RESEARCH ARTICLE

Classification of Eating Disturbance in Children and Adolescents: Proposed Changes for the DSM-V


Workgroup for Classification of Eating Disorders in Children and Adolescents (WCEDCA)

1Nationwide Children’s Hospital, Columbus, OH, USA
2Great Ormond Street Hospital, NHS Trust, London, UK
3Massachusetts General Hospital, MA, USA
4The Hospital for Sick Children and University of Toronto, Canada
5Western New York Comprehensive Care Center for Eating Disorders, NY, USA
6St George’s Hospital, University of London, Huntercombe Hospitals, London, UK
7Regional Eating Disorders Service, Oslo, Norway
8Stanford University, Pal Alto, CA, USA
9The University of Chicago, Chicago, IL, USA
10Fairleigh Dickinson University, Teaneck NJ and Mount Sinai School of Medicine, New York, NY, USA
11The Children’s Hospital at Westmead, Australia
12Western Psychiatric Institute, Pittsburgh, PA, USA
13Kartini Clinic for Childhood Eating Disorders, Portland, OR, USA
14Cleveland Clinic Children’s Hospital, Cincinnati, OH, USA
15Creighton University, Omaha, NE, USA
16Lund University Hospital, Lund, Sweden
17Duke University Medical Center and Duke University, Durham, NC, USA

Abstract

Childhood and adolescence are critical periods of neural development and physical growth. The malnutrition and related medical complications resulting from eating disorders such as anorexia nervosa (AN), bulimia nervosa (BN) and eating disorder not otherwise specified may have more severe and potentially more protracted consequences during youth than during other age periods. The consensus opinion of an international workgroup of experts on the diagnosis and treatment of child and adolescent eating disorders is that (a) lower and more developmentally sensitive thresholds of symptom severity (e.g. lower frequency of purging behaviours, significant deviations from growth curves as indicators of clinical severity) be used as diagnostic boundaries for children and adolescents, (b) behavioural indicators of psychological features of eating disorders be considered even in the absence of direct self-report of such symptoms and (c) multiple informants (e.g. parents) be used to ascertain symptom profiles. Collectively, these recommendations will permit earlier identification and intervention to prevent the exacerbation of eating disorder symptoms. Copyright © 2010 John Wiley & Sons, Ltd and Eating Disorders Association.

Keywords

eating disorders; classification; children; adolescents; malnutrition

*Correspondence

N. Zucker, Department of Psychiatry and Behavioral Sciences, Duke University Medical Center, P. O. 3842, Durham, NC 27705, USA. Tel: 1-919-308-9140; Fax: 1-919-681-7347.
Email: zucke001@mc.duke.edu

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Introduction

Anorexia nervosa (AN), bulimia nervosa (BN) and related eating disorders are psychiatric syndromes that convey significant risk for medical problems, particularly among youth. AN has a point prevalence of 0.48–0.7% in adolescent females (Ackard, Fulkerson, & Neumark-Sztainer, 2007; Hoek, 2006; Hoek & van Hoeken, 2003). BN occurs in approximately 1–2% of the adolescent population while clinically significant bulimic behaviours occur in an additional 2–3% (Hoek & van Hoeken, 2003). Lewinsohn, Striegel-Moore, and Steeley (2001) indicate that while the hazard rates for a diagnosis of AN peak between the ages of 16–17 years, slopes begin increasing around the age of 10 years old. For BN, a disorder that was previously reported to have a later age of onset than AN, the pattern is similar. The view put forward in this paper is that the current diagnostic classification system requires greater sensitivity to the expression of disordered eating in children and adolescents (Cooper, Watkins, Bryant-Waugh, & Lask, 2002; Nicholls, Chater, & Lask, 2000).

Disturbances in nutrition may be particularly harmful during vulnerable periods of brain development and physical growth. Adolescence is well-recognized as a period of profound alteration in multiple influential domains: the physical changes of puberty, the social changes associated with increased peer influence, the increased opportunities to engage in both maladaptive and adaptive decisions—all against a backdrop of intensified emotional reactivity (Casey, Getz, & Galvan, 2008; Casey, Giedd, & Thomas, 2000; Giedd et al., 1996; Giedd et al., 1999). Many of these changes are instantiated at the neural level. Accumulating evidence supports the observation that extensive neural remodelling and synaptic pruning occurs in the developing adolescent brain (Gogtay et al., 2004). Particularly, brain structures associated with motivated states and emotional experience have been reported to develop more rapidly than distributed neural circuitry associated with cognitive control, a developmental divergence that has been proposed to account, in part, for the increase in maladaptive decisions that occur during this developmental window as emotions hold more sway than rational cognition. Adaptive negotiation of this period leads to flexible, adaptive responding, i.e. the ability to use emotion to guide decisions, not dictate them (Ochsner & Gross, 2005).

To be sure, these are challenges of typical adolescents. Any insult or trauma during this period of vulnerability greatly complicates these developmental challenges, while trauma or neglect prior to this period may cause divergence from a healthy developmental trajectory that becomes potentiated via the maturational processes of adolescence (Cook, Ciorciari, Varker, & Devilly, 2009; De Bellis & Kuchibhatla, 2006). Frank malnutrition or the threat of self-imposed malnutrition (whether or not it is actually realized) are traumatic experiences that threaten the well-being and physical status at any stage of development. It stands to reason that nutritional disturbance or deficit, defining features of eating disorders, during particularly vulnerable developmental periods can result in excessive and potentially permanent physical and psychological consequences (De Souza, 2006). As adequate nutrition is the necessary substrate for all biological growth, the fact that the malnutrition of eating disorders principally begins in adolescence and can also onset prior to adolescence necessitates flexibility in diagnostic boundaries based on developmental stage. Modifications to adult-defined boundaries of illness are needed to address the associated morbidity in the short term and to prevent more serious morbidity in the longer term (Eisle, Dare, Russell, Szmukler, le Grange, & Dodge, 1997; Madden, Morris, Zunynski, Kohn, & Eliot, 2009; Peebles, Wilson, & Lock, 2006). The failure to capture unique age-sensitive nuances of eating disturbance impedes case identification and the opportunity for early intervention. Further, insensitivity to the developmental expression of nutrition inadequacy obscures differential diagnosis and inhibits identification and differentiation of related clinically significant childhood nutritional presentations and disorders (Cooper et al., 2002; Nicholls, Christie, Randall, & Lask, 2001; Watkins & Lask, 2002). This paper is intended to address these important issues by proposing changes to diagnostic criteria delineated in the current edition of Diagnostic and Statistical Manual of Mental Disorders (DSM-IV; American Psychiatric Association, 1994) to reflect pivotal developmental manifestations of symptom expression in children and adolescents. While this opinion was framed while considering the criteria outlined in the DSM-IV specifically, the issues
addressed are also germane to current revisions being considered for revisions to the International Statistical Classification of Diseases and Related Health Problems (ICD-10; World Health Organization, 2004).

**Recommendations**

A realistic goal for the current revision to the DSM-IV is the creation of empirically validated amendments to existing criteria that characterize developmentally sensitive alterations in symptom expression seen in children and adolescents. There are important gaps in the current research literature pertaining to child and adolescent eating disorder diagnosis as summarized by the following research questions. First, do eating disorders described in the DSM-IV appear in childhood in a similar manner to adults? If so, an adult with AN and a child with AN would present with the same features. Second, do currently defined DSM-IV diagnoses appear in childhood and young adolescence, yet manifest in ways developmentally consistent with cognitive and emotional maturity? If so, a child can be diagnosed with AN, but given, as an example, maturational limitations in abstract reasoning, she might be unable to identify or articulate the fears of fatness associated with adult illness manifestations and thus displays a child-specific manifestation of AN. Nevertheless, her behaviour might be entirely consistent with avoidance of any weight gain. Third, do children and adolescents manifest different eating disorders than described in older samples? For example, dietary restriction and weight loss without the stated fear of fatness may be a different psychological entity entirely to AN, and might not be associated with concerted efforts to avoid gaining weight. The working group determined the body of evidence was too limited to adequately adjudicate novel and separate diagnoses in children and adolescents (question 3), but considered this an area for future research. However, there is evidence that, in some cases, eating disorders as expressed in childhood and adolescence differ from their adult-expressed counterparts (question 1). Thus, our goal with this paper is to consider the current body of research in disordered eating in children and adolescents, to view the current diagnostic criteria in light of cognitive and developmental milestones of childhood and adolescence, and to integrate this body of evidence to arrive at proposed developmentally sensitive adaptations of existing criteria (question 2).

**Recommended changes to existing cognitive criteria in AN and BN**

**Recommendation 1:** Behavioural indicators should be permitted to substitute for internally referenced cognitive criteria.

**Recommendation 2:** Wording should be added that alerts healthcare professionals to developmental limitations that may preclude the ability to endorse a cognitive criterion.

Diagnostic criteria for eating disorders that require complex abstract reasoning may not be valid for children or adolescents. Formal thought is defined as the ability to integrate two or more lower-order, concrete processes to arrive at an intangible higher order process (e.g. refusing food and exercising excessively to manage fears of being overweight) (Marini & Case, 1994). In typically developing adolescents, formal thought emerges around 11–13 years old, while complex abstract reasoning continues to evolve into late adolescence (Rosso, Young, Femia, & Yurgelun-Todd, 2004). Capacity for complex abstract reasoning is necessary to meet current criteria. For example, to determine the value of body weight and appearance in the determination of self-worth, an individual needs to rank several abstract constructs (such as trustworthiness, loyalty, attractiveness and interpersonal competence) pertinent to self-evaluation. This capacity to describe internal experiences or compare and articulate multiple abstract concepts are not present in some children and adolescents.

The ability to perceive risk also continues to evolve throughout adolescence (Boyer, 2006). Risk perception requires a child or adolescent to appreciate relative probabilities and to weigh these relative perceptions both immediately and in the future (Boyer, 2006). For example, Criterion C for AN relates to denial of the seriousness of low body weight (Table 1). To perceive the seriousness of low body weight, a child must be able to consider the risk of their current weight relative to the risk of an alternative weight (such as being normal weight or overweight) and to project the long-term serious implications of this weight. While research has been inconsistent, a conservative interpretation is that some preadolescents have difficulty perceiving the
relative risk of alternative outcomes (Levin & Hart, 2003), while both young and older adolescents may exhibit difficulties in appreciating distal negative consequences.

Behavioural indicators may serve as valid substitutes when internal experiences cannot be articulated in a manner consistent with adult presentations. Importantly, we cannot distinguish whether lack of endorsement of a cognition-based criterion is due to developmental insensitivity or to differences in symptom expression. However, given aforementioned limitations in cognitive development, behavioural indicators should be permitted to serve as substitutions for reliable descriptions of internal experiences. For example, severe and determined food restriction or selectivity that is designed to yield a low body weight (e.g., refusal to eat any fat or carbohydrates) could be considered as a proxy for expressed fear of weight gain. There are several precedents for this type of accommodation within the existing body of DSM-IV criteria (e.g., anxiety disorders). Moreover, parents or other caregivers can be enlisted as additional informants in assessing the presence and severity of behavioural indicators of the psychological features of AN and BN, a strategy utilized in measures of other forms of psychopathology in children and adolescents (e.g., depression) (Table 1).

### Changes to weight loss criterion in AN

**Recommendation 3:** Wording should be added to existing weight loss criteria that emphasizes the importance of an individual’s previous growth and maturational trajectory in the determination of healthy weight status rather than population-referenced cut point in the determination of clinical significance.

While extreme weight loss or failure to make expected weight gain is a clinically significant symptom of AN (de Monleon et al., 1998), the sensitivity of current weight criteria in the detection of clinical severity has been questioned by numerous empirical investigations, professional organizations and national treatment guidelines (Hebebrand, Casper, Treasure, & Schweiger, 2004; Nicholls & Stanhope, 2000). Malnutrition has been convincingly demonstrated to negatively impact every system of the body both in individuals with AN and those who are malnourished due to environmental or medical causes (de Monleon et al., 1998; Misra et al., 2004; Misra et al., 2005; Misra et al., 2006; Nicholls & Stanhope, 2000). Of importance, however, is that the timing of severe weight loss may have more damaging effects at certain pivotal periods of development and may argue for more conservative definitions of weight loss during these sensitive periods (Nicholls, Wells, Singhal, & Stanhope, 2002). To illustrate, Peebles, Wilson, and Lock (2006) compared children with AN, defined as 13 years of age or younger, with older adolescent patients. Children were reported to exhibit a lower percentage ideal body weight, a shorter duration of illness, and a shorter temporal trajectory of weight loss (Peebles et al., 2006), highlighting the rapidity with which weight loss may confer potential harm. To be sure, a prepubescent child is likely to have lower fat stores than an adult (Nicholls et al., 2002). Thus, milder percentage weight loss may
have more immediate negative impact such as severe dehydration (Nicholls et al., 2002; Peebles et al., 2006). Of importance, while an adult needs to resolve symptoms to improve health, children and adolescents continue to grow and develop somatic processes with high-energy demands. Increased nutrition is necessary during this critical window to enable these processes to occur. To illustrate, malnourishment during adolescence is associated with low turnover of bone with increased bone resorption without concomitant bone formation, a pattern different from that seen in menopausal women (Lennkh et al., 1999). While weight restoration is an important determinant of bone mineral density, there appears to be a critical window during which such repair can occur (Brooks, Ogden, & Cavalier, 1998; Valla, Groenning, Syversen, & Hoeiseth, 2000). To avoid the negative impact of malnourishment and to reflect the valid nutritional status of a child or adolescent adequately, developmentally sensitive definitions are required to ensure children are able to access appropriate treatment.

Accordingly, an alternative strategy to reliably define ideal weight for children and adolescents is to consider developmental trends in growth and physical maturity for that individual. To be sure, current weight criteria were never meant to be interpreted literally, but rather to be used as a guide to determine when an individual’s weight history warranted concern. In practice, however, these guidelines are far too often literally interpreted with potentially deleterious effects as individuals may be denied access to care by third party payers due to failure to meet an arbitrary defined threshold. Thus, upcoming revisions have two challenges. First, weight criteria should be framed in a manner that is developmentally sensitive. Second, criteria must be framed so that they are clinically useful with no potential for misuse. Combined, such precautions would ensure that those who need care are not denied such treatment based on their weight loss history.

Current diagnostic complexity is due, in part, to the challenge of defining an optimal weight range for a growing child or adolescent. The degree of variation in physical development at any one age during puberty is wide; for example, some children may be prepubertal, while others may have reached full adult maturity by the age of 12. With regard to eating disorders, this range can be widened further, since underweight can result in pubertal delay or interruption. An average 16 year old female, with the onset of menarche at 12.5 years of age, could therefore have reached full adult height and weight, and thus be appropriately compared to centile charts or reported as standard deviations from population medians. However, if the eating disorder onsets early, she may still have the height of an 11 or 12 year old, making weight centiles more difficult to interpret. This is mainly an issue for premenarcheal onset AN, since linear growth decelerates after menarche with an average height gain averaging approximately 7 cm (2.8 inches) (Rosen, 2004). Further complicating this issue is that while there are strong correlations between degree of emaciation and the frequency and severity of the medical sequelae of starvation, there is no ‘threshold’ effect below which signs and symptoms of malnutrition occur (Swenne & Engstrom, 2005).

Reporting nutritional status in terms of BMI standard deviation units or centiles (or %BMI) provides an additional strategy to address problems with interpretation of weight by adjusting weight for height, age and sex. Many countries now have their own national reference centile charts for BMI for age. International BMI cut-offs for child overweight and obesity have been developed, and recently Cole, Flegal, Nicholls, and Jackson (2007) used similar methodology in order to improve international comparison of BMI in underweight growing children. While these guidelines provide a laudable advance in increasing the consistency in the definition of thinness across the developmental spectrum, such reference BMI centiles only account for age and sex and do not take developmental maturity into account. Finally, as evidence of the potential for harm when criteria are interpreted literally, Madden et al. (2009) characterized the expression of early onset eating disorders in a nationally representative sample of 5–13 year old Australian youth. Of importance, while only 51% met current weight criteria as defined by current diagnostic criteria, 61% had life-threatening complications of malnutrition (Madden et al., 2009). Accommodation for individual developmental trends is needed (Table 2).

### Changes to amenorrhea requirement in AN

**Recommendation 5:** Multiple physical systems should be evaluated for the clinical management of eating disturbance, but no single system should be required for diagnosis.
The requirement of amenorrhea in the diagnosis of AN is invalid for prepubescent children (Nicholls et al., 2000) inappropriate for males (Abraham, Pettigrew, Boyd, Russell, & Taylor, 2005), and is not reliably reported by patients (Abraham et al., 2005; Swenne, Belfrage, Thurfjell, & Engstrom, 2005). The inadequacy of this criterion is widely accepted (National Institute for Clinical Excellence, 2004; Society for Adolescent Medicine, 2003) as research investigations with both adolescents and adults do not consistently require this feature in defining their clinical populations, (Lock, Couturier, & Agras, 2006; Walsh et al., 2006). Further, treatment recommendations from professional organizations advise clinicians that while medical signs, symptoms and complications help characterize the clinical picture, they are not sufficient for diagnosis (National Institute for Clinical Excellence, 2004). Thus, what is the role or necessity of menstrual dysfunction in the diagnosis of child and adolescent eating disorders (ED)? The principle here is that for eating disturbance to be clinically significant there must be some evidence of physical risk or impairment. A review of the evidence suggests a middle ground: A recommendation that thorough diagnostic evaluation of children and adolescents take an expansive view of biological systems and consider a diverse array of biological abnormalities as contributing to the clinical picture of child and adolescent diagnosis, but not mandating specific abnormalities within a single system in the body for diagnosis.

The clinical profile of AN complicates determination of sexual maturation rating. In females, the emaciated physical state and low estrogen associated with AN may reduce breast size and distort a healthcare professional’s assessment of sexual maturity rating. In males, similar regression may be witnessed in the effects of lowered testosterone on rating of Tanner stage (Tanner & Preece, 1998).

Dysfunction in diverse physical systems may provide alternative or additional indicators of malnutrition resulting from disturbed eating in children and adolescents (Shamim, Golden, Arden, Filiberto, & Shenker, 2003). Several laboratories have supported the presence of significant medical abnormalities in diverse biological systems in both inpatient (Shamim et al., 2003) and outpatient (Misra et al., 2004) adolescents with ED. Such variation demonstrates that there is not likely to be one specific metabolic profile in children, adolescents or adults with AN. Rather, there may be an array of physiological signs in individuals with AN that aid in diagnosis (Table 3).

### Changes to binge eating criteria in BN and binge eating disorder

**Recommendation 6:** The experience of loss of control irrespective of the amount of calories consumed during an eating episode should be considered the hallmark of binge eating behaviour in children.

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**Table 2 Weight criteria for the diagnosis of anorexia nervosa**

<table>
<thead>
<tr>
<th>AN Criterion A: Refusal to maintain body weight at or above a minimally normal weight for age and height (e.g. weight loss leading to maintenance of body weight less than 85% of that expected or failure to make expected weight gain during period of growth, leading to body weight less than 85% of that expected)</th>
</tr>
</thead>
<tbody>
<tr>
<td>In children and adolescents, consideration should be given to weight and height trends such as deviation from previous growth trajectories, percentage of weight loss and/or BMI centiles for age, rather than reliance on particular weight values in the determination of clinical threshold and severity.</td>
</tr>
</tbody>
</table>

In children and adolescents, the deleterious effects of weight loss may appear at a lower percentage weight lost relative to adults.

**Table 3 Hypothalamic-pituitary axis dysfunction**

<table>
<thead>
<tr>
<th>Anorexia Criterion D: In post-menarcheal females, amenorrhea i.e. the absence of at least three consecutive cycles (A woman is considered to have amenorrhea if her periods occur only following hormone replacement, e.g. estrogen administration)</th>
</tr>
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<tbody>
<tr>
<td>The effects of malnutrition evidence great variability. Clinicians should evaluate changes in multiple systems (e.g. cardiac, endocrine, gastrointestinal) for clinical management but refrain from using a single physical sequelae as a diagnostic requirement (in children and adolescents).</td>
</tr>
</tbody>
</table>
**Recommendation 7:** Binge episodes should be a persistent symptom for diagnosis, but a lower frequency and duration are clinically significant in children. Episodes should occur at least once a month during the previous 3-month period for diagnosis.

Although adolescents with aberrant eating behaviours exhibit many features in common with adults, indicators of disordered eating in children are age-specific and require assessment strategies sensitive to developmental context. In the DSM-IV (American Psychiatric Association, 1994), binge eating is defined by consumption of an excessively large amount of food paired with the subjective experience of a loss of control over eating (American Psychiatric Association, 2000). Associated features of binge eating include the context in which eating occurs (e.g. in private), affective states that follow disturbed eating (e.g. shame and guilt), and the physiological state of the individual (e.g. feeling uncomfortably full following the episode). The assessment of these behaviours and experiences in children requires age-specific strategies. Such strategies include the use of concrete examples and developmentally sensitive metaphors (e.g. a loss of control is similar to ‘a ball rolling down a hill’) (Tanofsky-Kraff, Yanovski, Williley, Marmarosh, Morgan, & Yanovski, 2004). When such strategies are employed, a subgroup of children endorses symptom profiles consistent with adult definitions of binge eating. However, an additional group endorses the subjective experience of loss of control without accompanying excessive food intake. As highlighted by Marcus and Kalarchian (2003), the clinical significance of loss of control relative to the quantity of calories consumed may provide a more developmentally valid index of aberrant eating. Indeed, in cross-sectional studies, any loss of control (i.e. one or more episodes) reported over the previous 4-week period is associated with greater adiposity (Neumark-Sztainer & Hannan, 2000; Tanofsky-Kraff et al., 2004); elevations in psychiatric symptoms such as depression, anxiety and body image disturbance (Glasofer et al., 2007; Tanofsky-Kraff, Faden, Yanovski, Williley, & Yanovski, 2005); excessive weight gain (Tanofsky-Kraff, Yanovski, Schvey, Olsen, Gustafson, & Yanovski, 2009b); and increased vulnerability to the abundant environmental triggers for dysregulated eating such as excessive food availability (Tanofsky-Kraff et al., 2009a). Thus, consistent with the recommendations of Marcus and Kalarchian (2003) and supported by recent findings about the clinical implications of loss of control (Tanofsky-Kraff et al., 2009b), it may be that the experience of loss of control is an important consideration in the determination of clinical impairment irrespective of the amount of food consumed.

Despite similar symptom presentations in children and adolescents relative to adults, lower symptom thresholds are recommended. Research characterizing atypical eating patterns in adolescent BN and comparing threshold to subthreshold forms of this disorder indicate significant overlap in levels of impairment, patterns of psychiatric comorbidity, (Binford & Le Grange, 2005; Eddy, Celio-Doyle, Hoste, Herzog, & Le Grange, in press), and medical sequelae, (Le Grange, Loeb, Van Orman, & Jellar, 2004) supporting the inadequacy of current diagnostic thresholds. Indeed, this finding is not unique to adolescents (Le Grange et al., 2006). Current data suggest that the frequency and duration criteria for binge eating and purging in DSM-IV are not adequate (Crow, Agras, Halmi, Mitchell, & Kraemer, 2002; Sullivan & Kendler, 1998). Further, specific boundaries distinguishing subthreshold BN are extremely varied. Notwithstanding, in children, there is added motivation to consider lower thresholds for diagnosis given the greater impact of similar medical sequelae at fragile developmental stages. Evidence to date supports the clinical significance of at least one episode of loss of control eating occurring for 3 months, a recommendation describing both a lower symptom frequency and shorter duration of symptom expression. This practice is consistent with other childhood diagnoses (e.g. for dysthymic disorder the duration is 1 year instead of 2 years for adults). In sum, aberrant eating in children and adolescents overlaps significantly with adult manifestations, but behaviours do differ. The conservative strategy is to alert the healthcare community to divergent presentations and to consider a lower threshold of severity to protect children and adolescents from harmful sequelae of their disorders (Tanofsky-Kraff, Marcus, Yanovski, & Yanovski, 2008) (Table 4).

**Changes to criteria for inappropriate compensatory mechanisms in BN**

**Recommendation 8:** Lower thresholds of both symptom frequency and duration should be used to designate
clinical levels of inappropriate compensatory mechanisms in children and adolescents.

Given the potential severity of extreme weight loss strategies on growth and development, lower thresholds of symptom severity are needed for children and adolescents (Table 5). There is significant interindividual variation in the response to extreme weight loss behaviours, variation that complicates the determination of a threshold of severity. For example, while some children may manifest electrolyte abnormalities with subthreshold levels of symptom frequency, for others, laboratory results may fail to reveal significant medical sequelae until a chronic disease course has significantly progressed. Given the unpredictability of relating symptom frequency and duration to medical impairment, coupled with the severe cost of ‘missing’ a serious disorder due to insensitive thresholds, the conservative strategy is the consideration of lower symptom thresholds for both symptom frequency and symptom duration. This strategy would alert clinicians to the serious nature of extreme weight loss behaviours increasing the sensitivity of detection of severe clinical problems.

Subthreshold levels of extreme weight loss behaviours are clinically significant, predicting both increased symptom severity as well as concurrent impairment in functioning. For example, in a representative community sample of 2516 adolescents, Neumark-Sztainer, Wall, Guo, Story, Haines, and Eisenberg (2006) report that subthreshold levels of extreme weight loss behaviour predict a one-point increase in BMI relative to adolescents not engaging in these behaviours. Further, the presence of these behaviours at baseline resulted in a 3-fold increase in the likelihood of overweight at follow-up. Individuals who engage in extreme weight loss behaviours were less likely to engage in health-promoting weight maintenance strategies such as fruit and vegetable consumption (Story, Neumark-Sztainer, Sherwood, Stang, & Murray, 1998). Several studies comparing the diagnosis of BN to subthreshold forms of this disorder found no difference in comorbid symptom severity or treatment response (Binford & le Grange, 2005; Le Grange, Crosby, Rathouz, & Leventhal, 2007; Schmidt et al., 2007). These results portend harm: Adolescents engaging in even subthreshold levels of extreme weight loss strategies are positioned on a harmful trajectory predictive of unhealthy weight management and increased eating disturbance and appear to be as ill as threshold forms of BN.

Summary
The purpose of the DSM is to provide clear descriptions of diagnostic categories in order to enable clinicians and investigators to diagnose, communicate about, study and treat people with various mental

Table 4 Binge eating

Bulimia Criterion A: Recurrent episodes of binge eating. An episode of binge eating is characterized by both of the following:(1) 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 during a similar period of time and under similar circumstances. (2) A 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)

In children and younger adolescents, the subjective experience of a loss of control may be a particularly robust indicator of aberrant eating behaviour. However, care must be taken to ensure that children understand what is meant by loss of control through the use of developmentally appropriate assessment strategies such as concrete examples or age-appropriate metaphors (e.g. the experience is ‘like a ball rolling down a hill’ (Tanofsky-Kraff et al., 2004)). Behavioural indicators of loss of control eating such as secretive eating, food seeking in response to negative affect and food hoarding should be considered in children younger than 12 years of age.

Given the prognostic significance of binge eating in children and young adolescents in relation to symptom severity and chronicity, a lower threshold for diagnosis should be considered

Table 5 Compensatory behaviours

Bulimia Criterion B: Recurrent inappropriate compensatory behaviour in order to prevent weight gain, such as self-induced vomiting; misuse of laxatives, diuretics, enemas or other medications; fasting or excessive exercise

In children and younger adolescents, a lower threshold of symptom frequency and duration should be considered due to potential for acute medical sequelae in younger ages.
disorders’ (p. xxxvii) (American Psychiatric Association, 1994). The goal of this consensus opinion is to improve the description of AN, BN and Eating Disorder Not Otherwise Specified as they pertain to children and adolescents, to improve case identification, treatment and research efforts in that segment of the population at highest risk for onset of these serious eating disorders. In diagnosing child and adolescent eating disorders, the data on proposed criteria modifications are convincing and their import compelling, while the breadth of research is relatively small. This is due, in part, to the strategies that have been employed in the ascertainment of childhood eating disturbance, i.e. extrapolating from adult-defined symptoms. While clinically significant but atypical presentations of eating disturbance also appear prominently in the adult population, developmental considerations increase the likelihood of such symptom constellations obscuring true AN or BN ‘caseness’ among children and adolescents. The risks secondary to misdiagnosis during key periods of growth and development are grave. Advances in the diagnosis and research of eating disturbance in children and adolescents can only occur by adopting developmentally sensitive frameworks and methodologies. We recommend examining the topography and expression of symptoms of relevance for eating disorders through the lens of a developmental trajectory, and defining child and adolescent eating pathology at the intersect of deviation from both healthy development and adult-defined diagnostic criteria.

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References


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T. Bravender et al.


