Neuronal activity and reward processing in relation to binge eating

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Purpose of review
Studies increasingly show the importance of reward processing in binge eating and provide evidence of associated changes in the neurobiological reward system. This review gives an up-to-date overview of the neurobiological substrates of reward processing subconstructs in binge eating. Neural findings are linked to different behavioral theories and the clinical relevance is discussed.

Recent findings
Increased neural responses in the orbitofrontal cortex, anterior cingulate cortex as well as striatum during anticipation and receipt of food rewards are found in association to binge eating. Increased model-free learning is also found and associated with altered brain reward reactivity. Data in rest report reduced striatal dopamine release and lower frontostriatal connectivity. Mechanisms of onset of binge eating are less clear, but specific personality traits, related to frontostriatal dysconnectivity, probably increase the risk of binge eating onset.

Summary
Both structural and task-based imaging studies show differences in the neurobiological reward system in binge eating. These changes are linked to specific reward processing, such as altered reward responsiveness to food cues, reinforcement learning, and habitual behavior. Findings are lined with different behavioral theories of binge eating, and a staging model is described, from onset to full illness development. Understanding the specific underlying aberrant reward mechanism in binge eating, associated with different stages of the illness, enables caregivers to focus their treatment more precisely.

Keywords
binge eating, neurobiological reward system, reward processing

INTRODUCTION

Binge eating is a complex psychopathological behavior, potentially associated with the brain reward circuit [1▪▪]. In this overview of the literature, we summarize recent evidence on brain reward regions involved in binge eating, in association with specific subconstructs of reward processing. We will also discuss these neuroimaging findings considering current theoretical models of binge eating and evaluate their potential use in clinical practice.

BINGE EATING AND REWARD PROCESSING

Binge eating is characterized by overconsumption of highly palatable food in a short period. The amount of food exceeds what most individuals eat at a similar time under similar circumstances. People who binge eat experience a loss of control over their food intake and in general feel guilty and ashamed about their eating behavior [2]. Psychiatric disorders such as binge eating disorder (BED), bulimia nervosa, and anorexia nervosa binge/purge-type (AN-BP) all have binge eating as pivotal symptom [2]. Unfortunately, treatment options for binge eating are insufficient. Cognitive behavioral therapy and interpersonal therapy are the most used treatments for binge eating, but around 50% of patients are not able to permanently stop this behavior [3]. To develop better treatment strategies, a more thorough understanding of the underlying psychopathology involved in binge eating is needed.

Aberrant reward processing is considered a potential mechanism of action in binge behavior

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KEY POINTS

- In binge eating, increased brain reward responses during anticipation and receipt of food are found, as well as different brain response during reinforcement leaning, and more model-free decision-making.
- Lower frontostriatal connectivity in binge eating is associated to habitual-like behavior.
- Personality styles that acquire maladaptive expectancies about food increase the risk of binge eating onset.
- Understanding different components of reward processing in binge eating enables caregivers to tailor treatment more precisely.

[1▪▪,4,5]. Reward processing is a broad construct and has different subcomponents such as having a pleasurable experience from rewards, feeling motivated to obtain rewards, and associating rewarding stimuli with its consequences [6]. Several theoretical frameworks exist to better understand these subcomponents. The model of Berridge and Robinson [6] includes three subcomponents of reward processing: consummatory pleasure (liking), motivation to pursue rewards (wanting), or anticipation and reward learning. The more recent model of the Research Domain Criteria (RDoC) separates reward processing, or positive valence system, into nine subconstructs, including reward responsiveness (with subconstructs: reward anticipation, the initial response to reward, and reward satiety), reward valuation, or the attribution of subjective and motivational value (with subconstructs: risk/ambiguity, effort behavior, and delay) and reward learning (with subconstructs: reinforcement learning, prediction error, and habit) [7]. Reinforcement learning consists of model-free and model-based learning [8]. In model-free reinforcement learning, decisions are made based on the consequences of behavior after carrying out a specific action several times. In model-based reinforcement learning, decisions on how to act are predicted before performing it, thereby planning what to do beforehand. These nine subconstructs of reward processing in RDoC have been associated to several regions within the brain reward circuit and vice versa, such as the anterior cingulate cortex (ACC), the orbitofrontal cortex (OFC), the ventromedial prefrontal cortex (vmPFC) and the dorsolateral PFC (dlPFC), as well as the insula, the ventral striatum or nucleus accumbens (NAc) and the dorsal striatum with the caudate nucleus and putamen [9,10].

Data associating binge-eating behavior to brain regions of the reward circuit are growing in the literature and already summarized in several recently published reviews [11–13]. These reviews mainly focus on the association between disturbed eating behavior and/or binge eating with specific brain reward pathways but do not take valid subconstructs of reward processing into account [1▪▪]. However, a better understanding of the specific reward profile involved in a patient's precise behavior will enable caregivers to focus their treatment more on the underlying problem at hand [14▪▪]. In this article, we give an up-to-date review of evidence on specific subconstructs of reward processing involved in binge eating and their association with brain regions within the neurobiological brain reward circuit. We will also consider the relevance of these findings in existing theoretical models of binge eating and discuss clinical implications.

NEUROIMAGING FINDINGS RELATED TO REWARD PROCESSING IN BINGE EATING

Neural correlates of reward responsiveness in binge eating

When it comes to reward responsiveness, a recent meta-analysis by Yu and Desrivieres [15] finds a significant hyperactivation in the dorsal striatum (right putamen) and hypoactivation in cortical areas of the brain (left inferior parietal cortex) related to anticipatory responses in subjects with disordered eating behavior compared with controls. However, studies focusing their investigation on binge eating using tasks that probe reward responsiveness specifically for food, find a higher activity of the OFC, ACC as well as striatum during the anticipation and receipt of high-energy-density food, compared with lower activity during the anticipation and receipt of money [16,17]. This is in line with structural data, finding higher volumes in cortical areas, as well as NAc [18▪,19▪]. Interestingly, patients with bulimia nervosa may have even higher OFC volume and activation of ACC during food anticipation compared with BED [11,20].

With regards to the initial response to a food, results also suggest a different reaction towards money, compared with food in binge eating [21]. After intake of sucrose, bulimia patients have shown higher brain activity in the left dlPFC, brainstem, OFC, and parietal cortex than controls, but a blunted neural response in the reward network and related areas (e.g. insula) in response to sweet tastes is reported also in the past [22,23], However, no difference in satiation effects between controls and binge eating was found [22]. Different changes in brain activity in reaction to different taste stimuli have been reported in subjects who binge eat as well [24].

Neural correlates of reward learning in binge eating

Imaging studies probing reward learning in binge eating find different reinforcement processing in ACC, PFC, and striatum compared with control subjects, but the results vary dependent on the task and studied patient group [25,26]. Overall, more model-free learning is reported, indicating that individuals who binge eat are more likely to base their decisions on the outcomes of previous choices [27].

Patients with binge eating also have more habitual-like behavior compared with controls, linked to lower frontostriatal functional connectivity (i.e. less co-activation between the frontal cortex and striatum) and lower levels of metabolites (N-acetyl aspartate) in the frontal cortex [18▪,27]. Specifically, patients with binge eating show higher functional connectivity in striatal subregions compared with healthy controls, and a lower functional connectivity between cortical areas, as well as between frontal cortex and striatum [28,29]. These data suggest that individuals who binge eat have potentially more compulsive decision-making and that there is a functional disconnection between the frontal cortex and striatum.

Neural correlates of reward valuation in binge eating

Concerning reward valuation, data on neural correlates in binge eating is limited, and differences between healthy controls and binge eating patients are less clear [30]. For example, no differences in risk-taking or delay discounting are found [19▪,31]. However, healthiness and tastiness are rated differently by patients diagnosed with bulimia nervosa, which have been associated with altered vmPFC activity, but for healthiness, this was lower in binge eating patients [32].

Limitation of the literature on the neural correlation of reward processing in binge eating

Several limitations need to be considered when reviewing evidence on neural substrates of reward processing in binge eating. In a critical systematic review on neural findings of reward processing in binge eating using RDoC, Leenaerts *et al.* [1▪▪] found a publication bias in the literature in binge eating toward the subconstruct reward responsiveness. Indeed, over half of the imaging papers investigating brain reward areas in binge eating focus on reward responsiveness, in contrast to only one-third on reward learning and less than 10% on reward valuation. Also, different patient groups are examined, and most studies do not compare diagnostic subgroups. Over half of the studies include patients diagnosed with bulimia nervosa, 30% with BED, and 10–20% with AN-bp or individuals who binge eat without a specific diagnosis. In addition, half of the studies with BED do not include a weight-matched control group. Further, most studies only include women, and only a minority of studies have taken the impact of factors such as comorbidities, stage of illness, race, ethnicity, and socioeconomic status into account. Finally, tasks used in functional imaging are often not adapted to binge eating. For example, money and not food are used as a reward, or reward learning is not investigated in specific contexts, such as during moments of stress.

INTEGRATING IMAGING FINDINGS OF REWARD PROCESSING IN THEORETICAL MODELS OF BINGE EATING

The incentive sensitization theory and reward responsiveness

The incentive sensitization theory of addiction can help to understand current findings in imaging and behavioral studies investigating reward responsiveness in binge eating [6]. The theory states that sensitization to the anticipation of food in the reward system is caused by repeated binge eating episodes over time, leading to higher incentive salience (i.e. motivational value) for food stimuli, and increasing anticipation. The receipt of the reward (liking) by contrast, does not increase with sensitization and may even decrease, necessitating the intake of even bigger amounts of food to create the same response [33]. Evidence confirms that the brain of a patient with binge behavior is sensitized to food cues, causing a higher activity of the ACC, insula, and OFC during the anticipation of food, which is also in line with the increased striatal dopamine response to food reported in bulimia patients compared with healthy controls [24]. Indeed, repeated dopamine release to food cues may consequently downregulate presynaptic dopamine receptor activity, reducing presynaptic dopamine levels and dopamine release in rest, which has been reported in substance use disorders, as well as patients with binge eating [34▪,35]. The incentive sensitization theory is also confirmed in several case–control studies, finding that higher food anticipation in binge eating compared with non-binge eating subjects is convincingly reported and correlated with the severity of binge eating [36]. Moreover, dopamine release in the dorsal striatum, as well as other neurotransmitters such as serotonin

have significantly been associated to binge eating in BED [34▪,37].

Habit model and reward learning

Behavioral evidence on reward learning in binge eating shows increases in model-free learning, reduced inhibitory control, and habitual action in patients with binge eating, which is in line with the habit model [8,38]. The habit model states that overtrained behavior, such as repeated binging behavior, may, over time, result in a shift from model-based learning towards model-free learning, enhancing compulsive decision-making, reducing the impact of inhibitory control, and thus further perpetuating the behavior [8,39]. The associated structural and functional disconnect between the frontal cortex and striatum with habits found in patients with binge eating is also seen in patients with obsessive–compulsive disorder [18▪,40]. Moreover, it stipulates that reward processing does not only involve one brain region but comprises a network interaction between brain areas [41]. Indeed, the reward system also interacts with other brain circuits, including cognitive control, emotion regulation, and interoceptive processing, which also influence the development and maintenance of binge eating behaviors [14▪▪].

The acquired preparedness model, reward valuation, and onset of binge eating

How binge eating behavior begins, is not clearly explained by the incentive sensitization theory or habit model. However, valuation of food reward, as a trait, is hypothesized to play an important role here [14▪▪]. Trait impulsivity in adolescents is linked to loss of control eating [42]. A recent longitudinal study in a community-based sample of adolescent girls showed both increased reward responsiveness and elevated valuation for monetary rewards to predict the onset of binge behavior over time [43]. Moreover, binge eating severity was associated to higher ventromedial PFC and caudate responses in this sample.

These findings are in line with the acquired preparedness model, which hypothesizes that binge eating develops because of a stronger learning response during a situation where overeating is rewarding [44]. The model hypothesizes that certain individuals acquire maladaptive expectancies about food because they display more 'high-risk' personality traits that influence reward learning [45]. For example, negative urgency, or the tendency to act rashly when experiencing emotional dysregulation, makes someone more likely to binge eat to regulate their emotions, causing them to 'learn' the expectancy that alleviates NA and/or elevates PA [46]. Several authors associate negative urgency in people suffering from binge eating with the connection between the vmPFC and striatum [47,48].

STAGING MODEL OF REWARD PROCESSING IN BINGE EATING

Bodell and Racine [14▪▪] recently suggested a more integrated model of staging, or maladaptive scaling that states that reward processing evolves over the course of illness in binge eating, starting with the at-risk state, and ending as a fully developed, rigid illness. Taking this approach enables the integration of different theoretical models proposed in the literature and may be highly relevant when choosing the right treatment for binge eating. First, binge eating behavior is initiated within subjects who have general 'high-risk' coping traits and hypersensitivity to reward, making them learn more quickly to expect positive consequences of specific eating behaviors [42–45]. Second, binge eating behavior is further propelled, via repeated food reward exposure that sensitizes food cues and increases food anticipation [5,6]. In the third phase, binge behavior becomes less goal-directed but more compulsive and rigid with an overall reward hyposensitivity [8,25]. General anticipation to pursue rewards (wanting), as well as subjective pleasure (liking) to nonfood rewards may decrease in this phase [14▪▪]. Indeed, elevated anhedonia, or overall lack of pleasure experience, is reported across the spectrum of eating disorder diagnostic criteria including patients with binge eating, separate from depressive feelings [49]. Anhedonia is probably an interplay of both wanting and liking, not often separated in human research. Interestingly, lower pleasure experience in eating disorders is probably mainly driven by an altered anticipation or motivation not liking [5].

Neural evidence for this staging model of binge eating is still limited because there are no longitudinal imaging studies that follow subjects who binge eat for a long period, across different stages in their life [5,14▪▪]. However, a relation between higher OFC volume and reduced reward responsiveness in binge eating is found in adolescents, which is inversely reported in adults [50,51]. Furthermore, even though patients with binge eating compared with controls have no difference in the volume of the putamen and globus pallidus, a positive correlation has been found between the volume of these regions and binge eating onset [52]. These findings imply that the initial onset of binge eating is associated with different brain areas than binge eating itself.

CLINICAL IMPLICATIONS

Novel treatments for binge eating specifically targeting reward-based processes are already examined [53,54]. However, these treatments do not take the role of different subcomponents of reward processing into account, for example, in relation to the phase of the illness. First, according to evidence on at-risk individuals, the development of binge eating, and other maladaptive appetitive behaviors, might be prevented by targeting general reward hypersensitivity, such as tendencies to act rashly when stressed [45]. Second, when patients develop a specific sensitivity to food rewards, during the early-phase/mid-phase of the illness, treatment may focus on altering food cravings, responses to food cues, and/or mindful eating techniques [55]. Third, as binge eating behavior becomes more compulsive and model-free controlled, relevant treatment strategies may focus on shifting back towards model-based learning and goal-directed decision-making [56]. The overall reduced reward processing of non-food rewards may be targeted by treatments focusing on general affect and emotion regulation [57].

CONCLUSION

In this overview, we summarize findings confirming both structural and functional differences in the neurobiological reward system in binge eating. These changes are linked to specific subconstructs of reward processing, such as altered reward responsiveness to food cues, reinforcement learning, and habitual behavior. Understanding the precise dysfunction of reward processing in a specific patient is important because specific subconstructs of reward mechanisms are targeted by different treatments. Future research investigating reward processing in binge eating should focus specifically on food rewards, use a validated framework to fine grain reward processing and compare differences between eating disorder subgroups. Moreover, longitudinal neuroimaging as well as clinical research is needed to shed more light on differences in reward processing over different stages of the illness.

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There are no conflicts of interest.

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- ■ of special interest
- ■■ of outstanding interest

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