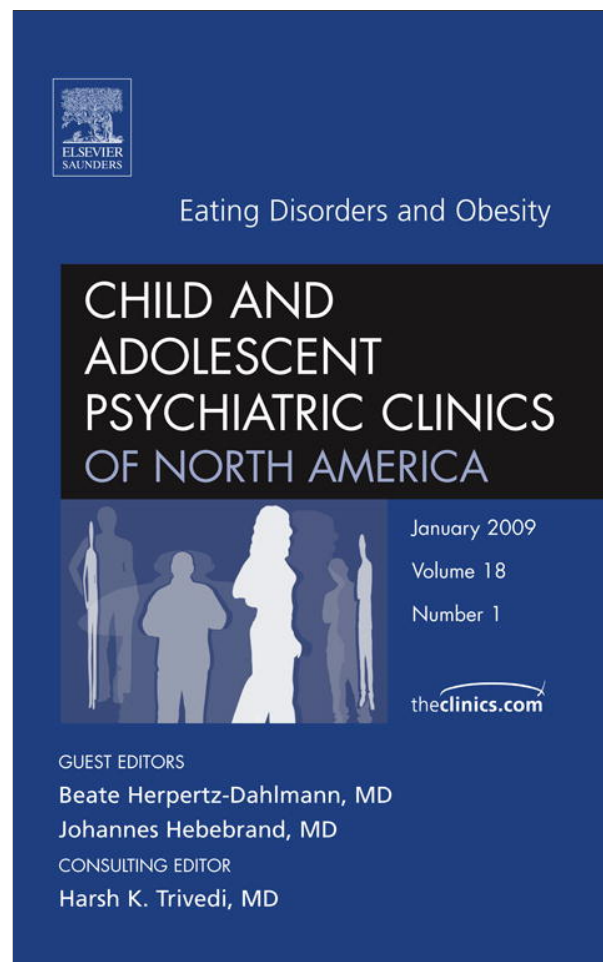


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Adolescent Eating Disorders: Definitions, Symptomatology, Epidemiology and Comorbidity

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KEYWORDS

- Eating disorders • Adolescence • Anorexia nervosa
- Bulimia nervosa • Epidemiology • Comorbidity • Review

Eating is a biological need, the importance of which is taken for granted by most human beings. However, eating disorders have morbidity and mortality rates that are among the highest of any mental disorders and are associated with significant functional impairment. Anorexia nervosa (AN) is the third most common chronic illness of adolescence, and bulimia nervosa (BN) affects more than 1% of adolescent girls.¹ In addition, many adolescents do not fulfill criteria for these distinct diagnoses, but are affected by subthreshold disorders of eating (eating disorder not otherwise specified, EDNOS), which are often as severe and long lasting as the classical conditions. With increasing incidence rates of obesity across all age groups, binge eating disorder (BED) has been a growing concern. Although subsumed under the diagnosis of EDNOS in the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (DSM-IV), binge eating disorder is described in more detail in the article by Hebebrand.

This article provides an up-to-date review on recent developments and increasing knowledge in adolescent AN, BN, and related disorders. It covers diagnoses and assessment, recognition of typical symptoms, medical and psychiatric comorbidities, and current trends in epidemiology (see also, the article by Hebebrand and Herpertz-Dahlmann, elsewhere in this issue).

DEFINITION AND CLASSIFICATION

AN, BN, and EDNOS all are characterized by the same distinct core of psychopathology—a morbid preoccupation with weight and shape. Fear of fatness dominates the

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life of the adolescent, and self-esteem is predominantly contingent on the ability to control one's weight and figure.

Anorexia Nervosa

In AN, the exaggerated wish for thinness leads to significant weight loss; individuals fall below a minimally normal weight for their age and height. Younger AN patients probably have not lost weight, but may weigh less than is expected with the corresponding growth in height. Low body weight is the result of severe and selective food restriction and/or excessive exercise. This exercise is not only voluntary in character, but is seemingly also in part triggered by starvation-induced biological changes.² Most of the patients experience their symptoms as ego-syntonic, and accordingly most, but not all, do not regard their state as an illness and deny serious associated medical complications. Weight loss is pursued beyond the bounds of reason and to the exclusion of other age-appropriate activities. It is often followed by social isolation and withdrawal, and emaciation is mostly interpreted as an achievement without feelings of remorse or suffering.

According to the DSM-IV, AN sufferers can be classified into two subgroups: restricting and binge eating/purging type. Patients with binge eating/purging type may engage in bingeing *and* purging, *only* bingeing (with intermittent periods of fasting or of excessive exercising), or *only* purging (ie, practicing self-induced vomiting, laxative abuse, or other extreme forms of weight control). It is still under debate whether the binge eating/purging type is associated with a worse outcome than the restricting type.^{3,4}

In contrast to AN in adults, the diagnosis of adolescent AN should take into account normal pubertal growth and growth-related increases in weight. For this reason, a fixed body mass index (BMI, calculated as weight in kilograms divided by height in meters²) criterion in children and adolescents is misleading; instead BMI centiles should be used to define "underweight." In Germany, a BMI lower than the 10th percentile is considered the threshold for a diagnosis of AN. If the patient has a BMI lower than the third percentile, many clinicians believe that she should be admitted to inpatient treatment (for a more detailed survey on weight and its definitions see Hebebrand).

Amenorrhea may be primary, because menarche will be postponed by starvation. Moreover, an adolescent will be considered to have amenorrhea when her menstruation occurs only after administration of sexual hormones (**Box 1**).

In anticipation of the revision of the DSM, there has been a lot of discussion about the usefulness of DSM-IV (and Tenth Revision of the International Classification of Diseases [ICD-10]) criteria for AN.

First, the hallmark criterion of AN—that is, maintenance of low body weight—is also found in some healthy individuals.^{5,6} In addition "refusal to maintain body weight at or

Box 1

Diagnostic criteria for AN according to DSM-IV (abbreviated form)

- Refusal to maintain a minimal body weight for age and height (less than 85% of that expected)
- Intense fear of gaining weight or becoming fat
- Disturbance in the way in which one's body weight or shape is experienced
- Amenorrhea

Subtypes: Restricting and Binge Eating/Purging Type.

above a minimally normal weight,” implies an active and purposeful process. However, at least at some point in the course of starvation, many patients admit that they are no longer in control of their body weight, and in several cases, an unintentional weight loss (eg, by a somatic disease) marks the beginning of the disorder. Criterion B according to the DSM-IV (fear of gaining weight) relies only on thoughts or feelings and cannot be easily objectified. Moreover, patients with a low premorbid body weight obviously demonstrate little drive for thinness,⁷ and although some of the patients do not deny the seriousness of their current condition (criterion C), they do not want to change it. Amenorrhea (criterion D) is not unique to AN, but is found in many conditions linked to a reduced energy intake. Furthermore, patients who still menstruate psychopathologically closely resemble those in their course of illness who do not.⁸

In sum, several investigators have recently expressed the need for alternative diagnostic criteria for AN that are more empirically based and endorse observation, than relying on interpretations of the patient's behavior (eg, see Bulik et al,⁵ and Hebebrand et al⁶).

Bulimia Nervosa

Bulimic patients are mostly within the normal weight range, although many have a body weight in the low normal range and some are slightly overweight. In BN, periods of dieting and fasting are interrupted by binge eating episodes accompanied by a feeling of loss of control. During binges, a large amount of food (“more than most people would eat in similar circumstances and similar periods of time”) is consumed. Bingeing is compensated by self-induced vomiting, laxative, diuretic, or other medication abuse or – more rarely – by non-purging strategies like exercising and dieting. Binges and purging behaviors are practiced mostly in secret. Patients are reluctant to seek help and feel ashamed of their behavior. Similar to AN, a morbid fear of becoming fat and overvaluation of shape and weight are core symptoms of BN. BN (**Box 2**) is also divided into two subgroups, the purging and non-purging type (see earlier discussion).

The frequency of binge attacks needed to make the diagnosis is based on convention. In the current DSM, a minimum frequency of 2 binges per week is required. However, there is no evidence that this is a valid criterion, because individuals with fewer binge attacks (eg, once a week) may have similar psychopathology or outcomes.⁹ In addition, there is a lack of specification for the “amount of food that is larger than most people would eat.” According to observations by Keel and colleagues,¹⁰ several self-identified binge-eaters did not eat more during a so-called “attack” than did “normal” college women, and those with binge attacks that were not objectively large did not differ from those with large binge attacks in terms of general and specific

Box 2

Diagnostic criteria for BN according to DSM-IV (abbreviated form)

- Recurrent episodes of binge eating
- Recurrent inappropriate compensatory behavior, for example, self-induced vomiting, laxative abuse, or fasting
- Frequency of binge eating at least twice a week for 3 months
- Self-worth is contingent on shape and weight
- Bulimic symptoms do not exclusively occur during the context of AN

Subtypes: Purging and non-purging type.

psychopathology. Moreover, the binge-eating criterion is questionable because of the lack of empiric evidence for its sub-criterion “loss of control.”

In sum, several of the diagnostic criteria for BN involving the frequency and duration of bingeing and the amount of food consumed are questionable because of little empiric support. In addition, some investigators support the subtyping of BN into two categories (with and without a history of AN) because patients with a history of AN seem to be more likely to have a protracted illness with repeated relapses into AN.¹¹

Eating Disorder Not Otherwise Specified

NOS diagnoses are residual categories by definition. Consequently, EDNOS is a heterogeneous diagnosis merging all those patients who narrowly miss full criteria for AN or BN, in addition to those with broader eating disorder spectrum symptoms (“partial ED”). According to Fairburn and colleagues Cooper¹² and other investigators, EDNOS—although a “residual” category—is “the most common eating disorder diagnosis encountered in clinical practice.” This fact is not only true for adults, but also for adolescents. In their comparison of different classification systems for eating disorders in childhood and adolescence, Nicholls and colleagues¹³ reclassified 226 children referred to a specialist clinic between the age of 7 and 16 years according to DSM-IV criteria. More than 50% of their sample fell into the DSM-IV category of EDNOS. In a very recent US study,¹⁴ 280 adolescents (mean age: 16 years, range: 12–19 years) seeking outpatient treatment through a specialized service were assessed for the diagnosis of an eating disorder. Again, more than 50% presented with EDNOS, most of whom could be best described as subthreshold AN or BN. Moreover, the investigators reported that individuals with EDNOS had higher levels of specific eating pathology and general psychopathology than those with DSM-IV AN. Moreover, the natural course did not differ between young women with partial or full syndrome of AN or BN.¹⁵ In our own investigation with a large representative sample in Germany comprising 1,800 11- to 17-year-olds, about one-third of the girls and 15% of the boys reported a partial eating disorder syndrome, which was most prevalent in overweight youth.¹⁶ There was also a significant association between partial eating disorder syndromes and psychopathology, including internalizing and externalizing behavioral problems.

Classification of Childhood and Adolescent Anorectic and Bulimic EDs

It has already been emphasized that many children and adolescents have to be classified in the residual category of EDNOS, but for different reasons from adults. Younger subjects are often not able to describe or even understand the nature of their disturbed eating behavior. Because several of the diagnostic criteria for DSM-IV AN or BN rely on feelings or thoughts, many of these youngsters will not fulfill them. Moreover, young individuals often fail to recognize the harmful nature of weight loss and purging behaviors and feel overwhelmed by their fear of gaining weight. The importance of weight- and shape-related self-esteem to core symptoms of AN or BN often cannot be validated in younger age groups. Additionally, the insubstantial usefulness of a fixed BMI criterion in developing weight-losing subjects has already been discussed.

Potential strategies have been proposed for changing classification criteria in childhood and adolescence. They include an amendment to classic eating disorder criteria for this age group, a specific classification category for eating disorders with onset in childhood or adolescence, or a broad classification, whereby a subcategory of eating

disorders in young age could be subsumed. Finally, diagnostic criteria could be primarily based on objective parameters like certain behaviors antagonistic to the maintenance of a normal body weight (eg, reduced intake, such as fasting or purging), elevated energy expenditure (eg, exercise) or physiologic measurements indicating semi-starvation (subnormal leptin levels, bradycardia, low T3⁶) (for a review see reference 17).

SYMPTOMS

As mentioned above, *anorectic* patients are intensely preoccupied by thoughts of food and their fear of fatness. The disorder almost always begins with dieting: girls often become vegetarians, skip meals, and confine themselves to “healthy stuff” like fruit, vegetables, and whole grain bread. A minority of anorectic patients are overweight before the onset of their illness. Many individuals celebrate their small meals, take a long time to finish them, and practice rituals while eating. Others have strange eating habits and cut their food into small pieces, rearrange food items several times on their plates, or take very small bites. Some develop an extensive interest in recipes and cooking. Younger adolescents or children may even refuse to drink because of an overwhelming fear of becoming fat.¹⁸

Several times a day these girls step on the scales, look in mirrors to assess their shape, and express concern about weight gain. Paradoxically the pursuit of thinness escalates despite the increasing weight loss. Continuous weight loss is considered a triumph. Many of the patients are physically hyperactive and practice sports or commit themselves to fitness training or gymnastics. Exercising and stepping on the scales can become highly ritualized and obsessive. Although mostly content and cheerful in the beginning, anorectic individuals often develop depressive symptoms and social withdrawal with increasing starvation. Several of them have delayed psychosexual development and an age-inadequate bonding to their family.

Moreover, many of these symptoms are not voluntarily driven, but are induced by prolonged semi-starvation. There is an inverted u-shaped relationship between leptin and activity levels, suggesting that hyperactivity is largely triggered by hypoleptinemia.¹⁹

In the so-called “Minnesota-experiment” conducted in the 1940s, many of the “typical” symptoms of anorexia or BN could be provoked in healthy young men during a 24-week laboratory–semi-starvation program.²⁰

Bulimic patients are usually older than anorectic patients. About one-fourth have a history of AN. They enter the bulimic cycle of fasting, bingeing, and purging by giving in to the intense urge to eat during a period of starvation. Only rarely does the binge–purge cycle begin de novo. Failure to adhere to a planned small amount of food is often followed by a binge attack to have enough food in the stomach to make vomiting easier. For the same purpose patients drink a lot of water during meals. A binge attack might comprise up to 11,000 kcal and mostly consists of food that is easy to swallow without much chewing. In contrast to anorectic individuals, many bulimic patients admit to having a strong appetite and a desire to eat. Overeating is nearly always a solitary and secretive habit. Although binge attacks are often provoked by emotional stress or feelings of emptiness at the beginning, they become more and more habituated during the course of the disorder, which makes treatment more difficult.

Although most bulimic patients are of normal weight, mild, moderate, and severe forms of obesity have been noticed in bulimic patients. Amongst a community-based cohort of bulimic females, childhood and parental obesity were reported in more than

one third of the cases, which significantly exceeds the respective rates in healthy and psychiatric controls.²¹

EPIDEMIOLOGY

It has often been proclaimed in the media that eating disorders are on the rise. However, changes in referral practices and diagnostic criteria often influence the results and lead to discrepant interpretations.

Anorexia Nervosa

Previous studies indicate that there was a global increase in the prevalence and incidence of AN until the 1970s, with more stable rates since then.²² In adolescents and young women, most studies found a *point prevalence* rate for AN according to DSM-IV between 0.3 and 0.9%.²³ These rates were assessed according to the current standard of a 2-stage selection model.²³ In the first step, a large population is investigated by means of a screening questionnaire to select the at-risk individuals. The persons at risk are then interviewed personally together with probands from a randomly selected sample of the general (not at risk) population (second step). In a recent study of 12- to 23-year-old girls and women attending public schools in Portugal, the exact point prevalence rate for adolescent AN was 0.39%.²⁴

Lifetime prevalence rates for 20- 40-year-old women are estimated to be between 1.2% and 2.2%.^{23,25} In community studies, it has been estimated that up to 50% of diagnosed cases are previously undetected by the health care system.²⁵

Recent *incidence rates* show an overall stabilization, but report an increase in the adolescent and young adult group. In a recent study from England, the general incidence rate derived from general practitioners was 4.7/100,000 persons (CI, 3.6–5.8) in the year 2000;²² in a Dutch study it was estimated to be 7.7/100,000 (CI, 5.9–10.0²³) during the years 1995-1999. In all studies, incidence rates were highest for female adolescent girls between 15 and 19 years of age. According to Hoek,²³ they constitute about 40% of all identified new cases. In the Netherlands a significant increase from 56 to 110/100,000 was observed in this age group between the time periods of 1985 to 1989 and 1995 to 1999.²⁶

Bulimia Nervosa

For BN, the *point prevalence* is calculated as being 1% to 2%. The rates differ substantially depending on the level of medical care (epidemiologic estimates vs. clinical estimates) with only a minority of individuals seeking mental health care.²³ In the above mentioned study based on data derived from general practices in the UK,²² there was an increase in the *incidence* of bulimia up until 1996 followed by a decline to 6.6/100,000 persons in 2000. For 10- to 19-year-old girls and women, the incidence was rated 35.8/100,000 (CI, 22.3–48.6). The decrease in incidence of BN was confirmed by a recent US study that investigated prevalence rates from 1982 through 2002²⁷ and by a 2-stage survey (see above) reporting a prevalence rate of only 0.3% in Portuguese adolescents.²⁴ However, it has to be kept in mind that changes in patients' referrals or service use (eg, increase in self-help groups vs. medical advice) and a reduced recognition of symptoms according to a decreased media representation could bias these findings.

Very few studies report epidemiologic data for men. The men-to-women ratio for AN is estimated at about 1:10 to 1:15 and at 1:15 to 1:20 for BN.^{22,27} According to recent studies, there has not been appreciable change in this ratio over time.

Eating Disorder Not Otherwise Specified

Although EDNOS represents the most common eating disorder in specialized eating disorder services, there are very few reports on its prevalence and incidence in the general population.

A recent 2-stage survey of a nationwide representative sample of 2,000 12- to 23-year-old Portuguese girls and women²⁴ (see earlier discussion) found a prevalence rate for EDNOS of 2.37%, using a reliable well-known screening instrument (EDE-Q) and an expert-rated interview (EDE).

COMORBIDITY

Medical Comorbidity

Eating disorders have a high rate of medical complications. In AN, mortality is significantly increased with 5% to 6% of patients suffering a premature death.³ In BN, the mortality rate is lower and is estimated to be about 2%.²⁸

The annual costs of treating eating disorders in the US²⁹ and in Germany³⁰ are very high and are even more expensive than the treatment of schizophrenia. For AN, the cost of illness in Germany based on data from the year 1998 was estimated to be about €195 million Euros (direct costs of €73 million for hospitalization and rehabilitation services, indirect costs of €124 million Euros for inability to work and premature death) and €124 million Euros for BN (€12 million direct and €112 million indirect costs).³⁰

The severity of comorbid medical illness commonly depends on the rapidity and extent of weight loss, the current degree of underweight, the duration of the eating disorder (eg, habituation to starvation), and the intensity of purging. In addition to typical medical problems in adults, AN has a severe impact on growth and pubertal development in children and adolescents. The most important physiologic changes and complications (but not all) are given in **Table 1**.

AN, and to a lesser extent BN, lead to a variety of endocrine changes. In general, these abnormalities are a consequence of semi-starvation, of abnormal eating behavior, or of both with poorly balanced meals and thus are mostly assigned as adaptive mechanisms to conserve energy and protein. Amenorrhea is one of the core symptoms of AN and is indexed as a diagnostic guideline in both the ICD-10 and DSM-IV. Besides the alterations of the hypothalamic–pituitary–gonadal axis, disturbances of hypothalamic–pituitary–adrenal axis, hypothalamic–pituitary–thyroid axis, and hypothalamic–pituitary–growth–hormone–IGF-1 axis are typical sequelae of adolescent eating disorders. Many of these endocrinologic changes are associated with changes in neuropeptides, such as leptin or ghrelin, which is described in more detail by Hebebrand Müller and colleagues, elsewhere in this issue.

Osteopenia and osteoporosis

Adolescents with eating disorders are at risk to develop osteopenia or osteoporosis associated with a 2- to 7-fold higher fracture risk in later life. Most of the bone mass is built up during adolescence. Any process interfering with normal bone mineral accrual in this critical period may lead to permanent deficits. Multiple factors contribute to a reduction of bone mass in AN (and to a lower extent in BN): hypogonadism, hypercortisolemia, low levels of insulin-like growth factor, malnutrition (eg, low protein or calcium intake), or excessive exercise, and bed rest (eg, as a “therapeutic strategy” during inpatient treatment). In addition, a critical role of neuropeptides like leptin, ghrelin, and peptide YY in bone metabolism has been discussed.² Normalization of body weight is believed to be the most important factor in counteracting bone loss in adolescent AN. However, there is still an ongoing debate about whether recovery from an

Table 1		
Medical alterations in adolescent eating disorders		
	AN	BN
Physical examination findings	Dry skin, lanugo hair formation (only with severe weight loss), acrocyanosis, alopecia, low body temperature, dehydration, retardation of growth and pubertal development	Erosion of dental enamel, parotid/salivary gland enlargement, scars on the skin of the back of the hand resulting from inducing the gag reflex, dehydration
Cardiovascular system	Bradycardia, ECG abnormalities (mostly prolonged QT-interval), pericardial effusion, edema (before or during refeeding)	ECG-abnormalities (cardiac arrhythmia, prolonged QT-interval)
Gastrointestinal system	Impaired gastric emptying, pancreatitis, constipation	Esophagitis, pancreatitis, delayed gastric emptying
Blood	Leukocytopenia, thrombocytopenia, anemia	
Biochemical abnormalities	Hypokalemia, hyponatremia, hypomagnesiemia, hypocalcemia, hypophosphatemia (during refeeding), low glucose levels, AST↑, ALT↑ (with severe fasting or beginning of refeeding), cholesterol ↑	Hypokalemia, hyponatremia, hypomagnesiemia (caused by diarrhea), hypocalcemia, metabolic alkalosis (in case of severe purging), metabolic acidosis (in case of severe laxative abuse)
Endocrine system	Cortisol ↑	n (↑)
	FSH, LH ↓	n (↓)
	Estradiol ↓	n (↓)
	FT3 ↓	n (↓)
	FT4 n (↓)	n (↓)
	TSH n (↓)	n
	GH ↑(n)	n (↑)
	IGF-1 ↓	n (↓)
	Leptin ↓	n (↓)

eating disorder is in fact associated with a complete restoration of bone mass³¹ (for a review, see Misra and Klebanski³²).

PSYCHIATRIC COMORBIDITY

It is well known that both AN and BN are often accompanied by other psychiatric conditions – either during the acute state or in the long-term course. The most frequent disorders are depression (major depressive disorder—MDD and dysthymia), anxiety disorders with a special emphasis on obsessive-compulsive disorder, substance abuse, and personality disorders. Several of these disorders are substantially affected by starvation and abnormal eating patterns.³³ Thus, one should always question whether depressive or anxious states are primarily the result of the physiologic consequences of malnutrition or had set in before the onset or after recovery from the eating disorder.

In addition, it should be kept in mind that rates of comorbidity are usually significantly higher in clinical settings than in field studies. This point is especially true for comorbid affective illnesses that enhance the probability of an individual seeking treatment. Thus, prevalences in most clinical samples are subject to a referral bias.

Affective Disorders

A wide range of depressive symptoms such as depressed mood, emotional emptiness, social withdrawal, loss of libido, and low self-esteem are prominent in malnourished anorectic patients. Studies that have used structured diagnostic interviews according to DSM-III-R or DSM-IV have reported a wide range of estimates, between 15% and 60%, for the percentage of treatment-seeking individuals with AN who meet lifetime criteria for a depressive disorder.³⁴ In adolescent AN, up to 80% of patients suffer from MDD during the acute stage of the illness^{35–37} with no significant difference between epidemiologic and clinical samples.³⁸

In bulimic patients, prevalence rates of MDD are similar to AN.³⁹ Keys and colleagues²⁰ were some of the first scientists to note mental changes during semi-starvation, such as emotional irritability, loss of libido, anhedonia, and difficulty making decisions. These changes may not only be evident in AN and BN patients with substantial weight loss, but also in bulimic girls with “only” erratic eating behavior. Thus, therapists should always wait for the effect of refeeding (because the mood of patients tends to lift with weight gain) or absence of purging before they start a specific “antidepressant” treatment.

There also seems to be a high prevalence of mood disorders in family members of eating disordered patients. However, a meta-analysis of several of these family studies revealed several methodological problems, so that the value of the results has to be questioned.⁴⁰ There is still an ongoing debate about whether shared genetic factors contribute to a common etiology of depression and eating disorders,⁴¹ family study and twin data do not always support a shared genetic diathesis.⁴²

Earlier investigations indicate that depression sometimes antedates the eating disorder^{43,44} and may even begin in childhood.⁴⁵ Others suggest that the eating disorders precede depressive disorders.

In the latter case, the eating disorder may in fact produce depressive symptomatology by effecting neurobiological changes. In the author's work on the effects of steroid hormones on the anatomy of the developing brain during puberty, growth of limbic structures was significantly associated with increasing estrogen levels.⁴⁶ Consequently, one could imagine that anorectic patients with long-lasting deficient estrogen levels are more vulnerable to typical psychiatric disorders associated with dysfunctions in limbic brain areas, such as mood or anxiety disorders.

The incidence of *suicide* and *suicidal attempts* is also a serious problem in eating disorders. About 10% to 20% of individuals with AN and 25% to 35% of patients with bulimia report a history of attempted suicide. Those with purging or bingeing/purging behavior admit to significantly more suicide attempts than those with the restricting subtype. Suicidal behavior is also related to severity of the eating disorder and the presence of a cluster-B personality disorder.^{47,48} The standardized mortality rate (representing the ratio of the observed number of deaths to the expected number of deaths in a matched population) for suicide in AN is estimated to be up to 5, whereas it does not appear to be elevated in BN. Unfortunately, data regarding rates for completed suicide and suicide attempts are lacking in adolescent AN and BN.

Anxiety Disorders (Other than OCD)

A wide range of anxiety disorders are common in patients with AN. Comparable to findings in depression estimates of comorbidity between 20% and 60% are suggested.^{34,49,50} In adolescent AN, similar rates are found.³⁵

During the acute phase of the illness, fears associated with eating, such as consuming certain foods, changing shape, and an avoidance of situations involving eating (eg, eating in a restaurant, at a party, at school), are prominent. In addition, typical anxiety disorders, such as agoraphobia, panic disorder, general anxiety disorder, PTSD, and especially social phobia are significantly more frequent in individuals with AN than in the general population.

In a study by Godart and colleagues⁵¹ using a predominantly adolescent sample, the most frequent lifetime anxiety disorder was social phobia (55%) followed by simple phobia (45%). However, the sample size was rather small, and it was based only on retrospective diagnoses.

At the time a comorbid anxiety disorder is present, it commonly predates the eating disorder and has its onset in childhood.^{49,51} In a very interesting study by Shoebridge and Gowers,⁵² the investigators reported significant differences between anorectic patients and healthy controls concerning “high concern” of mothers and separation anxiety in the children: anorectic patients had more infant sleep difficulties, severe distress at first separation from their parents, and a later age for first sleeping away from home. Mothers of patients reported higher trait anxiety levels and higher personal engagement in child care without the participation of the father in contrast to the control mothers.

In BN, prevalence rates of anxiety disorders also vary between 25% and 75% (see Swinbourne and Touyz⁵³ for a review) with social phobia again being the most prevalent disorder, present in up to 70% of cases.⁵³ Because bulimic patients are usually older than anorectic individuals, prevalence rates in exclusively adolescent samples are difficult to find.

In sum, current literature suggests that there is an important association between eating disorders and anxiety disorders that probably carries significant implications for etiology and treatment. Because of the high prevalence of social phobia in adolescent AN, the authors have implemented social competence training in the treatment protocol, containing situations with and without eating in public.

Obsessive-Compulsive Disorder (OCD)

Numerous studies have revealed a high rate of obsessive-compulsive disorder among patients with eating disorders. Some have even argued that eating disorders, especially AN, are variants of the obsessive-compulsive spectrum of illness.

For the restrictive anorectic patient, food-related obsessive-compulsive features, such as cutting food into a certain number of pieces, eating vegetables separately from meat, or having meals exactly at the same time of the day, are very common. As has been demonstrated by Keys and colleagues,²⁰ food-restricted rituals arise in laboratory-induced starvation states and are alleviated with refeeding.

However, there are additional thoughts and behaviors in eating disordered patients that would qualify for a comorbid diagnosis of true OCD. The most frequent phenomena identified in eating disordered patients are ordering and washing rituals and obsessions with things going wrong. Anorectic individuals are also often characterized by certain traits such as rigidity, perfectionism, and scrupulosity.

There is still an ongoing debate as to whether prevalence rates of OCD are higher in restrictive AN than the binge-purge type or when compared with BN. However, recent

studies have found similar rates of comorbid OCD in both AN and BN.⁴⁹ In the latter study, about 40% of the adult individuals received a lifetime diagnosis of OCD. In adolescent samples, about 20% of anorectic individuals suffer from OCD.^{35,51} The onset is mostly in childhood. In the study by Kaye and colleagues,⁴⁹ 23% of the subjects with eating disorders had childhood-onset OCD in contrast to only 2% of the community sample.

Although OCD co-occurs in individuals and families with eating disorders, there are still no distinct data that would clearly support a shared genetic etiology.⁴²

In sum, although the findings of prevalence rates of anxiety disorders are strikingly inconsistent, the comorbidity of both disorders brings up many important questions regarding the nature of the relationship. Childhood anxiety disorders (including OCD) may represent a risk factor for later eating disorders, suggesting that preventive strategies might be of value in this specific population. In addition, anxiety disorders are often still prominent in recovered eating disordered subjects,^{54,55} so that specific treatment strategies that also address anxiety disorders are warranted.

Substance Abuse

Substance abuse and eating disorders co-occur at a rate that is greater than by chance; however, the risk for comorbidity is lower than that of anxiety or mood disorders. For AN, it is important to distinguish between the binge-purge and the restricting subtype, because there seems to be no elevated risk for substance abuse in the latter. This was confirmed by a recent study on personality traits and comorbid disorders, which demonstrated that the obsessional (perfectionistic) personality type was protective against the development of substance abuse.⁵⁶

Unfortunately, few investigations have examined substance abuse in adolescent samples. In 1 study, among adolescent girls who were AN patients, about 8% suffered from substance abuse.³⁵ The disorder was 18 times as likely to co-occur with the AN binge-purging type than with the restricting type. In an adolescent sample suffering from bulimia, nearly two-thirds of individuals had tried alcohol at least once. A total of 40% of this sample reported using alcohol more than once per month, with 4% using it 2 to 4 days per week.⁵⁷ About one-third had tried illegal drugs at least once. The most commonly used drug was marijuana followed by cocaine (use occurring in about one-third of individuals with substance abuse) and amphetamines. Note, however, that both of these studies were executed with clinical populations.

Some researchers have hypothesized that shared impulsivity between bulimic disorders and substance abuse could be a common cause for both disorders, and some time ago Lacey and Evans⁵⁸ created the term “multi-impulsive bulimia.” Impulsivity in bulimia might also account for other “high risk behaviors” like shoplifting, self-injury, and suicide attempts.⁵⁹

Attention-Deficit Hyperactivity Disorder

Recent studies underline an association between ADHD and eating disorders. In a large prospective case-control study, 123 girls with ADHD and 106 controls were followed into late adolescence. Girls with ADHD were 3.6 times more likely to meet criteria for an eating disorder than control girls and 5.6 times more likely to fulfill criteria for BN.⁶⁰ Girls with both disorders also had increased rates of mood, anxiety, and disruptive behavior disorders. A similar result was obtained in a 5-year longitudinal study by Mikami and colleagues.⁶¹ The group of ADHD girls was divided into ADHD-combined and ADHD-inattentive subtypes, each of which was compared with controls. Girls with the combined subtype of ADHD were at the highest risk for bulimic behavior

and body image disturbances followed by the inattentive subtype. Childhood impulsivity symptoms best predicted adolescent eating problems.

Thus, girls with ADHD should be monitored for the development of an eating disorder in adolescence. Unfortunately, to the author's knowledge, there are only case reports, but no controlled studies that have investigated women with bulimic behavior for ADHD rates.

Personality Traits and Disorders

As has already been mentioned here, perfectionism, rigidity, and obsessiveness seem to be robust personality traits in AN patients, which are evident both during acute illness and after long-term recovery.^{55,62} Some investigators even hypothesize that perfectionism and obsessiveness might be a risk factor in childhood for later AN.^{49,63} In addition, anorectic individuals report high levels of anxiety, harm avoidance, and feelings of worthlessness, even after recovering from their eating disorder (data collected at least 1 month after recovery). Accordingly, the most prevalent personality disorders in adult AN patients are cluster-C disorders, which include obsessive-compulsive and avoidant or dependent personality disorder.^{53,55}

Perfectionistic traits are also often observed in bulimic patients.⁶⁴ As has been mentioned earlier, many bulimic individuals display impulsive or sensation-seeking behavior. Thus, in addition to cluster-C personality disorders, patients with the binge eating/purging subtype of AN and those with BN often receive a cluster-B diagnosis, such as borderline personality disorder.⁵⁰

As with other psychiatric disorders, personality disorders commonly predict a negative long-term course for both eating disorders.⁶⁵

ASSESSMENT

In typical AN, it is easy to make a diagnosis. However, early signs of the illness are sometimes difficult to recognize (**Box 3**). In contrast, diagnosis of BN is more complicated, because many patients feel ashamed of their behavior and deny their symptoms.

At the time a patient is admitted to a mental health or pediatric service, a full medical and psychometric assessment is necessary for diagnosis, treatment planning, and judgment of progress. The patient should always be weighed and should have her height measured. She should be asked about her ideal weight and how she feels about her actual shape. The clinician should obtain information about the highest weight in the past and the extent and rapidity of current weight loss. Frequently, patients do

Box 3

Early symptoms of eating disorders

- Growing interest in composition of food and calorie content
- Avoidance or skipping of main meals
- Restriction to healthy food
- Frequent weighing
- Increased energy or physical restlessness
- Discontent with weight and shape
- Increasing achievement motivation and social isolation

Box 4**Medical assessment recommended for eating disorders**

- Physical assessment (heart rate, blood pressure, body temperature)
- Complete blood count
- Biochemical profile (sodium, potassium, calcium, chloride, magnesium, phosphate, creatinine, urea, serum proteins, glucose, liver enzymes, amylase)
- ECG
- EEG, MRI, CT (in case of atypical eating disorder eg, men, children, or manifestation of seizures)

not seek medical advice for their eating disorder, but complain of hair loss, brittle nails, constipation, headache, or fatigue. The medical care provider or therapist must be patient in obtaining the patient's history of dietary restraint and other eating disorder symptoms, such as vomiting and purging, and menstrual irregularities. A thorough medical examination is recommended (**Box 4**). Some therapists prefer to have an anorectic patient weighed by the family practitioner or some other clinician not engaged in the treatment of the patient. However, restoration of body weight is one of the main targets of treatment, and the act of weighing may convey valuable information about the patient's body image concerns and weight phobia.

Various assessment methods have been developed for the measurement of specific eating disorder psychopathology. Valuable strategies comprise structured or semi-structured clinical interviews, self-report questionnaires, and self-monitoring. Commonly used clinical interviews include the Eating Disorder Examination (EDE⁶⁶) and the Structured Interview for Anorexia and Bulimia Nervosa (SIAB⁶⁷). Probably the most widely used self-report questionnaires are the Eating Disorder Inventory (EDI-2⁶⁸) and the Eating Attitudes Test (EAT⁶⁹). All of these instruments have been translated into different languages and have been validated in several countries and cultures.

In addition to specific eating disorder symptomatology, comorbidity with other mental disorders, especially depression, anxiety, and obsessions (see above), should be assessed carefully at admission and monitored during the process of refeeding.

For most patients, medical and psychological assessment is the first step to treatment. At this point it is often easier to help the patient recognize the relationship between her eating disordered behavior and medical or psychological problems than through direct confrontation or challenge. Both patient and family should be informed about the course and nature of the adolescent's eating disorder, including the extent of underweight and its medical consequences. Finally, treatment options have to be discussed in detail. As a rule, the threshold for intervention should be lower in adolescence than in adulthood, because research has clearly demonstrated that outcomes are worse for patients who are severely underweight.⁷⁰ A trusting and non-judgmental relationship between the therapist and patient at assessment can set the tone for change and be the beginning of successful treatment.

SUMMARY

In this article diagnostic and classification issues of adolescent eating disorders have been reviewed. Many clinicians have to face the problem of several of their young patients not fitting into the narrowly defined category of AN or BN. More than half of

childhood and adolescent eating disorder diagnoses seem to fall under EDNOS, so research on the symptoms, course, and outcome of this “residual” category is urgently needed. Other concerns involve the validity of current AN and BN criteria and an inadequate sensitivity for developmental aspects. Prevalence rates of AN seem to have remained stable during the last decades; however, recent epidemiologic studies indicate that AN is still on the rise in adolescent and adult women. It remains unclear whether there is a true decrease in the incidence of BN or whether this is the result of changes in patients’ referral or service use.

Eating disorders are serious, often chronic, and potentially life-threatening disorders. The clinician should be well aware of medical complications in severely underweight or purging adolescents. Significant strides have been made in the past 10 years in elucidating patterns of comorbidity. Anxiety disorders (including OCD) seem to be of special importance, probably even for our knowledge on nosology and prevention.

Special efforts should be made for the appropriate and right in-time identification of childhood and adolescent disorders so that earlier and probably more effective intervention can be applied.

REFERENCES

1. Nicholls D, Viner R. Eating disorders and weight problems. *BMJ* 2005;330:950–3.
2. Hebebrand J, Muller TD, Holtkamp K, et al. The role of leptin in anorexia nervosa: clinical implications. *Mol Psychiatry* 2007;12(1):23–35.
3. Steinhausen HC. The outcome of anorexia nervosa in the 20th century. *Am J Psychiatry* 2002;1284–93.
4. Eddy KT, Keel PK, Dorer DJ, et al. Longitudinal comparison of anorexia nervosa subtypes. *Int J Eat Disord* 2002;31:191–201.
5. Bulik CM, Hebebrand J, Keski-Rahkonen A, et al. Genetic epidemiology, endophenotypes, and eating disorder classification. *Int J Eat Disord* 2007;40:52–60.
6. Hebebrand J, Casper R, Treasure J, et al. The need to revise the diagnostic criteria for anorexia nervosa. *J Neural Transm* 2004;111(7):827–40.
7. Strober M, Freeman R, Morrell W. Atypical anorexia nervosa: separation from typical cases in course and outcome in a long-term prospective study. *Int J Eat Disord* 1999;25:135–42.
8. Garfinkel PE, Lin E, Goering P, et al. Should amenorrhoea be necessary for the diagnosis of anorexia nervosa? Evidence from a Canadian community sample. *Br J Psychiatry* 1996;68:500–6.
9. Garfinkel PE, Lin E, Goering P, et al. Bulimia nervosa in a Canadian community sample: prevalence and comparison of subgroups. *Am J Psychiatry* 1995;152:1052–8.
10. Keel PK, Mayer SA, Harnden-Fischer JH. Importance of size in defining binge eating episodes in bulimia nervosa. *Int J Eat Disord* 2001;29(3):294–301.
11. Eddy KT, Dorer DJ, Debra FL, et al. Should bulimia nervosa be subtyped by history of anorexia nervosa? A longitudinal validation. *Int J Eat Disord* 2007;40:67–71.
12. Fairburn CG, Cooper Z. Thinking afresh about the classification of eating disorders. *Int J Eat Disord* 2007;40:107–10.
13. Nicholls D, Chater R, Lask B. Children into DSM don't go: a comparison of classification systems for eating disorders in childhood and early adolescence. *Int J Eat Disord* 1999;28:317–24.

14. Eddy KT, Doyle AC, Hoste RR, et al. Eating disorder not otherwise specified in adolescents. *J Am Acad Child Adolesc Psychiatry* 2008;47(2):156–64.
15. Lewinsohn PM, Striegel-Moore RH, Seeley MS. Epidemiology and natural course of eating disorders in young women from adolescence to young adulthood. *J Am Acad Child Adolesc Psychiatry* 2000;39:1284–92.
16. Herpertz-Dahlmann B, Wille N, Ravens-Sieberer U, et al. Eating disordered behavior, associated psychopathology and health-related quality of life—results from the BELLA-study. *Eur J Child Adolesc Psychiatry*, in press.
17. Workgroup for Classification of Eating Disorders in Children and Adolescents (WCEDCA). Classification of child and adolescent eating disturbances. *Int J Eat Disord* 2007;40:117–22.
18. Lowinger K, Griffiths RA, Beumont PJ, et al. Fluid restriction in anorexia nervosa: a neglected symptom or new phenomenon. *Int J Eat Disord* 1999;26:392–6.
19. Holtkamp K, Herpertz-Dahlmann B, Hebebrand K, et al. Physical activity and restlessness correlate with leptin levels in patients with adolescent anorexia nervosa. *Biol Psychiatry* 2006;60(3):311–3.
20. Keys A, Brozek J, Henschel A, et al. *The biology of human starvation*. Minneapolis (MN): University of Minneapolis Press; 1950.
21. Fairburn CG, Welch SL, Doll HA, et al. Risk factors for bulimia nervosa. A community-based control study. *Arch Gen Psychiatry* 1997;54:509–17.
22. Currin L, Schmidt U, Treasure J, et al. Time trends in eating disorders incidence. *Br J Psychiatry* 2005;186:132–5.
23. Hoek HW. Incidence, prevalences and mortality of anorexia nervosa and other eating disorders. *Curr Opin Psychiatry* 2006;19(4):389–94.
24. Machado PP, Machado BC, Goncalves S, et al. The prevalences of eating disorders not otherwise specified. *Int J Eat Disord* 2007;40(3):212–7.
25. Keski-Rahkonen A, Hoek HW, Susser ES, et al. Epidemiology and course of anorexia nervosa in the community. *Am J Psychiatry* 2007;164(8):1259–65.
26. Van Son GE, van Hoeken D, Bartelds AI, et al. Time trends in the incidence of eating disorders: a primary care study in the Netherlands. *Int J Eat Disord* 2006;39(7):565–9.
27. Keel PK, Heatherton TF, Dorer DJ, et al. Point prevalence of bulimia nervosa in 1982, 1992, and 2002. *Psychol Med* 2006;36(1):119–27.
28. Fichter M, Quadflieg N. Twelve-year course and outcome of bulimia nervosa. *Psychol Med* 2004;34:1395–406.
29. Striegel-Moore RH, Leslie D, Petrill SA, et al. One-year use and cost of inpatient and outpatient services among female and male patients with an eating disorder: evidence from a national database of health insurance claims. *Int J Eat Disord* 2000;27(4):381–9.
30. Krauth C, Buser K, Vogel H. How high are the costs of eating disorders—anorexia nervosa and bulimia nervosa – for German society? *Eur J Health Econom* 2002;3:244–50.
31. Mika C, Holtkamp K, Heer M, et al. A 2-year perspective study of bone metabolism and bone mineral density in adolescents with anorexia nervosa. *J Neural Transm* 2007;114(12):1611–8.
32. Misra M, Klibanski A. Anorexia nervosa and osteoporosis. *Rev Endocr Metab Disord* 2006;7:91–9.
33. Pollice CP, Kaye WH, Greeno CG, et al. Relationship of depression, anxiety, and obsessiveness to state of illness in anorexia nervosa. *Int J Eat Disord* 1997;21:367–76.

34. Halmi KA, Eckert E, Marchi P, et al. Comorbidity of psychiatric diagnoses in anorexia nervosa. *Arch Gen Psychiatry* 1991;712–8.
35. Salbach-Andrae H, Lenz K, Simmendinger N, et al. Psychiatric comorbidities among female adolescents with anorexia nervosa. *Child Psychiatry Hum Dev* 2008;39:261–72.
36. Herpertz-Dahlmann B, Wewetzer C, Remschmidt H, et al. The predictive value of depression in anorexia nervosa. *Acta Psychiatr Scand* 1995;91:114–9.
37. Herzog DB, Keller MB, Sacks NR, et al. Psychiatric comorbidity in treatment-seeking anorexics and bulimics. *J Am Acad Child Adolesc Psychiatry* 1992; 31(5):810–8.
38. Rastam M. Anorexia nervosa in 51 Swedish adolescents: premorbid problems and comorbidity. *J Am Acad Child Adolesc Psychiatry* 1992;31:819–29.
39. Bushnell JA, Wells JE, McKenzie JM, et al. Bulimia comorbidity in the general population and in the clinic. *Psychol Med* 1994;24:605–11.
40. Perdereau F, Faucher S, Wallier J, et al. Family history of anxiety and mood disorders in anorexia nervosa: review of the literature. *Eat Weight Disord* 2008;13(1): 1–13.
41. Wade TD, Bulik CM, Neale M, et al. Anorexia nervosa and major depression: shared genetic and environmental risk factors. *Am J Psychiatry* 2000;157: 469–71.
42. Lilenfeld LR, Kaye WH, Greeno CG, et al. A controlled family study of anorexia nervosa and bulimia nervosa: psychiatric disorders in first-degree relatives and effects of proband comorbidity. *Arch Gen Psychiatry* 1998;55(7): 603–10.
43. Brewerton TD, Lydiard RB, Herzog DB, et al. Comorbidity of axis I psychiatric disorders in bulimia nervosa. *J Clin Psychiatry* 1995;56:77–80.
44. Piran N, Kennedy S, Garfinkel PE, et al. Affective disturbance in eating disorders. *Nerv Ment Dis* 1985;173:395–400.
45. Herpertz-Dahlmann B. Psychiatrische Erkrankungen im Vorfeld der Anorexia nervosa. *Klin Päd* 1988;200:108–12.
46. Neufang S, Specht K, Konrad K, et al. Sex differences and the impact of steroid hormones on the developing human brain. *Cerebr Cort*, in press.
47. Franko DL, Keel PK. Suicidality in eating disorders: occurrence, correlates, and clinical implications. *Clin Psychol Rev* 2006;26(6):769–82.
48. Bulik CM, Thornton L, Pinheiro AP, et al. Suicide attempts in anorexia nervosa. *Psychosom Med* 2008; [Epub ahead of print].
49. Kaye WH, Bulik CM, Thornton L, et al. Comorbidity of anxiety disorders with anorexia and bulimia nervosa. *Am J Psychiatry* 2004;161(12):2215–21.
50. Jordan J, Joyce PR, Carter FA, et al. Specific and nonspecific comorbidity in anorexia nervosa. *Int J Eat Disord* 2008;41(1):47–56.
51. Godart NT, Flament MF, Lecrubier Y, et al. Anxiety disorders in anorexia nervosa and bulimia nervosa: co-morbidity and chronology of appearance. *Eur Psychiatry* 2000;15:38–45.
52. Shoebridge P, Gowers SG. Parenteral high concern and adolescent-onset anorexia nervosa. A case-control study to investigate direction of causality. *Br J Psychiatry* 2000;176:132–7.
53. Swinbourne JM, Touyz SW. The co-morbidity of eating disorders and anxiety disorders: a review. *Eur Eat Disord Rev* 2007;15(4):253–74.
54. Wentz-Nilsson E, Gillberg C, Gillberg IC, et al. Ten-year follow-up of adolescent-onset anorexia nervosa: personality disorders. *J Am Acad Child Adolesc Psychiatry* 1999;1389–95.

55. Herpertz-Dahlmann B, Müller B, Herpertz S, et al. Prospective ten-year follow-up in adolescent anorexia nervosa—course, outcome and psychiatric comorbidity. *J Child Psychol Psychiatry* 2001;42:603–12.
56. Thompson-Brenner H, Eddy KT, Franko DL, et al. Personality pathology and substance abuse in eating disorders: a longitudinal study. *Int J Eat Disord* 2008; 41(3):203–8.
57. Fischer S, le Grange D. Comorbidity and high-risk behaviors in treatment-seeking adolescents with bulimia nervosa. *Int J Eat Disord* 2007;40(8):751–3.
58. Lacey JH, Evans CD. The impulsivist: a multi-impulsive personality disorder. *Br J Addiction* 1986;81:715–23.
59. Stein D, Lilenfeld LR, Wildmann PC, et al. Attempted suicide and self-injury in patients diagnosed with eating disorders. *Compr Psychiatry* 2004;45:447–51.
60. Biederman J, Ball SW, Monuteaux MC, et al. Are girls with ADHD at risk for eating disorders? Results from a controlled, five-year prospective study. *J Dev Behav Pediatr* 2007;28:302–7.
61. Mikami AY, Hinshaw SP, Patterson KA, et al. Eating pathology among adolescent girls with attention-deficit/hyperactivity disorder. *J Abnorm Psychol* 2008;117(1): 225–35.
62. Halmi KA, Sunday SR, Strober M, et al. Perfectionism in anorexia nervosa: variation by clinical subtype, obsessiveness, and pathological eating behaviour. *Am J Psychiatry* 2000;157(11):1799–805.
63. Fairburn CG, Cooper Z, Doll HA, et al. Risk factors for anorexia nervosa: three integrated case-control comparisons. *Arch Gen Psychiatry* 1999;56:468–76.
64. Lilenfeld LR, Stein D, Bulik CM, et al. Personality traits among currently eating disordered, recovered and never ill first-degree female relatives of bulimic and control woman. *Psychol Med* 2000;30(6):1399–410.
65. Nilsson K, Sundbom E, Hägglöf B. A longitudinal study of perfectionism in adolescent onset anorexia nervosa-restricting type. *Eur Eat Disord Rev* 2007; [Epub ahead of print].
66. Cooper Z, Cooper PF, Fairburn CG. The validity of eating disorder examination and its subscales. *Br J Psychiatry* 1989;154:807–12.
67. Fichter M, Herpertz S, Herpertz-Dahlmann B, et al. Structured interview for anorexia and bulimic disorder. *Int J Eat Disord* 1998;24:227–49.
68. Garner DM. Eating disorder inventory (EDI-2). 2nd professional manual. Odessa (FL): Psychological Assessment Resources, Inc; 1991.
69. Garner DM. Eating attitude test (EAT). *Psychological Medicine* 1979;9:273–9.
70. Hebebrand J, Himmelmann GW, Herzog W, et al. Prediction of low body weight at long-term follow-up in acute anorexia nervosa by low body weight at referral. *Am J Psychiatry* 1997;154:566–9.