

An update on eating disorders

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ARTICLE

SUMMARY

Eating disorders are heterogeneous disorders characterised by a maladaptive drive to lose weight and, for the most part, by extreme fear of weight gain and overvaluation of thin body image. Calorie restriction, overexercise and purging behaviours put some sufferers at high risk of physical morbidity and mortality. Mental preoccupations interfere with social, professional and general quality of life. Patients' defensive secrecy and compulsivity can make it hard to diagnose and treat such disorders despite the suffering they involve. Integrated medical and psychiatric intervention can save life and safely improve nutrition. Behavioural support – with family and carer involvement when appropriate – can counter the dysregulation that leads to vicious cycles of restriction–binge–purge, helping patients develop new skills to regulate emotion without weight losing. In the future, exciting developments in neuroimaging, neurosurgery and pharmacology may lead to ways to make the brain more responsive to therapy. Insights into risk factors may also improve preventive strategies in a climate of highly sophisticated international electronic communication.

LEARNING OBJECTIVES

After reading this article you will be able to:

- identify and assess the different types of eating disorder, and be aware of core clinical signs and symptoms
- confidently manage the physical and psychological consequences of eating disorders in accordance with current evidence-based guidelines
- understand current controversies in the field and evolving research themes.

KEYWORDS

Anorexia nervosa; bulimia nervosa; cognitive–behavioural therapies; comorbidity; eating disorders NOS.

all psychiatric disorders (Franko 2013). Behavioural and psychotherapeutic approaches are the mainstays of treatment.

This review summarises the current state of the specialty, covering eating disorders characterised by preoccupation with weight, shape and calorie balance. Disordered eating behaviour stemming from autistic, affective or other underpinnings warrants a separate review.

Contemporary models of eating disorders

Clinicians commonly hold an 'illness model' of eating disorders, whereas lay people, journalists and patients, particularly on social media, may assume that eating disorders represent a lifestyle choice. Charland and colleagues proposed that anorexia nervosa be considered a 'passion' – an overriding affect-laden philosophical priority (Charland 2013). This notion is fruitful in suggesting that treatment should 'fight passion with passion'.

Others emphasise endemic 'eating-disordered' symptoms in society, stressing the role of physiological dysregulation in perpetuating restriction–binge–purge cycles. Proponents of family-based treatment (FBT) (Lock 2015) observe that early refeeding alone treats many teenagers with anorexia nervosa. Such diverse opinion suggests heterogeneity despite many common features. Clinicians who address only physical disruptions are unlikely to engage their patients or provide alternative coping strategies, whereas those who neglect physiological maintaining factors may inadvertently perpetuate chronicity.

Diagnostic classification

Eating disorders share features of preoccupations and behaviours aimed at weight loss (Table 1). Both DSM-5 and ICD-11 (World Health Organization 2018) discriminate between anorexia nervosa and bulimia nervosa on the basis of weight. The hallmark of anorexia nervosa is significant weight loss, whereas those who are not underweight are diagnosed as having bulimia nervosa if they purge (induce vomiting or use laxatives) but binge eating disorder if they 'binge' without purging. Anorexia nervosa has existed for centuries, but bulimia nervosa appeared in the later 20th century. Binge eating disorder is an even more recent phenomenon. Adult eating disorder services specialise in disorders sharing the core

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Traditionally, the 'eating disorders' have been characterised by intense fear of gaining weight, although DSM-5, (American Psychiatric Association 2013) omitted fear of weight gain from criteria for binge eating disorder and some childhood eating disorders.

Far more than eating behaviour is disordered. Starvation, and consequences of purging and over-exercise, cause psychosocial and physical damage. Anorexia nervosa has the highest mortality rate of

TABLE 1 Main features of the eating disorders

	Anorexia nervosa	Bulimia nervosa	Binge eating disorder
Restrictive behaviours	Often extreme calorie restriction, with many dietary 'rules', including adoption of restrictive diets – extreme forms of veganism or 'clean eating' are common	As for anorexia nervosa, but less extreme than in the restrictive form of anorexia nervosa	Sufferer is less able to practise extreme food restriction
Binges	In the binge–purge subtype, binges occur. 'Subjective binges' describe unintended eating that is distressingly out of control though not objectively a large amount	The hallmark of bulimia nervosa is the large-scale, out-of-control consumption of amounts of food definitely more than would be normally consumed in a single sitting, and often food of a type not usually served as a meal (e.g. entire packets of biscuits, tubs of ice cream)	Binges are similar in quality to bulimia nervosa but usually less extreme in amount. The lack of control over such eating is experienced with shame and distress
Compensatory behaviours	Binge–purge subtype of anorexia nervosa involves purging by vomiting or laxatives. Restrictive subtype does not always, but may, change to binge–purge subtype in time. Deliberate overexercise to burn off calories is often a feature of restrictive anorexia nervosa	Binges are followed by such compensatory behaviours as self-induced vomiting, deliberate regurgitation or use of laxatives or diuretics. Deliberate overexercise to burn off calories also occurs	Such behaviours are less prominent in binge eating disorder
Affect and feeling	There is often pride in successful weight loss, with increasing levels of shame, anxiety and low mood as the disorder persists. Isolation and social avoidance contribute to this	Extremely high levels of shame are common, usually focused on bodily shame	Shame, depression and low self-esteem are common
Body-checking and body image avoidance	Many patients repeatedly check their body weight and composition by weighing, measuring, pinching their flesh, trying on garments to assess size, inspecting their appearance in mirrors or 'selfie' photographs, and making comments about their weight that are designed to elicit reassurance	Patients may engage in similar body-checking behaviours to those in anorexia nervosa, but many also practise body image avoidance and hide their bodies in public – being unable to wear swimwear, sportswear or other tightly fitting clothes. They may avoid being weighed out of shame and distress	Patients, particularly those who are overweight, are most likely to practise body image avoidance to avoid the distress and shame of exposure
Communication	Preoccupation with losing weight is extreme and interferes with other aspects of life. Web-based and social media activity is focused on losing weight	As in anorexia nervosa, preoccupation with losing weight may be extreme, although social life and work are often maintained. Web-based and social media activity is focused on losing weight	Body shame and low self-esteem may lead sufferers to engage in unrewarding, unduly deferential relationships

psychopathology of preoccupation with weight and shape.

Psychiatric classifications are limited by reliance on symptoms, whereas research is hampered by inaccurate diagnostic groupings. Treatment evidence is inextricably tied to existing categorisation. Clinicians should remember that a patient's failure to precisely meet listed criteria does not automatically exclude an eating disorder. Moreover, patients' diagnoses commonly migrate across categories during the course of illness.

Epidemiology

Eating disorders typically begin in early to mid-adolescence, but can emerge at any age (Nicholls 2011; Steinhausen 2015; Micali 2017). Lifetime

prevalence of anorexia nervosa in the general population in Western countries is about 1% among women and 0.5% among men. Gender distribution is less skewed in children. Adolescents have higher rates of full recovery and lower mortality than adults (mean mortality 2 v. 5%).

A recent Scandinavian register-based study (Reas 2018) found that, for both genders aged 10–49 years, incidence rates of anorexia nervosa were stable across the 7-year period to 2016, at 18.8–20.4 per 100 000 for narrowly defined and 33.2–39.5 per 100 000 for broadly defined anorexia nervosa. There was a significant annual increase in anorexia nervosa among 10- to 14-year-old girls. Overall rates of bulimia nervosa declined.

Register-based studies substantially underestimate true community prevalence, and it is reported that half of those who meet diagnostic criteria in the community do not access treatment. Not receiving treatment is even more likely in low- and middle-income countries, now seeing increased prevalence of formerly ‘Western’ disorders.

Risk factors

Genetics

Early family studies, large twin-based studies and smaller adoption studies demonstrate both a heritable component *and* significant influence of non-shared environmental factors in eating disorders. There is a strong familial component to anorexia nervosa, with estimates of heritability between 28 and 74%. The genetic contribution is polygenic. Large genome-wide association studies (GWAS) highlight the need to consider psychiatric and metabolic mechanisms involved in anorexia nervosa (Watson 2019). These studies, together with further research in the genetics of bulimia nervosa and binge eating disorder, may help elucidate new avenues of treatment.

Gene–environment interactions are complex. Recent work on gut microbiota has found reduced gut microbial diversity in anorexia nervosa, significantly associated with depression, anxiety and eating disorder symptoms (Kleiman 2015).

Structural and functional brain abnormalities

Neuroimaging often disappoints in revealing structural and functional abnormalities (Frank 2018). Studies in anorexia nervosa may reflect consequences of starvation rather than the psychiatric disorder. Rescans at normal weight cannot assume that brain nutrition is fully restored or that damage is reversible, although studies suggest that some brain tissue abnormalities recover with weight regain.

Reward and satiety circuits in the orbitofrontal cortex are important in both anorexia nervosa and bulimia nervosa. Structural magnetic resonance imaging has revealed that individuals with anorexia nervosa had increased right insula grey matter compared with controls (Frank 2018). This area is associated with self-recognition and interoceptive awareness. Prospective studies may explore whether these changes are biomarkers or sequelae.

Functional imaging, with symptom provocation, suggests abnormal responses in subcortical regions associated with stimulus-driven responses, and in prefrontal regions involved in evaluation and executive control (Kaye 2013). Abnormalities in reward circuit activation have also been found in anorexia nervosa (Monteleone 2018).

In anorexia nervosa the observed rigid thinking, poor central coherence and emotional avoidance may improve, at least partially, with re-nutrition (refeeding), but may persist and may also be observed in unaffected relatives (Kanakam 2013; Lang 2014).

Different eating disorders show different abnormalities of attentional bias. In bulimia nervosa and binge eating disorder there is increased attentional bias towards food-related cues, whereas individuals with anorexia nervosa have increased attentional bias towards disliked aspects of body image (Stojek 2018). This could guide specific interventions for different disorders.

Sociocultural factors

A prospective study of healthy adolescent girls (Stice 2017) found that impaired interpersonal functioning and negative affect were transdiagnostic risk factors. Pursuit of the thin ideal, body dissatisfaction, dieting and unhealthy weight-control behaviours increased risk for bingeing and purging disorders. Inherently lean girls, even if not pursuing the thin ideal, were at risk for anorexia nervosa.

The increase in eating disorders in lower-income societies follows Western lifestyle aspirations, nutritional habits and internalisation of the thin ideal. Sociocultural pressures appear more powerful in triggering bulimia nervosa and binge eating disorder than anorexia nervosa but may also trigger genetically vulnerable individuals to try to lose weight.

Childhood deprivation and trauma are important risk factors for many mental disorders and appear to be associated with increased risk of bulimia nervosa and binge eating disorder, but not anorexia nervosa (Larsen 2017).

Anorexia nervosa and bulimia nervosa are more common among females than males (Hoek 2006) and there are higher rates of disordered eating among sexual minorities, particularly transgender people (Calzo 2017).

Social media use and competitiveness can maintain and perhaps trigger eating disorders (Ferguson 2014; Mabe 2014). Treatment and prevention strategies might benefit from identifying differences between helpful and harmful behaviour online and in social media.

Prevention

A large number of prevention programmes are under development and evaluation. The most promising interventions use cognitive dissonance, cognitive-behavioural therapy (CBT) and media literacy. Although prevention programmes generally improve participants’ knowledge, and sometimes reduce risk factors, there is no evidence that they actually reduce development of diagnosable eating disorders.

Combined preventive approaches developed by eating disorder and obesity experts might avoid inadvertent triggering of disorders by focusing on the dangers of either extreme. Parent and carer groups regularly report onset of eating disorders following anti-obesity campaigns in schools.

Assessment of symptoms and risk

Prompt referral and treatment is important. Specialist intervention in the first 3 years gives the best prognosis (Stice 2017) and evaluation of a novel service in the UK – the First Episode and Rapid Early Intervention for Eating Disorders (FREED) service – suggests that early treatment of young adults reduces the need for later, more costly interventions (Fukutomi 2020).

Signs and symptoms

Signs and symptoms of eating disorders should be positively elicited by sympathetic enquiry. Individuals often present with physical symptoms – weight loss, weakness, fainting, constipation, nausea and amenorrhoea – or psychiatric symptoms such as depression and anxiety. The need to exclude organic causes should not delay assertive re-nutrition, particularly in younger patients. Any organ system may be affected, and Table 2 lists possible signs and symptoms.

Psychosocial disorder may be assessed by asking the patient, or better still a friend or relative, to describe a typical day. The Eating Disorder Examination Questionnaire (EDE-Q) (Fairburn 1994) is the gold-standard validated self-report questionnaire and is available free of charge. Widely used standard measures may be used to assess quality of life, anxiety and depression. The Clinical Impairment Assessment (CIA) questionnaire (Bohn 2008) assesses clinical severity.

Physical investigations

Blood tests may reveal the low white cell count of starvation and deranged liver function (starvation or substance use). At any weight there may be physical evidence of purging (e.g. low potassium) or of the ‘fluid loading’ in which many patients engage to fill their hungry bellies or to disguise their weight. Overhydration can cause low sodium levels and low gravity in urine samples. Electrolyte derangements may cause cardiovascular irregularities, seizures and even permanent damage. Asymptomatic low glucose levels in starved patients are less worrying than often feared, since the body adapts to metabolise ketones. Thyroid and other endocrine changes are mostly adaptive, to optimise energy expenditure.

After initial screening, patients at normal weight may need no further investigation. Those at low or changing weight, or with abnormal findings, require further monitoring.

Abnormal electrolytes, dizziness, fainting, palpitations, drug use or heart conditions should trigger electrocardiogram (ECG) investigation, looking for prolonged QTc interval. Patients at low weight, particularly with amenorrhoea, should undergo a non-urgent dual-energy X-ray absorptiometry (DEXA) bone density scan because of the risk of osteopaenia and osteoporosis (Schorr 2017). This allows advice on safe activity and provides a baseline to show response to nutrition. Bone mineral density loss is mostly reversible with healthy nutrition over several years.

Patients may omit prescribed medication if they believe it causes weight gain. This can include

TABLE 2 Possible signs and symptoms^a

Organ/system	Signs and symptoms
Cardiovascular	Bradycardia Hypotension Mitral valve prolapse Arrhythmia and sudden death Chest pain Dizziness and fainting
Pulmonary	Respiratory failure Spontaneous pneumothorax
Metabolic	Hypokalaemia Dehydration Nephropathy Metabolic acidosis
Neuropsychiatric	Depressed mood Anxiety Obsessive and compulsive symptoms Poor concentration and memory
Haematological	Low white cell count, particularly neutrophils Pancytopenia
Skin and hair	Dry skin Alopecia Lanugo hair Starvation-associated pruritis Russell's sign (calluses on the back of hand from self-induced vomiting)
Neurological	Cerebral atrophy
Endocrine	Amenorrhoea Reduced sex drive Infertility osteopaenia and osteoporosis Thyroid abnormalities such as sick euthyroid Hypercortisolaemia Hypoglycaemia
Gastrointestinal	Dental erosion Parotid gland swelling Gastrointestinal reflux Barrett's oesophagus Mallory–Weiss tear Constipation Refeeding pancreatitis Acute gastric dilatation Delayed gastric emptying, leading to pain and bloating Elevated liver enzymes

a. Note that any organ system can be affected by an eating disorder and this list is not exhaustive.

insulin, steroids or anticonvulsants, with catastrophic results.

'Compensatory behaviours' that may be hidden or subtly disguised include purging by self-induced vomiting, taking diuretics or laxatives, overuse of prescribed or acquired drugs associated with weight loss and compulsive overactivity. These may be prioritised over pleasurable and useful experiences.

Body-checking, reassurance-seeking and 'coaching' in ways to lose weight maintain the preoccupation with eating, shape and weight. Compulsive accessing of social media sites is a powerful maintaining factor (Mabe 2014).

Risk

Management of the acute physical risks of eating disorders is addressed further below (in the section 'Acute medical treatment'). Risk of self-harm and suicide should be considered and monitored – distress sometimes worsens as weight increases. Physical compromise increases the lethality of self-harm that is not necessarily intended to end life.

Risk assessment is particularly delicate when sufferers have social responsibilities such as parenting, nursing, medicine and teaching. Patients sometimes coerce others to eat, or neglect children to engage in compulsive exercise. Witnessing purging and self-harm is traumatic. Clinicians are also responsible for giving advice on safety to drive (Box 1).

Acute medical treatment

A thorough knowledge of medical complications can be life-saving, if backed up by clinician and service networks. The Management of Really Sick Patients with Anorexia Nervosa (MARSIPAN) guideline, first published in 2010, was developed in response to a number of avoidable deaths of patients with anorexia nervosa in medical units. The second edition (Royal College of Psychiatrists 2014), as well as the Junior MARSIPAN guideline (Royal College of Psychiatrists 2012), are currently under review and readers should ensure that they access the most up-to-date editions. General (family) practitioners and generalist health professionals should be aware of their local specialist eating disorder team, which will aim to work closely with an identified physician or paediatrician to enact MARSIPAN.

Psychological recovery from low-weight anorexia nervosa must be supported by medical management to prevent or mitigate lasting physical damage. Death is preventable with timely, effective treatment. The key to safe management is for key clinicians to have prepared and trained together, rather than improvise care plans in crisis.

BOX 1 Checklist to guide fitness to drive of people with eating disorders

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| <ol style="list-style-type: none"> 1. Is the patient suicidal or insufficiently caring of whether they live or die? 2. What medication is prescribed and taken? Might it interfere with driving? 3. Does the patient use alcohol, other over-the-counter, online-purchased or street drugs (including painkillers, diuretics, laxatives, etc.)? 4. Are there comorbid physical or psychiatric conditions that may be inadequately managed, e.g. diabetes, epilepsy. 5. Has the patient had any episodes of collapse, faints, falls? 6. On examination of heart and brain, is there risk of collapse? 7. Is the patient stable at their current weight or have there been fluctuations (in both directions)? 8. In particular, has the patient had a recent increase in their weight so that there will be increased metabolism without corresponding replenishment of glycogen stores? 9. Does the patient have adequate physical strength to do an emergency stop etc. if necessary? 10. Has the patient ever had a recorded low blood glucose? If so, has there been | <p>normal blood glucose (above 4) in the last four blood glucose tests (>4 using venous blood, >5 on blood glucose test strips)?</p> <ol style="list-style-type: none"> 11. Does the patient induce vomiting on the same days as driving? If so, how long elapses after vomiting before driving, and does the patient eat/drink again before driving? Frequent vomiting is likely to result in unstable electrolytes and fluctuating concentration. 12. Is the patient so preoccupied by obsessional thoughts, ruminations and calculations that they cannot prioritise road safety? Sometimes their behaviour in therapy will reveal preoccupations. 13. What about impulsivity and rage? Patients who have to satisfy their binges at all costs (e.g. shoplifting) may not be able to show the patience needed e.g. at traffic or pedestrian lights. 14. Does the patient eat while driving or engage in compensatory behaviours such as purging while driving? 15. Do 'checking behaviours' occur in the car, such as using mirrors to check appearance rather than for traffic, or using phones or calculators to add up calories while at the wheel? |
|--|--|

When MARSIPAN is enacted, medical rather than psychiatric wards can be the safest setting, meeting the need for frequent blood tests, intravenous treatment, continuous ECGs and intensive care unit (ICU) availability. Keeping the individual warm, rested, nourished and protected from infection is usually taken for granted with medical patients, but compulsive eating disorder behaviours may sabotage this and alienate staff. Both psychiatric nursing skills and dietetic expertise are indispensable in refeeding low-weight patients.

The potentially fatal refeeding syndrome is caused by the switch from fasting gluconeogenesis to carbohydrate-induced insulin release, inducing considerable intracellular uptake of potassium, phosphate and magnesium from the bloodstream. Unfortunately, avoidance of overfeeding can result in lethal underfeeding and MARSIPAN guidelines advise on this.

Continuing physical management involves balancing the need to relieve constipation without fuelling an obsessive laxative habit, improving bone density without recourse to ineffective treatments, and following up on emerging or relapsing instabilities and abnormalities. Infections and hypothermia are risks in winter. Regular dental assessment is

helpful. Contraceptive discussions are important even with low-weight patients.

Evidence-based psychological treatment

When physical stability, with appropriate monitoring, is assured, the most effective treatments for eating disorders are psychological therapies specifically adapted to target eating disorder pathology, as summarised in National Institute for Health and Care Excellence (NICE) guidelines (National Institute for Health and Care Excellence 2017).

Anorexia nervosa

Treatment for anorexia nervosa combines emotion regulation, distress tolerance, body image acceptance and interpersonal interventions alongside weight gain. In effect, the patient is exposed to a normal healthy weight and learns to tolerate this without eating disorder responses. In-patient or carer containment may be needed initially, followed by careful transition to the patient taking back responsibility.

Manualised FBT (Lock 2015) has the best evidence base for anorexia nervosa in the under-18s, with individual eating disorder focused cognitive-behavioural therapy (CBT-ED) as a second choice. Family-based models are being adapted for young adults, and for adults and their partners (Bulik 2011; Wierenga 2018).

NICE acknowledged the ‘low- to very-low’ quality of evidence for psychological treatments for adult anorexia nervosa, but recommended CBT-ED, the Maudsley Anorexia Nervosa Treatment for Adults (MANTRA) and specialist supportive clinical management (SSCM). Randomised controlled trials (RCTs) are underpowered to show superiority for any specific therapy. Even the best current treatments bring about significant improvement in only 50% of patients (Brockmeyer 2018; Murray 2019).

A systematic review and meta-analysis of 35 treatment-outcome RCTs for anorexia nervosa generated concern about lack of effect (Murray 2019) and disconnect between weight gain and psychological improvement. Clearly, further research is urgently needed.

Bulimia nervosa

There is more evidence for the effectiveness of CBT-ED for bulimia nervosa than for anorexia nervosa. Guided self-help appears beneficial and cost-effective, and individual CBT-ED is effective for adolescents as well as adults (Slade 2018). Treatment involves resetting dysregulated cycles of restriction–binge–purge. Patients establish regular meal-times and stabilise electrolyte levels. As hunger comes under control, avoided foods are reintroduced. Intrapsychic and interpersonal skills are learned for coping with life stresses.

Binge eating disorder

There is evidence that guided self-help and group CBT-ED improve binge frequency and EDE total score compared with waiting-list control (Vocks 2010), but there is no evidence that this treatment brings about weight loss.

Pharmacological treatment

Anorexia nervosa

Many patients are prescribed psychotropic drugs for associated symptoms of depression, anxiety and obsessive–compulsive disorder (OCD), although there are scant data on effectiveness in the presence of anorexia nervosa. The main medications directed at the eating disorder pathology are the atypical antipsychotics, particularly olanzapine, shown in some studies to reduce illness preoccupations and anxiety during refeeding. Surprisingly, weight gains are not significant in pooled meta-analyses of placebo-controlled trials. A recent out-patient RCT showed modest benefit on weight gain for olanzapine, but with no significant benefit on psychological symptoms (Attia 2019). Cannabinoid receptor agonist studies are at an early stage. A network meta-analysis is underway to build evidence for both pharmacological and non-pharmacological treatments for anorexia nervosa (Wade 2017).

Bulimia nervosa

High-dose antidepressant treatment has been shown to be of benefit in bulimia nervosa. CBT-based psychological therapies are more effective and longer lasting, but prescribing is an attractive choice if depressive symptoms are prominent, if CBT is unavailable or as adjunctive treatment.

Binge eating disorder

A recent meta-analysis (Peat 2017) found that lisdexamfetamine (LDX) increased binge abstinence more than second-generation antidepressant drugs. LDX is not approved for the treatment of binge eating disorder in the UK and European Union.

Mental health legislation

UK mental health legislation allows compulsory treatment when the illness is of a nature or degree that warrants hospital treatment and there are risks to health or safety. Any fully registered medical practitioner can detain patients for brief periods. For longer periods, psychiatrists approved under the relevant legislation must be involved. Clinicians should be aware of the relevant legislation where they work. Individuals with eating disorders also perceive high levels of ‘informal coercion’.

Ambivalence and the perception of coercion often fluctuate over the course of treatment.

Compulsory treatment may increase eating disorder behaviours in an attempt to regain control. Clinicians may then impose yet more restriction, so the vicious circle strengthens, unless this is managed by working to engage the patient. Studies have shown that compulsory treatment may improve survival, even when detained patients had poorer functioning before admission (Ward 2016). Detention may retain people in treatment long enough to establish an effective therapeutic alliance that will then be the basis for ongoing work towards recovery.

Prognosis

The standardised mortality ratio for anorexia nervosa is 5.9 – the highest mortality rate of all psychiatric disorders (Franko 2013). Most deaths result – shockingly – from starvation. Significant numbers are attributed to suicide. With treatment, around 50% of people with anorexia nervosa recover fully (Murray 2019). Lower body mass index (BMI) or higher age at admission, persistence of binge eating and purging behaviours, substance misuse and type 1 diabetes all predict poor outcome. Motherhood is related to better outcome, as is access to specialist eating disorder services.

Recovery from bulimia nervosa is widely considered greater than from anorexia nervosa. At 9-year follow-up, 68.2% of participants with bulimia nervosa had recovered, compared with 31.4% of those with anorexia nervosa; however, by 22-year follow-up, 62.8% of participants with anorexia nervosa had recovered (the recovery rate for bulimia nervosa remained at 68.2%) (Eddy 2017). In contrast, despite optimistic long-term results from centres of excellence (Franko 2018), other evidence suggests recovery rates from anorexia nervosa decrease over time, particularly after 12 years (Papadopoulos 2009).

There is disagreement about what constitutes ‘recovery’ from eating disorders, with consequent variation in outcome figures. Qualitative meta-analysis finds that recovered individuals value self-acceptance, autonomy and interpersonal relationships as much as reduced eating disorder symptoms (de Vos 2017).

Comorbidity

Psychiatric disorders

Behavioural symptoms of eating disorders often overlap with those of other psychiatric conditions, although the meaning of such behaviours may be different. Some apparent comorbidity may be

secondary to the eating disorder – for instance, starvation from any cause frequently causes depression.

Over 70% of people with anorexia nervosa report a lifetime mood disorder (Keski-Rahkonen 2016). Between 25 and 75% report a lifetime history of an anxiety disorder (Raney 2008), typically preceding the anorexic illness. Bulimia nervosa is even more robustly associated with affective and anxiety disorders (Becker 2018). Around 80% of people with anorexia nervosa experience OCD symptoms at some point. Prior OCD is associated with increased risk of subsequent bulimia nervosa (Hofer 2018). Register-based studies confirm aggregation of autism spectrum disorder in probands with anorexia nervosa and their relatives (Koch 2015).

The prevalence of alcohol misuse in restrictive anorexia nervosa is similar to that in the general population. People with bulimic illnesses have a higher prevalence of substance misuse (Root 2010). Eating disorder sufferers use harmful substances to reduce appetite, trigger vomiting, burn off calories or permit overexercise despite pain. There is increased prevalence of other disorders of impulsivity and lack of control in people with bulimia nervosa (Kim 2018; Sala 2018).

Insulin-dependent diabetes

Type 1 diabetes is a disease of insulin deficiency, resulting from destruction of pancreatic insulin-producing cells. This causes hyperglycaemia, glucosuria, ketone production and weight loss. Diabetics lose weight easily by omitting insulin rather than by diet, overexercise and purging. Between 30 and 40% of young people with diabetes omit or reduce insulin with the intention of losing weight (Hasken 2010). Longitudinal studies suggest an increase in eating disorders among young women with type 1 diabetes (Larranaga 2011). The peak onset of type 1 diabetes is at age 10–14, unfortunately just as puberty brings increased body image concerns.

The term ‘diabulimia’ (not recognised in official diagnostic criteria) implies that causing calories to be excreted in the form of glycosuria and high blood glucose levels is analogous to purging. In type 1 diabetes conventional eating disorder behaviours are unlikely, so we recommend use of the Diabetes Eating Problem Survey – Revised (DEPS-R) screening tool (Markowitz 2010).

In addition to the serious but mostly reversible complications of non-diabetic eating disorders, diabetic complications are cumulative and irreversible. They include retinopathy, kidney disease and peripheral neuropathy. Insulin reduction increases muscle breakdown, risk of dehydration, fatigue and risk of infection. The presence of an eating disorder significantly and greatly increases rates of

ketoacidosis, hospital admissions, morbidity and mortality and there is an association between poorer glycaemic control and self-reported eating disorder symptoms.

There is a potential for preventive approaches in diabetes clinics. Clinicians need awareness of eating disorder patients' acute sensitivity to weight and body image, and psychological 'denial' of the risks of diabetic complications. Such patients avoid giving insulin, avoid monitoring their blood glucose to prevent being confronted with 'bad' results and avoid clinic appointments for fear of being 'told off'.

Personality disorders

Around 30% of patients with an eating disorder meet criteria for at least one personality disorder (Solmi 2018). Many meet criteria for more than one. Some features are common to both disorders, and it is possible that some are consequences of the eating disorder or its treatment, rather than primary features of a personality disorder.

The presence of a comorbid personality disorder causes difficulties in terms of diagnosis and management. There are likely to be additional problems in engaging such patients, and increased risk of suicide and other mortality. The eating disorder may be used to manage the symptoms of the personality disorder, or the personality disorder may predispose to eating disorder behaviours. Individualised formulation helps both patient and clinicians to understand these functions. Alternative emotional coping behaviours can help patients to give up less adaptive behaviours.

Treatment access is complicated by service boundaries. NICE recommends that people with eating disorders are managed by specialists (National Institute for Health and Care Excellence 2017). People with personality disorders tend to be managed by community mental health services and may be referred between services according to dominant symptoms, receiving inadequate treatment in each when the disorders are intertwined. Eating disorder treatment focused on reducing purging may lead to an increase in trauma-related symptoms if purging was a way to cope with flashbacks. Difficulty managing relationships with different clinicians is a notorious aspect of personality disorder presentation, so joint working is essential.

Fertility, pregnancy and the puerperium

The effects of starvation on the hypothalamic–pituitary–gonadal axis often cause amenorrhoea. Women attending infertility clinics have a high prevalence of lifetime eating disorder. Conversely

some women become pregnant without having periods, so contraception is still relevant.

Body changes during pregnancy have various effects. Some women 'suspend' their eating disorder, attributing the need for nutrition to a cherished baby, but others report increased body image concerns and fear that weight will get out of control (Rocco 2005).

A mother's eating disorder can significantly affect fetal development, especially neural development. Women with anorexia nervosa are more likely to have smaller-than-average babies and increased risk of postnatal depression. Women with active bulimia nervosa have increased risk of miscarriage (Micali 2017).

Severe and enduring anorexia nervosa

Definitions of 'severe and enduring anorexia nervosa' (SE-AN) vary. The very concept is contentious. 'SE-AN' is evoked when the eating disorder does not appear to respond to our best available treatments or responds only with extreme distress. A rehabilitation model, shifting away from weight recovery to focus on quality of life, may be more appropriate, reducing adverse effects, achieving some symptom control and setting attainable goals (Bamford 2015).

Recent studies suggest that CBT-ED is effective for people with long-standing anorexia nervosa in community or in-patient treatment (Raykos 2018), but clinicians as well as patients may experience hopelessness. If this is not managed in supervision, 'therapeutic nihilism' can blight patients' chances. Patients may be considered 'treatment resistant' when not all appropriate treatment approaches have been attempted. A detailed independent second opinion can suggest other avenues to follow.

Emerging treatments

Current research activity is most active in exploring ever more powerful, more focused or novel psychological therapies such as cognitive remediation therapy and exposure-based therapy, and third-wave behavioural therapies such as radically open dialectical behaviour therapy. There is also research into improved delivery of treatments in a range of web-based or electronic formats, both to exploit media that may be most acceptable to patients and also to provide equitable delivery of care from small specialist services across remote or low-income populations.

One bold, and often controversial, approach to both the understanding and treatment of eating disorders is the practice of neurosurgery and deep brain stimulation.

Controversies, uncertainties and outstanding research questions

Evolving considerations about the nature and classification of the eating disorders affect the precision with which treatments can be researched. Most treatments involve biological, behavioural and psychosocial elements, making it hard to dissect out effects.

Psychological treatments rely crucially on therapeutic attachments. Relationships may be repeatedly interrupted when treatment has to span child and adult services, or local out-patient services follow distant in-patient treatment. Management of such crucial transitions is discussed in a recent report from the Royal College of Psychiatrists (Crockett 2017).

The role of social media in causation and maintenance of the eating disorders is a contemporary field for exploration, as is the potential for the proper exploitation of new technology in prevention and treatment. A linked concern is whether obesity prevention campaigns fuel eating disorders.

Ignorance of the scale of the problem, and public perception of eating disorders as ‘self-imposed lifestyle choices’, mean that services struggle for adequate funding. Research suggests that even within specialist services there is inadequate implementation of evidence-based treatments, and problems of ‘therapeutic drift’. Clinicians need skills-based supervision to deliver the most effective interventions rather than those with which they are most comfortable. Meanwhile services need to collect data on both clinical activity and outcomes.

Modern neuroscience holds promise for developing understanding and treatment approaches, while psychotherapeutic, ethical and philosophical work yield insights for overcoming obstacles to progress in these uniquely ego-syntonic disorders. We may learn how to amplify psychotherapeutic benefits by pretreating the brain with surgery, medication or electrical stimulation. In this world of endemic preoccupation with body image and valid concerns about obesity, psychiatry is starved of good evidence and desperate to discover effective prevention and treatment.

Conclusions

This review has not attempted to describe the whole range of psychologically driven disorders characterised by derangement of eating behaviour, since, in UK practice at least, specialist eating disorder services and academic research focus on those disorders characterised by a maladaptive drive to lose weight rooted in extreme fear of weight gain and fatness. Various restrictive and ‘compensatory’ behaviours pose physical dangers and involve

preoccupations and emotions that interfere with human experience and life achievements. Despite the appalling suffering, which also affects family and friends, the obsessive fear of weight gain often obliges sufferers to avoid rather than seek effective treatment.

Genetic research has identified a substantial, complex genetic contribution to causation and suggests that anorexia nervosa and bulimia nervosa, though overlapping, may be somewhat differently inherited. Adverse childhood experiences appear to have a non-specific relationship with all mental illness, but the body image competitiveness of modern society and social media may be triggering increased incidence in vulnerable young people, and preventive strategies are addressing this, particularly in high-risk groups.

There is little evidence for psychotropic medication as a stand-alone treatment, and most prescribing addresses comorbid psychiatric conditions. Meanwhile, integrated medical and psychiatric interventions save life and safely improve nutrition. Integrated medical expertise is most challenged when the patient is pregnant or has type 1 diabetes.

At our present stage of understanding, psychological therapies are the mainstay of treatment. The CBT model has been variously adapted so that both cognitive and behavioural exercises are used to challenge and tolerate the emotional distress that accompanies acceptance of a normal body weight. A strong, mutually respectful attachment to the therapist or therapeutic team is a crucial element of therapy. In younger patients, family-based models mobilise the strong attachment to parents to permit refeeding and toleration of the distress involved.

Author contributions

The two authors contributed equally to the writing and editing of the main text and preparation of the tables and the box.

Declaration of interest

None.

ICMJE forms are in the supplementary material, available online at <https://doi.org/10.1192/bja.2020.24>.

References

- American Psychiatric Association (2013) *Diagnostic and Statistical Manual of Mental Disorders* (5th edn) (DSM-5). APA.
- Attia E, Steinglass JE, Walsh BT, et al (2019) Olanzapine versus placebo in adult outpatients with anorexia nervosa: a randomized clinical trial. *American Journal of Psychiatry*, **176**: 449–56.
- Bamford B, Barras C, Sly R, et al (2015) Eating disorder symptoms and quality of life: where should clinicians place their focus in severe and enduring anorexia nervosa? *International Journal of Eating Disorders*, **48**: 133–8.

MCQ answers

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- Becker KR, Fischer S, Crosby RD, et al (2018) Dimensional analysis of emotion trajectories before and after disordered eating behaviors in a sample of women with bulimia nervosa. *Psychiatry Research*, **268**: 490–500.
- Bohn K, Doll HA, Cooper Z, et al (2008) The measurement of impairment due to eating disorder psychopathology. *Behaviour Research and Therapy*, **46**: 1105–10.
- Brockmeyer T, Friederich HC, Schmidt U (2018) Advances in the treatment of anorexia nervosa: a review of established and emerging interventions. *Psychological Medicine*, **48**: 1228–56.
- Bulik CM, Baucom DH, Kirby JS, et al (2011) Uniting Couples (in the treatment of) Anorexia Nervosa (UCAN). *International Journal of Eating Disorders*, **44**: 19–28.
- Calzo JP, Blashill AJ, Brown TA, et al (2017) Eating disorders and disordered weight and shape control behaviors in sexual minority populations. *Current Psychiatry Reports*, **19**(8): 49.
- Charland LC, Hope T, Stewart A, et al (2013) Anorexia nervosa as a passion. *Philosophy, Psychiatry & Psychology*, **20**: 353–65.
- Crockett P, Morris J, Winston AP (2017) *Managing Transitions When the Patient Has an Eating Disorder: Guidance for Good Practice (College Report CR208)*. Royal College of Psychiatrists.
- de Vos JA, Lamarre A, Radstaak M, et al (2017) Identifying fundamental criteria for eating disorder recovery: a systematic review and qualitative meta-analysis. *Journal of Eating Disorders*, **5**: 34.
- Eddy KT, Tabri N, Thomas JJ, et al (2017) Recovery from anorexia nervosa and bulimia nervosa at 22-year follow-up. *Journal of Clinical Psychiatry*, **78**: 184–9.
- Fairburn CG, Beglin SJ (1994) Assessment of eating disorders: interview or self-report questionnaire? *International Journal of Eating Disorders*, **16**: 363–70.
- Ferguson CJ, Munoz ME, Garza A, et al (2014) Concurrent and prospective analyses of peer, television and social media influences on body dissatisfaction, eating disorder symptoms and life satisfaction in adolescent girls. *Journal of Youth and Adolescence*, **43**: 1–14.
- Frank GK, Favaro A, Marsh R, et al (2018) Toward valid and reliable brain imaging results in eating disorders. *International Journal of Eating Disorders*, **51**: 250–61.
- Franko DL, Keshaviah A, Eddy KT, et al (2013) A longitudinal investigation of mortality in anorexia nervosa and bulimia nervosa. *American Journal of Psychiatry*, **170**: 917–25.
- Franko DL, Tabri N, Keshaviah A, et al (2018) Predictors of long-term recovery in anorexia nervosa and bulimia nervosa: data from a 22-year longitudinal study. *Journal of Psychiatric Research*, **96**: 183–8.
- Fukutomi A, Austin A, McClelland J, et al (2020) First episode rapid early intervention for eating disorders: a two-year follow-up. *Early Intervention in Psychiatry*, **14**: 137–41.
- Hasken J, Kresl L, Nydegger T, et al (2010) Diabulimia and the role of school health personnel. *Journal of School Health*, **80**: 465–9.
- Hoek HW (2006) Incidence, prevalence and mortality of anorexia nervosa and other eating disorders. *Current Opinion in Psychiatry*, **19**: 389–94.
- Hofer PD, Wahl K, Meyer AH, et al (2018) Obsessive-compulsive disorder and the risk of subsequent mental disorders: a community study of adolescents and young adults. *Depression and Anxiety*, **35**: 339–45.
- Kanakam N, Treasure J (2013) A review of cognitive neuropsychiatry in the taxonomy of eating disorders: state, trait, or genetic? *Cognitive Neuropsychiatry*, **18**: 83–114.
- Kaye WH, Wierenga CE, Bailer UF, et al (2013) Nothing tastes as good as skinny feels: the neurobiology of anorexia nervosa. *Trends in Neurosciences*, **36**: 110–20.
- Keski-Rahkonen A, Mustelin L (2016) Epidemiology of eating disorders in Europe: prevalence, incidence, comorbidity, course, consequences, and risk factors. *Current Opinion in Psychiatry*, **29**: 340–5.
- Kim HS, Von Ranson KM, Hodgins DC, et al (2018) Demographic, psychiatric, and personality correlates of adults seeking treatment for disordered gambling with a comorbid binge/purge type eating disorder. *European Eating Disorders Review*, **26**: 508–18.
- Kleiman SC, Watson HJ, Bulik-Sullivan EC, et al (2015) The intestinal microbiota in acute anorexia nervosa and during renourishment: relationship to depression, anxiety, and eating disorder psychopathology. *Psychosomatic Medicine*, **77**: 969–81.
- Koch SV, Larsen JT, Mouridsen SE, et al (2015) Autism spectrum disorder in individuals with anorexia nervosa and in their first- and second-degree relatives: Danish nationwide register-based cohort-study. *British Journal of Psychiatry*, **206**: 401–7.
- Lang K, Lopez C, Stahl D, et al (2014) Central coherence in eating disorders: an updated systematic review and meta-analysis. *World Journal of Biological Psychiatry*, **15**: 586–98.
- Larranaga A, Docet MF, Garcia-Mayor RV (2011) Disordered eating behaviors in type 1 diabetic patients. *World Journal of Diabetes*, **2**: 189–95.
- Larsen JT, Munk-Olsen T, Bulik CM, et al (2017) Early childhood adversities and risk of eating disorders in women: a Danish register-based cohort study. *International Journal of Eating Disorders*, **50**: 1404–12.
- Lock J, Le Grange D (2015) *Treatment Manual for Anorexia Nervosa: A Family-Based Approach* (2nd edn). Guilford Press.
- Mabe AG, Forney KJ, Keel PK (2014) Do you 'like' my photo? Facebook use maintains eating disorder risk. *International Journal of Eating Disorders*, **47**: 516–23.
- Markowitz JT, Butler DA, Volkeneing LK, et al (2010) Brief screening tool for disordered eating in diabetes: internal consistency and external validity in a contemporary sample of pediatric patients with type 1 diabetes. *Diabetes Care*, **33**: 495–500.
- Micali N, Martini MG, Thomas JJ, et al (2017) Lifetime and 12-month prevalence of eating disorders amongst women in mid-life: a population-based study of diagnoses and risk factors. *BMC Medicine*, **15**(1): 12.
- Monteleone AM, Castellini G, Volpe U, et al (2018) Neuroendocrinology and brain imaging of reward in eating disorders: a possible key to the treatment of anorexia nervosa and bulimia nervosa. *Progress in Neuro-Psychopharmacology & Biological Psychiatry*, **80**: 132–42.
- Murray SB, Quintana DS, Loeb KL, et al (2019) Treatment outcomes for anorexia nervosa: a systematic review and meta-analysis of randomized controlled trials. *Psychological Medicine*, **49**: 535–544.
- National Institute for Health and Care Excellence (2017) *Eating Disorders: Recognition and Treatment (NICE Guideline NG69)*. NICE.
- Nicholls DE, Lynn R, Viner RM (2011) Childhood eating disorders: British national surveillance study. *British Journal of Psychiatry*, **198**: 295–301.
- Papadopoulou FC, Ekblom A, Brandt L, et al (2009) Excess mortality, causes of death and prognostic factors in anorexia nervosa. *British Journal of Psychiatry*, **194**: 10–7.
- Peat CM, Berkman ND, Lohr KN, et al (2017) Comparative effectiveness of treatments for binge-eating disorder: systematic review and network meta-analysis. *European Eating Disorders Review*, **25**: 317–28.
- Raney TJ, Thornton LM, Berrettini W, et al (2008) Influence of overanxious disorder of childhood on the expression of anorexia nervosa. *International Journal of Eating Disorders*, **41**: 326–32.
- Raykos BC, Erceg-Hurn DM, Mcevoy PM, et al (2018) Severe and enduring anorexia nervosa? Illness severity and duration are unrelated to outcomes from cognitive behaviour therapy. *Journal of Consulting and Clinical Psychology*, **86**: 702–9.
- Reas DL, Ro O (2018) Time trends in healthcare-detected incidence of anorexia nervosa and bulimia nervosa in the Norwegian National Patient Register (2010–2016). *International Journal of Eating Disorders*, **51**: 1144–52.
- Rocco PL, Orbitello B, Perini L, et al (2005) Effects of pregnancy on eating attitudes and disorders: a prospective study. *Journal of Psychosomatic Research*, **59**: 175–9.
- Root TL, Pinheiro AP, Thornton L, et al (2010) Substance use disorders in women with anorexia nervosa. *International Journal of Eating Disorders*, **43**: 14–21.
- Royal College of Psychiatrists (2012) *Junior MARSIPAN: Management of Really Sick Patients under 18 with Anorexia Nervosa (College Report CR168)*. Royal College of Psychiatrists.

- Royal Colleges of Psychiatrists, Royal Colleges of Physicians, Royal Colleges of Pathologists (2014) *MARSIPAN: Management of Really Sick Patients with Anorexia Nervosa (2nd edn) (College Report CR189)*. Royal College of Psychiatrists.
- Sala L, Martinotti G, Carenti ML, et al (2018) Attention-deficit/hyperactivity disorder symptoms and psychological comorbidity in eating disorder patients. *Eating and Weight Disorders*, **23**: 513–9.
- Schorr M, Thomas JJ, Eddy KT, et al (2017) Bone density, body composition, and psychopathology of anorexia nervosa spectrum disorders in DSM-IV vs DSM-5. *International Journal of Eating Disorders*, **50**: 343–51.
- Slade E, Keeney E, Mavranzouli I, et al (2018) Treatments for bulimia nervosa: a network meta-analysis. *Psychological Medicine*, **48**: 2629–36.
- Solmi M, Collantoni E, Meneguzzo P, et al (2018) Network analysis of specific psychopathology and psychiatric symptoms in patients with eating disorders. *International Journal of Eating Disorders*, **51**: 80–692.
- Steinhausen HC, Jensen CM (2015) Time trends in lifetime incidence rates of first-time diagnosed anorexia nervosa and bulimia nervosa across 16 years in a Danish nationwide psychiatric registry study. *International Journal of Eating Disorders*, **48**: 845–50.
- Stice E, Gau JM, Rohde P, et al (2017) Risk factors that predict future onset of each DSM-5 eating disorder: predictive specificity in high-risk adolescent females. *Journal of Abnormal Psychology*, **126**: 38–51.
- Stojek M, Shank LM, Vannucci A, et al (2018) A systematic review of attentional biases in disorders involving binge eating. *Appetite*, **123**: 367–89.
- Vocks S, Tuschen-Caffier B, Pietrowsky R, et al (2010) Meta-analysis of the effectiveness of psychological and pharmacological treatments for binge eating disorder. *International Journal of Eating Disorders*, **43**: 205–17.
- Wade TD, Treasure J, Schmidt U, et al (2017) Comparative efficacy of pharmacological and non-pharmacological interventions for the acute treatment of adult outpatients with anorexia nervosa: study protocol for the systematic review and network meta-analysis of individual data. *Journal of Eating Disorders*, **5**: 24.
- Ward A, Ramsay R, Russell G, et al (2016) Follow-up mortality study of compulsorily treated patients with anorexia nervosa. *International Journal of Eating Disorders*, **49**(4): 435.
- Watson HJ, Yilmaz Z, Thornton LM, et al (2019) Genome-wide association study identifies eight risk loci and implicates metabo-psychiatric origins for anorexia nervosa. *Nature Genetics*, **51**: 1207–14.
- Wierenga CE, Hill L, Knatz Peck S, et al (2018) The acceptability, feasibility, and possible benefits of a neurobiologically-informed 5-day multifamily treatment for adults with anorexia nervosa. *International Journal of Eating Disorders*, **51**: 863–89.
- World Health Organization (2018) *ICD-11 for Mortality and Morbidity Statistics. Release version 04/2019*. WHO (<https://icd.who.int/browse11/l-m/en> [cited 5 Jan 2020]).

MCQs

Select the single best option for each question stem

1 Lifetime prevalence of anorexia nervosa in European women is estimated to be about:

- a 0.05%
- b 1%
- c 5%
- d 10%
- e 20%.

2 Current knowledge regarding the causation of eating disorders suggests that:

- a bulimia nervosa and binge eating disorder are inherited in a polygenic fashion, whereas anorexia nervosa is now thought to show autosomal recessive inheritance with partial penetrance
- b a history of childhood sexual abuse is strongly predictive of the development of anorexia nervosa in the early teens
- c there is a strong familial component to binge eating disorder, with estimates of heritability between 28 and 74%
- d scanning studies have shown that, compared with controls, individuals with anorexia nervosa have increases in brain areas associated with self-recognition and interoceptive awareness
- e an 'over-involved' style of parenting, particularly in the mother–daughter relationship, predisposes girls to develop eating disorders.

3 Regarding assessment of patients with probable eating disorders:

- a the Eating Disorder Examination Questionnaire (EDE-Q) is the gold-standard validated self-report questionnaire for assessment of eating disorders in adults
- b the MARSIPAN checklist assesses the psychosocial severity of an eating disorder
- c the Clinical Impairment Assessment (CIA) questionnaire rates weight change in recovering eating disorders
- d doctors have a responsibility to consider patients' fitness to drive only if the patient is significantly underweight
- e all patients with anorexia nervosa require an urgent DEXA scan of bone to screen for life-threatening osteoporosis.

4 There is evidence of at least moderate effectiveness for:

- a family-based treatments for individuals with anorexia nervosa who are under the age of 18
- b dialectical behaviour therapy for people with type 1 diabetes and an associated eating disorder
- c psychodynamically informed multi-family groups for adults with binge eating disorder
- d rebirthing therapies for teenagers with anorexia nervosa
- e single-session cognitive-behavioural therapy for bulimia nervosa in adults.

5 Which of the following statements about comorbidities in eating disorders is not true?

- a around 30% of patients with eating disorders meet criteria for at least one personality disorder
- b the most common cause of mortality in anorexia nervosa is starvation
- c prior obsessive-compulsive disorder increases the risk of subsequent bulimia nervosa
- d in individuals with comorbid type 1 diabetes and eating disorder, most diabetic complications are reversible
- e women attending infertility clinics have a high prevalence of lifetime eating disorder.