

## **CHAPTER 14 EATING DISORDERS**

**“You can never be too rich or too thin.”**

Duchess of Windsor (1896-1986)

**“Specific characteristics of eating disorders pathology did not change across time”**

Probst et al, 2018

Before plunging into the eating disorders – let’s take a broader view.

### **Asceticism**

Asceticism is a lifestyle in which the individual refuses worldly pleasures. The ascetic believes their chosen lifestyle is virtuous; their aim is usually to achieve greater spirituality. Asceticism has been practiced in all religions and by some non-religious individuals [who claim the practice increases the ability to think clearly and avoid destructive short-term impulses].

**St Catherine of Siena** (1347-80) was an ascetic. Her story may be the first recorded case of anorexia nervosa (Bell, 1985; Reda & Sacco, 2001). St Catherine was an influential figure, convincing Pope Gregory XI to restore the papacy from Avignon (Fr.) to Rome. She was later in dialogue with Pope Urban VI.



Illustration. St Catherine of Siena. Possibly the first described case of anorexia nervosa.

At 16 years of age St Catherine was admitted to the Third Order of St. Dominic. For three years she lived on a spoonful of herbs per day and slept only two hours per night. When her mother insisted that she eat, St Catherine began to throw meat under the table. She lost half her weight. When the local priest, Don Tommaso of Fonte persuaded her “in the name of God” to eat at least one once per day, she began to vomit. She was in the habit of prodding her throat with a stick of finnochio or a goose feather – causing vomiting. St Catherine had been in conflict with her family over a proposed marriage, she was perfectionistic and was never satisfied with the results she achieved. She died at 33 years of age.

**St Wilgefortis** lived some centuries earlier. The case for her being the first recorded case of anorexia nervosa is less strong (Lacy, 1982). St Wilgefortis lived some time between 700 and 1000 AD. She was the daughter of the King of Portugal. She had decided to enter the church. Her father arranged for her to marry the King of Sicily. To avoid the marriage St Wilgefortis prayed to God to be made unattractive. She became ascetic and ate little. She developed body hair and grew a beard. The King of Sicily withdrew his suit. Her father was so outraged he had her crucified. Lanugo, (fine baby-like hair) is frequently observed on the face and limbs of patients with anorexia nervosa – but, not a beard.



Illustration. The statue of St Wilgefortis in Henry VIII's Chapel, Westminster Abbey, depicts her with a beard flowing down to her chest. In the above Polish depiction, she is beardless, but very thin.

## Introduction to eating disorders

In the chapter “Feeding and Eating Disorders” the **DSM-5** describes, 1) Anorexia nervosa, 2) Bulimia nervosa, and 3) Binge-eating disorder, along with some minor curiosities.

Anorexia nervosa was first described as a medical condition in English by Sir William Gull in 1874. He drew attention to the diagnostic triad of 1) fasting, 2) amenorrhea and 3) hyperactivity. He described the disorder as “wasting without lassitude”.

The diagnosis of bulimia nervosa first appeared 30 years ago (Russell, 1979) as a variant of anorexia nervosa, in which there is dietary restriction, episodes of overeating, vomiting or laxative use, and the maintenance of about normal weight.

Binge-eating disorder appears for the first time as a recognized entity - recurrent episodes of bingeing in the absence of dietary restriction or other compensatory behaviours (except, in some cases, vomiting). It has been reported in 10-15% of female college students (Halmi et al, 1981).

## ANOREXIA NERVOSA (AN)

### DSM-5 Diagnostic criteria

- A. Restriction of energy intake relative to requirements, leading to significantly low body weight in the context of age, sex, developmental trajectory, and physical health.
- B. Intense fear of gaining weight or becoming fat, or persistent behaviour that interferes with weight gain, 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 persistent lack of recognition of the seriousness of the current low body weight.

AN is the most homogenous of all psychiatric disorders. AN can occur at any age, but with a peak in the late teen years. The earlier the onset the better the prognosis, with adult onset having a relatively poor outcome. There is a stereotypic presentation and course of illness - there is resistance to eating, powerful pursuit of weight loss, but paradoxically, there is preoccupation with food and eating rituals. There is distorted body image, denial of being underweight, a practice of energetic exercise, a lack of insight and resistance to treatment.

The **prevalence** of AN depends on the assessment tools and the population surveyed. One American authority (Hudson et al, 2007) gives a lifetime prevalence of 1% in women and <0.5% in men. The American Dietetic Association gives a slightly lower prevalence of 0.9% for women and 0.3% for men. Studies of western college-age women find: US, 1%; Italy, 1.3%, and Norway 2.6%. There is some suggestion of a western culture bound syndrome, as lower prevalence has been reported in China (0.01%) and Japan (0.025%) (Goyal 2012). However, a recent opinion suggests that AN is present in China, but that Western models do not accurately identify them easily (Getz, 2014). It has been studied in Han Chinese (Cheng et al, 2015). Iran is between east and west, with a prevalence of 0.8% (Garrusi and Baneshi, 2012).

In ballet and modelling schools the prevalence is about 7% (Garner & Garfinkel, 1980).

AN is about 10 times more common in females. The most common age of onset is 14-18 years, but has been reported in girls as young as 8 years. It is believed to be more common in the higher socio-economic groups. However, this has not been clearly demonstrated in epidemiological surveys. It is believed the incidence has increased over the last half century (Bulik et al, 2006; Hoek, 2006).

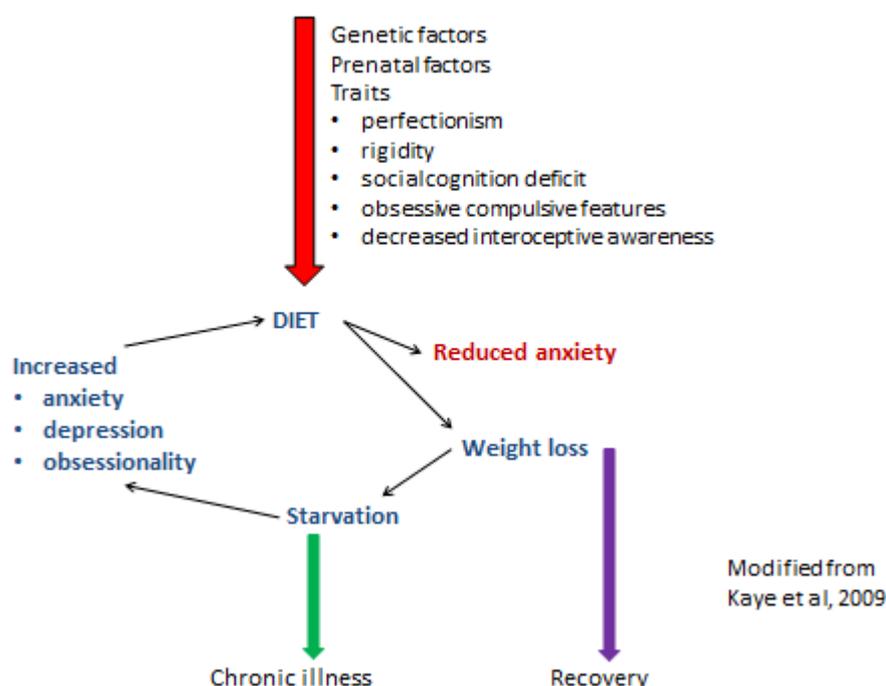


Illustration. Dieting reduces anxiety in the short term, but begins a destructive cycle of increasing anxiety, depression and obsessionality, and further dieting.

### Aetiology

Unclear. Evidence suggests a genetic predisposition, and important contributions from culture, early life experience (including neglect and abuse), personality type, the neurological and hormonal changes associated maturation, and the stressors of adolescence (including increasing autonomy).

The “hunger hormones” may play a role – but details are unclear. Leptin – an appetite suppressing hormone, is secreted by fat cells. Ghrelin – an appetite increasing hormone, is release by the stomach.

**Genetic studies.** The lifetime risk of AN is 7-12 times greater for individuals with a first degree relative with the disorder. Twin studies support high heritability (46-70%, Kendler et al, 1991; 33-84%, Bulik & Tozzi, 2004). Recent twin studies found heritability due to genetics 56%, shared environment 5% and unique environment 38% (Bulik et al, 2006).

Nevertheless, significant genome-wide findings are yet to be reported (Hinney & Volckmar, 2013).

The gene-environment interaction model views strict dieting as sufficient “insult” for expression of the phenotype (Bulik, 2005). Epigenetics is expected to become an area of active research – but little progress has been reported to this time.

Genetic factors could possibly be “switched on” by the hormonal changes and the particular stressors of puberty (Herpertz-Dahlman et al, 2011).

**Pregnancy and perinatal complications**, especially preterm birth, are risk factors.

The evidence for **childhood sexual abuse** as an aetiological factor in AN is lacking (Caslini et al, 2016).

**Adverse life events** may precipitate AN – but evidence is lacking (Thornton et al, 2017).

**Personality disorder** is found in at least 70% of those with AN; obsessive-compulsive personality disorder is the most common.

**Personality traits** are characteristic, and may represent risk factors, or an underpinning pathological process. Herpertz-Dahlmann et al (2011) describe patients as “usually good and successful”, however, often with “some peculiarities”. They describe rigidity and perfectionism, depression, anxiety. Interpersonal function may be reduced with non-assertive, submissive interpersonal style, poor theory of mind, and autistic traits, while Ahren et al (2011) found that these patients characteristically evaluate themselves by comparison with others. Many have described low self-esteem.

Patients with AN also appear to demonstrate diminished “interoceptive awareness”. Interoception includes a range of sensations beyond taste, including the perception of pain, temperature, itch, tickle, sensual touch, muscle tension, air hunger, stomach pH and intestinal tension. Integration of these provides a sense of the entire body and the self – provides a link between cognitive and affective processes and the current body state.

Altered interoceptive awareness might be a precipitating and reinforcing factor in AN (Khalsa et al, 2015).

Alexithymia is the term applied to an impaired ability to identify and communicate emotions. A recent review indicated that when alexithymia is present in AN, the prognosis is less favourable (Pinna et al, 2015).

Many such personality features improve with remission of the condition. However, tendency to negative emotional states, harm avoidance, perfectionism, desire for thinness and mild dietary preoccupation persist – suggesting these are underlying traits which contribute to the pathogenesis of AN (Kaye et al, 2009). At the same time, it must be remembered that such features are exaggerated by starvation (Keys et al, 1950).

**Anxiety and dysphoric mood.** Individuals with AN experience anxiety - dietary restraint reduces this unpleasant state, and eating stimulates dysphoric mood (Kaye et al, 2009). Dieting reduces feeling of worthlessness (Herpertz-Dahlmann et al, 2011).

**Socio-cultural factors** have shaped “the modern cult of thinness” in Western societies. It is believed individuals respond by dieting, and that genetically vulnerable individual’s progress through dieting to AN (Bulik, 2005).

**Socio-economic status.** Earlier reports found the prevalence of AN was higher in higher socio-economic schools, but the evidence is now equivocal.

### **Maintaining factors**

Mentioned under etiology, the relief of anxiety by dieting and dysphoric mood caused by eating, may also serve as a maintaining factor.

Starvation is another maintaining factor, inducing complex physiological and psychological reactions involving central and peripheral mechanisms. Such mechanism may have had evolutionary value, allowing animals to survive periods of food shortage, but in the current setting they serve only to perpetuate a vicious cycle of weight loss.

### **Neuropsychological testing**

Neuropsychological testing reveals **cognitive deficits** (Weider et al, 2013) which are related to severity of the disorder and may play a role in cause and outcome (Zakzanis et al, 2009). Executive control is impaired with problems in set-shifting, attention and decision-making (Treasure & Russell, 2011). A link has been demonstrated between amenorrhoea, brain structure and deficits in cognition, (Chui et al, 2008). Progress has been disappointingly slow (Jauregui-Lobera, 2014).

### **Neuroimaging**

Neuroimaging studies demonstrate structural and functional abnormalities. More work is needed. For a review see Phillipou et al (2013).

There is global reduction of grey (GM) and white matter (WM) during the acute stage. GM is reduced by 5-20%, and WM is reduced to a lesser extent.

GM loss is found in the anterior cingulate, hippocampi and the temporal, parietal and prefrontal regions. With recovery GM is restored, but most studies find small residual deficits remain. In one study, there was 60% restoration after 15 weeks of successful treatment.

Loss of GM volume is probably due to reduction in the size of neuronal and glial cell bodies, and reduction in the density of dendrites and synapses – as restoration of volume occurs with remission.

A recent study (Kaufmann et al, 2017) has discounted earlier studies which were thought to demonstrate reduced fractional anisotropy of the fornix in people with AN.

Magnetic resonance spectroscopy (MRS), which gives information on nerve cell damage by assessing brain metabolites, indicates altered cell membrane turnover which is reversible with recovery.

Functional magnetic resonance imaging (fMRI) using visual stimuli of food or body image has been reviewed (Garcia-Garcia et al, 2013). Differences between those with eating disorders and healthy controls located differences in two circuits, 1) limbic and paralimbic areas (associated with reward), and 2) prefrontal regions associated with cognitive functions and control.

The insula may be of particular importance (Kaye et al, 2009), as it integrates interoceptive information – confirmation is awaited.

The hippocampal volume of women with AN who are food restricting and exercising is larger than that of normal controls. Interestingly hippocampal volume of healthy individuals who engage in food restriction and exercise is also enlarged and is considered to have a protective function (Beadle et al, 2014).

### **Neurotransmitters and cells**

The pathogenic involvement of the serotonergic system in eating disorders is an established finding (Sigurth et al, 2013).

Dopamine (DA) dysfunction, particularly in striatal circuits, may contribute to altered reward centre responses (Kontis and Theochari et al, 2012).

### **The clinical picture**

The clinical picture is embodied in the **DSM-5** diagnostic criteria listed above.

The patient is usually a teenage female, brought in by her parents. There has been weight loss, cessation of the menses, fine hair growth on the face and limbs, refusal to eat in the manner expected for her age and family circumstances, particular avoidance of carbohydrate and fatty foods, frequent weighing, often vomiting and excessive laxative use, insomnia, irritability, sensitivity to cold, and withdrawal from friends.

The hands and feet are cold, the skin is dry, the pulse is slow (50-60/min) and the blood pressure is low (e.g., 90/60). There may be calluses on the dorsum of the second and third digits through frequent contact with the front teeth and erosion of tooth enamel due to regurgitated gastric acid. There may be disorder of hormones, including cortisol, gonadal and thyroid hormones.

History taking and subsequent management may be difficult. 'Denial of the illness, lies, cheating, manipulation, are characteristic of the behaviour of anorexics' (Russell, 2000).

The patient frequently believes that overweight is indicative of greed and deserving of social ostracism. She frequently maintains that she is overweight, in spite of evidence

to the contrary. There is a fear of gaining weight. Traditionally, terms such as 'distorted body image', 'over-valued ideas' and 'irrational beliefs' have been used, although similarities to delusional thinking have been noted. [Steinglass et al, (2007) found that 20% of 25 AN patients were delusional, and suggested a sub-group of AN patients whose concerns reach delusional proportions may be more refractory to treatment. This view may not be widely accepted.]

Depression is present in over 40%, and obsessive-compulsive disorder in over 20% of cases.

Gonadotrophins and oestrogens are low or undetectable. Pelvic ultrasound reveals reduced ovarian size. The 24 hour urinary cortisol is elevated and plasma tri-iodothyroxine is low (Krassas, 2003).

The complications of starvation include fluid and electrolyte disturbance, peripheral oedema, hypoglycaemia, myopathy, osteoporosis (Kandemir et al, 2018) and thrombocytopenic purpura. A recent account (Sheu et al, 2007) describes nutritional deficiency affecting both respiratory muscle strength and lung parenchyma, leading to emphysema. Death from medical causes may result from starvation or purging-related arrhythmias.

### **Outcome**

The mean mortality rate is 5% in adults and 2% in adolescents (Steinhausen et al, 2002).

Full recovery includes return to appropriate weight and continued growth and development, restoration of menstruation, and normal eating behaviour and attitude with regard to food and body shape. A Swedish study (Theander, 1985) followed patients up over 33 years, 18% had died. 29% recovered in less than 3 years, another 35% recovered by 6 years. Recovery after 12 years was rare. An Australian study (Wade et al, 2006) followed patients after 15 years and found that while 75% had a good outcome, less than 50% were asymptomatic.

At follow up, 20% are unable to support themselves independently (Treasure & Russell, 2011).

40% of AN death is by suicide (Papadopoulos et al, 2009). AN has the highest suicide rate of all mental disorders. However, people with AN do not have a higher attempted suicide rate than people with other eating disorders or the general public - they select more lethal means (suggesting a stronger desire to die; Guillaume et al, 2011).

Those who recover may do well in life – they have the ability to plan ahead, control their impulses and avoid harm.

## Treatment

As with all eating disorders, a collaborative approach and a multidisciplinary team is essential in the management of AN. There is a need for psycho-education and the building of a strong therapeutic relationship.

Early treatment is important. Outpatient treatment is the preferred approach, with admission to a psychiatric or special eating disorders unit reserved for cases which fail to progress. Admission to a medical ward is essential when there is risk to life.

**Family therapy** is often the first approach, especially for patients less than 19 years of age (Hay et al, 2014). It is effective in helping to correct miscommunication and misunderstandings within the family, and restore healthy eating habits.

**Individual psychotherapy** is usually offered, the type being influenced by the theoretical inclinations of the treating centres/clinicians (Hay et al, 2003). The patient remains psychologically inaccessible as long as starvation continues, thus, productive psychotherapy may not begin until there is at least some weight restoration.

Cognitive behaviour therapy (CBT) aims to restructure cognition, but has limited success in AN (American Dietetic Association, 2011). An unexpected finding (McIntosh et al, 2005) is that non-specific supportive clinical management was superior to specialized psychotherapy.

Evidence indicates that people with AN have reduced emotional awareness of others (Theory of Mind is the topic of Chapter 33). Psychotherapies that focus on emotional communication and the maternal bond have been recommended (Rommel et al (2013).

**Inpatient treatment** restores weight most rapidly (usually within three months). There may be difficulty in persuading the patient to remain in an inpatient program. Benefits include the omnipresence of skilled nursing staff who are able to provide psychotherapy and supervision. The patient is encouraged to take nutritious meals at regular times. It may be necessary to commence with smaller meals. Once eating is re-established the aim is to gain 200-300 grams per day.

Some experts find that initial in-patient treatment has no advantages over out-patient treatment, and that out-patient failures do poorly on transferred to in-patient facilities (Gowers et al, 2007). It seems where the patient is best managed depends on local factors.

**Drug treatment.** As mentioned, there is no FDA approved drug for the treatment of AN. In a recent review, Hay et al (2014) cautioned against using anxiolytics or antidepressants to relieve symptoms.

Antidepressants are indicated for the treatment of major depression, but they are ineffective in the presence of malnutrition.

**Compulsory treatment and naso-gastric feeding** depend on the local mental health legislation. This is generally avoided as it disrupts the trusting patient-clinician relationship and acceptance by the patient of responsibility for her/his actions. However, it becomes necessary should malnutrition pose an immediate threat to life.

## **BULIMIA NERVOSA (BN)**

### **DSM-5 diagnostic criteria**

- 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 an amount of food that is definitely larger than most people would eat during the same time and in the same circumstances
  2. a sense of lack of control over eating during the episode.
- B. Recurrent inappropriate compensatory behaviour 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 behaviour 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 Anorexia nervosa.

“Bulimia” is derived from the Greek words, *bous*, for ox, and *limos*, for hunger – indicating the *appetite of an ox*.

BN was first described by Russell (1979), and first appeared in the DSM (III) in 1980. The diagnostic features include binge eating followed by abnormal behaviour to avoid weight gain, including vomiting, purging and use of diuretics, fasting and excessive exercise. There are many similarities with AN, such as sustained periods of fasting. The main diagnostic difference is that with BN the weight is maintained about normal.

Binge eating (up to 10 000 calories; usually processed carbohydrates and fatty foods) may occur following prolonged fasts or in response to adverse emotional states, including low mood or feelings of rejection. Evidence also indicates that exposure to certain foods can trigger binges (Staiger et al, 2000), suggesting a similarity with drug use behaviour. Patients frequently describe a sense of pleasure during binging, which may indicate an anxiolytic effect. Concurrently, there may be a sense of loss of control, which is unpleasant. Purging (used here to include vomiting) occurs immediately, and may be associated with a sense of relief (erroneous) at having avoided weight gain. Purging is soon followed by a sense of self-disgust, frustration and regret.

BN is more common than AN, with a life-time **prevalence** in Western regions of 0.5% for males and 1.5% for females. Ten times more females than males present for treatment. BN typically develops in late adolescence and early adulthood. It is much more common in countries “where palatable food is plentiful yet thinness is esteemed” (Klein & Walsh, 2003).

## Aetiology

As with AN, **socio-cultural factors** are important. In the west, thinness in women has been prized for most of the last century. The “Flappers” of the 1920s are a prime example, and the female “mannequins” and “models” of all eras have been thin. That is not to deny the desirability of the voluptuous “pin-up girls” of the 1950, and the well rounded form of the sensuous type throughout history. Many of those with eating disorders have difficulty with sexuality and this may inspire some to seek the aesthetic form of the mannequin rather than the voluptuous form of the sex idol.

It is not only the female whom Western culture encourages to have a particular shape. In recent decades the ideal male depicted in underpants advertisements is shown to have abdominal muscles which are just as unattainable to the average male as the stick-figure is to the average female.

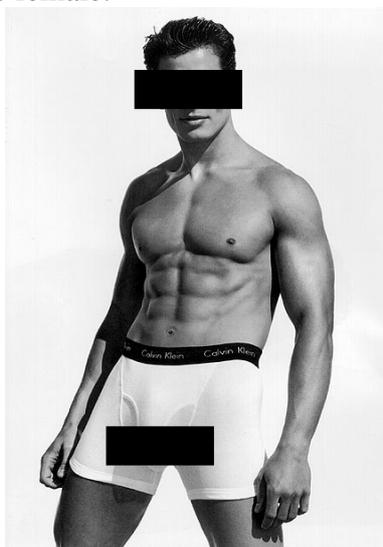


Illustration. Western culture encourages the female to aspire to thinness and the male to be thin but muscular. Both are almost unattainable by people with day-jobs. Our findings indicate that childhood adversities appear to be associated with an increased risk of BN and in particular EDNOS, whereas they seem to be either unassociated or associated with a decreased risk of AN.

**Childhood sexual abuse** is more common in people with BN than normal control populations. Interestingly, CSA is not a feature in AN (Larsen et al, 2017). Of course, CSA is not specific for BN, and is found in other psychiatric disorders. The majority of people with BN have not been exposed to this trauma.

**Personality disorder** is common in BN, particularly borderline personality disorder. In the absence of frank personality disorder, the temperament frequently features elevated novelty seeking and impulsivity. There is a high prevalence of drug and alcohol abuse, self-injurious behaviour such as cutting, and suicide attempts (Pearson, et al, 2017).

## Genetics

Studies in twins have estimated the heritability of BN as 28-83% (Bulik et al, 2000) – however no genetic risk factors have been conclusively identified. There is familial aggregation of AN and BN which suggests a shared vulnerability.

## **Neuroimaging**

Neuroimaging shows similar changes to those of AN: reduced global reduction of GM and WM, and 5HT-2A receptor binding abnormalities (Frank et al, 2004; Goethals et al, 2006).

An fMRI study using a food stimulus demonstrated reduced activation of the lateral prefrontal cortex and the anterior cingulate (Joos et al, 2011). (BN is a dysregulation syndrome and the lateral prefrontal cortex is a region of impulse control. BN is often associated with anxiety and distress, and the anterior cingulate plays a role in mood regulation.)

A more recent fMRI study demonstrated diminished frontostriatal brain activation in patients with bulimia nervosa contributing to the severity of binge eating symptoms (Skunde et al, 2016).

A diffusion tensor imaging (DTI) study has demonstrated white matter dysfunction of the corona radiata in bulimia nervosa (Mettler et al, 2013).

## **The clinical picture**

The clinical picture can be extrapolated from the diagnostic criteria. In contrast to the patient with AN, the patient with BN usually self-presents seeking help. Weight loss is not a prominent feature. Thinness and physical appearance are of great importance to her/him, and self-esteem is judged by this cultural yard-stick. There may be calluses on the dorsum of the second and third digits, erosion of dental enamel, and hypertrophy of the parotid glands. There is rarely disturbance of body chemistry. Arrhythmias occasionally occur secondary to electrolyte disturbances. Menstrual abnormalities are not uncommon, even when the body weight is normal.

Depression and low self-esteem are common. Anxiety disorders and obsessive-compulsive disorder are not uncommon.

**Intellectual function** (WAIS-III) may be reduced. Weider et al (2013) placed a sample with BN below healthy controls, but above a sample with AN.

## **Outcome**

50% or more are free of symptoms at 5 years (Steinhausen and Wever, 2009).

## **Treatment**

**Psychotherapy** is the first line therapy. CBT is used to restructure maladaptive thoughts which underpin the maladaptive behaviour (Slade et al, 2018). Dialectical behaviour therapy (DBT) may also have a place. Acceptance and Commitment Therapy (ACT) may have a place (Solomon, 2013).

**Antidepressants** are the second line of therapy. The selective serotonin reuptake inhibitors (SSRIs) are usually chosen (fluoxetine is the only FDA approved drug for this disorder). They are used in high doses (e.g., fluoxetine 60mg daily) and help

reduce the frequency of bingeing, irrespective of the presence of depression (Zhu & Walsh, 2002).

Tricyclic antidepressants and the anticonvulsant topiramate have been used (Flament, et al, 2012).

**Hospitalization** is rarely required, but may be indicated when psychotherapy and antidepressants fail to help.

## **BINGE-EATING DISORDER**

### **Binge-eating Disorder – DSM-5 diagnostic criteria**

[first appearance as a DSM fully-fledged disorder]

- A. Recurrent episodes of binge eating
  1. eating, in a discrete period of time an amount of food that is definitely larger than most people would eat during the same time and in the same circumstances
  2. a sense of lack of control over eating during the episode.
- B. Episodes associated with 3 (or more)
  1. eating more rapidly than normal
  2. eating until feeling uncomfortably full
  3. eating large amounts when not feeling physically hungry
  4. eating alone because of embarrassment about quantity
  5. feeling disgusted, depressed or guilty afterward.
- C. Marked distress because binge eating is present
- D. Occurs once a week for 3 months
- E. Not associated with recurrent use of inappropriate compensatory behaviour.

Individuals tend to be middle ages, rather than adolescents or young adults.

**Prevalence** among adults: females 1.6%, males 0.8%.

Commonly co-occurs with obesity (but may be found in normal weight individuals), and tends to be intermittent (Fairburn et al, 2011).

**Pathophysiology** – reduced brain mu-opioid receptor (MOR) availability has been described (Joutsa et al, 2018).

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