dropouts.
Wood, Reimherr, et al. (1985)	19	Phenylalanine	Double-blind, placebo crossover	2 weeks	587 mg/day	46% response rate (15% placebo)	Transient mood improvement only.
Mattes (1986)	13	Propanolol	Open	Mean = 9 weeks (3–50 weeks)	528 mg/day (40–640 mg/day)	85% response rate	Part of "temper" study.
Reimherr, Wender, et al. (1987)	12	Tyrosine	Open	8 weeks	50–150 mg/kg/day	66% response rate	14-day onset of action; tolerance developed; 4 dropouts.
Shekim, Masterson, et al. (1989)	18	Nomifensine maleate	Open	4 weeks	50–300 mg/day	94% response rate	Immediate response; one patient with allergic reaction.
Shekim, Antun, et al. (1990)	8	*S*-adenosyl-L-methionine	Open	4 weeks	≤2,400 mg/day	75% response rate	Mild adverse effects.
Wender & Reimherr (1990)	19	BPR	Open	6–8 weeks	359 mg/day (150–450 mg/day)	74% response rate	5 subjects could not tolerate lowest dose and dropped out; 10 subjects improved at 1 year.
Wilens, Biederman, et al. (1995)	37	Desipramine Nortriptyline	Open, retrospective	Mean = 50 weeks	183 mg/day (92 mg/day)	68% response rate	Comorbidity unrelated to response; 60% on stimulants, 84% on concurrent medications; response sustained in 54% of patients.
Adler, Resnick, et al. (1995)	16	Venlafaxine	Open	8 weeks	110 mg/day (25–225 mg/day)	83% response rate	4 subjects on other medications; 4 dropped out; 50% reduction in Sxs.
Hedges, Reimherr, et al. (1995)	18	Venlafaxine	Open	8 weeks	96 mg/day (50–150 mg/day)	50% response rate	Side effects led to 39% dropout rate; study divided by med tolerance.
Findling, Schwartz, et al. (1996)	10	Venlafaxine	Open	8 weeks	150 mg/day (7 of 9) (75–150 mg/day)	70% response rate	Improved anxiety scores; 1 dropout.

Wilens, Biederman, et al. (1996)	43	Desipramine	Double-blind, placebo parallel	6 weeks	147 mg/day	68% response rate (0% placebo)	Comorbidity or levels not related to response.
Ernst, Liebenauer, et al. (1996)	24	Selegiline	Double-blind, placebo parallel	6 weeks	20 mg/day, followed by 60 mg/day	Mild improvement	High placebo response, mild side effects; three arms; 60 mg dose best.
Spencer, Biederman, et al., 1998)	22	(A) tomoxetine	Double-blind, placebo crossover	7 weeks	76 mg/day	50% response rate (9% placebo)	Noradrenergic agent; well tolerated.
Wilens, Biederman et al. (1999)	32	ABT 418	Double-blind, placebo crossover	7 weeks	75 mg/day	40% response rate (13% placebo)	Nicotinic analog; attentional symptoms improved preferentially
Taylor & Russo (2000)	22	Modafinil *d*-AMPH	Double-blind, placebo crossover	7 weeks	207 mg/day 22 mg/day	48% response rate 48% response rate	Improved neuropsychological functioning with both Tx.
Cephalon, Inc. (2000)	113	Modafinil	Double-blind, placebo crossover	7 weeks	100 & 400 mg/day	No difference versus placebo	Cephalon, Inc. report.
Taylor & Russo (2001)	17	Guanfacine *d*-AMPH	Double-blind, placebo crossover	7 weeks	1 mg/day (0.25–2 mg/day) 10 mg/day (2.5–20 mg/day)	Both Tx improved versus placebo	Well tolerated; neuropsychological profile improved.
Wilens, Spencer, et al. (2001)	40	BPR SR	Double-blind, placebo parallel	6 weeks	362 mg/day (100–400 mg/day)	52% response rate (11% placebo)	Delayed onset of action; well tolerated.
Upadhyaya, Brady, et al. (2001)	10	Venlafaxine	Open	12 weeks	75–300mg/day	Improved ADHD and alcohol use	Substance use disorder study; 4/10 subjects completed 12 weeks.
Kuperman, Perry, et al. (2001)	30	BPR SR MPH	Double-blind, placebo parallel	7 weeks	Maximum 300 mg/day Maximum 0.9 mg/kg/day	64% response rate BPR 50% response rate MPH (27% placebo)	Not statistically significant versus placebo; n = 8–11/group.
Levin, Evans, et al. (2002)	11	BPR	Single-blind	12 weeks	400 mg/day (250–400 mg/day)	47% response rate	Cocaine abusers; reduced cocaine use.
Wilens, Prince, et al. (2003)	36	BPR SR	Open	6 weeks	370 mg/day (200–400 mg/day)	70% response rate by CGI	Bipolar + ADHD in adults; no manic activation.
Michelson, Adler, et al. (2003); Simpson & Plosker (2004)	536	ATX	Double-blind, placebo parallel	10 weeks	60, 90, or 120 mg/day	58% response rate	Combination of two separate multisite studies; improved functioning and less disability.

(continued)

TABLE 35.4. *(continued)*

Study (year)	*N*	Medication	Design	Duration	Total dose (mean and/or range)	Outcome	Comments
Adler, Spencer, et al. (2005, 2008); Marchant, Reimherr, et al. (2011)	384	ATX	Open	Mean = 40 weeks	99 mg/day	Decrease on CAARS 33.2%	Continuation of Michelson, Adler, et al. (2003); safety and efficacy established in adults with ADHD.
Wilens, Haight, et al. (2005)	162	BPR XL	Double-blind, placebo parallel	8 weeks	393 mg/day	53% response rate ADHD-RS (31% placebo)	Medications provided benefit throughout day versus placebo; no serious or unexpected AEs.
Wilens, Waxmonsky, et al. (2005)	6 adult	Donepezil	Open	12 weeks	9 mg/day (2.5–10 mg/day)	55% improved on CGI	Not well tolerated.
Reimherr, Hedges, et al. (2005)	47	BPR SR	Double-blind, placebo parallel	6 weeks	298 mg/day (100–400 mg/day)	41% response rate on CGI (22% placebo)	Not statistically significant versus placebo.
Adler, Dietrich, et al. (2006)	218	ATX	Double-blind, multicenter	—	80 mg every day versus 40 mg twice daily	Both treatments efficacious	Changes in dosing are not associated with greater AE's or safety risks. twice daily treatment had greater effect.
Wilens, Verlinden, et al. (2006)	11	ABT-089	Double-blind, placebo crossover	8 weeks	4, 8 and 40 mg/day	ABT-089 improved CGI and CAARS	Nicotinic partial agonist; no safety or side effect profiles were observed; study interrupted.
Biederman, Mick, et al. (2006a)	28	Galantamine	Double-blind, placebo parallel	12 weeks	20 mg/day (8–24 mg/day)	22% response rate on CGI (11% placebo)	Study did not support the use of galantamine; no statistically or clinically significant greater reduction in ADHD symptoms.
Levin, Evans, et al. (2006)	98	MPH SR BPR SR	Double-blind, placebo parallel (MPH, BPR)	12 weeks	10–80 mg/day 100–400mg/day	Response rates: 34% MPH and 49% BPR, (46% placebo)	SUD study; MPH & BPR did not provide a clear advantage over placebo (AARS).
Wilens, Klint, et al. (2008)	126	NS2359	Double-blind, placebo parallel	8 weeks	0.5 mg/day	33% response rate on ADHD (27% placebo)	Triple amine reuptake inhibitor; no serious AEs; some attentional improvement on neuropsychological testing; ADHD RS pNS.
Wilens, Adler, et al. (2008)	147	ATX	Double-blind, placebo parallel	12 weeks	90 mg/day (25–100 mg/day)	Reduced ADHD symptoms and less heavy drinking	Substance use disorder study; no serious AEs or specific drug–drug reactions related to current alcohol

							use; no effect on relapse rate versus placebo.
Levin, Mariani, et al. (2009)	20	ATX	Open	12 weeks	80 mg/day (20–100 mg/day)	50% response rate on AARS	Cocaine abusers; little to no effect on cocaine abuse.
Johnson, Cederlund, et al. (2010)	20	ATX	Open	10 weeks–1 year	85 mg/day (40–100 mg/day)	50% response rate on CGI	Side effects led to 95% dropout rate by 10 weeks; only one patient continued treatment for 1 year.
Adler, Liebowitz, et al. (2009)	442	ATX	Double-blind, placebo parallel	14 weeks	83 mg/day (40–100 mg/day)	ATX improved sxs of ADHD and anxiety	Comorbid social anxiety disorder; rates of insomnia, nausea, dry mouth, and dizziness were higher with ATX than with placebo.
Adler, Spencer, et al. (2009)	501	ATX	Double-blind, placebo parallel	6 months	85 mg/day (25–100 mg/day)	ATX was effective at 10 weeks and 6 months	Long-term study; AEs similar to previous trials.
Wilens, Prince, et al. (2010)	32	BPR SR	Open	6 weeks	100–400 mg/day	66% response rate on ADHD RS	Substance use disorders study; 19/32 completed 6-week protocol; no clinically significant reductions observed in self-report of substance use disorder or CGI substance use disorder scores.
Surman, Hammerness, et al. (2010)	45	ATX	Open	6 weeks	79 mg/day (50–20 mg/day)	64% response rate on CGI and AISRS	ADHD-NOS population, similar outcome versus full ADHD; no serious AEs.
Adler, Guida, et al. (2010)	18	ATX	Open	10 weeks	25–120 mg/day	Improved ADHD, reduced cravings	Substance use disorder study; 12/18 completed.
Marchant, Reimherr, et al. (2011)	384	ATX	Open	Mean = 60 weeks	50–160 mg/day	Improved CAARS and CGI scores	Those with emotion dysregulation had greater improvement.
Takahashi, Takita, et al. (2011)	45	ATX	Open	8 weeks	114 mg/day (40–120 mg/day)	Improved CAARS and CGI scores	No serious AEs were reported.
Young, Sarkis, et al. (2011)	502	ATX	Double-blind, placebo-controlled	24 weeks	90 mg/day (40–100 mg/day)	68% response rate (42% placebo)	AEs overall and for on-label or slow titration to ATX were similar and consistent with previous adult ATX studies.

(continued)

TABLE 35.4. *(continued)*

Study (year)	*N*	Medication	Design	Duration	Total dose (mean and/or range)	Outcome	Comments
Apostol, Abi-Saab, et al. (2012)	171	ABT-089	Double-blind, placebo crossover	Two 4-week treatment periods separated by a 2-week washout	2, 5, 15, and 40 mg/day 40 mg twice daily	Significant improvements compared to placebo	Side effects: headache, upper respiratory tract infection, irritability, insomnia, and hasopharyngitis.
Arnold, Feifel, et al. (2014)	330	Modafinil	Double-blind, placebo-controlled	9 weeks	225–510 mg/day	No benefit on ADHD Sxs	Drug was reasonably tolerated and some observations among patients who completed warrant further investigation.
Manor, Ben-Hayun, et al. (2012)	120	Metadoxine ER	Double-blind, placebo-controlled	6 weeks	1,400 mg/day	Metadoxine improved CAARS ADHD symptom score, rate of response, TOVA and AAQOL score	Improvement in ADHD symptoms were seen as early as 2 weeks after receiving medication.
Sutherland, Adler, et al. (2012)	241	ATX Buspirone	Double-blind, placebo-controlled	8 weeks	91 and 90 mg/day ATX/buspirone and ATX, respectively	Response rates of ATX (69%) &ATX/buspirone (78%)	Similar outcomes of ATX and buspirone versus ATX monotherapy.
Bain, Robieson, et al. (2013)	243	ABT-894	Double-blind, placebo crossover	Two 4-week treatment periods separated by a 2-week washout	1, 2, and 4 mg/day or 4 mg two times a day	Significantly reduced ADHD symptoms compared to placebo	Five patients did not receive study drug and 36 patients discontinued treatment.
Total (*N* = 51)	*N* = 5,488 6–536 (range)	BPR: 9 ATX: 15 Others: 27	Double-blind: 27 Single-blind: 1 Open: 23	2 weeks–1 year	BPR: 100–450 mg/day ATX: 25–320 mg/day	Variable response	Some delay in therapeutic response—may be related to titration schedule. Response rates typically less than stimulants.

Note. Response rate refers to subject reported much to very much improved (i.e., by clinical global improvement) or with clinically significant reduction in symptoms on ADHD rating scales. AARS, Adult ADHD Rating Scale; AE, adverse event; AISRS, Adult ADHD Investigator Symptom Rating Scale; ATX, atomoxetine; BPR, bupropion; CAARS, Conners' Adult ADHD Rating Scale; CGI, Clinical Global Impression; *d*-AMPH, dextroamphetamine; Dx, diagnosis; MPH, methylphenidate; NOS, not otherwise specified; RS, rating scale, SR/XL, sustained release; Sx, symptom; Tx, treatment; TOVA, Test of Variables of Attention; AAQOL, Adult ADHD Quality-of-Life Scale.

side effects. Extensive testing was undertaken to look at the ability of patients with relatively slow metabolic activity at 2D6 (approximately 7% of the sample) to metabolize ATMX. These pediatric studies indicate that while patients with slow metabolizer status experienced increased rates of common side effects, they were generally able to tolerate ATMX. In such situations or when ATMX is coadministered with medication known to inhibit 2D6 (e.g., fluoxetine, paroxetine), clinicians should consider reducing the dose. In addition to the treatment of both inattention and hyperactivity–impulsivity in adults with ADHD, ATMX may be particularly useful when anxiety, mood, or tics co-occur with ADHD. For example, in a large, 14-week, multisite study of ATMX in adults with ADHD and social anxiety disorder, Adler, Liebowitz, and colleagues (2009) reported clinically significant effects on both ADHD and anxiety.

Although untested because of its lack of abuse liability (Heil, Holmes, et al., 2002), ATMX may be particularly useful for adults with substance use issues. For instance, in a 12-week controlled trial, Wilens, Adler, and colleagues (2008) demonstrated that treatment with ATMX in recently abstinent alcoholics was associated with reduced ADHD symptoms and reduced drinking, although absolute abstinent rates were unaffected. Moreover, ATMX has not been reported to have significant or serious drug interactions with alcohol or marijuana (Adler, Wilens, et al., 2009). Since pharmacotherapy of ADHD is often chronic, missed doses of medication can be expected and may be problematic.

Clinically, ATMX is often prescribed in conjunction with stimulants. Although the safety, tolerability, and efficacy of this combination have not been fully studied, reports suggest that this combination may be well tolerated and effective (Brown, 2004; Hammerness, Georgiopoulos et al., 2009; Wilens, Hammerness, et al., 2009). Therefore, although the full safety of administering stimulants and ATMX together has not been fully established, there are good data from which to extrapolate, and clinicians must balance the risks and benefits in each patient.

Although generally well tolerated, the most common side effects observed with ATMX appear to reflect increased noradrenergic tone. The most common side effects of ATMX include dry mouth, insomnia, nausea, decreased appetite, constipation, decreased libido, dizziness, and sweating (Michelson, Adler, et al., 2003). Furthermore, 9.8% of males experienced difficulty attaining or maintaining erections. During these trials, extensive laboratory testing indicated that ATMX causes no organ toxicity, and there were no discontinuations in the clinical trials due to abnormal laboratory tests. However, there have been reports of hepatotoxicity in two patients taking ATMX (out of 2 million patients exposed to ATMX). Both patients recovered upon discontinuation of ATMX. ATMX should be discontinued in patients with jaudice, and patients should contact their doctors if they develop jaundice, pruritis, dark urine, right-upper-quadrant tenderness, and/or unexplained "flu-like" symptoms. Laboratory monitoring outside of routine medical care does not appear necessary. While the impact of ATMX on the cardiovascular system appears minimal (Habel, Cooper, et al., 2011; Wernicke, Faries, et al., 2003), ATMX was associated with mean increases in heart rate of 6 beats per minute, and increases in systolic and diastolic blood pressure of 1.5 mm Hg. Adults should have their vital signs checked prior to initiating treatment with ATMX and periodically thereafter.

ALPHA-ADRENERGIC AGONISTS

Clonidine, an imidazoline derivative with alpha-adrenergic agonist properties, has been used primarily in the treatment of hypertension (Roden, Nadeau, et al., 1988). At low doses, it appears to stimulate inhibitory, presynaptic autoreceptors in the central nervous system (CNS) (Buccafusco, 1992). In 2010, the FDA approved an ED oral formulation of clonidine, clonidine ER (Kapvay) as a treatment for ADHD in youth ages 6–17 years (*Physicians' Desk Reference*, 2013). This formulation is approved both as monotherapy and as adjunctive treatment with stimulants. Although clonidine reduces symptoms of ADHD (Hunt, Inderaa, et al., 1986), its overall effect is less than that of the stimulants (Connor, Fletcher, et al., 1999), and likely smaller than ATMX, TCAs, and bupropion. Clonidine appears to be particularly helpful in patients with ADHD and comorbid conduct disorder (CD) or oppositional defiant disorder (ODD) (Connor, Barkley, et al., 2000; Hazell & Stuart, 2003; Schvehla, Mandoki, et al., 1994), tic disorders (Singer, Brown, et al., 1995; Steingard, Biederman, et al., 1993), ADHD-associated sleep disturbances (Prince, Wilens, et al., 1996; Wilens, Biederman, et al., 1994), and may reduce anxiety and hypervigilance in traumatized children (Donnelly, 2003).

Clonidine is a relatively short-acting compound with a plasma half-life ranging from approximately 5.5 hours

(in children) to 8.5 hours (in adults). Clonidine ER is usually initiated at a dose of 0.1 mg (at bedtime) for several days and titrated up to a maximum recommended dose of 0.2 mg twice daily. Immediate-release clonidine usually is initiated at the lowest manufactured dose of one-half or one-fourth of a 0.1-mg tablet. Usual daily doses ranges from 3 to 10 mcg/kg given generally in divided doses, twice daily (bid), three times daily (tid), or four times daily (qid), and there is a transdermal preparation. The most common short-term adverse effect of clonidine is sedation, which tends to subside with continued treatment. It can also produce, in some cases, hypotension, dry mouth, vivid dreams, depression, and confusion. A recent summary of the safety of Kapvay is available at *www.fda.gov/downloads/advisorycommittees/committeesmeetingmaterials/pediatricadvisorycommittee/ucm319363.pdf*. Overdoses of clonidine in children under 5 years of age may have life-threatening consequences (Klein-Schwartz, 2002). Since abrupt withdrawal of clonidine has been associated with rebound hypertension, slow tapering is advised (Leckman, Ort, et al., 1986; Nami, Bianchini, et al., 1983). In addition, extreme caution should be exercised with the coadministration of clonidine with beta-blockers or calcium channel blockers (Jaffe, Livshits, et al., 1994). Although concerns about the safety of coadministration of clonidine with stimulants have been debated (Wilens, Spencer, et al., 1999), recent data support the tolerability, safety, and efficacy of this combination (Childress & Sallee, 2012). Current guidelines are to monitor blood pressure when initiating and tapering clonidine, but electrocardiographic (ECG) monitoring is not usually necessary (Gutgesell, Atkins, et al., 1999).

Guanfacine, the most selective alpha$_{2A}$-adrenergic agonist currently available, appears to act by mimicking NE binding in the prefrontal cortex (Arnsten, 2010). In 2009, an ED formulation, Guanfacine ER (Intuniv), was approved by the FDA for the treatment of ADHD in youth ages 6–17 years as monotherapy or as adjunctive treatment with stimulants (*Physicians' Desk Reference*, 2013). Guanfacine ER is usually started at 1 mg daily at bedtime and titrated to a maximum dose of 4 mg. Possible advantages of guanfacine over clonidine include less sedation and longer duration of action, and since it has little affinity for the brain stem imidazoline I1 receptors, it may have a milder cardiovascular profile (Arnsten, 2010). Recent information from the FDA about postmarketing experience with Intuniv is available at *www.fda.gov/downloads/advisorycommittees/committeesmeetingmaterials/pediatricadvisorycommittee/ucm255105.pdf*. Guanfacine treatment is associated with minor, clinically insignificant decreases in blood pressure and pulse rate. The adverse effects of guanfacine include sedation, irritability, and depression. Several cases of apparent guanfacine-induced mania have been described, but the impact of guanfacine on mood disorders remains unclear (Horrigan & Barnhill, 1999). Alpha-adrenergic medications may be particularly useful in youth with primarily a hyperactive–impulsive and/or aggressive component (Sallee, Connor, et al., 2013).

However, there is a dearth of data on using the alpha agonists in adults with ADHD. Taylor and Russo (2001) reported results from 17 adults treated with either *d*-AMP or guanfacine IR and found similar reductions in ADHD symptoms compared to placebo, with a similar response between active treatments. To date, no studies of clonidine for adults with ADHD have been completed. Given the paucity of efficacy data and concerns about sedative and hypotensive effects, use of alpha agonists in adults remains unclear.

ANTIDEPRESSANTS

Bupropion, a novel-structured antidepressant, has been reported to be moderately helpful in reducing ADHD symptoms in children (Casat, Pleasants, et al., 1987). There have been at least three open trials and six controlled trials using bupropion in adults with ADHD. In a 6-week, double-blind, placebo-controlled trial, bupropion SR at 200 mg BID (final mean dose 386 mg/day) resulted in a 42% reduction of symptoms in the ADHD Rating Scale (ADHD-RS), with 52% of subjects treated with bupropion considered responders. Similar results were found by Reimherr, Hedges, and colleagues (2005) and by Wilens, Haight, and colleagues (2005) using an alternative once-daily preparation. Dosing of 400–450 mg (SR or extended release [XL] preparations) is usually necessary for best efficacy. Side effects include insomnia, edginess, and a theoretical risk for seizures with IR preparations. Despite the small number of adults studied, bupropion may be helpful in ADHD, particularly when associated with comorbid depression (Daviss, Bentivoglio, et al., 2001), substance abuse (Levin, Evans, et al., 2002; Wilens, Prince, et al., 2010), bipolar disorder (Wilens, Prince et al., 2003), or in adults with cardiac abnormalities (Gelenberg, Bassuk, et al., 1991). Bupropion appears to be more stimulating

than other antidepressants and is associated with higher rate of drug-induced seizures than other antidepressants (Gelenberg, Bassuk, et al., 1991). These seizures appear to be dose-related (> 450 mg/day) and elevated in patients with bulimia or a previous seizure history. Bupropion has also been associated with excitement, agitation, increased motor activity, insomnia, tremors, and tics.

Despite an extensive experience in children and adolescents there are only two studies of TCAs in adult ADHD (Wilens, Biederman, et al., 1995). Compared to the stimulants, TCAs have negligible abuse liability, single daily dosing, and efficacy for comorbid anxiety and depression. However, given concerns about potential overdose and the availability of ATMX, use of the TCAs has been significantly curtailed. Generally, TCA daily doses of 50–250 mg are required, with a relatively rapid response to treatment (i.e., 2 weeks) when the appropriate dose is reached. TCAs should be initiated at 25 mg and slowly titrated upward within dosing- and serum-level parameters until an acceptable response or intolerable adverse effects are reported. Common side effects of the TCAs include dry mouth, constipation, blurred vision, weight gain, and sexual dysfunction. While cardiovascular effects of reduced cardiac conduction, elevated blood pressure, and elevated heart rate are not infrequent, if monitored, they rarely prevent treatment. Because serum TCA levels are variable, they are best used as guidelines for efficacy and to reduce CNS and cardiovascular toxicity.

The MAOI antidepressants have also been studied for the treatment of ADHD. Whereas open studies with pargyline and deprenyl in adult ADHD indicated moderate symptom reduction (Wender, Wood, et al., 1983, 1985), a controlled trial of selegeline (deprenyl) yielded less enthusiastic findings, with researchers reporting dose-dependent symptom reduction in ADHD on selegeline (Ernst, Liebenauer, et al., 1996) that was not significant when compared to a high placebo response. Although a pilot child-based study demonstrated efficacy of the reversible MAOI moclobemide (Trott, Friese, et al., 1992), data on its effectiveness for ADHD are limited to case reports (Myronuk, Weiss, et al., 1996; Vaiva, De Lenclave, et al., 2002). The concerns about diet- or medication-induced hypertensive crisis limit the usefulness and safety of these medications, especially in a group of patients with ADHD who were vulnerable to impulsivity. Additionally, other adverse effects associated with the MAOIs include agitation or lethargy, orthostatic hypotension, weight gain, sexual dysfunction, sleep disturbances, and edema, often leading to the discontinuation of these agents (Gelenberg, Bassuk, et al., 1991).

MISCELLANEOUS MEDICATIONS

Modafinil, approved for the treatment of narcolepsy (U.S. Modafinil in Narcolepsy Multicenter Study Group, 1998), has generated interest as a potential treatment for ADHD. Although controlled trials on the use of modafinil in children and adolescents with ADHD demonstrated efficacy (Biederman, Swanson, et al., 2005; Wigal, Biederman, et al., 2006), it did not receive approval from the FDA (2007) due to safety concerns specifically related to possible serious skin reactions, including erythema multiforme (EM), Stevens–Johnson syndrome (SJS), and toxic epidermal necrolysis (TEN). Although one double-blind, placebo-controlled crossover design in 22 adults suggested reductions in ADHD symptoms (Taylor & Russo, 2000), results of large, company-sponsored multisite trials in adults with ADHD have not shown the same results (Table 35.4). Recently, Arnold, Feifel, and colleagues (2014) reported on a multicenter (18 locations) randomized, double-blind treatment study of modafinil versus placebo for 330 adults with ADHD. The study lasted 9 weeks, and although a significant difference in ADHD symptoms was not found for those on medication versus placebo, results from individual subjects warrant further investigation.

Given the cognition-enhancing properties of nicotine (Rezvani & Levin, 2001), nicotinic agents have been studied in the treatment of ADHD. Whereas smaller crossover studies of nicotinic analogues with either full or partial agonistic properties demonstrated efficacy in adults with ADHD (Apostol, Abi-Saab, et al., 2012; Wilens, Biederman, et al., 1999; Wilens, Verlinden, et al., 2006), on follow-up, larger, multisite, parallel design studies failed to show a significant effect of this compound on reducing ADHD symptomatology (Wilens, Gault, et al., 2011) and the role of nicotinic agents remains investigational.

Trials with the amino acids were in part undertaken with the assumptions that ADHD may be related to a deficiency in the catecholaminergic system, and that administration of precursors of these systems would reverse these deficits (Reimherr, Wender, et al., 1987; Wood, Reimherr, et al., 1982, 1985). In these studies, transient improvement in ADHD symptoms was lost

after 2 weeks of treatment. Therefore, amino acids have a limited role in the treatment of adults with ADHD. Prohistaminergic agents, although appealing given the endogenous histamine effects on the attention arousal systems, have been disappointing (Herring, Wilens, et al., 2012).

Although compelling based on efficacy in Alzheimer's disease and some positive initial experience in patients with ADHD (Wilens, Biederman, et al., 2000) trials in ADHD adults with donepezil (Wilens, Waxmonsky, et al., 2005) and galantamine (Biederman, Mick, et al., 2006a) were negative. At this time, no data support the use of these cholinergic agents in the treatment of ADHD. Recently, Surman, Hammerness, and colleagues (2013) openly treated 34 adults with ADHD with the *N*-methyl-D-aspartate (NMDA) receptor antagonist memantine. In this pilot study memantine, titrated to a maximum dose of 10 mg bid, was generally well tolerated and resulted in improvements in measures of ADHD symptoms and neuropsychological measures. These encouraging but preliminary results warrant controlled study.

Recently, MG01CI, an ER formulation of metadoxine, has been studied in adults with ADHD. Metadoxine is an ion-pair salt of pyridoxine (vitamin B_6) and 2-pyrrolidone-5-carboxylate, used in Europe for over 30 years in the treatment of acute alcohol intoxication and withdrawal. In a short-term, controlled trial with MG01CI, subjects experienced reductions in ADHD symptoms, as well as improved neuropsychological measures and overall functioning (Manor, Ben-Hayun, et al., 2012).

SUGGESTED MANAGEMENT STRATEGIES

Having been diagnosed with ADHD, the adult needs to know the risks and benefits of pharmacotherapy, the availability of alternative treatments, and the likely adverse effects. Patient expectations need to be explored, and realistic goals of treatment need to be clearly delineated (Haavik, Halmoy, et al., 2010). Likewise, the clinician should review with the patient the various pharmacological options available and that each will require systematic trials of the anti-ADHD medications for a reasonable duration and at clinically meaningful doses. Treatment-seeking adults with ADHD who manifest substantial psychiatric comorbidity, have residual symptomatology with treatment, or report psychological distress related to their ADHD (i.e., self-esteem issues, self-sabotaging patterns, interpersonal disturbances) should be directed to appropriate psychotherapeutic intervention with clinicians knowledgeable in ADHD treatment.

Adults with ADHD often require more comprehensive treatment for their ADHD given the sequalae associated with a chronic disorder, its effect on psychological development, and residual psychiatric and ADHD symptoms, even with aggressive pharmacotherapy. To this end, the use of structured cognitively based psychotherapies appears helpful especially when used conjointly with pharmacotherapy (Safren, Sprich, et al., 2010; Solanto, Marks, et al., 2010). For adults considering advanced schooling, educational planning and alterations in the school environment may be necessary.

The stimulant medications and ATMX are FDA-approved and are the most rigorously investigated pharmacotherapies (see Tables 35.2, 35.3, and 35.4) and are considered the first-line therapy for ADHD in adults. Although there are no evidence-based guidelines in selecting a first choice of medication for adults with ADHD, clinicians ought to make their recommendations after considering issues of comorbidity, tolerability, efficacy, and duration of action (Greenhill, Pliszka, et al., 2002; Kooij, Bejerot, et al., 2010; Weiss & Weiss, 2004). The European Network Adult ADHD published a consensus outlining guidelines for ADHD treatment with stimulants. The guidelines recomend that the severity of ADHD and comorbid disorders should be the first guide to select treatments, with stimulants being the medication of choice. Long-lasting, ER formulations are preferred for reasons of adherence to treatment, protection against abuse, avoidance of rebound symptoms, and provision of symptom relief throughout the day, without the need for multiple doses. Every few days the dose may be increased to optimize response. Frequently, patients benefit from adding AMP-IR or MPH-IR in combination with longer-acting preparations in order to sculpt the dose to the patient's individual needs (Adler, Reingold, et al., 2006), although the efficacy of this practice is not well studied. Additionally, psychotherapy is recommended in combination with stimulant medication treatment in order to relieve additional impairements (Kooij, Bejerot, et al., 2010).

Consideration of another stimulant or ATMX is recommended if an ADHD adult is unresponsive or has intolerable side effects to the initial medication. Given their pharmacodynamic differences (Wilens & Spencer, 1998), if a MPH product was initially selected, then moving to an AMP-based medication is appropriate. Although some adults are able to take ATMX once daily, many adults benefit from dosing twice daily (Mi-

chelson, Adler, et al., 2003). Patients must also be made aware that the full benefits of ATMX may not occur for several weeks, and they may not "feel" anything like what they may have felt with the stimulants. Monitoring routine side effects, vital signs, and the misuse of the medication is warranted.

Adult ADHD is a heterogeneous disorder associated with considerable comorbidity with antisocial disorders, anxiety and mood disorders, as well as substance use disorders (Biederman, 2004; Biederman, Faraone, et al., 1993). Adults with ADHD and comorbid mood or anxiety disorders may respond differently to ADHD pharmacotherapy, depending on the clinical state of their co-occurring disorders. The effect of stimulants on comorbid anxiety and depression have not been systematically assessed in adults with ADHD. While it is possible for stimulants to exacerbate anxiety and depression, patients may present with chronic anxiety/demoralization related to their untreated ADHD. Often, in these cases, symptoms of anxiety and demoralization diminish with treatment for their ADHD. Also, one can treat anxiety and ADHD simultaneously. Weiss and Hechtman (2006) found that adults receiving paroxetine or *d*-AMP and paroxetine demonstrated greater reductions in mood and anxiety symptoms compared to adults receiving *d*-AMP or placebo alone. Likewise, patients presenting for treatment of depression may have their ADHD overlooked (Alpert, Maddocks, et al., 1996).

Other concurrent psychiatric disorders also need to be assessed and, if possible, the relationship of the ADHD symptoms with these other disorders should be delineated. In subjects with ADHD plus bipolar mood disorders, for example, the risk of mania and/or hypomania needs to be addressed and closely monitored during treatment of the ADHD (Wilens, Biederman, et al., 2003). In cases such as these, mood stabilization is the priority and usually involves both the introduction of antimanic medications and discontinuation of ADHD treatments, as is done when treating children (Findling, Short, et al., 2007; Scheffer, Kowatch, et al., 2005). Once the mood is euthymic, conservative introduction of anti-ADHD medications along with mood stabilizing agents should be considered.

TREATMENT-REFRACTORY PATIENTS

Despite the availability of various agents for adults with ADHD, there appear to be a number of individuals who either do not respond or cannot tolerate adverse effects of medications used to treat their ADHD. In managing difficult cases, several therapeutic strategies are available. If psychiatric adverse effects develop concurrent with a poor medication response, alternative treatments should be pursued. Severe psychiatric symptoms that emerge during the acute phase may be problematic, irrespective of the efficacy of the medications for ADHD. These symptoms may require reconsideration of the diagnosis of ADHD and careful reassessment of the presence of comorbid disorders. For example, it is common to observe depressive symptoms in an adult with ADHD that are independent of the ADHD or treatment. If reduction of dose or change in preparation (i.e., IR vs. ER) does not resolve the problem, consideration should be given to combined pharmacotherapies, such as stimulants and nonstimulants or alternative treatments. Concurrent nonpharmacological interventions such as behavioral or cognitive therapy may assist with symptom reduction.

In summary, the aggregate literature indicates that pharmacotherapy provides an effective treatment for adults with ADHD. Effective FDA-approved pharmacological treatments for adults with ADHD to date have included the use of the stimulants and ATMX. Bupropion, TCAs, and modafinil have also been studied in the treatment of adult ADHD, and have a role in its treatment. Although interest remains high, data on the efficacy of cognitive enhancers remains minimal, and their role is limited and research-based at this point. The use of agents with catecholaminergic and novel mechanisms (e.g., Metadoxine ER) for ADHD in adults are under way. Structured psychotherapy may be effective when used adjunctively with medications. Further controlled investigations assessing the efficacy of single and combination agents with known and unique mechanisms of action for adults with ADHD are necessary, with careful attention to diagnostics, comorbidity, symptom and neuropsychological outcome, long-term tolerability and efficacy, and use in specific ADHD subgroups.

KEY CLINICAL POINTS

- ✓ The aggregate literature indicates that pharmacotherapy provides an effective treatment for adults with ADHD.
- ✓ Effective FDA-approved pharmacological treatments to date for adults with ADHD have included the use of the stimulants and ATMX.

- ✓ Bupropion, TCAs, and modafinil have also been studied in the treatment of adult ADHD, and have a role in its treatment.
- ✓ Although interest remains high, data on the efficacy of cognitive enhancers remains minimal, and their role is limited and research-based at this point.
- ✓ The use of agents with catecholaminergic and novel mechanisms (e.g., metadoxine ER) for ADHD in adults are under way.
- ✓ Structured (cognitive-behavioral) psychotherapy may be effective when used adjunctively with medications.
- ✓ Further controlled investigations assessing the efficacy of single and combination agents with known and unique mechanisms of action for adults with ADHD are necessary and require careful attention to diagnostics, comorbidity, symptom and neuropsychological outcome, long-term tolerability and efficacy, and use in specific ADHD subgroups.

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