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Eating Disorders
A Review

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IMPORTANCE Eating disorders are characterized by disturbances in eating behavior and occur worldwide, with a lifetime prevalence of 2% to 5%. They are more common among females than males and may be associated with medical and psychiatric complications, impaired functioning, and decreased quality of life.

OBSERVATIONS Common eating disorders include anorexia nervosa, bulimia nervosa, binge-eating disorder, and avoidant/restrictive food intake disorder. These disorders may be associated with changes in weight, electrolyte abnormalities (eg, hyponatremia, hypokalemia), bradycardia, disturbances in reproductive hormones (eg, decreased estradiol levels in females), and decreased bone density. Individuals with anorexia nervosa, bulimia nervosa, and binge-eating disorder have high lifetime rates of depression (76.3% for bulimia nervosa, 65.5% for binge-eating disorder, and 49.5% for anorexia nervosa) and higher rates of suicide attempts than those without eating disorders. Anorexia nervosa is associated with a mortality rate of 5.1 deaths per 1000 person-years (95% CI, 4.0-6.1), nearly 6 times higher than that of individuals of the same age without anorexia nervosa; 25% of deaths among individuals with anorexia nervosa are from suicide. First-line treatments for eating disorders include nutritional support, psychotherapy, and pharmacotherapy. Behaviorally focused therapies, including cognitive behavioral therapy, may be effective, especially for bulimia nervosa and binge-eating disorder. Youth with anorexia nervosa benefit from family-based treatment with parental oversight of eating, resulting in a remission rate at 6 to 12 months of 48.6% vs 34.3% with individual treatment (odds ratio, 2.08; 95% CI, 1.07-4.03; $P = .03$). Fluoxetine and other antidepressants decrease episodes of binge eating in individuals with bulimia nervosa, even in those without depression (fluoxetine vs placebo, standardized mean difference = −0.24 [small effect size; 95% CI, −0.41 to −0.08]). Antidepressants and the central nervous system stimulant lisdexamfetamine reduce binge frequency in binge-eating disorder compared with placebo (antidepressants vs placebo, standardized mean difference = −0.29 [small effect size; 95% CI, −0.51 to −0.06]; lisdexamfetamine vs placebo, Hedges $g = 0.57$ [medium effect size; 95% CI, 0.28-0.86]). There are currently no effective medications for treatment of anorexia nervosa. Individuals with serious medical or psychiatric complications of eating disorders such as bradycardia or suicidality should be hospitalized for treatment.

CONCLUSIONS AND RELEVANCE Globally, eating disorders affect 2% to 5% of individuals during their lifetime and are more common in females than males. In addition to weight changes, eating disorders may cause electrolyte abnormalities, bradycardia, disturbances in reproductive hormones, and decreased bone density, and are associated with increased risk of depression, anxiety, and suicide attempts. First-line treatments of eating disorders include nutritional support, psychotherapy, and pharmacotherapy.

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The *Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition, Text Revision* (*DSM-5-TR*)[1] describes 3 common and well-characterized eating disorders (anorexia nervosa, bulimia nervosa, and binge-eating disorder). Avoidant/restrictive food intake disorder (ARFID), first defined by the *DSM-5* in 2013,[2] is less well characterized. Eating disorders occur worldwide in individuals of all racial and ethnic backgrounds.[3-5] Sociocultural attitudes about thin appearance have been associated with body dissatisfaction and disordered eating behaviors among adolescents in many countries.[3-5]

Binge-eating disorder, bulimia nervosa, and anorexia nervosa are estimated to affect approximately 1.5 million, 500 000, and 175 000 adults in the United States, respectively.[6] These disorders occur more frequently among females than males, with a ratio of 12:1 for anorexia nervosa, 5.8:1 for bulimia nervosa, and 3:1 for binge-eating disorder.[6]

Eating disorders have both genetic and environmental risk factors. Large-scale twin studies indicate a genetic susceptibility to eating disorders, with heritability rates (rates range from 0 [no genetic contribution] to 1 [100% genetic]) of 0.38 to 0.74 for anorexia nervosa, 0.55 to 0.62 for bulimia nervosa, and 0.39 to 0.45 for binge-eating disorder.[7] Malnutrition and life stresses arising from gestation through adolescence may interact with genetic susceptibility in the development of these disorders. The economic costs of eating disorders in the United States due to medical care and loss of workplace productivity were estimated to be $64.7 billion in 2018 to 2019.[8]

This Narrative Review summarizes current evidence on epidemiology, diagnosis, and treatment of anorexia nervosa, bulimia nervosa, and binge-eating disorder.

Methods

We searched PubMed from June 2023 through October 2024 for English-language studies of the epidemiology, diagnosis, assessment, and treatment of eating disorders, excluding pica and rumination disorder. Systematic reviews, clinical practice guidelines and recommendations, and randomized clinical trials (RCTs) published in the preceding 5 years were prioritized for inclusion. Additional articles were identified from references of selected articles. We identified 668 articles and included 79 of them, consisting of 13 meta-analyses and systematic reviews; 9 narrative reviews; 7 clinical practice guidelines and recommendations; 25 RCTs; 21 epidemiologic, observational, or longitudinal studies; and 4 diagnostic manuals. Many recent, high-quality meta-analyses included in this review presented effect sizes instead of absolute rates. Standardized mean difference, Cohen *d*, and Hedges *g* are each interpreted as 0.2 = small effect, 0.5 = medium effect, and 0.8 = large effect.

Discussion

Distinguishing Clinical Features

The diagnostic criteria for eating disorders provided by the *DSM-5-TR*[1] and the World Health Organization's *International Classification of Diseases, 11th Revision*[9] are similar. The defining feature of an eating disorder is a substantial disturbance in eating or eating-related behavior, with various behavioral disturbances associated with each disorder (**Figure**).

Risk factors for developing an eating disorder include a family history of an eating disorder and childhood maltreatment such as emotional, physical, or sexual abuse, although many individuals with eating disorders report none of these risk factors.[10] Participation in activities emphasizing body shape or weight (eg, gymnastics, ballet, modeling) have been associated with the development of anorexia nervosa and bulimia nervosa.[11,12] Sexual and gender minority groups (eg, individuals who do not identify as heterosexual or who identify as a gender different from sex at birth) have a higher lifetime prevalence of eating disorder diagnoses than majority groups. For example, in a survey that included 36 309 adults in the United States, 3.6% of those who reported being a sexual minority had an eating disorder diagnosis vs 1.6% of those who did not report being a sexual minority (odds ratio [OR], 1.96; 95% CI, 1.31-2.94).[13]

Patients may transition between different eating disorders over time. For instance, someone with anorexia nervosa who has regained weight may later develop binge-purge behaviors that meet criteria for bulimia nervosa. In a 6-year follow-up of 793 patients with eating disorders, 33 of 197 patients (16.8%) with anorexia nervosa developed bulimia nervosa.[14]

Eating disorders often co-occur with other psychiatric disorders, including mood disorders (eg, major depressive disorder, bipolar disorder), anxiety disorders, obsessive-compulsive disorder, and substance use disorders such as alcohol use disorder and stimulant use disorder. Among individuals with eating disorders, major depression and anxiety disorder are the most common comorbid psychiatric disorders. In a national US survey of 36 309 adults, the lifetime prevalence of major depression among people with bulimia nervosa was 76.3%; with binge-eating disorder, 65.5%; and with anorexia nervosa, 49.5%.[15] Lifetime prevalence of anxiety disorder among people with bulimia nervosa was 44.6%; with binge-eating disorder, 59.0%; and with anorexia nervosa, 40.5%.[15] In a study that included 36 171 survey respondents, prevalence estimates of suicide attempts were 31.4% for individuals with bulimia nervosa, 24.9% for those with anorexia nervosa, and 22.9% for those with binge-eating disorder.[16] Comprehensive evaluation for psychiatric and substance use disorders is therefore recommended for individuals with eating disorders.

Anorexia Nervosa

Anorexia nervosa is characterized by restricted caloric intake resulting in a low body weight (eg, body mass index [BMI, calculated as weight in kilograms divided by height in meters squared] <18.5 in adults). Although most adolescents who eliminate desserts or increase exercise do not develop an eating disorder, these behaviors may intensify in some individuals, leading to substantial weight loss or failure to gain weight as expected developmentally. Individuals with anorexia nervosa often have rigid rules about food intake and may engage in excessive exercise, including exercising despite illness or injury. Patients with anorexia nervosa are typically preoccupied with their weight and body shape and fail to recognize the potentially serious health risks associated with low body weight (**Table 1**). Although some individuals with anorexia nervosa exclusively restrict food intake (restricting subtype), others also engage in binge-eating and purging behaviors (binge-eating/purging

Figure. Eating Disorder Differential Diagnosis, Assessment, and Treatment Considerations

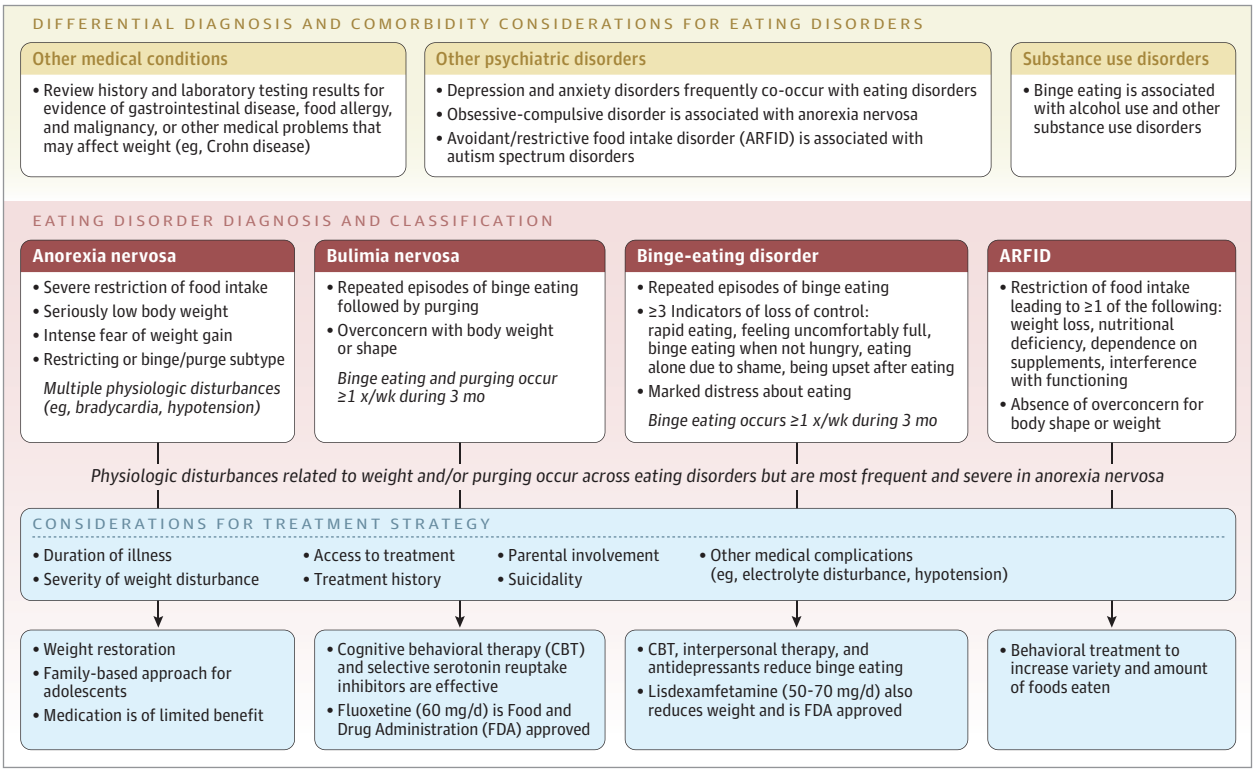

Table 1. Common Medical and Physical Complications Associated With Anorexia Nervosa and Bulimia Nervosa[a]

	Anorexia nervosa	Bulimia nervosa
Body weight and vital signs	• Low weight • Bradycardia • Hypotension, including orthostasis • Hypothermia	• Weight usually in normal range, occasionally overweight/obese
Skin	• Lanugo (fine hair growth on trunk and face) • Hair loss or thinning • Carotenemia	• Calluses on dorsum of hand from abrasion by teeth during self-induced vomiting (Russell sign; uncommon)
Head and mouth		• Enamel erosion of lingual surfaces of front teeth • Salivary gland hypertrophy
Cardiovascular	• Prolonged QTc • Peripheral edema	
Gastrointestinal	• Delayed gastric emptying • Constipation	• Elevated serum salivary amylase
Hematologic	• Neutropenia • Normochromic anemia	
Electrolyte/metabolic	• Hypoglycemia • Hyponatremia • Hypercholesterolemia • Elevated liver function test results	• Hypokalemia • Hyponatremia • Hypochloremia • Alkalosis
Endocrine	• Low luteinizing hormone • Low follicle-stimulating hormone • Low estrogen or testosterone • Low or normal thyroxine • Elevated cortisol • Amenorrhea • Decreased bone mineral density	• Oligomenorrhea or amenorrhea

Abbreviation: QTc, corrected QT interval.

[a] The complications associated with bulimia nervosa are primarily related to self-induced vomiting and may also occur among individuals with anorexia nervosa who engage in this behavior.

subtype). Prolonged anorexia nervosa can result in decreased bone mineral density, potentially leading to osteoporosis and higher fracture risk.[17]

Individuals with anorexia nervosa may develop cognitive disturbances such as difficulty concentrating; anemia, leukopenia, hyponatremia, and hypokalemia; and, if severely underweight,

hypophosphatemia and hypomagnesemia. Endocrine disturbances include the sick euthyroid syndrome and hypothalamic amenorrhea resulting from markedly reduced levels of luteinizing hormone, follicle-stimulating hormone, and estradiol. In a meta-analysis of 5 studies of 548 females with anorexia nervosa, the frequency of oligomenorrhea or amenorrhea was 77.7%.[18] Individuals with anorexia nervosa may develop bradycardia and QT-interval prolongation on electrocardiogram.

The term *atypical anorexia nervosa* was introduced in *DSM-5* in 2013 to describe individuals who lose substantial amounts of weight and develop many of the psychological, behavioral, and physiologic characteristics of anorexia nervosa but whose weight remains in the normal or overweight range.[2,18] Emerging data suggest the lifetime prevalence of atypical anorexia nervosa (2.9%) may be similar to that of typical anorexia nervosa (3.1%).[19] However, because there is limited information about the course and treatment response of individuals with atypical anorexia nervosa, it will not be discussed further in this review.

Bulimia Nervosa

Bulimia nervosa is characterized by frequent consumption of excessive amounts of food, with a sense of loss of control over eating (binge eating), followed by inappropriate compensatory behaviors to prevent weight gain, such as self-induced vomiting; misuse of diuretics, laxatives, or weight loss medications; and excessive exercise. Diagnostic criteria for bulimia nervosa include binge eating and purging occurring at least once weekly for greater than or equal to 3 months. Individuals with bulimia nervosa fear weight gain but typically have a normal or above-normal body weight. In addition to binge eating and compensatory purging, individuals with bulimia nervosa may engage in other impulsive behaviors such as nonsuicidal self-injury and substance use.[20]

Physical complications of bulimia nervosa are primarily caused by purging behavior (Table 1).[21] Frequent vomiting exposes the teeth to gastric acid, which can lead to loss of enamel and dental erosion. Parotid gland hyperplasia is also common among patients with bulimia nervosa. Chronic dehydration from purging may lead to elevated aldosterone levels, causing fluid retention, and peripheral edema may develop with abrupt cessation of purging. Self-induced vomiting and laxative and diuretic misuse are associated with fluid and electrolyte disturbances such as hyponatremia, hypokalemia, and acid-base imbalance. Rare but severe complications of bulimia nervosa include Mallory-Weiss esophageal tears from forceful vomiting.[21]

Binge-Eating Disorder

Individuals with binge-eating disorder engage in frequent episodes of overeating similar to the binge-eating characteristic of bulimia nervosa. However, individuals with binge-eating disorder do not engage in inappropriate behaviors to avoid weight gain such as purging or excessive exercise, as observed in bulimia nervosa.

To meet the *DSM-5-TR* criteria for binge-eating disorder, individuals must engage in binge eating at least once weekly for 3 months or more and have at least 3 indicators of loss of control over eating such as eating until uncomfortably full, eating more rapidly than normal, and eating alone because of embarrassment about their eating behavior. A prospective cohort study of US adolescents (Adolescent Brain Cognitive Development Study; N = 10 035; 2016-2020) reported that 1.2% of individuals had developed binge-eating disorder by the 2-year follow-up.[22]

Binge-eating disorder is associated with overweight and obesity. In a clinical sample of 174 individuals with binge-eating disorder, 71% had obesity, defined as BMI greater than 30.[23] Complications of obesity, such as the development of type 2 diabetes, are also associated with binge-eating disorder.[24] In 1 US study, individuals with binge-eating disorder (n = 318) had a mean of 2.3 (SE, 0.18) chronic conditions vs 1.4 (SE, 0.02; $P < .05$) among individuals without an eating disorder (n = 35 709); among individuals with binge-eating disorder, 13.3% reported having diabetes and 31.2% reported hypertension compared with 9.3% and 25.0%, respectively, for those without an eating disorder.[15] Binge-eating disorder is also associated with food insecurity. In the Adolescent Brain Cognitive Development Study, 15.8% of adolescents had food insecurity, which was associated with 1.67 higher odds of developing binge-eating disorder compared with those without food insecurity.[15] Food insecurity may lead individuals to consume inexpensive, calorie-dense, processed food that may be associated with binge eating.

ARFID

Avoidant/restrictive food intake disorder, first defined by the *DSM-5* in 2013,[2] involves restrictive eating unrelated to concerns about body image. Individuals with ARFID avoid specific foods according to color or texture, restrict intake after an adverse experience such as choking or vomiting, or have diminished interest in eating that can lead to substantial weight loss. The disorder develops more frequently in youth with neurodevelopmental disorders, including autism spectrum disorder and certain psychiatric disorders such as anxiety. A recent population-based study of Swedish children reported that 12.1% (74 of 611) of those with ARFID vs 0.9% (276 of 30 092) of control participants had autism spectrum disorder (OR, 13.7; 95% CI, 10.3-18.3). This study also found that children with ARFID had higher rates of anxiety disorder, 30.7% (123 of 401) vs 9.8% (1756 of 17 884) of controls (OR, 4.08; 95% CI, 3.24-5.13).[25] Avoidant/restrictive food intake disorder usually develops during early childhood, when picky and unusual eating patterns are common and may be considered normative; a recent study from the United Kingdom found that individuals with ARFID presented at a mean age of 11.2 years.[26]

Avoidant/restrictive food intake disorder may be associated with nutritional deficiencies, poor growth, delayed puberty, amenorrhea, vitamin and mineral deficiency, weakened bones and muscles, and psychosocial impairment.[25] The mortality and suicide rates associated with ARFID have not been well defined. Because limited evidence exists about how ARFID should be treated, it will not be discussed further in this review.

Assessment and Diagnostic Evaluation of Individuals With Eating Disorders

The initial evaluation of a patient with a possible eating disorder should exclude alternate medical diagnoses, including hyperthyroidism, hypothyroidism, or gastrointestinal conditions such as Crohn disease. Early identification of eating disorders is associated with improved clinical outcomes, including more rapid recovery. However, the diagnosis is often delayed because patients may be reluctant to seek help, and parents and clinicians may not recognize eating

Box. Treatment of Eating Disorders

1. What Should Clinicians Know About Anorexia Nervosa?

Anorexia nervosa is a potentially life-threatening disorder that can develop in individuals from all racial, ethnic, and socioeconomic backgrounds. Treatment focuses on weight restoration and normalization of eating behaviors and may take place in an outpatient, day-treatment, or hospital setting, depending on the severity of symptoms. Adolescents with anorexia nervosa benefit from family-based treatment.

2. What Are Effective Treatments for Bulimia Nervosa?

Evidence-based treatments for bulimia nervosa include cognitive behavioral therapy (CBT) and antidepressant medications. The selective serotonin reuptake inhibitor fluoxetine (60 mg/d) is Food and Drug Administration (FDA) approved for use in bulimia nervosa.

3. Which Treatments Are Helpful for Binge-Eating Disorder?

Binge-eating disorder symptoms typically improve with psychotherapy (eg, CBT) and pharmacotherapy (eg, antidepressant medications). The stimulant lisdexamfetamine is the only FDA-approved medication for treatment of binge-eating disorder.

disorders.[27] The American Psychiatric Association (APA),[28] the American Academy of Pediatrics (AAP),[29] and the Society for Adolescent Health and Medicine (SAHM)[30] recommend clinicians inquire about eating disorder symptoms and history, especially for patients whose weights are below or above the expected normal range. If a diagnosis of an eating disorder is suspected, especially in association with a coexisting psychiatric disorder such as anxiety and depression, referral for a comprehensive psychiatric evaluation should be strongly considered.

Although individuals with eating disorders often have a normal physical examination result, those with anorexia nervosa may have hypotension, bradycardia, and hypothermia (Table 1), and some develop fine body hair (lanugo) on the face, neck, and arms. Individuals who self-induce vomiting may develop erosions on the lingual surface of front teeth. Practice guidelines from the APA,[28] the AAP,[29] and the SAHM[30] recommend that the physical examination of patients with a suspected eating disorder include orthostatic vital signs for those with lightheadedness, bradycardia, or hypotension.

Additionally, for patients with a suspected eating disorder, an electrocardiogram should be obtained and laboratory testing should include a complete blood cell count; serum glucose, potassium, sodium, calcium, magnesium, phosphorous, albumin, and prealbumin levels; and tests of kidney, liver, and thyroid function. Reproductive hormones (luteinizing hormone, follicle-stimulating hormone, and estradiol) should be assessed in females experiencing oligomenorrhea or amenorrhea.[28] Practice guidelines suggest that bone mineral density be measured with a dual-energy x-ray absorptiometry scan for females with amenorrhea for 6 or more months.[28,29,31]

Treatment

Treatment of eating disorders (**Box**) should involve an empathetic therapeutic alliance between clinician and patient. Normalization of eating behaviors; improvement in distorted beliefs about body shape, weight, and food; and weight restoration, if indicated, are the goals of treatment. Clinicians should offer nutritional advice and nonjudgmental explanations of eating disorders, as well as monitor weight and symptoms.

Psychological and pharmacologic treatments are used for eating disorders. Treatment settings range from outpatient sessions to structured programs that include meal supervision to residential and inpatient programs. Selection of treatment setting is based on the patient's severity of symptoms, vital signs, laboratory evaluation results, age, eating disorder diagnosis, suicidality, history of response to previous treatments, cost, and availability. If patients are clinically stable and eating behavior goals are being met, the least restrictive treatment setting meeting patients' preferences should be selected. Treatment includes a multidisciplinary clinical team (eg, primary care clinician, therapist, dietitian). Indications for hospitalization are described in **Table 2**.

Table 2. Indications for Higher Level of Care Treatment Intensity[a]

Indication	Clinical characteristic
Behavioral	
Lack of improvement with outpatient treatment	
Potentially severe related disturbances (eg, elevated risk of suicide)	
Inability to access outpatient treatment	
>10% Weight loss in 6 mo or >20% in 12 mo	
Physiologic	
Weight	For adults, BMI <15
	For adolescents, <75% of median for age and sex
Temperature	<36 °C
Bradycardia	<50/min
BP	<90 mm Hg systolic
Orthostatic change in BP	>20 mm Hg decrease in systolic BP
ECG	QTc >450 ms
Serum parameters[b]	
Glucose	<60 mg/dL
Sodium	<130 mEq/L
Potassium	<3.0 mEq/L
Phosphate	<2.0 mg/dL
Magnesium	<1.5 mg/dL

Abbreviations: BMI, body mass index (calculated as weight in kilograms divided by height in meters squared); BP, blood pressure; ECG, electrocardiogram; QTc, corrected QT interval.

SI conversion factors: To convert glucose to mmol/L, multiply by 0.0555; to convert magnesium to mmol/L, multiply by 0.4114.

[a] For example, hospitalization. Indications are based in part on the American Psychiatric Association practice guideline.[28]

[b] Normal ranges for these parameters may differ among laboratories.

Acute Medical and Nutritional Considerations

Anorexia Nervosa

Weight restoration, medical and nutritional improvement, and psychological care are first-line treatments for anorexia nervosa. Target weight restoration recommendations should be individualized. For adults, considerations include preillness weight and achieving

Table 3. Psychotherapy Interventions for Eating Disorders

Intervention	Description	Comments	Evidence from RCTs
CBT	CBT posits that overconcern with shape and weight leads to excessive food restriction, which predisposes to binge eating. Treatment focuses on normalizing eating behavior and addressing overconcern with shape and weight. Typically delivered via ≈20 individual sessions during ≈6 mo.	CBT is considered first-line treatment for BN and BED. Remission rates at the end of treatment are ≈50%. CBT is not associated with significant weight change. CBT is of benefit for adults with AN but not superior to other psychotherapies.[39]	CBT vs waiting list: BN, Hedges *g* = 0.97 (95% CI, 0.44-1.50)[40]; BED, Hedges *g* = 1.13 (95% CI, 0.71-1.55).[41]
Self-help CBT	Patient learns principles of CBT from reading text, uses techniques described in a manual. In guided self-help, also meets with a therapist.		Self-help CBT vs waiting list or treatment as usual: BN, Hedges *g* = 3.44 (95% CI, 2.05-5.78); BED, Hedges *g* = 4.82 (95% CI, 3.20-7.27).[41]
IPT	IPT focuses on interpersonal difficulties and social deficits that are linked to disturbances in eating behavior. Typically delivered via ≈20 individual or group sessions during ≈6 mo.	Considered first- or second-line treatment for BN and BED. Effect of CBT is somewhat more rapid than that of IPT, but, over time, benefits appear similar.	BN: 2 large RCTs found that long-term benefits were similar to those from CBT.[42,43] BED: 3 large RCTs found that long-term benefits were similar to those from CBT.[44-46]
FBT	FBT focuses on treatment of adolescents by empowering parents to help change patients' eating behavior with guidance from a therapist. Typically delivered in weekly sessions during ≈6 mo.	Considered first-line treatment for adolescents with AN.	FBT therapy for AN or BN vs other treatment. Remission at end of treatment: odds ratio, 1.90 (95% CI, 0.91-3.94). Remission at 6-12 mo: odds ratio, 2.14 (95% CI, 1.29-3.53).[47]

Abbreviations: AN, anorexia nervosa; BED, binge-eating disorder; BN, bulimia nervosa; CBT, cognitive behavioral therapy; FBT, family-based therapy; IPT, interpersonal therapy; RCT, randomized clinical trial.

a weight associated with normal physiologic function (eg, resumption of menses in females), with the target BMI typically 18.5 to 24.9.[32] For adolescents with anorexia nervosa, individual growth curves help estimate an age-appropriate healthy BMI range. For adolescents and adults, individualized goals for caloric intake and weight gain include initial nutritional targets of approximately 1500 to 2000 kcal/d, with incremental increases up to an intake of 3000 to 4000 kcal/d until target weight is reached; intake is then reduced to support maintenance of healthy weight.[28,33,34] For individuals whose oral caloric intake is inadequate to achieve weight gain, nasogastric tube feeding may be considered. Nasogastric tubes, when necessary, are preferable to percutaneous endoscopic gastrostomy tubes because they do not require a surgical procedure and are easily removed. Nasogastric tube feeding, which may be court-ordered for patients who lack decision-making capacity and refuse oral nutrition, safely facilitates weight gain. The APA guideline recommends against use of parenteral nutrition (eg, total parenteral nutrition) for treatment of eating disorders.[28]

Early and faster rates of weight gain (eg, 0.9-1.8 kg/wk in inpatient or residential settings and 0.5-0.9 kg/wk in structured outpatient settings) are associated with improved outcomes such as shorter inpatient stays, normalization of vital signs, and improved remission rates.[33-35] Refeeding syndrome (ie, shifts in fluids and electrolytes with rapid refeeding in the context of starvation) is rare, typically mild to moderate in severity, and managed with regular monitoring of electrolytes such as phosphate along with nutrient and electrolyte repletion.[33,36] Most of the effects of starvation such as electrolyte, nutritional, and cognitive disturbances such as difficulty concentrating improve with weight gain, but bone density may not recover, requiring ongoing monitoring and management.[17]

Most patients with anorexia nervosa treated in inpatient and structured outpatient programs achieve full weight restoration; for example, a study of 265 adults and 92 adolescents (11-17 years) reported that, with inpatient treatment, 71.8% and 80.4% reached target weights in 27.7 and 35.4 days, respectively.[36] However, eating disorder relapse rates after acute weight restoration are high, with studies reporting relapse of 40% to 50% of patients with anorexia nervosa within 1 year of discharge from inpatient or intensive day program treatment.[37,38]

Bulimia Nervosa and Binge-Eating Disorder

Medical complications occur less frequently among individuals with bulimia nervosa and binge-eating disorder than among those with anorexia nervosa. However, individuals with bulimia nervosa should be evaluated for complications associated with purging behaviors (Table 1).

Psychotherapy for Eating Disorders

Clinical trials have reported efficacy of several forms of psychotherapy for eating disorders (**Table 3**).[39,42-46] Behaviorally focused therapies, particularly cognitive behavioral therapy (CBT), which address beliefs about body shape and weight and disordered eating behaviors, have been demonstrated to be helpful, especially in reducing binge eating for individuals with bulimia nervosa or binge-eating disorder.[48] Dialectical behavioral therapy, a treatment that helps individuals develop skills to reduce impulsive behaviors, has not been extensively studied in patients with eating disorders but may be useful, especially for individuals with coexisting borderline personality disorder, impulsivity, and emotional dysregulation.[49]

Anorexia Nervosa

For patients with anorexia nervosa, practice guidelines from the APA,[28,50] the American Academy of Child and Adolescent Psychiatry,[51] the AAP,[29] and the SAHM[30] recommend eating

disorder–focused psychotherapy with individualized treatment goals that include normalizing eating and eliminating inappropriate weight control behaviors (eg, purging, excessive exercise), restoring weight, and addressing associated psychological symptoms. An RCT in Germany compared 3 forms of psychotherapy (psychodynamic psychotherapy, CBT, and optimized treatment as usual [outpatient psychotherapy and structured care from a family physician]) for 242 adults with anorexia nervosa. This trial reported that, after 10 months of treatment, BMI increased in all study groups (0.73 with psychodynamic therapy, 0.93 with CBT, and 0.69 with optimized treatment), with no significant differences among groups.[52] Systematic reviews of psychological treatments for adults with anorexia nervosa also demonstrate no superiority of any single psychological treatment over others (Table 3).

For children and adolescents with anorexia nervosa, family-based treatment is recommended by the APA,[28] the American Academy of Child and Adolescent Psychiatry,[51] and Canadian practice guidelines.[53] Family-based treatment, an outpatient treatment offered during 6 to 12 months, informs parents that they are not responsible for their child's eating disorder and guides them through their child's full weight restoration. The treatment involves either joint family sessions or separate sessions with parents and patient. Family-based treatment is associated with substantial weight gain in anorexia nervosa and with a greater remission rate at 6- to 12-month follow-up (48.6% vs 34.3% for individual treatment; OR, 2.08; 95% CI, 1.07-4.03; $P = .03$).[47] For individuals who do not improve with family-based treatment or do not have access to it, other eating disorder treatments such as CBT should be considered.

Bulimia Nervosa

Cognitive behavioral therapy is a first-line treatment for bulimia nervosa and is superior to other interventions, including supportive psychotherapy.[40] A meta-analysis of 3 RCTs involving 109 patients reported that the odds of achieving abstinence from binge eating were more than 5 times greater among patients randomized to CBT vs to a waiting list (OR, 5.25; 95% CI, 1.60-17.19).[40]

Binge-Eating Disorder

Therapist-delivered CBT and self-help versions of CBT can be effective for individuals with binge-eating disorder.[41] A 2023 study randomly assigned 18 of 31 patients with binge-eating disorder who had not responded adequately to medication (naltrexone combined with bupropion), behavioral therapy, or both to CBT for 16 weeks[54]; 11 of 18 (61%) of those assigned to CBT achieved remission vs 1 of 13 (7.7%) who did not receive CBT ($P = .003$). Interpersonal psychotherapy focusing on understanding illness behaviors may also be effective at reducing binge-eating episodes.[55]

Pharmacotherapy for Eating Disorders

Anorexia Nervosa

There are currently no guideline-recommended medications for anorexia nervosa. Compared with placebo, selective serotonin reuptake inhibitors and other antidepressant medications do not promote weight gain or improve psychological symptoms in patients with anorexia nervosa (Table 4).[56,59,60] However, selective serotonin reuptake inhibitor treatment may be considered for individuals with anorexia nervosa and persistent depression, anxiety, or obsessive-compulsive disorder. Olanzapine, an appetite-stimulating antipsychotic medication, may be useful in selected patients to promote weight gain but is not a sufficient stand-alone treatment.[56] In the largest trial to date, use of olanzapine in 152 patients with anorexia nervosa was associated with a mean (SD) increase in BMI of 0.259 (0.051) per month vs 0.095 (0.053) with placebo ($P = .03$), the equivalent of 0.7 kg/mo vs 0.26 kg/mo for an average-height patient (eg, 165 cm), without adverse effects such as hyperglycemia and hyperlipidemia.[64]

Use of oral hormone replacement therapy in patients with anorexia nervosa does not improve bone mineral density and masks amenorrhea.[67] Small studies (20-70 patients with anorexia nervosa) have reported minor increases in bone mineral density with transdermal estradiol,[68] alendronate,[69] and risedronate with low-dose testosterone[70] and modest increases with denosumab.[71]

Bulimia Nervosa

Clinical trials have reported that antidepressants are more efficacious than placebo in reducing binge-eating frequency and reducing episodes of purging in patients with bulimia nervosa. A recent meta-analysis reported that, compared with placebo, use of selective serotonin reuptake inhibitors was associated with decreased binge eating (10 RCTs; 911 participants; standardized mean difference, −0.29; 95% CI, −0.51 to −0.08) and decreased purging (9 RCTs; 884 participants; standardized mean difference, −0.51; 95% CI, −0.81 to −0.21).[57] Fluoxetine is currently the only Food and Drug Administration–approved medication for bulimia nervosa, and the therapeutic effect occurs within 3 weeks of treatment initiation. An RCT (n = 387) reported that individuals assigned to fluoxetine 60 mg/d compared with placebo were more likely to have a decrease of 50% or more in binge-eating episodes (63% vs 43%) and vomiting episodes (57% vs 26%; $P < .001$ for both comparisons).[63] Antidepressants (eg, fluoxetine,[63] desipramine[72]) may be effective for individuals with bulimia nervosa even in the absence of major depression and for individuals who have not improved with psychotherapy.[73] The APA guideline recommends continuing selective serotonin reuptake inhibitor medication in treatment responders with bulimia nervosa for at least 9 months.[28]

Binge-Eating Disorder

The stimulant lisdexamfetamine, which was approved by the Food and Drug Administration in 2015 for treatment of binge-eating disorder, is associated with reductions in binge-eating episodes and in body weight. An RCT of 259 individuals with binge-eating disorder reported that the number of days in which binge episodes occurred decreased by a mean (SD) of 4.1 (1.52) per week among those randomized to lisdexamfetamine (50 mg/d) vs 3.2 (2.04) among those randomized to placebo ($P = .008$) during 11 weeks; weight decreased by 4.9 kg in the lisdexamfetamine group vs 0.1 kg in the placebo group ($P < .001$).[74]

Two RCTs reported reduced frequency of binge eating and reductions in weight- and eating disorder–related psychopathology with topiramate compared with placebo[65,66]; in the larger study (n = 404), during 16 weeks binge eating decreased by a mean (SD) of 3.7 (1.9) episodes per week among patients randomized to topiramate (25-400 mg/d) vs 2.4 (2.1) episodes per week among those randomized to placebo ($P < .001$).[66] Multiple small RCTs of antidepressants, including selective serotonin reuptake inhibitors

Table 4. Pharmacotherapy Interventions for Eating Disorders

Drug	Anorexia nervosa	Bulimia nervosa	Binge-eating disorder	Other considerations
SSRIs	No good evidence of clinical utility in treatment of underweight patients or in preventing relapse after weight restoration.[56]	Fluoxetine (60 mg/d) significantly reduces binge eating and purging and is FDA approved. Frequency of binge eating, effect size vs placebo: Hedges *g*, 0.203 (95% CI, 0.007-0.399), 5 trials;[56] SMD, −0.24 (95% CI, −0.41 to −0.08), 6 trials.[57] RCTs of other SSRIs indicate they also reduce binge eating and purging.	RCTs of several SSRIs demonstrate a significant reduction in frequency of binge eating but generally not in weight: fluoxetine,[58] citalopram,[59] duloxetine,[60] fluvoxamine,[61] and sertraline.[62]	The dose of fluoxetine for bulimia nervosa (60 mg/d) is superior to the dose of 20 mg/d usually used to treat depression.[63] Most effective doses of other SSRIs for bulimia nervosa and binge-eating disorder are not well established but are typically used according to approved doses for the treatment of depression.
Antipsychotics	Olanzapine (5-10 mg/d) is associated with a small increase in weight gain vs placebo (Hedges *g*, 0.283; 95% CI, 0.051-0.515), 6 trials.[56]			Olanzapine likely increases food consumption but has a limited effect on psychological symptoms. Metabolic adverse effects (eg, elevations in serum glucose level) are rare.[64]
Stimulants			Lisdexamfetamine (50-70 mg/d) significantly reduces binge eating and weight and is FDA approved. Reduction in binge eating: Hedges *g*, 0.57 (95% CI, 0.28-0.86), 3 trials. Weight loss: Hedges *g*, 0.259 (95% CI, 0.071-0.446), 3 trials.[56]	Lisdexamfetamine treatment is associated with modest weight loss, although weight regain may occur after drug discontinuation. Lisdexamfetamine is associated with small increases in heart rate and blood pressure, so these parameters should be monitored during treatment; patients should be assessed for cardiovascular disease before initiation of lisdexamfetamine.
Anticonvulsants			2 RCTs reported topiramate (300 mg/d) led to significant reductions in binge eating and weight.[65,66]	Topiramate may be associated with cognitive adverse effects (eg, brain fog), which may be reduced by gradually increasing dose to a maximum of 300 mg/d.

Abbreviations: FDA, Food and Drug Administration; RCT, randomized clinical trial; SMD, standardized mean difference; SSRI, selective serotonin reuptake inhibitor.

(eg, fluoxetine,[58] fluvoxamine,[61] sertraline[62]), have reported reductions in binge eating (antidepressants vs placebo, standardized mean difference = −0.29; 95% CI, −0.51 to −0.06) but typically limited effects on weight compared with placebo.[75]

Relapse and Recovery

Among patients with anorexia nervosa, relapse rates are high after weight restoration treatments (eg, approximately 50% during the following year[37]). An 8.3-year follow-up study reported that among 212 adolescents hospitalized for anorexia nervosa, 45.7% were rehospitalized 2 or more times, suggesting that multiple treatment efforts are often necessary.[76]

Most studies about eating disorders include relatively short-term outcome data (6- to 12-month follow-up), making accurate prediction of the longer-term prognosis challenging. Longitudinal studies of clinical and community samples suggest that recovery often occurs only after extended periods. A longitudinal cohort study of 228 patients reported recovery rates for anorexia nervosa of 31.4% at 9 years and 62.8% at 22 years.[77] Recovery rates for bulimia nervosa were 68.2% at 9 years and 68.2% at 22 years.[77] A recent meta-analysis of 415 cohort studies and clinical trials with 88 372 individuals with eating disorders (mean follow-up, 38.3 months [SD, 76.5 months]) reported a pooled recovery rate (46% overall) across all eating disorders that increased with duration of follow-up (42% at <2 years; 43% at 2 to <4 years; 54% at 4 to <6 years; 59% at 6 to <8 years; 64% at 8 to <10 years; and 67% at ≥10 years), without significant differences among eating disorder diagnostic groups.[78]

Mortality

Individuals with eating disorders have a higher mortality rate than controls. A meta-analysis of 36 studies and 17 272 participants with eating disorders reported that anorexia nervosa had a mortality rate of 5.1 deaths per 1000 person-years (95% CI, 4.0-6.1; standardized mortality ratio, 5.86; 95% CI, 4.17-8.26); 3.3 deaths per 1000 person-years for the *DSM-IV* category of "eating disorder not otherwise specified," which includes binge-eating disorder (standardized mortality ratio, 1.92; 95% CI, 1.46-2.52); and 1.7 deaths per 1000 person-years for bulimia nervosa (standardized mortality ratio, 1.93; 95% CI, 1.46-2.52).[79] Anorexia nervosa has among the highest mortality rates of any psychiatric disorder, and 25% of deaths of individuals with anorexia nervosa are due to suicide.[79]

Limitations

There are several limitations to this review. First, a formal quality assessment of the included literature was not performed. Second, the available studies, especially those focusing on treatment, often included small numbers of participants. Third, treatment studies rarely included a comparison between 2 active treatments, which limits information about the comparative effectiveness of treatments. Fourth, included studies varied in research design and some had inconsistent findings. Fifth, some relevant studies may have been missed.

Conclusions

Globally, eating disorders affect 2% to 5% of individuals during their lifetime and are more common in females than males. In addition to weight changes, eating disorders may cause electrolyte abnormalities, bradycardia, disturbances in reproductive hormones, and decreased bone density, and are associated with increased risk of depression, anxiety, and suicide attempts. First-line treatments of eating disorders include nutritional support, psychotherapy, and pharmacotherapy.

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Submissions: We encourage authors to submit papers for consideration as a Review. Please contact Kristin Walter, MD, at kristin.walter@jamanetwork.org.

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