

Section 1
Chapter 1

Definitions and prevalence
Definitions and epidemiology

Eating disorders are associated with significant psychosocial and physical disability. They pose a heavy burden on the family, community and health services.

Anorexia nervosa

Anorexia nervosa is low in prevalence, but high in medical consequence. The most common age of onset is 15 years (range 9–24 years). Females are diagnosed with anorexia nervosa ten times more often than males. During their lifetime, 0.9–2.2% of women and 0.2–0.3% of men are diagnosed with anorexia nervosa. In addition, one-third of the cases of anorexia nervosa are not included in statistics, because they never seek treatment. The incidence of anorexia nervosa, the rate at which new cases occur, has not changed in the last five decades, although the incidence in adolescent girls may have increased a little. Anorexia nervosa has the highest mortality rate of any psychiatric condition, with a standardised mortality rate approximately ten times that of the general population. Suicide accounts for one half of the deaths, medical causes, especially arrhythmias, for the other half.

The diagnostic features of anorexia nervosa in the DSM-IV and ICD-10 are similar and are shown in Table 1.1 and Table 1.2.

The essential feature of anorexia nervosa is a constant, inexplicable fear of being fat or eating, which increases with weight loss and does not change with reason. Anorexia nervosa is egosyntonic; this means the patient believes their concerns about weight and shape are normal, they do not think there is anything wrong with them and they are therefore not interested in treatment. Weight loss, in anorexia nervosa, is attained and sustained by insufficient food intake, usually coupled with an increase in energy expenditure caused by compulsive activity. However, one half of cases of anorexia nervosa also binge eat, then vomit (binge eating/purging subtype). Equivalents of purging that may be used in addition to, or instead of, vomiting are laxatives, enemas, suppositories, diuretics, ipecac, fasting, weight loss pills or pills to increase the metabolic rate, spitting out food, self-phlebotomy and self-gavage.

Often patients will begin with restricting their food intake and then add exercise, which can become compulsive. Often, 6 months to 2 years later, food craving and overeating begins, presumably related to malnutrition. This drive to eat leads to bingeing, which then leads to purging. In a quarter to a half of patients with anorexia nervosa the binge–purge cycle becomes chronic.

Bulimia nervosa

Bulimia nervosa involves habitual binge eating followed by purging or a purge equivalent. The purging subtype is defined by the use of vomiting, laxatives, diuretics or enemas. The

The Medical Management of Eating Disorders

Table 1.1 DSM-IV diagnostic criteria for anorexia nervosa

(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).
(b) Intense fear of gaining weight or becoming fat, even though underweight.
(c) Disturbance in the way in which one’s body weight or shape is experienced, undue influence of body weight or shape on self-evaluation, or denial of the seriousness of the current low body weight.
(d) In postmenarcheal females, amenorrhea, i.e., the absence of at least three consecutive menstrual cycles. (A woman is considered to have amenorrhea if her periods occur only following hormone, e.g. estrogen, administration.)
Specify type:
Restricting type: During the current episode of anorexia nervosa, the person has not regularly engaged in binge eating or purging behavior (i.e., self-induced vomiting or the misuse of laxatives, diuretics or enemas)
Binge eating/Purging type: During the current episode of anorexia nervosa, the person has regularly engaged in binge eating or purging behavior (i.e., self-induced vomiting or the misuse of laxatives, diuretics or enemas)
Source: <i>Diagnostic and Statistical Manual of Mental Disorders</i> , Fourth Edition. Washington, DC, American Psychiatric Association, 1994.

Table 1.2 ICD-10 diagnostic criteria for anorexia nervosa

(a) Body weight is maintained at least 15% below that expected (either lost or never achieved), or Quetelet’s body mass index is 17.5 or less. Prepubertal patients may show failure to make the expected weight gain during the period of growth.
(b) The weight loss is self-induced by avoidance of ‘fattening foods’ and one or more of the following: self-induced vomiting; self-induced purging; excessive exercise; use of appetite suppressants and/or diuretics.
(c) There is body-image distortion in the form of a specific psychopathology whereby a dread of fatness persists as an intrusive, overvalued idea and the patient imposes a low weight threshold on himself or herself.
(d) A widespread endocrine disorder involving the hypothalamic–pituitary–gonadal axis is manifest in women as amenorrhoea and in men as a loss of sexual interest and potency. (An apparent exception is the persistence of vaginal bleeds in anorexic women who are receiving replacement hormonal therapy, most commonly taken as a contraceptive pill.) There may also be elevated levels of growth hormone, raised levels of cortisol, changes in the peripheral metabolism of the thyroid hormone and abnormalities of insulin secretion.
(e) If onset is prepubertal, the sequence of pubertal events is delayed or even arrested (growth ceases; in girls the breasts do not develop and there is a primary amenorrhoea; in boys the genitals remain juvenile). With recovery, puberty is often completed normally, but the menarche is late.
Source: <i>Classification of Mental and Behavioural Disorders</i> (ICD-10). Geneva, World Health Organization, 1992.

non-purging subtype uses other behaviours that compensate for the binge. Patients with bulimia nervosa usually have a normal weight, are still active at school or work, and binge and purge covertly. However, bulimia nervosa is an egodystonic disorder; the patients want to be cured! But, perhaps not at the price of weight gain.

Definitions and epidemiology

Table 1.3 DSM-IV diagnostic criteria for bulimia nervosa

(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).
(b) Recurrent inappropriate compensatory behavior in order to prevent weight gain, such as self-induced vomiting; misuse of laxatives, diuretics, enemas or other medications; fasting; or excessive exercise.
(c) The binge eating and inappropriate compensatory behaviours both occur, on average, at least twice a week for 3 months.
(d) Self-evaluation is unduly influenced by body shape and weight.
(e) The disturbance does not occur exclusively during episodes of bulimia nervosa.
<i>Specify type</i>
Purging type: During the current episode of bulimia nervosa, the person has regularly engaged in self-induced vomiting or the misuse of laxatives, diuretics or enemas
Non-purging type: During the current episode of bulimia nervosa, the person has used other inappropriate compensatory behaviours, such as fasting or excessive exercise, but has not regularly engaged in self-induced vomiting or the misuse of laxatives, diuretics or enemas
Source: <i>Diagnostic and Statistical Manual of Mental Disorders</i> , Fourth Edition. Washington, DC, American Psychiatric Association, 1994.

Table 1.4 ICD-10 diagnostic criteria for bulimia nervosa

(a) There is a persistent preoccupation with eating, and an irresistible craving for food; the patient succumbs to episodes of overeating in which large amounts of food are consumed in short periods of time.
(b) The patient attempts to counteract the 'fattening' effects of food by one or more of the following: self-induced vomiting; purgative abuse; alternating periods of starvation; use of drugs such as appetite suppressants, thyroid preparations or diuretics. When bulimia occurs in diabetic patients they may choose to neglect their insulin treatment.
(c) The psychopathology consists of a morbid dread of fatness and the patient sets herself or himself a sharply defined weight threshold, well below the premorbid weight that constitutes the optimum or healthy weight in the opinion of the physician. There is often, but not always, a history of an earlier episode of anorexia nervosa, the interval between the two disorders ranging from a few months to several years. This earlier episode may have been fully expressed, or may have assumed a minor cryptic form with a moderate loss of weight and/or a transient phase of amenorrhoea.
Source: <i>Classification of Mental and Behavioural Disorders</i> (ICD-10). Geneva, World Health Organization, 1992.

Bulimia nervosa affects 1.5–2.0% of women and 0.5% of men. The incidence increased rapidly after the 1970s, but currently appears to be stable or possibly decreasing from a peak in the 1990s. The onset is usually in the late teenage years, later than anorexia nervosa. Table 1.3 and Table 1.4 list the DSM-IV and ICD-10 definitions of bulimia nervosa. The essential feature of bulimia nervosa is recurrent binge eating. A binge is defined as an episode of eating which is excessive for the context and is accompanied by a subjective sense

The Medical Management of Eating Disorders

Table 1.5 Binge eating disorder

(a) Recurrent episodes of binge eating. An episode is characterized by: (1) Eating a larger amount of food than normal during a short period of time (within any 2-hour period). (2) Lack of control over eating during the binge episode (i.e. the feeling that one cannot stop eating).
(b) Binge eating episodes are associated with three or more of the following: (1) Eating until feeling uncomfortably full. (2) Eating large amounts of food when not physically hungry. (3) Eating much more rapidly than normal. (4) Eating alone because you are embarrassed by how much you're eating. (5) Feeling disgusted, depressed or guilty after overeating.
(c) Marked distress regarding binge eating is present.
(d) Binge eating occurs, on average, at least 2 days a week for six months.
(e) The binge eating is not associated with the regular use of inappropriate compensatory behaviour (i.e. purging, excessive exercise, etc.) and does not occur exclusively during the course of bulimia nervosa or anorexia nervosa.
Source: <i>Diagnostic and Statistical Manual of Mental Disorders</i> , Fourth Edition. Washington, DC, American Psychiatric Association, 1994.

of loss of control. The binge may be associated with reduced caloric intake, stress, a particular location (e.g. the bathroom at home), a particular time of day (e.g. at night), particular people, changes in mood, loneliness, pain, insomnia or fatigue.

Binge eating disorder

Binge eating disorder (BED) is classified in the DSM-IV as ‘a provisional diagnosis worthy of further study’. The lifetime prevalence of BED is estimated to be 3.5% among adult females and 2.0% in males and the prevalence appears to be increasing. It occurs in black as well as white ethnic groups. BED is present in about a third of patients with obesity. Table 1.5 gives the definition of BED.

The essential feature of BED is episodes of overeating. The compulsion to overeat is often associated with the taste and quality of food, whereas in bulimia nervosa binges are more likely to be ritualistic and to include food that is easy to vomit afterwards. In BED there is no extreme compensatory behaviour, like vomiting or laxative use. This behaviour is also seen in genetic forms of obesity like Prader–Willi syndrome and Kleine–Levin syndrome. There is less over-investment in weight and shape for self-esteem than in bulimia nervosa.

Eating disorder not otherwise specified

This category is used, at present, for all those whose quality of life is severely affected by an eating disorder, but who do not meet all the criteria of one of the other eating disorder diagnoses. Some cases of anorexia nervosa are classified as EDNOS during an early stage of illness or recovery. However, there are other combinations of symptoms that can lead to a similar disruption in quality of life. Examples of EDNOS include: someone of normal weight who engages in purging such as vomiting or laxative use in the absence of bingeing episodes (‘purging disorder’), or someone with anorexia nervosa whose weight is not low enough to satisfy the criteria for anorexia nervosa, or whose periods have not stopped). This difficulty with classification is particularly evident when diagnosing children and adolescents.

Section 2
Chapter
2

Cause
Causal and maintaining factors

The causal models of eating disorders should explain the differential epidemiology between the restricting and bingeing forms of eating disorders. Why has the prevalence of anorexia nervosa remained low over its long history, in contrast to the exponential increase of bulimia nervosa and then binge eating disorders, particularly in women but also in the case of binge eating in men born in the latter half of the twentieth century? Why is bulimia nervosa more common in urban environments? It is probable that the cultural factors that account for the rapid increase in the prevalence of obesity within this time frame also contribute to the risk of bingeing in the form of both bulimia nervosa and binge eating disorder. Cultural factors may contribute to the maintenance of all forms of eating disorders, making them impermeable to therapy.

For those interested in examining risk factors in more depth, the primary sources are contained in a systematic review of Jacobi and colleagues (Jacobi). Figure 2.1 illustrates how genetic, environmental and developmental factors can interact with the biological, psychological and social matrix over the life-trajectory, to increase the risk of developing an eating disorder. In the following subsection we will describe in more detail each of these domains.

Cultural factors

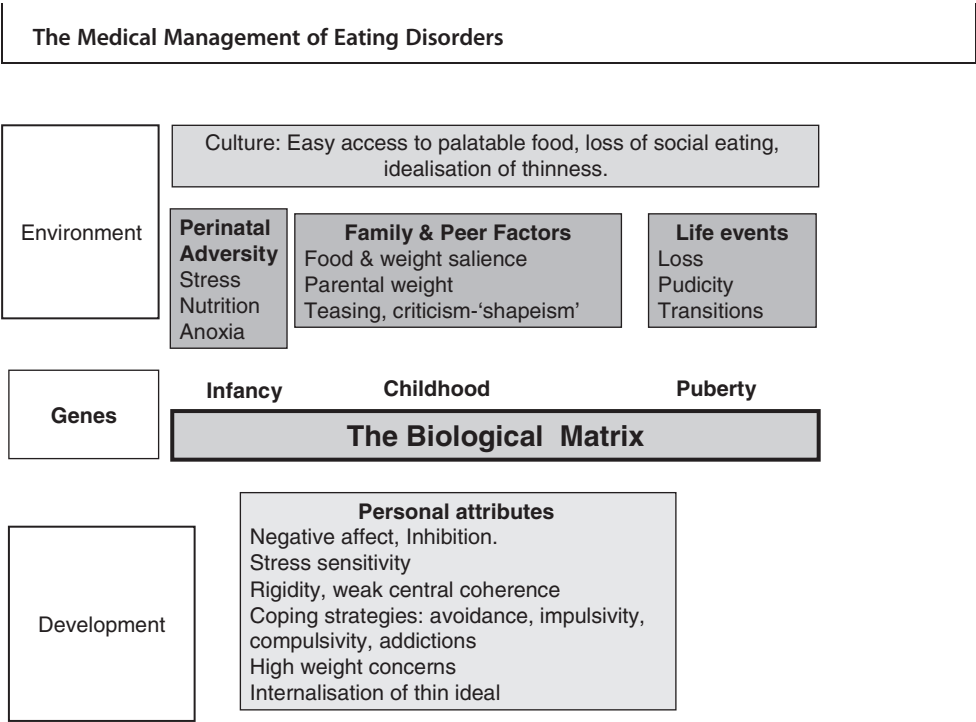
The rapid increase in bulimia nervosa and binge eating disorder and their predominance in urban centres suggests that cultural factors are relevant. Western culture has fostered the idealisation of thinness and self-discontent. Advertising fosters the possibility of a self ‘makeover’ given the expenditure of enough resources. The tension between fear of the consequences of eating and the easy accessibility of highly palatable and appealing food promotes anxiety and disordered eating.

Specific environmental risk factors

Both the general cultural risk factors and the more specific environmental triggers (such as teasing about weight or shape, or hobbies, career goals where weight and shape are salient) within the family and peer environment impact on the individual over the life course and moderate the susceptibility.

Perinatal risk factors

People who develop eating disorders are more likely to have a history of obstetric complications such as prematurity, small-for-dates and cephalohaematoma. Adverse perinatal factors such as higher levels of maternal stress are also more likely. Thus, hypoxic damage



Causal and maintaining factors

The risk of eating disorder is increased tenfold in families with an affected individual. Several family-based linkage studies have identified genetic loci for anorexia nervosa, bulimia nervosa or associated behavioural traits. Also, associations between eating disorders and the genes associated with brain-derived neurotrophic factor (BDNF), opioid and serotonin receptors have been reported by several groups. It is probable that a number of genes, each of which has a small effect, may contribute to an increase in risk. Furthermore the risk associated with these genes may only be expressed in the certain circumstances, for example after puberty or in the presence of weight loss. Puberty appears to be a critical developmental stage during which environmental triggers causing stress, loss of weight or a change in meal content or patterning can exaggerate or activate latent traits.

Development changes in psychology and physiology

Genetic and environmental effects interact in the developing individual and produce changes in the psychological and physiological template of the individual. Eating disorders begin at a time in brain development when there are marked changes such as pruning down of links between neurons and myelination of the connecting tracts; both factors make the function of the brain more efficient. The frontal area of the brain, which is the centre of self-regulation, is the last to mature. Some theories suggest that problems in self-regulation may trigger or maintain eating disorders.

Abnormalities in the structure and chemistry of the brain

A simple diagram outlining the brain structures involved in the control of appetite is shown in Figure 2.2. There are three basic elements. First, there is the nutrostat system, which

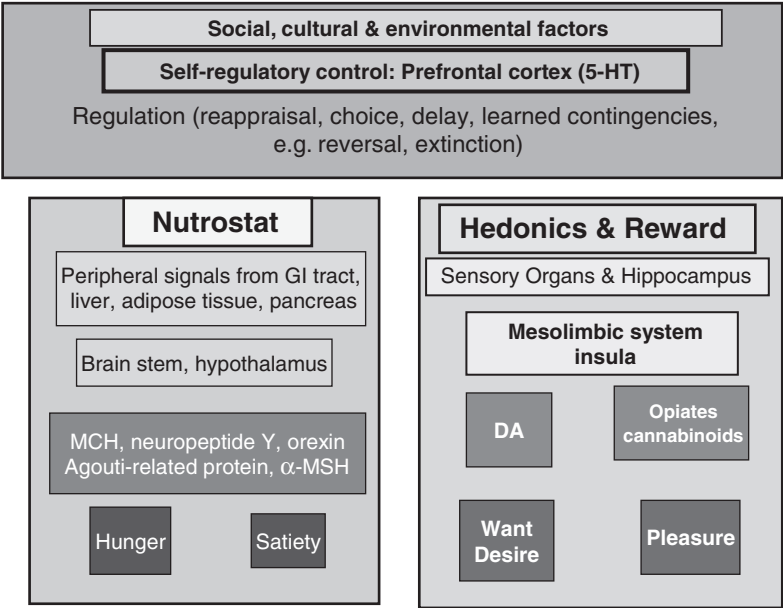


Figure 2.2 Brain structures involved in the control of appetite.

The Medical Management of Eating Disorders

regulates food intake according to metabolic signals relating to body weight and energy expenditure. Second, the hedonic system is responsible for the reward associated with eating and the drive and motivation to attain food. Finally there is the self-regulatory system, which controls the other two systems and sets eating into a social context and involves choice, decision making and forms of planned behaviour.

The brain is shrunken and the ventricular spaces enlarged in the acute phase of anorexia nervosa. Specific reductions in grey matter mass have been reported in all of the centres which are involved in eating. The degree of recovery of brain mass after recovery remains uncertain.

Abnormalities in the brain chemistry of people with eating disorders remain after recovery. These alterations in the number of monoamine receptors are biological factors associated with eating disorders, so-called endophenotypes. Kaye has proposed a hypothesis, which is that an abnormality in the chemical balance between 5-hydroxytryptamine (5-HT, serotonin) and dopamine might lead to some of the temperamental factors that are known to predispose to an eating disorder and also maintain the problem. 5-HT_{1A} post-synaptic receptors are increased and 5-HT_{2A} receptors are decreased in people with eating disorders, a pattern associated with behavioural inhibition and anxiety. Poor nutrition reduces tryptophan, a precursor of 5-HT, and it is thought that this can be rewarding to people with an anxious disposition as it produces a sense of calmness.

Also increased dopamine D₂/D₃ binding is present in the reward systems. Dopamine plays a key role in reward processing. The release of dopamine can be altered by diets containing high fat and high sugar and so bingeing on foods containing these elements may make such foods more pleasurable. People with eating disorders show an unusual pattern of brain response to reward tasks, with a greater activation in the deliberative parts of the brain suggesting a cognitive response and less activation in the parts of the brain associated with intuitive or emotional response.

Cognitive, emotional, perceptual and neurophysiological vulnerabilities

There are a wide variety of biomarkers associated with eating disorders in the metabolic, electrophysiological, endocrinological, anatomical and cognitive/psychological/neuropsychological domains. Many are state effects and are the consequences of nutritional deficiencies. Others appear to be traits that run in families and have a genetic origin and have been termed endophenotypes.

People with eating disorders have problems with information processing in the self-regulatory system. For example, they have a reduced ability to change set and show a rigidity and perseveration. Also they have problems with decision making. It is possible that stress and nutritional problems interfere with effective self-regulatory control as many of these problems are restored after recovery. However, these problems with self-regulatory control may account for the increase in compulsive behaviours and poor emotional and social regulation associated with eating disorders, and contribute to the maintenance of eating disorders. This failure of the self-regulatory control system may account for the emergence of binge eating which occurs in a substantial proportion of people with eating disorders within a few years of the onset of anorexia nervosa.

People with eating disorders often show an enhanced skill in processing detail. In some cases, especially in the acute state, this is at the expense of the ability to integrate information

Causal and maintaining factors

into a global perspective. This eye for detail probably contributes to obsessive–compulsive personality traits such as sensitivity to error and perfectionism. This skill enables individuals to master the laws of thermodynamics and so show ‘talent’ in weight loss.

An over-exaggerated stress response and an anxious temperament may be a consequence of perinatal or later developmental adversity. This alone, or in conjunction with emotional experiences in childhood, can lead to poor emotional regulation.

These cognitive and emotional difficulties may contribute to low self-esteem and social inferiority. One method to gain acceptance is to strive to attain culturally sanctioned goals, which in many modern societies is to be thin.

Factors that maintain eating disorder symptoms

Maintaining factors are those variables that predict symptom persistence over time among initially symptomatic individuals. The pattern of secondary consequences such as the problems with self regulatory control evolves over time. The early phase is common to all forms of eating disorder; however, the pathways diverge depending on whether a restrictive or a binge eating course evolves. Identifying maintaining factors is important because successful treatment of them is often necessary for recovery. Furthermore, unlike triggering factors they are currently in ‘action’ and so can be remediated in therapy.

Nutritional problems in the brain

There have been remarkable changes in understanding about brain function. At one time it was thought that there was little change in the structure or function of the brain throughout the life course. However, the brain is a highly plastic organ. Experience, learning and memory modify the structure and function of the brain. The brain’s nutritional requirements are approximately 500 kcal per day, a fifth of the total caloric requirements. The failure to meet the brain’s nutritional needs explains the change in structure (loss of grey and white matter) and function (reduction in aspects of self-regulatory control) in people with anorexia nervosa.

Figure 2.3 outlines some of the inherent traits that become more pronounced once self-regulation is reduced.

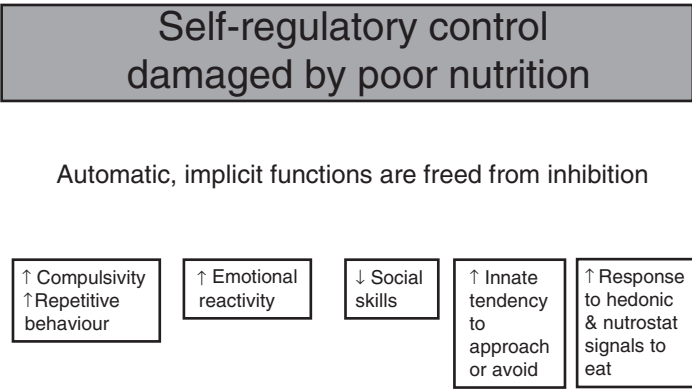


Figure 2.3 Some of the inherent traits that become more pronounced once self-regulation is reduced.

The Medical Management of Eating Disorders

For example, weight loss allows compulsive traits and rigidity to become more dominant. These traits enhance mastery over the rules of dieting and so rapid and protracted weight loss can ensue. The extreme loss of weight causes the sensitivity and drive from the nutrostat and hedonic systems of the brain to become increased. In many cases this leads to binge eating or the fear of succumbing to this. The tendency to avoid and the increased sensitivity to punishment means that the individual withdraws from many activities and becomes more anxious and isolated. The impairment in emotional and social regulation and the focus of reward onto food means that the individual becomes impermeable to interpersonal influences. Similarly the poor concentration and decision making means that the capacity to assimilate treatment and the concerns of responsible others is impaired. The longer starvation persists, the more fixed and rigid the brain becomes, new learning is stunted and normal brain development is interrupted.

Thus all of these nutritional consequences on the brain may cause eating disorder symptoms to be more firmly entrenched over time and may explain how recovery from anorexia nervosa becomes more difficult the longer the illness persists. Thus once the symptoms of an eating disorder develop, a positive feedback trap can be sprung.

Positive reinforcement for eating disorder symptoms

Psychological and physical factors

Some of the other biological and psychological consequences of severe restriction are reinforcing because they have meaning for the individual. For example, the reduction in secondary sexual drives can be seen as positive for those with anxieties relating to their sexuality. Thus in these cases, the reduction in the secretion of sex hormones is welcomed. It may provide relief from the challenges of menstruation and from sexual demands. Other physical consequences may be perpetuating. Physical debility and poor concentration can heighten the patient’s sense of vulnerability and personal inadequacy. This need to bolster a frail sense of self drives them to continue a pattern of behaviour that gives them a sense of mastery. Secondary effects on gastrointestinal function, such as slowed gastric emptying and constipation, may contribute to the discomfort and bloating that are the common complaints of individuals when they start to eat.

Social factors

The social response to the illness can be positive, including admiration for weight loss. In the early phase, peers and family may applaud the individual with an eating disorder for her zealous application to food and exercise rules, which give the impression of following a healthy lifestyle (Figure 2.4). Usually, there is, in addition, a more concentrated application to academic work with less interest in social activities, and this is also seen as commendable. For the individual herself, the success and sense of mastery that follows the rigid application to the detail of dietary rules is rewarding. As she attains the success of weight loss, this goal becomes more salient.

High expressed emotion (overprotection and criticism) from family members and other behaviours in response to the eating disorder symptoms can inadvertently enable the abnormal eating behaviours to persist. A variety of secondary gains can occur in the