Eating disorders

Janet Treasure

Abstract

Eating disorders are common problems affecting 5–10% of young people. The bulimic forms of eating disorders became dominant in the last half of the 20th century and are caused in part by tensions between the easy availability of high-palatability food that promotes excess eating and a culture that idealizes thinness. Genetic factors, high anxiety and environmental stress also contribute. Anorexia nervosa is associated with high levels of physical disability, and problems also occur in social, vocational and parenting functioning. Psychological forms of treatment, particularly those that incorporate education and behavioural change skills such as cognitive behavioural therapy, are effective. With anorexia nervosa, it is particularly helpful if family members are involved as they may have been drawn into unhelpful patterns of interaction. Antidepressants and antiepileptic drugs have a small effect on binge eating, and there is interest in adding drugs used in obesity and stimulants for binge-eating disorders.

Keywords Anorexia nervosa; binge-eating disorder; bulimia nervosa; eating disorders

Key points

- Approximately 10% of young women have some form of eating problem
- Binge-eating disorder has rapidly increased in prevalence and is the most common form of eating disorder, although most cases do not present for treatment
- Bulimia nervosa and binge-eating disorder can be treated by cognitive behavioural therapy (self-help adaptations are available)
- Pharmacotherapy can supplement treatment for binge eating
- It is hard to engage people with anorexia nervosa (AN) in treatment as they hold fixed attitudes about AN and do not want to change
- An alternative diagnosis should be suspected if there is a raised erythrocyte sedimentation rate (or C-reactive protein level)
- Factors such as the age, stage and severity of AN are used to triage treatment for AN
- Admission should be considered if there are signs of any of the following: myopathy, reduced core temperature, circulatory decompensation, abnormal biochemistry and haematology
- Wherever possible, give nutrient and mineral replacements orally. Close physical monitoring is essential to prevent re-feeding syndrome
- Stabilization of high-risk AN requires the gradual introduction of food (starting with approximately 1000 kcal) supplemented with standard doses of vitamin and mineral supplements
- Sharing information and skills with family members improves a patient's well-being and may improve the outcome of the eating disorder

Definition

The diagnostic criteria for eating disorders were expanded and made more lenient in the latest (5th) edition of the Diagnostic and Statistical Manual of Mental Disorders, from the American Psychiatric Association, to better capture the form in which these problems now present. The distinct clinical entities now include anorexia nervosa (AN), bulimia nervosa (BN), binge-eating disorder (BED) and avoidant/restrictive food intake disorder (ARFID) (usually seen in children). The key feature of AN is a weight 15% below a person's expected weight for their age, height and sex associated with an intense reluctance to gain weight, and a preoccupation with weight and/or food.

BN shares some features with AN, such as an intense preoccupation with weight and shape; however, it is characterized by regular episodes of uncontrolled eating (>1000 kcal in one sitting) associated with various methods of counteracting weight gain (vomiting and laxative abuse being most common). BED is characterized by significant binge eating in the absence of extreme compensatory behaviours, so is associated with obesity. ARFID does not have the typical psychopathology of AN such as weight concerns.

Epidemiology

Eating disorders and related behaviours are a common problem in pre-adolescents and adolescents. A recent study on a large sample of American young people (aged 9–14 years) found that 34% of boys and 43.5% of girls had some eating disorder trait.

The lifetime female prevalence rates (percentage of the population) are approximately 0.9% for AN, 1.5% for BN, 3.5% for BED, and 10% for subclinical disorders. However, fewer than 20% of cases of eating disorder present for treatment. The incidence of cases of BED presenting to primary care in the UK increased to 25 per 100,000 between 2000 and 2010, whereas the incidence (22 per 100,000) of BN[1] stabilized following a rapid increase between 1980 and 1995. The incidence of AN has been stable at 15 per 100,000 over the last four decades.

Abundant historical records of AN date from the 19th century. The clinical and behavioural features are present in diverse cultural settings, but the content of the psychopathology varies over

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time and place. BN was recognized as a new syndrome in 1979. It is more common in urban areas. The peak onset of AN is 16 years and of binge eating 18 years. (A degree of autonomy and disconnection from the social aspects of eating facilitates the development of the disrupted pattern of eating.)

The gender bias varies across diagnosis and setting. For example, the female:male ratio is 10:1 for AN and BN, but in the community and in child and adolescent settings, the ratio is 3:1, and in BED there is less of a disparity between the sexes. The finding that the proportion of male patients presenting to clinical services is less than that found in the community suggests that there are barriers in terms of awareness, recognition and stigma about having a 'girl's illness'.

BED is particularly common in clinical populations with obesity and diabetes. Co-morbidity with other psychiatric disorders (depression, anxiety, obsessive–compulsive disorder) is the rule rather than the exception and there are also links with temperamental traits or developmental disorders such as ADHD, or emotional unstable personality disorder (with BN and BED) and obsessive–compulsive personality disorder (AN).

Pathology and pathogenesis

Family, biological, social and cultural factors can play a role in either the development or the maintenance of eating disorders. The risk of eating disorders in first-degree relatives is increased tenfold, and twin studies suggest that this is due to inherited factors. Other psychiatric disorders such as depression, generalized anxiety disorder and obsessive–compulsive disorder are slightly over-represented in the families of people with eating disorders. Family members also have a three-fold risk of obsessive–compulsive personality disorder (see *Anxiety disorders*, pp xxx–xxx of this issue).

Furthermore, obsessive–compulsive traits, such as perfectionism and rigidity in childhood increase the risk of developing an eating disorder. Eating disorders are associated with traits associated with some neurodevelopmental disorders (AN with autism spectrum disorders, and BED and obesity with attention-deficit hyperactivity disorder). Prematurity, particularly if the baby was small for gestational age, increases the risk of developing AN sixfold. Later, adversity during childhood, such as sexual and physical abuse, increases the risk of bulimic disorders. In addition, the risk of BN is increased in individuals who had a robust appetite during childhood, were heavy and were teased or criticized about their weight or eating. A model of the evolution of risk factors into the development of eating disorders is shown in Figure 1.[2]

Diagnosis

A positive diagnosis of an eating disorder is usually made from the history. For AN, informants can help. It is useful to screen for eating disorders in populations with high risk (e.g. diabetes, college students) and in pregnancy where the risk for the unborn child can be increased (Figure 2). The screening questions should include asking for a pattern of extreme weight fluctuations (especially if they have caused concern to others), an increased salience of food and shape or weight issues, and extreme weight control behaviours (vomiting, fasting, exercise, etc.). In a typical case, it is necessary to exclude other medical conditions (Table 1). A variety of medical complications can occur as a consequence of starvation or other weight control measures.[3]

Course

Over 50% of cases of AN have a protracted course over 6 years, and one-third of cases never make a full recovery. Approximately, one-third of cases develop bulimic features. BN has a relapsing and remitting course, and the median duration is >10 years. BED also persists over many years.

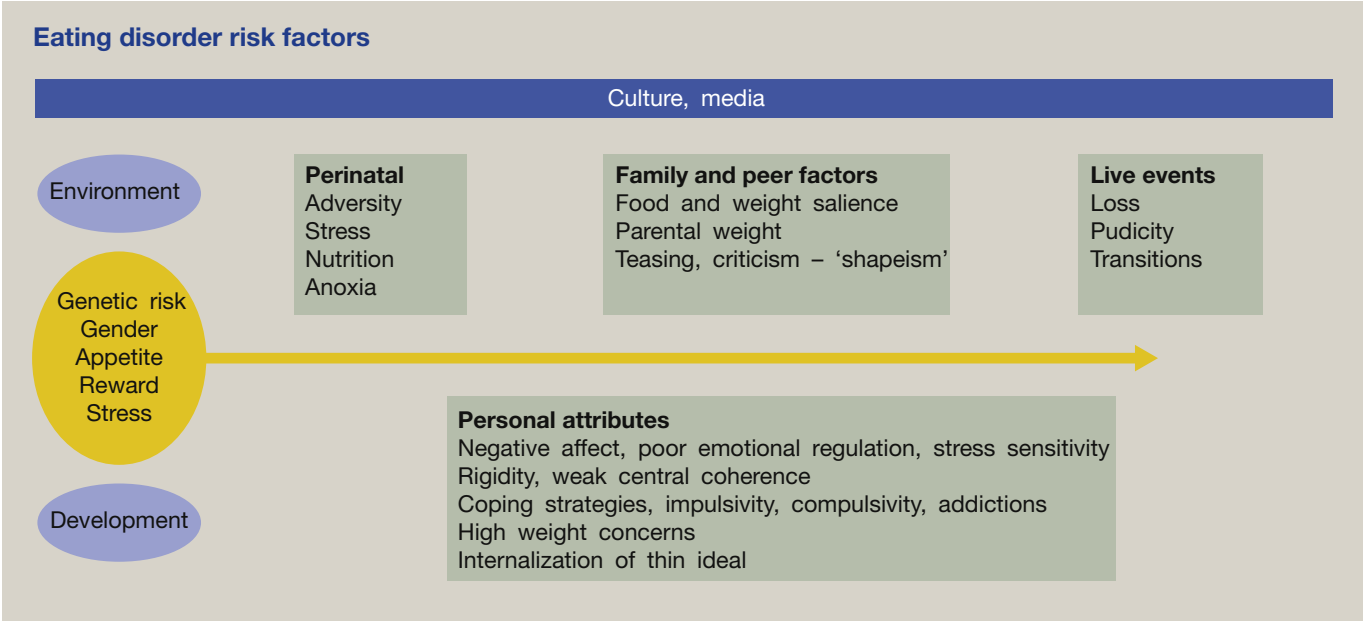

Figure 1

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