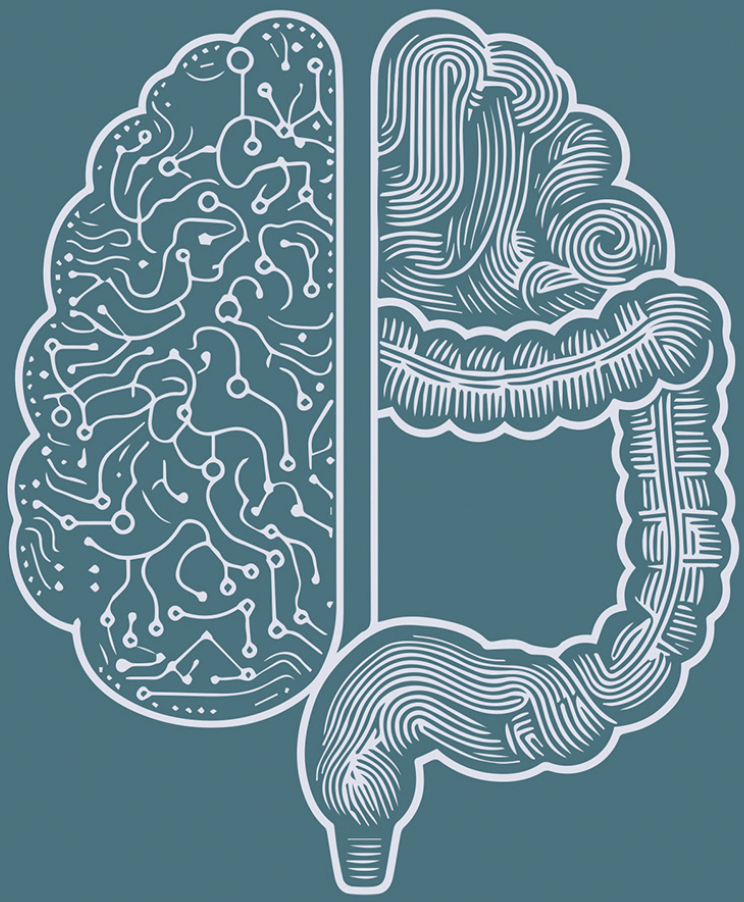
EDGI
Training Project

# COMPREHENSIVE NUTRITION THERAPY FOR CO-OCCURRING GASTROINTESTINAL & EATING DISORDERS



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THE EDGI TRAINING PROJECT

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## Continuing Professional Education

This edition of *Comprehensive Nutrition Therapy for Co-Occurring Gastrointestinal & Eating Disorders* offers readers 12 hours of Continuing Professional Education (CPE) credit expiring December 31, 2028. Readers may earn credit by completing the interactive online quiz at:

https://edgitraining.com/cpe

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# FOUNDATIONS

CHAPTER 1

# Introduction

## Foreword

We all met by word of mouth. We needed to form our own community of peers because none existed for us.

> "Do you know anyone else who works with both eating disorders and gastrointestinal disorders?"
>
> "I need to consult about one of my clients—traditional nutrition therapy just isn't helping."
>
> "I didn't plan to specialize in disordered eating, but almost all of my digestive disorder patients seem to have a troubled relationship with food, so I had to learn quickly."
>
> "I know my own diagnosis radically changed my relationship with food. How can I give better support to clients than I had from my providers?
>
> "This is a really big problem; what do we do next?"

The EDGI Training Project, collective author of *Comprehensive Nutrition Therapy for Gastrointestinal and Eating Disorders,* is a group of clinicians dedicated to helping others find peace with food through their lived experience, clinical practice, and peer-reviewed research. They saw a critical need to form a new community of multidisciplinary professional peers to better serve

their clients and other healthcare providers. In 2022, they committed to create The EDGI Training Project.

The contributors are excited to share their expertise with a growing community of practitioners who recognize the importance of the intersection of gut health and disordered eating. They want eating disorder-informed care to become the standard of practice in the field of gastrointestinal nutrition. Their greatest hope is that individuals who pursue healing from disordered eating can find tangible gastrointestinal symptom relief from professionals without being told, "It's all your eating disorder."

The academic literature on these co-occurring disorders is in its infancy, with no formal clinical treatment guidelines available. Much of the literature is siloed into journals and conferences that are specific to either psychology, nutrition, or gastroenterology, but not all three. As a result, most affected patients live straddled between separate worlds. They have their eating disorder treatment team and their GI medical team, but the providers rarely talk to each other. **Not enough providers are trained in treating both conditions,** and it is our opinion, based on thousands of patient care hours, that **patient outcomes are worse when co-occurring eating disorders and gastrointestinal disorders are not simultaneously addressed.**

The goal of this handbook and course is to cross-train providers in both specialties so that members of each group feel confident in offering help without harming, walking alongside a client to the next stop in their healing journey, and connecting them with more specialized help when indicated. We want providers to specialize in the area of nutrition that excites them and expand their reach, helping more people than they would be able to without this training.

The authors developed these guidelines with the intention of highlighting inclusive and intersectional practices. This is essential to meet the needs of our clients. We are a group of ten individuals with thousands of hours of personal, clinical, and research experience in the healing of disordered eating and gastrointestinal distress. We recognize, however, that our best efforts are also limited by our privileges and lived experiences; we understand that our own biases might cause harm. We welcome any and all feedback that could help to ensure all people are represented fairly and accurately.

By using the tools provided throughout this handbook, our goal is that your practice will become deeper, richer, and more impactful. You will be able to build on the skills in your specialty and expand your competence and clinical compassion to improve patients' quality of life with food and body.

This guide is founded on existing research in both GI and ED fields, and we thank the researchers and research participants for their contributions. We acknowledge that most of the past and current methods for gathering, processing, disseminating, and interpreting research are steeped in bias and lack the representation necessary to assume the findings will apply to the majority of people, especially those from marginalized communities. If we wait for the evidence to be gathered, too many people will continue to be harmed by siloed understandings of their health and gaps in care. We must advance clinical practice and research at the same time. We will be identifying research disparities and addressing them with our clinical experience and best practices. We hope this inspires those with the power to study these co-occurring conditions formally to center the experiences of those who are most impacted—our patients.

# Overview of Co-Occurring Gastrointestinal and Eating Disorders

The confluence of eating disorders (ED) and gastrointestinal (GI) disorders is common, complex, and often overlooked in healthcare settings. Research regarding the risk factors, prevention, diagnosis, and treatment of EDs within individuals with GI disorders is very limited. Many of the known risk factors for EDs and GI disorders are shared, and causality is likely not unidirectional, presenting a chicken-or-the-egg quandary. Both literature and clinical experience describe a higher incidence of disordered eating thoughts and behaviors in individuals with diet-related chronic health conditions[1,2]. Self-led dietary exclusions appear to predict ED onset and, of those presenting at GI clinics, an estimated 23% of patients will have disordered eating behaviors[3,4,5].

Inversely, of those presenting with an eating disorder, up to 98% will meet criteria for a functional GI disorder[6]. Even more common than the development of distinct, diagnosable GI disorders is the occurrence of GI symptoms ranging from chronic manifestations (e.g., reflux, postprandial distress, bloating, irregular bowel movements) to acute, potentially life-threatening presentations (e.g., upper GI bleeding, esophageal rupture, intractable vomiting related to superior mesenteric artery syndrome, acute liver failure). The lifetime prevalence of EDs is 8% in women and 2% in men, worldwide, and 5% in women and 2% in men in the U.S.; research is limited regarding gender-diverse populations[7]. However, the majority of EDs go undiagnosed and are rarely screened for in clinical settings, representing lost opportunities to identify patients who may be unaware of the severity of the ED and its relationship to their GI symptoms.

Eating disorder thoughts, behaviors, and traits can resist detection by unfamiliar clinicians, causing GI symptoms to take center stage

while the ED goes unaddressed. GI issues linked to ED-related physiological adaptations may be misattributed to other etiologies, leading clients down diagnostic rabbit holes and away from nutritional rehabilitation and psychological treatments as the primary interventions. Even when ED risk or status is known, providers may be tempted to offer "help" in the form of targeting GI symptoms and disorders by any means available. However, there is considerable risk for GI-focused dietary therapies to trigger the initial onset of an ED, to precipitate relapse, or to harm those actively struggling with an ED. For this reason, we recommend prioritizing interventions that have the least harmful effect on an individual's mental health—which may mean sidestepping sweeping nutrition changes in favor of non-diet interventions. When indicated, diet interventions should be utilized with great care and support in order to prevent inadvertent harm.

## A Brief Overview of the Handbook

Psychological Foundations covers the diagnostic criteria and treatments for EDs, as well as the impacts of marginalization and systemic oppression on individuals with and seeking care for EDs.

GI Foundations covers "normal" anatomy and physiology of the GI tract.

Alterations in GI Anatomy and Physiology describes structural and functional aberrations due to EDs and provides an overview of many common GI disorders (including their prevalence, overlap with EDs where available, diagnostics, and general treatment approaches).

Psychogastroenterology Foundations introduces this new field and describes its tools which may serve those with both GI disorders and mental health conditions.

Screening & Referral covers the purpose of screening and developing mental health-informed referral networks, tools used for screening, suggested communication methods during screening, and the RD scope of practice with respect to screening for EDs.

Nutrition Assessment, Nutrition-Related Diagnosis, and Nutrition Intervention walk readers through the initial steps of the nutrition care process, from the assessment of nutritional status to specific common nutrition deficits, and finally to interventions.

Monitoring & Evaluation provides tips on the ongoing reassessment of individuals with co-occurring GI disorders and EDs.

The book ends with resources for providers to use for further education and use with patients.

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CHAPTER 2

# Psychological Foundations

The mind-body connection is the link between a person's thoughts, attitudes, behaviors, and physical health. Up until recently, the traditional medical model attempted to address physical ailments independent of the mind and psychological ailments independent of the body. This approach to healthcare is not only ineffective, but harmful; it has created a huge gap in our ability to address actual health concerns by conflating symptoms and root causes. Nowhere is this highlighted more than in the assessment and treatment of eating disorders. Without an understanding of eating disorders and their consequences, many individuals are overlooked, misdiagnosed, and untreated.

## DSM 5 Diagnostic Criteria for Eating Disorders

As a general overview, eating disorders are a unique set of psychiatric conditions that involve a disruption in a person's relationship with their body and food. Because food is requisite for life, the physical consequences are severe and impact just about every organ system in the body. As a result, those living with eating disorders often present in a variety of different settings, attempting to treat the physical consequences of their disorder while being completely unaware or actively avoiding the root cause[1]. The Diagnostic and Statistical Manual of Mental Disorders (DSM), 5th edition, is the handbook used by healthcare professionals in the United States and much of the world as the authoritative guide to

the diagnosis of mental disorders[2]. It has included the following disorders in its most recent iteration: Pica, Rumination Disorder, Avoidant Restrictive Food Intake Disorder (ARFID), Anorexia Nervosa (AN), Bulimia Nervosa (BN), Binge-Eating Disorder (BED), Other Specified Feeding or Eating Disorder (OSFED), and Unspecified Feeding or Eating Disorder (UFED). Out of the conditions listed above, AN, BN, and BED are the most commonly recognized, but OSFED is the most commonly diagnosed feeding and eating disorder as it accounts for 44% of all eating disorder diagnoses[3]. Below is an overview of each diagnosis and their corresponding criteria.

## Avoidant Restrictive Food Intake Disorder (ARFID)

***Overview:*** ARFID is a disorder characterized by avoiding food without concern for body image. Typically, it can present as an apparent lack of interest in eating or food, as avoidance of particular sensory characteristics of food, or as fear of aversive consequences of eating (e.g., bloating, pain, diarrhea, choking). The "fearful" subtype of ARFID is prevalent in the GI space, but more research is needed to understand how to best treat this population[4].

***Prevalence:*** In clinical samples of children and adolescents, ARFID prevalence estimates have been reported between 32 and 64%[5]. This sample included individuals from a diverse range of settings including eating disorder clinics, pediatric hospitals, medical outpatient programs and gastroenterology clinics. In community-based studies, the estimated prevalence ranges greatly: between 0.3% and 15.5%. It affects boys more often than girls and appears to impact 3.2% of children aged 8-13 years. The best estimates we have for adults suggests that 0.3% of people over 15 years have ARFID and that these rates could be much higher in adults with gastrointestinal issues. A study of adults who presented for a GI

evaluation found that 23.6% of people had some symptoms of ARFID, 17.3% of the group most likely had ARFID with more assessment needed, and 6.3% of the adults met the full criteria[2,6].

*DSM 5 Criteria:*

- An eating or feeding disturbance (e.g., apparent lack of interest in eating or food; avoidance based on the sensory characteristics of food; concern about aversive consequences of eating) as manifested by a persistent failure to meet appropriate nutritional and/or energy needs associated with **one (or more)** of the following:
    - Significant weight loss (or failure to achieve expected weight gain or faltering growth in children)
    - Significant nutritional deficiency
    - Dependence on enteral feeding or oral nutritional supplements
    - Marked interference with psychosocial functioning
- The eating disturbance does not occur exclusively during the course of anorexia nervosa or bulimia nervosa, and there is no evidence of a disturbance in the way in which one's body weight or shape is experienced
- The disturbance is not better explained by lack of available food, a culturally sanctioned practice, or a concurrent medical condition

## Anorexia Nervosa

***Overview:*** Anorexia nervosa is a disorder characterized by restriction of food (e.g., skipping meals, small portions, limiting or excluding food groups), fear of weight gain, lack of insight into the seriousness of the condition, an inaccurate body perception (i.e.,

feel larger than they are), and an overvaluation of body image in a person's identity.

***Prevalence:*** The estimated lifetime prevalence of AN in the United States is 0.8%. The overall incidence rate of AN is around 7 per 100,000. The incidence is increasing in Italy and other Western countries, likely due to earlier diagnosis or earlier onset. The lifetime prevalence of anorexia nervosa in the LGBTQIA+ community is 1.7%, which is slightly more than double the rate for the general population. It's estimated that 10-25% of all anorexia cases are males, which translates into a lifetime prevalence of 0.3%[7]. It's important to note that available data on men and boys is limited[8]. There aren't any significant ethnic or socioeconomic differences in prevalence rates for anorexia nervosa.

*DSM 5 Criteria:*

- Restriction of energy intake relative to requirements, leading to a significantly low body weight in the context of age, sex, developmental trajectory, and physical health ("significantly low weight" is defined as less than minimally normal or, for children and adolescents, less than that minimally expected)
- Intense fear of gaining weight or becoming fat, or persistent behavior that interferes with weight gain, even though at a significantly low weight
- Disturbance in the way in which one's body weight or shape is experienced, undue influence of body weight or shape on self-evaluation, or persistent lack of recognition of the seriousness of the current low body weight
- Subtypes:
    - Restricting type: no binge eating or purging in the last 3 months

- Binge eating/purging type: recurrent episodes of binge-eating or purging behavior in the last 3 months

## Binge Eating Disorder

***Overview:*** Binge eating disorder is characterized by a person eating a large amount of food in a short period of time, a sense of being on "autopilot" during the episodes, and a lack of engagement in purging behaviors following the episodes.

***Prevalence:*** BED behaviors are described in 0.85% of the population and are frequently associated with medical comorbidities such as type II diabetes mellitus, hypertension, and dyslipidemia. There is also a high incidence in people undergoing bariatric surgery. The lifetime prevalence for BED in the LGBTQIA+ community is 2.2%, which is 2.5 times higher than cisgender heterosexual population[9].

### *DSM 5 Criteria:*

- Recurrent episodes of binge eating. An episode of binge eating is characterized by both of the following:
  - Eating, in a discrete period of time (e.g., within any 2-hour period), an amount of food that is definitely larger than most people would eat in a similar period of time under similar circumstances
  - The sense of lack of control over eating during the episode (e.g., a feeling that one cannot stop eating or control what or how much one is eating)
- Binge-eating episodes are associated with 3 or more of the following:
  - Eating much more rapidly than normal
  - Eating until feeling uncomfortably full

    - Eating large amounts of food when not feeling physically hungry
    - Eating alone because of being embarrassed by how much one is eating
    - Feeling disgusted with oneself, depressed, or very guilty after overeating
- Marked distress regarding binge eating is present
- The binge eating occurs, on average, at least 1 day a week for 3 months
- The binge-eating is not associated with the regular use of inappropriate compensatory behavior and does not occur exclusively during the course of anorexia nervosa or bulimia nervosa

## Bulimia Nervosa

***Overview:*** Bulimia nervosa is a disorder characterized by eating large amounts of food in a short period of time and inappropriately compensating for that intake by purging the food consumed in some way (i.e., self-induced vomiting; misuse of laxatives, diuretics, or other medications; excessive exercise; fasting).

***Prevalence:*** The prevalence of BN is 0.28%. The highest rate of BN is 300 per 100,000 persons in women between ages 16 and 20, although the age at onset is decreasing. It is important to note that black and Hispanic teens are 50% more likely than their white peers to experience symptoms of BN[10]. The lifetime prevalence of BN in the LGBTQIA+ community is 1.3%, which is almost 4 times higher than that of cisgender heterosexual adults[9]. Compared with heterosexual men, gay and bisexual men had a significantly higher prevalence of lifetime bulimia nervosa, subclinical bulimia, and any subclinical eating disorder.

*DSM 5 Criteria:*

- Recurrent episodes of binge eating. An episode of binge eating is characterized by both of the following:
  - Eating in a discrete period of time an amount of food that is definitely larger than what most individuals would eat in a similar period of time under similar circumstances
  - A sense of lack of control over eating during the episode
- Recurrent inappropriate compensatory behavior in order to prevent weight gain
- The binge eating and compensatory behavior occur on average at least once a week for 3 months
- Self-evaluation is unduly influenced by body shape and weight
- The disturbance does not occur as part of a pattern of anorexia nervosa (binge-eating/purging subtype)

**What's the distinction? AN, BN, or BED**

The addition of AN, binge-purge type into the DSM-V brought with it some confusion about how to differentiate this diagnosis from the other eating disorders that include binge-eating and/or purging episodes as a part of the criteria. When differentiating between each diagnosis the best approach involves paying attention to two things: the level of restriction the client is engaging and their body weight.

Severe food restriction is not a part of the criteria for either BN or BED but it is primary for Anorexia Nervosa, including Atypical Anorexia Nervosa. It is common for people with BN

and BED to also engage in some level of restriction but it is not the primary way that they manage their disordered relationship with food.

Another distinctive difference is the low body weight associated with Anorexia Nervosa. It is important to remember that the DSM-V currently requires that an individual must be significantly underweight in order to meet the criteria for AN. Those with BN and/or BED are more often "normal" or "above normal" weight by BMI standards.

## Pica

***Overview:*** Pica is a disorder categorized by an individual regularly consuming a non-food substance (e.g., ice cubes, dirt, hand sanitizer).

***Prevalence:*** The DSM 5 states, regarding pica, "prevalence is unclear." A 2022 study reiterates that there's a lack of data related to pica due to it being understudied[2,11].

### *DSM 5 Criteria:*

- Persistent eating of nonnutritive, nonfood substances over a period of at least 1 month
- The eating of nonnutritive, nonfood substances is inappropriate to the developmental level of the individual
- The eating behavior is not part of a culturally supported or socially normative practice
- If the eating behavior occurs in the context of another mental disorder (e.g., a neurodevelopmental disorder, schizophrenia) or medical condition (e.g., pregnancy,

nutritional deficiencies, sickle cell), it is sufficiently severe to warrant additional clinical attention

## Rumination Disorder

***Overview:*** Rumination disorder is characterized by regurgitation of food. Unlike vomiting, it is not usually forceful and can be voluntary. It is thought to be an unintentionally acquired habit and possibly a learned adaptation of the belch reflex[12]. People suffering with this disorder may either re-chew, re-swallow, or spit out the food regurgitated. This diagnosis is defined by the DSM 5 as a feeding/eating disorder and also by the Rome Foundation as a GI disorder (see Chapter 4).

***Prevalence:*** The DSM 5 describes rumination disorder prevalence data as "inconclusive." A 2022 global study suggested a worldwide prevalence of 3.1% (ranging from 1.7% to 5.5%)[13].

### *DSM 5 Criteria:*

- Repeated regurgitation of food over a period of at least 1 month
- The repeated regurgitation is not attributable to an associated gastrointestinal or other medical condition (e.g., gastroesophageal reflux, pyloric stenosis)
- The eating disturbance doesn't occur exclusively during the course of anorexia nervosa, bulimia nervosa, binge-eating disorder, or avoidant/restrictive food intake disorder
- If the symptoms occur in the context of another mental disorder, they are sufficiently severe to warrant additional clinical attention

## Other Specified Feeding or Eating Disorder (OSFED)

***Overview:*** OSFED encompasses a handful of feeding and eating disorders that are serious and require treatment but fall short of fully meeting the rigid criteria of the main diagnoses listed above.

***Prevalence:*** OSFED is the most commonly diagnosed eating disorder making up approximately 44% of all eating disorder diagnoses[3]. This translates to 3.82% of females and 1.61% of males having OSFED in their lifetime[14].

### *DSM 5 Criteria:*

The following feeding and eating disorders are examples of OSFED presentations that differ in frequency, duration and intensity from the aforementioned diagnoses; these manifestations do not necessarily indicate lesser severity or medical risk.

- Atypical Anorexia Nervosa: All of the criteria of anorexia nervosa are met except the individual presents with a BMI ≥18.5 kg/m$^2$
- Bulimia Nervosa (of low frequency and/or limited duration): All of the criteria for bulimia nervosa except that it occurs less than once per week for less than 3 months
- Binge Eating Disorder (of low frequency and/or limited duration): All of the criteria for BED except it happens less than once per week for less than 3 months
- Purging Disorder: Recurrent purging behavior to influence weight or shape (e.g., self-induced vomiting; misuse of laxatives, diuretics, or other medications; excessive exercise) in the absence of binge eating and presenting as "underweight"

- Night Eating Syndrome: recurrent episodes of night eating, as manifested by eating after awakening from sleep or by excessive food consumption after the evening meal. There is awareness and recall of the eating. The night eating is not better explained by external influences such as changes in the individual's sleep-wake cycle or by local and/or social norms

## Unspecified Feeding or Eating Disorder (UFED)

***Overview:*** This diagnosis is best used when a provider is unable to complete a thorough evaluation to tease apart the subtleties of an individual's diagnosis, but there is a clinical disruption in the person's ability to feed themself appropriately.

***Prevalence:*** There is virtually no data available on the frequency of UFED as a standalone diagnosis.

### *DSM 5 Criteria*

UFED may apply to presentations that cause clinically significant distress, but the clinician chooses not to specify. Common presentations that might warrant this diagnosis would include:

- Someone that meets most of the criteria for AN but may not have a low enough body weight to meet the full criteria for this diagnosis *or* weigh enough (i.e., "normal" weight) to meet the criteria for Atypical AN
- Someone that meets most of the criteria for BN but does not have binge episodes as frequently as the criteria requires (i.e., episodes occur over several months or years)
- Someone that presents to the emergency department with severe dehydration as a result of excessive laxative

use, but they only engage in purging behaviors during stressful situations rather than regular intervals

## What the DSM Has Missed

The DSM lays an important foundation for what eating disorders are and how they should be conceptualized. However, it is important to acknowledge the various longstanding biases that have negatively impacted the mental health field and its accompanying text. Most of the large bodies of research that have informed how we understand, diagnose, and treat eating disorders are largely geared towards white, cisgender, heterosexual, teenage girls with restrictive eating patterns. This has created the false narrative that those who don't fit into that demographic do not develop eating disorders and, if they do, that those disorders are not as severe. Contrary to popular belief, some overlooked populations are at equal or greater risk for developing certain eating disorders.

### Fat People/Individuals of Size

Internalized weight bias–the belief that negative stereotypes about weight apply to the self–is a significant concern, as it has been linked with a host of physical and psychological issues including eating disorders[15]. Unfortunately, externalized weight bias–negative weight-related attitudes and beliefs that impact how one might view and treat people of size–has influenced eating disorder treatment such that two people can have the same symptoms of anorexia nervosa but not receive the same diagnosis or treatment due to differences in body weight, shape, or size.

### Black, Indigenous, and People of Color (BIPOC)

People within the BIPOC population have been largely left out of the eating disorder discussion by nature of who has been present

in treatment centers and who has been present in research. This has resulted in diagnostic criteria which overlook the reality that there are a variety of unrealistic beauty standards (e.g., flat stomach, perfectly proportioned curves) that can trigger disordered eating in the same way a "drive for thinness" does; additionally, acculturation can contribute to the development of eating disorders by impacting individuals' relationship with body image and eating practices[16,17]. Historically, when research did include people of color, it identified the differences in beauty standards (i.e., acceptance of larger body sizes) as protective factors, which encouraged providers and the general public to assume that eating disorders do not exist in these communities[18]. We are now aware that eating disorders do not discriminate and can impact people from all racial and ethnic backgrounds[19]. For example, there are some eating disorders (i.e., bulimia nervosa, binge eating disorder) that impact some BIPOC communities at greater rates than their white counterparts[20-22].

## LGBTQIA+

The current DSM criteria is more inclusive of all genders than previous editions, but there is still work to be done to ensure that heteronormative standards and binary views of gender aren't creating biases in how providers diagnose and assess eating disorders. Historically, anorexia nervosa criteria required significantly low body weight *and* loss of the menstrual cycle, which automatically eliminated individuals without uteri from receiving a diagnosis[23]. The current criteria no longer requires amenorrhea but still has a long way to go to acknowledge the different ways in which gender dysphoria, discrimination, and internalized homophobia and transphobia can increase a person's risk for developing an eating disorder.

## Boys and Men

Boys and men have also been left out of the eating disorder community for similar reasons as those listed for BIPOC individuals; they don't have a large presence in treatment settings, so it has been assumed that eating disorders do not impact them. This is similar to what happened in the early days of substance abuse research, where it was thought to be a "man's disease" simply because there weren't many women present in the rehab facilities. It has largely been assumed that men do not have eating disorders because so much focus was initially placed on anorexia nervosa, which used to require a significantly low weight *and* loss of a menstrual cycle. As discussed above, this automatically eliminated those assigned male at birth from the diagnosis, but it also ignored that some men may actively avoid muscularity and seek out a thin body ideal. There is currently no formal diagnosis that accurately describes disordered eating in men who hyper-fixate on muscularity (e.g., bodybuilders, wrestlers). Informally, this is referred to as "bigorexia" (see "Eating Disorders Not Yet Defined by the DSM" box for more info).

## People with Disability

Eating disorder advocacy and media have long been divorced from disability justice work, which has led to a gross under-representation in research. There are very few studies that specifically look at this population, which is unfortunate, considering that an estimated 12.6% of Americans live with some form of disability[24]. Because we know research is the guiding force behind the development of the diagnostic criteria for eating disorders, it is safe to say there are likely a variety of ways the DSM has overlooked important signs and/or symptom differences in this population that impact the way eating disorders are diagnosed and treated. We do not yet know the specific ways in which the DSM is harmful to this population, as there is no data expounding

on this topic, but we believe it is important to speak about this issue and highlight it as an area of future research[25].

### Eating Disorders Not Yet Defined by the DSM

The following terms refer to diagnoses that are not formally recognized by the DSM for a variety of reasons, the most common being that the symptomatology may overlap with other mental health conditions (e.g., Obsessive Compulsive Disorder), making it challenging to categorize exclusively as a feeding or eating disorder. In spite of this, these diagnoses are known to impact an individual's relationship with food and/or body image and can have severe physical and psychological health consequences.

**Orthorexia:** An obsession with "correct" or "healthy" eating. This obsession becomes so intense that individuals struggling with this disorder may experience malnutrition or impairment in their psychosocial functioning (e.g., social life, career advancement)[26-28].

**Diabulimia:** Anyone with insulin-dependent diabetes who manipulates their insulin in an effort to lose weight or engage in disordered eating behaviors. Signs may include restricting certain food groups to avoid insulin usage, intentionally overdosing insulin to justify a binge, and/or fearing that insulin may lead to weight gain[29].

**Pregorexia:** When a person excessively attempts to control their weight during pregnancy through a variety of disordered behaviors, including caloric restriction, restriction of specific food groups, over-exercise, and/or purging behaviors. Possible complications include bone loss for the pregnant person, fetal developmental problems, low birth weight,

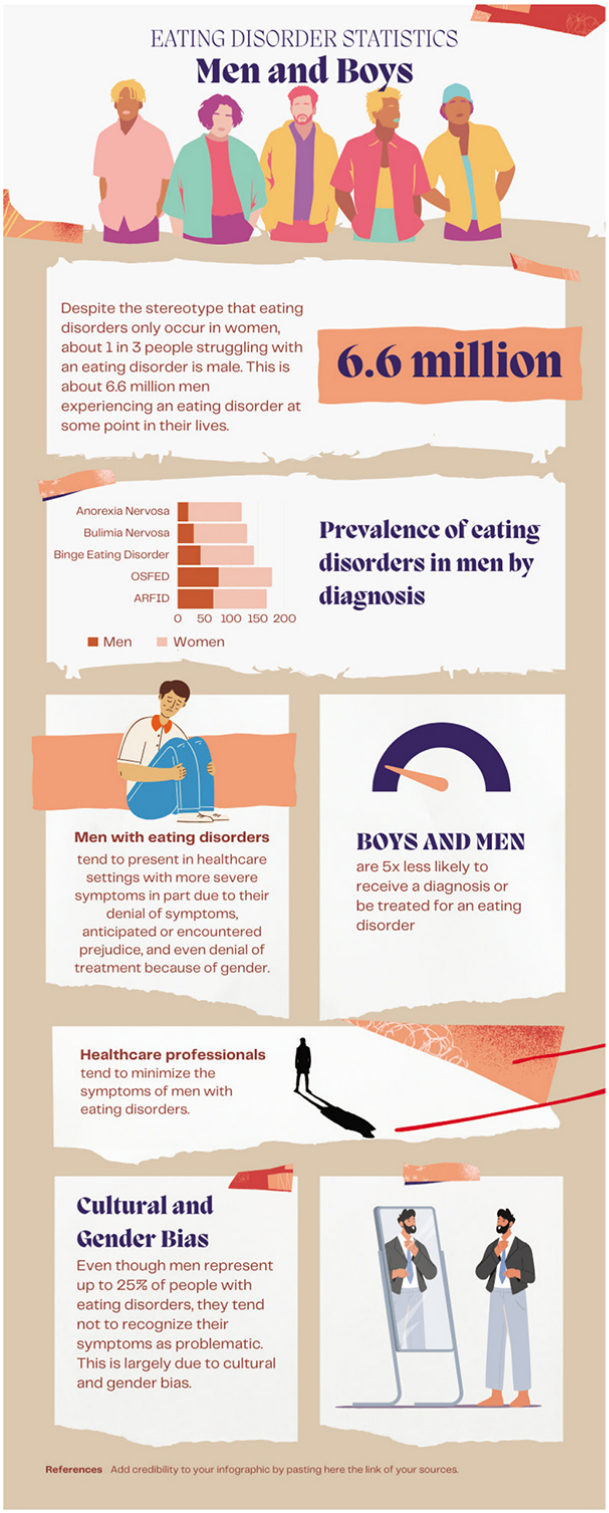
impaired growth, and an increased risk of prolonged labor and/or miscarriage[30].

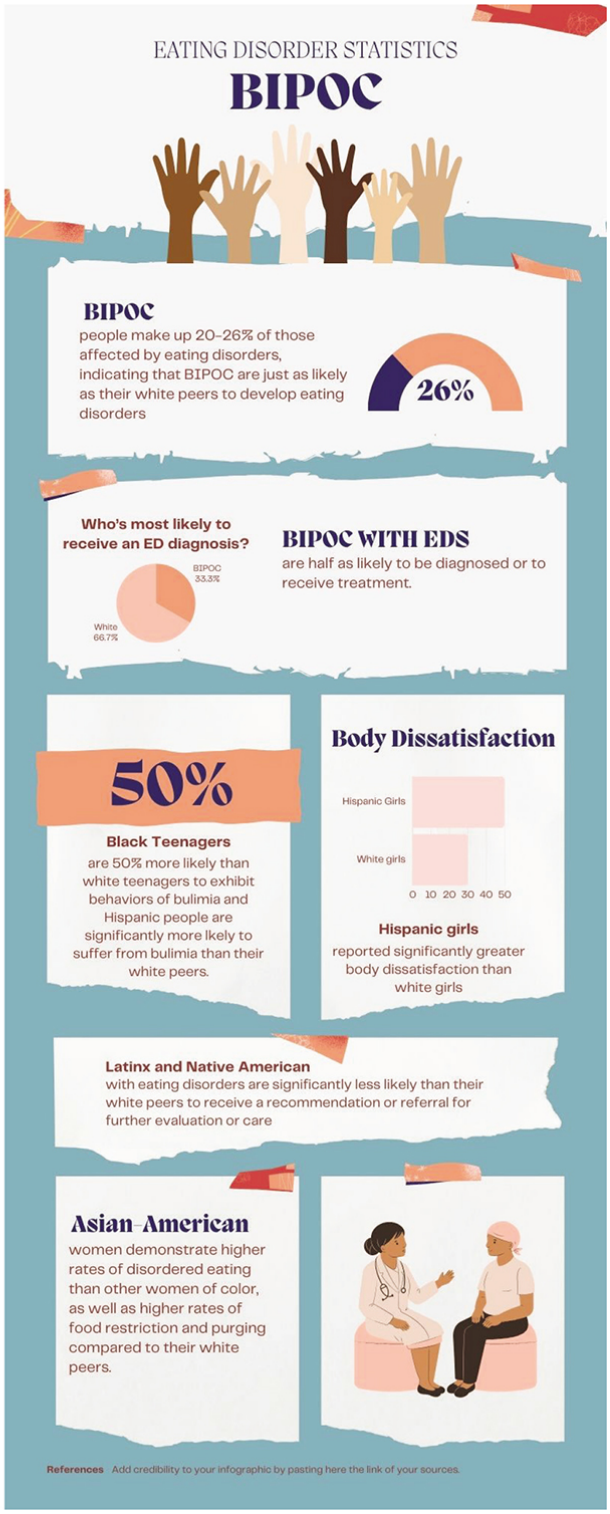
**Drunkorexia:** When a person restricts calories to compensate for calories consumed from drinking alcohol. The primary reason for this behavior is to avoid weight gain[31].

**Bigorexia:** A body dysmorphic disorder characterized by the individual's desire to have less fat mass and an obsession with increasing muscle mass. This appears to be influenced by environmental factors such as media, peers, and dietary attitudes in addition to personality traits, such as perfectionism and insecurity[32].

# Eating Disorder Statistics by Community

Source: *Eating Disorder Statistics*, ANAD, 2023[7]



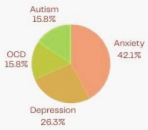
EATING DISORDER STATISTICS

# People with Co-Occuring Conditions

## AUTISM X EATING DISORDERS

20-30 % of adults with eating disorders also have autism.

**20-30%**




## PEOPLE WITH ED'S

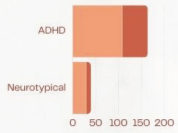
Have between 1 and 4 additional psychiatric disorders.

**58%**

**ARFID x Eating Disorders**

Between 13-58% of ARFID patients also have Autism Spectrum Disorder.

## ADHD x Eating Disorders




**Girls with ADHD**

are 3.6 times more likely to have an eating disorder in general and 5.6 times more likely to have bulimia in particular

**Eating Disorders x Type I Diabetes**

In a study, girls with type 1 diabetes aged 9-13 were evaluated for 14 years and by the time they were in their 20s 40.8% met criteria for a full- to sub-threshold eating disorder, and 59.2 % took part in dangerous disordered eating behavior.

## OCD x Eating Disorders

Between 10 and 35% of patients with eating disorders have OCD unrelated to the eating disorder.

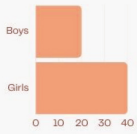
**References** Add credibility to your infographic by pasting here the link of your sources.

EATING DISORDER STATISTICS

# People in Bodies of Size

In a study of college and university students, just **2% of those who met criteria for eating disorders were "underweight."** For the overall populace, the figure is less than 6%.

**2%**




## PEOPLE IN LARGER BODIES

are at a higher risk of using unhealthy weight control behaviors. About 40% of "overweight" girls and 20% of "overweight" boys use disordered eating behaviors.

**14x**

**People with Atypical Anorexia Nervosa**

were 14 times less likely to receive the recommended treatment for Anorexia Nervosa than those who were underweight.

## Weight Stigma x Eating Disorders

**Among those who experience weight stigma**

two-thirds were stigmatized by doctors, leading many to avoid seeking healthcare.

**People who experience weight discrimination**

are 60% more likely to die regardless of their body mass index (BMI).

**60%**

## Weight stigma

can trigger changes in the body, such as increased cortisol levels, that lead to poor metabolic health and an increased likelihood of alcohol and substance abuse.

**References** Add credibility to your infographic by pasting here the link of your sources.

EATING DISORDER STATISTICS

# LGBTQIA+

## LGBTQ YOUTH

Nearly nine in ten (87%) LGBTQ youth reported being dissatisfied with their body


## TRANS YOUTH

Rates of body dissatisfaction were higher among transgender and nonbinary youth (90%) compared to cisgender youth (80%).

**32%**

Transgender

people report using their eating disorder to modify their body without hormones

### Gay Men

are seven times more likely to report binge-eating and twelve times more likely to report purging than heterosexual men.

**Gender dysphoria and body dissatisfaction** in transgender people is often cited as a key link to eating disorders.

## Non-Binary People

may restrict their eating to appear thin, consistent with the common stereotype of androgynous people in popular culture.

**References** (Giachin, 2023)

# Eating Disorder Risk Factors

Unfortunately, the exact cause of eating disorders is unknown. What we do know is that eating disorders are biopsychosocial illnesses, meaning they are a result of various risk factors and vulnerabilities[3]. The most common include age, gender, family history, weight stigma, psychological illness, dieting, and extracurricular activities[33].

## Age

The most common age of onset for eating disorders is between 12-25 years. They are thought to occur more frequently in younger populations because of the stress and various transitions that take place during that period of life. However, there has been a recent increase in hospital admissions of folks over age 50 for untreated eating disorders requiring medical stabilization, and the age group with the highest prevalence of eating disorders is between 45-59 years[34].

## Gender

Hereditary patterns of eating disorders have been shown to disproportionately affect individuals assigned female at birth[35]. Eating disorders are also fairly common among sexual and gender minority populations[9]. For both groups, this is thought to in part be the result of a vulnerability created by discrimination and/or minority stress. For gender minority populations (i.e., transgender and gender-expansive individuals), gender dysphoria is another factor that may make them more susceptible to eating disorders. Gender dysphoria is characterized by distress, discomfort, or even disgust relating to how one's gendered experience may be incongruent with their gender identity. Gender dysphoria is often worsened by experiences of being misgendered, negative body remarks, internalization of appearance ideals, and body

surveillance[36]. For individuals whose eating disorder may be a tool to alleviate gender dysphoria, it can be useful to center ways of alleviating gender dysphoria without using harmful behaviors, such as the use of gender-affirming supplies or shifts to clothing choices.

## Genetics and Family History

Recent research has identified specific genetic markers that increase a person's risk for developing an eating disorder. One 2009 study found that only half of the genetic risk factors predicting drive for thinness and body dissatisfaction in females predicted the same traits in males[37]. This is thought to be a result of inherited variation in an estrogen (a sex hormone found in males and females, though higher in females) receptor gene (ESR1) that significantly increased risk of restrictive eating and the subsequent development of anorexia nervosa, restrictive subtype[38]. According to the Mayo Clinic, people with first-degree relatives with an eating disorder are at higher risk of developing one themselves[39]. The heritability of eating disorders is likely related to the combination of genetic vulnerability as well as the behavior modeled by the relative in their relationship with food and body.

## Weight Stigma

Weight stigma is the act of holding bias against people in higher-weight bodies and making assumptions about their character and health based on their appearance. These types of attitudes and beliefs are deeply harmful as they can cause people, in all body types, to internalize the idea that being in a higher-weight body is inherently negative. Internalized weight bias increases the likelihood that a person will engage in disordered eating behaviors and diets to avoid the negative social consequences of being at a higher weight[40,41].

## Psychological Illness

Most people who are diagnosed with an eating disorder have co-occurring mental health concerns. These could include low self-esteem, anxiety, depression, obsessive-compulsive disorder, substance use disorder, and/or difficulty forming and maintaining relationships with family members and peers. Any one of these psychological challenges generates stress and may create a vulnerability to developing an eating disorder[33].

## Dieting

There is a popular misconception that risk is contingent on extremity (i.e., that eating disorder risk is only impacted by "crash dieting" while it is safe to engage in so-called "sensible dieting"). However, any restrictive diet can increase the risk of developing an eating disorder. Even dietary restrictions undertaken for reasons other than weight management can increase a person's risk. Examples might include a low-FODMAP diet for GI symptom improvement or reducing intake to cope with food insecurity and stretch the grocery budget. In short, dietary restriction is not a benign intervention, regardless of the intention behind it[42-44].

## Extracurricular Activities and Profession

Being part of a social group is generally an important aspect of mental wellness. However, if an individual participates in a social group or career where appearance or body shape/size influences their status or financial security, there is an increased risk for developing an eating disorder. Some examples include television personalities, performing artists, models, certain athletes, social media influencers, fitness instructors, and military personnel, among others. These people may be pressured by peers, coaches, workplace culture, and even parents to conform to expectations, putting them at risk for an eating disorder[33].

## Eating Disorder Psychological Assessments

There are a variety of eating disorder assessments available that are tailored to certain diagnoses, age groups, and presentations. The assessments listed below are only available for use by trained mental health professionals. Assessments that can be self-administered or completed with the help of an RDN or other health provider are described in Chapter 6.

- Body Shape Questionnaire (BSQ)
- The Compulsive Exercise Test
- Dietary Rules Inventory (DRI)
- Drive for Muscularity Scale (DMS)
- The Eating Disorder Assessment for DSM-5 (EDA-5)
- Eating Disorder Diagnostic Scale (EDDS)
- Eating Disorder Examination (EDE)
- Eating Pathology Symptoms Inventory (EPSI)
- Emotional Eating Scale (EES)
- Exercise and Eating Disorders
- Pica, ARFID, and Rumination Disorder Interview (PARDI)
- Structured Interview for Anorexic and Bulimic Syndromes (SIAB-EX)

## The Recovery Process

The most recent data shows that fewer than half of adults ever recover from either AN or BN[45]. This is sobering information as it highlights how little is known about how to best treat eating disorders. The path to recovery can be circuitous and, for some individuals, chronic, despite their best efforts. Each therapeutic modality might conceptualize eating disorders differently, but all of them generally agree there are three main areas of recovery, which include physical, behavioral, and psychological aspects.

### Physical Recovery

Physical recovery involves weight restoration, normalizing electrolyte and hormone levels, resuming menstruation (if applicable), and rectifying other health consequences associated with having an eating disorder. Most of these are reversible, but some health consequences can persist independently of the eating disorder (e.g., low bone density, some cases of gastroparesis, etc.).

### Behavioral Recovery

This aspect of recovery involves extinguishing all of the behaviors associated with having an eating disorder, such as restriction, excessive exercise, purging, and/or binge-eating. Behavioral abstinence alone is not an indication of a robust recovery.

### Psychological Recovery

This element of recovery involves treating the emotional and cognitive perturbations that underpin and/or occur alongside an eating disorder. Targets for treatment might include commonly co-occurring mental health disorders (e.g., anxiety, depression, obsessive-compulsive disorder, substance abuse disorder) or perpetuating traits (e.g., perfectionism, negative body image, cognitive rigidity).

## Eating Disorder Treatment

There is a network of eating disorder specialists across disciplines, including dietitians, psychotherapists, primary care physicians, and psychiatrists. In most cases, these providers have sought specialized training above and beyond what they received in their graduate programs, as most programs do not require any specific eating disorder training as a requisite for graduation or licensure. This is deeply problematic considering the high prevalence rate of eating disorders in the United States.

The most effective treatment involves a coordinated multidisciplinary approach[46-47]. Treatment can occur on an outpatient basis, or it can occur in a treatment center or hospital. Most treatment centers in the United States are for-profit businesses that require some form of health insurance to access, and they have different levels of care depending on the individual's severity of symptoms:

- Inpatient Hospitalization – 24 hours per day treatment with full-time nursing and medical monitoring and care
- Residential Treatment Centers – 24 hours per day recovery program with full-time nursing staff and scheduled access to physician care
- Partial Hospitalization (PHP) – provides treatment for half to full days (6-10 hours per day), up to six days per week and does not include treatment overnight (contrary to what the name suggests, PHPs don't usually take place in a hospital setting)
- Intensive Outpatient (IOP) – treatment typically occurs at least three times per week for two or more hours each day
- Outpatient treatment – may occur with each member of the treatment team, ranging in frequency from once monthly for stable and/or disengaged clients to multiple times per week for more acute and/or engaged clients

The following chart describes the criteria for different levels of care as well as the signs and symptoms that may determine a person's eligibility for each level.

**Table 2.1. APA Level of Care Guidelines for Patients with Eating Disorders**

| | Level 1: Outpatient | Level 2: Intensive Outpatient | Level 3: Partial Hospitalization | Level 4: Residential Treatment Center | Level 5: Inpatient Hospitalization |
|---|---|---|---|---|---|
| **Medical Status** | Medically stable to the extent that more extensive medical monitoring, as defined in levels 4 and 5, is not required | | | Medically stable to the extent that intravenous fluids, nasogastric tube feedings, or multiple daily laboratory tests are not needed | ***For adults:*** Heart rate <40 bpm; blood pressure <90/60 mmHg; glucose <60 mg/dl; potassium <3 mEq/L; electrolyte imbalance, temperature <97.0°F; dehydration; hepatic, renal, or cardiovascular organ compromise requiring acute treatment; poorly controlled diabetes<br>***For children and adolescents:*** Heart rate near 40 bpm, orthostatic blood pressure changes (>20 bpm increase in heart rate or >10 mmHg to 20 mmHg drop), blood pressure <80/50 mmHg, hypokalemia, hypophosphatemia, or hypomagnesemia |
| **Suicidality** | If suicidality is present, inpatient monitoring and treatment may be needed depending on the estimated level of risk | | | | Specific plan with high lethality or intent; admission may also be indicated in patient with suicidal ideas or after a suicide attempt or aborted attempt, depending on the presence or absence of other factors modulating suicide risk |
| **Weight as percentage of healthy body weight** | Generally >85% | Generally >80% | Generally >80% | Generally <85% | Generally <85%; acute weight decline with food refusal even if not <85% of healthy body weight |
| **Motivation to recover, including cooperativeness, insight, and ability to control obsessive thoughts** | Fair-to-good motivation | Fair motivation | Partial motivation; cooperative; patient preoccupied with intrusive, repetitive thoughts >3 hours/day | Poor-to-fair motivation; patient preoccupied with intrusive, repetitive thoughts 4-6 hours/day; patient cooperative | Very poor to poor motivation; patient preoccupied with intrusive, repetitive thoughts; patient uncooperative with treatment or cooperative only in highly structured environments |

| | | | | | |
|---|---|---|---|---|---|
| | | | | with highly structured treatment | |
| **Co-occurring disorders (substance use, depression, anxiety)** | Presence of comorbid condition may influence choice of level of care | | | | Any existing psychiatric disorder that would require hospitalization |
| **Structure needed for eating/gaining weight** | Self-sufficient | Self-sufficient | Needs some structure to gain weight | Needs supervision at all meals or will restrict eating | Needs supervision during and after meals or nasogastric/special feeding modality |
| **Ability to control compulsive exercising** | Can manage compulsive exercising through self-control | Some degree of external structure beyond self-control required to prevent patient from compulsive exercising; rarely a sole indication for increasing the level of care | | | |
| **Purging behavior (laxatives and diuretics)** | Can greatly reduce incidents of purging in an unstructured setting; no significant medical complications, such as electrocardiographic or other abnormalities, suggesting need for hospitalization | | | Can ask for and use support from others or use cognitive and behavioral skills to inhibit purging | Needs supervision during and after all meals and in bathrooms; unable to control multiple daily episodes of purging that are severe, persistent, and disabling, despite appropriate trials of outpatient care, even if routine laboratory test results reveal no obvious metabolic abnormalities |
| **Environmental stress** | Others able to provide adequate emotional and practical support and structure | | Others able to provide at least limited support and structure | Severe family conflict or problems or absence of family so patient is unable to receive structured treatment in home; patient lives alone without adequate support system | |
| **Geographic availability of treatment program** | Patient lives near treatment setting | | | Treatment program is too distant for patient to participate from home | |

*Adapted from: American Psychiatric Association Practice Guideline for the Treatment of Patients with Eating Disorders, 2023.*[48]

Various modalities can be used to treat eating disorders in each of the above levels of care. Certain modalities are more or less suited to particular diagnoses and/or levels of care, as will be mentioned below.

## Psychotherapy

Talk therapy has been shown to be an effective method for addressing the psychological symptoms of eating disorders. There are several different therapeutic options that all share similar goals, which include normalizing eating patterns and improving emotional regulation skills, distress tolerance, and body image. The Mayo Clinic identifies behavior therapy, family-based therapy, and group therapy as important modalities in the treatment of eating disorders[49].

### *Behavioral Therapies*

The most common and widely behavioral therapies include cognitive behavioral therapy (CBT), dialectical behavior therapy (DBT), and acceptance and commitment therapy (ACT). Some modalities offer specialized approaches (i.e., CBT-E, CBT-AR, RO-DBT) but, in general, every behavioral therapy is based on the idea that psychological problems are rooted in faulty thinking and learned patterns of unhelpful behavior; when people learn alternate ways of coping, they become more effective in managing thoughts and behaviors and symptoms dissipate. Of these therapeutic options, CBT has been shown to begin improving eating disorder symptoms in just six sessions, with early reduction in restrictive behaviors being the best indicator of a positive outcome[50]. A 2017 review also found that CBT is effective at treating all types of eating disorders and is either equally or more effective than other therapeutic approaches[51].

### *Family-Based Treatment (FBT)*

Family-based treatment is a unique approach to treating eating disorders that empowers an individual's family members and/or immediate support people to play a central role in helping the person recover. This approach teaches participating loved ones how to guide the individual in building appropriate eating patterns

that lead to a biologically appropriate weight. Once this is accomplished, the individual working toward recovery is given increasing food autonomy to practice feeding themselves in a similar pattern until they are ready to address the underlying causes of the eating disorder. This form of therapy is the most effective approach for adolescents with AN and has shown promise in young adults and other diagnoses as well. In one randomized control trial (RCT) comparing FBT with adolescent-focused therapy (AFT), there were no differences between the two groups at the end of treatment, but significantly more patients receiving FBT had achieved full remission at 6 months (FBT 40%, AFT 18%) and 12 months follow-up (FBT 49%, AFT 23%)[52].

### *Group Therapy*

Group therapy is a type of therapy that involves meeting with a therapist and several other individuals with similar eating challenges and working through the thoughts, feelings, and behaviors of having an eating disorder as a community rather than alone. This approach provides a support network of peers, making group therapy an important form of treatment that addresses the social isolation and secrecy often associated with having an eating disorder. A recent study found that group psychotherapy is as effective as other common treatments and is more cost-effective than the most popular treatment[53].

### *Trauma Therapy*

The majority of people with AN, BN, and BED report a history of interpersonal trauma and rates of eating disorders are higher in people who have experienced trauma and post-traumatic stress disorder (PTSD)[54,55]. This makes effective trauma therapies, such as Eye Movement Desensitization and Reprocessing (EMDR) and Somatic Experiencing, important modalities to utilize when trauma is at the heart of an eating disorder. EMDR is a non-narrative

approach that allows clients to remember their trauma without the emotional charge by setting up a learning state that allows these experiences to be stored appropriately in the brain. All of this is done without an individual having to relive the details of the event and possibly retraumatize themselves. Somatic Experiencing is another modality that also focuses on relieving the negative consequences of a traumatic experience, and instead of focusing on the mind's memory, it focuses on the body's memory of the trauma and seeks to assist individuals in discharging the associated physiological tension. It is important to note that the only controlled studies examining EMDR's effectiveness at relieving the ED symptomatology found some improvements in its ability to lower negative body image memories and lower body dissatisfaction post-treatment[56], which highlight the reality that both of these therapy modalities are best used in conjunction with nutritional and behavioral approaches that directly improve eating disorder symptomatology.

## Pharmacotherapy

Neurobiological studies have shown that there are multiple systems of neurotransmitters and neurohormones involved in the control of appetite and eating and that they do not work in isolation[57]. Serotonin and dopamine disturbances have been reported across the different eating disorder diagnoses but manifest uniquely depending on the individual's symptoms, nutritional state, and comorbidities. For this reason, when discussing pharmacotherapy and its application to the treatment of eating disorders, it is best understood by looking at individual diagnoses and the effects different classes of psychotropic medications have on their related symptoms.

### *Anorexia Nervosa*

Different classes of drugs have been utilized in the treatment of AN with the goal of improving mood, restoring appetite and/or weight, improving motivation, and overall reducing the core symptoms of AN. Unfortunately, there is very little empirical data to support the efficacy of pharmacotherapy (specifically antidepressants) in this population, and the regulatory agencies in the United Kingdom and the United States have not approved any specific medication for the treatment of AN[58,59]. Despite this, most individuals with AN are treated with some form of psychotropic medication, and most guidelines for the treatment of AN include medication recommendations[60]. Currently, there is one class of medication, Atypical Antipsychotics (AAs), that has shown clinical promise in treating clients with AN. The rationale for using these medications is grounded in several bases: a) the neurobiology of AN, with the alterations of dopamine and serotonin pathways in the brain; b) the antidopaminergic properties of these medications that could improve sufferers' obsessional thinking towards weight and body shape; c) AA's positive effects on safety, anxiety, eating psychopathology and depression; d) the increase in appetite and food intake that AA's entail, consequently enhancing weight restoration[61].

A few of those medications are Olanzapine (Zyprexa), Aripiprazole (Abilify), and Quetiapine (Seroquel). In one randomized controlled study, participants taking Olanzapine achieved more rapid weight gain and exhibited fewer obsessive symptoms than individuals taking the placebo[62]. In a chart review of 106 adolescents with anorexia nervosa, some of whom were treated with Aripiprazole and others who were not, those treated with the medication were found to have a greater increase in BMI and BMI percentile compared to the group that did not receive the medication[63]. This medication has also been found to reduce some eating-related preoccupations and rituals[61]. Quetiapine has not been shown to improve weight restoration, but it does appear to decrease

symptoms of anxiety, depression, and obsessive-compulsive symptoms[64]. It is important to note that even with these findings, AAs remain experimental drugs used off-label in more severe cases of AN.

### *Bulimia Nervosa*

The effectiveness of medication in the treatment of BN has been well-established, and there have been many medication trials in this population that have consistently proven the use of antidepressant medications, such as selective serotonin reuptake inhibitors (SSRIs) and tricyclic antidepressants (TCAs), can improve a client's symptoms of BN. The most common medication used to treat BN and the only medication approved by the Food and Drug Administration (FDA) for the treatment of bulimia nervosa is fluoxetine (Prozac), a type of selective serotonin reuptake inhibitor (SSRI)[65]. Fluoxetine has been shown to reduce binge eating, purging behaviors, and relapse rates[66]. There are approximately 13 double-blind, placebo-controlled trials that show these same results for fluoxetine, which makes it the gold standard medication for treating bulimia nervosa[65].

### *Binge Eating Disorder*

Pharmacotherapy has routinely been useful and effective at treating the symptoms of BED. Stimulant medications have been a major focus of BED due to their appetite-suppressant side effects. In 2015, lisdexamfetamine (Vyvanse), a type of stimulant that is also used to treat attention deficit hyperactivity disorder (ADHD), was approved by the FDA for the treatment of binge eating disorder[67]. Antidepressants may be routinely prescribed for mood regulation. They have similar effects in BED as they do for BN and have been shown to reduce binge-eating episodes. A variety of SSRIs have a similar impact and do not outperform each other in any notable way[65]. Weight management medications are commonly

prescribed as well, due to concerns about unchecked appetite and weight gain in BED; these medications are sometimes prescribed without consideration for the individual's actual eating behaviors or natural body size. The most common weight management drug used for BED is Topiramate, which showed a side effect of weight loss in other populations. While this medication has been shown to reduce binge-eating frequency, it has many known contraindications, including fatigue, cognitive impairment, metabolic acidosis, interactions with other medications or alcohol, paresthesia, dry mouth, nausea, and headache[65,68]. The most recent off-label medication to gain popularity for the treatment of BED belongs to the class of GLP-1 agonists that go by the popular names of Ozempic, Wegovy, Saxenda, and Mounjaro, to name a few. These medications are known as incretin mimetics and are a group of medications frequently used for managing Type 2 diabetes by stimulating insulin secretion, slowing stomach emptying, and lowering blood glucose levels[68]. It is important to note that there is very little research to support the use of this medication for BED, but what is available suggests that those who took GLP-1 agonists had a greater reduction in BED symptomatology compared to other common weight management medications, and they have an effect on central satiety signaling. As a note of caution, taking any medication with an appetite-suppressant effect without therapeutic support to address the thoughts and behaviors associated with BED could lead to a worsening of the binge-deprivation cycle known to impact the intensity and frequency of binge episodes.

### *Avoidant-Restrictive Food Intake Disorder*

Prescription medication is not usually a first-line treatment for ARFID. There are currently no randomized controlled trials that support the use of medication for this diagnosis, nor are there any FDA-approved drugs. There are several off-label medications often used to address the symptoms of ARFID, including anxiety at

mealtime, lack of appetite, and rigidity in food beliefs. Cyproheptadine (Periactin) is an antihistamine that can stimulate appetite and assist in weight gain; mirtazapine (Remeron) is an antidepressant that also stimulates appetite and has shown some promise in its ability to reduce mealtime fear; lorazepam (Ativan) and olanzapine (Zyprexa) are used to reduce anxiety related to eating and cognitive rigidity[69].

## Nutrition Therapy

Nutrition therapy is an integral part of an individual's recovery from an eating disorder. While it may seem that having a dietitian involved in the treatment of eating disorders is an obvious necessity given the overlap between nutritional diagnoses and the symptoms of an eating disorder, many manualized treatments for eating disorders have no defined role for dietitians. This is, in our opinion, a grave oversight that is fortunately rectified in practice, as dietitians play an important role in multidisciplinary teams across every level of care in the treatment of eating disorders. The role of the dietitian is often individualized depending on the team members and the client's needs. Responsibilities may include (but are not limited to) reviewing growth charts and determining a biologically appropriate weight (BAW), advising the team on the client's energy requirements and refeeding regimen, monitoring a client's micronutrients in response to nutritional rehabilitation, recommending and reviewing food logs, creating meal plans to normalize eating patterns, and establishing behavioral goals to help a client expand their diet and engage in cognitive restructuring focused on deeply held beliefs and thought patterns relating to food[70].

| Complementary and Alternative Medicine (CAM) for Eating Disorders |
| --- |
| |

Several alternative treatments have potential to improve the symptoms of eating disorders. It is important to note that these options are still considered experimental, as there is limited and low-quality evidence to support their use. If pursued, these modalities should be utilized as an adjunct to traditional therapy rather than as the primary approach to healing.

*Acupuncture* is a traditional Chinese therapy that involves inserting fine, sterile needles into specific points in the body. It is thought that this type of stimulation can promote relaxation and improve overall health.

*Arts therapy* is a discipline developed between artists, psychotherapists, educators, and social/health workers that prioritizes creativity in the healing process. It values non-verbal communication including symbolism, imagery, and metaphor as a link to psychological and emotional states[71].

*Biofeedback* involves electronic monitoring of a bodily function to develop consciousness and control over that function. It can address anxiety by digitally displaying heart rate while encouraging the participant to breathe deeply.

*Equine therapy* is an approach to psychotherapy in which horses play an integral part of the therapeutic process. It includes a mental health clinician, a horse, and may also include an equine specialist. The human-horse bond is used as a healing mechanism[72].

*Massage therapy* is a practice of manipulating the body's soft tissues with varying pressure. It may be combined with aromatherapy, heat, meditation, or other methods to enhance the experience.

*Psychedelics-Assisted Therapy* refers to the therapeutic use of potent psychoactive substances. The individual attends a drug-free preparatory session, followed by sessions under the influence of the substance. Individuals are continuously monitored throughout and supported by trained mental health professionals following available guidelines. It is currently utilized in clients with severe presentation that appear treatment-resistant[73].

*Yoga* is a mind-body spiritual practice originating in South Asia. It combines breath control, meditation and holding specific body postures. Many eating disorder treatment facilities have started to incorporate yoga into their therapeutic offerings to promote mindfulness and non-compensatory movement.

## Spotlight on Treatment for Less Common Eating and Feeding Disorders

Much of the current evidence-based treatment protocols are for the diagnoses of AN, BN, BED, and unspecified ED (UFED). Treatment for feeding/eating disorders with little or no body image distress, such as ARFID, rumination disorder, and pica, is generally behavior-based with nutrition and pharmacological therapies playing a secondary role.

### *ARFID*

There is currently no gold standard treatment protocol for ARFID. Current modalities under research and development include: Cognitive Behavioral Therapy–ARFID (CBT-AR), Exposure and Response Prevention (ERP), and The Neurodiversity Affirming Model®. For those with presentations that include a fear of vomiting, there are additional emetophobia protocols.

***Rumination Syndrome***

Diaphragmatic breathing and re-swallowing after rumination are used in "habit reversal training." Other modalities include a speech and language pathologist and esophageal manometry (see more in Chapter 4).

***Pica***

Neuropsychological evaluations and nutritional evaluations should be completed before beginning treatment to rule out any organic causes for eating non-food substances. After that, the primary treatment for pica is psychotherapy. The most common approaches include mild aversive therapy, behavioral therapy, and differential reinforcement. Each of these approaches may include behavior avoidance and learning new coping mechanisms to deal with the accompanying urges.

## Barriers to Eating Disorder Recovery

A recent study conducted by Project Heal, a nonprofit organization focused on addressing the inequities in eating disorder healing, looked at the most common barriers to eating disorder treatment. The findings identified six categories of barriers that can be described as systemic, cultural, healthcare, financial, logistical, and personal[74].

### *Systemic*

Systemic barriers include lack of diversity in research, lack of diverse representation as well as explicit/implicit biases amongst treatment providers, and weight requirements in the DSM-5 that have resulted in weight discrimination within admission criteria for higher levels of care.

### *Cultural*

Examples of cultural barriers identified include social stigma around seeking mental healthcare, anti-fat bias and the resulting thin ideal, lack of body diversity in media, and cultural norms that are permissive of eating disorder behaviors.

### *Healthcare*

In the United States, the insurance system is expensive, and the comprehensiveness of coverage is dependent on employment, leaving immigrants, people with lower levels of education, and those with disability at a disadvantage. People with quality insurance still face barriers to accessing treatment, such as unfair denials of coverage and premature discharges determined by subjective evaluations. In addition, many providers do not have training related to screening for or treating eating disorders and many specialized outpatient providers don't accept insurance.

### *Health Status*

Due to the siloed nature of most healthcare facilities, having co-occurring medical or psychological conditions makes eating disorder treatment more difficult to access. Clients may be turned away from eating disorder treatment programs due to the added complexity of managing additional conditions, such as type 1 diabetes, food allergies, kidney conditions, substance use disorders, enteral or parenteral nutrition feeds, and others. Furthermore, thorough treatment options for lesser-known eating disorders (e.g., ARFID, "atypical anorexia") may be lacking.

### *Financial*

Most eating disorder treatment in the United States is exorbitantly expensive, even with health insurance, due to high deductibles, high out-of-pocket maximums, and high co-pays. Furthermore, most higher levels of care require patients to take time off work for

treatment, which may jeopardize their financial security. While most facilities offer some type of scholarship, and there are nonprofit organizations that attempt to fill the gap, these initiatives fall short of addressing the high need amongst individuals experiencing poverty and lower incomes.

### *Logistical*

Geographic barriers, language barriers, and technological barriers may hinder access to treatment. Many people seeking eating disorder treatment live in locations that are far from where treatment centers or in-person outpatient providers operate. It can also be challenging to find trained providers that offer services in languages other than English. Lastly, while virtual care would seem to address the geographical issues, most virtual care requires reliable high-speed internet, technological devices, and privacy, all of which can be a challenge.

### *Personal*

Healing from an eating disorder can run counter to the cultural reinforcement of disordered behaviors. Individuals may experience denial or even shame that they struggle to recover on their own. Other personal barriers to recovery might include lack of family or social support, lack of clear diagnosis, and/or past treatment trauma.

**Harm Reduction in Eating Disorders**

Harm reduction refers to a range of practices and public health policies designed to lessen the negative social and/or physical consequences associated with stigmatized human behaviors. It was first introduced as an approach to substance use disorders where abstinence, though ideal, was complicated by some of the same barriers we see in the

eating disorder world (e.g., financial barriers, stigma, lack of social support, etc.). In the eating disorders field, there has been a growing call to incorporate these harm reduction strategies, especially for those who have not found success in abstinence-focused approaches. Under a harm reduction model, providers and clients explore ways to minimize the risk of physical and emotional harm while engaging in disordered eating behaviors[75].

There may be ethical concerns for providers who use harm reduction strategies, as it has the potential to hinder treatment outcomes by not pushing clients to resolve their ambivalence and move towards abstinence. Proponents argue, however, that with recovery rates as low as they currently are, it is equally harmful for providers to deny clients strategies that might help them maintain a better health status than they would otherwise achieve with traditional models of care. Current guidance suggests that harm reduction should only be used for folks with enduring symptoms who have tried multiple abstinence-focused modalities without success and are considering terminating treatment altogether. Harm reduction is not considered a viable option for patients with AN who are younger or those who are not at a significantly low weight[75].

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CHAPTER 3

# Gastrointestinal Foundations

## Overview of Gastrointestinal Anatomy and Physiology

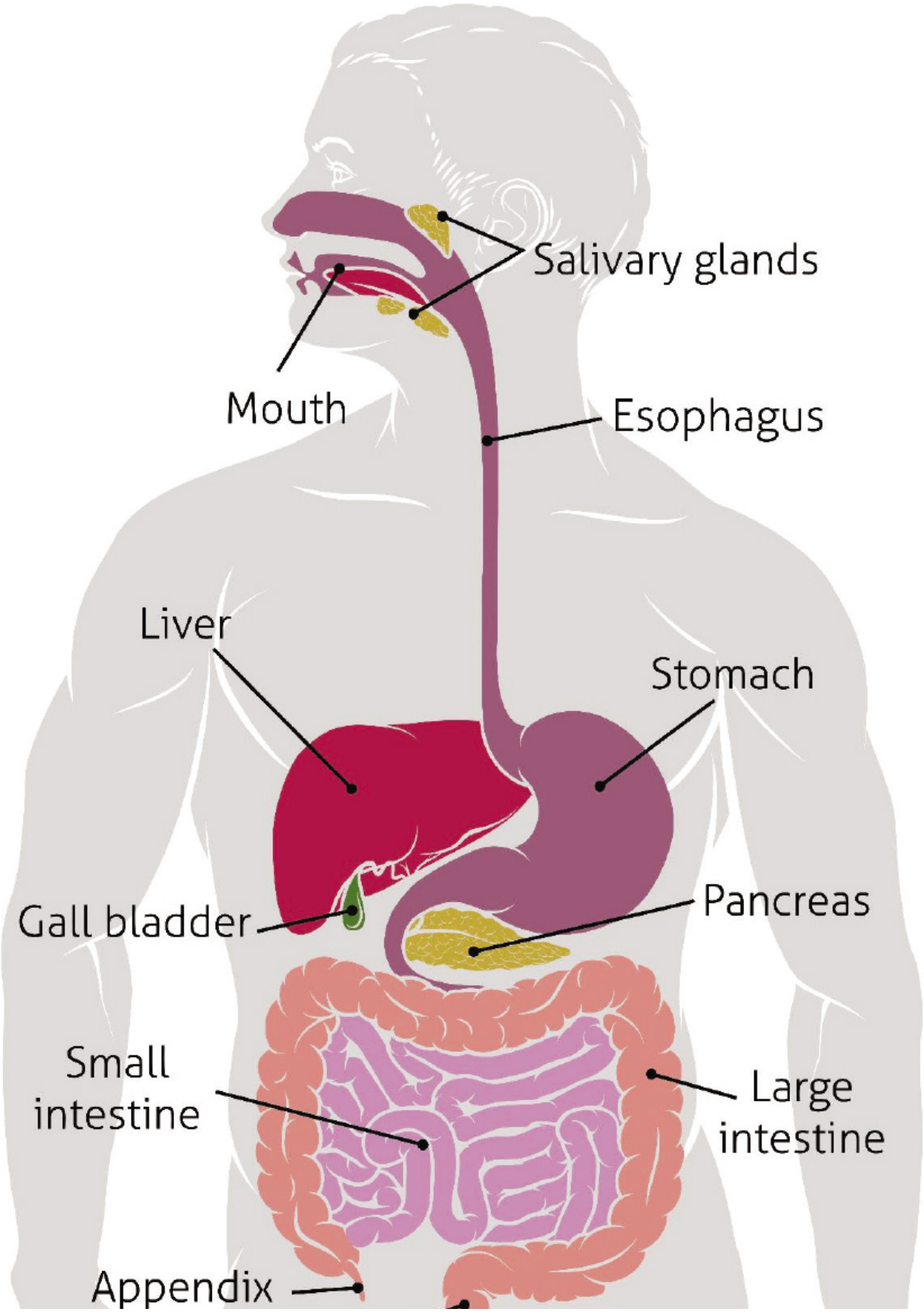
The gastrointestinal (GI) tract is a highly specialized group of organs tasked with breakdown (digestion) and extraction of nutrients (absorption) from the food we eat, maintenance of fluid and electrolyte balance, protection from pathogens (immune function), and mediation between the microbiome and health. Each portion of the GI tract is highly specialized to carry out specific functions, with the only conscious efforts required in the journey food makes from mouth to anus being those of chewing and defecation.

## Key Structures of the Gastrointestinal Tract

### The Tubular GI Tract

The GI tract consists of a series of tubular structures running from the mouth to the anus: the esophagus, stomach, small intestine (made up of duodenum, jejunum, and ileum), and large intestine or colon (made up of the cecum, ascending colon, transverse colon, descending colon, sigmoid colon, and rectum illustrated in Figure 3.1). Several portions of the tubular GI tract are separated by muscular barriers known as sphincters, which open and close to allow forward and prevent backwards passage of the food bolus.

The upper esophageal sphincter (UES) and lower esophageal sphincter (LES), respectively, are contracted at baseline to prevent passage of stomach contents into the esophagus (LES) and oral cavity and airway (UES), and actively open to allow food into the stomach. The pylorus regulates the outflow of food contents from the stomach into the small intestine, and the ileocecal valve allows passage of digested food into the colon. Finally, the anal sphincters prevent leakage of stool at baseline in their contracted states and relax to permit defecation.


Salivary glands
Mouth
Esophagus
Liver
Stomach
Gall bladder
Pancreas
Small intestine
Large intestine
Appendix

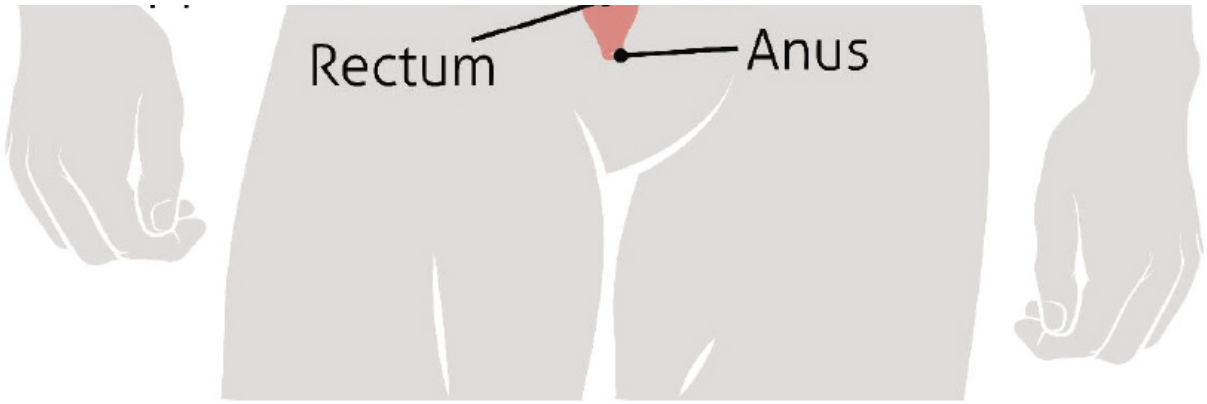



**Figure 3.1.** *Anatomical structures of the gastrointestinal system*

### Table 3.1. Layers of the Tubular GI Tract

| Layer | Functions |
|---|---|
| Mucosa (innermost) | Directly contacts food contents in the lumen and is made up of epithelial (surface lining) cells with various functions: secretion, absorption, or hormone production |
| Submucosa | Connective tissue layer beneath mucosa with blood vessels, lymphatics, and nerves |
| Muscular layer | Inner circular layer, outer longitudinal layer, and in the stomach a third oblique innermost layer. The muscular layer regulates movement of food contents through the GI tract |
| Serosa (outermost) | Thin layer of connective tissue reinforcing the GI tract and covered by epithelium, which protects the GI tract and decreases friction of organs as they move in the abdominal cavity |

## Accessory Organs of the GI Tract

Several accessory organs secrete into the GI tract to help with digestion of food. The salivary glands produce saliva, which acts as a lubricant to a food bolus and contains the digestive enzymes, liqual lipase and salivary amylase. The pancreas has both endocrine (insulin) and exocrine (digestive enzymes) function. The liver and pancreas secrete bile and digestive enzymes through the common bile duct and main pancreatic duct, respectively, through the shared ampulla of Vater, and into the second portion of the

duodenum to mix with food contents. The gallbladder is connected to the common bile duct and stores extra bile to secrete in the event that a higher fat meal is consumed.

## Nervous System of the GI Tract

Nerves signal the gut to move, secrete, and absorb, and several branches of the nervous system play a role in normal GI function. The GI tract is influenced by both the central nervous system, or CNS (i.e., brain, brain stem, and spinal cord), and the enteric nervous system, or ENS. The CNS includes the autonomic nervous system (ANS), which is made up of two divisions—sympathetic ("fight, flight, freeze, or fawn") and parasympathetic ("rest and digest")—with greater sympathetic tone shunting blood flow away from the GI tract and decreasing normal GI function and the latter increasing GI functions. The vagus nerve, cranial nerve I, is the major nerve involved in the parasympathetic division of the ANS, though 80% of its fibers are afferent, carrying information from peripheral organs, such as the GI tract, back to the CNS[1].

The GI tract also has its own intrinsic nervous system, the enteric nervous system (ENS). The ENS is often referred to as "the second brain" due to its complex organization of over 500 million nerves, more than 20 neurotransmitters used, and breadth of functions controlled[2]. The ability of the ENS to function independently from the central nervous system (e.g., brain, spinal cord) was shown by a famous experiment in which the tubular GI tract was able to move a food pellet from the end of the mouth to the end of the anus despite being disconnected from the body[3]. The communication between the brain and GI tract is often referred to as the "brain-gut axis," with communication occurring in both directions: brain to GI tract and GI tract to brain[4]. Disorders of gut-brain interaction (DGBIs), such as irritable bowel syndrome (IBS), may occur when the brain-gut axis is disrupted.

### Blood Supply of the GI Tract

The GI tract receives its supply of oxygenated blood from three branches off of the aorta (the largest artery in the body): the celiac artery, superior mesenteric artery, and inferior mesenteric artery. Blood returns to the liver through the portal vein and then to the right side of the heart, where it is pumped to the lungs to pick up further oxygen. From the lungs, the oxygenated blood returns to the left side of the heart to be pumped out into systemic circulation through the aorta once again.

## A Trip Through the GI Tract

What follows is a detailed, integrated description of what happens to food from the point that it enters the mouth until it exits the anus as stool. However, the mere thought, smell, and/or sight of food can cause salivation and secretion of stomach acid. Thus, the table is figuratively set for digestion to occur before food even enters the body. See the box below for a brief experiential mindfulness exercise related to this.

**Mindfulness Exercise**

Read the following passage, and then close your eyes to imagine the scene described.

*Imagine that you are in the kitchen. On the counter is a ripe, juicy lemon, a knife, and a cutting board. You pick up the lemon and roll it around in your hand, feeling the texture of the peel and the gentle give that it has. As you gently squeeze it, you notice a faint scent of lemon in your nostrils. You place the lemon on the cutting board, pick up the knife, and cut the lemon in half. A small amount of lemon juice squirts onto the counter and the smell of the lemon is even more intense. You place the half of the lemon still in your hand to your nose and*

*inhale a strong lemon smell. Finally, you take a small bite of the edge of the cut lemon and taste its sourness.*

Reflection questions:

- What did you notice during the exercise?
- Were certain senses—sight, smell, or taste—easier for you to imagine?
- Did you notice yourself salivating and/or your face/lips puckering when you imagined taking a bit of lemon?

## Mouth and Esophagus: Chewing and Swallowing

Food entering the body passes through three phases of deglutition ("swallowing")—oral, pharyngeal, and esophageal—which prepare and carry food from the mouth down the esophagus to the stomach.

### *Oral phase*

As soon as food and drink enter the mouth, the tongue and teeth begin mechanically breaking them into smaller pieces. Different types of teeth cut (incisors), tear (canines), and grind (molars) food during mastication, or "chewing." Chewing involves the strongest muscle per size in the body, the masseter, which closes the jaw to crush food. Despite the common belief that chewing is voluntary (e.g., a mother telling her child to "Chew your food!"), there are both voluntary and involuntary mechanisms at play. Stretching of muscles of mastication leads to reflexive contraction, followed again by relaxation, to rhythmically chew food. Food is also mixed with saliva from the salivary glands, which contains water, mucin (a protein which helps with lubrication), antimicrobial compounds, buffering agents, and digestive enzymes to begin chemical digestion. Saliva contains both salivary amylase for carbohydrate digestion, as well as lingual lipase for fat digestion. Roughly 600

milliliters (2.5 cups) of saliva are produced per day in adults[5]. At the end of the oral phase, the tongue separates a small portion of chewed, softened, and lubricated food and pushes it to the back of the throat, or oropharynx. From this point on, the remainder of the journey of food through the GI tract is completely involuntary until defecation occurs.

### *Pharyngeal phase*

When the bolus of food hits the highly innervated oropharynx, a series of reflexive movements occur to move the food into the esophagus: the soft palate at the posterior aspect of the roof of the mouth lifts to close and keep food from entering the nasal passages, the epiglottis folds backwards to close and prevent aspiration of food into the airway, and the upper esophageal sphincter (UES) relaxes from its contracted, resting state to allow food into the upper esophagus.

### *Esophageal phase*

As soon as the food bolus passes through the UES, peristalsis begins to push the food bolus down the esophagus and into the stomach. Peristalsis occurs as a coordinated, sequential contraction above the bolus and relaxation ahead the bolus and progresses down the esophagus until the food empties into the stomach. Primary peristalsis occurs when a swallow is initiated, whereas secondary peristalsis is the intrinsic capability of the esophagus to initiate peristaltic contractions in the upper esophagus without a swallow. Secondary peristalsis often occurs when an initial swallow does not clear a food bolus from the esophagus or as a response to clear acid reflux back into the stomach. Despite some contribution of gravity to the esophageal phase, humans can swallow upside down and still have peristalsis push the food bolus into the stomach. As peristalsis approaches the distal esophagus, a second muscular ring that is contracted at baseline, the lower

esophageal sphincter (LES), relaxes to allow passage of food into the stomach. The LES is a muscular ring with contributions from the phrenoesophageal ligament, crura of the diaphragm, and circular muscle of the distal esophagus. The LES relaxes periodically to help vent gas from the stomach, and these transient LES relaxations (TLESRs) are also one of the main contributors to gastroesophageal reflux disease (GERD)[6].

## Stomach

The stomach is responsible for mechanical and chemical digestion. The portion of the stomach nearest the opening of the esophagus is the cardia. The fundus is the rounded upper/proximal portion of the stomach just next to the cardia, and the largest portion of the stomach is the proximal, tubular structure known as the gastric body. The distal portion of the stomach just proximal to the pylorus is the antrum.

The lower esophageal sphincter (LES) relaxes with each swallow to allow food to enter the stomach and then promptly closes to prevent reflux into the esophagus. In preparation to make room to receive food, the fundus expands in a process known as accommodation. The stomach is able to expand up to five-fold its baseline volume after meals. As the stomach fills with food, the LES (inflow of the stomach) and pyloric sphincter (outflow of the stomach) close, and the stomach contracts in increasingly strong wave-like patterns to grind food against the antrum and closed pylorus.

The stomach serves as more than a mere vessel for storage and grinding of food. At the same time as mechanical digestion is occurring, the stomach also contributes to chemical digestion of food. The stomach is strongly acidic, with a pH of 1-2 (the pH of the human body is much more alkaline at 7.4), though acid is only part of chemical digestion. The lining of the stomach contains many

specialized cells that contribute to gastric secretions that perform many different and interconnected functions. See Table 3.2.

## Table 3.2. Gastric Cells and Their Functions/Products

| | |
|---|---|
| Parietal cells[7] | Located in the body and fundus, parietal cells produce hydrochloric acid (HCl) and intrinsic factor<br><br>*Hydrochloric acid*<br>• Helps digest protein, kill bacteria, and inactivates salivary amylase<br>• Produced at a basal rate that is 10 to 15% of the maximum capacity<br>• Secretion is increased by 1) muscarinic acetylcholine receptors when stimulated by acetylcholine from the parasympathetic nervous system, 2) histamine type 2 receptors when stimulated by histamine released by enterochromaffin-like cells (ECL), and 3) gastrin receptors when stimulated by gastrin released by G-cells of the antrum. Both gastrin and acetylcholine directly stimulate parietal cells to secrete acid, and gastrin also indirectly does so by stimulating ECL cells to make histamine which stimulates receptors on parietal cells. Histamine potentiates the effects of acetylcholine and gastrin to allow smaller amounts of these to cause acid secretion.<br><br>*Intrinsic factor*<br>• Needed for absorption of vitamin B12 and the only product of gastric secretion that humans cannot live without |
| Chief cells | *Pepsinogen*<br>• Pepsinogen, the inactive precursor of pepsin, is the enzyme that breaks down protein into amino acids<br>• Acid required for the conversion of pepsinogen to pepsin |
| Mucus cells | *Mucus*<br>• Mucus with bicarbonate forms a layer that lines the stomach to protect it from acid and pepsin |
| G-cells | *Gastrin*<br>• Secreted from G-cells in response to stretch of the stomach, protein, and/or increased pH (i.e., less acidity) |

| | |
|---|---|
| | • Major direct stimulus for parietal cells to make acid<br>• Indirect stimulus for parietal cells to make acid by stimulating ECL cells to make histamine, which then acts on parietal cells |
| Enterochromaffin- like (ECL) cells | *Histamine*<br>• Stimulated by gastrin, then potentiates effects of gastrin and acetylcholine to stimulate parietal cells to make acid |
| D-cells | Located in the antrum (and less so in the duodenum and pancreas) and release somatostatin<br><br>*Somatostatin*<br>• Released to inhibit gastric secretion, slow gastric emptying and intestinal motility, and to inhibit several other non-GI hormones (e.g., thyroid stimulating hormone, prolactin, human growth hormone) |

Gastric secretion occurs in three phases[7]:

1. ***Cephalic phase:*** Occurs before food is ingested, stimulated by the sight, smell, and/or thought of food, and accounts for 30 to 50% of gastric secretion. Mostly driven by vagal nerve release of acetylcholine to stimulate parietal cells to secrete acid and chief cells to make pepsinogen (which is cleaved to the active pepsin in an acidic environment). Vagal efferents also stimulate G-cells using gastrin-releasing peptide (not acetylcholine) to release gastrin, which indirectly stimulates acid secretion by acting on parietal cells. The cephalic phase is inhibited by stimuli of the sympathetic nervous system, such as physical or psychological distress.
2. ***Gastric phase:*** Begins when food enters the stomach, stimulated by stretch/distention and breakdown products from proteins (i.e., amino acids and peptides), and accounts for 40 to 60% of gastric secretions. Distention triggers vago-vagal nerve reflexes in which

the stretch is sensed and efferent fibers release acetylcholine to stimulate parietal cells and gastrin-releasing peptide to stimulate gastrin release from G-cells. Increased pH from food buffering the baseline acidic environment also stimulates G-cells to release gastrin. The gastric phase of secretion is inhibited by somatostatin from D-cells, which are activated by decreased pH (i.e., increased acidity). Somatostatin inhibits parietal cells from secreting acid, ECL cells from producing histamine, and G-cells from producing gastrin.

3. ***Intestinal phase:*** Triggered by partially digested proteins entering the small intestine and stimulating duodenal G-cells to secrete gastrin and other hormones that lead to gastric acid secretion. Accounts for 5 to 10% of gastric secretions.

The stomach empties in a highly regulated manner as mechanical and chemical digestion turn food into chyme, a sludge like consistency. Neurohormonal control of gastric emptying is so precise that fatty acids longer than 12 carbon molecules long stimulate the production of a peptide, cholecystokinin (CCK), which inhibits gastric emptying[8]. Liquids empty much more quickly than solids and leave the stomach through a groove that runs along the lesser curve (top) of the stomach called "Magenstrasse." The pylorus opens to allow solid food particles that are 2 to 3 millimeters through. The typical amount of time it takes for the stomach to empty is 2 to 4 hours[9].

## Small Intestine

Food broken down to chyme enters the small intestine from the stomach and passes through three segments of the small intestine –the duodenum, jejunum, and ileum–over an average of 2 to 6

hours. The small intestine is the primary site of absorption of nutrients, most of which are absorbed in the duodenum and jejunum. The terminal portion of the third segment, the ileum, is the site of absorption of vitamin B12, bile acids, and fat-soluble vitamins. See Figure 3.2 for the sites of absorption of macro- and micronutrients (i.e., vitamins and minerals).

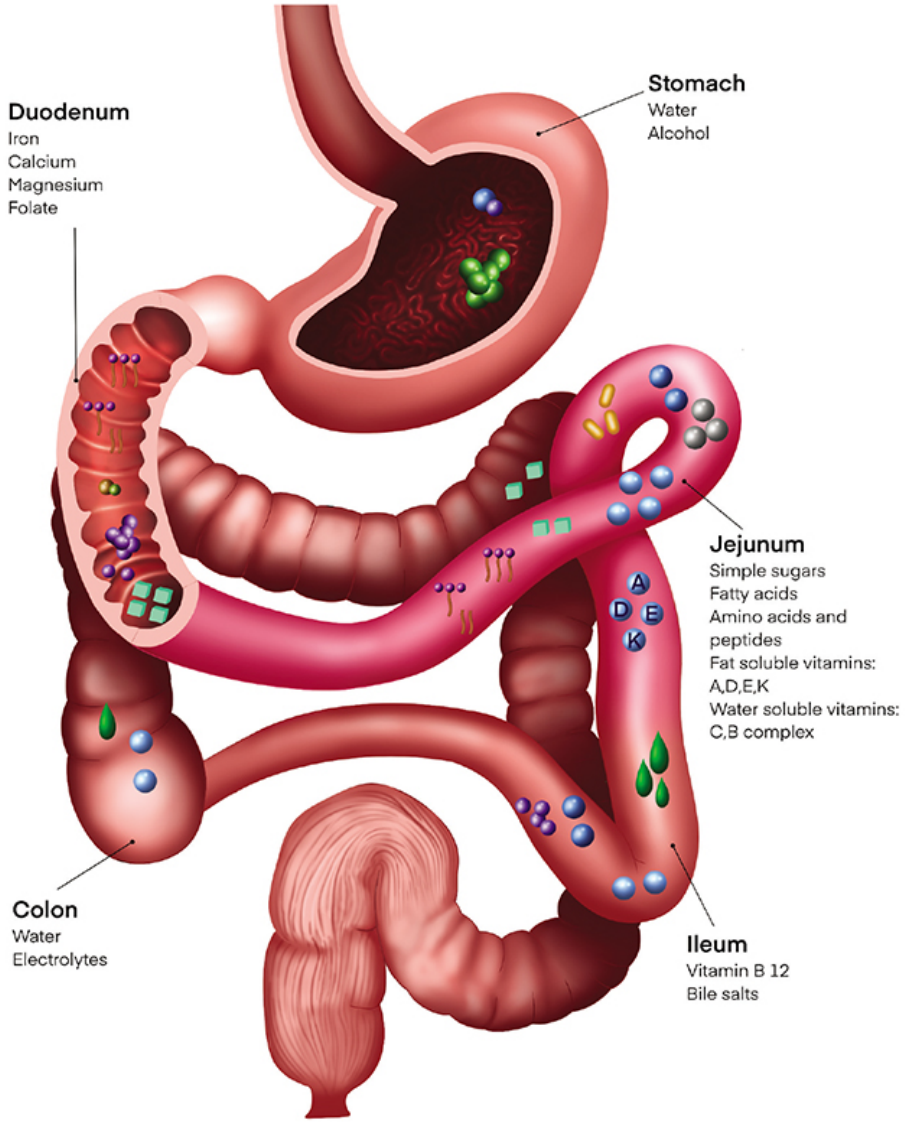



*Figure 3.2. Gastrointestinal tract absorption sites*

The small intestine is 15 to 22 feet (450 to 670 cm) long in adults. The surface area of the small intestine is approximately the size of a

tennis court due to its length and microvillus projections. Each villus consists of a capillary to absorb glucose and amino acids and a lacteal to absorb fatty acids. Capillaries drain to the portal vein and then the liver and lacteals to the thoracic duct.

Movement of food through the small intestine consists of several types of contractions, each with their own function. Peristalsis is the propulsion of bowel contents forward, with contraction of the bowel proximal and relaxation distal to the contents. Segmentation breaks contents into small portions to avoid overwhelming the digestive capacity of the small intestine. Mixing movements lead to a back-and-forth movement of contents to mix chyme with digestive enzymes from the pancreas and bile acids from the liver and gallbladder and to maximize contact with microvilli.

The migrating motor complex (MMC) is a specific series of patterns of electrical activity that occur every 90 to 230 minutes in the GI tract during fasting between meals that sweep indigestible components of food from the stomach into the colon[7]. Phase I of the MMC (40 to 60% of the time) consists of electrical slow waves that do not reach the point of causing action potentials and thus do not result in contractions. Phase II of the MMC (20 to 30% of the time) consists of increasing frequency of action potentials and contractions but in an erratic, non-propulsive manner. Phase III of the MMC (5 to 10 minutes) results in large amplitude contractions starting at the distal gastric body and antrum that migrate to the distal ileum. While phase III of the MMC occurs, the pylorus and ileocecal valve relax to allow the MMC to move residual intestinal contents into the colon. Phase IV is the gradual inhibition of contractions and return to the quiescent phase I. Eating and vagal stimulation immediately inhibit the MMC. Failure or frequent disruptions of the MMC can lead to small intestinal bacterial overgrowth, or SIBO, which is associated with multiple chronic GI

and non-GI conditions and can result in bloating, gas, altered stools (diarrhea more often than constipation), and malabsorption.

## Pancreas

The pancreas has endocrine and exocrine functions. The endocrine pancreas releases insulin from beta cells and glucagon from alpha cells to regulate blood glucose levels and mediate energy storage and metabolism. In response to elevated blood glucose levels after meals, insulin is secreted to store glucose as glycogen in cells throughout the body, thereby reducing blood glucose levels. Glucagon is released when blood glucose levels are low, leading to breakdown of glycogen from the liver to release glucose into the bloodstream for energy.

## Liver and Gallbladder

For the purposes of digestion, the main role of the liver is the production of bile. Bile is produced by the liver in a continuous fashion and flows out the ampulla of Vater and into the second portion of the duodenum to mix with food as it is released into the small intestine from the stomach. Bile acts as an emulsifier of fat to break it into smaller pieces in order to allow pancreatic lipase to further cleave fats into glycerol and fatty acids. Additional bile is stored in the gallbladder and is available to help break down higher fat meals. Contraction of the gallbladder occurs in response to CCK, which is released by enteroendocrine cells in the duodenum in response to large amounts of fat in the small intestine.

Other functions of the liver are listed in Table 3.3.

**Table 3.3. Liver Functions**

| | |
|---|---|
| Bile acid production | Bile is produced by the liver to act as an emulsifier to aid in the digestion and absorption of fat. |
| | |

| | |
|---|---|
| Protein synthesis | Proteins are built from amino acids in the liver and utilized to create factors that both help form and break down clots, carrier proteins for other substances (e.g., transferrin for iron), and thrombopoietin, a protein that stimulates the bone marrow to make more platelets (a type of blood cell that helps initiate clot formation). |
| Cholesterol synthesis | 80% of cholesterol production occurs in the liver, with only 20% from the diet. Cholesterol is then used as a key component of cell membranes, chemical messengers for signaling between cells, and is the precursor for vitamin D and all steroid hormones such as cortisol, estrogen, and testosterone. |
| Detoxification from toxins and drugs | Modifies drugs and toxins by making them water or fat soluble to be excreted in the urine or stool, respectively. |
| Storage of nutrients | Storage of glycogen (storage form of glucose), vitamins A, D, E, K, and B12, as well as iron and copper. |
| Immune functions | The liver is rich in immune cells which help remove bacteria and other pathogens from the blood that flow from the intestines back to the liver. |
| Red blood cell production | Erythropoiesis, or red blood cell production, begins before birth in the liver, spleen, and yolk sac of the fetus. By the time of birth and afterward, it occurs in bone marrow of many bones. |

## Colon (Large Intestine)

The colon is 5 feet (150 cm) long and divided into cecum (proximal-most portion where the appendix is attached and where the terminal ileum empties into), ascending colon, transverse colon, descending colon, sigmoid colon, and rectum. The ileocecal valve regulates emptying of small bowel contents into the colon at the junction of the ileum and cecum and prevents backflow of colonic contents and bacteria into the small intestine. The colon is responsible for absorption of water and electrolytes and propelling the remaining feces to the rectum for defecation, with passage through the colon usually taking 30 to 40 hours and ranging from 10 to 59 hours. The bacteria of the colon produce several B vitamins, vitamin K, and short chain fatty acids.

## Anorectum/Pelvic Floor Muscles

The final passageway of the remnants from ingested food is the anorectal canal. Here, a combination of voluntary and involuntary processes occurs to regulate defecation. The anus and rectum are exquisitely sensitive and are able to sense the differences between solid, liquid, and gas contents to avoid accidents. Two sphincters, the internal anal sphincter (IAS) and external anal sphincter (EAS), are contracted at baseline to prevent loss of feces. The EAS is a skeletal muscle and is under voluntary control. Control of the IAS is involuntary, and it is contracted at baseline. A third key muscle, the puborectalis, is a sling-like muscle originating from the pubic bone and extending posteriorly around the anorectal junction. The muscle is contracted at baseline, is under voluntary control, and acts in a purse string-like fashion to close the anorectal canal at baseline and opens to allow passage of feces. When the rectum fills with stool, the distention causes both the urge to defecate and relaxation of the puborectalis and IAS muscles to allow feces to move into the distal rectum and closer to the anus. The EAS is held contracted until ready to defecate. With relaxation of the puborectalis straightening the anorectal angle and relaxation of the IAS and EAS allowing easy passage of the stool, a gentle contraction of the abdominal wall should generate enough pressure to push the feces out the anal canal and complete defecation.

## Microbiome

The GI tract contains a similar number of bacteria as there are human cells in the body—30 to 40 trillion—which weigh a mere half of a pound (0.2 kilograms). The stomach contains $10^{2}$ to $10^{3}$ bacteria and the colon $10^{9}$ to $10^{12}$, while the small intestinal bacterial counts range from stomach-like amounts in the duodenum to $10^{4}$ to $10^{7}$ in the terminal ileum[11]. The microbiome plays significant roles in health and disease, including nutrient

metabolism, drug and toxin metabolism, maintenance of the integrity of the mucosal barrier, modulation of the immune system, and protection against pathogens.

Microbiome changes have been associated with many disease states. Despite various methods aimed at manipulating the microbiome, the ability to do so for therapeutic purposes is still in its infancy for most conditions, and the marketing of microbiome treatments has outpaced science. It has also been difficult to ascertain when the microbiome changes are contributing to disease versus a downstream outcome of the underlying conditions. Diet is one of the key modifiable factors contributing to the composition of the microbiome, and significant ongoing research is investigating dietary treatments for specific conditions.

### GI Immune System

The GI tract runs through the core of the body, yet it is open to the environment at both ends. Given the potential exposure of the GI tract to ingested pathogens and toxins, several measures are in place to protect humans from infection and injury. The epithelial cell layer, mucin, and stomach acid all serve as physical barriers. In addition, the GI tract is lined with up to 70% of the body's immune system cells that protect from infection by ingesting and neutralizing harmful pathogens[12]. The cells of the immune system in the GI tract influence and are influenced by the enteric nervous system and microbiome to maintain homeostasis.

## Potential Role of Gut Microbiome in Eating Disorder Etiology and Pathogenesis

The etiology and pathogenesis of eating disorders are multifactorial and complex. Emerging and growing evidence indicates the composition of the gut microbiota may play a

significant role in the development and maintenance of eating disorders in individuals who have increased genetic susceptibility to their development. This review will highlight the known relationships between microbiota and eating disorders, mechanisms by which the microbiome influences mental health, and proposed nutritional approaches as a targeted therapeutic intervention in the treatment of eating disorders and mental health concerns.

## Introduction

Humans harbor vast amounts of microbes, which linger on the skin, in hair, and inside mucosal cavities, like the nose and mouth. The community of bacteria, archaea, fungi, and other microorganisms that reside within the gastrointestinal tract are referred to as the *gut microbiota*. The gut microbiota, their metabolites, signaling molecules, and environmental conditions are collectively called the *gut microbiome*[13] (Figure 3.3). Much like the Amazon rainforest, this inner ecosystem can foster an incredible amount of biodiversity, and each microorganism's relationship with its host and one another is the subject of intense research. A particular species might exert an effect on the host, depending on its characteristics, population, and surroundings. Adding to the complexity, that same species' *suppression* may also have a pronounced effect by leaving a vacuum in which other species can thrive. A race is underway to understand the relationships between the various bacteria that make up the microbiome. Researchers are asking:

- *Which microbes positively affect human health?*
- *Which are detrimental?*
- *At what level of abundance do they switch from being beneficial to detrimental?*

- *What is required to nurture desirable microbes?*
- *Are changes to microbial diversity permanent, or do they require ongoing "management"?*
- *What are the long-term effects of induced microbiome changes?*

These are just a sampling of questions currently being investigated for the purpose of maintaining human health and treating a variety of illnesses. Significant microbiome research has occurred in the eating disorder field due to promising distinctions between affected individuals and "healthy" controls that point to development of potential therapies. However, the majority of this research is limited to applications in anorexia nervosa.

## Findings

Anorexia nervosa (AN) has the highest mortality of all psychiatric disorders. Treatment is not always accepted or sustained, and many co-occurring conditions can complicate the road to recovery (e.g., other psychological disorders, gastrointestinal disorders, etc.). Even among AN patients receiving intensive treatment, it is estimated that more than 10% do not achieve full recovery, and their eating disorder develops into a chronic condition[14]. Understanding the biological mechanisms and risk factors underlying AN (and all forms of eating disorders) might allow for alternative therapies that achieve better recovery outcomes.

Growing research shows profound alterations of the gut microbiome associated with AN-induced starvation. Findings show a reduced amount of total bacteria in the gut microbiome of those with AN. Additionally, *alpha diversity*, which is a measure of the number of different species in an individual's sample, has been shown to be reduced in acutely ill AN patients. Alpha diversity also appears to increase with weight rehabilitation. It is speculated that

lower alpha diversity implies disequilibrium of the microbial community and a lesser ability to withstand disturbances. Some studies have additionally found the *beta diversity* (a measure of the microbiome differences between samples) to be higher amongst AN patients compared to controls, demonstrating more heterogeneity across the sampled AN population[15-18].

Weight gain in the case of patients with AN does seem to impact the microbiome. Several studies demonstrate species-specific changes with weight restoration[16,19]. However, even with short-term weight recovery, AN patients' microbiomes still appeared more similar to their own microbiome under the conditions of acute starvation than those of healthy controls[15,16,17,19]. This suggests there may be microbiome markers of AN–a theory that is underscored by the fact that differences in the microbiome of restrictive and binge/purge subtypes of anorexia nervosa have also been noted[17].

## Mechanisms

Many studies have demonstrated differences in the gut microbiome between eating disorder patients and controls; however, the pathology and mechanisms of this difference are only just beginning to be investigated. Several studies propose a *gut-brain-microbiome (GBM) axis* model[20]. They suggest bidirectional communication between the GI tract, central nervous system, and gut microbiome–with diet likely playing a moderating role. The resulting interactions may have the potential to affect gut function, blood-brain barrier function, hormones, neurotransmitters, and the development of neuropsychiatric diseases[19,20] (Figure 3.3).

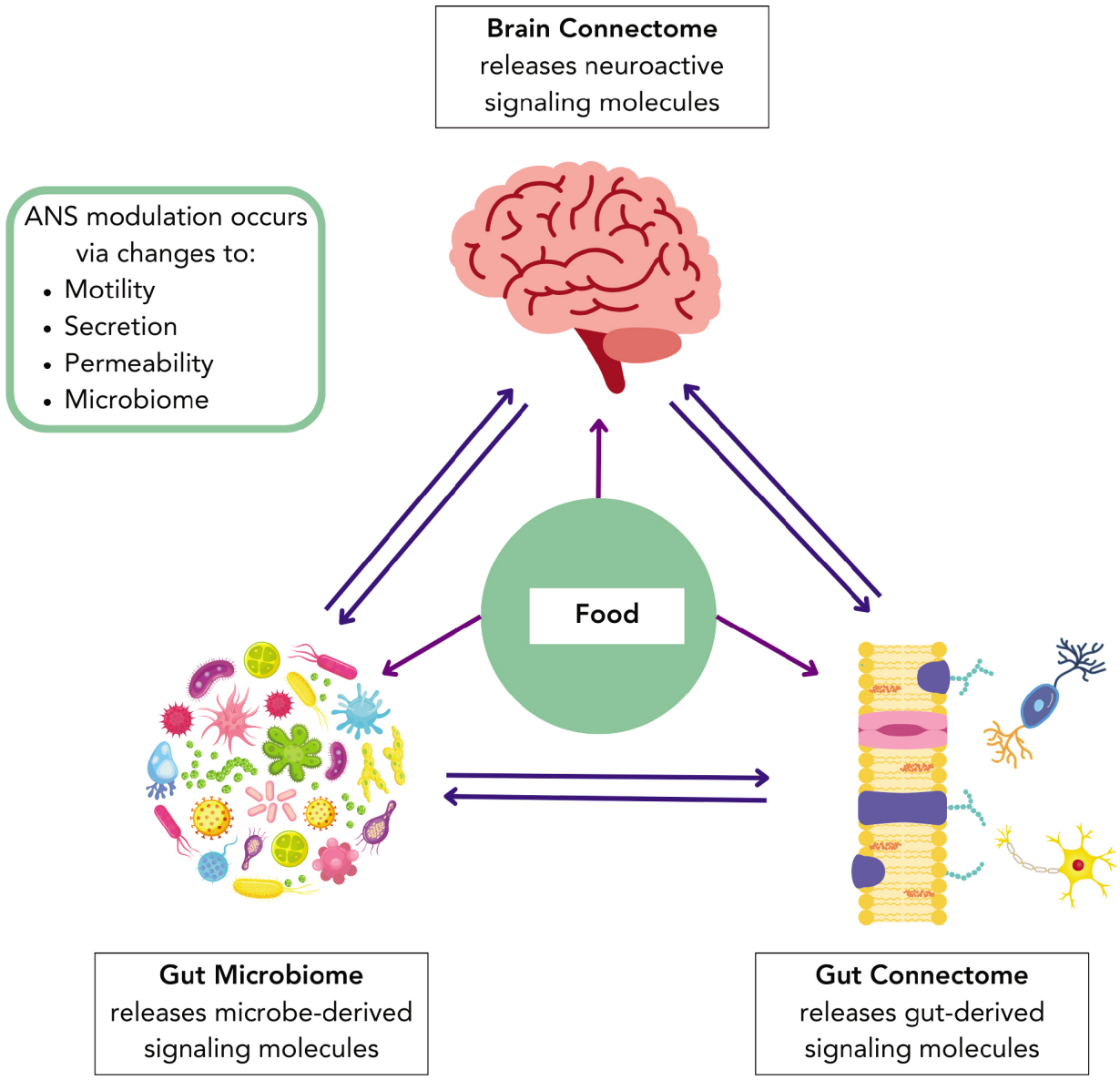



*Figure 3.3.* **The proposed gut-brain-microbiome axis model.** *Adapted from Martin et al., 2018.*

The concept of the gut-brain-microbiome axis offers a model by which researchers are exploring how to harness the microbiome for the potential treatment of mental disorders. The microbiome is capable of chemically transforming substrates into gut signaling molecules. The sources of these substrates can be host-derived, diet-derived, microbe-derived, or newly synthesized. The resulting

signaling molecules can influence immune, nerve, and endocrine cells in the GI tract as well as other organs, including the brain[20].

An example of one such pathway involves serotonin. Depending on the diet of the host, gut microbes might produce short-chain fatty acids and branched amino acids. These signaling molecules stimulate serotonin release by specialized enterocytes called enterochromaffin cells. The released serotonin might connect with receptors in the brain (affecting sleep, intake, mood, pain) or in the gut (affecting motility, secretion). Other pathways involving downstream nutritional metabolites are somewhat known, but more remain to be elucidated[20].

## Limitations

The microbiome is a promising area of study for the treatment of eating disorders. The research that exists, though, is severely limited by its homogeneity. The vast majority of relevant studies focus on anorexia nervosa, and most without distinction between restricting types, binge-purge types, and “atypical” presentations.

Furthering the oversimplification of anorexia nervosa, several studies use modest, short-term weight gain as a proxy for “recovery” (meaning that mental, emotional, and behavioral aspects of robust eating disorder recovery are not taken into account).

In studies involving human subjects, the participants are overwhelmingly female; conversely, the subjects of animal studies are usually male rats, making it difficult to extrapolate some findings. Future research will need to consider more diagnoses and strive for gender, body size, and racial diversity across study participants in order to truly serve people affected by eating disorders.

## Conclusion

While the microbiome offers potential avenues for future targeted therapies for eating disorders, the current body of evidence is limited in its ability to point to individual nutrition prescriptions. There is modest support for a varied, plant-forward, anti-inflammatory dietary pattern to support a diverse microbiome and reduce eating disorder-related comorbidities[20-21]. However, current research is dominated by observational studies using inconsistent measures of broad mental health concerns, such as depression and cognitive decline. Further research should aim to design robust studies that can demonstrate the potential benefits (or lack thereof) of diet on the development or prognosis of eating disorders specifically. These kinds of findings could have immense impacts in the prevention and treatment of eating disorders, further reducing blame, recidivism, and suffering for affected individuals and implementing a more multifaceted treatment approach.

### Too Soon to Recommend

Although probiotic products are widely available, current research does not yet support their use in most clinical scenarios. There are studies that suggest certain probiotics strains or combinations may positively impact symptom scores, but this type of analysis is too much in its infancy to provide standard recommended doses[23]. The American Gastroenterology Association Clinical Practice Guidelines for the Role of Probiotics in the Management of Gastrointestinal Disorders[24] states specific strains may be useful in the case of *C. difficile* infection, antibiotic-associated diarrhea, and pouchitis. We do not generally recommend probiotics outside of these issues.

Microbiome testing has also become popular in recent years and measures the number of microbe species and their relative populations in the GI tract via stool samples. Current research has not established standards for microbiome biodiversity or reference ranges for microbial populations, rendering the testing results useless at this time.

## Fiber Basics

Dietary fiber manipulation can play a significant role in managing GI symptoms. Fiber is nondigestible plant matter that is not absorbed by the human body. Due to its inability to be fully broken down, it serves as a fuel source for the microbiota throughout the GI tract. Understanding the different properties of fiber can help the RDN tailor recommendations to the client without overly restricting their diet. Fiber is defined by three main properties: fermentability, viscosity, and solubility.

Fermentability refers to the extent to which fiber can be broken down by microbes within the gut. The process of fermentation yields gas and sometimes has an osmotic effect, drawing water into the large intestine. While this is a normal process that happens in the absence of disease, people with certain digestive disorders may experience exacerbated symptoms due to fermentation.

Viscosity refers to the ability of fiber to thicken when it encounters water and form a gel-like consistency.

Solubility refers to the ability of fibers to dissolve in water. Insoluble fibers do not hold water and their bulk stimulates peristalsis, contributing to decreased transit time. Soluble fiber attracts water, thereby keeping stool soft and easy to pass.

Dietary fiber recommendations for adults range from 28-35 grams per day but may need to be modified to increase tolerability in

certain GI conditions[25].

**Table 3.4. Types of Fiber Based on Its Solubility, Fermentability, and Viscosity**

| | Insoluble Fiber | Soluble Fiber | | |
|---|---|---|---|---|
| **Fermentability** | Poorly fermented | Fermentable | | Poorly fermented |
| **Viscosity** | Non-viscous | | Viscous | |
| **Examples** | Cellulose<br>Hemicellulose<br>Lignin[26]<br>Wheat bran, whole grain husks, nuts, flax, most fruit/ vegetable skins, nuts/seeds | Acacia gum<br>Inulin<br>Partially hydrolyzed guar gum (PHGG)<br>Oligofructose (OF)<br>Fructo-oligosaccharides (FOS)<br>Galacto-oligosaccharides (GOS)<br>Resistant starch[26,27]<br>Chicory root, onion, garlic, artichokes, asparagus, beans, beets potatoes, pasta[26,27] | ß-glucan<br>Pectin<br>Mucilages<br>Raw guar gum[26,27]<br>Bananas, oats, citrus, seaweed[26], flesh of peeled fruit | Psyllium |
| **Functions and Clinical Applications** | Increases stool mass.[26]<br>Mechanically irritates lining of colon resulting in water and mucus secretions.[28]<br>May have laxative effect by decreasing bowel transit time.[26]<br>Caution for those with intestinal strictures or obstructions. | Hold water and resist dehydration through colon.[28]<br>Increases stool bulk through increased mass and fermentation byproducts (SCFAs, gas).<br>Digested by gut microbiota to produce short-chain fatty acids (SCFAs), which may lower proinflammatory cytokines.[27]<br>Increases fat and cholesterol excretion in feces.[27]<br>May reduce urgency and frequency of loose stools.<br>High intakes may result in abdominal pain, bloating, flatulence.<br>For improved tolerance, gradually increase intake by 1-3 g/day every | | Forms gel with stool and remains viscous in large intestine due to resistance to fermentation.<br>May both solidify loose stools and soften hard constipated stools. |

| | | | |
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| | | 1-3 days. | |
| | | | Slows transit time to maximize digestion and absorption which may reduce hunger and food intake.[28] |

## Modifying "Roughage"

While not discussed in the above table, particle size can also influence fiber tolerance. Reducing particle size of insoluble fiber by blenderizing, cooking, peeling, and/or deseeding may make those foods easier to pass in cases of gastroparesis, strictures, pelvic floor dysfunction, or other conditions. These dietary modifications may be referred to collectively as "small-particle size," "low-roughage," or "GI gentle" approaches.

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CHAPTER 4

# Alterations in Gastrointestinal Anatomy and Physiology

## Possible Gastrointestinal Consequences of Eating Disorders

Patients with eating disorders may present with a variety of gastrointestinal symptoms (i.e., patient-reported) and signs (i.e., abnormal findings on physical exam or diagnostic testing) which may or may not have been previously linked to underlying eating disorders[1].

**Table 4.1 Possible Gastrointestinal Consequences of Eating Disorders**

| Parts of the GI Tract Involved | Pathophysiology of Physical Changes in Eating Disorders | Common Physical Signs and Symptoms Due to Eating Disorders |
|---|---|---|
| Dental | • Erosion of enamel (the protective outer coating of teeth) from stomach acid causing thinning, chipping, and sensitivity<br>• Nutritional deficiencies may impact health of | • Dental caries (i.e., cavities), decay, and/or erosions<br>• Gingival (i.e., gum) disease |

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| | the jaw bones, teeth, gums<br>• Altered teeth brushing hygiene<br>• Bingeing sugary foods, acidic foods, carbonated beverages | |
| Oral | • Autonomic nervous system response to frequent vomiting with accumulation of granules in acinar cells impairing saliva secretion<br>• Repeated trauma from insertion of fingers/instrument to induce vomiting, and acid exposure from vomiting<br>• Vascular injury | • Parotid/salivary gland hypertrophy (sialadenosis)<br>• Damage to oral mucosa (e.g., petechiae, edema, erosions, bleeding)<br>• Necrotizing sialometaplasia (a benign, inflammatory condition of the salivary glands mimicking a malignancy) |
| Esophagus | • Increased acid exposure in the esophagus, increased frequency of lower esophageal sphincter relaxation<br>• Increased acid exposure in the esophagus<br>• Trauma from forceful vomiting<br>• Alterations in motility<br>• Alterations in brain-gut interactions from repeated vomiting, acid exposure, and/or weight loss | • Gastroesophageal reflux disease<br>• Barrett's esophagus (a precancerous change in cell type at the end of the esophagus) and esophageal cancer<br>• Mallory-Weiss tears (superficial tears from forceful vomiting that can cause bleeding)<br>• Boerhaave syndrome (full thickness esophageal tear/rupture)<br>• Food impaction<br>• Abnormal esophageal motility and/or sensation (e.g., dysphagia, difficulty swallowing) |
| Stomach | • Alterations in gastric | • Gastroparesis |

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| | emptying and/or gastric accommodation<br>• Alterations in cholecystokinin and ghrelin (hormones that regulate appetite)<br>• Mass effect of a large food bolus on the gastric mucosa and/or obstruction of the gastric outlet (i.e., pylorus) | • Functional dyspepsia<br>• Acute gastric dilation<br>• Gastric bezoars<br>• Gastric outlet obstruction<br>• Gastric perforation |
| Small and Large Intestines | • Alterations in motility and/or sensation related to caloric restriction, electrolyte abnormalities from purging or laxative abuse, and medications<br>• Alterations in the gut microbiome<br>• Loss of the mesenteric fat pad leading to decreased angle between the superior mesenteric artery and the third portion of the duodenum leading to superior mesenteric artery (SMA) syndrome | • Constipation<br>• Diarrhea<br>• Fecal incontinence<br>• Irritable bowel syndrome and other disorders of gut-brain interaction (DGBIs)<br>• Superior mesenteric artery (SMA) syndrome<br>• Necrotizing colitis<br>• Pelvic floor dysfunction<br>• Rectal prolapse<br>• Melanosis coli<br>• Small intestinal bacterial overgrowth (SIBO) |
| Liver | • Autophagy<br>• Decreased blood flow<br>• Oxidative stress leading to lipid peroxidation and triglyceride deposition resulting in inflammation, cell death, and fibrosis | • Abnormal liver tests<br>• Liver failure<br>• Hypoxia-induced hepatitis<br>• Metabolic dysfunction associated steatotic liver disease (MASLD)<br>• Elevated transaminases (liver tests) |
| Pancreas | • Microlithiasis<br>• Ischemia<br>• Structural damage | • Acute pancreatitis<br>• Pancreatic atrophy |

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| | | • Exocrine pancreatic insufficiency |

## Applying a Weight-Inclusive Lens to Discussions of Gut Health

Medical care has historically been weight-centric, meaning weight management (and minimization) is viewed as a primary means of health promotion. Adiposity is popularly considered a major contributor to the development of disease, and patients who are unable to meet the weight management expectations of providers are seen as self-negligent and noncompliant.

These anti-fat attitudes mask the reality that current research is not clear about higher weight causing poor health. Often, an association can be observed between higher weight and particular disease states, but these correlations fail to account for fat deposition related to disease progression (e.g., polycystic ovarian syndrome, lipedema), the role of internalized weight bias, overt weight stigma and weight-based discrimination (especially in medical settings), and weight cycling. Furthermore, studies documenting health improvements "as a result of weight loss" are likely capturing the effect of health-promoting behaviors used for the purpose of weight loss (e.g., increased movement, higher fiber intake, smoking cessation, stress management)[2].

Regardless of the role of adiposity in disease, the current state of medical research does not support the existence of effective interventions to achieve permanent weight loss. No studies exist demonstrating a weight loss intervention that yields significant weight reduction for the majority of participants beyond 2-5 years. In fact, an overwhelming majority of people who go on a diet will regain the weight within 3-5 years, and 33-66% of those people will gain more than they initially lost[3]. Weight cycling—losing and

gaining weight over multiple attempts, as is typically the result of yo-yo dieting—is the most likely outcome of dieting and carries its own risks. Weight cycling increases inflammation (which, in turn, increases the risk of many diseases) as well as the risk of emotional distress, body dissatisfaction, and disordered eating behaviors[4]. It is not safe, effective, or non-maleficent to recommend weight loss to patients.

This handbook positions itself in contrast to anti-fat attitudes by taking a weight-inclusive approach. The nutritional, behavioral, and medical interventions that appear in this text are recommended without concern for body size. We also encourage providers to examine their own reactions to patients' weight and weight changes. In populations with eating disorders and gastrointestinal disorders, weight loss can be cause for tremendous concern and weight gain can be a sign of healing; providers need to be prepared to view weight data with the greatest degree of neutrality in order to best serve the patient in front of them.

# Gastrointestinal Disorders and Diagnoses

## Abdominal Bloating and Distention

***Overview:*** Bloating is the subjective sensation of fullness, swelling, tightness, or trapped gas in the abdomen. Distention is a visible increase in abdominal girth that may be conveyed as feeling "like being pregnant." Bloating and distention may be the primary symptom that DGBIs patients suffer from or may be part of other conditions such as IBS. The causes of bloating and distention include food intolerance (such as lactose, fructose, fructan intolerance) and hypersensitivity (such as histamine intolerance), small intestinal bacterial overgrowth (SIBO), celiac disease, non-celiac gluten sensitivity (NCGS), motility disorders, anorectal and pelvic floor disorders, and abdomino-phrenic dyssynergia (APD).

APD is a cause of distention characterized by the pathologic contraction and descent of the diaphragm and relaxation and protrusion of the abdominal wall[5]. Many individuals with bloating (the sensation of increased abdominal pressure/tension) and distention (the objective, visible increase in abdominal girth) do not have increased intestinal gas volumes. APD is thought to play a role in bloating and distention seen in DGBIs such as IBS, functional dyspepsia, aerophagia (swallowing of excessive air leading to bloating, distention, and belching), and functional bloating and distention. Despite their commonality, bloating and distention have many potential causes, are often not associated with increased overall gas in the gastrointestinal tract, and identifying the primary cause can prove challenging.

***Prevalence:*** Functional bloating and distention occur in up to 3.5% of the world's population, with significantly greater prevalence of > 50% when other DGBIs are present (e.g., IBS, functional dyspepsia). No epidemiology specific to prevalence of bloating and distention in patients with eating disorders exists. No studies have investigated the prevalence of APD in patients with EDs, though bloating and distention are common across all EDs. Despite being common symptoms, bloating and distention can be due to many causes and may prove very frustrating for patients when clear causes are not able to be identified and/or remedied.

***Diagnosis:*** Bloating and distention can be diagnosed using the Rome IV criteria: 1) recurrent bloating and/or distention occurring on average at least 1 day/week, and 2) abdominal bloating and/or distention predominates over other symptoms, there are insufficient criteria to diagnose IBS, functional constipation, functional diarrhea, or functional dyspepsia postprandial distress syndrome, and the criteria must be fulfilled for the last 3 months with onset of symptoms at least 6 months prior to diagnosis. Despite the existence of breath testing for lactose and sucrose

intolerances, food intolerances are often best tested using a 2-week elimination diet, which poses risks in eating disorder populations. When further testing is done, breath testing (e.g., CSID breath testing) or small bowel biopsies for enzyme activity may be performed. SIBO/IMO (see below) are often suspected and treated empirically but may be tested for with hydrogen-methane breath testing using glucose or lactulose as substrates and less often with jejunal aspirates. Celiac disease testing (see below) is recommended as bloating and distention are common in patients with celiac disease, non-celiac gluten sensitivity, and gluten intolerance. Motility disorders, such as gastroparesis and slow transit constipation, as well as pelvic floor dysfunction (e.g., dyssynergic defecation) may contribute to bloating and distention and may warrant gastric emptying studies to rule out gastroparesis and anorectal testing with digital rectal exam, anorectal manometry with balloon expulsion, and/or defecography. Radiographic testing with ultrasound, X-ray, or CT scan may be considered in certain patients. There are no standard tests to diagnose APD. Studies have used CT scans to find morphological and volumetric differences during severe distention versus minimal or no distention are related to electromyography (EMG) activity of the abdominal wall. Other studies have used abdominal inductance plethysmography to measure changes in abdominal girth, ultrasound to evaluate movement of the diaphragm, and manometry to monitor esophageal and gastric pressures.

***Treatment:*** Treatment of bloating requires careful consideration of the most likely contributors to patient symptoms. Treatment of constipation with medications and/or pelvic floor physical therapy, as well as restriction of carbohydrates, FODMAPs, and/or gluten and its components (e.g., fructans) may relieve bloating and distention. However, any restrictive or elimination diet should be avoided in patients with histories of eating disorders unless used under the supervision of a dietitian with experience with eating

disorders. Antibiotics and prokinetics may help to treat SIBO and decreased motility. Central neuromodulators and brain-gut therapies such as gut-directed hypnotherapy and cognitive behavioral therapy are very efficacious and effective in managing conditions such as IBS and functional dyspepsia. No standardized treatments currently exist for bloating and distention. However, EMG biofeedback, diaphragmatic breathing, specific treatments for other DGBIs such as the low FODMAP diet, behavioral therapies, and neuromodulators may have a role in treatment. In addition, treatment of associated comorbidities such as IBS, functional dyspepsia, pelvic floor dysfunction, endometriosis, anxiety, depression, and PTSD may be warranted.

## Bile Acid Diarrhea (BAD)

***Overview:*** The liver produces bile acids and approximately 95% are reabsorbed at the terminal ileum as part of routine digestion. Bile acid malabsorption (BAM) occurs when this process is disrupted, resulting in excess bile acids reaching the colon. This can cause chronic diarrhea, known as bile acid diarrhea (BAD), by means of altering water and sodium transport, inducing mucus secretion, changing motility and/or stimulating bowel movements[6]. There are three types of BAD, based on etiology: Type 1—presence of ileal disease (e.g., Crohn's disease), resection, or bypass of the terminal ileum; Type 2—no ileal disease, sometimes referred to as primary bile acid diarrhea; and Type 3—other forms not covered by Types 1 and 2, including small intestinal bacterial overgrowth (SIBO), post-cholecystectomy, post-vagotomy, celiac disease, radiation enteritis, and chronic pancreatitis.

***Prevalence:*** Accurate prevalence rates are difficult to come by, as many surveyed gastroenterologists report rarely or never investigating BAD in patients presenting with chronic diarrhea. Many people with BAD may be misdiagnosed with IBS-D due to

the overlap in symptom profiles and the misconception that BAD is rare[7]. A systematic review in 2015 of 6 studies with 908 patients with IBS-D showed rates of BAD ranging from 16.9% to 35.3%, with a pooled rate of 28.1%[8]. A 2021 study found a BAD prevalence rate of 28.1% among patients at an outpatient gastroenterology clinic who presented with chronic diarrhea[9]. Another 2021 study found a prevalence rate of 52.6% of patients with chronic diarrhea, after IBD, celiac disease, pancreatic insufficiency, parasites and C. difficile had been ruled out[10]. Thus, BAD should be considered in all patients with chronic diarrhea, at least as a part of the pathophysiology of diarrheal symptoms. Prevalence of BAD in patients with known EDs is unknown in the literature to date.

***Diagnosis:*** There are three commonly described tests for BAD: $^{75}$Selenium-homotaurocholic-acid-test ($^{75}$SeHCAT) using a < 10% retention threshold, fasting serum 7-α-hydroxy-4-cholesten-3-one (C4), and total fecal bile acid excretion over 48 hours. However, none of these tests is routinely available in the U.S., so most providers suspecting BAD use a "therapeutic trial" of a bile acid sequestrant.

***Treatment:*** Treatment of BAD includes the use of bile acid sequestrants, as well as treatment of underlying disorders contributing to BAD when possible. Cholestyramine, colestipol, and colesevelam are the three bile acid sequestrants available in the United States. Common side effects of these medications include nausea, gas, bloating, abdominal pain, and constipation. Cholestyramine and colestipol may interfere with absorption of certain medications and, as such, medications should be taken 1 hour before or at least 4 hours after bile acid sequestrants.

## Celiac Disease (CeD)

***Overview:*** Celiac disease (CeD), also known as gluten-sensitive enteropathy, is an autoimmune inflammatory condition of the small intestine caused by sensitivity to gluten and related proteins in susceptible individuals. Common symptoms in individuals with CeD include abdominal pain, bloating/distention, chronic diarrhea (although constipation does not exclude the diagnosis), vomiting, and failure-to-thrive or weight loss. In addition, there are several extraintestinal manifestations: neurologic and behavioral symptoms, arthritis/arthralgias, abnormal liver tests, chronic fatigue, short stature, pubertal delay, iron deficiency anemia not responding to oral iron supplements, dermatitis herpetiformis-like rash, dental enamel hypoplasia of permanent teeth (in a symmetric distribution), recurrent aphthous ulcers, and osteopenia/osteoporosis. Many individuals with CeD have other chronic, autoimmune conditions such as type 1 diabetes mellitus, autoimmune thyroid disease, juvenile idiopathic arthritis, selective IgA deficiency, and autoimmune liver disease. In addition, certain genetic conditions carry a much higher risk for CeD: Down syndrome, Turner syndrome, and Williams syndrome. Sensitivity to gluten, as well as other components of wheat (e.g., fructans) without evidence of CeD is common and referred to as non-celiac gluten sensitivity (NCGS). These individuals report many of the same symptoms as those with CeD with ingestion of wheat/gluten despite negative CeD testing. True wheat allergy, which is IgE-mediated, is associated with stereotypical signs of allergy: hives, edema, shortness of breath, abdominal pain, and anaphylaxis. For a multitude of reasons, from perceived health benefits and health fads to self-management of symptoms, up to 5% of adults in the U.S. exclude wheat from their diets on their own.

***Prevalence:*** The worldwide prevalence of CeD is 1.4% based on blood tests and 0.7% based on biopsy results. The prevalence is higher in females than males and, when screened, is significantly greater in children than adults. The prevalence of those with eating

disorders having celiac disease is 8.88%. Compared to adults without an ED, the risk for AN patients of developing CeD is 2.35 times higher[11,12]**.** Among ARFID patients, an estimated 14% have CeD[13]. Of note, 0.5-13% of the population have non-celiac gluten sensitivity, and true wheat allergy has a prevalence of 0.2 to 1% of children, but most outgrow this by the age of 12[14,15]. Those with both CeD and an active ED are more likely to intentionally eat gluten as a means of "purging" calories or modifying body shape/weight.

***Diagnosis:*** The gold standard to confirm the diagnosis of CeD are biopsies of the duodenum showing increased intraepithelial lymphocytes, blunted/atrophic villi, and crypt hyperplasia (elongation of the grooves between villi). Endoscopic appearance suggestive of CeD includes flattening of normal folds and villi, fissures over folds, and mosaic appearing patterns of the villi. Blood tests (serologies) for antibodies against components of wheat that are suggestive of CeD including tissue transglutaminase (tTG), deamidated gliadin peptide (DGP), and endomysial antibody (EMA). The recommended screening serologies for CeD are anti-tTG IgA antibodies, as well as a total IgA level to ensure that a patient is not IgA deficient. Selective IgA deficiency is the most common primary immunodeficiency and occurs in roughly 1 in 500 individuals, with variations across ethnicities. However, in patients with CeD, the prevalence of IgA deficiency is 2-5%. If a patient is IgA deficient, the tTG IgA levels may be falsely normal, and thus a tTG IgG is required to assess for CeD. Biopsies and serologies are used to monitor disease activity, and both normalize in patients on a gluten-free diet, making the diagnosis of celiac disease challenging in patients who have already excluded gluten from their diets. The typical recommendation for patients who have already excluded gluten from their diets is to do a gluten challenge consisting of 3 grams per day of gluten (the equivalent of 1.5 slices of white bread per day) for 8 weeks, followed by endoscopy with

biopsies of the duodenum[16]. For patients unable to tolerate or unwilling to undergo a gluten challenge and endoscopy, blood tests for genetic markers of predisposition to CeD (i.e., HLA DQ2/DQ8) can be checked. The markers of predisposition are present in roughly 40% of the population, and they may or may not express over an individual's lifetime. For this reason, genetic markers are more helpful when negative to rule out CeD. In patients with positive HLA markers and noted adverse reaction to consuming wheat/gluten, care should mirror the treatment of patients with known CeD.

***Treatment:*** The treatment of CeD is a gluten-free diet (GFD). Roughly 20-30% of the U.S. population excludes gluten to some extent, and the availability of gluten-free foods in grocery stores and restaurants is widespread[17]. Thus, many patients and providers may be inclined to begin a GFD on their own. However, patients are often unaware of sources of hidden gluten which are a common culprit for ongoing symptoms and/or continued disease activity. Serologies and endoscopy are used to monitor disease activity, and both should normalize with a GFD. Typically serologies are checked at 6 and 12 months post-diagnosis, and then annually for patients who achieve remission. Nonresponsive celiac disease (NRCD)—when either symptoms and/or biological markers of CeD do not resolve as expected—occurs in 30% of people with CeD and is most often due to dietary indiscretion (either intentional or unintentional)[17]. Gluten exposure should be ruled out by a detailed interview from a specialized dietitian and/or stool or urine detection of gluten-immunogenic peptides. Celiac disease serologies (i.e., autoantibodies) are not always sensitive to small levels of gluten intake, however. If villous atrophy is suspected, intestinal biopsies should be examined to evaluate for abnormal Marsh score characteristic of active CeD whether or not serology is elevated. Approximately 20% of people with celiac disease on a gluten-free diet will have intestinal injury despite normal tissue

transglutaminase antibodies[18]. The next most common causes of NRCD are co-occurring diseases, such as irritable bowel syndrome, small intestinal bacterial overgrowth/intestinal methanogen overgrowth (SIBO/IMO), microscopic colitis, or exocrine pancreatic insufficiency. If a patient is not found to have high likelihood of gluten ingestion, it is recommended to evaluate for co-occurring disorders; these disorders should be evaluated from their own diagnostic criteria. Patients with CeD may require other health maintenance including pneumococcal vaccination (given the association of CeD with decreased function of the spleen), screening for osteoporosis, correction of micronutrient deficiencies, and treatment of associated skin conditions such as dermatitis herpetiformis. Refractory CeD (RCD) occurs in 0.3–4% of patients with CeD and accounts for 8–23% of NRCD and requires immunosuppressive medications to treat[17]. Two studies have demonstrated a positive impact of a gluten contamination elimination diet (GCED) on resolution of patients previously thought to have RCD[19]. This highly restrictive diet should be approached with extreme caution, as it eliminates all processed/packaged foods and social dining. In our opinion, this diet is a risk factor for exacerbation or onset of ED, and we recommend thorough medical evaluation by a gastroenterologist specialized in RCD prior to initiating.

## Constipation

***Overview:*** There are many terms that describe constipation. In 2013, the AGA referred to it as "chronic constipation" and described subtypes: normal-transit constipation (understood to mean *functional constipation*), slow-transit constipation, and pelvic floor and/or outlet dysfunction (i.e., defecatory disorders)[20]. More recently, in 2023, joint guidelines by the AGA and ACG refer to "chronic idiopathic constipation," which appears to lump together various subtypes[21]. Regardless of the terminology used,

constipation is a bowel disorder marked by infrequent and/or difficult bowel movements. It typically presents without abdominal pain or visceral hypersensitivity, making it distinct from constipation-predominant irritable bowel syndrome (IBS-C). Similar interventions are used in the treatment of normal-transit/functional constipation and slow-transit constipation.

Pelvic floor dysfunction refers to lack of appropriate muscle tone and/or muscular coordination in the pelvic bowl. Several causes exist, including chronic constipation, injury, and trauma, and it has a specific course of treatment (detailed below)[22]. Pelvic floor dysfunction may manifest as constipation and/or fecal incontinence, small/hard/pencil-thin stools, sense of incomplete evacuation, sense of fecal urgency, and need to manually disimpact stool. Fecal incontinence may occur due to pelvic muscle weakness or as part of an "overflow" phenomenon, whereby liquid stool passes around a hard, slow-moving mass of stool. Posterior pelvic floor disorders include dyssynergic defecation (failure of the puborectalis and external anal sphincter muscles to relax or paradoxical contraction of these muscles with attempted defecation), pelvic organ prolapse (e.g., bowel, bladder, urethral, rectum, or uterine), and neurogenic bowel disorders (e.g., in patients with multiple sclerosis, Hirschsprung's disease, and spinal cord injury).

***Prevalence:*** Many studies refer to "functional constipation" without indicating in their methodology whether they distinguished between normal-transit and slow-transit or just combined the two. That said, reportedly, "functional constipation" affects an estimated 2-27% of North Americans and 10-17% of people worldwide[23,24]. In a 2013 study surveying inpatient eating disorder unit admissions, 27% of patients were found to meet criteria for functional constipation (although this study used the Rome II criteria, which was phased out in 2006)[25]. A similar study in 2014

showed that up to 27% of eating disorder patients met Rome III criteria for functional constipation[26]. Up to 98% of inpatients receiving treatment for EDs have symptoms of a DGBI, with constipation-predominant DGBIs being the most common[27].

Dyssynergic defecation affects over one-third of patients with chronic constipation and has a prevalence of 7% in the general population[28]. One study of patients with AN found that 84% of patients had dyssynergic defecation, and that 73% of patients with AN had fecal incontinence[29]. One study of 1,899 patients in a urogynecology clinic found that 17% had a history of sexual trauma[22]. Additional studies have demonstrated no difference in anorectal physiology testing between patients with and without a history of sexual trauma who present for defecatory problems, but the literature is inconsistent with regards to whether a history of sexual trauma leads to greater symptom severity[30,31].

***Diagnosis:*** Functional constipation is currently defined by Rome IV criteria (updated Rome V criteria are expected to be available in 2026). The patient history should demonstrate 2 or more of the following (ongoing for ≥3 months, with symptom onset ≥6 months prior to diagnosis): fewer than 3 spontaneous bowel movements per week, straining, hard or lumpy stools, sensation of incomplete evacuation, sensation of anorectal blockage/obstruction, use of manual maneuvers to facilitate stool passage, and/or loose stools are rarely present without the use of laxatives (Rome IV Criteria).

Slow transit constipation is diagnosed using radiopaque (Sitzmarkers) or wireless motility capsule studies. Sitzmarkers studies involve ingestion of a capsule with 24 radiopaque markers on day 0 followed by an X-ray on day 5 with assessment of the number of markers remaining. With normal transit, less than or equal to 5 markers should remain in the colon. Distribution of markers can also suggest slow transit versus outlet dysfunction

constipation, with markers collecting in the left side of the colon and rectum in outlet dysfunction instead of being evenly distributed. Wireless motility capsules are swallowed and use temperature, pressure, and pH to determine gastric, small intestinal, and colonic motility, with normal colonic motility indicated if the capsule passes through the colon in 10-59 hours.

Dyssynergic constipation and other posterior pelvic floor disorder causes of constipation should be investigated whenever chronic constipation does not resolve with standard treatment. Dyssynergic defecation is diagnosed using a digital rectal exam, anorectal manometry with balloon expulsion, and/or defecography. With dyssynergic defecation, a rectal exam of simulated defecation allows palpation of failed relaxation or paradoxical contraction of the puborectalis and external anal sphincter muscles; manometry demonstrates the same phenomenon using a pressure-sensing catheter in the anorectal canal; balloon expulsion testing reveals inability to expel a balloon filled with 50 milliliters of water from the rectum within 60 seconds; and defecography (using either MRI or barium and X-ray) demonstrate failure of the perineum to descend and the anorectal angle to straighten adequately. Unfortunately, few gastroenterologists perform rectal exams, especially for the assessment of posterior pelvic floor muscles, and manometry testing is not widely available in community practices. The balloon expulsion test can be performed in an exam room with access to a toilet if manometry is unavailable. Fecal incontinence can be assessed beyond patient reports by using a digital rectal exam or anorectal manometry.

***Treatment:*** Treatment of constipation includes over-the-counter oral options, such as fiber supplementation, senna, bisacodyl, polyethylene glycol, and magnesium, as well as suppositories and enemas. In addition to fiber supplements, consumption of prunes or kiwifruits have shown to be effective for the treatment of

constipation. Prescription laxatives called secretagogues work by stimulating peristalsis and causing secretion of water and electrolytes into the lumen of the colon by direct stimulation of chloride channels in the colon or stimulation of serotonin receptors. Some secretagogues are approved for use in both chronic idiopathic constipation and constipation-predominant IBS (IBS-C). Increasing fluid intake may help those who don't consume enough, but, in general, this intervention alone has limited benefit due to the ability of the colon to increase water absorption from 1.5 to 2 liters in normal conditions up to 6 liters per day in the setting of increased intake or alterations in small bowel secretion/absorption. In rare instances of severe slow transit constipation, surgery may be necessary and typically involves colectomy with either ileoanal anastomosis (re-connection of small intestine to the anus) or end-ileostomy (small intestine brought up to the abdominal wall as an ostomy).

Treatment for most pelvic floor disorders, such as dyssynergic defecation and incontinence, as well as many cases of pelvic organ prolapse, begins with pelvic floor physical therapy (PT). Pelvic floor PT may include biofeedback and various other modalities to relax, strengthen, and/or coordinate muscle movements. Surgery for pelvic floor organ prolapse may be required in severe cases that are not improved with non-invasive treatments, such as pelvic floor PT and/or pessaries. Of note, while slow transit related to EDs often improves with ED recovery, pelvic floor dysfunction may not, which some prior studies suggest may be due to damage to the puborectalis muscles[32].

## Dyspepsia and Functional Dyspepsia

***Overview:*** Dyspepsia, sometimes referred to as "indigestion," is a clinical diagnosis that includes non-GERD upper GI symptoms (i.e., those other than heartburn and reflux) such as epigastric pain, burning, belching, bloating, distention, nausea, and early satiety.

Dyspepsia can be caused by *Helicobacter pylori* (*H. pylori*) infection, peptic ulcer disease, medications (e.g., non-steroidal anti-inflammatory drugs such as ibuprofen, alendronate, iron, potassium, selective serotonin reuptake inhibitors, and certain antibiotics such as erythromycin), gastrointestinal malignancy, and functional dyspepsia. Functional dyspepsia is a DGBI diagnosed when *H. pylori* testing, medication trial or proton pump inhibitors, and/or endoscopy are negative. Approximately 10% of individuals with dyspepsia have endoscopic findings that explain their symptoms, leaving the remaining 90% with functional dyspepsia[33]. Functional dyspepsia is considered a disorder of gut-brain interaction in which sensation and motility are altered, including impaired gastric accommodation and fundic relaxation. Functional dyspepsia can be categorized as epigastric pain syndrome (EPS), characterized by epigastric pain after eating, or postprandial distress syndrome (PDS), marked by early satiety, bloating, and belching.

***Prevalence:*** Dyspepsia affects roughly 20% of adults throughout the world. Dyspepsia and nausea are common in patients with EDs, with post-meal discomfort reported in up to 90% or more of patients with EDs and nausea in 21%. In patients presenting with symptoms of gastroparesis/functional dyspepsia, roughly 40% met criteria for ARFID, with symptoms thought to be due to ED-related alterations in gastric emptying and/or accommodation[34]. In patients with EDs, functional dyspepsia PDS is much more common than EPS. In a systematic review, criteria for PDS were fulfilled in 90% of patients with AN and EDNOS (eating disorder not-otherwise specified, an earlier iteration of "Other Specified Feeding/Eating Disorder") attending an outpatient department for eating disorders and by 83.3% of patients with BN, while only one patient with BN had EPS[26,35,36].

***Diagnosis:*** The initial investigations seek to rule in/out *H. pylori* infection using stool antigen testing, urea breath test, or endoscopy with biopsies. When *H. pylori* is not present or has been eradicated and symptoms persist, upper endoscopy is often done to look for structural/inflammatory causes of dyspepsia. If *H. pylori* is ruled out/eradicated and an upper endoscopy is normal, then the diagnosis is functional dyspepsia. Endoscopy may be performed upfront in cases with red flag symptoms such as weight loss, new onset of dyspepsia after age 60, anemia, melena (black, tarry stools indicative of upper GI bleeding), or severe pain. Furthermore, a recent study suggested improved cost-effectiveness performing endoscopy upfront compared to after failure of medication trial and/or *H. pylori* eradication[37].

***Treatment:*** If *H. pylori* is present, then treatment consists of combinations of antibiotics and PPIs with or without bismuth subsalicylate for 2 weeks, followed by testing for eradication using any of the above modalities and must be done with patients off PPIs for 2 weeks and off antibiotics and bismuth-containing medications for 4 weeks. When *H. pylori* is not present or if symptoms persist after eradication, a trial of PPIs is standard of care. Patients with early satiety in the setting of PDS may benefit from a fundic relaxing agent, such as the anxiolytic, buspirone, which has been shown to improve symptoms and fundic relaxation. Patients with predominant pain symptoms of EPS may benefit more from neuromodulators such as tricyclic antidepressants (e.g., amitriptyline), selective serotonin reuptake inhibitors (e.g., duloxetine), or alpha-2 adrenergic receptor antagonists (e.g., mirtazapine). In addition, behavioral therapies such as cognitive behavioral therapy and hypnosis may be used to treat functional dyspepsia. Research is limited on dietary interventions for functional dyspepsia, though patients with PDS may derive more benefit from dietary treatment than those with EPS.

## Gastroesophageal Reflux Disease (GERD)

***Overview:*** Classic symptoms of GERD are heartburn and reflux of stomach contents into the esophagus. Acidic contents of the stomach can cause symptoms, as well as objective findings of erosive esophagitis (ulcerated lower esophagus) due to the caustic nature of acid, esophageal strictures due to chronic inflammation, and Barrett's esophagus, a precursor lesion to esophageal cancer. The most common cause of GERD is an increase in transient lower esophageal sphincter relaxations (TLESRs). TLESRs are a normal physiologic phenomenon to help vent gas from the stomach but may be increased in some individuals. Certain foods (such as those high in fat, acidic and citrus foods, peppermint, chocolate, and carbonated beverages) may worsen reflux, possibly due to relaxation of the LES, increase in gastric acidity, and/or increase in intragastric gas. In addition to TLESRs and food, hiatal hernias are a common contributor to GERD. Hiatal hernias occur when the upper portion of the stomach bulges through the opening in the diaphragm through which the esophagus connects to the stomach. Hiatal hernias disrupt the normal anti-reflux barrier made by the LES. The normal response of the esophagus to reflux is to contract to push acidic contents back into the stomach. Thus, abnormal motility of the esophagus can contribute to GERD and vice versa. The current weight-centric model of care in gastroenterological institutions is to attribute GERD to weight gain and intra-abdominal pressure from visceral fat. However, it should be noted that people across the weight and size spectrum can develop GERD.

***Prevalence:*** GERD affects approximately 18.1-27.8% of adults in Western countries, though several studies have demonstrated increases in GERD[38]. Furthermore, many epidemiologic studies of GERD have noted the likelihood that the literature underestimates GERD due to the widespread availability of over-the-counter GERD medications. One small study of adolescent females with AN found

that only 8.7% of individuals with GERD symptoms had abnormal acid reflux tests[39].

***Diagnosis:*** GERD is a clinical diagnosis that may be supported by therapeutic trials of medications. Upper endoscopy can suggest GERD when erosive esophagitis or Barrett's esophagus is present. In addition, upper endoscopy allows for potential identification of a hiatal hernia, which is a structural risk factor for GERD. Formal diagnosis of GERD requires pH testing, either using a pH-impedance catheter inserted through the nose and into the esophagus or Bravo wireless pH monitoring, which requires an upper endoscopy to affix the device to the lower esophagus. Both pH tests measure the acid exposure (i.e., pH < 4) over a period of time, as well as correlation of symptoms with reflux events.

***Treatment:*** In some cases, GERD may be managed by lifestyle modifications such as avoidance of relevant trigger foods, avoidance of eating close to bedtime, and raising the head of the bed during sleep to allow for a 30- to 45-degree angle. However, many patients require medications to manage their GERD, which range from antacids (e.g., calcium carbonate or TUMS) to histamine-2 receptor antagonists (e.g., famotidine, cimetidine) to proton pump inhibitors (e.g., pantoprazole, omeprazole, esomeprazole, rabeprazole, dexlansoprazole, lansoprazole). In the setting of a hiatal hernia, severe reflux refractory to medications, and/or inability to tolerate medications, anti-reflux procedures, including Nissen fundoplication (surgical), transoral incisionless fundoplication/TIF (endoscopic), or magnetic sphincter augmentation (surgical), can be considered.

## Gastroparesis (GP)

***Overview:*** Gastroparesis (GP) is a motility disorder that delays gastric emptying of solid food without mechanical obstruction[40]. The symptoms associated with GP may become worse upon eating

a meal and can include early satiety, postprandial distress, nausea, vomiting, and upper abdominal pain. Many of the symptoms of GP overlap with those of functional dyspepsia, with the main difference being the degree of nausea and vomiting and objective evidence of delayed gastric emptying.

***Prevalence***: It has been predicted that GP impacts 1.3-1.4% of the general population, though epidemiologic data on GP have been limited and difficult to interpret due to inconsistencies in citing patients with suspected versus confirmed GP[41,42]. A 2022 retrospective database study stated the prevalence of GP was 267.7 per 100,000 US adults based on insurance claims. The same study reported a "definite" GP diagnosis in 21.5 per 100,000 persons[42]. People who struggle with eating disorders may exhibit gastroparesis-like symptoms and as such current guidelines do not recommend routine gastric-emptying tests, as symptoms are likely to get worse as their eating disorder progresses[1]. In our expert opinion, a GES study can be an effective tool to rule-in GP in order to validate the patient's symptoms with tangible evidence and possibly help to resolve patient ambivalence or denial about negative impact of disordered eating behaviors. It also can be an effective tool to rule out GP and guide the clinician to other diagnoses and treatments such as reduced gastric accommodation and functional dyspepsia. Sometimes, when patients are told they do not have GP, there are no further treatment options offered. (See dyspepsia section given significant overlap of symptoms such as early satiety, bloating, and pain in both functional dyspepsia and gastroparesis).

***Diagnosis:*** The gold standard for diagnosing GP is a nuclear medicine scintigraphic gastric emptying study of a standardized solid meal[40]. After consumption of the meal, images are collected at hourly intervals for 4 hours. GP is diagnosed when 10% or more of the solid meal is retained in the stomach at the 4-hour mark[40].

Wireless capsule endoscopy, which evaluates gastric, small intestinal, and colonic motility using temperature, pressure, and pH sensors can also suggest the diagnosis of gastroparesis. Lastly, several breath tests have been studied for the diagnosis of GP, with the spirulina carbon-13 breath test approved by the FDA in 2015.

***Treatment:*** Dietary modification is considered first-line therapy for GP, with small, frequent meals low in fat and insoluble fiber and/or large particle size, typically recommended. Alcohol, tobacco, and carbonated beverages are generally discouraged, as they may further slow gastric emptying and cause excessive gastric distention. Optimization of blood sugar in people with diabetes may improve gastric emptying. Prokinetics stimulate gastric emptying (e.g., metoclopramide, erythromycin, and domperidone); antiemetics treat nausea; tricyclic antidepressants moderate pain. Some cases might call for gastric decompression and post-pyloric feeding with gastrojejunostomy tubes to keep patients nourished. Pyloric directed therapies such as endoscopic and laparoscopic pyloromyotomy (cutting the pyloric muscle to improve outflow of the stomach) are potential options in refractory cases, and gastric electrical stimulation may improve symptoms without having significant impact on gastric emptying. In patients with restrictive eating disorders, resolution of GP may occur spontaneously with nutrition rehabilitation and/or weight restoration[43].

## Inflammatory Bowel Disease (IBD)

***Overview:*** Inflammatory bowel disease (IBD) is comprised primarily of Crohn's disease (CD) and ulcerative colitis (UC). In CD, inflammation can involve any portion of the GI tract from the mouth to the anus, and is often characterized by skip lesions, with area of inflammation separated by normal-appearing mucosa. The inflammation in CD is transmural (full thickness), which leads to many of the complications of CD such as stricturing and fistulizing

disease. UC involves superficial inflammation of the colon, with the rectum always involved and inflammation extending in a continuous fashion proximally. Inflammation in UC may be limited to the rectum (proctitis), may involve the rectum and sigmoid colon (proctosigmoiditis), or may involve the whole colon (pancolitis). Both subtypes of IBD are also associated with extraintestinal manifestations that may include inflammation of the ocular, musculoskeletal, skin, and/or hepatobiliary systems, as well as other less common sites.

***Prevalence:*** Prevalence of IBD in US adults is 1%[44]. Compared to adults without an ED, the risk for AN patients of developing IBD is 1.44 times higher[11,12]. Among ARFID patients, an estimated 10% have IBD, and 2% have UC[13].

***Diagnosis:*** IBD is suspected based upon symptoms such as abdominal pain, diarrhea, blood in the stools, unintentional weight loss, and extraintestinal manifestations. Laboratory testing that may raise suspicion for IBD includes low hemoglobin (i.e., anemia) on a complete blood count (CBC), elevated fecal inflammatory markers such as calprotectin or lactoferrin, iron deficiency, and elevated serum inflammatory markers such as C-reactive protein (CRP) and erythrocyte sedimentation rate (ESR). However, up to 20-25% of patients with active flares of IBD may not mount elevations in serum inflammatory markers. Imaging using CT or MRI may identify thickening of the gastrointestinal wall in involved areas and/or complications such as stricturing with or without bowel obstruction, fistulae, and/or intra-abdominal abscesses. The gold standard for confirming the diagnosis of IBD involves colonoscopy with endoscopic findings of inflammation (e.g., erythema and ulcerations) and histologic evidence of inflammation on biopsies.

***Treatment:*** Treatment of IBD is focused on improvement of symptoms, as well as normalization of laboratory, endoscopic, and

histologic evidence of inflammation. When all of these have normalized, patients are said to be in "deep remission." Steroids such as prednisone may be used for induction of remission but have long-term complications with chronic use. It is unfortunately not uncommon for new or ongoing symptoms to be treated with steroids without confirming the presence of inflammation. For UC, 5-aminosalicylates (5-ASA) such as mesalamine are used in oral and rectal (enemas and suppositories) formulations to induce and maintain remission[14]. For moderate to severe UC or 5-ASA failures, patients are transitioned to intravenous/intramuscular biologic agents (anti-TNFs such as infliximab, adalimumab, golimumab; anti-IL-12/-23 inhibitor in the form of ustekinumab; or anti-integrin antibody in the form of vedolizumab) or oral small molecule inhibitors (JAK inhibitors tofacitinib or upadacitinib; sphingosine-1-phosphate receptor modulator ozanimod)[46]. Patients with acute severe UC may require admissions for intravenous steroids, infliximab, or cyclosporine, and they may require emergency surgery with total procto-colectomy[47]. Elective colectomy may be performed in patients who fail or do not tolerate medications or who have precancerous (dysplasia) lesions or colorectal cancer. Surgery cures UC given that the disease only involves the colon, but pouchitis is common and can resemble a UC flare. Patients with CD are treated with immunomodulators such as azathioprine, 6-mercaptopurine, or methotrexate if they have mild disease. Those with moderate to severe disease are treated with intravenous/intramuscular biologics (anti-TNFs such as infliximab, adalimumab, certolizumab; anti-IL-12/-23 ustekinumab; anti-IL-23 in the form of risankizumab; anti-integrin antibody in the form of vedolizumab) or oral small molecule drugs (JAK inhibitor upapacitinib)[48]. Surgery may be necessary for stricturing disease, fistulae, and abscesses, though the risk of recurrence at the site or re-anastomosis (reconnection) of the bowel is high and typically requires post-operative treatment with medications to reduce said risk. Current guidelines do not recommend a specific diet for the

induction or maintenance of remission in moderate to severe Crohn's disease patients or in any disease severity of ulcerative colitis patients[49]. Exclusive enteral nutrition (EEN) or the Crohn's disease exclusion diet (CDED) with partial enteral nutrition (PEN) can be considered for induction of remission for pediatric patients with mild to moderate Crohn's disease[49]. In adults with mild to moderate Crohn's disease, the CDED with or without PEN can be considered for induction of remission[49]. There is insufficient data to support a diet for the maintenance of remission in mild to moderate Crohn's disease[49]. One of the most common causes of ongoing symptoms in the absence of evidence of objective inflammation on diagnostic testing is IBS. The IBD-IBS overlap occurs in upwards of 39% of patients and is more common in patients with Crohn's disease than ulcerative colitis. The low-FODMAP diet may be considered in quiescent IBD patients experiencing symptoms but it does not appear to impact inflammation[50]. Patients with Crohn's disease and inflammation limited to the small bowel may be more likely to have normal blood, stool, and endoscopic evaluations and may require radiographic imaging and/or capsule endoscopy to identify inflammation not found on routine upper endoscopy and colonoscopy[51].

## Microscopic Colitis

***Overview:*** Microscopic colitis is a chronic inflammatory condition of the colon and is a subtype of IBD (it is listed separately from IBD due to distinctions in long-term risks and treatment approaches). It is clinically characterized by watery, non-bloody diarrhea. Microscopic colitis consists of lymphocytic and collagenous colitis, with the two entities as presenting clinically the same and the differences noted on histology. Microscopic colitis is associated with other autoimmune diseases such as autoimmune thyroiditis, type 1 diabetes mellitus, and celiac disease.

***Prevalence:*** There are currently no studies reporting on the overlap of microscopic colitis in patients with EDs. A systematic review and meta-analysis found a pooled incidence rate of 4.85 (95% CI, 3.45-6.25) for lymphocytic colitis and 4.14 (95% confidence interval (CI) 2.89-5.40) per 100,000 person-years for collagenous colitis[52]. The median age of diagnosis of microscopic colitis is 65 years. Microscopic colitis is most common in women. PPIs, selective serotonin reuptake inhibitors (SSRIs), and NSAIDS are associated with increased risk of microscopic colitis.

***Diagnosis:*** Microscopic colitis should be suspected in patients with chronic diarrhea, especially in middle-aged and older adults. Exclusion of other causes of chronic diarrhea may include stool studies for *Clostridium difficile*, Giardia, and other infectious etiologies, and celiac disease testing using serologies. Use of stool tests such as fecal calprotectin to rule out colonic inflammation is not recommended for diagnosing microscopic colitis, as it would be for assessing and monitoring CD and UC. The gold standard for diagnosing microscopic colitis is a colonoscopy with random biopsies from the right and left colon, given that the histologic findings of microscopic inflammation are often patchy and may be missed in up to 10% of patients when biopsies are only taken from the left side (e.g., via flexible sigmoidoscopy). The colon mucosa appears normal and histologic findings show either a colonic subepithelial collagen band (i.e., collagenous colitis) or ≥ 20 intraepithelial lymphocytes per 100 surface epithelial cells. Crypt architecture is not distorted as is seen in CD and UC.

***Treatment:*** Management of microscopic colitis includes removal of potential culprit medications, such as NSAIDs, SSRIs (which require consultation with prescribing physician and/or tapering), and PPIs[53]. Anti-diarrheals such as loperamide may help with symptoms of chronic diarrhea. If patients have more severe diarrhea, first line treatment is typically budesonide, a locally active

corticosteroid with low systemic absorption. Most patients have significant symptom improvement within days, but complete resolution can take 6 to 8 weeks or longer. After at least 8 weeks, budesonide can be tapered off or to the lowest effective dose. Other medications that may be added as adjunctive therapies for those with incomplete resolution of symptoms include cholestyramine and bismuth subsalicylate. Rarely biologic agents, such as infliximab, adalimumab, azathioprine, and vedolizumab, are used. Current research does not support the use of any particular dietary intervention in these patients, with the exception of those with concomitant IBS-D and/or celiac disease. Because of this, no specific MNT interventions for microscopic colitis are recommended in Ch. 7, and RDNs are advised to personalize based on reported triggers.

## Irritable Bowel Syndrome (IBS)

***Overview:*** Irritable Bowel Syndrome (IBS) is a DGBI in which patients experience abdominal pain and alterations in bowel movements. IBS is subtyped based upon the predominant form of stool present (i.e., diarrhea-predominant, constipation-predominant, mixed and unspecified). IBS has no single cause and is thought to be multifactorial with alterations in GI motility, visceral hypersensitivity, inflammation, enteric immune activation, microbiome changes including bacterial overgrowth, bile acid malabsorption, food sensitivities, psychological distress and trauma, and genetic predisposition all recognized as possible contributors.

***Prevalence:*** IBS is prevalent in the U.S., with a recent study of 88,607 subjects using the Rome IV criteria reporting a prevalence of 6.1%[54]. Many prior studies found prevalences ranging from 10-15% of the population but were based upon older diagnostic criteria and studies with significant heterogeneity[55]. Only 30% of patients with IBS symptoms seek medical care. The prevalence

rates of IBS in those with AN and BN are reported to be as high as 41–69%[56].

***Diagnosis:*** IBS is diagnosed using the Rome IV criteria based on abdominal pain, change in the frequency of bowel movements, and change in the consistency of bowel movements. There are four subtypes of IBS based on the frequency and appearance of bowel movements: IBS with diarrhea predominance (IBS-D), where more than 25% of bowel movements fall into the 6-7 range on the Bristol stool scale and less than 25% of bowel movements fall into the 1-2 range on the Bristol stool scale; IBS with constipation-predominance (IBS-C), where more than 25% of bowel movements fall into the 1-2 range on the Bristol stool chart and less than 25% fall into the 6-7 range; IBS with mixed bowel habits (IBS-M), where more than 25% of bowel movements fall into the 1-2 range on the Bristol stool scale and more than 25% fall into the 6-7 range; and IBS unspecified (IBS-U) where patients meet diagnostic criteria but don't fall into one of the other subtypes[57].

***Treatment:*** Treatment of IBS includes diet, laxatives, antidiarrheals, antispasmodics, FDA-approved medications for the treatment of IBS based upon subtype, neuromodulators, and behavioral therapies. Diet typically involves the removal of lactose or gluten from the diet, and/or the use of the low-fermentable oligosaccharides, disaccharides, monosaccharides, and polyols (FODMAP) diet. The low-FODMAP diet involves three phases: elimination, reintroduction, and personalization. The elimination phase is a 2-6-week trial during which high-FODMAP foods are removed from the diet. In patients whose symptoms improve during the elimination phase, the reintroduction phase is used to reintroduce food groups in small, medium, and large quantities over 3-day periods of time to identify triggering food groups and amounts. Finally, the personalization phase is used to create plans for minimization of symptoms while still enjoying an expanded diet

with the ability to predict what foods may trigger symptoms. The low-FODMAP diet, as with any elimination diet, can be problematic in individuals with eating disorders and disordered eating, and it is recommended to screen for these behaviors prior to initiation of the low-FODMAP diet and, when possible, work with a registered dietitian[58]. For mild symptoms, antispasmodics can be used for pain, and either antidiarrheals or laxatives can be used for diarrhea and constipation, respectively. Antispasmodics include both prescription medications such as hyoscyamine and dicyclomine, as well as enteric-coated peppermint oil capsules. For moderate to severe symptoms in IBS-C, five FDA-approved medications currently exist including four secretagogues (lubiprostone, linaclotide, plecanitide, and tenapanor), as well as the sodium/hydrogen exchange antagonist (tenapanor). Lubiprostone, linaclotide, and plecanitide are also approved for chronic idiopathic constipation. For moderate to severe symptoms in IBS-D, three FDA-approved medications exist, including the gut-selective antibiotic rifaximin; mixed opioid receptor agonist/antagonist, eluxadoline; and serotonin receptor antagonist, alosetron[59,60]. Neuromodulators are used primarily for pain, though they may impact stool urgency, frequency, and consistency. The most commonly used neuromodulators are tricyclic antidepressants (TCAs) such as amitriptyline and nortriptyline, and selective serotonin reuptake inhibitors (SSRIs) which were once recommended but are no longer considered first-line treatment. Selective serotonin norepinephrine reuptake inhibitors (SNRIs) have benefits for pain without the degree of GI and sexual side effects that SSRIs have. Finally, behavioral therapies, namely gut-directed hypnotherapy and cognitive behavioral therapy, are efficacious for the treatment of all subtypes of IBS. They have been studied in face-to-face, in-person settings, as well as telephone and internet-/app-delivered therapies, and they are shown to have lasting effects in responsive patients (i.e.,

beneficial outcomes are demonstrated even years after the therapy has concluded).

## Lactose Intolerance

***Overview:*** Lactose intolerance refers to the inability to digest lactose, which is a naturally occurring sugar found in milk and many dairy products. This condition occurs when the small intestine does not produce enough of the lactase enzyme to digest lactose, resulting in lactose malabsorption. Common symptoms of lactose intolerance include abdominal pain, cramping, gas, bloating, and diarrhea (and, less often, constipation). Severity of symptoms varies and often depends on the amount of lactose consumed.

***Prevalence:*** Lactose intolerance is fairly common, affecting up to 68% of the global population and approximately 36% of the U.S. population[61].

***Diagnosis:*** Though some people are born without the ability to produce lactase, many people develop lactose intolerance with age as a result of reduced production of lactase or as a result of injury, infection, or disease affecting the small intestine. Diagnosis can be made with a hydrogen breath test, in which a known concentration of lactose is consumed, levels of breath hydrogen are monitored, and symptoms are recorded. High levels of breath hydrogen indicate lactose malabsorption and lactose intolerance. Diagnosis may also involve a trial elimination or reduction of lactose-containing foods for 2 weeks and monitoring for symptom improvement, though many patients notice improvement sooner than 2 weeks.

***Treatment:*** Treatment of lactose intolerance involves either partial or complete elimination of lactose-containing foods and/or use of lactase supplements.

## Metabolic Dysfunction-Associated Steatotic Liver Disease (MASLD) (previously Non-Alcoholic Fatty Liver Disease)

***Overview:*** As the field of hepatology is embracing new terminology, this section will refer to both MASLD (new language) and non-alcoholic fatty liver disease/NAFLD (language appearing in most research studies to date). MASLD is characterized by the presence of fat in the liver in the absence of a secondary cause, such as alcohol consumption[62]. NAFLD includes NAFL, and non-alcoholic steatohepatitis (NASH) is the most common liver disease in the Western world. NAFLD is associated with high BMI, hypertension, dyslipidemia, and insulin resistance or overt diabetes. MASLD can progress to cirrhosis and is becoming a leading cause for liver transplantation. One of the challenges with MASLD is difficulty predicting which patients will progress to cirrhosis. Patients with MASLD may have normal liver labs in the setting of advanced liver disease or may have abnormal liver labs with no evidence of advanced liver disease. Though metabolic abnormalities most often accompany MASLD, MASLD can be seen in patients without these findings, as well, sometimes referred to as "lean MASLD."

***Prevalence:*** The prevalence of MASLD in the U.S. is 10-46% depending on the study, with biopsy-proven NASH present in 3-5%. Worldwide, the estimated prevalence of NAFLD is 32%, with increasing prevalence seen from 26% in studies prior to 2005 to 38% in studies from 2018 and beyond[63]. Binge-eating disorder was present in 23.1% of patients with NAFLD in one study[64].

***Diagnosis:*** MASLD often has no physical exam findings prior to the development of advanced liver disease, though patients may have hepatomegaly on exam. Labs may show mild to moderate elevation in aminotransferases (aspartate aminotransferase/AST and alanine aminotransferase/ALT), as well as elevations in alkaline phosphatase, though albumin and total bilirubin are typically

normal until patients develop cirrhosis. MASLD is usually suggested by imaging (typically ultrasound) demonstrating steatosis (i.e., fat) in the liver, though it may be seen on CT and MRI imaging as well. Elastography, either ultrasound or MRI, can be used to assess liver stiffness as a surrogate for liver biopsy and histology. Patients with physical exam findings suggestive of cirrhosis, age over 45 with comorbidity (e.g., diabetes, or high BMI per a weight-centric model), and ferritin > 1.5 times the upper limit of normal may be candidates for liver biopsy to assess the stage of liver fibrosis. In addition, patients with abnormal laboratory tests and/or suspicion of secondary causes of liver disease such as autoimmune or drug-induced liver disease may be recommended for biopsy to attempt to identify other causes of liver disease, which may have specific treatment to prevent progression.

***Treatment:*** Initial treatment of MASLD often involves dietary modifications that favor a plant-forward intake (e.g., "Mediterranean" diet), as well as regular exercise where appropriate/desired. Abstinence from alcohol, dietary supplements and some medications avoids further injury to the liver. Diabetes medications such as glucagon-like peptide-1 (GLP-1) agonists and pioglitazone may be used in certain patients with NASH due to observed improvements in various metabolic markers[65,66]. Under a weight-centric model, weight loss of 5-7% is typically recommended (with bariatric surgery suggested to some patients), despite risk of severe harm to individuals with eating disorders/disordered eating.

## Rumination Syndrome

***Overview:*** Rumination syndrome is a disorder of gut-brain interaction in which ingested food is effortlessly regurgitated into the mouth after most meals. The food is either spit out or re-swallowed. Rumination syndrome is often misdiagnosed for considerable amounts of time as nausea and vomiting or as

gastroesophageal reflux disease. Patients with rumination syndrome may lose significant amounts of weight due to frequent postprandial regurgitation, as well as avoidance of food intake due to fear of regurgitation.

***Prevalence:*** Rumination syndrome affects 3.1% to 5.8% of the general population[67,68]. One study found that criteria for rumination syndrome were met in 7% of individuals with EDs[26].

***Diagnosis:*** Rumination syndrome is a clinical diagnosis made using criteria from Rome IV or the DSM 5. Per Rome IV, patients must meet all of the following conditions: (1) persistent or recurrent regurgitation of recently ingested food into the mouth with subsequent spitting or rechewing and swallowing and (2) regurgitation not preceded by retching. Additional features that may be present include effortless regurgitation events usually not preceded by nausea, regurgitant contains recognizable food that might have a pleasant taste, and cessation of rumination when the regurgitant material becomes acidic. Testing is focused on ruling out mechanical obstruction with upper endoscopy, and occasionally, imaging such as an upper GI barium study and/or computed tomography (CT) or magnetic resonance (MR) enterography. Impedance pH manometry can also be used to demonstrate reflux events extending to the proximal esophagus and associated with an increase in gastric pressure to > 30 mm Hg. It can be helpful to ask patients or family members to record videos of the events to assess the degree of retching preceding regurgitation.

***Treatment:*** Treatment of rumination syndrome includes education and diaphragmatic breathing to reduce postprandial intragastric pressure and increase pressure at the junction of the esophagus and stomach[69]. Diaphragmatic breathing should be timed after meals for 10 to 15 minutes or until the sensation of regurgitation

resolves. A psychologist and/or speech language pathologist may be able to instruct on the use of diaphragmatic breathing, as well as address underlying and contributing mood disorders and/or mealtime anxiety. For refractory cases, use of baclofen, a muscle relaxant, may help reduce rumination episodes. There are no well-supported nutrition interventions for rumination disorder, so this condition will not be featured in Ch. 7.

## Small Intestinal Bacterial Overgrowth (SIBO) and Intestinal Methanogen Overgrowth (IMO)

***Overview:*** Small intestinal bacterial overgrowth (SIBO) is a condition in which colonic bacteria are seen in excess in the small intestine; intestinal methanogenic overgrowth (IMO) is similar and occurs when archaea are seen in excess throughout the intestines. Both cause symptoms of gas, bloating, abdominal pain, diarrhea and, occasionally, malabsorption (although IMO is more commonly associated with constipation). Gastric acid, bile, digestive enzymes, secretory IgA antibodies, the migrating motor complex (MMC), and the ileocecal valve all work to prevent overgrowth of bacteria in the small intestine. SIBO/IMO may occur in patients with motility disorders (e.g., IBS, narcotic use, neuromuscular conditions such as scleroderma, multiple sclerosis, and diabetes), anatomic alterations of the GI tract (e.g., Roux-en-Y gastric bypass, tumors of the GI tract, strictures due to radiation or IBD, and diverticulosis of the small intestine, bowel resection that includes the ileocecal valve), immune deficiencies (e.g., common variable immune deficiency/CVID, human immunodeficiency virus/HIV; and selective IgA deficiency), metabolic and systemic disorders (e.g., diabetes, cirrhosis, and pancreatic insufficiency), and medications (e.g., antibiotics and proton pump inhibitors). In patients with eating disorders, loss of the MMC may occur, resulting in SIBO/IMO. In addition to symptoms, patients may become deficient in vitamin B12 due to bacterial competition and malabsorption.

***Prevalence:*** The prevalence of SIBO/IMO is not well established, with estimates ranging from 2.5% to 22% and wide variations depending on the study population and the method of testing used. No current studies have evaluated the prevalence of SIBO/IMO in eating disorders.

***Diagnosis:*** The gold standard for SIBO/IMO is upper endoscopy with jejunal aspirates for microbial culture showing > $10^3$ colony forming units per milliliter. However, this is rarely done in practice due to the invasive nature of the procedure, requirement for sedation, few laboratories willing to quantify microbial burden, and risk of contamination leading to false positive results. More commonly, breath testing using a carbohydrate substrate such as glucose or lactulose is performed. A baseline breath is collected and analyzed for hydrogen and methane and, in some cases, hydrogen sulfide. Then a carbohydrate substrate is consumed, which is metabolized by microbes into compounds (hydrogen, methane, and/or hydrogen sulfide) that are absorbed into the bloodstream and exhaled in the breath. Peaks in hydrogen of 20 parts per million or greater, methane of 10 parts per million or greater, and/or hydrogen sulfide of 3 parts per million or greater are considered abnormal[70].

***Treatment:*** SIBO/IMO is treated with antibiotics, with rifaximin considered first-line for elevated hydrogen and both rifaximin and neomycin for elevated methane (studies are ongoing but have suggested a role for bismuth subsalicylate for elevated hydrogen sulfide). Alternative antibiotic regimens may be required if insurance does not cover rifaximin, which is not FDA-approved for SIBO but is for IBS-D (for which SIBO is often a major driver of symptoms). Correction of micronutrient deficiencies with vitamin B12, niacin, thiamine, fat-soluble vitamins, and iron may be necessary. One study suggests that a two-week elemental diet may improve symptoms[71], but cost, palatability, risk of recurrence, and

general risk of harm to those with ED limit the utility of this treatment.

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CHAPTER 5

# Psychogastroenterology Foundations

Psychogastroenterology is a new area of practice and research within the field of gastroenterology. Psychological interventions for GI conditions (referred to as *brain-gut psychotherapies*) have extensive research demonstrating their effectiveness at improving GI symptoms and quality of life[1]. Unlike other psychological treatment modalities, brain-gut psychotherapies are generally short-term (e.g., as few as 4 sessions) and focused on GI symptom management; their skills-based interventions target unpleasant GI sensations, reduce avoidance behaviors, and increase resilience in the face of a chronic condition.

## Overview

Current brain-gut psychotherapies utilized by gastrointestinal mental health practitioners (GI-MHPs) include cognitive behavioral therapy (CBT), gut-directed hypnotherapy, acceptance and commitment therapy (ACT), written exposure therapy (WET), prolonged exposure therapy (PE), diaphragmatic breathing, interoceptive exposure, mindfulness-based therapies (MBT), and psychodynamic therapies. There are more than 30 randomized controlled trials for CBT and 11 for hypnosis in IBS alone that demonstrate their efficacy[1].

The Rome Foundation, a nonprofit organization that spearheads research and the development of clinical criteria for disorders of gut-brain interaction (DGBIs), recommends that brain-gut psychotherapies be delivered by a specialized GI-MHP. Medical work-up should be completed and a formal diagnosis made prior to engaging in these interventions. Coordination and collaboration should occur between the GI-MHP and other members of the healthcare team. These specialized services are often covered by insurance.

Brain-gut psychotherapies are considered inappropriate for clients with untreated or unstable mental health conditions (separate from their GI issues), clients with cognitive limitations or language barriers, and clients experiencing active substance use disorders. Another contraindication identified by the Rome Foundation is having a BMI <17 (or otherwise untreated ED), as brain-gut psychotherapies are not designed to address eating disorder symptoms directly. If a person struggles with an active eating disorder, it is recommended to use GI-CBT in addition to existing treatments. More research is needed to determine the optimal type of brain-gut psychotherapies for people with eating disorders.

Mental health comorbidities, including eating disorders, are extremely common in patients with gastrointestinal disorders. Within this population, anxiety, depression, suicidality, obsessive compulsive disorder (OCD), PTSD, and sexual dysfunction are seen in rates higher than or equal to the general population. Early adverse life events and PTSD are associated with irritable bowel syndrome (IBS), defecation disorders, and functional constipation. Those with abnormal anorectal manometry and balloon expulsion test have higher rates of prior emotional abuse and poorer mental health[2].

## Cognitive Behavioral Therapy

Cognitive Behavioral Therapy (CBT) is one of the most commonly studied psychological interventions for IBS and can be an effective treatment for patients who do not respond to standard pharmacotherapy[3]. It has not been well studied in the treatment of other DGBIs but is a promising approach. The modality can help people with IBS understand the relationship between their thoughts, feelings, and behaviors in order to shift those thoughts and implement effective coping strategies to improve quality of life. Strategies may include psychoeducation, stress management, self-monitoring, muscle relaxation, problem solving, and relapse prevention training. CBT may also be combined with exposure therapy and/or mindfulness practices[4]. It is proposed that CBT positively affects IBS by alleviating psychological distress, such as anxiety and depression, and improving communication via the gut-brain axis[4].

There are many different forms of CBT for IBS, including face-to-face or online, individual or group settings. Internet-based CBT can provide a more cost-effective and accessible option to IBS patients.

## Gut-Directed Hypnotherapy

Gut-directed hypnotherapy (GDH) has been used as a therapeutic technique for IBS since the 1980s. It affects the gut-brain axis through several pathways and results in reduced global symptoms (e.g., bloating, pain, visceral hypersensitivity, hyper-/hypomotility) and lowered levels of stress, anxiety, and depression[1]. While some research exists supporting the use of GDH for GI conditions such as ulcerative colitis and reflux, the bulk of the research has been conducted on patients with IBS. According to a systematic review and network meta-analysis conducted in 2020, GDH and CBT had the largest evidence base and were most effective long term[5].

In 2016, researchers from Monash University conducted a study for participants with IBS comparing the effects of a low-FODMAP diet versus GDH. While the effects of each therapy improved GI symptoms such as pain, bloating, gas, and stool consistency, the GDH intervention was more effective for improving anxiety and depression[6].

GDH is not the same as *stage hypnosis*. It is a therapeutic technique delivered by trained clinicians and is different from other psychological therapies in that it works on the subconscious mind. Typically, hypnosis begins with induction and deepening techniques that allow the subject to go into an altered consciousness or trance state. In this trance state, the client is still in full possession of their faculties while the clinician provides relaxing imagery, suggestions, and metaphors to the subconscious mind, which are then more accessible to the client outside of sessions[7]. Examples of relevant imagery and metaphors might include:

**Table 5.1 Sample Language Used in Gut-Directed Hypnotherapy**

| Imagery/Metaphor Examples | Suggestion Examples |
|---|---|
| *Imagine a wave of medication moving through from your stomach down into your intestines. This medication coats your intestines, making them less and less sensitive to pain and discomfort.*<br><br>*Imagine that your belly is a balloon. Notice now that you are holding a pin. Go ahead and take that pin to pop the balloon. Little by little the air starts to release, lowering the pressure and decreasing the bloating and distention.* | *Your bowel habits will continue to improve day by day, week by week.*<br><br>*You will be able to eat without discomfort and pain.*<br><br>*Your digestive tract will begin to function with a healthy soothing rhythm.* |

## Mindfulness-Based Stress Reduction

Mindfulness-Based Stress Reduction (MBSR) therapies train the individual to notice thoughts and situations in a nonreactive and accepting manner[8]. Techniques used in MBSR include body scans, meditation, and yoga.

MBSR has been studied in many GI conditions such as IBD and IBS, with suggested improvements in stress, depression, quality of life, health-related quality of life, symptom scores, and possibly inflammatory markers[9-12]. Although mindfulness strategies may seem universally beneficial, they should still be employed thoughtfully; some individuals may experience an increase in psychological symptoms such as increased depressive symptoms, interpersonal sensitivity, paranoid ideation, and psychosis[13].

## The Intersection of Trauma and GI Conditions

Preliminary research by GI-MHPs shows that a significant portion of individuals with chronic and/or life-threatening gastrointestinal conditions experience post-traumatic stress syndrome (PTSS). PTSS due to medical trauma can cause flashbacks, nightmares, hypervigilance, disrupted sleep, and low mood. Researchers found that 25-30% of patients with IBD met criteria for PTSS, which was more likely to occur in participants with hospitalizations, surgical bowel resection, worse disease severity, and the diagnosis of a life-threatening illness[14]. Participants of one qualitative study described the following factors to be significantly detrimental to their mental health: viewing medical results without explanation, the uncertainty of IBD trajectory, feeling out of control of one's body, intense physical pain, psychiatric symptoms, stigmatizing physical changes, prolonged delays in diagnosis, and NG tube placement. Protective factors included positive communication with providers, feeling listened to and provided with thorough information, education about their condition and treatment

options, shared medical decision-making, and non-stigmatizing language from providers[15].

Though not yet demonstrated in psychogastroenterology literature, we authors have seen PTSS and PTSD manifest as onset of a new eating disorder and/or flare of previous eating disorder. Future directions must address both the link between PTSS in GI disorders and disordered eating, as well as effective therapies for treating these co-occurring disorders. Modalities effective in treating trauma and related conditions that may expand into the GI sphere in the future include eye movement desensitization and reprocessing (EMDR) therapy and written exposure therapy (WET)[16].

## Accessing Psychogastroenterology Modalities

Depending on a client's geographical location, finances, and presentation, they may have access to in-person services, digital prescription programs and/or app-based programs. Trained psychogastroenterology providers can be found through the Rome Foundation's online directory (https://romegipsych.org/) or Dr. Olafur S. Palsson's online directory (https://ibshypnosis.com/). Digital programs are attempting to fill the provider gap, and entities known at the time of publishing can be found in the Resources section.

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SECTION II

# NUTRITION CARE PROCESS

CHAPTER 6

# Screening & Referral for Disordered Eating

Screening and referral may occur before and during the nutrition care process. The goal of screening and referral is to identify individuals who can benefit from nutrition intervention using appropriate tools and to collaborate with interdisciplinary team members to identify and refer to other appropriate interventions by non-RD team members.

It is important to acknowledge the limitations that currently exist with regard to screening of and referral for co-occurring EDs and GI disorders: lack of consideration for diverse populations in the research and design of screening tools; non-validated screening tools in disordered eating and co-occurring GI conditions; inequitable access to healthcare resources (including specialty referrals); and bias in provider discretion as to whether or not to screen for disordered eating, among others.

## The Registered Dietitian Nutritionist's Role

The RDN's scope of practice includes identifying patients with disordered eating behaviors and collecting information about eating behaviors that may help inform a psychological diagnosis[1]. While some screening tools must be administered by a trained mental health professional (see Chapter 2), many can be self-

administered with the help of an RDN, which are included in Table 6.1.

After identifying a client with disordered eating symptomatology, the RDN should refer to an appropriate physician to assess and manage potential medical complications and to an appropriate mental health provider for psychological diagnosis and care (if the client has not already established care). The RDN should then participate in interdisciplinary collaboration with these providers to determine an appropriate care plan and provide clinical updates as needed.

It is not within the RDN's scope of practice, unless otherwise conferred by appropriate credentials and training, to administer assessment tools reserved for licensed mental health professionals, provide a mental health diagnosis, develop a care plan independently, or deliver treatment protocols reserved for licensed mental health professionals.

## Screening Tools for Eating Disorders

Screening tools can be useful in gathering information about the patient's relationship with food and thus what type of interventions may be warranted. They are NOT diagnostic of an eating disorder and can be subject to both error and bias. Most assessments are "face valid," which means the wording is such that if a patient wants to misrepresent their symptoms (i.e., lie and/or hide), it is fairly easy to do so. Additionally, most of these tools were developed in groups that are not reflective of the ethnic, gender, or sexual diversity found in the United States; these tools should be used with awareness that the results may not fully or accurately capture an individual's symptoms.

These tools can and should be used by allied health professionals, including RDNs, to assess risk and identify appropriate referrals for

a proper diagnosis. If a patient does not score positively on the screening tool, it may not prompt the RDN to refer to a psychologist. If they do score positively, the screening tool can serve as a conversation starter to explain and support additional referrals and/or interventions.

Only a few of the below tools have been validated in individuals with gastrointestinal disorders, highlighting the limitations of the other screening tools. Validation of existing tools in populations with diet-related chronic health conditions is a necessary area of further research. Table 6.1 below contains examples of various ED screening tools and considerations for their use. Refer to Resources for the Registered Dietitian Nutritionist section for links to web-based and printable versions of many of these screening tools.

**Example of Clinical Application**

Choose one or more screening tools to integrate into your nutrition care process during an initial assessment, upon reassessment, or prior to enrolling in a group program. For example, combine the use of the EDE-Q + NIAS to detect both weight- and shape-related eating disorder risk and non-body image related maladaptive eating behaviors, as recommended by recent international consensus on patient-centered outcomes in eating disorders[2].

**Table 6.1. Common Eating Disorder Screening Tools**

| Tool | Format | Validated Population | Notes on Use |
|---|---|---|---|
| **Binge Eating Scale (BES)** | 16 items<br>multiple choice | Individuals with BMI >30 | Self-reported questionnaire that measures behavioral symptoms (rapid eating, eating large amounts of food, eating in secret, eating until feeling ill) and affective/cognitive symptoms (guilt, feeling lack of control during eating)[3].<br>More recently, there have been efforts to expand the BES's utility to the general population, where it has been found to have good reliability and validity[4]. |
| **Coeliac Disease Food Attitudes and Behaviors (CD-FAB)** | 11 items<br>7-point scale | Adults with celiac disease | Assesses eating attitudes and behaviors related to handling of food (cross-contamination), trust, risk-taking, and food safety. Higher scores are associated with psychological distress and impaired quality of life and suggest increased maladaptive eating. Beyond the celiac population, the CD-FAB may have a broader clinical application, so future research is warranted[5]. |
| **Disordered Eating Screen for Athletes (DESA-6)** | 6 items<br>multiple choice & yes/no | Adolescent athletes ages 13-19 | A brief, easy-to-administer screening tool for recognizing disordered eating in athletes for the purpose of applying early interventions[6]. |
| **Eating Disorder Screen for Primary Care (ESP)** | 5 items<br>yes/no | Primary care population and university students | Brief questionnaire where research has determined its two most effective questions for assessing ED risk are *Does your weight affect the way you feel about yourself? Are you satisfied with your eating patterns?*[7]. |
| **Eating Disorder Examination–Questionnaire (EDE-Q 6.0)** | 28 items<br>7-point scale | Individuals ages 14+ | Self-report questionnaire based on the clinician-led diagnostic tool Eating Disorder Examination. Assesses range and severity of features associated with diagnosis of eating disorder based on 4 subscales and a global score. The subscales are restraint, eating concern, shape concern, and weight concern. Global score of 4+ is considered clinically significant. Overall reliability and validity are comparable to the full examination. Some ethnic and racial variations demonstrated[8]. |
| **Eating Disorder Examination–Questionnaire Short (EDE-QS)** | 12 items<br>7-point scale | Adults in eating disorder treatment UK, 90% female<br>dx: OSFED, AN-R, AN-BP, BN, BED | Derived from the EDE-Q that can be used for outcome monitoring in treatment and research, as well as screening. The EDE-QS was designed to have high reliability and validity, including strong positive correlations with the EDE-Q, other measures of eating disorder pathology, measures of comorbid psychopathology, and strong negative correlations with measures of quality of life. It is considered sufficiently sensitive to identify patients with an ED[9]. |
| **Eating Attitudes Test (EAT-26)** | 26 items<br>yes/no & 6-point scale | Adolescents, adults and athletes (not validated among those with diet-related chronic health conditions and binge-eating disorder); available in multiple languages | Self-report questionnaire capturing the behaviors and attitudes characteristic of EDs. Items on the EAT-26 that may score higher in those with diet-related chronic health conditions compared with controls include: "I find myself preoccupied with food"; "I feel that food controls my life"; "I display self-control around food"; and "I give too much time and thought to food"[10]. |

| | | | |
|---|---|---|---|
| **EcSatter Inventory (ECSI 2.0)** | 16 items<br>5-point scale | Adults (although most participants have been female, white, educated, BMI 25-30, physically active, and food secure, without eating disorder diagnoses)[12] | Self-report questionnaire assessing the four domains of the Satter Eating Competence model (ecSatter): eating attitudes, food acceptance, food regulation, and contextual skills[11]. Higher eating competency scores correlate with increased weight satisfaction, physical activity, and preference for fruits and vegetables. Higher scores also correlate with decreases in restrained eating, disinhibition, hunger, weight dissatisfaction, food dislikes, drive for thinness, and other indicators of eating disorders[12]. |
| **Fear of Food Questionnaire (FFQ-18)** | 18 items<br>5-point scale | Individuals with IBS, IBD, phagophobia, emetophobia, suspected ARFID | Assesses whether a fear of food that impacts quality of life exists and is novel in that it may serve as a screening tool for ARFID (specifically Fear-ARFID) that appears to be valid in multiple populations (those with IBS, IBD, emetophobia, phagophobia). Further research is needed although initial results appear promising[13]. |
| **Food-Related Quality of Life (FRQOL)** | 29 items<br>4-point scale | Individuals with IBD | Assesses the prevalence and magnitude of the psychosocial impact of IBD and IBS. Specific measures include those that capture the impact food restrictions and fear of relapse have on family celebrations and religious occasions, uncertainty around bowel function, and IBD's impact on travel and autonomy due to the perceived need to be near a toilet. Higher scores indicate better food-related quality of life[14]. This screen has been similarly validated in IBS populations[15]. |
| **Nine Item ARFID Screen (NIAS)** | 9 items<br>6-point scale | American adults | Questionnaire with three subscales that represent each ARFID presentation (sensory, anhedonic, fear of aversive consequences). Use of NIAS is recommended in tandem with another ED screening tool (such as EDE-Q or EDE-QS) to avoid incorrectly classifying patients with other EDs as having ARFID[16]. |
| **Screen for Disordered Eating (SDE)** | 5 items<br>yes/no | Adult female veterans age 18-70 | First short tool to be inclusive of BED, AN, and BN, recommended for use in primary care settings by allied health providers[17]. |
| **SCOFF Questionnaire** | 5 items<br>yes/no | Low-normal weight clinics and students | Best used for young women at risk of AN and BN. Short screening tool that is easily used in a variety of settings but less sensitive to BED[18]. |

## Suggested Language for Screening for EDs in GI Practice

Given that there are no validated screening tools in this population, it is our expert opinion that the above screening tools be used in addition to the following open-ended questions to help identify disordered eating. The following questions may elicit "flags" that can be further assessed. If the practitioner does not feel

competent at fully assessing these behaviors, they may use these screening questions to determine that a more appropriate referral is indicated.

In many settings, screening and assessment can blend into one another. For example, a screening question with a positive answer can naturally prompt additional questions that help form the nutrition assessment and diagnosis. While this practice is done typically during the nutrition assessment, it is not always possible when time or circumstances are limited to just screening. In that case, screening may lead to nutrition assessment at a future date if the RDN intends to continue care.

We recommend the use of open-ended questions for screening. Open-ended questions allow for a "soft delivery" where the patient may divulge as much as they feel comfortable sharing. They are most appropriate for someone who is precontemplative or unaware of their need to address disordered eating. Direct or closed-ended questions sometimes elicit honesty, but many patients with disordered eating will want to conceal their disordered behaviors and/or be embarrassed to share their experience. If a close-ended question is asked, it is best to then follow up with an open-ended question to elicit a more complete response and understanding. It is recommended to ask these questions individually with the patient and not in front of parents or family members. Family members may be asked about any concerns with the client's eating behaviors on an individual basis (provided there is consent from the client).

**Table 6.2 Informal Screening Questions for Disordered Eating**

| Question | Flags for Disordered Eating |
|---|---|
| How would you describe your relationship with food? | • Feelings of anxiety about food, not enjoying food, fear of eating in situations with little control |

| | |
|---|---|
| | • Moral associations with food ("good" food or "bad day of eating")<br>• Any skipping of meals, loss-of-control eating |
| Do you avoid eating certain foods? For what purpose? | • Body image/weight control<br>• Morality ("good/bad" foods)<br>• "Optimal" health<br>• Feeling judged or ashamed<br>• Food insecurity<br>• Palatability/texture<br>• Management or avoidance of digestive symptoms |
| How do you feel that your attitude or behavior toward eating impacts your daily life? | • Avoiding eating in social settings (e.g., at work with colleagues, going out with friends, family meals)<br>• Choosing not to dine out due to feeling limited by options or lack of control with ingredients, portions, etc.<br>• Avoiding travel or feeling anxious when traveling with regard to food options<br>• Negative impact on relationships (e.g., friendships, marriage, relationship with parents/kids, etc.)<br>• Preoccupation with food impacting school, work or other activities |
| What do you do for movement? How do you feel about moving your body? | • Primary motivation is related to weight control, burning calories, maintaining a certain physique<br>• Stress about getting enough activity<br>• Using physical activity as direct compensation for calorie intake<br>• Tracking/changing food intake (not for purpose of getting adequate intake/supporting energy needs)<br>• Continued physical activity despite worsening GI symptoms or other health issues |
| Are you open to changing how you eat? Liberalizing your diet? Incorporating fear foods? Challenging food rules/beliefs? | • Anxiety to the point of unwillingness/inability to change<br>• Creation of food rules that can mask disordered eating |

| | |
|---|---|
| | • Ready to improve symptoms but not if it will cause weight changes/gain |
| If eating more food and/or fear foods resolved most of your GI symptoms, would you be willing to do so?<br>If gaining weight resolved many of your GI symptoms, would you be willing to do so?<br>Gaining 5-10 lbs<br>How about 15-20 lbs?<br>Returning to pre-sickness weight? | • Unwillingness to gain weight to a biologically appropriate weight<br>• Setting limitations to the dietary work, such as a weight limit or not addressing certain food rules<br>• Acceptance of ongoing symptoms over discomfort of challenging new foods, food rules, etc. |
| Do you have a history of or an active eating disorder? | • History of eating disorder with/without treatment followed by other disordered eating flags<br>• Recent history of ED behaviors <6-12 months ago |
| Do you ever self-induce vomiting?<br>Do you ever use laxatives when you are not constipated?<br>Why do you think you vomit/need laxatives so frequently? | • Presence of self-induced vomiting<br>• Presence of excessive and/or compulsive laxative use |
| Can you describe how bloating feels to you? | • Confusing a normal feeling of fullness after eating with significant bloating and/or visible distention with other symptoms<br>• Refusal to challenge any new food if the first challenge caused some mild bloating |
| Can you describe a typical day of meals/snacks, from waking up to going to sleep?<br>Do you feel that you have enough energy to get through your day? | • Insufficient intake overall<br>• Fasting for periods of time while awake, delaying meals, skipping meals, exercise without fueling<br>• Refusal or unwillingness to alter eating habits (such as amount of food consumed in a meal, types of food consumed) |
| Do you follow any food rules?<br>Where does this preference come from? | • Presence of voluntary dietary restrictions<br>• Unwillingness to challenge food rules based on RD guidance |

| | |
|---|---|
| How long have you had this rule?<br><br>What was the original goal of this food rule? | • Not open to education about value of reintroducing excluded foods<br><br>*Some voluntary dietary restrictions may mirror disordered eating behaviors but are not necessarily indicative of disordered eating. It's important to further investigate the reasons behind these choices and rules. Examples include avoidance of processed foods, veganism, or use of diet food products (such as "preference" for egg whites, lower-carbohydrate alternatives, etc.).* |
| How do you think an elimination diet, modifying your diet, or tracking your food intake would impact your relationship with food? | • Response notes the following are likely:<br>  ○ Increase in obsessive thoughts about food<br>  ○ Hypervigilance over weight/body image<br>  ○ Would lead to counting calories<br>  ○ Would spur judgmental food thoughts |

## Referral to Appropriate Treatment

Screening and treatment for disordered eating may occur at any point in the nutrition care process. Many individuals' disordered eating is not apparent at the initial assessment, even to seasoned professionals experienced with eating disorders. This phenomenon will be addressed further in "What to Expect Working with Eating Disorders." Furthermore, disordered eating can arise or worsen in the midst of the nutrition care process, whether or not it is related to the initial diet intervention. In either case, reassessment and change in interventions is warranted. The RDN is highly encouraged to conduct ongoing screenings for disordered eating and/or informally gauge behaviors throughout their relationship with a client.

The goal of screening and referral is to identify individuals who can benefit from nutrition intervention and to collaborate with interdisciplinary team members and refer for care outside of the RDN scope of practice when indicated. In many cases, screening may suggest that additional team members and/or a higher level of support for disordered eating are appropriate for the patient.

If the RDN is well-versed in assessing and treating eating disorders, then it is appropriate to collect more information to assess the duration and severity of eating disorder pathology. The RDN should connect the individual to a physician specialized in treating eating disorders for an assessment of medical stability and of conditions that may have resulted from long-standing malnutrition (e.g., osteopenia, etc.). Likewise, a referral to a mental health provider specialized in eating disorders should also be offered. If the individual has already established care with a physician and a mental health provider, then care should be coordinated. Treatment decisions and plans should be agreed upon by the group and consistent messaging should be provided to the client.

If the RDN is not experienced at treating eating disorders, then a referral should be made to an eating disorders-informed dietitian so an assessment and treatment plan can be developed. The RDN is strongly encouraged to have an awareness of specialty providers in their area to facilitate referrals when needed.

There may be times where a patient is not ready or willing to change or does not have the resources to be able to access appropriate recommended treatment. In those cases, it may be appropriate for the RDN to establish rapport and continue to be an advocate for change until readiness for change becomes apparent. The RDN should also continue to monitor for medical and mental health stability, communicating their observations to the members of the healthcare team. The RDN should document all interventions and patient engagement.

Research shows that patients see the best outcomes when they are treated by an interdisciplinary team of a primary care physician, a mental health provider, and a registered dietitian, all of whom specialize in treating eating disorders. The disparity in those who display disordered eating symptoms versus those who are

screened and then actually receive care for these symptoms calls for more providers to be competent in treating this population.

## Where to Refer

Tables 2.2 and 2.3 in Psychological Foundations provides an overview of the level of care guidelines for eating disorders. As a general rule, according to APA, an individual is inappropriate for outpatient care if they meet the criteria for inpatient hospitalization due to the high risks of delaying treatment. Exceptions to this may occur in a harm reduction model of treatment in which individuals are fully informed of and consenting to these risks. There may be times when a patient is appropriate for a higher level of care (e.g., residential, PHP, IOP), but cannot or does not want to access it due to resources, life circumstances, prior ineffectiveness, lack of availability of appropriate programs (e.g., not trauma-informed, not equipped to handle co-occurring diagnoses, etc.), or other reasons. It is then up to the RDN, in coordination with the physician and mental health provider, to determine whether it is safe and/or ethical to continue working with the client at the outpatient level of care. Factors in this decision may include symptom severity, medical acuity, co-existing mental health issues, client's support system, and client's motivation and readiness to change.

## Having the Conversation

Introducing the need to connect a patient to a provider specializing in eating disorders can be a difficult conversation to have. Expressing care, understanding, and compassion are essential when explaining the need for a change in nutrition care, so patients do not feel abandoned or dismissed by the GI team. It may be helpful to explain the connection between eating disorders and GI disorders, as well as the need to prioritize treatment of the eating disorder (and resulting malnutrition) in order to potentially resolve the GI symptoms. It is important to also clear up any

misconceptions about eating disorders being related to size, weight, or BMI, and share that eating disorder severity is indicated by thoughts, behaviors, and impact on one's life.

GI providers are generally not equipped to give a specific eating disorder diagnosis or recommendations for a specific level of care or treatment modality. Specialist providers are best able to assess what is needed. Because eating disorders are psychological conditions, telling an individual, "You just need to eat," or similar expressions will not be helpful. Commenting on body, appearance or weight, including degree of malnutrition, is unlikely to motivate the patient in the intended way, and may inadvertently fuel the eating disorder.

Providers should recommend a potential higher level of care as an opportunity for increased or more specialized support. They should explain that they can continue to meet with the patient either concurrently or in the future when it is more relevant. In many cases, nutrition interventions will not be effective without adequate support or when other comorbidities are more pressing.

**Table 6.3. Sample Referral Conversation Using Therapeutic Language**

| Conversation Goal | Expressing It |
|---|---|
| Rapport-Building | Thank you so much for taking the time to answer all of my questions and sharing what you've been experiencing lately. |
| Validation | I can't imagine what it's been like for you these last few months feeling so uncomfortable and not knowing what was going on. |
| Asking for Consent | May I offer you some background information on the mind-body connection and how it impacts overall health before I share my recommendations? |
| Psychoeducation | I'm sure you've heard the phrase "mind-body connection," but let's talk about what that actually means. In the simplest |

| | |
|---|---|
| | terms, that mind-body connection is the idea that what we do to our body directly impacts how we think and feel, and how we think and feel directly impacts how our bodies function. An example of this is when you were a kid in school and your teacher called on you for an answer and you weren't paying attention. Remember the butterflies in your stomach? Or the cold sweat you broke out into? Or how your voice felt tight? That's the mind-body connection! You were caught off guard, realized you didn't have the answer, and all of a sudden there was a cascade of physical reactions. |
| Name Concerns | Based on our conversation, I have concerns that your approach to nourishing yourself may be exacerbating your physical symptoms. When the body isn't able to have its biological needs met (including nutrition), it creates stress, which can intensify any discomfort you already have. |
| Describe Approach | Because the mind and body work in tandem, I want to offer you some dietary and psychological tools that might help alleviate your symptoms. Let's start with the diet. Based on the symptoms you've described (X, Y, Z), here are the nutritional and non-dietary interventions I might suggest… [describe appropriate interventions and theoretical mechanisms of action]. |
| Referral | I also noticed throughout our conversation that a lot of your thoughts and behaviors related to food and your body seem rigid and distressing to you. Have you ever been evaluated for disordered eating? ... I think addressing the mental health piece could be a really positive addition to our work together and, if you're open to it, I'd like to refer you to someone who I think could be a good fit. Healing the food issues will make healing the GI issues easier to navigate. |
| Ask for Buy-In | We covered a lot today. How are you feeling about these recommendations? What are you open to trying? |

## What to Expect Working with Eating Disorders

Treating a client with an eating disorder can be markedly more challenging than working with clients with GI conditions alone. Many eating disorders are *ego-syntonic*; they can be fueled by an individual's personality and beliefs. For example, restrictive eating disorders often reinforce rigid, perfectionistic attitudes about

health and encourage behaviors that are socially accepted and praised as "disciplined." Thus, it's hard for the person struggling with restrictive eating to recognize the intensity of their behaviors or even believe they need help. The overarching challenge is to caringly convey to the client that their beliefs and behaviors are not only unsubstantiated, but also harmful to their health. So, how might this manifest in sessions? It may present as resistance, bewilderment, a constant questioning of your expertise, poor follow-through on agreed-upon goals, defensiveness, frequent requests for you to justify the treatment plan, being less than truthful, premature termination of treatment, and expressing negative feedback about the care provided. Though discouraging, it is important to remember that this is a consequence of a malnourished brain that has impaired flexibility.

## Building Rapport

Building rapport with a client with an eating disorder is key to the beginning stages of treatment. While the brain is still malnourished and unable to fully integrate new information, the best foundation for recovery lies in providers' ability to engender trust, despite the difficult changes recommended throughout treatment.

Motivational Interviewing is a communication strategy that seeks to build rapport and momentum toward behavioral change in people who are experiencing ambivalence. This modality is ideal for dietitians and other disciplines outside of the mental health field, as it requires no therapeutic training and can be adapted to most settings. The role of the provider is to guide the client through a discussion of a behavior change *without* the use of coercion or unsolicited advice. Motivational Interviewing is influenced by the Transtheoretical Model of Change, which posits that individuals can be categorized into different stages of change depending on their expressed readiness[19] (see Table 6.4).

## Table 6.4. Characteristics of the Stages of Change

| Stage | Characteristics |
|---|---|
| Precontemplation | Client has no intention of changing behavior in the next six months |
| Contemplation | Client is strongly inclined to change behavior in the next six months |
| Preparation | Client intents to take action in the next month |
| Action | Behavior change has been implemented for at least six months |
| Maintenance | Behavior change has been incorporated for more than six months and the likelihood of returning to old behavior is low |

*Source: Adapted from Prochaska & Norcross, 2001*[19].

Providers are likely to find that the vast majority of their patients with restrictive eating behaviors are in the Precontemplation Stage, regardless of whether they have had treatment before. This is likely due to the normalization of disordered behaviors within the culture (e.g., dieting, excessive exercise, fasting, limiting food groups). Individuals struggling with disordered behaviors that are stigmatized (e.g., bingeing, self-induced vomiting) are more likely to be in the Contemplation Stage. Table 6.5 offers examples of how clients might express precontemplation and strategies for responding.

## Table 6.5. Responding to Precontemplation

| Theme | What Client May Say | Strategy |
|---|---|---|
| Reluctance | "I'm not ready for that"<br>"I'm not sure I need to…"<br>"I'm afraid to…"<br>"I can't" | Listen actively and provide feedback in a sensitive and empathic manner |
| Rebellion | "I'm telling you right now, I'm not…"<br>"You can't make me…" | Offer options so that the client can begin to consider |

| | | |
|---|---|---|
| | "I can do what I want in my own home" | what step they might be willing to take |
| Rationalization | "Everyone does it"<br>"Other people do way worse to their bodies"<br>"My mother did the same" | Express empathy and use reflections to find ways to align with the client and establish trust in the provider |
| Resignation | "There's nothing I can do about it"<br>"I've always managed things"<br>"It's too hard"<br>"It's impossible" | Offer hope for their ability to change and explore both benefits of recovery and costs of not changing |
| Reception/Deception | "I'll do whatever you say"<br>"I know I need help"<br>"What should I do?" | Be wary of whether client may be making efforts to appear agreeable and say what they think is expected of them; kindly point out later discrepancies between their words and actions, if applicable |

*Adapted from Diclemente & Velasquez, 2002*[20]

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CHAPTER 7

# Nutrition Assessment

The Nutrition Care Process consists of Assessment, Diagnosis, Intervention, Monitoring and Evaluation steps (ADIME). The goal of nutrition assessment is to collect relevant information to identify nutrition-related problems and, ultimately, develop a nutrition care plan.

Throughout the nutrition assessment, the RDN may encounter limitations in the tools they choose to use. These may include non-diverse research populations supporting the use of these measures, lack of validated assessment tools for co-occurring EDs and GI disorders, provider bias or inexperience in conducting the assessment (e.g., what to ask, how to ask, how to interpret), and reinforce the false notion of absolute "expertise" as opposed to taking into consideration lived experience.

## Medical History

A full medical history would include history of psychiatric diagnoses, family medical history, current medications, biochemical data, medical tests, and procedure data to serve as the basis for implementing nutrition prescription, goals, and interventions.

### Laboratory and Testing Results by Diagnosis

Biochemical data, medical tests, and procedures may further inform the assessment. The RDN should review available medical records to formulate their assessment. While the RDN will likely not be the ordering provider for these tests, they are well poised to help clients understand their results and collaborate with prescribing providers and monitor changes over time.

"Abnormal" findings are frequently absent even in cases of severe eating disorder pathology and physical malnutrition. A "normal" result should not be

used as evidence of health in this patient population, whereas an abnormal finding is very significant.

### Table 7.1. General Tests and Studies[1]

| Test or Study | Abnormal Finding | Interpretation |
|---|---|---|
| Complete Blood Count (CBC) | ↓ white blood cells (WBC)/leukopenia<br>↓ red blood cells (RBC)/anemia<br>↓ platelets/thrombocytopenia | poor nutrition |
| Comprehensive Metabolic Panel (CMP) | ↓ glucose<br>↓ sodium<br>↓ potassium<br>↓ chloride<br>↓↑ bicarbonate<br>↑ blood urea nitrogen (BUN)<br>↓↑ creatinine<br>↓↑ calcium<br>↓ phosphate<br>↓ magnesium<br>↓ total protein/albumin<br>↓↑ aspartate aminotransaminase (AST), alanine aminotransaminase (ALT), alkaline phosphatase | poor nutrition<br>water loading, laxatives, or syndrome of inappropriate antidiuretic hormone secretion (SIADH)<br>Vomiting, laxatives or diuretics<br>Vomiting, laxatives<br>↑ vomiting, ↓ laxatives<br>dehydration<br>↑ dehydration, renal dysfunction<br>↓ low muscle mass<br>↑ dehydration, ↓ poor nutrition<br>poor nutrition, early refeeding syndrome<br>poor nutrition, laxative use<br>malnutrition<br>poor nutrition |
| Thyroid Hormone Testing | ↓ thyroid stimulating hormone (TSH)<br>↓ thyroxine (T4)<br>↓ triiodothyronine (T3) | euthyroid sick syndrome, below metabolically appropriate weight |
| Gonadotropins and Sex Steroids | ↓ luteinizing (LH)<br>↓ follicle stimulating hormone (FSH)<br>↓ estradiol<br>↓ testosterone | prepubertal levels in hypothalamic amenorrhea |
| Pancreatic Enzymes<br>Amylase, Lipase | ↑<br>↑ | recurrent binge eating, self-induced vomiting<br>starvation-induced pancreatitis, binging-induced pancreatitis |
| Electrocardiogram (ECG) | ↓ heart rate < 60 (bradycardia)<br>↑ QTC < 450 msec<br>other arrhythmia | poor nutrition, purging, over-exercise |
| | | |

| | | |
|---|---|---|
| Orthostatic Vitals (Lying to Standing) | ↓ blood pressure by >10-20 points<br>↑ heart rate >20 points | slowed autonomic nervous system caused by malnutrition |

## Table 7.2. Tests and Studies Related to Abdominal Bloating & Distention

| Test or Study | Abnormal Finding | Interpretation |
|---|---|---|
| Kidney, Ureter, Bladder (KUB) X-ray | Evidence of dilation/obstruction | Used to assess for obstruction, non-obstructive ileus, or acute colonic pseudo-obstruction. May also identify large stool burden, foreign bodies, and/or air outside of the bowel in the setting of perforation (pneumoperitoneum). |
| Physical Exam | Increased girth measurement from laying to standing<br>soft/tender<br>hard/rigid | All patients with bloating/distention should be examined. Main components include inspection, auscultation (listening with stethoscope) for changes in bowel sounds, percussion (tapping to determine density of underlying structures, e.g., air versus solid), and palpation (pressing to feel for masses and to assess for pain) |
| SIBO/IMO Breath Testing | SIBO—↑hydrogen (20 ppm)<br>IMO—methane >10 ppm | SIBO with elevated hydrogen at 90 minutes, IMO with elevated methane at 90 minutes. Patients may have both. |
| Gastric Emptying Study | ≥10% of a test meal remaining in the stomach at 4 hours after ingestion (or more than 60% at 2 hours) | Abnormally slow gastric emptying due to gastroparesis, opioid use, GLP-1 agonists, malnutrition |
| Anorectal Manometry to Assess for Dyssynergic Defecation and Other Outlet Disorders | ↑ resting anal sphincter pressure, ↓ squeeze strength and/or endurance, ↓ relaxation or paradoxical contraction of the anal sphincters with simulated defecation | Dyssynergic defecation |

## Table 7.3. Tests and Studies Related to Celiac Disease & Gluten-Related Disorders

| Test or Study | Abnormal Finding | Interpretation |
|---|---|---|
| Tissue transglutaminase (tTg)<br>IgA, IgG | ↑ celiac disease, especially if >10x upper limit of normal range | All serum antibody levels should trend downward over 6-18 months on gluten-free diet (GFD) to confirm positive response. |

| | | |
|---|---|---|
| | ↑ mildly elevated (<3x normal range) in other autoimmune disease such as Hashimoto's, Lupus, DMT1 | Changes are not likely to indicate low/infrequent levels of gluten ingestion in people with CeD and are not always correlated with intestinal healing.<br><br>If IgA forms are measured, it is necessary to measure total IgA. If IgA deficiency is present, utilize IgG forms of these tests. |
| Endomysial Antibody IgA | ↑ titer above 1:10 in celiac disease | |
| Deamidated Gliadin Peptide IgA, IgG | ↑ celiac disease, sometimes non-celiac gluten sensitivity and other autoimmune disease | |
| HLA DQ2 and DQ8 genes (serum or saliva) | presence of either gene | Genetic susceptibility for developing CeD over lifetime<br><br>Not diagnostic of CeD alone, but can be helpful to rule out CeD as highly unlikely when both genes are negative/absent |
| Endoscopy<br><br>Pathology of Biopsies from Duodenum (Both Bulb and Distal) and/or Jejunum | Loss of folds<br><br>+ Fissures<br><br>+ Nodular appearance/scalloping folds<br><br>↑ intraepithelial lymphocytes<br><br>↑ crypt hyperplasia<br><br>+ villous atrophy | On its own, not diagnostic of CeD. Must be combined with other criteria.<br><br>Marsh 0-1 indicates adequate positive response to GFD if compared to previous pathology reports on gluten-containing diet that were Marsh 2-4. |
| | Marsh score 1<br><br>Marsh 2<br><br>Marsh 3a-b<br><br>Marsh 4 | Marsh 2-4 indicative of duodenal enteritis, which could be from CeD or other causes. When combined with positive serum antibodies and genetics for celiac disease, then diagnostic of CeD. |
| **Micronutrients**<br><br>Vit A, D, E, K<br><br>Vit B6, B9, B12<br><br>Ferritin, Copper, Zinc | ↓ in CeD | Typically normal in non-celiac gluten sensitivity |
| DEXA | ↓ bone density | Likely to normalize after 1 year on GFD without supplementation or medication |

## Table 7.4. Tests and Studies Related to Constipation

| Test or Study | Abnormal Finding | Interpretation |
|---|---|---|
| KUB | "Mild/moderate/severe stool burden" visualized | Constipation |
| Digital Rectal Exam | ↑ resting anal sphincter pressure, ↓ squeeze strength and/or | Dyssynergic defecation |

| | | |
|---|---|---|
| | endurance, ↓ relaxation or paradoxical contraction of the anal sphincters with simulated defecation | |
| Anorectal Manometry to Assess for Dyssynergic Defecation and Other Outlet Disorders | ↑ resting anal sphincter pressure, ↓ squeeze strength and/or endurance, ↓ relaxation or paradoxical contraction of the anal sphincters with simulated defecation | Dyssynergic defecation |
| Balloon Expulsion Testing (Usually Done with Anorectal Manometry) | Inability to expel balloon filled with 50 cc of water from the rectum within 60 seconds | Dyssynergic defecation |
| Defecography (Barium or MRI) | Inability to evacuate or ≥ 50% retention of barium during defecography and/or straightening of the anorectal angle of < 20 degrees during simulated defecation | Dyssynergic defecation |
| Sitzmarker Study | > 5 of 24 markers remain on day 5 x-ray | Slow transit constipation, if distributed throughout the colon<br><br>Outlet/pelvic floor dysfunction constipation, if distributed in the rectosigmoid (left/distal) colon |
| Nuclear Scintigraphy<br><br>*Rarely performed due to lack of testing sites, may be performed with whole gut transit time* | ↓ rate of passage of isotope. | Slow transit constipation |
| Colonic Manometry<br><br>*Rarely performed due to lack of testing sites, prolonged procedure time, and burden to patients* | ↓ frequency or strength of contractions, non-propulsive/uncoordinated contractions, ↑ anorectal tone and incomplete relaxation of the anorectum | Slow transit or outlet/pelvic floor dysfunction constipation |

## Table 7.5. Tests and Studies Related to Diarrhea[2,3]

| Test or Study | Abnormal Finding | Interpretation |
|---|---|---|
| Tissue Transglutaminase Antibody IgA, total IgA | ↑ | Rule out CeD |
| *C. Difficile*, Giardia | Positive | Rule out fecal infection |
| Fecal Calprotectin, Fecal Lactoferrin | ↑ | Rule out IBD |
| Fecal Fat, 24-hour Stool Study | ↑ | Fat malabsorption, pancreatic insufficiency, bile acid malabsorption |
| | | |

| | | |
|---|---|---|
| Fecal Elastase & Coefficient of Fat Absorption (CFA) | ↓ | Rule out pancreatic insufficiency |
| Breath Tests: Lactose, CSID, SIBO, Fructose | ↑ | Rule out carbohydrate intolerance & SIBO |
| Liver Functioning Tests | ↑ | Rule out excessive alcohol intake |
| HgbA1c, Fasting BG | ↑ | Rule out hyperglycemia |
| Thyroid Function Panel: TSH | ↓ | Rule out hyperthyroidism |
| CBC, Iron Panel, Serum Ferritin | ↓ | Rule out anemia and iron deficiency |
| Vitamin B12 | ↓ | Loose stools may indicate a vitamin B12 deficiency |
| Folate | ↓ | Loose stools may indicate a folate deficiency |
| Fat-soluble Vitamins, Serum Retinol | ↓ | Steatorrhea, malnutrition, vitamin A deficiency |

## Table 7.6. Tests and Studies Related to Dyspepsia

| Test or Study | Abnormal Finding | Interpretation |
|---|---|---|
| *H. Pylori* Testing (Stool Antigen, Urea Breath Test, or Endoscopic Biopsies for Histology)<br><br>*IgG antibody testing will demonstrate if patient was ever infected but will not tell if infection has cleared* | Positive | *H. pylori* infection (resolution after treatment indicates eradication). |
| Upper Endoscopy | Inflammation (e.g., esophagitis, gastritis), ulcer (e.g., peptic ulcer disease) | Functional dyspepsia, alternative explanations with structural causes, *H. pylori* infection (based on biopsy) |

## Table 7.7. Tests and Studies Related to Gastroparesis

| Test or Study | Abnormal Finding | Interpretation |
|---|---|---|
| HgbA1c | Prediabetes: 5.7-6.4%<br><br>Diabetes: ≥6.5% | Diabetes mellitus is a risk factor for gastroparesis |
| ANNA-1 or Anti-Hu | ↑ | A rare cause of malignancy (e.g., small cell lung cancer) triggering development of anti-neuronal antibodies leading to destruction of the enteric nerves and gastroparesis |
| Barium Upper GI Series | ↓ | Used to noninvasively assess for gastric outlet obstruction |

| | | |
|---|---|---|
| Gastric Emptying Study | ≥10% of a test meal remaining in the stomach at 4 hours after ingestion (or more than 60% at 2 hours) | Abnormally slow gastric emptying due to gastroparesis, opioid use, GLP-1 agonists, malnutrition |
| $^{13}$C-Spirulina Gastric Emptying Breath Test | Positive | Gastroparesis |
| Upper Endoscopy | Structural abnormalities, residual food | Used to rule out gastric cancer or gastric outlet obstruction. Food found incidentally in the stomach after a prolonged period of fasting should prompt more work-up for gastroparesis |

## Table 7.8. Tests and Studies Related to Gastroesophageal Reflux Disease

| Test or Study | Abnormal Finding | Interpretation |
|---|---|---|
| Barium Esophagram | Reflux and/or strictures | Possible need to dilate in response to strictures |
| Upper Endoscopy | Esophagitis, precancerous cellular changes, peptic strictures, inflammation | Reflux esophagitis, Barrett's esophagus, eosinophilic esophagitis |
| Esophageal Manometry | Dysmotility | Esophageal motility disorder |
| Ambulatory Esophageal pH Monitoring (Catheter or Implanted) | ↑ acid exposure time (AET)<br>Inconclusive: 4.0%-6.0%<br>Abnormal: > 6.0% | GERD, functional heartburn |

## Table 7.9. Tests and Studies Related to Inflammatory Bowel Disease

| Test | Abnormal finding | Interpretations |
|---|---|---|
| *C. Difficile*, Giardia | Positive | Rule out fecal infection |
| Fecal Calprotectin, Fecal Lactoferrin | ↑ | Active IBD (less sensitive in checking for proctitis or small bowel inflammation compared to left-sided or pancolitis)<br>AGA Guidelines recommend checking in clinical remission every 6-12 months |
| C-Reactive Protein (CRP), Erythrocyte Sedimentation Rate (ESR) | ↑ | Active IBD<br>~15% of people do not mount response during active IBD inflammation |

## Table 7.10. Tests and Studies Related to Lactose Intolerance

| Test or Study | Abnormal Finding | Interpretation |
|---|---|---|
| Hydrogen Breath Testing | Positive | Lactose intolerance |

| | | |
|---|---|---|
| (Using Lactose as Substrate) | | |
| Glucose Tolerance Test (Using Lactose as Substrate) | Failure of blood glucose to rise 2 hours after ingestion of lactose | Lactose intolerance |
| Duodenal Biopsy for Disaccharidases | Low lactase | Lactose intolerance |

## Table 7.11. Tests and Studies Related to Metabolic-Associated Steatotic Liver Disease

| Test or Study | Abnormal Finding | Interpretation |
|---|---|---|
| Liver Biopsy<br>*Not often used due to invasive nature* | Fat deposits, fibrosis, inflammation | Used to determine etiology of abnormal liver labs and/or imaging and the presence of fibrosis/cirrhosis in patients with abnormal liver labs, MASLD, MASH |
| Liver Ultrasound<br>*Visualization may be less accurate in higher-weight individuals* | Enlargement, steatosis, nodular contour, evidence of portal hypertension, screening for liver cancer in patients with cirrhosis | MASLD, MASH |
| MRI or CT Scan | Enlargement, steatosis, bile duct obstructions | Steatosis, liver lesions (e.g., nodules, cysts), liver/bile duct problems |
| Liver Fibrosis Prediction Calculations (Fatty Liver Index, Steato Test, NAFLD Liver Score, FIB-4) | High risk score | Estimates hepatic fibrosis using a variety of metrics such as BMI, aminotransferases, platelets, etc. |
| Transient Elastography (Fibroscan) | Fibrosis | Noninvasive test to determine degree of steatosis and likelihood of advanced fibrosis/cirrhosis in MASLD/MASH |
| Enhanced Liver Fibrosis (ELF) Test | Low risk of progression: <9.8<br>Mid risk: 9.8-11.29<br>High risk: >11.29 | Estimates risk of progression of MASLD/MASH to cirrhosis |
| Liver Tests (AST, ALT, Bilirubin, Alkaline Phosphatase) | ↑ | Hepatocellular injury (elevated AST, ALT), cholestatic/bile duct injury (elevated bilirubin, alkaline phosphatase) |
| CBC | Thrombocytopenia | Many causes, nonspecific, can suggest advanced fibrosis/cirrhosis and portal hypertension |

## Table 7.12. Tests and Studies Related to Small Intestinal Bacterial Overgrowth & Intestinal Methanogenic Overgrowth

| Test or Study | Abnormal Finding | Interpretation |
|---|---|---|
| Breath Testing | Rise in hydrogen concentrations of ≥20 ppm from baseline within 90 minutes<br><br>Methane levels ≥10 ppm at any point before 90 minutes | SIBO, hypermotility of GI tract |

**Clinical Call-Out**

There are several additional tests that patients may have questions about, including but not limited to: Immunoglobulin-G (IgG) food sensitivity testing, LEAP/MRT (mediator release assay), applied kinesiology muscle testing, hair analysis, or at-home stool and microbiome studies (e.g., GI MAP). At this time, there is inadequate scientific evidence to support the use of these studies for clinical assessment of gastrointestinal conditions, and they are not recommended[4,5].

## Anthropometric Data

### *Body Mass Index (BMI)*

The concept of BMI was originally developed in Western Europe as a statistical tool to evaluate population-wide height and weight averages among a male Caucasian demographic. Today, it is widely used as a proxy for health status, though its clinical relevancy is severely limited. BMI fails to account for gender, age, race, musculature, frame size, cardiometabolic parameters, and a host of other factors, all of which are relevant when assessing health status. Further, BMI categorizations can lead to stigma, shame, anxiety and maladaptive behaviors, which may result in avoidance of care, misdiagnosis, and clinical focus on weight rather than more effective treatment[6].

### *Weight History and Weight Changes*

Weight should be interpreted within an individual context. Learning about a client's weight history may offer insight into their biologically appropriate weight range, or the span of weight that supports optimal functioning. A discussion of weight history can also be an opportunity to identify intentional weight control behaviors that have been or are currently being used.

Weight changes can provide clinical details relevant to both EDs and GI disorders, so clinicians are encouraged to consider a wide array of causes. For example, weight gain may be attributed to steroid medications, water loading/retention, bingeing behaviors, or improved nutritional parameters[7,8]. Weight loss may suggest the onset of restrictive eating patterns, malabsorption, or other severe GI disease. In these cases, it is important to consider the possible role of malnutrition for all clients, as malnutrition can occur at any weight.

### *Growth Charts*

Pediatric growth charts are expressed as percentiles and individuals generally track along a particular curve. Weight loss in children and adolescents runs counter to their predicted growth and development and is always cause for further investigation. Clinicians should take note of significant weight/percentile changes in either direction and are recommended to frequently assess for possible malnutrition[9]. A weight-inclusive approach would use percent of weight loss to identify malnutrition, rather than weight or BMI alone. When weight restoration is warranted, a biologically appropriate weight range should be determined based on pre-illness height and weight trajectories, as well as pubertal onset and current pubertal stage[10].

## Gastrointestinal Symptoms

Gastrointestinal symptoms can vary in type, frequency, and root cause. The RDN should ask the patient about current and past gastrointestinal symptoms and consider the patient's perceived food triggers, history of symptom management, previous dietary interventions tried, and quality of life issues associated with their symptoms.

| Alarm Features |
| --- |
| "Alarm" or "red flag" features refer to symptoms or other details that suggest higher acuity and require appropriate investigation by a gastroenterologist. These clients may be appropriate for nutrition counseling after organic disease has been identified or ruled out. The RDN should refer to a gastroenterologist immediately when confronted with any of the following:<br>• Unintentional weight loss<br>• Family history of bowel diseases or cancer |

- GI bleeding (or black, tarry stool indicative of bowel bleeding)
- Iron-deficiency anemia
- Unexplained fever
- Recurrent vomiting
- Persistent daily diarrhea
- Nocturnal bowel movements
- Symptoms worsening over time

## Functional GI Symptoms

Determining the most effective GI intervention is dependent on a global understanding of the client's symptoms. There are many potential causes shared by various diseases. For example, bloating can be due to IBS, IBD, SIBO, and others. Disease-specific recommendations for navigating these symptoms are provided in the Nutrition Intervention section. The following table offers clinical suggestions for navigating symptoms in the absence of a diagnosis. In order to properly investigate GI concerns, it's important that clients have been screened for certain conditions and other conditions have been ruled out. The RDN should relay diagnostically relevant information to appropriate providers (e.g., informing the gastroenterologist that the patient is having symptoms in response to high-fat meals).

**Table 7.13. Inquiring About Gastrointestinal Symptoms**

| Symptoms |
|---|
| **Appetite Changes** |
| *Has your appetite changed, and if so, is the change related to disease onset, symptoms, medications, diet history, etc.?*<br>*Do you get full before finishing your meals?*<br>*Do you ever feel that you cannot get full enough?* |
| **Reflux** |
| *When does your reflux feel most intense?*<br>*Does your reflux improve after eating or throughout the day?*<br>*Does your reflux seem to be exacerbated when you're feeling constipated?*<br>*Do you have symptoms of hoarse voice, cough, dental erosion, or metallic taste in the mouth?* |
| **Nausea & Vomiting** |
| *When does your nausea feel most intense?*<br>*Does your nausea seem to be exacerbated by certain meals or foods? Which ones?*<br>*Does your nausea seem to be exacerbated when you're feeling constipated?*<br>*Is your nausea impacted by sleep quality, smells or sights?*<br>*Is your vomiting spontaneous or self-induced in an effort to relieve the nausea?* |

| |
|---|
| *Do you vomit a small or large quantity?*<br>*Is there anything that helps relieve your nausea or vomiting once it has started?*<br>*Do you regurgitate food without a feeling of nausea or the action of retching? Do you chew, swallow, or spit it out?* |
| **GI Pain** |
| *Where does your pain occur? (Clarify above/below the navel, which side(s), fixed location vs migrating pain, etc.)*<br>*When is abdominal pain likely to occur? When does your pain feel most intense?*<br>*Is the pain alleviated by passing stool or gas?*<br>*Does your abdominal pain seem to be exacerbated by certain meals or foods? Which ones?* |
| **Gas (Flatulence, Eructation)** |
| *Do you experience "farting" gas, "burping" gas, or both?*<br>Flatus: 10-20 times per day is considered "normal"[11]<br>Belching: Up to 30 times per day is considered "normal"[12]<br>*Does your flatulence smell particularly bad (malodorous)?*<br>*Can you control passing gas?*<br>*Does your gas seem to be exacerbated by certain meals or foods? Which ones?* |
| **Bloating** |
| *Where does your bloating occur?*<br>*When does your bloating feel most intense?*<br>*Does your bloating seem to be exacerbated by certain meals or foods?*<br>*Does your bloating seem to be exacerbated when you're feeling constipated or alleviated by passing gas or a bowel movement?* |
| **Diarrhea** |
| *Do you have diarrhea/loose stools, defined as Bristol Stool Scale 5-7?*<br>*Do you feel a sense of urgency?*<br>*Do you experience cramping?*<br>*Do you have the urge to poop shortly after passing a bowel movement?*<br>*Do you have the urge to poop shortly after eating?*<br>*Is your stool an unusual color (e.g., pale or orange, black, etc.)? Does it have an oily/greasy appearance?*<br>*Do your loose stools seem to be exacerbated by certain meals or foods?*<br>*Do you ever experience incontinence or "accidents" with bowel movements?* |
| **Constipation** |
| *How often are you having a bowel movement?*<br>*What number(s) on the Bristol Stool Scale (BSS) does your stool most resemble? (Client might indicate a range)*<br>*How often do you strain while having a bowel movement? Do you find that you have to strain even when the stool is soft?*<br>*Do you ever feel your bowel movement is incomplete?*<br>*Do you ever use a finger or other object to remove stool or "splint" the perineum/vagina to pass stool?*<br>*Do you constantly feel the urge to have a bowel movement?*<br>*How long does it take for you to evacuate your bowel movements?* |

# Disordered Eating Behaviors and Related History

The Registered Dietitian should seek to understand the patient's current use of potentially disordered eating behaviors (including frequency and/or intensity) and their history. Seemingly disordered eating behaviors may be attributable to comorbid conditions and should be discussed with other healthcare providers, as the resulting collaborative information may help guide effective interventions for improving or resolving GI concerns. Specifically, the Registered Dietitian should inquire about:

**Table 7.14. Inquiring About Disordered Eating Behaviors**

| Behavior | Suggested Language | Implications |
|---|---|---|
| **Restriction**<br>*Purposeful underfueling or limiting intake to control weight/shape and/or manage anxiety; may include pursuing inappropriate calorie/macronutrient targets, use of diet plans, food avoidance/elimination (with or without intention of managing a medical condition), adherence to "food rules"* | • Do you intentionally limit your intake out of concern for weight or body shape?<br>• In your experience, how linked are intake and anxiety?<br>• How do you determine what or how much to eat?<br>• How difficult would it be to eat more on a daily basis, if that was recommended?<br>• What kinds of foods (if any) do you avoid? For what reason? Where did you learn about this approach?<br>• Do you have any rules that guide your eating? How flexible are you with these rules?<br>• Do you knowingly skip taking your medication? | • Relative energy deficiency<br>• Malnutrition<br>• Micronutrient deficiencies<br>• Sarcopenia<br>• Possible impact on gastrocolic reflex, transit time<br>• Possible body image distress, willingness to table weight concerns in favor of addressing GI symptoms<br>• Patient's history interacting with the medical system vs. tendency for self-diagnosis<br>• Cognitive rigidity<br>• Quality of life |
| **Bingeing**<br>*Episodes characterized by a sense of loss of control, eating rapidly, eating in the absence of hunger, and/or eating until extremely full; generally associated with* | • Do you ever feel out of control with regard to food?<br>• How often do you find yourself consuming a remarkably large amount of food in a short period of time? | • Possibility of "subjective binges" (food consumption that feels excessive and/or shameful to the patient but is clinically unremarkable in amount)<br>• Possible impact on bowel regularity<br>• Possible impact on intake later on/the following day |

| | | |
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| *depression, shame, or guilt*[13]. | • What types of foods do you gravitate toward in those situations?<br>• How do you feel afterwards?<br>• Is secrecy part of this kind of eating behavior? | |
| **Compensatory Behaviors**<br>*Actions meant to counteract intake; may include self-induced vomiting, laxative misuse, diuretic misuse, excessive exercise, colonic cleanses, diet pills, insulin adjustments, inappropriate use of other medications, chewing and spitting, etc.* | • What do you do when you feel that you have eaten "too much" or the "wrong" kinds of foods?<br>• How often do you take those kinds of measures?<br>• Do you find it distressing to feel the physical sensations of fullness and/or digestion? | • Medical risk (i.e., electrolyte disturbances, cardiac events, fainting/collapse, esophageal damage or rupture, damage to tooth enamel, diabetic neuropathy/retinopathy/nephropathy, etc.)<br>• Level of distress surrounding normal physiological processes |
| **Excessive Physical Activity***<br>*Physical activity that surpasses the individual's ability to fuel or otherwise reduces quality of life*<br>**also a compensatory behavior* | • What is your motivation for moving your body?<br>• Does your physical activity feel depleting or repleting?<br>• Do you alter your social plans to ensure that physical activity occurs?<br>• How does your eating change depending on your physical activity?<br>• Conversely, how does your physical activity change depending on what you eat? | • Low energy availability<br>• Cognitive rigidity<br>• Compensatory urges<br>• Possible downstream effects on GI symptoms<br>• Risk of injury |
| **Rumination**<br>*Repeated regurgitation of food not attributable to an associated gastrointestinal or other medical condition*[14] | • Do you ever find your food coming back up after you've swallowed it?<br>• In your experience, is there any relationship between regurgitation and feeling anxious about what you've eaten? | • Self-awareness of co-occurring anxiety, if present<br>• Rule out GI or other medical condition |

| | | |
|---|---|---|
| | • Have you had this pattern evaluated by a specialist? | |
| **Pica**<br>*Ingestion of nonnutritive substances*[14] | • Do you ever crave, taste, chew, or swallow non-food items? Common examples include ice, dirt, clay, cleaning supplies, drywall, hair, small objects, etc.<br>• Do these events coincide with any changes in your GI symptoms?<br>• What vitamins/supplements are you currently taking?<br>• Have you been evaluated for possible nutrient deficiencies? | • Possible nutrient deficiency<br>• Possible sensory processing difficulties, neurodivergence, developmental delay |
| **Mealtime Rituals**<br>*Behaviors to manipulate energy intake (e.g., spreading out fat/sauces/cheese to appear as if they had been eaten, hiding food, chewing and spitting)*<br>*Behaviors to dilute/suppress appetite (e.g., excessive intake of fluids with meals, excessive intake of water/caffeinated beverages, and artificial sweeteners)*<br>*Behaviors indicative of aversions (e.g., limiting foods based on color, temperature, food group, or other characteristics)*<br>*Behaviors that make socializing difficult (e.g., excessive cutting, mixing, or separating foods; comparing intake with others; eating in solitude)* | • Are there any behaviors you take part in to influence how others view your eating?<br>• Are there any behaviors you take part in to suppress your appetite or not feel your hunger?<br>• Do you notice any sensory difficulties with certain foods?<br>• To what degree do your eating habits impact your ability to eat socially with others?<br>• Do you ever feel like your meals/snacks need to happen in a certain way or proceed in specific order? | • Cognitive rigidity<br>• Possible comorbid conditions (e.g., OCD)<br>• Possible sensory processing difficulties or neurodivergence |

| | | |
|---|---|---|
| *Behaviors indicative of obsessive-compulsive thinking (e.g., eating with rigid order, timing, or counting)* | | |
| **Body Checking**<br>*Assessing for changes in body weight/shape/composition to manage anxiety; may include studying self in mirror/reflective surfaces, body pinching, self-weighing, checking the fit of clothing, measuring waist or wrist circumference, etc.* | • What do you do when you feel as though your weight or body may have changed?<br>• How often do you perform this "check"?<br>• How do you feel after you've "checked" on your body in this way?<br>• How do you feel if you aren't able to perform this "check"? | • Cognitive rigidity<br>• Possible comorbid conditions (e.g., OCD) |
| **Previously Seeking Treatment**<br>*Previous encounters with providers and/or programs meant to address medical and/or mental health concerns* | • What kind of providers have you talked to about this?<br>• What previous recommendations and treatment options have you been given?<br>• What was your experience like?<br>• What made that approach helpful/unhelpful? | • What has or hasn't worked<br>• Attachment style<br>• Stage of change<br>• Possibility of past medical neglect/trauma<br>• Possibility of misinformation |

Within each of the above categories, the Registered Dietitian may consider asking about the behavior's history. Such questions may include:

- *When and how did these behaviors develop?*
- *Does the client understand the function of the behaviors?*
- *Are the underlying stressors that fuel the behaviors still present in some form?*
- *What (if any) medical complications has the client experienced as a result of these behaviors?*
- *What (if any) treatment or support has the client received for these issues?*

## Food and Nutrition-Related History

The Registered Dietitian should assess food intake, nutrient intake, and use of supplements (including herbals, probiotics, and over-the-counter supplements), as well as the patient's intake and attitudes toward food. This information may highlight specific foods or dietary patterns that exacerbate GI symptoms, as well as potential barriers to implementing the Registered Dietitian's medical nutrition therapy recommendations. Points of inquiry should include:

- 24-hour dietary recall and/or usual intake. *Include timing of both intake and GI symptoms to assess whether the two are related.*
- Past/current eating patterns. *Assess for caloric adequacy (regardless of ED diagnosis), nutrient gaps, chaotic eating, food preferences, cultural foods, prescribed or self-administered diets, etc.*
- Allergies, avoided foods, "safe" foods. *Assess for medical necessity/appropriateness of avoided foods.*
- Nutrition-related beliefs and attitudes. *This may include culture, family influence, sources of nutrition information, and observable food anxiety.*

The way nutrition assessment questions are asked has the potential to be triggering for someone with an ED. Some examples of less-triggering language are below:

**Table 7.15. Rephrasing Questions for Eating Disorder Clients**

| Questions or comments that might be triggering to someone with an ED | A better way to communicate your message |
|---|---|
| "Have you gained (or lost) weight recently?" | "Have you noticed any changes in the way your clothes fit?" |
| "Wow, that's a large portion of food," or "You have a really big appetite!" | "How do you feel after you consume that meal? Do you experience any unpleasant symptoms afterwards?" |
| "I'm surprised to hear that you want to lose weight. You look so healthy!" or "Do you have a target weight?" | "Tell me more about your motivations for this desire to lose weight." |
| "That food isn't triggering your symptoms. You don't have an intolerance to it." | "Tell me more about why you avoid that food. How do you feel when you consume it?" |
| "It looks like you have a lot of self-control." | "It looks like you're very detail-oriented when it |

| | |
|---|---|
| | comes to food and nutrition. How does that impact your life?" |

Using affirming, empathetic language in your conversations with clients with co-occurring GI conditions and eating disorders validates their experience and can foster an environment where the individual will feel more comfortable working with you. Examples of empathetic language include:

- "That sounds really hard."
- "I hear you."
- "Thank you for sharing that with me."
- "I believe you."

## Other Factors Affecting Quality of Life

A comprehensive patient history will consider a variety of factors that influence quality of life. In addition to reviewing past medical history, disordered eating history, gastrointestinal symptoms, usual intake and food-related beliefs and attitudes, the Registered Dietitian may be able assess other related domains and connect the patient to needed resources. The Registered Dietitian may consider asking about the following:

- Food security. *"Within the last 12 months, I worried whether my food would run out before I got money to buy more. Within the last 12 months, the food I bought just didn't last, and I didn't have money to get more." Yes, or no?*[15]
- Stress management
- Sleep hygiene
- Access to medical, mental, and/or gender-affirming healthcare. *Are essential members of the healthcare team in place?*
- Family/social support

## Nutrition-Focused Physical Exam

The Nutrition-Focused Physical Exam (NFPE) is an opportunity for the Registered Dietitian to use sight and touch to evaluate the patient for malnutrition, disruptions in fluid status, and/or nutrient deficiencies. Considerations for each are described below. The Registered Dietitian should

use their discretion as to which parts of a NFPE are appropriate for patients; for example, skin-fold measurements, open weights, and palpating the abdomen may add no significant benefit while harming the patient.

## Malnutrition

Malnutrition can be evaluated by considering energy intake, weight loss, subcutaneous fat loss, muscle mass, fluid accumulation, and/or grip strength (see Table 7.16).

**Table 7.16. Joint AND/ASPEN Malnutrition Criteria**

| | Chronic Illness | | Acute Illness | |
|---|---|---|---|---|
| **Indicator** | **Moderate** | **Severe** | **Moderate** | **Severe** |
| Energy Intake | <75% energy needs for >7 days | ≤50% energy needs for ≥5 days | <75% energy needs for ≥1 month | ≤75% energy needs for ≥1 month |
| Weight Loss | 1-2% 1 week<br>5% 1 month<br>7.5% 3 months | >2% 1 week<br>>5% 1 month<br>>7.5% 3 months | 5% 1 month<br>7.5% 3 months<br>10% 6 months<br>20% 1 year | >5% 1 month<br>>7.5% 3 months<br>>10% 6 months<br>>20% 1 year |
| Loss of Subcutaneous Fat | Mild | Moderate | Mild | Severe |
| Loss of Muscle Mass | Mild | Moderate | Mild | Severe |
| Fluid Accumulation | Mild | Moderate-Severe | Mild | Severe |
| Grip Strength | N/A | Measurably Reduced | N/A | Measurably Reduced |

*Adapted from White et al., 2012*[16]

Muscle wasting or fat loss may appear less pronounced in a larger-bodied individual and reinforce the stereotype that eating disorders only occur in thin or emaciated people. The Registered Dietitian is discouraged from using this assessment to rule out the presence of disordered eating concerns.

The joint AND-ASPEN malnutrition criteria is based on data that may or may not be available to the Registered Dietitian, and it may not capture all clients who present with malnutrition. For additional malnutrition screening tools, consider use of the following:

- Global Leadership Initiative on Malnutrition (GLIM)

- Malnutrition Universal Screening Tool (MUST)
- Subjective Global Assessment (SGA)
- Saskatchewan Inflammatory Bowel Disease Nutrition Risk (SaskIBD-NR)
- Strength, Assistance walking, Rising from a chair, Climbing stairs and Falls (Sarc-F) screening tool

## Nutrient Deficiencies

The Registered Dietitian can look for signs of nutritional deficiencies by evaluating areas such as the hair, skin, nails, eyes, and mouth. If possible, suspected deficiencies should be confirmed via laboratory findings and treated accordingly.

## Other Relevant Assessments

The Registered Dietitian may also consider abnormal resting heart rate (<60bpm) and/or orthostatic vitals in assessing medical stability. Orthostatic hypotension is considered a change in blood pressure of ≥20mmHg systolic and/or ≥10mmHg diastolic when changing positions from laying down to sitting, or from sitting to standing. This change in blood pressure may also be accompanied by a change in heart rate of ≥20bpm, which signals cardiac decompensation.

In addition to appropriate clinical training, it should be noted that the NFPE requires an understanding of trauma-informed care. Due to the high prevalence of trauma within the eating disorders population, it's imperative that the Registered Dietitian explains what they intend to do, ask for consent to proceed, and check in with the patient's comfort level throughout each procedure.

In-person NFPEs may become less common as the practice of telehealth expands. Virtual NFPEs may require adaptations that are summarized in Table 7.17.

**Table 7.17. Nutrition-Focused Physical Examination (NFPE) Adaptations for Telehealth**

| NFPE Component | Clinician Assessment Actions | Questions to Ask Patient |
|---|---|---|

| | | |
|---|---|---|
| Functional status | • Observe posture/position/physique<br>• Notice if patient is sitting/lying in bed, if able to maintain posture | • Do you notice any signs of weakness?<br>• Has your activity level changed? If so, how?<br>• Ask about activities of daily living/time spent on activities |
| Fat and muscle wasting | • Notice visible losses of fat or muscle<br>• Guide patient to show each area of body<br>• Explain positioning<br>• Consider asking for photos for monitoring and follow-up evaluation | • Have you noticed any changes in weight?<br>• Do your clothes, belt, jewelry, glasses, dentures, etc. fit differently? |
| Fluid status | • Teach patient how to assess quality of edema<br>• Teach patient skin turgor assessment (back of hand, forearm, sternum) | • Have you noticed any increase in puffiness/fluid accumulation in your ankles/around your feet?<br>• How far up your leg does swelling occur?<br>• Is there indentation when you press on your skin? How long does the indentation last?<br>• Pinch skin; after letting go, does the skin return to its original position? How long does it take? |
| Micronutrients: skin | • Observe facial skin, notice any discoloration, rashes, or poor pallor | • Have you noticed any rashes or changes in your skin, areas of flaky skin, redness, wounds? |
| Micronutrients: hair | • Observe hair; ask to see arm hair | • Have you noticed any difference in your hair?<br>• Has your hair been falling out more? Are you waking up with hair on your pillow?<br>• Does arm hair look like a corkscrew? |
| Micronutrients: nails | • Ask to see nails up close on camera<br>• Ask patient to push on nail bed to demonstrate blanching | • Have you noticed any difference in your nails?<br>• Are your nails breaking more easily? |
| Micronutrients: eyes | • Observe eyes<br>• Ask patient to lightly pull down on bottom eyelid to | • Have you noticed any changes in eye health or color? |

| | | |
|---|---|---|
| | assess conjunctiva color | • Have you had any trouble seeing in general or seeing at night?<br>• Are you experiencing dry, stinging, or burning eyes? |
| Micronutrients: mouth | • Observe mouth and lips<br>• Ask to see tongue, inner bottom lip, and gums | • Have you noticed any sores in your mouth or changes with your teeth? Any trouble with bleeding gums?<br>• Are you having difficulty chewing/swallowing?<br>• Any taste changes? |

*Adapted from: Mauldin 2021*

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CHAPTER 8

# Nutrition-Related Diagnosis

Nutrition-related diagnoses identify problems that can be treated within the scope of practice of the RDN. They are written in a standardized format called a "PES statement." The PES statement outlines the nutrition-related problem (P), related to a likely etiology (E), as evidenced by supporting signs and symptoms (S).

PES statements can help justify the RDN's role in providing medical nutrition therapy, although they are not required documentation in most settings. The concept of nutrition-related diagnosis is distinct from medical and psychological diagnoses.

## Problem (P)

A nutrition problem is one that can be resolved or improved through medical nutrition therapy. Nutrition problems fall into Intake, Clinical, or Behavioral-Environmental domains.

## Etiology (E)

The etiology is the likely root cause of the nutrition problem and is identified so that the nutrition intervention can be appropriately tailored. Etiology domains include: Belief-Attitudes, Physiologic-Metabolic, Psychological, Cultural, Knowledge Deficits, Access, Physical Function, Social-Personal, Treatment, and Behavior.

## Signs and Symptoms (S)

Reported signs and symptoms, as well as other clinical information, are documented to support the existence of the nutrition problem and its etiology.

For those who find PES statements useful, we outline several templates that we have found useful through years of clinical experience with the ED-GI patient population:

*Disordered eating related to* [ED diagnosis or preoccupation with food and weight] *as evidenced by* [patient report, staff reports, nutrition-focused physical examination, laboratory findings/test results, etc.]

*Altered GI function related to* [diagnosis or hypothesis] *as evidenced by* [patient report, staff reports, nutrition-focused physical examination, laboratory findings/test results, etc.]

*Altered nutrition-related labs related to* [diagnosis or hypothesis] *as evidenced by* [patient report, staff reports, nutrition-focused physical examination, laboratory findings/test results, etc.]

Detailed examples are included in Table 8.1 below. For a more comprehensive resource, please see the Nutrition Care Process Terminology[1].

**Table 8.1. Examples of PES Components Relevant to the EDGI Population**

| Problem: Intake | Etiology | Signs & Symptoms |
|---|---|---|
| Inadequate protein-energy intake | related to fear of weight gain (*Belief-Attitude*) | as evidenced by current weight 80% of usual weight, estimated intake less than 75% of estimated needs, and client reports of restricting calorie intake to prevent weight gain |

| | | |
|---|---|---|
| Inconsistent timing of nutrient intake | related to inability to change ostomy bag independently (*Physical Function*) | as evidenced by client reports of avoiding food intake until caregiver is present |
| Disordered eating pattern | related to anorexia nervosa-restricting type (*Psychological*) | as evidenced by client reports of limiting intake to 800 kcal/day and fear of weight gain |
| **Problem: Clinical** | **Etiology** | **Sign & Symptoms** |
| Altered GI function | related to suspected gastroparesis (*Physiologic*) | as evidenced by recurrent vomiting, nausea, early satiety, postprandial distress, and inadequate kcal intake |
| Altered GI function | related to diarrhea predominant-irritable bowel syndrome (*Physiologic*) | as evidenced by reported diarrhea up to 8x/day accompanied by urgency, bloating, and abdominal pain |
| Altered nutrition-related labs | related to diagnosis of SIBO (*Physiologic*) | as evidenced by positive lactulose breath test and vitamin B12 level of 200 |
| Predicted suboptimal intake | related to planned j-pouch surgery (*Treatment*) | as evidenced by expected hospital stay of 5-7 days with 2-4 days of liquid diet and 24 hours fasting |
| Altered nutrient needs | related to fat malabsorption (*Physiologic*) | as evidenced by unintentional weight loss of 10 lbs over 4 weeks coinciding with reports of steatorrhea |
| Severe malnutrition in the context of chronic illness | related to active Crohn's disease (*Physiologic*) | as evidenced by weight loss >10% over 6 months, severe orbital fat loss, and severe temporalis muscle wasting |
| **Problem: Behavioral-Environmental** | **Etiology** | **Sign & Symptoms** |
| Limited adherence to nutrition recommendations | related to lack of adequate education on gluten-free diet (*Knowledge Deficit*) | as evidenced by client reports of consuming oats not labeled GF approximately 5x/week and |

| | | |
|---|---|---|
| | | elevated tissue transglutaminase level |
| Harmful beliefs/attitudes about food | related to familial pressure to treat diagnosis without medication (*Social-Personal*) | as evidenced by client reports of following "carnivore diet" and eliminating all plant foods for 3 months, elevated LDL-chol, and imaging demonstrating progression of liver fibrosis |
| Inconsistent timing of food intake | related to limited finances for food (*Access*) | as evidenced by intake of one large meal daily and client stating their SNAP benefits usually run out before the end of the month |

*Adapted from Nutrition Diagnosis Etiology Matrix 2013*[2].

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CHAPTER 9

# Nutrition Intervention

The goal of Nutrition Intervention is to address, improve, or resolve the nutrition-related problem by targeting the etiology and/or signs and symptoms originally identified. In the AND Nutrition Care Process, interventions are categorized as: food and nutrient delivery, nutrition education, nutrition counseling, and coordination of nutrition care. For our purposes, we have divided interventions into "Diet" and "Non-Diet" Interventions, to remind practitioners that treatment interventions include more than dietary modification and restriction. The following considerations should guide the Nutrition Intervention:

- Center the client's goals
- Prioritize the nutrition diagnoses
- Utilize relevant practice guidelines, as available and appropriate
- Set goals and design treatment plan collaboratively with the client
- Communicate the plan to other providers

The interventions in this chapter are based on current standards of practice, evidence-based research and, when robust evidence is lacking, expert clinical experience. There is no universal way to

treat these conditions; treatment recommendations should be tailored to a client's individual needs.

## General Eating Disorder- and GI-Informed Interventions

The goal of EDGI Nutrition Intervention is to support and encourage recovery from the eating disorder, including psychological, behavioral, and physical symptoms, keeping in mind the distinct needs of those who experience gastrointestinal symptoms and disorders. Treating the eating disorder may involve explicitly challenging or contradicting health behaviors that are considered common practice by the current medical establishment in order to promote an individual's overall and long-term wellbeing. In our collective experience, an eating disorder is a greater threat to one's health than any particular food, outside of very few medical conditions. When we treat EDs and GI disorders in their separate silos, the client suffers. Comprehensive care requires that ED interventions be tailored to GI needs and GI interventions be ED-informed.

In this section, we will address what we feel are the main ED treatment interventions and how they apply to people with GI symptoms. Certain difficulties may arise when implementing nutrition interventions for co-occurring EDGI disorders. For example, the RDN may find that the nutrition intervention for the ED and that of the GI diagnosis are in conflict with one another. In addition, many RDNs may feel that their counseling skills and psychology education is limited and hinders their ability to provide effective care for those with EDs. Where applicable, additional information is provided to guide the RDN in choosing and executing the described interventions.

### Food Philosophies and Care Models

For the purpose of eating disorder treatment, it is recommended that the RDN have an understanding of or be trained in various common psychological interventions. Examples include cognitive behavioral therapy (CBT), dialectical behavioral therapy (DBT), and acceptance and commitment therapy (ACT), among others.

In addition, there are several food philosophies and care models that underlie many nutrition interventions. These philosophies and models are incorporated into the EDGI interventions described in the rest of this section. Each of these merits training and education beyond the scope of this text, so we have provided resources for further learning in the Resources for the RDN section.

### *All Foods Fit & Food Neutrality*

Common concepts in eating disorder treatment, food neutrality and the phrase "all foods fit" aim to reduce judgment of food choices, preferences, and eating behaviors; self-criticism is often a precursor to disordered eating behaviors because it cues the need for perfectionism, numbing/dissociation, or other forms of coping.

Some people with gastrointestinal and other chronic health conditions will find the phrase "all foods fit" to feel inaccurate, given that there are cases in which eliminating foods may be medically necessary. In this case, "food neutrality" can be more accessible as a concept to remove the moral connotations of categorizing foods as "good or bad," "safe or unsafe," "healthy or unhealthy." Instead, the choice to eat or not to eat a food is framed as morally neutral, regardless of whether that food causes physical harm in the body.

### *Intuitive Eating*

This framework is supported by over 200 studies that show a reduction in the risk of eating disorders and weight cycling, as well

as improvements in body dissatisfaction and cardiometabolic risk factors. The Ten Principles of Intuitive Eating include:

- Reject the diet mentality
- Honor your hunger
- Make peace with food
- Challenge the food police
- Discover the satisfaction factor
- Feel your fullness
- Cope with your emotions with kindness
- Respect your body
- Movement–feel the difference
- Honor your health–gentle nutrition

Intuitive Eating was developed and copyrighted by Elyse Resch, MS, RDN and Evelyn Tribole, MS, RDN. For more information, visit intuitiveeating.org.

### *Mindful Eating*

Teaching mindfulness and mindful eating encourages clients to be fully present without judgment while eating meals and snacks. This framework is appropriate for some, but not all, clients (especially in the beginning of ED treatment when hunger and fullness are not accurate cues for guiding nutrition). Furthermore, those with gastrointestinal distress may find it unhelpful to be more mindful of their body sensations while eating. When a person is able to engage with mindfulness and form a more positive association with the experience of eating, this can be a helpful framework. Examples of mindful eating include eating without any other

distractions, checking in with body sensations before and after eating, and noticing hunger and fullness, among others.

### *Harm Reduction*

ED behaviors, thoughts, and feelings are often protective or adaptive to the individual in some way, despite being physically harmful. Harm reduction strategies can acknowledge and honor this dialectic. They can reduce shame for engaging in behaviors and encourage feelings and acts of self-care. Examples of harm-reduction interventions include:

- Decreasing frequency of ED behaviors rather than eliminating altogether
- Modifying the behavior to be less severe or risky
- Identifying what need the behavior fulfills and practicing self-compassion around use of the behavior

### *Health at Every Size® (HAES)*

This is a care and advocacy framework developed by fat activists and trademarked by the Association for Size Diversity and Health (ASDAH). Health at Every Size® is rooted in civil rights and health equity discourse and holds that people should not be excluded or prevented from accessing quality healthcare based on their size. Furthermore, higher weight is seen as a neutral trait and not indicative of health status or characterized as a disease. Health-promoting behaviors, if pursued, should be sought without moral obligation, judgment, shame, or oppression. For more information, visit ASDAH.org.

### *Trauma-Informed Care*

Trauma-informed care respects the client's lived experience and focuses on creating a felt sense of safety in the relationship with

their provider. It emphasizes client consent with regard to the treatment plan and prioritizes body autonomy over the clinician's comfort or expectations. This can be a reparative experience for those who have felt disempowered by past medical encounters and/or other trauma and who have difficulty expressing their needs and boundaries in the present. Examples may include explaining an intervention to a client and asking for their feedback and consent prior to implementing it or encouraging the client to voice when they would like to pause or stop the intervention.

### *Neurodiversity-Affirming Care Model*

A fairly new model coined by Naureen Hunani, RD of RDs for Neurodiversity, the Neurodiversity-Affirming Care Model seeks to provide eating disorder treatment that validates and accommodates an individual's neurodivergence rather than dismissing, invalidating, or pathologizing that person's needs. This model sees neurodivergence (for example, autism spectrum disorders, sensory processing disorders, attention deficit disorders) as a neutral part of human diversity, rather than as pathological. It seeks to find healing and growth as directed by the individual's own wants and needs rather than objective standards. Examples may include:

- Asking a client if they want to change their eating behaviors, what motivations they might have for doing so, and if that motivation is related to internalized stigma
- Helping a client identify internalized stigma in their judgments of their own eating behaviors, and how that further interferes with their relationship with food
- Not engaging in sensory desensitization or Exposure and Response Prevention (ERP) therapies if that intervention is experienced as unhelpful at best and traumatizing at worst

For more information, please visit rdsforneurodiversity.com.

## Education and Counseling Interventions

In the treatment of GI disorders, the provider's knowledge base and treatment plan can often provide a succinct path to resolution. In eating disorders, the path can be much slower and winding, meaning the "how" of interventions is just as important as the "what." Education and Counseling Interventions will review the "how."

### *Set expectations for dietary treatment for disordered eating/eating disorders*[1].

- The number of sessions with the RDN may be much more frequent than medical nutrition therapy for other conditions, often occurring on a weekly or biweekly basis, over months or years to provide adequate support for behavioral change.
- ED progress may not be linear; clients may experience plateaus, lapses or worsening behaviors, as well as phases of progress toward recovery. A common refrain amongst ED practitioners is that "recovery is not a straight line," similar to other life transitions (e.g., education, career, relationships).
- Set the expectation that ED lapses may arise after progress is made. Discourage black-and-white thinking that would equate lapse or setback with relapse to prevent fatalistic thinking.
- Be transparent that weight and body composition is likely to change. Weight-suppressed clients may find they gain weight in the abdomen initially and that it redistributes over the course of approximately one

year[2,3]. It is important for clients to be informed of this process, even if they find it extremely challenging to accept.

- Invite the client to acknowledge how current/past eating patterns have interfered with various aspects of wellbeing. If needed to increase client engagement, encourage the client to view nutrition recommendations as a short-term trial or experiment. This may also decrease the pressure to pursue the "perfect" treatment plan and allows space for ambivalent feelings about treatment.

*Educate on the interplay between nutritional rehabilitation and gastrointestinal symptoms.*

- Hunger and fullness cues will likely be unreliable and confusing in hypermetabolic states and across many medical conditions and treatments[4].
- GI discomfort anywhere throughout the digestive tract is common in the early stages of nutrition rehabilitation as the body adapts to digesting and metabolizing more or different foods[5]. Providers, however, should not assure the client that all discomfort will resolve with eating disorder recovery because some will find that their GI symptoms persist beyond recovery.
- Visceral hypersensitivity is also a common co-occurrence with eating disorders and educate on the difference between discomfort and harm in the body (for example, consuming milk with a diagnosis of lactose intolerance causing uncomfortable flatulence versus consuming gluten with a diagnosis of celiac disease, causing intestinal damage)[6].

- Changes in appetite and GI symptoms will likely be dysregulating to the client[6]; acknowledge the discomfort of these sensations. Frame them like other pains of change that are uncomfortable but not harmful. For example:
    - Puberty—body changes are very difficult, but "normal" and inevitable.
    - Difficult feelings—feeling sad, angry, or disappointed, may all feel unpleasant or even intolerable, but they are also "normal" to feel and don't actually cause lasting harm to the body.
    - Learning a new skill or sport—initially new muscles may feel sore, but the body adapts over time.

*Dispel harmful nutrition information.*

- Clients are bombarded with nutrition misinformation and may implement inappropriate recommendations to manage their symptoms or disease. When counseling clients, ask about the source of their nutrition/supplement information, and clarify dietary management that's relevant to their diagnosis[1].
- Wherever possible, recommend a liberalized approach towards diet.

*Challenge food-related morality.*

- Foods are often seen in a binary of "good" or "bad" ("healthy" or "unhealthy"), with personal responsibility for eating these foods conferring a sense that the person is also "good" or "bad." This commonly leads to restriction driven by food morality and ED behaviors to compensate

for eating "bad" foods. Potential cognitive reframes for "good" and "bad" food categories may include:

  - "Even if this is true, it is not helpful to me"
  - "What food might feel good to one person might not feel good to me"

- Help the client understand how the ED is perpetuated by black-and-white thinking and reduce the mental burden of food-related thoughts.
- Consider the use of exposure and habituations in order to dismantle food morality (e.g., give assignments to obtain and eat so-called "bad" foods, participate in social eating opportunities, make "bad" foods available at home).

*Convey empathy and validation.*

- Many individuals with EDGI may have experienced past trauma related to feeling invalidated or not believed. Use language that validates their struggle. For example:
  - "It makes sense that..."
  - "I believe you."
  - "This is really hard."
  - "Of course these symptoms are making ED behaviors worse, and of course these eating behaviors are impacting your GI symptoms."

*Reassess treatment progress and goals with the client*[1].

- When appropriate and at well-timed occasions, engage the client actively in reassessing their own recovery progress. This is best done when client is not

hypervigilant or overly critical of their own markers of progress

- Utilize these discussions to acknowledge progress in some areas, even if the client feels that they are not making progress in others, and develop buy-in for referral to higher level of care or other treatment, if additional support is needed.

*Help client understand and interpret medical information.*

- The RDN should serve as a bridge between clients and other medical providers by confirming the client's understanding of medical diagnoses, test results, and procedures (to the extent that it is within their scope of practice both ethically and legally)[7,1]. This can reduce medical anxiety, build rapport and trust, and offer an opportunity to outline the role (or limitations) of dietary interventions.

*Discuss aspects of management for the GI diagnosis that may be activating for the ED.*

- Assist the client in differentiating behaviors required for adequate management of GI conditions from past/present ED behaviors. Examples may include:
  - Reading food labels for ingredients, nutrition facts, or health claims
  - Avoiding/declining food in social situations
  - Finding limited options when dining out at restaurants rather than being able to choose according to preference, appetite, etc.
  - Taking small bites, chewing thoroughly, small/frequent meals

- Challenge ED thoughts, beliefs, and behaviors while adhering to necessary dietary modifications (e.g., using myth-busting, positive self-talk, reincorporating pleasurable foods)[4].

*Explore client's relationship with food and body as it relates to GI diagnosis.*

- Examine how GI symptoms and/or medical encounters have impacted the client's food choices and experience of being in their body.
- Dismantle internalized shame and blame for diagnosis and/or symptoms, as the development of health conditions is multifactorial and shame often exacerbates ED behaviors.

*Encourage lifestyle habits that support overall wellbeing.*

- Endorse stress management techniques. Stress can exacerbate symptoms of DGBIs, visceral hypersensitivity, hypervigilance, and anxiety. Various modalities may be accessible or of interest to different clients, including deep breathing, meditation apps, soothing music, progressive muscle relaxation, community groups, etc.[8]
- Encourage movement, when appropriate. Movement can play a role in improved digestion, mood, sleep quality, and other aspects of wellbeing. Activities of daily living, formal exercise, or a combination of the two can contribute to improved health outcomes (this recommendation hinges on the client's relationship with exercise and nutritional status; physical ability and accessibility should also be considered)[1].

- Promote restful sleep, as it has a significant impact on both mental health and gastrointestinal health. Many people benefit from taking steps to optimize their sleep. This may include creating a wind-down routine, reasonably consistent bedtime and wake-up times, reducing screen time before bed, and/or keeping the bedroom dark and comfortably cool, if possible.
- Recommend comfortable clothing. Tight, fitted clothing (especially around the abdomen or neck) may exacerbate visceral hypersensitivity, upper GI distress, and/or body surveillance[5].

## Dietary Interventions

Eating disorders present a greater threat to one's health and wellbeing than nearly all GI concerns. As such, ED nutritional rehabilitation takes precedence over GI-focused dietary changes. The following tenets establish ED care as primary or concurrent with GI disease or symptom management.

### *Recommend an eating pattern, also called a "meal plan" or "nutritional rehabilitation plan."*

- In the absence of competent eating or medical contraindications, encourage food intake approximately every 2-4 hours
- An eating pattern or meal plan can[9]:
  - reduce food decision-making stress and anxiety for the client
  - communicate clear guidelines to promote accountability
  - assist caregivers who are involved with food intake

    - re-establish hunger and fullness cues at regular intervals which mirror usage of liver glycogen stores

- Encourage "self-care eating" or "mechanical eating" when clients have difficulty responding to hunger/fullness cues or in cases of altered appetite related to medical conditions and medications.

*Consider needs for macronutrients, micronutrients, fiber and fluids considering anatomy, physiology and hypermetabolism.*

- Many individuals will have specific needs for individual nutrients based on their anatomy and physiology. Institute appropriate diet without focusing on "numbers" such as grams or calories[9].

- Those who are in the process of refeeding may become hypermetabolic and begin to lose weight with a stable food intake.
    - Do not explicitly discuss numbers with the client unless it is part of a planned intervention with psychological support.
    - The RD should consider an increase of approximately 500 kcals 1-2 times per week to continue the desired rate of weight gain if weight is plateauing and/or decreasing[5,4].
    - It is common for energy needs to range between 40-100 kcals/kg current body weight to reach biologically appropriate weight[10].
    - It is common for protein intake to be >2.0 g/kg with high-calorie meal plans; monitor renal and metabolic labs to ensure safety[10].

- It is common for liver enzymes to be elevated with refeeding, both due to the positive balance of nutrients being processed and stored in the liver, as well as due to autophagy[7,5]. Refer to physician to monitor liver enzymes rather than decrease energy intake.

*Review medications and supplements to provide appropriate diet intervention.*

- Many medications and supplements will have nutritional contraindications[5]. Identify possible food-drug interactions and suggest changes in timing, form, dose, or frequency (where within scope of practice).
- Identify potential side effects that are influencing GI symptoms[5]. Examples include excessive vitamin C supplementation or antibiotics contributing to diarrhea, or iron supplementation or opioids contributing to constipation.

*Oral nutrition supplement (ONS) use may be indicated.*

- The RDN should recommend ONS as a more accessible means of meeting energy and protein needs when[11]:
  - A client feels that they cannot physically eat more due to texture or sensory aversions, physiology and anatomy, absence of hunger cues, or exacerbation of gastrointestinal symptoms.
  - A client is psychologically unable to eat the food offered due to eating disorder or trauma-related feelings, thoughts, or beliefs.
  - Energy needs exceed a realistic dietary intake.

    - Energy needs would mimic past binge-eating behaviors that would interfere with client's progress.
- Be wary of when ONS contradicts other therapeutic goals, such as increasing oral food intake or transitioning off nutrition support dependence.

*Assess for enteral or parenteral nutrition needs.*

- EN and PN nutrition support are generally contraindicated in eating disorders due to increased medical complications (such as bacterial infections, new wounds) and increased opportunity for eating disorder behaviors (such as purging or restricting by manipulating the equipment delivering nutrition support)[7,5].
- In many individuals with co-occurring GI conditions, however, EN or PN may be necessary and/or facilitate ED recovery. Example scenarios include when indicated due to anatomy/physiology, as a short-term bridge in nutrition rehabilitation when oral intake is not yet high enough to support physiological needs, or to allow for nutritional rehabilitation and/or weight gain in clients who have multiple barriers to eating orally but are ready for treatment[7,5].

*Liberalize diet orders or guidelines.*

- While a certain diet may be recommended for a given medical condition, it is also essential to encourage an eating pattern that promotes eating disorder recovery. The RDN, client, and treatment team must triage which dietary guidelines are most important at that time[4].
- Encourage a liberalized diet that promotes food variety, eating for enjoyment, and social eating, as many EDGI

individuals will avoid tolerated foods and stick to a narrow selection of foods that "feel safe"[4].

- If the client is not open to liberalizing, explore why, and collaborate with their mental health practitioner, if available[4].

*Utilize a positive nutrition counseling approach.*

- Focus on foods to include rather than foods to avoid.
- Frame necessary avoidance of foods for physical health promotion rather than morality or virtue and find acceptable substitutes and alternatives for any necessary exclusions.
- Explore if any substitutions feel restrictive or depriving and seek to either resolve that or process it.
- Ensure that the client's original eating pattern is respected (i.e., make changes starting with what they're already eating rather than suggesting an entirely new selection of foods)[5].

*Address micronutrient abnormalities.*

- Coordinate with appropriate clinician to assess and monitor tests for micronutrients[5].
- Recommend the most accessible form of the nutrient (e.g., physically accessible, financially reasonable, best absorbed, best tolerated).
- Supplementation should be prioritized when current food intake is not likely to change quickly enough to address the abnormality, when deficiency/toxicity sequelae are already present, or when

anatomy/diagnosis does not allow for adequate intake or absorption of the nutrient[4].

*Facilitate food exposure and habituation for avoided foods.*

- Coordinate care with the client's therapist for increased psychological support[1].
- Educate client on the avoidance-anxiety relationship, in which avoidance of the stimulus for anxiety actually increases and perpetuates the anxiety toward that stimulus. Habituation (repeated exposure with tools for coping with dysregulation) to the stimulus is more effective in reducing anxiety than avoidance[12].
- Coach the client to identify foods that are avoided, and rate them by how challenging it would be to (re)introduce them. This is sometimes called a "food exposure list," "fear foods list," or "food inventory." A few different ways to rate and categorize these include:
  - Traffic light system: red light foods, yellow light foods, green light foods
  - Subjective Units of Distress scale (SUDS): rate on scale of 1-10
  - ED language: safe foods and fear foods (people with sensory-specific ARFID or in the neurodivergent community may use the terms "yes" and "no foods" to convey a similar meaning or "same foods" to indicate repetitive food choices)
- Explore the client's personal motivations for (re)introducing foods to make the work more meaningful. Examples include participating in social gatherings, meeting a particular nutrient need, expanding

accessibility to acceptable foods, or reducing fear and anxiety around fear foods.

- Discuss the food exposure prior to tasting it[13].
  - Dispel any misinformation about the food and discuss benefits and neutral characteristics of the food.
  - Help the client identify what led to that particular food avoidance, as there may be different reasons for different foods (e.g., body image concerns, food trauma, fear of symptoms, sensory differences, etc.). If needed, offer modifications that reduce risk of symptoms recurrence or trauma response (e.g., have the food in a more tolerated form/portion or more preferred texture).
- Create a supportive environment for food (re)introduction. Examples include:
  - With distractions to reduce somatic experience of the food and feelings (e.g., listening to music, watching videos)
  - With social support (e.g., with a friend, family member)
  - In session with the RDN and/or therapist
  - Free from other triggers (e.g., in a quiet space if loud noises are dysregulating, away from others who may speak about the food in an unhelpful way)
- In order to fully incorporate client consent, allow for a change of mind or pace as needed by the client. Uphold their autonomy and do not convey a sense of coercion, disappointment, or threat; pressuring food exposure can be retraumatizing and push the client to engage with ED behaviors to regain a sense of autonomy.

## Interdisciplinary Provider Team Interventions

Best practice is to coordinate care with all the members of a client's care team, and in an ideal world, this would include offering feedback about the impact of other providers' interventions to continually improve care[1]. In actual practice, the RDN may encounter unresponsiveness, ego, or other challenges to collaboration. Every effort should be to reach out to the other parties, regardless of whether contact is ultimately established or ongoing.

- The RDN should seek to establish contact with members of the client's healthcare team to coordinate care. To facilitate this, the RDN should request Release of Information forms for any providers they deem integral to the care plan.
- The RDN should communicate the following to the team[1]:
  - their nutrition-related assessment, diagnosis, and plan
  - client-reported eating disorder behaviors, beliefs/thoughts, physical symptoms
  - client's level of engagement or other provider concerns
- Coordinate to assess and monitor nutrition-related labs and treat as indicated[5].

## Treatment Goals by Eating Disorder Behavior

Eating disorder behaviors can occur across multiple diagnoses. Treatment goals should be based on behaviors—reducing them and coping with the distress that happens in their absence.

*Restriction*

- Build up to adequate caloric intake by starting with client's current intake or at a level where they feel moderately challenged[5].
- Work through plateaus by investigating barriers to increasing intake through open dialogue[5].
- Promote food variety and encourage client to challenge "fear foods" (e.g., high-calorie, high-fat, containing sugar, large portions, caloric beverages, different textures/flavors).
- Honor preferences and limitations based on medical necessity and client autonomy.
- Challenge food rules and meal-time rituals, as well as cognitive rigidity outside of the ED (e.g., lack of spontaneity, emotional guardedness, self-denial).

*Bingeing*

- Encourage adequate eating to reduce the risk of rebound/deprivation-driven eating[5].
- Reduce shame and guilt related to eating by viewing bingeing as a means of coping.
- Encourage mindfulness and body awareness during binges to stay attuned to fullness cues and emotional data (this may be inappropriate for clients with active trauma symptoms)[5].
- Develop "toolbox" of coping strategies as alternatives (or delays) to bingeing[5].

- Disrupt routines and rituals that lead up to a binge[5,12]. *Total abstinence from bingeing is not a reasonable treatment goal; the provider should view bingeing as a neutral event and increased understanding of motivations to binge as significant progress. Over time, bingeing episodes are likely to decrease.

*Compensatory Behaviors (Including Excessive Physical Activity)*

- Build understanding for how compensatory behaviors manage anxiety and/or offer a false sense of control.
- Develop "toolbox" of coping strategies to ride out urges to use compensatory behaviors.
- Redesign environment to reduce ease of behavior use (e.g., avoiding the bathroom after meals to prevent self-induced vomiting, throwing away laxatives, committing to group exercise only)[5].
- Avoid reinforcing all-or-nothing thinking by celebrating reductions in behavior use (and not just behavior abstinence).

*Rumination*

- Develop understanding of when rumination happens consciously versus unconsciously and precipitating factors.
- Practice diaphragmatic breathing during and after meals to reduce occurrence of regurgitation.
- If eating quickly or to point of over-fullness increases rumination, encourage pattern of moderate portions of food intake at regular intervals throughout the day.

*Pica*

- Explore how eating non-nutritive substance may be soothing to the client[14].
- Develop "toolbox" of alternative behaviors to approximate the perceived "benefit" of eating non-nutritive substances.
- Address any micronutrient deficiencies or other medical causes of the cravings for non-nutritive substances (Rajput, 2020).

*Body Checking*

- Build awareness of frequency of body checking, precipitating factors, and whether the behaviors elicit positive or negative self-talk.
- Reduce the number of occurrences and/or duration by setting timers, scheduling behaviors, developing distraction tools, etc.
- Redesign environment to reduce ease of behavior use (e.g., cover mirrors, throw out clothes that serve as body measurement tools, ask a loved one to hide the scale).
- *It is the RDN's role to hold the client's body in unconditional positive regard, despite physical changes they may experience. The RDN should take great care to project neutrality about the client's weight, shape, and size.

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# ED-Informed GI Disease-Specific Interventions

The following recommendations offer ED-informed interventions for specific GI conditions, both to treat GI disease in the presence of an active eating disorder and to reduce the risk of ED development. The RDN should use their clinical judgment of the client's food fear, rigidity, recovery progress, and other circumstances to determine which GI interventions are appropriate to offer. Interventions are grouped by GI condition; co-occurrence of multiple GI conditions is common, so the reader may have to consult multiple tables to adequately treat clients.

## Abdominal Bloating & Distention

Abdominal bloating and/or distention are common GI complaints and can be symptoms of various diagnoses, including IBS, constipation, and others[1]. However, in the case of functional abdominal bloating/distension (which includes abdomino-phrenic dyssynergia/APD), bloating can be a seemingly stand-alone issue. Nutritional guidance in this arena is extremely limited, and most interventions are based on clinical experience rather than robust research.

| Nutrition Intervention | Considerations |
|---|---|
| Consider small, regular meals throughout the day | A "three square meals" approach may exacerbate distention for those with functional bloating. Eating opportunities comprised of smaller meals and larger snacks spaced throughout the day may be better tolerated.<br><br>Delayed or skipped meals due to fear of bloating/distention may trigger bingeing later in the day, perpetuating disordered eating behaviors and creating even more bloating. |
| Introduce low-roughage dietary | Foods high in insoluble fiber take longer to break |

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| modifications | down in the stomach and are more likely to cause distention. Examples include popcorn, trail mix, dried fruit, and large portions of raw vegetables. Breaking down the fiber in the cooking process (e.g., cooking, blenderizing) reduces the burden of mechanical digestion for the stomach. |
| Assess hydration needs | Encourage clients to take fluids after or outside of mealtimes to reduce the risk of fluids crowding out more calorie-dense foods. |
| Determine appropriateness of using the low-FODMAP diet | FODMAPs are types of short chain carbohydrates that are poorly absorbed in the small intestine, which can trigger distention of the intestines by drawing in excess water and producing gas via fermentation by the gut bacteria.<br><br>A more detailed description of the low FODMAP diet can be found in the IBS intervention section.<br><br>If the client experiences a lot of flatulence (not related to stool back up/constipation), they may respond well to removing some high FODMAP foods that are suspected triggers (beans, lactose, onions, apples, etc.). Consider a trial elimination of just these foods (also known as "cherry picking"). Recommend substitutions for these foods to ensure adequate energy intake[2].<br><br>Sugar alcohols (sorbitol, manitol, xylitol, etc.) are within the polyol category of FODMAPs. They are generally malabsorbed and can be a bloating/gas trigger even for people without GI conditions. If you notice clients are consuming multiple foods with sugar alcohols per day, consider replacing these foods with non-sugar free versions[3].<br><br>Because the low-FODMAP diet is restrictive in nature, the full version is not appropriate for individuals with active EDs. |

| Non-Diet Intervention | Considerations |
|---|---|
| Trial simethicone | Gas and swallowed air can fill up the stomach and aggravate symptoms of bloating. Simethicone taken before meals can break up gas bubbles and offer some relief. Common brands include Gas-X and Phazyme[4]. |
| Refer to pelvic floor physical | In the case of bloating due to abdomino-phrenic |

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| therapy | dyssynergia, physical therapy and biofeedback can help retrain the abdominal muscles to move in coordination with the stomach and diaphragm. A pelvic floor PT is more likely to be familiar with this than general PTs.<br><br>Use https://pelvicrehab.com/ to search for pelvic floor physical therapists in the client's area. |
| Recommend diaphragmatic breathing after meals | Diaphragmatic breathing (or "belly breathing") can help train the abdominal wall muscles and diaphragm to work together. If the RDN is unfamiliar with diaphragmatic breathing, they may defer to the mental healthcare provider and/or pelvic floor physical therapist.<br><br>Belly breathing can be triggering for some clients with ED, as it can bring more attention to the size/distention of the abdomen. |
| Encourage client to speak with their doctor about neuromodulating medications | Improper nerve signaling may contribute to functional abdominal bloating. Neuromodulating medications such as muscle relaxants, tricyclic antidepressants (TCAs), and anticonvulsants may help[5]. |
| Refer to GI psychologist or relevant app to improve gut-brain interaction | Various therapeutic modalities, including CBT and gut-directed hypnotherapy, may improve visceral hypersensitivity and overall QOL[6,7,8,9]. Using non-diet therapies can be protective for people with EDs, as they reduce the focus on food. |

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## Bile Acid Diarrhea (BAD)

| Nutrition Intervention | Considerations |
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| Consider a low- or moderate-fat diet | High-fat diets are believed to exacerbate bile acid diarrhea by triggering release of bile acid. While limited evidence suggests a lower-fat diet may offer improvements (especially with regard to abdominal pain and nocturnal defecation), this intervention is not believed to fully manage the condition[1,2].<br><br>Restriction of dietary fat may hinder nutritional rehabilitation efforts and/or lead to disordered eating behaviors and should be approached with caution. |

| Non-Diet Intervention | Considerations |
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| Encourage client to discuss bile acid-sequestering medication with their physician/prescriber | Bile acid-sequestrants are the first-line approach in managing bile acid diarrhea and reduce the need for dietary restrictions. Medications taken <1 hour before or <4 hours after a bile acid sequestrant may not be properly absorbed[3,4,5]. |

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| | Common bile acid sequestrants available by prescription include:<br>• Colestipol (tablets)<br>• Cholestyramine (powder)<br>• Colesevelam (tablet or powder)<br>Medication titration is necessary for achieving the desired effect; some clients may do better with powdered forms that can allow for variable doses. |

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# Celiac Disease (CeD)

| Nutrition Intervention | Considerations |
|---|---|
| Implement a gluten-free diet | A strict gluten-free diet is the only treatment available for celiac disease. The gluten-free diet requires total elimination of wheat, barley, rye, and products made with or derived from these ingredients.<br>Fruits, vegetables, meat, eggs, seafood/fish, poultry, most dairy products, beans, legumes, seeds, whole grains (e.g., quinoa, buckwheat, millet, rice, oats labeled GF, etc.) are all naturally gluten-free and can be consumed safely on a gluten-free diet in their natural form. There also are many available gluten-free |

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| | substitutes for gluten-containing foods (e.g., breads, pastas, crackers, etc.) that individuals with celiac disease can consume safely.<br><br>Educate patients on label reading for a gluten-free diet and help them understand how to identify the presence of gluten on a food label. |
| Assess for micronutrient deficiencies | Individuals with celiac disease may experience micronutrient deficiencies related to malabsorption (in untreated celiac disease or those not following a GFD) or inadequate intake. The most common deficiencies in this population include vitamin D, zinc, iron, ferritin, B vitamins, calcium, and magnesium[1,2].<br><br>Work with patients on increasing intake of these nutrients through food. Supplementation may be indicated in some clients (especially those with co-occurring EDs) to quickly improve nutritional status and avoid long-term complications. |
| Encourage client to plan ahead to assure gluten-free options are available to them | Gluten-free food options are not always readily available, so researching eateries in advance and/or bringing snacks along offers clients opportunities for safe and regular nourishment. |
| Encourage client to explore a variety of gluten-free foods | Due to the restrictive nature of the gluten-free diet, it's important that clients have a variety of foods they can eat that will bring them joy. This may include incorporating fun foods, trying gluten-free substitutes for favorite foods, experimenting with new recipes, and sharing dishes with friends and family. |
| Equip client to self-advocate at restaurants, catered events, and social situations | Ask staff about the production process for meals for someone with celiac disease. Questions to consider asking include:<br>• Does your kitchen have a dedicated preparation and cooking area/pans/cutting board for preparing gluten-free meals?<br>• Does staff change their gloves when moving between preparing gluten-containing meals and gluten-free meals?<br>• Does your kitchen have a dedicated gluten-free fryer?<br>• How do you go about ensuring that your gluten-free meals are safe for people with celiac disease?<br><br>Celiac disease and its dietary limitations are not always well understood by staff. Clients are encouraged to |

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| | reiterate the seriousness of their dietary needs using phrases such as "sensitive to cross-contamination" and "allergy." |
| Eliminate sources of gluten cross-contamination | Recommend safety measures to reduce gluten cross-contamination:<br>• If the household is not completely gluten-free, thoroughly wash utensils, pans, cutting boards, prep areas, etc. when switching between cooking gluten-containing and gluten-free food. Washing cooking equipment with soap and water is adequate for removing gluten[3,4].<br>• In shared kitchen environments where equipment and surfaces are difficult to clean, suggest cooking gluten-free food on surfaces that provide a barrier to the exposed surface, such as dedicated baking sheets, trays, foil, or "toaster bags"[5,3].<br>• Choose oats/oat products that are labeled gluten-free and have clear and transparent GF testing standards. While the majority of people with CeD do not react negatively to pure oats, monitor for tolerance[6].<br>• Look at packaged foods being consumed to confirm gluten is not hidden in the ingredients list. If the labeling is unclear, consider calling companies to find out about their production process to ensure less than 20 ppm gluten for safe consumption.<br>• Assess restaurant meal intake and safety (above) as well as meals at other people's houses (frequency and safety).<br>• At this time, the use of gluten detection devices, sensors, and gluten detection dogs are not recommended as reliable sources of gluten contamination[6,7].<br>Monitor for signs of over-restriction and hypervigilance due to fear of cross contact with gluten. Help the client to prioritize safety without an all-consuming preoccupation regarding contamination. Advise clients about the high prevalence of misinformation online about the gluten-free diet and direct them to reliable sources of information (e.g., Celiac.org, BeyondCeliac.org, NationalCeliac.org). |

| Non-Diet Intervention | Considerations |
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| Consider referral to mental health professional | Assess need for additional support in managing a new diagnosis/chronic medical condition. Clients may express fear and anxiety related to eating and/or grief related to lifestyle changes. |
| Encourage the client to seek out community support | Clients may benefit from celiac support groups, apps for finding gluten-free restaurants (e.g., Find Me Gluten-Free), and patient advocacy organizations (e.g., Celiac.org, BeyondCeliac.org, NationalCeliac.org).<br><br>Many social media groups and forums are not moderated by health professionals and may feature misinformation. |
| Support client through body changes related to intestinal healing | Recovering from celiac-related intestinal inflammation may lead to weight changes, stronger hair and nails, improved energy levels, and improved digestive symptoms. Some body changes may trigger emotional reactions. Explore the source of the fear and work towards improving body image as needed. |
| Refer to gastroenterologist to assess for non-responsive and refractory celiac disease if no improvement on strict gluten-free diet | Non-responsive celiac disease is defined as continuing persistent symptoms, elevated antibodies, or small intestinal damage even after following a strict gluten-free diet for 6-12 months.<br><br>Refractory celiac disease is defined by lack of response to a strict gluten-free diet after 6-12 months with symptoms, intestinal damage, and abnormal population of white blood cells in the gut (called *abnormal intraepithelial lymphocytes*). This iteration of celiac disease significantly increases cancer risk. |
| | Non-responsive and refractory celiac disease are uncommon, and an effort should be made to identify sources of gluten cross-contamination in the diet before making this referral. |

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## Chronic Constipation

Constipation may have several etiologies, including normal-transit (sometimes referred to as "functional"), slow-transit, and pelvic floor dysfunction. In the absence of a clear diagnosis, the RDN may suspect pelvic floor dysfunction when the client fails to respond to (or symptoms are exacerbated by) traditional nutrition interventions.

| Nutrition Intervention | Consideration(s) |
|---|---|
| Encourage regular meals throughout the day | Meal intake stimulates the gastrocolic reflex, which can help spur colonic motility. High-fat and high-volume meals can trigger a more pronounced reflex[1]. |
| Encourage adequate intake of water (1.5-2L/day) | Fluids are essential for maintaining soft, easy-to-pass stool[2]. Individual hydration needs vary, and comorbid conditions may require more or less fluid. If no contraindications, encourage clients to consume |

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| | enough fluids that their urine maintains a faint yellow color. |
| Encourage increased fiber intake | This may be especially helpful for clients with low baseline fiber intake[3-5].<br><br>A fiber supplement may offer faster relief while gradually increasing fiber via dietary sources (see Non-Diet Interventions for Chronic Constipation).<br><br>For a client already consuming high amounts of fiber, consider rebalancing fiber sources to favor soluble (e.g., grains/starches, squash, root vegetables, chia seeds) over insoluble fiber (e.g., cruciferous vegetables, leafy greens, whole nuts)[6]. In some cases, a reduction in fiber intake may be warranted to help stool output keep pace with fiber intake[7,8]. |
| Emphasize soluble fiber and limit insoluble fiber*<br><br>**Consider for clients presenting with suspected or confirmed pelvic floor dysfunction* | Among people with pelvic floor dysfunction, soluble fiber may be better tolerated than insoluble fiber. Soluble fiber helps form and soften stool and can be found in foods like oatmeal, chia seeds, peeled fruits and veggies, etc. Insoluble fiber ("roughage") can be difficult to evacuate if the pelvic floor muscles are not coordinating properly and include foods like raw veggies, leafy greens, popcorn, fruit skins, etc. While insoluble fiber does not need to be eliminated, it should not be the primary fiber source[9]. |
| Trial a low-fiber diet*<br><br>**Consider for clients presenting with suspected or confirmed pelvic floor dysfunction* | For patients with a suspected high stool burden, trial of a low-fiber diet (less than 8 grams) may be helpful for reducing uncomfortable bloating symptoms while they await medical treatment for pelvic floor dysfunction[10]. |
| Recommend consumption of two kiwi per day or a serving of prunes | Several studies involving kiwis demonstrate a positive effect on complete spontaneous bowel movements[11,12]. At least one study shows the efficacy of kiwis is not statistically different from that of prunes or psyllium. Prunes are generally well tolerated but also contain sorbitol, which may trigger gas and/or diarrhea in sensitive clients. |
| Recommend regular consumption of flaxseed (linseed) | Regular consumption of flaxseed has been shown to increase frequency of bowel movements and improve stool form and evacuation. The typical amount used in these research studies has been 20-50g (~2-5 tablespoons whole, ~3-8 tablespoons ground) split over 2 doses[13-15]. |
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| Recommend drinking coffee (regular or decaf) in the morning | Coffee helps stimulate colonic motility and can be part of a daily bowel regimen[16]. |

| Non-Diet Intervention | Consideration(s) |
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| Educate client on optimal toileting position | Clients who defecate while in a squatted position (with their feet propped up so that the knees are higher than the hips) may benefit from improved bowel evacuation, reduced straining and shorter bowel movement duration[17]. Squatting can be achieved through use of a Squatty Potty or any similar household object (e.g., small step stool, toilet paper roll under each foot, stack of books, yoga blocks, etc.). |
| Consider a functional fiber supplement | A fiber supplement may help increase a client's overall fiber intake and/or offer therapeutic properties that help with evacuation[18]. Common fiber products include:<br>• Methylcellulose<br>• Wheat dextrin (not appropriate for celiac disease unless labeled gluten-free)<br>• Partially Hydrolyzed Guar Gum (PHGG)<br>• Psyllium husk (best for alternating hard and loose stool)<br>• Polycarbophil (this fiber is technically insoluble fiber but works like a soluble fiber and can be effective for reducing incontinence related to pelvic floor weakness)<br>Fiber supplements may also be helpful in cases of pelvic hypotension (to bulk stool and make it easier to pass), fecal incontinence (to bind liquid stool), and rectocele (to prevent stool from getting trapped in the rectocele). They are not usually recommended in the case of dyssynergia and pelvic hypertension because the bulk can make it harder to evacuate. |
| Consider the use of a daily osmotic laxative | Osmotic laxatives may increase bowel movement frequency and improve stool form[10]. Start at the low end of the recommended range, dose in the evening, and slowly increase as needed.<br>• Polyethylene glycol, or PEG, comes as a powder. Typical dose is 1-2 "capfuls" per day. |

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| | • Magnesium supplementation (e.g., magnesium oxide, citrate) can come in pill, powder, gummy or liquid form; check for poorly tolerated sugar alcohols in flavored products. Typical dose is 400-1000mg per day[19,20]. |
| | If the client appears to need more than the highest dose indicated here, see sections on Stimulant Laxatives or Prescription Medications. If the client is unresponsive to the highest doses indicated here, see section on Pelvic Floor Physical Therapy.<br><br>High-dose magnesium is not appropriate for clients with impaired kidney function (i.e., chronic kidney disease)[21]. |
| Consider the use of daily stimulant laxatives | Stimulant laxatives increase bowel movement frequency by inducing colonic motility[10]. Start at the low end of the recommended range indicated by package instructions and slowly increase as needed. Do not increase beyond the maximum indicated dose.<br><br>• Senna glycoside<br>• Bisacodyl<br><br>Inquire about the client's history with stimulant laxatives, including past misuse, to inform recommendations. Many formal ED treatment centers will not permit use of these medications while under their care. |
| Consider use of glycerin suppository or saline laxative enema to facilitate a bowel movement | Suppositories and saline laxative enemas can offer temporary relief for constipation and bloating from PFD while a patient is waiting for physical therapy or other treatments[22].<br><br>Follow package instructions; do not increase beyond the maximum indicated dose. Many formal ED treatment centers will not permit use of these products while under their care. |
| Encourage client to discuss possible medical prescriptions with their physician/prescriber | Common classes of medications that improve motility[23] include:<br><br>• Serotonin agonists<br>• Secretagogues<br>• Bile acid-modifying agents<br>• Vibrating capsule (e.g., Vibrant[24]) |
| Encourage setting aside | Some clients may struggle to adequately relax and |

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| adequate time to relax and pass a bowel movement | allow passage of a bowel movement due to past trauma, avoidance of public or semi-private restrooms, demanding schedule, and/or anticipatory stress related to bowel habits. Consider referral for mental health therapy if anxiety around bowel movements is impacting daily life. |
| Encourage joyful movement | Physical activity reduces overall GI transit time[4].<br><br>The recommendation for increased physical activity may not be appropriate for clients struggling with inadequate intake, exercise compulsion, or other active eating disorder symptoms. |
| Screen for pelvic floor dysfunction and refer out to pelvic floor physical therapist, if indicated | Pelvic floor-targeted physical therapy is the gold standard treatment for pelvic floor dysfunction. Pelvic floor physical therapists can provide biofeedback, muscle re-education, breathing exercises, manual therapy, and client education on bowel habits. Possible indicators of pelvic floor dysfunction include[25]:<br>• Increased dietary fiber worsens bloating, constipation, and/or discomfort<br>• Unresponsive to laxatives<br>• Incomplete evacuation<br>• Pelvic and/or lower back pain<br>• Urinary or fecal incontinence<br>• Pain with sexual intercourse<br>• History of injury to pelvis (e.g., falling on tailbone, childbirth injury, sexual assault)<br><br>Use one of the following directories to find pelvic floor physical therapists near you:<br>• https://pelvicrehab.com/<br>• https://pelvicguru.com/ |
| Encourage client to self-apply bowel massage | Bowel massage is an inexpensive and noninvasive intervention that can improve colonic transit time and defecation frequency, as well as pain and quality of life[26].<br><br>The client can be instructed to massage their colon with firm pressure, moving in a clockwise direction, from the ascending through the transverse to the descending colon. This can be practiced for 10 minutes, three times weekly[27]. |
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| Ask about the client's use of splinting | Splinting is a technique used to evacuate stool without straining, in which a clean finger is inserted into the vagina to apply pressure to the wall between the vagina and anus to provide support as stool passes. Encourage its use if advised by MD and/or PT. |
| Referral to therapist, ideally GI psychologist | Gut-brain psychological therapies such as cognitive behavioral therapy, gut-directed hypnotherapy, and mindfulness-based stress reduction have shown to be effective in treating irritable bowel syndrome. These therapies may also provide benefit for PFD, especially those that have rectal hypersensitivity.<br><br>Prevalence of trauma history is high in patients with PFD[28]. Therapy may be an important adjunct treatment. |
| Encourage patient to speak to MD about additional treatment options | In the case of limited improvement from dietary support and target physical therapy, the client can be encouraged to explore additional therapies with their doctor (e.g., diazepam rectal suppositories, surgery, Botox injections, etc.). |

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## Dyspepsia & Functional Dyspepsia (FD)

| Nutrition Intervention | Considerations |
|---|---|
| Consider small, regular meals throughout the day | A "three square meals" approach may exacerbate symptoms for those with FD. Eating opportunities comprised of smaller meals and larger snacks spaced throughout the day may be better tolerated (i.e., every 3-4 hours). |
| Minimize beverages with meals | Due to postprandial distress and early satiety, clients with FD should prioritize food at mealtimes and |

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| | spread beverages out throughout the day. Encourage clients to stay hydrated in between meals with small sips instead of gulping fluids. |
| Chew food thoroughly | Chewing food to "applesauce" consistency can help with slowing down during meals. |
| Introduce low-roughage dietary modifications | Foods high in insoluble fiber take longer to break down in the stomach and are more likely to contribute to pain and postprandial distress. Examples include popcorn, trail mix, dried fruit, and large portions of raw vegetables. Food may be better tolerated when cooked to a "fork tender" consistency or otherwise broken down/blenderized. |
| Advocate for a low- or moderate-fat diet | Foods higher in fat may delay gastric emptying and increase hypersensitivity via gastrointestinal hormones. Liquid sources of fat may be more tolerable than solid [1]. Similarly, fat intake spread out more evenly throughout the day is likely to be better tolerated than boluses. Fat intake should be tailored to individual tolerance.<br><br>In malnourished clients, dietary fat restriction may not be an appropriate first-line therapy. |
| Promote inclusion of ginger in diet | Ginger has been shown to help reduce nausea and enhance gastric emptying[1]. |
| Encourage moderation of alcohol intake | Alcohol interferes with normal gastric physiology and may exacerbate FD symptoms. |
| Assess the client's diet for gastric irritants | The client's FD symptoms may or may not be adversely affected by these foods in their diet: coffee, tea, alcohol, inulin/chicory root fiber, spicy foods, cocoa/chocolate, carbonated beverages, tomato products, peppermint, capsaicin/chili peppers. |
| Include two kiwi fruit per day in the diet | Kiwifruit has a positive influence on abdominal pain and indigestion[2]. |

| Non-Diet Intervention | Considerations |
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| Practice deep breathing prior to meals | Preliminary research shows that slow, deep breathing for 5 minutes per day can help increase drinking capacity and quality of life for those with functional dyspepsia[3]. |
| Encourage a gentle walk after meals | Gentle, rhythmic steps can promote gastric emptying and reduce postprandial distress. This intervention |

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| | may not be appropriate or accessible for all clients. |
| Assess medication list for potential medication-induced dyspepsia | Medications that are associated with dyspepsia may include but are not limited to:<br>• Acabose (precose)<br>• Antibiotics<br>• Bisphosphonates<br>• Corticosteroids<br>• Herbs (e.g., chaste tree berry, feverfew, garlic, ginkgo, saw palmetto, white willow bark)<br>• Metformin<br>• Miglitol (glyset)<br>• NSAIDs (including cyclooxygenase-2 inhibitors)<br>• Opiates<br>• Orlistat (xenical)<br>• Theophylline[4] |
| Refer to gastroenterologist to rule out *H. pylori* infection | 20% of patients infected with *H. pylori* will experience a comorbid condition, which may include dyspepsia. |
| Trial appropriate supplements and over-the-counter medications based on client symptom profile | Several products exist that purport to improve upper GI symptoms. Monitor for improvement to ensure client does not continue with unnecessary and ineffective supplements:<br>• FDGard—shows improvement in global symptom scores[5]<br>• Iberogast (STW5)—possible improvement across dyspeptic symptoms[6]<br>• Gas-X (simethicone)—reduces upper GI gas burden<br>• Mylanta—reduces upper GI gas burden and acts as an antacid |
| Inquire about past medication use to determine whether medical referral is appropriate | Several medications may help control FD symptoms:<br>• Tricyclic antidepressants—can moderate epigastric pain[7]<br>• Antiemetics—reduce nausea and subsequent vomiting<br>• Busprione—improves fundic accommodation and postprandial fullness[8] |
| Refer to GI psychologist or mental health practitioner | Preliminary research suggests interpersonal therapy, CBT, stress management and mindfulness, and gut- |

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| | directed hypnotherapy may possibly benefit people with FD[9]. |

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# Gastroesophageal Reflux Disease (GERD)

| Nutrition Intervention | Considerations |
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| Assess common dietary triggers to determine whether limitations are needed | Dietary restrictions are often a first-line lifestyle modification for management of GERD. However, people with GERD vary in their sensitivity to these foods:<br>• Gastric irritants (e.g., alcohol, spicy food, acidic foods like tomato and citrus)<br>• LES relaxants (e.g., high-fat meal composition, mint, onion and garlic, coffee, chocolate)<br>• Gas pressure (e.g., carbonated beverages, excessive gum chewing)<br><br>Sweeping dietary restrictions are not recommended. Instead, the client and clinician should observe which foods (if any) are actually problematic. If it's appropriate to minimize these foods in the diet, the RDN should offer acceptable substitutes or help the client find tolerated portion sizes (e.g., choosing low-fat options or low-acid coffee)[1-5]. |
| Consider small, regular meals throughout the day | A "three square meals" approach may exacerbate symptoms for those with GERD. Eating opportunities comprised of smaller meals and larger snacks spaced throughout the day may be better tolerated (i.e., every 3-4 hours). |
| Inquire about possible co-occurring constipation that may impact upper GI symptoms | Stool burden can worsen GERD symptoms by increasing pressure on the stomach. If suspected, refer to Chronic Constipation section. |
| Advise client to avoid eating past fullness | Eating past comfortable fullness puts pressure on the LES and can exacerbate GERD symptoms. Consider coaching clients to recognize fullness cues, practice elements of mindful eating, or limit distractions if needed. |
| Consider low-roughage diet modifications | Focusing on softer, easily digestible foods can promote gastric emptying and prevent the patient from feeling overly full. Ensure to avoid triggering recommendations and instead, focus on simply adding in low-roughage modifications[6]. |
| Chew food thoroughly | Chewing food to "applesauce" consistency can help with ease of digestion and slowing down during meals[6]. |

| Non-Diet Intervention | Considerations |
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| Allow time between eating and going to sleep | Most people with GERD benefit from having adequate time for gastric emptying before bedtime (usually 2-3 hours). If client is malnourished or in active ED recovery, adequate intake should be prioritized and may necessitate an evening snack. In these cases, consider leaving *some* time between eating and laying down, possibly limiting fat and fiber in the snack, and/or choosing a snack that is full liquid consistency[3]. |
| Encourage client to elevate their head and torso while sleeping | Encourage patients to elevate the head of the bed 6-8 inches to prevent reflux when sleeping. Use bed stilts for the head of the bed or a wedge pillow under head of mattress[1]. |
| Encourage the client to lay on their left side when going to sleep | This sleeping position may reduce reflux due to the anatomy of the stomach[7,1]. However, it may not be accessible to all clients. |
| Encourage a gentle walk after meals | Gentle, rhythmic steps can promote gastric emptying and reduce postprandial distress. This intervention may not be appropriate or accessible for all clients. |
| Encourage gentle and/or upright exercise | Research regarding recommended type and frequency of exercise for the symptom management of GERD is limited. However, research shows running, in particular, to be correlated with increased relaxation of the LES, reduced esophageal emptying rate and increased abdominal pressure. These findings might be extended to other high-impact activities, and so it is recommended to first focus on gentle and/or upright forms of physical activity (e.g., walking, strength training, dancing). Vigorous exercise can be included as appropriate depending on symptom management and the individual's relationship with exercise[8]. |
| Practice stress management strategies | Increased stress is associated with worsened GERD symptoms[9]. Stress management strategies should be individualized and patient-centered. Consider whether dietary restrictions increase stress and assess appropriateness. |
| Encourage smoking cessation | Smoking is positively correlated with increased GERD symptoms[10]. |
| Encourage clients to wear loose clothing that doesn't constrict the abdomen | Increased pressure from clothes, shapewear, "waist trainers," chest binders, etc. (especially around the abdomen) can exacerbate reflux symptoms. |
| Consider use of over-the-counter medications and refer to physician for | Several classes of medications may help manage GERD symptoms and allow for liberalization of the |

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| possible prescription medication management | diet. Some can be found over the counter, while others require a prescription:<br>• Antacids<br>• H2 blockers<br>• Proton pump inhibitors (PPIs)<br>• GABA(b) agonist (used off-label and not FDA approved for the management of GERD at the time of this publication)[1] |

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## Gastroparesis (GP)

| Nutrition Intervention | Considerations |
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| Consider small, regular meals throughout the day | A "three square meals" approach may exacerbate symptoms for those with GP. Eating opportunities comprised of smaller meals and larger snacks spaced throughout the day may be better tolerated (i.e., every 3-4 hours). Many people with GP find tolerance is best in the mornings and declines throughout the day[1]. |
| Chew food thoroughly | Chewing food to "applesauce" consistency can help with slowing down during meals. |
| Reduce particle size of diet | Foods with a large particle size take longer to break down in the stomach and are more likely to contribute to pain and postprandial distress. Foods should be modified via cooking, peeling, deseeding, shredding, blenderizing, pureeing, and other methods to make them more tolerable to clients with GP[1]. |
| Advocate for a low- or moderate-fat diet | Foods higher in fat may delay gastric emptying and increase hypersensitivity via gastrointestinal hormones. Liquid sources of fat may be more tolerable than solid[2]. Similarly, fat intake spread out more evenly throughout the day is likely to be better tolerated than boluses. Fat intake should be tailored to individual tolerance.<br><br>In malnourished clients, dietary fat restriction may not be an appropriate first-line therapy. |
| Prioritize calorically dense foods/fluids | If the client struggles with nutritional adequacy or maintaining a biologically appropriate weight, recommend calorically dense food and/or beverages such as juices, smoothies, milk, soup, stews, nutritional supplements (e.g., Ensure, Orgain, Kate Farms), etc.[1] Due to postprandial distress and early satiety, clients with GP should minimize consumption of low-calorie foods and beverages. |

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| | Encourage clients to stay hydrated in between meals with small sips instead of gulping fluids. |
| Promote inclusion of ginger in diet | Ginger has been shown to help reduce nausea and enhance gastric emptying[3]. |

| Non-Diet Intervention | Considerations |
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| Encourage a gentle walk after meals | Gentle, rhythmic steps can promote gastric emptying and reduce postprandial distress. This intervention may not be appropriate or accessible for all clients. |
| Inquire about past medication use to determine whether medical referral is appropriate | Several medications may help address GP symptoms:<br>• Prokinetics—increase rate of gastric emptying<br>• Antiemetics—reduce nausea and subsequent vomiting<br>• Tricyclic antidepressants—can moderate epigastric pain[4] |
| Assess medication list for potential medication-induced delayed gastric emptying | Medications that are associated with GP symptoms may include but are not limited to:<br>• Aluminum hydroxide antacids<br>• Anticholinergic<br>• Antipsychotics<br>• Beta-agonists<br>• Calcium channel blockers<br>• Cyclosporine<br>• Dexfenfluramine<br>• Diphenhydramine<br>• Glucagon hydrochloride and glucagon-like peptide-1 analogs<br>• H2-receptor antagonists<br>• Opioids<br>• Progesterone<br>• PPIs<br>• Tobacco<br>• Tricyclic antidepressants[1] |
| Encourage clients to stay upright for at least 1 hour after eating | The supine position (laying down) is most likely to exacerbate GP-related symptoms. Encourage patients to stay upright for at least 1 hour after meals to expedite gastric emptying and to elevate the head of the bed 6-8 inches to prevent reflux when sleeping. |

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| | Use bed stilts for the head of the bed or a wedge pillow under the head of the mattress[5]. |

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## Inflammatory Bowel Disease (IBD)

Patients with IBD may have ongoing inflammation with limited symptoms (quiescent disease) or may have severe symptoms with no evidence of active inflammation (clinical remission)[1]. Therefore, one of the greatest challenges in patients with established IBD is determining whether ongoing symptoms (e.g., pain, diarrhea) are due to active inflammation or from an alternate etiology. If patients with IBD have overlapping comorbidities (such as IBS) and their disease is in endoscopic or histologic remission, consider additional interventions in the corresponding sections.

| Nutrition Intervention | Considerations |
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| Screen for malnutrition and related complications | Patients with IBD are at risk for malnutrition and sarcopenia and should be screened for both at time of diagnosis and thereafter on a regular basis[1,2]. |

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| | Malnutrition should be treated immediately, as malnutrition worsens the prognosis, complication rates, mortality, and quality of life in individuals with IBD[2]. Individuals with co-occurring EDs may be at an increased risk for malnutrition. Additionally, food group avoidance, perioperative status, and weight suppression increase risk of malnutrition[2,3].<br><br>In clients struggling with disordered eating and/or body image, frequent discussion of weight (even for the purpose of assessing malnutrition) can be triggering. Consider evaluating malnutrition risk by reviewing labs and asking about changes to appetite, stool form, and nausea/vomiting.<br><br>Follow ASPEN guidelines for prevention of refeeding syndrome (RS) for those at high risk, with special consideration of phosphate and thiamine[2,4].<br><br>Inquire with clients about negative past experiences with food that may affect food acceptance (e.g., foods that caused obstruction, overreliance on formula for weight maintenance or in eating disorder treatment). |
| Promote increased calorie and protein intake during active flare | While in remission, energy and protein requirements for clients with IBD are the same as the general population. However, acute inflammation demands increased energy and protein intake. Research indicates that, during an active flare, caloric needs may start at 30-35kcal/kg/day and protein needs may be 1.2-1.5g/kg/day[2].<br><br>In clients with EDs, detailed discussion of diet may be harmful and encourage rigid tracking of intake. Consider encouraging tolerable protein-rich foods or ONS throughout the day. |
| Increase tolerated fluids for those at risk or showing signs of dehydration | Consider and correct dehydration in IBD clients[2].<br><br>Consider monitoring fluid output and urine sodium monitored in IBD clients with severe diarrhea or a high output jejunostomy or ileostomy[2]. For those same clients and those post colectomy or J-pouch, recommend use of a well-tolerated oral rehydration solution[5,3].<br><br>Reevaluate use of fluids that appear to cause an osmotic reaction, thus worsening diarrhea, bloating, and/or gas. |
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| Recommend a daily multivitamin | All clients with IBD (including those with disordered eating) should take a multivitamin due to increased risk of nutrient deficiencies and malnutrition[5]. Exceptions may include:<br>• IBD clients in endoscopic remission who eat a well-balanced diet<br>• Microscopic colitis clients who eat a well-balanced diet<br>• Clients whose inflammation is restricted to the rectum, eat a well-balanced diet and have no nutrient deficiencies<br><br>Clients with IBD should be screened regularly for deficiencies[2], especially when on restrictive diets. |
| Assess and treat iron deficiency | Since patients with IBD are at an increased risk for iron deficiency, refer any patient presenting with fatigue, bleeding, or signs of active disease for the following labs: CBC, serum ferritin, CRP, and iron panel (or just transferrin saturation)[2,1,6]. Follow the Crohn's & Colitis Foundation of America Anemia Care Pathway based on the lab results[6].<br><br>Oral iron can be used as first line treatment for iron deficiency in patients with mild anemia, inactive disease, and in those who have not previously had issues tolerating oral iron[2].<br><br>Intravenous iron should be used as the first line treatment for iron deficiency in those with active inflammation, those with previous oral iron intolerance, those with hemoglobin levels <10.0 g/dL, and in those requiring erythropoiesis-stimulating agents[2]. |
| Consider supplementation of calcium and vitamin D | Calcium and vitamin D supplementation is indicated in any IBD client avoiding dairy products, taking corticosteroids, with known low bone mineral density, or with known vitamin D deficiency[2]. |
| Consider supplementation of vitamin B12 | Vitamin B12 should be supplemented in those with clinical deficiency (1000ug of vitamin B12 via intramuscular injection every other day for a week then every other month for life) or when ≥20cm of distal ileum is resected in CD patients (1000 ug of vitamin B12 monthly via intramuscular injection)[2]. |
| Consider modifications to fiber type, texture, and | Clients with IBD who present with a symptomatic response to fiber, as well as those with stricturing CD, |

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| amounts | may benefit from adjusting the fibrous foods in their diet[2,7].<br>• Type: Some clients may benefit from minimizing insoluble dietary fiber (e.g., fruit/vegetable skins, popcorn, corn on the cob, cruciferous vegetables, raw nuts, seeds with a tough outer shell, etc.). See Fiber Basics section for more information.<br>• Texture: The smaller and smoother the particle size of the food, the better it is tolerated. For example, raw spinach may not be tolerated but spinach blended into a smoothie may be much better tolerated despite having the same amount of fiber.<br>• Amount: In some cases, slowly increasing the portion of fiber over time can help improve tolerance.<br><br>Consider prophylactic use of psyllium husk or loperamide (e.g., Imodium) to minimize symptoms with the introduction of more roughage in the diet. For stricturing Crohn's clients, discuss the risk of blockages with the client's gastroenterologist and/or surgeon to allow for the most expansive diet possible. |
| Assess for the risk of hyperoxaluria | The following sub-groups of IBD may be at an increased risk for hyperoxaluria[2]:<br>• Intestinal insufficiency related to intestinal failure after multiple intestinal resections, or short bowel syndrome<br>• CD with short bowel syndrome with a colon[1]<br>• Bile acid malabsorption after ileal resection or inflammation of the terminal ileum<br>• SIBO<br>• Disaccharidase deficiency<br>• Malabsorption due to other GI conditions, such as celiac disease<br><br>In recommending an oxalate-modified diet, only recommend swapping out high-oxalate rich foods (e.g., spinach, beets, rhubarb, chocolate, cocoa, carob, peanuts, almonds, bran flakes, >2 cups of tea a day, parsley) for lower oxalate foods. Make sure the client is consuming adequate calcium, or supplements if needed[1]. For individuals that might be triggered by |

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| | dietary restriction, supplementing calcium alone may be the most prudent course of action.<br><br>Consider educating client on lower-fat food options if they are experiencing co-occurring steatorrhea[2]. |
| Assess for the need for the appropriate IBD dietary intervention on an individual basis | ESPEN guidelines do not recommend a specific diet to promote remission in all IBD patients with active disease[2]. Instead, they recommend following an individualized diet with a focus on general healthy eating and avoidance of individual trigger foods[2]. Regardless, some dietary approaches have been studied and may be considered:<br><br>**Exclusive Enteral Nutrition (EEN)**<br><br>EEN involves replacing all intake with formula, typically for 6+ weeks. It is arduous but has been shown to induce remission in children with mild to moderate Crohn's disease[8].<br>• In adults with mild to moderate CD, EEN may be recommended to those who are preoperative or who have fistulas, in order to improve nutritional status, promote closure of fistulas, induce remission, and reduce postsurgical complications[3].<br>• In patients with overlapping eating disorders or disordered eating, this diet will likely not be appropriate.<br>• There's insufficient evidence to recommend use of EEN in UC patients at this time.<br><br>**Partial Enteral Nutrition (PEN)**<br><br>PEN involves supplementing the diet with a nutrient dense formula. In a meta-analysis of mild to moderate CD patients, researchers found the rate of clinical relapse was significantly lower in those receiving PEN (with 400-1800 kcal coming from PEN) compared to those not on PEN[9]. Consistent enteral nutrition should be considered for weight maintenance or weight gain during active IBD if oral intake is insufficient[2].<br><br>**Crohn's Disease Exclusion Diet (CDED)**<br><br>ESPEN guidelines recommend considering this diet for the induction of remission instead of EEN in children with mild to moderate CD[2]. ESPEN guidelines also recommend considering this diet with or without |

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| | PEN for the induction of remission in adults with mild to moderate CD[2]. To learn more about delivering the CDED, visit the Modulife website: https://mymodulife.com/experts/.<br><br>EEN, PEN and CDED are not typically recommended for clients with an active ED. Some clients request dietary interventions due to hesitation about starting immunosuppressant drugs to control their IBD; however, there can be deleterious effects of forgoing medication and implementing restrictive diets, and the risks should be discussed.<br><br>While some other diets have low quality studies suggesting they may help reduce symptoms, ESPEN guidelines don't recommend any of the following diets for the induction or maintenance of remission in UC or CD patients, due to insufficient evidence[2]: Mediterranean diet, Specific Carbohydrate Diet (SCD), modified SCD, inflammatory bowel disease autoimmune diet (IBD-AID), Autoimmune Protocol (AIP), ulcerative colitis exclusion diet (UCED), semi-vegetarian diet, dairy-free diet, gluten-free diet, CD-TREAT, low-sulfur diet, low-emulsifier diet, carrageenan-free diet, low-fat diet, low-meat diet (FACES), food exclusion based on IgG antibodies, CRAFT diet, whole foods diet, 4 SURE diet, GAPS diet, ayurvedic diet. |
| Assess need for parenteral infusions | Consider fluid and electrolyte parenteral infusions in IBD patients with continued high output stomas[2]. |
| Factor 2020 IOIBD dietary guidelines into overall dietary recommendations, when appropriate | The IOIBD 2020 diet guidelines make recommendations to add or limit certain foods depending on diagnosis:<br>• CD: increase fruits and vegetables<br>• UC: increase omega-3 fatty acids (e.g., fatty fish, flaxseed oil, chia, walnuts) and decrease intake of red and/or processed meats<br>• Emerging evidence in animal studies suggest that gluten and certain additives (artificial sweeteners, maltodextrin, carboxymethylcellulose, polysorbate 80, carrageenan, sulfates, titanium dioxide) may be pro-inflammatory<br><br>It may not be appropriate to discuss the IOIBD diet guidelines with certain ED clients, as they may enhance food fears. When choosing to draw from the IOIBD guidelines, explain that even foods that increase |

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| | the risk for active disease can be consumed in moderation; for example, UC patients that consume red meat once a week or less are five times less likely to flare than those who consume it daily[10]. In this case, encouraging consumption of a variety of protein options may decrease their overall red meat intake without promoting harmful dietary restriction. |
| Review common trigger foods | Common trigger foods and beverages in IBD include:<br>• Spicy foods<br>• High-fat foods<br>• Red or processed meat<br>• Foods with additives of concern (see IOIBD 2020 guidelines[10])<br>• Gluten<br>• Concentrated sugars<br>• Artificial sweeteners<br>• FODMAPs<br>• Insoluble fiber (especially larger particle size)<br>• Coffee<br>• Alcohol<br><br>Foods should only be limited if they consistently trigger symptoms and should be replaced with better tolerated alternatives to avoid disruptions to intake. When clients suspect a food trigger, invite curious observation of how they respond to that food rather than eliminating it immediately. |
| Assess and educate patients on perioperative nutrition prior to surgery[11,2,12] | Pre-operative recommendations include:<br><br>**Correct Malnutrition**<br>• Pre-op malnutrition (via Subjective Global Assessment or MUST screening tool) is associated with increased risk for post-op complications and mortality. Clients at risk of malnutrition should receive nutrition treatment (ONS, EN, and/or PN if indicated) for at least 7-14 days to reduce the risk for post-op complications and anastomotic leaks. Encourage the client to sample several brands so they can choose the most palatable option.<br>• Clients should also be screened for nutrient deficiencies and treated accordingly. Preoperatively, choose IV iron for IBD clients with active disease. |

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| | **Support Perioperative Nutrition**<br>• Avoid excessive pre-op fasting; a light meal up to 6 hours pre-op is safe.<br>• Carbohydrate loading via pre-op drinks the evening before and 2 hours before surgery helps to improve pre-op well-being, reduce post-op insulin resistance, decrease protein breakdown, and maintain lean body mass and muscle strength. They may also reduce complications, improve recovery time, and decrease the risk of post-op nausea and vomiting.<br><br>**Support Post-Operative Nutrition**<br>• Encourage immediate resumption of oral intake, as it has been associated with decreased rates of infectious complications and expedited recovery. Encourage eating as early as 4 hours after surgery. Low-residue diet (instead of clear liquids) after colorectal surgery has been associated with less nausea, faster return of bowel function, and a shorter length of hospital stay.<br>• Provide oral nutrition supplements (ONS), along with diet, to reach protein and calorie requirements 7 days post-op (or longer if needed). |
| Consider dietary interventions unique to those with a J-pouch | For UC patients who have undergone Ileal pouch anal anastomosis (IPAA) surgery:<br>• Consider psyllium husk powder or eating starchy foods spread throughout the day to help improve stool consistency[13]<br>• Aim for at least 1.5-3.5 (or more) servings of fruit/day as it may decrease the risk for pouchitis[14,15].<br>• Consider adjusting meal volume, frequency, and timing to reduce BM frequency[13,16].<br>• Recommend regular bone density screenings, as clients with J-pouches are at an increased risk for bone demineralization[13]. |
| Consider dietary interventions unique to those with stricturing obstructing CD | Depending on the severity of the stricture, tolerance to fiber can vary dramatically. Encourage adjustment in texture of fibrous foods and tough meats/seafood rather than restriction, wherever possible. Collaborate with the client's gastroenterologist and/or surgeon to assess risk of blockages to inform fiber |

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| | recommendations and allow the client the most expansive diet possible. |
| Consider dietary interventions unique to those with short bowel syndrome (with a colon) | The energy requirements for those with short bowel syndrome may range from 30-60 kcal/kg/day. Protein needs may range from 1.25-1.5g/kg/day of protein[1]. Encourage consumption of protein-rich foods throughout the day to meet these needs instead of tracking protein intake.<br><br>Recommend a diet high in complex carbohydrates and low in fat and consider supplementing with medium chain triglycerides (MCT) if needed.<br><br>Recommend a low oxalate diet (see above) to prevent nephrolithiasis in this vulnerable population. |
| Consider dietary interventions unique to those with a jejunostomy | Clients with jejunostomies are recommended to consume the same carbohydrate and fat intakes as the general population but may be at risk for excessive sodium loss. In these cases, recommend:<br><br>• Enjoyable high-sodium foods and liberal salt use<br>• Oral rehydration solutions to optimize hydration<br>• Limited hypotonic/hypertonic oral fluids (≤500mL/day)[1] |
| Encourage client to discuss IBD-related medication options with their gastroenterologist | It's important for clients to discuss the risks of medications versus untreated disease with their doctor. Some clients may want to use dietary approaches to manage symptoms and/or inflammation and others may purposely go off their medications to exacerbate disease to encourage more weight loss[17]; however, when an overlapping ED is present, medications, non-diet therapies, and gentle nutrition approaches are the recommended treatment.<br><br>Medication options for IBD include the following[18-20]:<br><br>• Corticosteroids (e.g., prednisone, budesonide, prednisolone)<br>• 5 Aminosalasilic acids/5-ASAs (e.g., mesalamine, balsalazide, sulphasalazine)<br>• Immunomodulators (e.g., Azathioprine, Imurin, 6 mercaptopurine, Methotrexate)<br>• Anti-TNF inhibitors (e.g., Remicade, Humira, Cimzia, Simponi)<br>• Interleukin-23 (e.g., Skyrizi)<br>• Interleukin-1 (e.g., Stelara) |

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| | • Anti-Integrins (e.g., Entyvio)<br>• JAK Inhibitors (e.g., Xeljanz, Rinvoq)<br>• S1P receptors (e.g., Zeposia)<br><br>Medications and substances that may be used to manage symptoms of IBD include:<br><br>• Anti-diarrheal (e.g., Imodium, psyllium husk powder)<br>• Laxatives<br>• Prosecretory agents<br>• Proton pump inhibitors<br>• Antidepressants (e.g., tricyclic antidepressants, selective serotonin reuptake inhibitors)<br>• Antispasmodics (e.g., dicyclomine, peppermint oil)<br>• Neuropathic-directed agents<br>• Antibiotics (e.g., rifaximin, ciprofloxicin, metronidazole)<br>• Bile-acid sequestrants (e.g., cholestyramine)<br>• Digestive enzymes (e.g., lactase, alpha-galactosidase)<br>• Cannabis/cannabinoids: these compounds do not appear to impact clinical remission or endoscopic remission but may improve symptoms and quality of life[21].<br>• Curcumin: may help induce remission in those with mild to moderate UC and CD. Most research has been conducted using 500-3000mg per day, with the higher doses typically split in two doses a day. Supplementation should be accompanied by serial monitoring of liver functioning tests as there have been reports of liver toxicity[1,22]. |
| Discuss food/nutrient and medication interactions with client | Nutrient considerations for specific medications include[1,2,5,23,24]:<br><br>• Zeposia (Ozanimod): this medication is impacted by tyramine consumption. Educate the client on the tyramine content in food, as this will help avoid deleterious effects[25]<br>• Corticosteroids: supplement with 500mg calcium and 400 IU vitamin D twice daily<br>• Sulfasalazine, methotrexate: supplement with 1mg folic acid daily |

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| | • PPIs: may interfere with absorption of iron, vitamin B12, vitamin C, vitamin D, possibly magnesium<br>• Cholestyramine: can interfere with absorption of fat-soluble vitamins, iron, vitamin B12<br>• Psyllium husk: can interfere with absorption of medications and should be spaced at least one hour apart |
| Recommend probiotic in certain cases | Clients with chronic pouchitis that respond to antibiotics may benefit from supplementing with Visbiome Extra Strength probiotic to reduce risk of recurrence[26,1]. Probiotics are not currently recommended for primary prevention of pouchitis or for the treatment of infrequent pouchitis[26].<br><br>Probiotics should not be recommended in CD clients for the induction or maintenance of remission[2].<br><br>Mixed evidence exists for the use of probiotics for clients with mild to moderate UC patients; the 2023 ESPEN Guideline on Clinical Nutrition in Inflammatory Bowel Disease suggests certain probiotics or probiotic-containing preparations may be considered if 5-Aminosalicylic Acid standard therapy is not tolerated[2]. |
| Encourage incorporation of resistance exercise, if appropriate | Resistance training can be recommended to improve muscle mass and risk of osteoporosis[2]. General movement may reduce symptoms and improve overall quality of life[27].<br><br>Any exercise recommendation should be secondary to ED recovery and accessibility concerns. |
| Address body size concerns in clients | Pursuit of weight loss is highly discouraged for clients with active IBD per ESPEN guidelines[2]. A 2017 meta-analysis found that people in larger bodies have a less severe disease course[28], and a 2022 study found that being in a larger body was not associated with an increased risk for hospitalization, IBD-related surgery, or serious infections[29]. |
| Refer to GI psychologist for adjunct therapy | GI psychology intervention may help reduce the client's focus on food and offer benefits in its own right:<br><br>Gut-directed hypnotherapy may improve IBD-related inflammation and quality of life, separate from any improvement in functional symptoms. Refer to GI |

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| | psychologist or app-based treatment program as adjunctive therapy[30-32].<br><br>Cognitive behavioral therapy appears to improve overall IBD quality of life[33].<br><br>Acceptance and commitment therapy may reduce stress and improve overall QOL[34].<br><br>Mindfulness-based therapies may improve inflammatory markers, reduce depression scores, and improve quality of life[35,36]. |
| Refer to gynecologist if needed | Individuals who menstruate may report worsening of symptoms during menstruation. Initiating birth control or ruling out other conditions (e.g., endometriosis) may help to decrease symptoms and reduce risk of unnecessary dietary restrictions[37]. |

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## Irritable Bowel Syndrome (IBS)

| Nutrition Intervention | Considerations |
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| Adjust portion sizes and timing of food consumption | The gastrocolic reflex (GCR) stimulates colonic motility in response to eating and is more sensitive to high-fat and high-volume meals[1,2]. As such, people with IBS-D may benefit from consuming smaller, more frequent meals (e.g., every 3-4 hours) as opposed to larger meals.<br><br>In patients with IBS-C, larger volumes of food may help send a stronger signal to the colon to empty and promote regular laxation[3].<br><br>These recommendations should be secondary to implementation of a nutritionally adequate meal plan for individuals with eating disorders. |
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| Encourage consumption of a variety of plant-based foods | Consuming a wide range of fiber-containing foods promote diversity of the gut microbiota[4] and can be beneficial for DGBIs.<br><br>Patients with IBS and EDs may be fearful of certain foods and food groups that they associate with symptoms. Help clients understand what foods are most likely to be well tolerated and expand dietary variety from that starting point. |
| Adjust the texture, type, and amount of fiber in the diet | People with IBS may suffer from inadequate or excessive fiber in the diet. A daily intake of 28-35 grams, as recommended to the general population, should be sufficient[5].<br><br>While both insoluble and soluble fiber contribute to gut health, many clients with IBS may benefit from emphasizing soluble fiber sources (e.g., oats, squash, root vegetables, chia seeds, etc.) to regulate bowel movements.<br><br>When clients struggle with gas and/or bloating, it may be appropriate to prioritize less fermentable (i.e., low-FODMAP) fiber sources[6]. However, it is important to consider the impact of restricting certain foods in ED patients.<br><br>In some cases, fiber supplementation may be a helpful strategy. See Non-Diet Intervention section on fiber supplementation for more information.<br><br>In IBS-D, increasing the amount of soluble fiber in the diet may improve the texture of stools and transit time, reducing symptoms of urgency. |
| Recommend consumption of two kiwi per day or a serving of prunes | Several studies involving kiwis demonstrate a positive effect on complete spontaneous bowel movements[7,8]. At least one study shows the efficacy of kiwis is not statistically different from that of prunes or psyllium. Prunes are generally well tolerated but also contain sorbitol, which may trigger gas and/or diarrhea in sensitive clients. |
| Educate client on physiological effects of trendy food additives | "Diet" foods and other products often contain additives that can trigger IBS symptoms. For example, sugar-free, low-calorie, and/or keto foods often contain poorly digested sugar alcohols (e.g., sorbitol) or added fibers (e.g., inulin, chicory root). |
| Determine appropriateness of using the low-FODMAP | FODMAPs are types of short chain carbohydrates that are poorly absorbed in the small intestine, which can |

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| diet | trigger distention of the intestines by drawing in excess water and producing gas via fermentation by the gut bacteria. This intervention is designed to reduce FODMAP-containing foods in the diet to alleviate symptoms associated with IBS[6].<br><br>The low-FODMAP diet is a 3-phase approach that involves restriction or elimination of FODMAPs, systematic reintroduction, and personalization. The goal is to identify dietary triggers of symptoms and liberalize the diet as much as possible while maintaining symptom improvement[6].<br><br>Contraindications and limitations for the low-FODMAP diet include:<br>• IBS-C: The low-FODMAP diet may not be helpful for improving stool consistency in IBS-C, as it reduces stool water content in the colon, leading to stool hardness. In addition, the diet does not increase gut transit time[9].<br>• IBS-D / IBS-M: Research shows that the low-FODMAP diet is only effective for up to 60-80% of patients[9,10], meaning some patients may require other interventions (e.g., medication, behavioral therapies, etc.).<br>• EDs: Because the low-FODMAP diet is restrictive in nature, the full version is not appropriate for individuals with active EDs. |
| Consider alternative approaches to using tenets of the low-FODMAP diet if a full elimination is contraindicated | Modified approaches to the low-FODMAP diet may be more appropriate in clients with EDs[6].<br><br>A "FODMAP gentle" approach only eliminates the most common trigger foods that are very high in FODMAPs. Foods commonly removed using this approach include milk, yogurt, wheat, rye, garlic, onion, leeks, cauliflower, mushrooms, apples, pears, watermelon, stone fruits, dried fruits, and legumes[11].<br><br>If specific high-FODMAP foods appear frequently in the client's diet and are suspected triggers for symptoms, consider a trial elimination of just these foods (also known as "cherry picking"). Recommend substitutions for these foods to ensure adequate energy intake[9]. |
| Look for possible exacerbation of symptoms | Clients with IBS may experience an uptick in symptoms or discomfort in response to the following food types: |

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| in response to non-fermentable trigger foods | • High-fat foods<br>• High-insoluble fiber/high-volume foods (e.g., popcorn, entree salads)<br>• Stimulants (i.e., caffeine)<br>• Alcohol<br>• Gas promoters (e.g., carbonated beverages, gum, drinking from straw)[12]<br>• Spicy foods<br><br>Work to only limit foods that reliably trigger symptoms and suggest appropriate substitutions for problematic foods they enjoy. |
| Counsel clients on maintaining adequate hydration | Drinking enough fluid is critical for the function of the digestive tract. In people with IBS-C, increasing fluid intake may help soften stool and relieve symptoms. In people with IBS-D, extra fluids and electrolytes may be beneficial if they are experiencing regular bouts of diarrhea. |

| Non-Diet Intervention | Considerations |
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| Refer to GI psychologist or relevant app to improve gut-brain interaction | Various therapeutic modalities, including CBT and gut-directed hypnotherapy, may improve visceral hypersensitivity and overall QOL[13-16]. Using non-diet therapies can be protective for people with EDs, as they reduce the focus on food. |
| Consider use of a fiber supplement to improve stool form | Supplementation may support adequate fiber intake in those with reduced intake of fiber or those with irregular stool form. These supplements are often taken at night according to package instructions to improve morning bowel movements. Specific subtypes of IBS may benefit from different types of fiber[17,18]:<br>• IBS-D<br>  ○ Psyllium husk<br>  ○ Methylcellulose<br>  ○ Wheat dextrin<br>  ○ Partially hydrolyzed guar gum/PHGG[19]<br>  ○ Acacia fiber<br>• IBS-M<br>  ○ Psyllium husk |

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| | • IBS-C<br>  ◦ Calcium polycarbophil<br>  ◦ Psyllium husk |
| Consider use of an enterically coated peppermint oil supplement | Peppermint oil acts as a natural antispasmodic and analgesic that can help relax the smooth muscle tissue of the gut and reduce abdominal pain. This product should be dosed 30-90 minutes before meals[20,21]. |
| Consider L-glutamine supplementation in the case of suspected post-infectious IBS | L-glutamine is an amino acid that fuels enterocyte proliferation. While the data supporting L-glutamine supplementation is limited, it is a safe intervention that may offer benefit. The dosing reported across several studies was 5 grams three times per day for 6-8 weeks[22,23]. |
| Consider the use of over-the-counter laxatives to increase frequency of bowel movements | Use of laxatives may be considered in IBS-C. There are two types[3,24]:<br>• Osmotic laxatives draw water into the colon, softening stools and decreasing transit time (e.g., high-dose magnesium, polyethylene glycol).<br>• Stimulant laxatives increase contractions of the inner lining of the colon, improving transit time (e.g., bisacodyl, senna).<br>Inquire about the client's history with stimulant laxatives, including past misuse, to inform recommendations. |
| Educate client on optimal toileting position | Clients experiencing incomplete evacuation may benefit from defecating while in a squatted position (with their feet propped up so that the knees are higher than the hips)[25]. This can be achieved through use of a Squatty Potty or any similar household object (e.g., small step stool, toilet paper roll under each foot, stack of books, yoga blocks, etc.)[26]. |
| Consider the use of targeted digestive enzyme supplements | Supplementation of specific digestive enzymes may help break down certain types of FODMAPs, making them easier to tolerate. Some of these products are well-studied and others have limited research backing them (i.e., inulinase/fructan hydrolase, glucose isomerase/xylose isomerase). Collectively, they show clinical promise and are low-risk interventions for potentially relieving gas and bloating associated with FODMAP intake. They may also help liberalize the diet when ED clients have known FODMAP triggers. |
| | Digestive enzymes should be taken at the start of a |

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| | meal. The active ingredients in the digestive enzyme supplements determine when they are appropriate to use[27-29].<br><br>For example:<br>• Inulinase/fructan hydrolase covers consumption of fructans (animal studies only)<br>• Alpha-galactosidase covers consumption of galactooligosaccharides (GOS)/galactans (supported by research in human subjects)<br>• Lactase covers consumption of lactose (supported by research in human subjects)<br>• Glucose isomerase or xylose isomerase covers consumption of fructose (no human or animal research studies at this time)<br><br>*Some digestive enzyme supplements contain other high-FODMAP ingredients (e.g., mannitol, sorbitol), which may worsen symptoms. |
| Refer to physician for medical prescription management | Encourage clients to discuss medication options for IBS management with their doctor. Common medications include:<br>• Antispasmodics (e.g., dicyclomine, hyoscyamine)<br>• Antidiarrheal medications (e.g., loperamide/Imodium, diphenoxylate-atropine/Lomotil)<br>• Antidepressants (e.g., tricyclic antidepressants, serotonin-norepinephrine reuptake inhibitors)<br>• Secretagogues (e.g., lubiprostone/Amitiza, linaclotide/Linzess, plecanatide/Trulance)<br>• Sodium-hydrogen exchange 3 inhibitor (e.g., tenapanor/IBSrela)<br>• Serotonergic agents (e.g., alosetron/Lotronex, tegaserod/Zelnorm)<br>• Mixed opioid (e.g., eluxadoline/Viberzi)<br>• Antibiotics (e.g., rifaximin/Xifaxan)<br>• Vibrating capsule (e.g., Vibrant[30]) |

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## Lactose Intolerance

| Nutrition Intervention | Consideration |
|---|---|
| Assess tolerance to lactose-containing foods and modify diet as needed | Higher lactose foods include cow's milk, yogurt, cottage cheese, ice cream, and certain cheeses (e.g., American cheese, ricotta)[1].<br><br>Individual tolerance to lactose may vary. To mitigate GI symptoms, higher lactose dairy products may need to be reduced or removed from the diet. This may be approached by trialing portions of lactose-containing foods using a phased approach (e.g., small → medium → large portions) to determine how much lactose the client can tolerate or by simply swapping for lactose-removed dairy products.<br><br>When recommending acceptable food substitutes, consider whether a reduction in dairy consumption will contribute to protein, calcium, or vitamin D gaps in the diet. |

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| Consider lactase enzyme supplementation | Lactase supplementation may help reduce or eliminate symptoms associated with lactose consumption. Take 9000FCC immediately prior to meals and snacks containing lactose[2]. |
| Consider calcium and vitamin D supplementation | Clients who have been avoiding dairy may not be consuming enough calcium and vitamin D, putting them at an increased risk for bone fractures and osteoporosis. This risk may be higher in those with an ED. Consider supplementation of calcium and vitamin D if necessary. |
| Refer to physician to assess bone health | Clients who have been avoiding dairy may be at an increased risk for bone fractures and osteoporosis, especially with a comorbid ED. Consider referring to a physician for evaluation of bone density. |

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## Metabolic Dysfunction-Associated Steatotic Liver Disease (MASLD) (previously Non-Alcoholic Fatty Liver Disease)

| Nutrition Intervention | Considerations |
|---|---|
| Encourage behavior modification instead of weight loss efforts | Weight loss is often recommended for improving MASLD, but it is not an appropriate recommendation for people who struggle with their relationship with food and body. Various health-promoting behaviors can lead to improvement and should be prioritized[1]. Improvements following weight loss have yet to be established as directly caused by changes in fat mass (as compared to increases in fruit/vegetable consumption, physical activity, or other common strategies employed in the pursuit of weight loss). See Ch. 4 for a more detailed discussion of the limitations of weight loss research. |
| Encourage refined | Proper blood sugar management may minimize |

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| carbohydrates to be accompanied by other macronutrients or fiber | triglyceride formation and subsequent liver deposits. Meals and snacks containing refined carbohydrates are best paired with other foods (i.e., protein, fat, fiber sources) that can buffer the blood sugar effect. |
| Consider a plant-forward, culturally appropriate diet (e.g., Mediterranean-style diet) | These dietary patterns prioritize plant-based foods, lean proteins, whole grains, and unsaturated fats[2]. |
| Recommend minimization of alcohol intake | Alcohol is a known exacerbator of MASLD and intake of it should be reduced as much as is acceptable by the client. |
| Encourage liberal coffee consumption, if enjoyed | Coffee consumption is inversely associated with GGT, liver function tests, and liver disease severity. The relationship is particularly strong at an intake of 2+ cups of caffeinated coffee per day[3]. |

| Non-Diet Intervention | Considerations |
|---|---|
| Advocate for regular physical activity | Physical activity helps manage blood sugar, sensitize the body to insulin and mobilize triglycerides, and therefore has MASLD-specific benefits that extend beyond general wellbeing. |
| Consider omega-3 supplementation | Omega-3 fatty acid supplementation may improve triglyceride, total cholesterol, and HDL levels in patients with fatty liver[4]. A dose of 2-4 grams per day is most often observed in the research. |
| Recommend screening for low vitamin D | Vitamin D supplementation has been shown to be beneficial in adults with NAFLD* who do not have co-occurring diabetes. Amounts ranging from 1,000 IU per day to 50,0000 IU weekly appear to improve insulin resistance, glucose homeostasis, and ALT levels[5].<br><br>*Research at present has been conducted using the previous nomenclature but appears applicable to people with metabolic associated steatotic liver disease (MASLD), without diabetes. |
| Consider vitamin E supplementation in advanced forms of MASLD | Vitamin E supplementation of 800 units per day is supported for people with non-alcoholic steatotic hepatitis (NASH)* who do not have diabetes[6,7].<br><br>*Research at present has been conducted using the previous nomenclature but appears applicable to |

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| | people with metabolic associated steatohepatitis (MASH), without diabetes. |

## References: MASLD


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## Small Intestinal Bacterial Overgrowth (SIBO) & Intestinal Methanogen Overgrowth (IMO)

| Nutrition Intervention | Considerations |
|---|---|
| Avoid recommending unnecessarily restrictive diets | At time of publication, there is not robust evidence to recommend any particular dietary intervention for SIBO, including the following:<br>• Low-FODMAP diet<br>• Specific Carbohydrate Diet (SCD)<br>• Low fermentation diet<br>• Biphasic diet |

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| | • Gut and Psychology Syndrome (GAPS) diet<br>• Elemental diet (this diet has one weak study supporting it, but the evidence has not been replicated, and this approach is extraordinarily restrictive[1]).<br><br>For people with eating disorders, the potential and undemonstrated benefits of these kinds of diets are not worth the risk and harm of disrupted, and possibly inadequate, intake. |
| Tailor dietary recommendations to reported symptoms | The impact of diet on SIBO/IMO-related symptoms is varied. Consider the use of a food/symptom log to identify correlations between symptoms and specific types of foods. Some noted examples include:<br><br>FODMAPs: These foods are best minimized using a "gentle" or "cherry-picking" approach (See IBS Dietary Intervention section). Clients' symptom reports may indicate only a single FODMAP category. If a client has already implemented a low-FODMAP diet, moving into the reintroduction phase may be less restrictive than their current eating pattern.<br><br>Fat: SIBO may cause fat malabsorption due to a reduction in bile salts[2]. As such, fat tolerance will vary among individuals. Work with the client to spread fat intake out over the course of the day and/or ensure presence of all macronutrients at each meal.<br><br>Fiber: Due to altered bowel habits related to SIBO, some clients may benefit from modifying the texture/consistency of fiber-containing foods (via peeling, deseeding, cooking to fork-tender consistency, etc.).<br><br>For clients with eating disorders, any dietary restrictions should be made thoughtfully and abandoned if they may contribute to setbacks for the individual's mental health. |
| Aim to space meals 2+ hours apart | Meal spacing allows for activation of the migrating motor complex (MMC), which is a motility wave that sweeps bacteria toward the colon. Interference with regular MMCs may promote abnormal bacteria load in the small intestine, contributing to the development of SIBO/IMO. Client should be encouraged to have intentional meal and snack times rather than grazing throughout the day[3,4]. |

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| | Clients with eating disorders should prioritize their weight restoration meal plan and/or responding to hunger cues over this recommendation. |

| Non-Diet Interventions | Considerations |
|---|---|
| Monitor for common SIBO-related nutrient deficiencies: folate, vitamin B12, fat-soluble vitamins (A, D, E, K) and iron | If possible, consider filling nutrient gaps with food sources. Otherwise, supplement as needed.<br><br>Be mindful of recommending iron supplementation, as this can worsen constipation.<br><br>If fat-soluble vitamin status is not repleted using traditional supplements, consider recommending a water-soluble vitamin A/D/E/K formulation[5]. |
| Work on habits that promote bowel regularity | A bowel regimen should be established and prioritized, especially in those who likely developed SIBO as a result of slow motility. This may include osmotic laxatives, proper toileting positioning, diaphragmatic breathing, a full breakfast, a warm beverage in the morning, bowel massage, setting time aside to sit on the toilet, and others (see Chronic Constipation section)[6]. |
| Refer to physician for antibiotic treatment | Antibiotics are the only established treatment for SIBO/IMO and must be prescribed by a physician. Typical courses include rifaximin (Xifaxan) for SIBO or rifaximin + neomycin for IMO, although alternate options exist. Some clients may require multiple courses of treatment[6]. |
| Suggest the use of comfortable clothing | Tight clothing may exacerbate sensations of pain, bloating, and discomfort. |
| Discourage the use of “alternative” treatment methods | As there are limited options for SIBO/IMO treatment, a cottage industry of supplements has popped up purporting to treat the condition and has been bolstered by a poorly designed 2014 study from Johns Hopkins[7]. Antimicrobial supplements, herbal “protocols,” and probiotics are not recommended at this time, as there is no significant evidence to support their use. |
| Manage the underlying condition that may have predisposed the client to develop overgrowth | Common predisposing factors and/or associated conditions include IBS, abdominal surgery, PPI use, frequent antibiotic use, motility disorders, and unmanaged GI conditions that can ultimately impact motility. If it is identified that any of these associated |

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| | conditions are playing a role, the condition should be managed to reduce the risk of SIBO/IMO recurrence[4]. |

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CHAPTER 10

# Monitoring and Evaluation

Nutrition monitoring and evaluation are necessary to determine whether and how much progress has been made during the nutrition intervention. During this step, the RDN reassesses appropriateness of the interventions and decides between continuation, discontinuation, or modification of the interventions. This sets the stage for the Nutrition Care Process to repeat itself throughout future reassessments.

While nutrition monitoring and evaluation are imperative to practice, limitations certainly exist within the co-occurrence of EDs and GI disorders. For example, in the absence of standardized screening tools and years of experience, providers are reliant on subjective clinical judgment, which may be ill-informed or biased. Siloed healthcare also inhibits interdisciplinary collaboration that could impact medical nutrition therapy effectiveness.

## The RDN's Role

Continuous care involves reassessment across various spheres of client health. The RDN is equipped to evaluate adherence to the nutrition plan, indicators of nutrition status, subjective gastrointestinal symptoms, disease prognosis, disordered eating symptoms, co-occurring conditions, and quality of life. The RDN may be informed by client interviews, anthropometric measures and vital signs, objective GI- and ED-related laboratory tests and

findings, and reports from collaborating providers. Changes relative to baseline may be indicative of effectiveness of intervention and/or progression of disease state.

## Best Care Practices

While the ADIME steps of the Nutrition Care Process provide a foundation for evidence-based practice, there are additional considerations that will enhance client care and outcomes.

### Communicate With Other Providers

Different providers within the care team may be privy to different sets of information. Maintaining a communicative relationship with the client's other care team members can be extremely beneficial to the client; it may prevent them from having to repeat information leading to streamlined care, reduce the likelihood of relevant information slipping through the cracks, and help the client feel supported.

### Elicit Feedback from the Client

When clinically appropriate, be transparent with the client about progress or lack thereof in nutrition goals and elicit feedback from the patient about their own perception of progress and effectiveness of interventions. Having an opportunity for feedback between the RDN and client can strengthen the therapeutic relationship and offer a window into the client's current level of motivation. The RDN is also encouraged to observe nonverbal clues such as "people-pleasing" tendencies, body language, and emotional cues.

### Connect Client with Additional Support, Where Possible

The RDN is often in a position to identify gaps in services that impact ED recovery, disease prognosis, and/or adherence to

nutritional recommendations. To the extent possible, the RDN is encouraged to assist the client in accessing necessary forms of support. Examples include referrals to providers with a demonstrated understanding of the client's identities, government benefits (e.g., SNAP, WIC, unemployment insurance), meal services, and group support.

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SECTION III

# Resources for the Registered Dietitian Nutritionist

## Combined ED-GI Resources

*Dietitians in Gluten and Gastrointestinal Disorders (DIGID)*

https://www.dmntdpg.org/subunits/digid
Subgroup within the Dietitians in Medical Nutrition Therapy (DMNT) dietetic practice group (DPG) of the Academy of Nutrition and Dietetics (AND); for members only

*Identification and Management of Disordered Eating in Gastrointestinal Disorders*

https://www.monashfodmap.com/online-training/online-dietitian-courses/
Online course hosted by Monash University

*Digestive Disorders & Eating Disorders: A Complicated Mix*

https://marcird.teachable.com/p/dd-ed-training
Online course developed by Marci Evans, MS, CEDS-S, LDN and Lauren Adler Dear, MS, RDN

## Gastrointestinal-Specific Resources

Apps

*Fig*

https://foodisgood.com/
Allows users to input their dietary restrictions then scan the barcode of food items to quickly determine if they align with their dietary needs.

*Find Me Gluten Free*

https://www.findmeglutenfree.com/
Allows users to browse the area for gluten free restaurants and read reviews from other users.

*Monash University Low FODMAP App*

https://www.monashfodmap.com/ibs-central/i-have-ibs/get-the-app/
Using Monash University's FODMAP-tested food database, the app allows users to search for specific food items, then indicates whether they are low, moderate, or high in FODMAPs based on a particular portion size.

*Nerva*

https://try.nervaibs.com/hypnotherapy-nerva/
A self-directed IBS management app utilizing a six-week gut-directed hypnotherapy program. In addition to hypnotherapy, the app also includes interactive content, advice, and an in-app chat feature.

*MySymptoms*

https://www.mysymptoms.net/
Allows users to log and analyze foods, medications, sleep, stress, exercise, and symptoms to help identify potential IBS triggers. The app also allows users to share their results with their providers.

*Spoonful*

https://spoonfulapp.com/
Users scan the barcode of packaged food items and receive information about whether it aligns with the dietary restrictions or diet they follow.

*Toilet Finder*

http://toiletfinder.net/
Using the location feature of a phone, the app indicates nearby, free public toilets. The app will also find toilets that are handicap accessible.

*Trellus*

https://trellushealth.com/
A virtual program designed for Crohn's disease, ulcerative colitis, and IBS management allowing users to review self-paced modules, work with a multidisciplinary GI team, and track progress. The app utilizes a holistic approach and focuses on the mind-body connection, nutrition, resilience, and social support.

*We Can't Wait (CCF)*

https://www.crohnscolitisfoundation.org/patientsandcaregivers/wecantwait
Designed for patients with IBD, this app allows users who are in urgent need of a restroom to find one nearby. Restroom availability is identified through crowdsourcing and businesses/establishments who are understanding of the struggles of IBD.

## Books

*The Bloated Belly Whisperer by Tamara Duker Freuman, MS, RD, CDN*

Freuman TD. *The Bloated Belly Whisperer: A Nutritionist's Ultimate Guide to Beating Bloat and Improving Digestive Wellness*. New York: St. Martin's Press; 2020.
https://us.macmillan.com/books/9781250195272/thebloatedbellywhisperer
Resource for patients and practitioners that discusses common etiologies of bloating and evidence-based management strategies

### *Regular by Tamara Duker Freuman, MS, RD, CDN*

Freuman TD. *Regular: The Ultimate Guide to Taming Unruly Bowels and Achieving Inner Peace*. New York: Hachette Go; 2023.
https://www.hachettebookgroup.com/titles/tamara-duker-freuman-ms-rd-cdn/regular/9780306830785/?lens=hachette-go
Resource for patients and practitioners that discusses common conditions that impact bowel regularity and evidence-based management strategies

### *Mind Your Gut by Kate Scarlata, MPH, RDN and Megan Riehl, PsyD*

Scarlata K, Riehl M. *Mind Your Gut: The Science-Based Whole-Body Guide to Living Well with IBS*. New York:Hachette Go; 2024.
https://www.hachettebookgroup.com/titles/kate-scarlata/mind-your-gut/9780306832338/
Evidence-based resource for patients and practitioners that discusses IBS management strategies

## Organizations & Conferences

### *American Gastroenterological Association (AGA)*

https://gastro.org/
Organization representing gastrointestinal medical professionals and the journal, *Gastroenterology*

*American College of Gastroenterology (ACG)*

https://gi.org/
Professional organization representing GI clinicians and support staff and the *American Journal of Gastroenterology*

*FOOD: The Main Course*

https://www.foodthemaincourse.com/
Annual conference for gastrointestinal nutrition hosted of the University of Michigan Medicine

*American Society for Parenteral and Enteral Nutrition (ASPEN)*

https://www.nutritioncare.org/
Professional organization that represents clinicians in nutrition support and defines clinical criteria for malnutrition

*Crohn's and Colitis Foundation*

https://www.crohnscolitisfoundation.org/
Non-profit dedicated to funding research and improving quality of life for individuals living with IBD

*DIGID Digestive Disease Nutrition Series*

https://www.eatrightstore.org/dpg-and-mig-products/dmnt/2023-digestive-diseases-nutrition-series
Biannual webinar series hosted by the DIGID subgroup of the DMNT DPG

*Digestive Disease Week*

https://ddw.org/
Annual GI conference focused on emerging research and hosted by the AGA

*Crohn's and Colitis Congress*

*https://crohnscolitiscongress.org*
Annual conference focused on IBD hosted by the Crohn's and Colitis Foundation and the AGA

# Eating Disorder-Specific Resources

## Apps

*Blind Weight Scale*

*https://blindweight.com/products/blind-weight-scale*
Allows patients to do blind weights and share the results with their providers. There is no subscription fee, only the cost of the scale.

*MyClearStep*

*https://www.myclearstep.com/*
Eating disorder recovery app that allows the treatment team to remotely monitor patients' weight and blood pressure by pairing MyClearStep numberless devices with the app.

*Recovery Record*

*https://www.recoveryrecord.com/*
An eating disorder recovery app allowing patients to share photos and descriptions of their meals and snacks with their treatment team, without calorie or nutrient tracking. The app also features meal plans, skill development, and messaging between the patient and treatment team.

## Groups/Organizations

*Academy for Eating Disorders (AED)*

*https://www.aedweb.org/home*

Organization dedicated to eating disorders research, education, treatment, and prevention

### *Alliance for Eating Disorders Awareness*

*https://www.allianceforeatingdisorders.com/*
National non-profit organization that provides eating disorder-related outreach, education, early intervention, and advocacy

### *American Psychiatric Association*

*https://www.psychiatry.org/*
Organization of psychiatrists who provide information and resources on various disorders, including publication of the Diagnostic and Statistical Manual of Mental Disorder, 5th edition (DSM 5)

### *American Psychological Association*

*https://www.apa.org/*
Professional organization encouraging the development of psychology by promoting research and application of findings and establishing standards for the field

### *Australia & New Zealand Academy for Eating Disorders (ANZAED)*

*https://www.anzaed.org.au/*
Organization that supports ED professionals in prevention, treatment, and research

### *Behavioral Health Nutrition Dietetic Practice Group (BHN)*

*https://www.bhndpg.org/*
DPG within the AND focused on substance use disorders, eating disorders, intellectual and developmental disabilities, and mental health conditions

*Eating Disorder Foundation*

*https://eatingdisorderfoundation.org/*

Nonprofit organization supporting education, support, and advocacy efforts for people with EDs, their friends, family, and allies

*Eating Disorder Registered Dietitians and Professionals (EDRDPro)*

*https://edrdpro.com/*

Membership-based organization providing online training and education in ED-related topics

*Eating Disorders Coalition*

https://www.eatingdisorderscoalition.org/

Nonprofit organization devoted to helping people with eating disorders and their supports via education and advocacy

*FEDUP Collective (Fighting Eating Disorders in Underrepresented Populations)*

*https://fedupcollective.org/*

Nonprofit organization that provides eating disorder recovery support groups and a vetted directory of practitioners who work with marginalized communities, including LGBTQIA+, intersex, and gender diverse people

*International Federation of Eating Disorder Dietitians (IFEDD)*

*http://www.eddietitians.com/*

Professional organization of RDNs that offers a community listserv, conducts eating disorder research, and advocates for access to eating disorder treatment and care

*Multi-Service Eating Disorders Association (MEDA)*

*https://www.medainc.org/*
Nonprofit organization focused on training professionals, expanding access to treatment, and advocating for equitable policy related to eating disorders

*National Association of Anorexia Nervosa and Associated Disorders (ANAD)*

*https://anad.org/*
Nonprofit organization that provides support, raises awareness, and advocates for the prevention of eating disorders

*Project HEAL*

*https://www.theprojectheal.org/*
Nonprofit organization that offers financial assistance, help navigating insurance, and access to free clinical assessment for those looking for eating disorder recovery resources

## Books

*Academy's Pocket Guide to Eating Disorders, 2nd edition by Jessica Setnick, MS, RD, CEDRD-S*

https://www.eatrightstore.org/product-type/pocket-guides/pocket-guide-to-eating-disorders-second-edition
Miniguide for RDNs encountering individuals with dysfunctional eating behaviors

*Fat Talk*

Sole-Smith V. *Fat Talk: Parenting in the Age of Diet Culture*. New York, NY: Henry Holt and Co; 2023.
https://us.macmillan.com/books/9781250831217/fattalk

This book shares parenting strategies for navigating the body shaming and fatphobia present in society.

### *Intuitive Eating: An Anti-Diet Revolutionary Approach, 4th edition*

Tribole E, Resch E. *Intuitive Eating: An Anti-Diet Revolutionary Approach*. 4th ed. New York, NY: St. Martin's Press; 2020.
https://www.intuitiveeating.org/
This formative book provides readers with steps to move away from diet culture influences and rebuilding a healthy relationship with food and body; the authors developed an Intuitive Eating Counselor certification process for providers

### *Intuitive Eating Workbook*

Tribole E, Resch E. *The Intuitive Eating Workbook: Ten Principles for Nourishing a Healthy Relationship with Food*. Oakland, CA: New Harbinger Publications; 2017.
https://www.newharbinger.com/9781626256224/the-intuitive-eating-workbook/
This workbook created by the authors who developed the Intuitive Eating Counselor certification process takes readers through the ten principles of intuitive eating.

### *Nutrition Counseling in the Treatment of Eating Disorders, 2nd edition*

Herrin M, Larkin M. Nutrition Counseling in the Treatment of Eating Disorders. 2nd ed. New York, NY: Routledge; 2013.
https://www.routledge.com/Nutrition-Counseling-in-the-Treatment-of-Eating-Disorders/Herrin-Larkin/p/book/9780415642576
This book highlights research-based approaches and clinically refined tools for managing food-related issues, including bingeing, purging, excessive exercise, and weight restoration

## *Sick Enough: A Guide to the Medical Complications of Eating Disorders*

Gaudiani JL. *Sick Enough: A Guide to the Medical Complications of Eating Disorders*. Routledge; 2018.
https://www.sickenough.com/
A primer on the medical issues that can arise throughout the course of an eating disorder

## *The Body is Not An Apology*

Taylor SR. *The Body Is Not an Apology: The Power of Radical Self-Love*. Berrett-Koehler Publishers; 2021.
https://www.sonyareneetaylor.com/books/the-body-is-not-an-apology-the-power-of-radical-self-love-h2x6p
A book containing reflective prompts to encourage readers to divest from the "body hierarchy" that reinforces various forms of supremacy.

## *The Body is Not an Apology Workbook*

Taylor SR. *Your Body Is Not an Apology Workbook: Tools for Living Radical Self-Love*. Oakland, CA: Berrett-Koehler Publishers; 2021.
https://penguinrandomhousehighereducation.com/book/?isbn=9781523091164
This workbook allows readers to put the ideas in *The Body Is Not an Apology* into practice.

## *How to Raise an Intuitive Eater*

Brooks S., Severson A. *How to Raise an Intuitive Eater: Raising the Next Generation with Food and Body Confidence*. New York, NY: St. Martin's Essentials; 2022.
https://us.macmillan.com/books/9781250786609/howtoraiseanintuitiveeater
This book focuses on how to create an environment for children centered around intuitive eating and teaches parents how to raise

children to have a healthy relationship with food.

## Eating Disorder Screening Tools

Binge Eating Scale (BES): https://psychology-tools.com/test/binge-eating-scale

Eating Attitudes Test 26 Item (EAT-26): https://psychology-tools.com/test/eat-26

Ellyn Satter Eating Competence Inventory 2.0 (ECSI 2.0): https://www.ellynsatterinstitute.org/ecsi-2-0-sdor-2-6y/

Eating Disorder Examination Questionnaire (EDE-Q): https://www.corc.uk.net/outcome-experience-measures/eating-disorder-examination-questionnaire-ede-q/

Eating Disorder Examination Questionnaire Short (EDE-QS): https://www.corc.uk.net/media/3339/ede-qs.pdf

Eating Disorder Screen for Primary Care (ESP): https://nedc.com.au/assets/PHN/Fact-sheets/Screening-and-assessment-tools-for-eating-disorders.pdf

Nine Item Avoidant/Restrictive Food Intake Disorder Screen (NIAS): https://peds.arizona.edu/sites/default/files/intake_forms-child_v12.2018.pdf

Screen for Disordered Eating (SDE): www.nyeatingdisorders.org/_assets/pdf/screen_for_disordered_eating.pdf

SCOFF Questionnaire: https://www.nutritionhealth.com.au/site/assets/files/1064/scoff-questionnaire.pdf