Chapter 21: Feeding and Eating Disorders

Cornelia Kaufmann

Academic Department of Psychiatry, Kolling Institute, Northern Clinical School, Faculty of Medicine and Health, The University of Sydney, Australia, and CADE Clinic and Mood-T, Royal North Shore Hospital, Northern Sydney Local Health District, Australia.

Introduction

Feeding and eating disorders are highly distinctive and serious mental disorders characterised by persistent disturbance of eating behaviours which is responsible for impairment of physical and mental health and psychosocial functioning. The following disorders are in this category: Pica, rumination disorder, avoidant/restrictive food intake disorder, anorexia nervosa, bulimia nervosa and, recently added in DSM-5, binge eating disorder. These disorders share some diagnostic criteria, whilst other criteria are mutually exclusive and only one diagnosis can be assigned. However, due to the fact that there are a number of common psychological underpinnings and shared behavioural features, patients afflicted by these disorders can "migrate" from one disorder to another. For instance, someone might initially develop anorexia nervosa in middle adolescence and transition to binge eating and/or bulimia nervosa when starvation becomes untenable and impossible to control.

Aetiology

Biological, psychological and socio-cultural factors can all contribute to the development of an eating disorder. Genetic factors influence risk, psychosocial and interpersonal factors can trigger onset and changes in the neural network can sustain the illness (Zipfel et al., 2015). There is evidence that exposure to media images that promote the thin-ideal, may contribute to the development of eating disorders by causing body dissatisfaction, negative moods, low self-esteem, and eating disorders symptoms among women (Hawkins et al., 2004). Strict dieting as a response to body dissatisfaction can in itself trigger the development of an eating disorder.

A study from the Anorexia Nervosa Genetics Initiative (ANGI) and the Eating Disorders Working Group of the Psychiatric Genomics Consortium (PGC-ED) conducted a genome-wide association study of 16,992 cases of anorexia nervosa and 55,525 controls, identifying eight significant loci. The study found the genes associated with anorexia nervosa overlapped with other psychiatric disorders such as obsessive-compulsive disorder, depression, anxiety, and schizophrenia (Watson et al., 2019).

Psychologically, a low self-esteem, perfectionism and an anxious temperament seem to predispose individuals to an eating disorder. Impulsivity and emotional dysregulation are more common in bulimia nervosa. Life events, trauma and disrupted attachment in early childhood increase vulnerability to the development of an eating disorder (Brewerton, 2007). Being in an environment which encourages a certain physique, for instance working as a fashion model, professional ballet dancing, gymnastics or athletics can contribute to the development of an eating disorder.

An eating disorder often is a maladaptive coping attempt to regain control over unwanted and distressing emotions or life-situations. The individual feels that achieving in the eating disorder shows increased self-efficacy, self-control and "success".

Epidemiology

Anorexia nervosa typically begins in mid adolescence and early adulthood. Lifetime prevalence in the general population is 1.9% for females (Wade et al., 2006) and around 0.1% in men (The National Eating Disorders Collaboration, 2010a). Bagaric et al. (2020) in their study found that the lifetime prevalence of bulimia nervosa was 1.21% (95% CI [0.87, 1.67]) for males and 2.59% (95% CI [2.07, 3.22]) for females. The lifetime prevalence for binge eating disorder was 0.74% (95% CI [0.49, 1.11]) and 1.85% (95% CI [1.42, 2.40]) for males and females, respectively, which is higher than reported in previous research.

Anorexia Nervosa (AN)

History

Reports of anorexia nervosa date back to the 1689 when it was described by Richard Morton, a British physician as a "nervous consumption". It was first described as a diagnostic entity named *anorexia nervosa* in the 19th century by Sir

William Gull, who was Queen Victoria's personal physician. Awareness of the condition was limited to the medical profession until Hilde Bruch, a German-American psychoanalyst, created wider awareness in her seminal work "The Golden Cage" in 1978 (Bruch, 2001). Since then, research into this disorder has advanced our knowledge of the genetic and biological processes and some of the psychological underpinnings.

Diagnostic Criteria

The diagnostic criteria for anorexia nervosa are a striving for weight loss that cannot be explained by another medical illness and an associated overvaluation of one's weight and shape. There is an intense fear about weight gain and often behaviours are present which interfere with treatment attempts to increase food intake and hence, weight. This can take the form of using excessive amounts of laxatives, diet pills or diuretics as well as excessive exercise or self-induced vomiting. Individuals often do not acknowledge how unwell they are and experience their illness as ego-syntonic. It is often the relatives or partners of the individual who encourage engagement in treatment. The individual needs to have significant low weight which is defined as weight that is less than minimally normal, or for children and adolescents less than the minimally expected BMI percentile (Mild: BMI >17 kg/m^2; Mod: BMI> 16-16.99kg/m^2; Severe: BMI 15-15.99 kg/m^2; Extreme: BMI < 15kg/m^2).

Two forms of anorexia nervosa have been established:

- *Restrictive subtype*: weight loss is accomplished mainly through fasting, dieting and/or excessive exercise. No binge eating or purging occurs.
- *Binge-eating/purging subtype*: recurrent episodes of binge-eating and purging occur.

Severe and enduring anorexia nervosa (SEAN) is a group of individuals with a prolonged course of anorexia and several treatment failures. They generally present with significant physical and psychiatric sequelae as well as compromised social, interpersonal and occupational functioning.

Bulimia Nervosa (BN)

History

In contrast to anorexia nervosa - which appears to have been noted throughout history - bulimia nervosa appears to be a more modern development. It was first described in 1979 by British psychiatrist, Gerald Russell, who published his

seminal paper, "*bulimia nervosa: an ominous variant of anorexia nervosa*", a case series of 30 patients who reported self-induced vomiting as an attempt to mitigate the effects of episodes of overeating. It was determined that these represented a syndrome that was distinct from anorexia nervosa but shared the same fear of weight gain.

Diagnostic Criteria

DSM-5-TR diagnostic criteria are recurrent episodes of binge eating with inappropriate compensatory behaviours in order to mitigate the caloric effect of binge eating (American Psychiatric Association, 2022). These behaviours can take the form of self-induced vomiting, laxative abuse, using diuretics or diet pills and engaging in prolonged fasting or excessive exercise. Binge eating episodes are discrete episodes of eating a much larger amount of food than what most individuals would eat in the same time (about 2-3x normal meal sizes). These episodes have to occur at least once per week.

Binge Eating Disorder (BED)

History

Binge eating disorder was first described by psychiatrist Albert Stunkard, who coined the term "Night Eating Syndrome" (Stunkard et al., 1955). He later specified that binge eating could occur without the nocturnal component (Stunkard, 1959). In DSM-IV, Binge eating disorder was listed under the category "Eating Disorders Not Otherwise Specified" (EDNOS), but now in DSM-5-TR it has its own place as an eating disorder diagnosis (American Psychiatric Association, 2022).

Diagnostic Criteria

Individuals with this disorder will eat a larger than normal amount of food with a sense of loss of control. They will eat much more rapid than normal and until feeling uncomfortably full, despite not always feeling hungry during those episodes. These episodes are followed by feelings of shame, disgust and embarrassment. Importantly, there are no compensatory behaviours following the binge eating episodes in the form of food restriction, excessive exercise, self-induced vomiting, taking of laxatives, diet pills or diuretics.

Other Specified Feeding or Eating Disorders (OSFED)

In this category, any Feeding and Eating disorders are grouped when they do not meet full criteria for any of the above disorders, but still cause significant psychological, social, occupational distress.

- Atypical anorexia nervosa - criteria for AN are met, except that despite significant weight loss, the individual's weight is within or above the normal range.
- Bulimia nervosa (of low frequency and/or limited duration) - inappropriate compensatory behaviours occur less than once per week.
- Binge eating disorder (of low frequency and/or limited duration) - binge eating occurs less than once per week.
- Purging disorder - recurrent purging behaviours without binge eating.

Assessment and Diagnosis

A full psychiatric assessment needs to be undertaken, as individuals with eating disorders experience high rates of other mental disorders, with reports of up to 97% having a comorbid condition. The most common of these are depression, anxiety disorders, followed by substance abuse and personality disorders (58%) (The National Eating Disorders Collaboration, 2010b; The National Eating Disorders Collaboration, 2012). Of note is that anxiety, depression, OCD and cognitive impairment can all represent the reversible effects of starvation. Additional to a full psychiatric assessment, it is important to ask questions specific to eating disorders:

- Food intake ("*what do you eat on a typical day?*")
- Compensatory mechanisms: exercise, self-induced vomiting, laxatives, diet pills or diuretics.
- Body image ("*what do you see when you look into the mirror*" and *"does your weight influence how you see yourself?"*) This can reveal significant disturbances.
- It is advisable to ask about the highest and lowest weight during the course of the illness and prior to its onset. *"How do you feel about gaining weight?"* can reveal in intense fear of weight gain.
- Medical complications specific to eating disorders: amenorrhoea, reduced bone density, bradycardia, dental issues, constipation, lack of energy and fatigue, feeling cold, dizziness and fainting.

Physical Examination

Additional to a comprehensive physical examination:

- Measure weight and height to calculate BMI
- Blood pressure (postural), HR, temperature
- Bloods: FBC/ EUC/ LFT's/ Ca/ Mg/ PO4/ Glucose/ TFT's/ Amylase/ Lipase/ Serum Osmolality/ lipids
- ECG- bradycardia, ST segment elevation, T wave flattening, more serious: arrhythmias, U waves, QTc -prolongation
- Consider a Bone Mineral Density scan every 12 months (adolescents) to 2 years (adults)

Look out for:

- Hypotension, postural hypotension, bradycardia, cardiac arrhythmias
- Cachexia
- Conjunctival pallor, skin pallor
- Brittle nails and hair
- Dry flaky skin, Carotodermia
- Bruising
- Lanugo (new development of fine hair on face and body)
- Hypothermia
- Dehydration
- Delayed capillary refill, acrocyanosis (peripheral cyanosis)
- Peripheral oedema
- Parotid hypertrophy, dental decay and Russell's sign (calluses on the dorsum of the fingers due to purging)
- Stress fractures

Medical Complications

All eating disorders have a high prevalence of concomitant medical complications. This applies particularly to anorexia nervosa (see Table 21.1) due to the often severely underweight status of these individuals (Mehler and Brown, 2015). All body systems are adversely affected by malnutrition. Most medical complications are reversible with weight restoration, or cessation of binge/purge behaviours but some are not, for instance osteoporosis is unlikely to be reversible after puberty.

Table 21.1 Medical complications of anorexia nervosa.

Organ System	Pathology	Possible Outcome
Cardiovascular	Bradycardia, Hypotension	Syncope
	Cardiac arrhythmias	Sudden death
	Mitral valve prolapse	Chest pain
	Takotsubo cardiomyopathy	Heart failure
Endocrine	Low levels of reproductive hormones	Amenorrhoea Osteoporosis and stress fractures, arrested growth
		Hypoglycaemia
	Loss of hepatic glycogen	Hypernatraemia
	Central diabetes insipidus	Hypothyroidism
	Hypercortisolaemia	
	Low T3 and T4 (TSH often normal)	
Renal	↓Na, ↓K, ↓ Cl, ↓ Mg, ↓ Ca, ↓ PO4,	Cardiac arrhythmias Refeeding syndrome
	Loss of HCL from purging	Metabolic hypokalaemic alkalosis
	Volume depletion (laxatives/ diuretics/ restriction)	Secondary hyperaldosteronism (Pseudo Barter syndrome)
Gastrointestinal	Gastroparesis	Constipation, atonic colon
	Parotid hypertrophy	Can be painful when purging is ceased
	Refeeding pancreatitis	Epigastric pain, nausea, vomiting
	Liver transaminitis	
	Increased HDL	Hepatic steatosis
	Loss of adipose tissue between spine and aorta	Hypercholesterolaemia Superior mesenteric artery syndrome– upper quadrant abdominal pain
Neurological	Loss of adipose tissue	Local compression neuropathies (peroneal nerve palsy),
	Loss of cerebral grey matter and white matter, reduced connectivity between limbic system and frontal lobe	Difficulties with set shifting, and evidence of cognitive rigidity, impaired emotional regulation, motivation and memory
	High levels of cortisol lack of glucose, fatty acids and thiamine	affecting memory and learning
Haematological	Pancytopenia due to fatty atrophy of bone marrow	anaemia, leukopenia and thrombocytopenia
Ophthalmologic	Orbital fat atrophy	Lagopthalmus

Anorexia Nervosa Medical Complications

Refeeding Syndrome

Reefeding syndrome is an extremely rare but serious and potentially fatal complication of refeeding. The cause is a rapid uptake of electrolytes from blood plasma into cells in response to the release of insulin to metabolise carbohydrates, causing a significant fluid shift. The consequence can be hypophosphataemia, hypomagnesaemia and hypokalaemia in the blood plasma, especially as stores of electrolytes tend to be low already. This can cause muscle weakness, cardiac arrhythmias, cardiac failure, seizures and confusion. Osmotic shifts of fluids into cells can cause hypertension and oedema. Peripheral oedema can occur, and concerningly so can pulmonary oedema. Most at risk are individuals who are severely underweight. A wide range of incidence has been cited (0-45%) due to a heterogeneity in population and definition of refeeding syndrome (Cioffi et al., 2021). Refeeding should be done with care and in consultation with physician colleagues if needed. Most difficulties occur in patients with conditions other than anorexia nervosa and when carbohydrate rich Total Parenteral Nutrition (TPN) was used (Madden et al., 2015).

Bulimia Nervosa Medical Complications

Treatment tends to be stepped and should ideally include a range of inpatient and outpatient options. Ideally there is a smooth transition between inpatient and outpatient settings, according to the patients need and level of recovery (Hay and Claudino, 2012). Relevant factors to consider regarding the level of care are medical (in)stability, level of cognition and insight, ability to cooperate with treatment and the individual's level of community supports. Table 21.2 displays a summary of medical complications of bulimia nervosa.

Table 21.2. Medical complications of bulimia nervosa.

Organ system	Pathology	Potential outcome
Metabolic	↓K, ↓Cl from self-induced vomiting	Metabolic alkalosis Cardiac arrhythmias, sudden death
	Dehydration	Volume depletion, Pseudo-Barter syndrome
	Loss of bicarbonates due to laxatives	Metabolic acidosis
Dental	Tooth enamel erosion	Teeth sensitivity, increased tooth decay
Gastrointestinal	Self-induced vomiting	Gastroesophageal reflux, Barrett's oesophagus Russel's sign (callus on the dorsal aspect of the hand)
	Mallory Weiss tear	Haematemesis
	Laxative use	Chronic constipation, atonic colon
Ophthalmologic	Increased intraocular pressure due to self-induced vomiting	Subconjunctival haemorrhage

Engagement

The first step in treatment is always engagement of the individual and their family, which can be difficult due to the ego-syntonic nature of the disorder – this means that, particularly for anorexia nervosa, the illness is perceived as congruent with the individuals' values and needs, and hence any change in the status quo is resisted. Ideally, self-determination and self-management are encouraged with the aim of conducting treatment in the least restrictive context. Psychoeducation and discussion about the medical implications of the disorder is an important part of this process.

Medical stabilisation is the next step to achieve a reversal of physical and cognitive effects of starvation. This means refeeding and cessation of compensatory behaviours. Inpatient treatment is preferred if the patient is medically unstable and/or BMI <14, an inpatient medical treatment is indicated.

Indicators of medical instability (in adults):

- HR < 40bpm or tachycardia on standing (>20bpm increase)
- blood pressure < 90/60 mmHg or with a postural drop of >20mmHg
- temperature <35.5^{0}C.
- Admission is also indicated if there has been rapid weight loss, several days of no oral intake at all and uncontrolled purging, exercise or suicidality.

Involuntary treatment is a last resort, but when done sensitively does not have to be the end of a therapeutic relationship.

Outpatient treatment is an option if the patient is medically stable and should ideally consist of a multidisciplinary team with involvement of the patient's GP, a psychiatrist, dietician and psychologist (if possible, all should have clinical experience with eating disorders). Day programs, if available can be a step-down from the inpatient setting.

Refeeding:

- Options include oral intake of meals, use of nutrition supplements (Ensure, Fortisip etc.) and nasogastric feeding
- Establishment of regular and appropriate meals and snacks sufficient to achieve nutritional rehabilitation is the first step. Involvement of a dietitian is desirable.
- Some components of refeeding will be considered non-negotiable (e.g. steady weight gain, being weighed, consuming a specified amount of nutrition and calories, not engaging in compensatory behaviours). Excessive negotiation about these basic components is best avoided.
- A combination of nasogastric feeding overnight and oral intake of food during the day is an option.
- Determining the adequate energy requirement for refeeding can be difficult due to metabolic changes occurring during refeeding, diversion of food (e.g. into pockets and serviettes) and secret compensatory behaviours (purging, exercise etc.). Recommendations regarding actual kcal/kg/day can vary and is individual. It is not advisable to discuss calories with the patient.
- The patient and family should be provided with a meal plan in order to optimise consistency in approach an minimise debates about alterations to food portion sizes or food types.

- Establishing increased social eating with family members and friends will help to normalise eating.
- Minimise eating disordered behaviours at mealtimes (e.g. no playing with food, not taking very small bites or cutting food into tiny pieces, sitting at the table for a specified time)
- Post meal support can help with anxiety and agitation and can involve socialising, relaxation or distraction.
- In order to prevent purging behaviours, access to the bathroom might be restricted for one hour after meals.

Psychological therapies with evidence for eating disorders:

- Maudsley family-based treatment (for children and adolescents) is a family-based therapy with three definitive phases: Phase 1 consists of weight restoration with parents taking control of all meals. Phase 2 works on returning control overeating to the adolescent and the goal of phase 3 is establishing a healthy adolescent identity.
- Cognitive Behaviour Therapy for Eating Disorders (CBT-Enhanced) – challenges eating disorder related behaviours and patterns of thinking (Fairburn et al., 2015)
- The Maudsley model of anorexia nervosa treatment for adults (MANTRA) – a combination of motivational interviewing, cognitive remediation and flexible involvement of carers.
- Focused psychodynamic therapy for anorexia nervosa (Zipfel et al., 2014)
- Specialist supportive clinical management (SSCM) with a primary focus on resumption of normal eating and restoration of weight, but also addresses life issues impacting on the eating disorder.

Pharmacotherapy in eating disorders:

- Evidence for medication treatment in eating disorders is weak to moderate
- There is support for the use of high dose fluoxetine in bulimia nervosa and binge eating disorder (Hay, 2012)
- Lisdexamphetamine also has shown efficacy in binge eating disorder (Fornaro et al., 2016)
- Low dose antipsychotic medication may be useful in anorexia nervosa especially in the context of high anxiety and obsessive eating disorder related ruminations (Hay and Claudino, 2012).

Prognosis

In anorexia nervosa, there is an almost 18-fold increase in mortality including a high suicide rate. Among the surviving patients, less than one-half recovered on average, whereas one-third improved, and 20% remained chronically ill. The normalization of the core symptoms, involving weight, menstruation, and eating behaviours, was slightly better when each symptom was analysed in isolation. The presence of other psychiatric disorders at follow-up was very common. Longer duration of follow-up and, less strongly, younger age at onset of illness were associated with better outcome. There was no convincing evidence that the outcome of anorexia nervosa has improved in the second half of the last century (Hans-Christoph, 2002).

Most people with bulimia nervosa, binge eating disorder or other specified eating and feeding disorders (OSFED) have a relatively good prognosis with 50% or more free of symptoms after five years of treatment (Steinhausen and Weber, 2009).

Avoidant/Restrictive Food Intake Disorder (ARFID)

This disorder has some similar criteria to anorexia nervosa, however, here the predominant feature is a concern about the sensory qualities about food (texture, smell, colour, taste or temperature of food) or worry about a potential aversive consequence of eating certain foods (choking, vomiting, nausea, gastrointestinal discomfort, etc). This leads to a failure to meet adequate nutritional and/or energy needs and causes significant weight loss. However, in contrast to anorexia nervosa, there is no concern about weight or body shape. Some individuals become dependent on nutritional supplements or even nasogastric feeding. ARFID is more common in children than any other age group. Anxiety disorders, autism spectrum disorder, obsessive-compulsive disorder and attention-deficit/hyperactivity disorder may increase the risk for developing ARFID.

Pica

Pica is the persistent eating of non-nutritive and non-food substances (for instance soil) which is inappropriate for the individual's developmental level and is not part of a socially and cultural normative practice. Pica often occurs in the context of another mental disorder like autism spectrum disorder or intellectual developmental disorder, but can also occur in medical conditions, including pregnancy. Onset is most often in childhood, but can occur throughout the ages. Neglect and lack of supervision can increase the risk for this condition.

Rumination Disorder

Repeated regurgitation of food after eating, which then may be re-chewed and re-swallowed or spat out. This occurs in the absence of a medical disorder affecting the gastrointestinal system and the behaviour occurs without any apparent disgust, involuntary retching or voluntary purging behaviours. It cannot occur together with a diagnosis of another feeding and eating disorder. Rumination disorder is more common in individuals with intellectual disability and can occur at any age. Predisposing factors can be lack of stimulation, neglect, stressful life events and difficulties in the parent-child relationship.

Further Reading

Mehler, P. S., Anderson, K., Bauschka, M., Cost, J., & Farooq, A. (2023). Emergency room presentations of people with anorexia nervosa. *Journal of Eating Disorders*, *11*(1), 16.

Rowe, E. (2017). Early detection of eating disorders in general practice. *Australian Family Physician*, *46*(11), 833-838.

Cite as:

Kaufmann, C. (2024). Eating and feeding disorders. In Boyce, P., Harris, A., and Malhi, G.S. (Eds.), *The Sydney textbook of psychiatry* (pp. 272–285). The University of Sydney.

References

American Psychiatric Association (2022) *Diagnostic and statistical manual of mental disorders : DSM-5-TR*. Washington, DC: American Psychiatric Association Publishing.

Bagaric M, Touyz S, Heriseanu A, et al. (2020) Are bulimia nervosa and binge eating disorder increasing? Results of a population-based study of lifetime prevalence and lifetime prevalence by age in South Australia. *European Eating Disorders Review* 28(3): 260-268.

Brewerton TD (2007) Eating disorders, trauma, and comorbidity: Focus on PTSD. *Eating disorders* 15(4): 285-304.

Bruch H (2001) *The golden cage: The enigma of anorexia nervosa*. Harvard University Press.

Cioffi I, Ponzo V, Pellegrini M, et al. (2021) The incidence of the refeeding syndrome. A systematic review and meta-analyses of literature. *Clinical Nutrition* 40(6): 3688-3701.

Fairburn CG, Bailey-Straebler S, Basden S, et al. (2015) A transdiagnostic comparison of enhanced cognitive behaviour therapy (CBT-E) and interpersonal psychotherapy in the treatment of eating disorders. *Behaviour research and therapy* 70: 64-71.

Fornaro M, Solmi M, Perna G, et al. (2016) Lisdexamfetamine in the treatment of moderate-to-severe binge eating disorder in adults: systematic review and exploratory meta-analysis of publicly available placebo-controlled, randomized clinical trials. *Neuropsychiatric Disease and Treatment*. 1827-1836.

Hans-Christoph S (2002) The outcome of anorexia nervosa in the 20th century. *American Journal of Psychiatry* 159(8): 1284-1293.

Hawkins N, Richards PS, Granley HM, et al. (2004) The impact of exposure to the thin-ideal media image on women. *Eating disorders* 12(1): 35-50.

Hay PJ and Claudino AM (2012) Clinical psychopharmacology of eating disorders: a research update. *International Journal of Neuropsychopharmacology* 15(2): 209-222.

Madden S, Miskovic-Wheatley J, Clarke S, et al. (2015) Outcomes of a rapid refeeding protocol in adolescent anorexia nervosa. *Journal of eating disorders* 3(1): 1-8.

Mehler PS and Brown C (2015) Anorexia nervosa–medical complications. *Journal of eating disorders* 3: 1-8.

Steinhausen H-C and Weber S (2009) The outcome of bulimia nervosa: findings from one-quarter century of research. *American Journal of Psychiatry* 166(12): 1331-1341.

Stunkard AJ (1959) Eating patterns and obesity. *Psychiatric quarterly* 33(2): 284-295.

Stunkard AJ, Grace WJ and Wolff HG (1955) The night-eating syndrome. A pattern of food intake among certain obese patients. *American Journal of Medicine* 19: 78-86.

The National Eating Disorders Collaboration (2010a) Eating disorders prevention, treatment & management: An evidence review. Sydney: NEDC.

The National Eating Disorders Collaboration (2010b) Eating disorders: The way forward: An Australian national framework. Sydney: NEDC.

The National Eating Disorders Collaboration (2012) An integrated response to complexity – National eating disorders framework 2012. Sydney: NEDC.

Wade TD, Bergin JL, Tiggemann M, et al. (2006) Prevalence and long-term course of lifetime eating disorders in an adult Australian twin cohort. *Australian & New Zealand Journal of Psychiatry* 40(2): 121-128.

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(8): 1207-1214.

Zipfel S, Giel KE, Bulik CM, et al. (2015) Anorexia nervosa: aetiology, assessment, and treatment. *The Lancet Psychiatry* 2(12): 1099-1111.

Zipfel S, Wild B, Groß G, et al. (2014) Focal psychodynamic therapy, cognitive behaviour therapy, and optimised treatment as usual in outpatients with anorexia nervosa (ANTOP study): randomised controlled trial. *The Lancet* 383(9912): 127-137.