Eating Disorders in Children and Adolescents: State of the Art Review

This is the 3rd in our series on Adolescent Health.

abstract

Despite their high prevalence, associated morbidity and mortality, and available treatment options, eating disorders (EDs) continue to be underdiagnosed by pediatric professionals. Many adolescents go untreated, do not recover, or reach only partial recovery. Higher rates of EDs are seen now in younger children, boys, and minority groups; EDs are increasingly recognized in patients with previous histories of obesity. Medical complications are common in both full and subthreshold EDs and affect every organ system. No single cause of EDs has emerged, although neurobiological and genetic predispositions are emerging as important. Recent treatment paradigms acknowledge that they are not caused by families or chosen by patients. EDs present differently in pediatric populations, and providers should have a high index of suspicion using new *Diagnostic and Statistical Manual, 5th edition* diagnostic criteria because early intervention can affect prognosis. Outpatient family-based treatment focused on weight restoration, reducing blame, and empowering caregivers has emerged as particularly effective; cognitive behavioral therapy, individual therapy, and higher levels of care may also be appropriate. Pharmacotherapy is useful in specific contexts. Full weight restoration is critical, often involves high-calorie diets, and must allow for continued growth and development; weight maintenance is typically inappropriate in pediatric populations. Physical, nutritional, behavioral, and psychological health are all metrics of a full recovery, and pediatric EDs have a good prognosis with appropriate care. ED prevention efforts should work toward aligning with families and understanding the impact of antiobesity efforts. Primary care providers can be key players in treatment success. *Pediatrics* 2014;134:582–592

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Despite their high prevalence, associated morbidity and mortality, and available treatment options, eating disorders (EDs) continue to be under-diagnosed by pediatric professionals. Many adolescents go untreated, do not recover, or reach only partial recovery. This article provides an update of the current ED literature in children and adolescents. Anorexia nervosa (AN), bulimia nervosa (BN), and other EDs are presented. The epidemiology, etiology and pathogenesis, clinical presentation, and diagnosis of EDs are reviewed. New diagnostic criteria from the *Diagnostic and Statistical Manual, 5th edition* (DSM-5), updates on complications of EDs, and advances in their evidence-based treatment are highlighted, as is the role that primary care providers (PCPs) have in positively affecting treatment outcomes and the prevention of EDs in children and adolescents.

EPIDEMIOLOGY

Pediatric EDs are more common than type 2 diabetes, and the epidemiology is changing, with higher rates of EDs in younger children, boys, and minority groups.[1–3] The lifetime prevalence of AN is between 0.5% to 2%,[4] with a peak age of onset of 13 to 18 years.[5] AN has a mortality rate of at least 5% to 6%,[6,7] the highest mortality rate of any psychiatric illness.[8] The lifetime prevalence of BN is higher at between 0.9% and 3%,[9,10] with an older age of onset of 16 to 17 years.[11] Although mortality rates in BN are estimated to be ~2%,[12] the risk of lifetime suicidality and suicide attempts in BN are much higher.[6] On the basis of the criteria from the fourth edition of the DSM, most adolescents were diagnosed with EDs not otherwise specified (EDNOS), a group of heterogeneous disorders composed primarily of subthreshold AN or BN.[13] The estimated lifetime prevalence of EDNOS in adolescents is 4.8%.[14] Rates of medical complications in EDNOS are similar to full-threshold disorders.[15] Although female patients account for most ED diagnoses, males have accounted for 10% of ED cases over the past years,[4] with some studies reporting up to 25% of cases being male.[16] Furthermore, younger patients diagnosed with EDs are more likely to be boys, with a female to male ratio of 6 to 1, compared with a 10 to 1 ratio in adults.[3,17] Dieting behaviors are a risk factor for developing an ED and are highly prevalent; ~50% of girls and 25% of boys report dieting during the past year.[18] Moreover, 30% of girls and 15% of boys had disordered eating behaviors severe enough to warrant medical evaluation, and 9% of girls and 4% of boys reported daily self-induced vomiting.[19]

ETIOLOGY

The exact etiology of EDs is unknown; there is thought to be an interface between genetic and biological predispositions, environmental and sociocultural influences, and psychological traits. Evidence continues to increase that EDs are heritable, with relatives of ED patients having 7 to 12 times greater risk of developing an ED.[20–22] Twin studies have estimated heritability of AN between 33% and 84% and BN between 28% and 83%.[4,22] Research is ongoing to identify specific chromosomes, genes, and proteins that may play a role in the development of AN and BN.[4] There are also neurobiological factors being studied in EDs, but it is uncertain whether they contribute to the development of EDs or result from the physiologic alterations caused by EDs.[23]

CLINICAL PRESENTATION

Adolescence is a critical period of development and a window of vulnerability during which EDs can develop. The explosive physical and cognitive development that occurs during this period lends itself to substantial differences in the presentation of EDs in children and adolescents; pediatricians are frequently the front-line providers diagnosing these disorders. An ED should be suspected in a patient of any weight who presents with weight loss, unexplained growth stunting or pubertal delay, restrictive or abnormal eating behaviors, recurrent vomiting, excessive exercise, trouble gaining weight, or body image concerns. Younger patients are likely to have atypical presentations; instead of rapid weight loss, they may present with failure to make expected gains in weight or height and may not endorse body image concerns or engage in binge eating or purging behaviors.[17,24] Boys and children and adolescents who are overweight or obese are at risk for delayed diagnoses and significant complications[3,25,26]; these populations require heightened vigilance by providers. Adolescents with chronic illnesses, especially insulin-dependent diabetes mellitus, are also at higher risk of developing ED behaviors and should be screened regularly.[27–31]

Screening tools, such as the brief SCOFF questionnaire,[32] although only validated in adults, are used in the primary care setting for ED screening in adolescents (Table 1). In addition, providers should evaluate all patients for high-risk behaviors, such as dieting or excessive exercise, and follow their growth trajectories and BMI to assess

TABLE 1 The SCOFF Questionnaire[a]

1. Do you make yourself Sick because you feel uncomfortably full?
2. Do you worry you have lost Control over how much you eat?
3. Have you recently lost more than One stone (14 lb/6.3 kg) in a 3-month period?
4. Do you believe yourself to be Fat when others say you are too thin?
5. Would you say that Food dominates your life?

[a] One point for every "yes"; a score of ≥2 indicates a likely case of anorexia nervosa or bulimia.

for weight loss or failure to make appropriate gains. If an ED is suspected, it is important to obtain a comprehensive medical, family, and social history and a complete review of systems and to perform a thorough physical examination to evaluate for physical stigmata and medical complications of EDs (Table 2). Obtaining the history from both the patient and caregiver(s) is important; although time alone with the adolescent is recommended, history from caregiver(s) can be crucial in elucidating behaviors or cognitions that the adolescent may not report. In addition, the providers should always consider a complete differential diagnosis when evaluating a patient with a potential ED (Table 3).

DIAGNOSIS

New diagnostic criteria for EDs are published in the DSM-5, released in 2013.[33] Significant changes were made in an effort to improve the accuracy and precision of ED diagnoses, which will potentially allow for more targeted treatment. One major limitation of the fourth edition of the DSM was the diagnostic category of EDNOS, which accounted for the majority of ED diagnoses in most pediatric series.[34] EDNOS was a nonspecific diagnostic category that encompassed a wide spectrum of EDs, including subthreshold AN, subthreshold BN, and binge ED (BED).[14] This ambiguity led to misunderstandings of the clinical significance of the disorder and difficulty choosing the most effective therapy. To address these issues, the DSM-5 broadens the inclusion criteria for both AN and BN, BED is now a formal diagnosis, and other EDs have been further clarified.[35]

Adolescents with AN often present with dramatic weight loss or poor growth and may be preoccupied with food and weight. Restriction of entire food groups (ie, new-onset vegetarianism) or calories, and the development of food rituals are commonplace. They commonly refuse to eat foods they once enjoyed, avoid meals with family and friends, and overexercise in a rigid manner. Pubertal milestones such as linear growth or menstrual cycles are often affected.[5,16,24] DSM-5 criteria for AN consider expected weight and growth[36] in children and adolescents versus comparisons to population norms. They describe a restriction of energy intake relative to requirements, leading to a lower than expected body weight. In addition, behavioral criteria are considered equivalent to cognitive criteria, equating fear of weight gain to failure to gain weight in the face of low body weight or growth stunting.[5] Amenorrhea has been removed as a criterion because its use was never validated[36] and excluded males, premenarchal females, and adolescents who remain eumenorrheic despite low body weight.[37] Finally, body image distortion or an unusual focus on weight or shape are still included as

TABLE 2 History Questions and Physical Examination Findings in EDs

History	Physical Examination Findings
Past medical history	Anorexia nervosa and other restrictive disorders
Time course of weight loss, including minimum and maximum weight during adolescence.	Sinus bradycardia
Perceived goal weight/healthy weight	Cardiac arrhythmias including QT prolongation
Body image concerns	Orthostatic changes in pulse >20 or blood pressure >10
Dietary habits including 24-hr recall, history of restricting, binge eating, and/or purging	Hypotension
Exercise history	Hypothermia
Previous therapy	Dry, pale skin
Secretive behaviors	Orange discoloration of skin
Symptoms of systemic illnesses, such as inflammatory bowel disease, diabetes mellitus, celiac, lupus	Lanugo
Relevant review of systems: presyncope, syncope, headaches, fatigue, exercise intolerance, sleep disturbance, dry skin, increased shedding of hair/hair loss, cold intolerance, easy bruising, delayed wound healing, mood changes	Bruising/abrasions over spine
Family history	Acrocyanosis
Eating disorders, obesity, depression, anxiety, alcoholism or drug abuse, bipolar affective disorder, schizophrenia	Thinning scalp hair
Pubertal/menstrual history	Facial wasting
Menarche, last menstrual period, changes in regularity, duration	Cachexia
Timing of thelarche and pubarche if premenarchal	Atrophic breasts
Growth rate deceleration	Scaphoid abdomen
For males: history of decreased erections or nocturnal emissions	Dependent edema
Social history	Flat or anxious affect
Recent stressors, family, school, friends	BN and other purging disorders
Tobacco, alcohol, illicit drug use	Sinus bradycardia or cardiac arrhythmias including QT prolongation
History of physical or sexual abuse	Orthostatic changes in pulse >20 or blood pressure >10
Use of pro-eating disorder Web sites	Calluses or abrasions over knuckles due to self-induced vomiting
Mood changes	Parotid enlargement
Time with friends	Dental enamel erosions, caries, oral ulcerations
Engagement in fun activities	Mood lability
Recent stressors, family, school, friends	Scleral hemorrhage
	Palatal petechiae
	Loss of gag reflex

TABLE 3 Differential Diagnosis of EDs

Endocrine disorders
    Hyperthyroidism or hypothyroidism
    Diabetes mellitus
    Hypercortisolism
    Adrenal insufficiency
Gastrointestinal disorders
    Inflammatory bowel disease
    Celiac disease
    Infectious diarrhea
    Immunodeficiency or chronic infections (ie, HIV,
        tuberculosis)
Psychiatric disorders
    Depression
    Obsessive compulsive disorder/anxiety
    Substance abuse
Other disorders
    Superior mesenteric artery syndrome
    Malignancies
    Central nervous system tumors (ie,
        prolactinoma)
    Pregnancy
    Excessive exercise/energy imbalance
    Rheumatologic disease
    Wilson's disease
    Porphyria

criteria but are not required if the patient persistently fails to recognize the seriousness of his or her low body weight.

The hallmark of BN is recurrent episodes of binge eating accompanied by inappropriate compensatory behaviors. An objective binge episode involves eating more food in a discrete period of time than most people would eat, coupled with feeling a loss of control. DSM-5 criteria for BN require objective binge episodes and subsequent compensatory behaviors at least once per week for 3 months. Patients with BN may be of any weight and often have frequent weight fluctuations from fluid shifts. Caregivers or peers may notice the development of mood swings, surreptitious behaviors (ie, increased time in the bathroom after meals, hiding food), or periods of fasting or excessive exercise.[9,33]

The distinguishing feature between BED and BN is that episodes of binge eating are not associated with inappropriate compensatory behaviors. Patients with BED and BN display marked distress regarding binge eating and will often binge in secret. The frequency of recurrent episodes of binge eating was decreased in the DSM-5 similar to BN.[33,38]

Additional new categories in the DSM-5 with likely impact are avoidant restrictive food intake disorder, other specified feeding and EDs, and unspecified feeding and ED. "Avoidant restrictive food intake disorder is not uncommon in children[35,39] and comprises a variety of restrictive eating behaviors (ie, swallowing phobias, textural aversions) that do not involve a fear of weight gain or distorted cognitions but lead to significant physical and emotional impairment. Other specified feeding and EDs refers to atypical AN (normal-weight AN), subthreshold BN, purging disorder, and night eating syndrome. Unspecified feeding and ED comprises any other clinically significant EDs that do not fit the aforementioned categories.[33,39]

Complications

EDs can affect every organ system, and complications can occur at any weight.[16,24,40,41] It is important for providers to act quickly and decisively when they suspect an ED in all patients to avoid complications and the potential for chronicity.

CARDIOVASCULAR SYSTEM

Cardiac complications are common[15,40,42]; patients with EDs often present with bradycardia, hypotension, arrhythmias, and changes in heart rate variability.[15,40,43–46] Hypotension and postural changes in heart rate and blood pressure can result from decreased cardiac mass leading to systolic dysfunction, in addition to volume depletion and autonomic dysfunction.[16,24,42–44,47,48] Associated physical symptoms may include headache, presyncope, syncope, and exercise intolerance, although patients are frequently asymptomatic even in the face of profound vital sign instability.[16,24,48–50]

Patients with chronic purging are at risk for cardiomyopathy, and up to one-third of hospitalized patients with AN have mitral valve prolapse and pericardial effusion.[41,43,46,51] Several small studies have demonstrated almost complete reversibility of both structural and functional derangement,[43,51] although ipecac abuse can lead to an irreversible cardiomyopathy.[52–55] Serious cardiac complications are not unique to AN but are also seen in other normal-weight EDs, particularly atypical AN and BN.[15,56–62]

Gastrointestinal

Gastrointestinal complications may occur secondary to malnutrition, vomiting, or binge eating. Complications secondary to malnutrition include delayed gastric emptying,[63] constipation, mild transaminitis, dyslipidemias, and superior mesenteric artery syndrome.[48,64–66] Patients who vomit risk esophagitis, and in severe cases, esophageal rupture and pneumomediastinum. They may present with reflux, hematemesis or parotid swelling.[67,68] Patients with AN typically report abdominal bloating, nausea, and postprandial fullness. Patients with binge eating behaviors are at risk for gastric dilation or rarely gastric rupture and pancreatitis.[69,70]

Electrolytes

Electrolyte disturbances occur in patients who engage in vomiting, laxative abuse, or diuretic use, with hypokalemia and hypophosphatemia being the most common.[71,72] Hypochloremic metabolic alkalosis may develop in patients who vomit, and hyperchloremic metabolic acidosis may develop in those who abuse laxatives.[73] Patients with malnutrition are at risk for refeeding syndrome during treatment, which includes hypophosphatemia,[74] hypokalemia,[73,75,76] and hypomagnesemia.[77]

Endocrine

Patients with AN typically have hypothalamic suppression with low normal to low gonadotropin and sex hormone levels. Girls and boys may present with decelerated linear growth, pubertal delay, or pubertal regression, and menstrual dysfunction is common in females.[78–81] Low insulin-like growth factor-I and low to low-normal thyroxine and triiodothyronine levels are seen.[40,48] Sick euthyroid syndrome can be seen in severely malnourished patients and resolves with reversal of the malnourished state.[40,79,80] ED adolescents also risk reduced bone mineral density primarily due to poor nutritional intake, low BMI, and reduced fat mass.[45] Leptin plays a key role in energy homeostasis, and levels are low in malnourished states. A recent study demonstrated that if leptin levels are normalized, menstrual function and thyroid and bone markers improve in hypothalamic amenorrhea.[82]

Renal

ED Patients can develop dehydration and renal insufficiency due to severe fluid restriction or vomiting. Other renal abnormalities include pyuria and, less commonly, proteinuria and hematuria, which both clear with hydration and reversal of malnutrition.[40] Patients with AN may lose renal concentrating ability, which can result in high urine output and inaccurate specific gravity measurements on urinalyses.[83–86]

Hematologic

Bone marrow hypoplasia is seen in low-weight EDs, primarily leukopenia and anemia, with rare cases of thrombocytopenia. Leukopenia is not thought to increase infection risk, and all dyscrasias resolve with the reversal of malnutrition. It is important to evaluate for iron and vitamin B_{12} deficiency in anemic patients because these are easily reversed with supplementation. Finally, when evaluating for systemic illness, it is important to note that malnourished patients typically have a low sedimentation rate, usually below 5 mm/hr.[16,24,45]

Neurologic

Malnutrition significantly affects the brain in children and adolescents because of the dynamic changes that are occurring in cognitive and structural brain development during this period.[87–101] Severely ill patients with AN have been shown to have reduced brain tissue volume and impaired neuropsychological functioning.[87] One study demonstrated persistent gray-white matter deficits and cerebrospinal fluid levels elevations on magnetic resonance imaging of weight restored patients with AN.[102] However, several studies have demonstrated mixed results in the permanence of neurologic deficits.[87,103] Abnormalities in brain structure have been associated with low body weight and cortisol levels, whereas cognitive deficits are associated with menstrual function.[87]

Psychiatric

Psychiatric comorbidities are common in EDs but may be premorbid, comorbid, or present after recovery. Common disorders are depression, anxiety, obsessive-compulsive disorder, post-traumatic stress disorder, personality disorders, substance abuse disorders, and self-injurious behaviors.[104–110] In AN, the lifetime prevalence of depression and anxiety disorders is 50% to 68% and 30% to 65% respectively.[107] In BN the lifetime prevalence of mood disorders is 50% to 70%, anxiety disorders is 13% to 65%, substance use disorders is 25%, and personality disorders is 20% to 80%.[107]

TREATMENT MODALITIES

In 1995, the Society for Adolescent Medicine issued a statement that the treatment threshold for ED adolescents should be low because of potentially irreversible effects of EDs on growth and development, their mortality risk, and evidence that early treatment improves outcomes.[111] Children and adolescents are triaged to outpatient treatment, partial hospitalization, residential programs, and inpatient hospitalization based on severity of illness, duration of disease, safety considerations, and familial preferences. Treating patients in a home setting is preferred, but other models of care may be necessary and appropriate. In 2005, the American Academy of Pediatrics released criteria for inpatient hospitalization in patients with AN and BN (Table 4), which were reaffirmed in 2010.[16] This review focuses on emerging outpatient treatment modalities.

A paradigm shift in EDs is evident in newer treatment modalities. In older paradigms, patients with EDs were

TABLE 4 American Academy of Pediatrics Criteria for Inpatient Hospitalization in Eating Disorders

Anorexia Nervosa	Bulimia Nervosa
Heart rate <50 beats/min daytime; < 45 beats/min nighttime	Syncope
Systolic blood pressure <90 mm Hg	Serum potassium <3.2 mmol/L
Orthostatic changes in pulse (>20 beats/min) or blood pressure (>10 mm Hg)	Serum chloride <88 mmol/L
Arrhythmia	Esophageal tears
Temperature <96°F	Cardiac arrhythmias including prolonged QTc
<75% ideal body weight or ongoing weight loss despite intensive management	Hypothermia
Body fat <10%	Suicide risk
Refusal to eat	Intractable vomiting
Failure to respond to outpatient treatment	Hematemesis
	Failure to respond to outpatient treatment

thought to develop maladaptive eating behaviors in part because of overly controlling caregivers. This approach focuses on developing insight into the etiology of the disorder in psychodynamically informed individual treatment and/or cognitive behavioral therapy (CBT). These therapies focus on the patient's distorted body image and undue influence of weight and shape with a drive for thinness. A newer paradigm takes into account the biological and genetic contributions to EDs and views caregivers as critical allies in treatment.[112] In this approach, nutritional rehabilitation is considered an important factor in improving cognitions and is the primary initial focus of treatment rather than causation, with age-appropriate insight developing over time. This corresponds with the tenets of family-based treatment (FBT).[113,114]

Evidence for effective treatments in EDs in children and adolescents is growing but remains limited. Primary treatment modalities in pediatric AN are individual therapy, CBT, and FBT. FBT has the largest evidence base of any treatment of efficacy in adolescent and young adult AN populations with multiple clinical trials; there is also growing evidence that it is useful for children under age 12 with restrictive EDs.[113–116] FBT is superior to other currently available treatment methods, but its use is limited by a paucity of qualified practitioners. Although some differences between FBT and individual treatment diminished at long-term follow-up, FBT was more protective against relapse.[115] CBT has been studied in adolescents with BN and shows promise, but there is growing evidence that FBT is also effective[9,117–120]; additional research is needed comparing the 2 methods. CBT has also demonstrated efficacy in BED.[121] In subthreshold disorders, it is recommended that the patient be treated based on the full

syndrome to which their disorder is most similar.[122] Children with textural aversions or swallowing phobias may also benefit from targeted occupational therapy.[123,124] Finally, translational research is underway targeting known deficits in neurocognitive processes, neurotransmitters affected in EDs, and neuroanatomic changes found on imaging studies to tailor treatment and improve treatment response in patients with EDs.[88–101]

Family-Based Treatment

In FBT an agnostic view is taken and the focus is not on the etiology of the disorder. Caregivers are not blamed but instead empowered to refeed their child back to health. The therapist and any other providers are considered consultants to caregivers in this work. Siblings are also supported in this treatment because they frequently have numerous concerns about their sick brother or sister.[125] Additionally, the disorder is externalized from the child to release blame toward the child for their disorder.[114] FBT progresses through 3 phases that target the goals of treatment in children and adolescents with EDs: physical, behavioral and psychological recovery. Phase I of FBT focuses on coaching the caregivers to refeed their child to recovery through specific therapeutic interventions. Food exposures are commonly used to target anxieties and aversions to certain foods or food groups; caregivers are encouraged to incorporate foods their children used to enjoy before the ED rather than to practice avoidance. Once the child is weight-restored, FBT progresses to Phase II, which focuses on gradually transferring developmentally appropriate control of eating back to the child or adolescent. Phase III works on relapse prevention and any other remaining developmental considerations, and then treatment termination. FBT typically is

conducted over a 6- to 12-month time period.[114] Whereas in traditional treatments, fewer than half of AN patients fully recover within 2 to 5 years, a third partially recover, and 20% develop chronic illness,[126] 50% to 60% of patients in FBT achieve full remission within 1 year, another 25% to 35% partially recover (showing improvement but not full remission), and only 15% are nonresponsive to treatment. Thus, FBT is emerging as a first-line treatment in pediatric EDs.[113]

The role of the PCP during FBT is important to the success of treatment.[127] The PCP can serve as a consultant to both the caregiver(s) and the FBT provider, in addition to providing ongoing comprehensive medical assessments and monitoring. It is essential for the PCP to support the FBT provider in explaining the seriousness of the medical complications and prognosis of the ED and the importance of early and aggressive treatment, in addition to removing blame for the ED from either the caregiver(s) or the child. Finally, it is imperative that the PCP support complete weight restoration in the adolescent and full remission of the ED. Skills learned by the PCP in supporting FBT are useful regardless of the modality chosen for ED treatment because they help PCPs align with and remain respectful of caregivers.[113]

Pharmacotherapy

Pharmacologic agents are often used in patients with EDs, despite few studies demonstrating efficacy. There have been no published randomized controlled trials (RCTs) for antidepressant treatment in AN conducted in children and adolescents, and selective serotonin reuptake inhibitors and tricyclic antidepressants have not been shown to be better than placebo in weight gain or improvement in ED symptoms in adult AN. There are also no large RCTs on the use of atypical antipsychotics in the

treatment of AN, although they may be useful in reducing anxiety and rigidity and improving early weight gain. There is no evidence to suggest that pharmacotherapy in AN should be first line, but it may play a role in individual patients resistant to treatment or with premorbid psychiatric conditions.[107,128–130] In BN, several RCTs in adults have found that antidepressants are effective in decreasing binge eating and purging symptoms. Specifically, fluoxetine has a strong evidence base and is approved by the Food and Drug Administration for use in adults with BN; thus far there is some evidence that the effects are similar in adolescents.[107] Other medications such as topiramate and ondansetron are currently being studied for use in adults with BN but are not routinely used.[107] In adults with BED, selective serotonin reuptake inhibitors seem to be effective in short-term reduction of binge eating but do not seem to be superior to CBT alone.[107]

RECOVERY GOALS

Although different metrics for recovery exist in the literature, most agree that behavioral recovery includes normalizing eating patterns and the return of flexibility in eating. Psychological recovery includes improved self-esteem and age-appropriate interpersonal, psychosocial, and occupational functioning. Weight and body shape should no longer have an undue influence on self-evaluation, and normal growth and pubertal patterns are restored.[131–135] Physical recovery includes full weight restoration, return of menses and/or pubertal progression, linear growth if expected, and reversal of most or all organ damage.[136,137] Nutritional restoration involves reaching a goal weight and the ability to eat a varied and balanced diet, but it is important to remember that a "maintenance weight" is often inappropriate in pediatric

populations. Children and adolescents continue to grow and develop throughout puberty and into young adulthood.[113,138] Body composition and activity changes will mandate changes in weight even if a final adult linear height has been achieved. This is an important concept to highlight for parents and patients when working toward recovery.

The determination of goal weight in this population is complex: the provider typically works with a registered dietician experienced in treating EDs and must consider previous weight and linear growth trajectories if previously normal, genetic potential with the use of midparental height, and the median body weight using standardized Centers for Disease Control and Prevention BMI growth curves for height, age, and gender.[138,139] It is important to note that children and adolescents are not "little adults," and because of their hypermetabolic state, once nutrition is introduced, their caloric needs are high, typically between 3000 and 6000 kcal daily. They may remain hypermetabolic for up to 2 years,[113,140] so it is important not to reduce caloric intake prematurely once they reach their weight goal; instead, the treatment team can work on activity increases as development of muscle mass requires continuation of caloric goals. Recent studies have affirmed the safety of more aggressive nutrition approaches in ED treatment.[141–146] RDs can help reinforce these concepts; in newer approaches registered dieticians meet with caregivers to answer questions rather than with patients individually. Caregivers should avoid deferring all nutrition decisions to the registered dietician but rather use this consultation to better empower their efforts at refeeding.

PREVENTION

Developing effective primary and secondary prevention efforts is critical in

EDs because of their high rate of future medical complications, psychiatric comorbidities, and risk of suicidality and relapse.[147] Several features of successful ED prevention programs from a recent meta-analysis of ED prevention programs are described in Table 5[147]; secondary prevention efforts may benefit from targeting caregivers as well.[148] Table 6 delineates 5 public health recommendations focused on shifting the focus from weight to the promotion of a healthy lifestyle for adolescents and their families to facilitate the creation of a positive body image in adolescents.[149] There are growing concerns that an antiobesity focus in pediatric public health may result in an increase in EDs, and future obesity prevention and treatment efforts should track ED cognitions as well as extreme weight control behaviors.[25,26]

TABLE 5 Features of Successful ED Prevention Programs

1. Target high risk adolescents over 15 y of age
2. Deliver intervention by trained individuals
3. Intervention content should include body acceptance and dissonance induction[a]

[a] Involves taking an active stance against the culturally mediated thin ideal, which leads to cognitive dissonance and a shift in belief systems toward an antithin ideal.

TABLE 6 Recommendations for Preventing ED and Obesity for Health Care Providers from Eating Among Teens

1. Inform adolescents that dieting, and particularly unhealthy weight-control behaviors, may be counterproductive. Instead, encourage positive eating and physical behaviors that can be maintained on a regular basis.
2. Do not use body dissatisfaction as a motivator for change. Instead, help teens care for their bodies so that they will want to nurture them through healthy eating, activity, and positive self-talk.
3. Encourage families to have regular, and enjoyable, family meals.
4. Encourage families to avoid weight talk. Talk less about weight and do more to help teens achieve a weight that is healthy for them.
5. Assume overweight teens have experienced weight mistreatment and address with teens and their families.

CONCLUSIONS

EDs in children and adolescents are prevalent and have serious medical and psychological consequences. Children and adolescents have increased potential for long-term complications, thus it is imperative that providers recognize the risk factors and screen for EDs in their patients. Early recognition and aggressive treatment is needed to prevent complications and chronicity. Treatment efforts that focus on weight restoration, reducing blame, and actively incorporating caregivers and families have emerged as particularly effective. The evidence base for ED treatment modalities continues to grow, but to be successful, the treatment team, the family and the PCP must work in collaboration to promote remission and to prevent relapse in this population. Future research is needed to refine treatment in pediatric ED patients and to clarify the role of pharmacotherapy in the treatment of these disorders. Primary and secondary prevention of EDs are also important in improving the health of children, adolescents, and their families.

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