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Emotional Eating, Binge Eating and Animal Models of Binge-Type Eating Disorders

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Abstract
Purpose of Review The objective of this paper is to review the role that hedonic factors, emotions and self-regulation systems have over eating behaviours from animal models to humans.
Recent Findings Evidence has been found to suggest that for some high-risk individuals, obesity/binge eating may develop as an impulsive reaction to negative emotions that over time becomes a compulsive habit. Animal models highlight the neural mechanisms that might underlie this process and suggest similarities with substance use disorders.
Summary Emotional difficulties and neurobiological factors have a role in the aetiology of eating and weight disorders. Precise treatments targeted at these mechanisms may be of help for people who have difficulties with compulsive overeating.

Keywords Emotional eating · Neurobiology · Animal model · Bulimia nervosa · Binge eating disorder

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Introduction

Gerald Russell first described bulimia nervosa (BN) in 1979 [1], and binge eating disorder (BED) has only been recently accepted as a diagnostic category in the fifth edition of the Diagnostic and Statistical Manual of Mental Disorders [2]. BN and BED are characterised by a loss of control over eating and the rapid consumption of large amounts of food [2]. In BN, compensatory behaviours are used to prevent weight gain (e.g. self-induced vomiting/compulsive exercising). These disorders are often co-morbid with obesity [3] and share similar risk and maintaining factors. Anorexia nervosa (AN) is characterised by under-eating and weight loss, although a substantial proportion of people with AN also binge eat and/or use compensatory behaviours [2].

In primary care, an increasing number of people received a diagnosis for an eating disorder in the 1980s and in the early 1990s [4–6]. This trend has continued into the beginning of the millennium [7]. The incidence rates of obesity have also increased at the same time worldwide [8]. One explanation for these trends is an increased level of awareness of eating disorders amongst the general population and clinicians. Another possibility is that this pattern of changes is due to new factors within the environment that may be impacting on eating and weight control.

There have been rapid changes in the food environment over the last 60–70 years with food technology changing what and how we eat [9]. For instance, nutrients have been ultra-processed and or purified, and foods have been modified to make them more accessible, cheaper and palatable [10]. The impact of these environmental changes on eating behaviour might be modified by individual factors [11]. For example, genetic factors may modulate the vulnerability to develop abnormalities in eating behaviours [12•], whilst the hedonic value of food [13] and emotions [14•] are other key factors that

may influence eating behaviour. These factors may promote either approach or avoidance behaviours towards food due to the processes of reward and punishment (see [15, 16] for detailed reviews of these motivational systems). Moreover, they may override the homeostatic control of eating behaviour and over time lead to overeating.

This paper will summarise animal models of pathological eating behaviour to help delineate the various mechanisms that may increase the risk for disordered eating behaviour. It will then outline recent findings in humans and suggest potential treatment targets. Please see Fig. 1 for an overview of the key risk factors that will be covered within this review.

Animal Models of Eating Behaviour—Possible Translational Insights

A variety of animal models have been developed in order to characterise the possible mechanisms involved in pathological eating behaviour and weight homeostasis in humans [17–20]. These models are based upon findings from lesion studies, pharmacological manipulations and by controlling the environmental conditions of rodents. Many of the models developed have included the factors that are thought to increase the risk of eating and weight disorders in humans. Therefore, they might help to understand the mechanisms that underpin abnormal eating behaviour.

Homeostatic Mechanisms and Eating Behaviour

Innate homeostatic mechanisms control nutritional balance. The hypothalamus plays a key role in the homeostatic control of eating [21]. For example, lesion studies in rats have allowed the identification of the specific structures involved, such as the hypothalamic ventromedial, paraventricular and dorsomedial nuclei as satiety centres and the lateral hypothalamus as a hunger centre [22, 23].

Circuits including the nucleus accumbens, the amygdala, the lateral hypothalamus and the ventral tegmental area are involved in hedonic food consumption. For recent reviews regarding the role of these circuits in eating behaviour and reward, please see [24, 25]. Also, please refer to [26] for an outline of the role of endocrine factors (i.e. leptin, ghrelin and insulin) on eating behaviour beyond the scope of this review. However, the specific neurochemicals involved in hedonic eating are worth discussing.

Dopamine

Research has shown that daily binge eating on a palatable sugar or fat diet is associated with increased dopamine release in the nucleus accumbens [27]. It is thought that the pleasure experienced from highly processed foods is positively correlated with the amount of dopamine released in the striatum. Over time, this leads to the downregulation of the expression D2R dopamine receptors in the striatum [28, 29]. This downregulation is associated with food consumption becoming

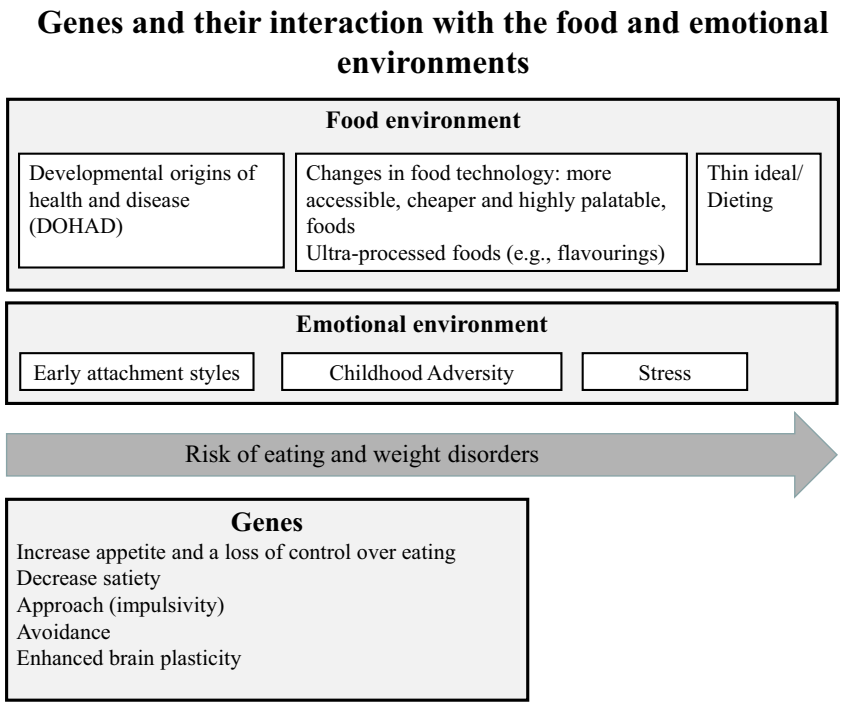

Fig. 1 Genes and their interactions with the food and emotional environments. This highlights the role that genetics and environmental factors (i.e. food and emotion related) have in the development of eating and weight disorders

more compulsive in its nature [31, 32•] and is similar to the rewarded processes observed in response to drugs of abuse [30]. Dopamine's role in food reward and addiction-like eating will be discussed below.

Opiates

Central opioid signalling pathways are also involved in food reward [33]. For instance, rats that overeat palatable food (i.e. glucose) show increased opioid receptor binding in the nucleus accumbens shell, the locus coeruleus, the cingulate cortex and the hippocampus [34, 35]. Furthermore, opioids are involved in the hedonic aspects of food consumption (e.g. pleasantness). Naloxone, an opioid antagonist, decreases the overall consumption of a sucrose diet [36]. Also, opioid antagonists decrease the intake of preferred foods to a higher degree than non-preferred foods [37], thus demonstrating opioid's role in reward experienced from hedonic food consumption.

Oxytocin

The effects of the neuropeptide oxytocin on food consumption are complex, as it appears to be involved in not only the maintenance of homeostasis but also in the regulation of hedonic eating [38, 39]. Oxytocin is thought to suppress the activation of reward pathways [41]. During palatable food intake, the release of endogenous opioids acts to inhibit oxytocin neurons, contributing to inhibitory control over intake [42]. As such, research shows that the central administration of an oxytocin receptor agonist reduces the tendency to restrict palatable food intake in a novel/stressful eating environment [40]. Moreover, mice whose oxytocin gene expression was knocked out consumed significantly more sucrose solution compared to their wild-type cohorts, implying that oxytocin pathways play a role in hedonic eating by increasing inhibitory control over intake [42].

Rodent Models of Non-Homeostatic 'Binge-Like' Eating

A variety of permutations of the environment have been found to induce rats to 'binge eat' (i.e. to overeat rather than to eat in accordance with homeostatic principles). Most of these involve limiting food intake, increasing food reward or associating food with punishment.

– Limited access models [43].

Intermittent, limited access to palatable food has been used to change eating behaviours and promote overconsumption. Importantly, after a fasting period, increased food intake continues even after basic metabolic needs are met.

– A stress-induced hyperphagia model [44].

This model suggests that food restriction combined with stress (e.g. an electric foot shock) induces binge-like increases in energy intake greater than that caused by restriction alone.

– Sham feeding models [45].

This model involves using a gastric fistula to induce drainage of consumed food before it enters the intestine [20]. Under such states, rats show an increase in binge eating behaviour as compared to control rats [46]. This sham feeding is often used to reproduce the compensatory behaviour of vomiting, impaired satiety and overeating amongst individuals with BN [17].

Food Palatability (Reward)

There has been a recent change in the food environment caused by technological modification of foods to increase their palatability. Several animal models include exposure to such palatable foods as a means of modulating the hedonic response to food. Some people and some rodents are more susceptible to change their eating behaviour in response to these foods than others [47, 48]. In support of this notion, it is possible to separate out binge eating prone (BEP) and binge eating resistant (BER) rats following exposure to palatable food [49]. BEP rats consistently consume more than twice the amount of palatable food compared to BER rats, although they show an appropriate homeostatic eating response when sated and when hungry [49]. The rats prone to overeat with palatable food over time become obese. These traits vary within wild-type rat strains, and there has been selective breeding to produce rats with sensitivity to dietary-induced obesity. Obesity-prone rats show greater changes in limbic and motivational circuits in response to palatable food [25, 48, 50•]. Moreover, these foods also produce a marked increase in AMPA glutamate receptor in the nucleus accumbens [25]. These findings suggest that there is an interaction between genotype and availability of highly palatable foods.

Stress (Punishment)

Exposure to aversive environments has been used to change eating behaviour in rodents. Short-term stressors such as cold exposure, water exposure or social defeat can reduce feeding behaviour in animals [44, 51]. However, chronic stress can lead to overfeeding on palatable food [52]. Pijlman and

colleagues [53] examined the impact of emotional stress (i.e. rats watch another rat receiving foot shocks) on eating behaviour versus physical stress (i.e. repeated foot shocks) in rodents. They found that emotional stress was associated with a preference of saccharin and hyperactivity, whereas physical stress was associated with a preference for water and anhedonia. Therefore, emotional distress has been found to increase hedonically driven eating in rodents.

'Addiction-Like' Eating Behaviour: How Binge Eating in Animals Relates to Substance Use Disorders

The change from impulsive responding and approach to the reward of highly palatable food into a more compulsive behaviour, which occurs even in the face to adverse consequences, is thought to be the mechanism that accounts for 'food/eating addiction'. This is a similar process to what occurs with substances of abuse (e.g. alcohol, cocaine use).

Indeed, there are similarities in the brain circuits and neurochemistry found with addiction to both substances and food [54]. For example, diets consisting of intermittent access to high-sucrose drinks are associated with a change in the balance of dopamine and acetylcholine in the brain. When these animals are given naloxone, extracellular dopamine and increased acetylcholine in the nucleus accumbens decreases [55]. Moreover, animals given nalaoxone show somatic signs of withdrawal, demonstrating symptoms such as anxiety, teeth chattering, forepaw tremor and aggression [55–58]. This suggests that opiate mechanisms are involved in this model of food/eating addiction [56]. Furthermore, the finding that rats are motivated to seek out highly palatable foods regardless of shocks to their feet is comparable to compulsive eating in humans, in which the behaviour persists despite negative consequences [59]. However, it is important to note that the magnitude of the effect is comparatively smaller for food than substances of abuse.

In Summary

- Changes in the food environment (palatable food, restriction and intermittent exposure) and also stress can lead to changes in eating behaviour in rodents. The context is important.
- The duration of stress (i.e. chronic or acute) and the kind of stressor (i.e. emotional or physical) differentially impact on eating behaviour.
- 'Addiction-like eating behaviour' occurs in animals when they continue to seek food even though they receive negative consequences (e.g. punishment with electric foot shocks.
- Changes in brain chemistry (e.g. dopamine and endogenous opiates) underpin eating behaviour in these animal models. For example, opiate-like withdrawal symptoms develop after the administration of naloxone and opiate antagonists.

In the following section, we will examine parallels between these processes and human eating behaviour.

Humans: Risk and Maintenance Factors

The Barker Hypothesis

In 1990, David Barker hypothesised that pre-natal and early post-natal experiences, such as under-nutrition during gestation or a low birth weight, predict the later onset of illness (see [60] for a review). This is now known as Developmental Origins of Health and Disease (DOHAD) and has a well-documented impact on body weight and metabolism [61]. Longitudinal studies of people exposed during early gestation (i.e. during the first trimester) to severe starvation in the Dutch famine of 1944–1945 have found higher levels of obesity amongst other adverse health markers in later life [62, 63]. A systematic review has found evidence that pre-natal exposure to diabetes, famine and cigarette smoking was linked to childhood overweight and obesity [64]. It has been suggested that these pre-natal factors alter the development of the endocrine system [64]. A range of obstetric complications have also been positively associated with the development of AN (e.g. maternal diabetes) and BN (e.g. low birth weight) [65].

What Is the Evidence for a Genetic Vulnerability for Problems in Eating Behaviour in Humans?

Some people appear to have a genetic susceptibility to anomalies in appetitive traits and behaviours [66–68]. Many genetic loci associated with obesity, such as variants in FTO, MC4R and BDNF genes, are expressed primarily within the hypothalamus and are thought to impact appetitive behaviours. For example, children with the AA genotype of the FTO (rs9939609) were reported by their parents to have inefficient satiety responsiveness [69]. Moreover, children with AA alleles and those with a single A allele for FTO have been found to consume significantly more than participants who were homogeneous for the T allele on a test meal [70•].

Furthermore, individuals with a higher polygenic risk score (based upon 32 genetic loci) for obesity have been reported to have increased emotional and uncontrolled eating [71•]. Specifically, variants of FTO, ZC3H4, MTCH2 and TNNI3K were positively associated with emotional eating. A recent study [72] also examined whether the polygenic risk

score (based upon 32 genetic loci) was associated with eating disorders. The FTO gene in particular was linked with the development of binge eating in adolescence. Another group of researchers [73] found that 34.7% of children homogenous/heterogeneous for the A allele self-reported a loss of control over eating versus 18.2% of the children homogenous for the T allele of FTO and those participants either homogenous/heterogeneous for the A allele ate highly palatable, energy-dense foods more frequently although in the context of a test meal, no differences in total energy intake occurred.

Genetic factors are also associated with disorders of under-eating (for a recent review, see [12•]). To date, genome-wide association studies of AN have not been sufficiently powered to indicate significant genetic loci [74, 75]. Nonetheless, they have suggested that studies with greater sample sizes could highlight genetic loci associated with AN and whether there is a specific polygenic risk profile for either of the AN subtypes (i.e. restricting or binge/purge) [e.g. 74]. This emerging research is helping to clarify the role of genetic factors in eating behaviours.

Early Adversity and Eating Behaviour

The early emotional environment also impacts on eating behaviour. Early attachment experiences increase the risk of eating disorders [76•, 77] and obesity [78]. Furthermore, adolescents [79, 80] and adults [81, 82] with AN have insecure patterns of attachment. A recent meta-analysis concluded that this effect was large (Cohen's d = 1.3) [76•].

Early adverse experiences impact on eating behaviour and weight control. A variety of abusive experiences increase the risk of BN and binge eating behaviour [83]. These types of experiences (i.e. psychological abuse, sexual abuse, physical abuse and neglect) are also more common in obese individuals [84, 85].

Hedonic Eating

Obese people appear to be oversensitive to approach food and experience higher levels of food craving [86–88]. This is associated with increased activation of the nucleus accumbens [89]. Moreover, longitudinal studies have found that atypical activation of nucleus accumbens in response to food cues is associated with weight gain [90, 91].

People with BN have been found to have an increased nucleus accumbens volume relative to healthy controls [92]. Furthermore, people with BN and BED are reported to have greater medial orbitofrontal cortex (OFC) volumes in comparison to people without eating disorders [92] and show increased activation of the OFC in response to food cues [93]. The medial OFC has been implicated in both reward processing [94] and impulsivity [95].

What Role Does Emotional Eating Have in the Development of Changes in Eating Behaviour and/or Weight Homeostasis?

The propensity to eat in response to positive [96•] and negative emotions [97, 98] is called '*emotional eating*' [99, 100]. Laboratory-based research has shown that emotional eating is predictive of increased food intake in young people [101]. However, there is uncertainty about the association between emotional eating and weight gain in children and adolescents. Cross-sectional research has shown greater levels of emotional eating in young people who are overweight relative to those within the healthy weight range [102, 103]. Other studies have not supported those findings (e.g. in girls [104]). A potential explanation is that, during childhood and adolescence, the relationship between food intake and BMI is non-linear, as weight gain may be more greatly influenced by other genetic and behavioural factors at this stage of development [104]. Indeed, eating in the absence of hunger at age 7 has been found to predict binge eating problems in adolescents (i.e. at age 15), with a higher BMI, dietary restraint, body dissatisfaction and negative affect elevating the risk [105]. Another recent prospective study showed that after a 1-year interval, emotional eating alone was not related to weight gain in adolescents; however, in conjunction with loss of control over eating, there was weight gain at the 1-year follow-up [106•].

In adulthood, there is a clearer association between emotional eating and obesity [107, 108]. Cross-sectional questionnaire-based research has shown that overweight/obese women report greater levels of emotional eating than participants within the healthy weight range [109, 110]. A 2-year prospective study has shown that emotional eating moderated a relationship between the over-consumption of food and weight gain [111].

With regards to BED, Masheb and Grilo found that emotional eating, particularly anxiety, is positively associated with eating psychopathology [112]. More recently, researchers have found that anger, feeling hurt by others, feeling disappointed, sadness and feeling guilty were associated to binge eating, suggesting that emotional eating may be related to a wide range of negative emotions and problematic interpersonal relationships [113]. This is in line with research that has suggested that people with eating disorders are often highly sensitive to social threat and rejection [114, 115].

Table 1 Emotion-based models of binge eating psychopathology

Model	Description
Emotion regulation model [120, 121]	The central tenet of this model is that binge eating is a maladaptive coping strategy for negative emotions. It suggests that binge eating reduces negative affect after eating excessive amounts of food. This strategy is negatively reinforced due to the temporary alleviation of distress that eating causes.
Escape theory [122]	The main proposition of this theory is that binge eating is a means of escaping or detaching oneself from negative aversive states. This is because during binge eating episodes, cognitive narrowing occurs whereby attention becomes solely focused towards the immediate environment such as food and disinhibited eating follows. This model suggests that binge eating is negatively reinforced as negative affect decreases during the episode of overeating.
The dual-pathway model of BN [123, 124]	The dual-pathway model suggests that there are two pathways that can lead to the development of BN symptoms. The first pathway involves dieting, and the second involves eating to reduce negative affect. For example, internalisation of the thin ideal may promote feelings of body dissatisfaction and negative affect. As a result, people may resort to extreme diets in order to try and achieve the thin ideal and improve their mood. However, dieting is often unsuccessful leading to further feelings of body dissatisfaction and increased negative affect. These pathways can lead to the development of BN symptoms.
Three-phase model of socioemotional functioning in eating disorders [125]	This model suggests that there are three phases of socioemotional functioning in eating disorders. The first phase involves predisposing factors for the illness such as adverse perinatal events, early attachment and temperament styles (e.g. shyness). The second phase describes maintaining factors of the illness such as difficulties in emotion regulation and a heightened sensitivity to rejection. The third stage is the impact of the eating disorder on close others. For instance, it may lead to an increase in carer anxiety or avoidance behaviours. This, in turn, may lead to deterioration in the patient's socioemotional functioning.

Several theoretical models have been proposed to outline the role of emotions in the development and maintenance of binge eating in humans

Ecological momentary assessment (EMA) techniques have been used to investigate the effect of emotions on binge eating episodes for people with BN and BED [116–118]. A meta-analysis of the EMA literature found that people report increased levels of negative affect prior to binge eating (medium effect size = 0.63) and that this negative affect increases even further following the episode (medium effect size = 0.5). For people with BN, negative affect was found to decrease following episodes of purging (medium effect size = −0.46) [119]. However, a limitation of EMA techniques is that this approach can only suggest an association, not causation, between negative affect and binge eating.

To help address this limitation, Cardi et al. did a meta-analysis of experimental studies that induce a negative or positive mood within the laboratory and assess subsequent eating behaviour on a test meal in comparison to a neutral condition. This review found that negative mood induction leads to increased food consumption, with greater effects in restrained eaters (very large effect size = 1.5) and people with BED (large effect size = 0.74) [14•]. It also showed that positive mood induction leads to increased food consumption in HCs (small effect size = 0.3). For people with BN, a limited number of studies suggested that positive mood induction could help to reduce food consumption. Please see Table 1 for an overview of several emotion-based theoretical models for the development and maintenance of the binge eating episodes.

Control Over Eating Behaviour: Impulsive and Compulsive Traits

Emotions are linked to drives to approach or withdraw from food; impulsive or compulsive patterns of behaviour can develop in response to these tendencies [126•, 127]. Smith and Robbins propose that overeating behaviours in obesity and BED may begin as an impulsive behaviour driven by reward [127]. However, with repetition, excessive habit formation may cause the behaviour to become triggered by cues such as negative affect rather than by reward. This occurs due to stimulus response learning and may be similar to the processes involved in addiction in substance use disorder (for a recent comprehensive review, see [128]).

Pearson et al. have suggested that impulsive and compulsive traits might also underlie the psychopathology of BN

Table 2 Translating animal models of overeating to humans: targets for new treatment approaches

Animal models of overeating	Translation to humans	Examples of targeted treatment approaches
Food rations are reduced.	Dieting and prolonged fasting	Implementation intentions (if-then planning approaches to help counter dieting) (e.g. [141])
They are given highly palatable, high sugar/fat food at irregular and unpredictable times.	Cravings lead to an impulsive episode of eating snack food, which is ubiquitous in Western societies.	Food-specific inhibition training (e.g. [142]) Attention bias modification (e.g. [143]) Approach-avoidance training (e.g. [144])
Stress induces overeating.	The trigger to diet is often a stressful event or difficulty. This leads to episodes of overeating.	Vodcasts: the use of calming music, imagery and guided relaxation scripts to help induce a positive mood for people with eating disorders (e.g. [145])
Downregulation of reward-based neural pathways.	Binge eating becomes compulsive in its nature.	Oxytocin administration (e.g. [146]).
Their stomach contents are drained after eating.	Vomiting and satiety disturbance in BN.	Implementation intentions (if-then planning approaches to help counter purging)

In this table, we link what is known to predispose overeating in animals to what happens in humans. This has implications for the use of targeted treatment approaches

[129•]. They also proposed that emotions have a core role in the development and maintenance of symptoms. This model hypothesises that there are two pathways towards the development of binge eating. The first *state-based* pathway suggests that when people experience negative emotions, there is a reduction in self-control. As a result, people may binge eat as they are unable to maintain the effortful demands of dietary restraint. The second pathway suggests that *trait-based* factors have a role in the development of BN. Specifically, the model suggests that the personality trait of negative urgency may make individuals vulnerable to over consume food, which is easily accessible during times of distress, with the expectation that it will provide relief. At the beginning of the illness, this behaviour is rewarding as it provides a distraction from negative emotions, whilst purging helps to lower feelings of distress that follow the binge. However, as the illness develops, these symptoms become compulsive as they continue despite the serious risks to health that are associated with them (e.g. cardiac problems). Furthermore, the function of the binge eating episodes shifts from being a distraction from negative emotions to a way of avoiding them completely.

A key mechanism that might underpin both impulsivity and compulsivity in obesity, BED and BN is impaired inhibitory control [126•]. This may be defined as the inability to stop an action [130]. Indeed, a systemic review of the literature [131] has shown that people with BED and BN have difficulties in inhibitory control (small effects −0.26 for BN and −0.16 for BED), with enhanced difficulties for illness-specific stimuli in BN (i.e. large effect sizes for food/eating = −0.67 and body-related stimuli = 0.61). In keeping with this notion, research has found that impaired inhibitory control is positively associated with a poorer treatment outcome for overweight children [132, 133]. It is thought that this inefficient self-regulatory system might be due to abnormal brain activation in the fronto-striatal networks [134–137]. Consequently, difficulties in inhibitory control may be one mechanism that helps to explain why people with obesity and binge eating develop eating behaviours that can become highly persistent and difficult to change. This predisposition to compulsively overeat may underpin obesity and eating disorders and is a possible target for treatment [138].

In Summary

- Food intake in humans is regulated by homeostatic/hedonic factors and memory (a full review of their role was beyond the remit of this article; nevertheless, please see [139, 140] for comprehensive reviews).
- People with obesity and binge eating crave highly palatable, energy-dense foods and find them highly rewarding.
- Evidence has been found to suggest that there is a genetic susceptibility for appetitive traits, obesity, binge eating and emotional eating.
- Emotional eating appears to have a role in the development and maintenance of obesity and binge eating.

- Difficulties in inhibiting approach or avoidance tendencies in response to emotions can lead to abnormal eating behaviours.
- Difficulties in inhibitory control can produce compulsive responding and habit formation.

Clinical Implications

This review has potential implications for the treatment of obesity/binge-type eating disorders. Recently, there has been an increasing focus on the use of novel treatment enhancers for eating and weight disorders. In line with this, please see Table 2 for an overview of how animal models of overeating translate to humans and examples of possible targeted treatment approaches.

Conclusion

This paper has covered recent studies relating to risk and maintenance factors for obesity and binge-type eating disorders. It suggests that there may be a genetic risk for overeating and binge eating behaviours, and these may develop as an impulsive coping strategy for negative emotions. This symptom may become compulsive in nature due to the process of excessive habit formation. Animal models highlight neurochemical changes that underlie inhibitory and reward pathways, which contribute to the maintenance of compulsive overeating. Additionally, through inducing forced abstinence, intermittent access, restriction and environmental stress, they offer valuable insight that is comparable to eating disorder populations. Moreover, they allow researchers to draw comparisons to substance use disorders, by inducing withdrawal and tolerance. Consequently, it may be of benefit to draw upon the use of novel targeted treatment approaches to further improve treatment outcomes for people with these conditions.

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Compliance with Ethical Standards

Conflict of Interest Robert Turton, Rayane Chami and Janet Treasure declare they have no conflict of interest.

Human and Animal Rights and Informed Consent This article does not contain any studies with human or animal subjects performed by any of the authors.

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