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# Behind binge eating: A review of food-specific adaptations of neurocognitive and neuroimaging tasks

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# Abstract

Recurrent binge eating, or overeating accompanied by a sense of loss of control, is a major public health concern. Identifying similarities and differences among individuals with binge eating and those with other psychiatric symptoms and characterizing the deficits that uniquely predispose individuals to eating problems are essential to improving treatment. Research suggests that altered reward and control-related processes may contribute to dysregulated eating and other impulsive behaviors in binge-eating populations, but the best methods for reliably assessing the contributions of these processes to binge eating are unclear. In this review, we summarize standard neurocognitive and neuroimaging tasks that assess reward and control-related processes, describe adaptations of these tasks used to study eating and food-specific responsivity and deficits, and consider the advantages and limitations of these tasks. Future studies integrating both general and food-specific tasks with neuroimaging will improve understanding of the neurocognitive processes and neural circuits that contribute to binge eating and could inform novel interventions that more directly target or prevent this transdiagnostic behavior.

# Keywords

Binge eating; Bulimia nervosa; Binge eating disorder; Reward; Inhibition; Neuroimaging

# 1. Introduction

Binge eating, or the consumption of an objectively large amount of food in a discrete time period while experiencing a sense of "loss of control" [1], is associated with significant impairment and represents a major public health concern. Binge eating disorder (BED),

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defined in part by at least weekly binge eating for a three-month period, is the most prevalent eating disorder, estimated to impact 1.9% to 3.9% of the population [2,3]. Roughly 30-42% of individuals with BED also meet criteria for obesity [2,4-6], and BED shares many overlapping medical complications and health risks with obesity, including cardiovascular disease, diabetes, and metabolic syndrome [3,7,8]. Independent of the high degree of comorbidity with obesity, individuals with BED may experience increased levels of morbidity and mortality [9,10]. BED is also associated with increased likelihood of mood and anxiety disorders [11–14] and decreased quality of life [15,16]. Diagnosis of bulimia nervosa (BN), like BED, requires regular binge eating in the last three months but also requires recurrent compensatory behaviors, including but not limited to self-induced vomiting [1]. BN affects 1-3% of women [1,2] and also is associated with significant disability, medical complications, and high rates of comorbid psychopathology [1,17,18]. Anorexia nervosa (AN) has the highest mortality rate of any mental illness, and more than half of individuals who start with pure restricting-type AN develop regular binge eating and/or purging (i.e., the binge-eating/purging subtype of AN, or AN-BP) [19]. High levels of impairment, morbidity, and mortality in populations who engage in binge eating highlight a crucial need for effective treatments.

Depending on the disorder and setting, evidence-based treatments for binge eating produce only modest long-term outcomes for 30–60% of individuals seeking treatment [4,20–23]. Given the costly individual and societal impact of binge eating, identification and characterization of neuropsychological abnormalities that could serve as targets for new prevention efforts and interventions are crucial. The National Institute for Mental Health has supported a Research Domain Criteria (RDoC) initiative to examine shared mechanisms across diagnoses [24]. The study of unique mechanisms underlying different categorical eating disorder groups and presentations is useful and important, but identification of potential shared alterations and deficits across eating disorder diagnostic groups that have behavioral symptoms in common may ultimately improve treatment for all presentations. As a result, prior reviews and meta-analyses have integrated findings from individuals with BN, BED, AN-BP, and subthreshold binge eating behavior e.g., [25–27]. In the current review, we discuss neurocognitive tasks that have been or could be used to shed light on the mechanisms that may contribute to binge eating.

To date, research suggests that an imbalance between reward processes that promote consumption and self-regulatory control processes that limit food consumption may contribute to binge eating [28–30]. A variety of well-established stimulus-exposure paradigms and neurocognitive tasks have been used to gather neural and behavioral data in reward and self-regulatory domains and across a range of psychiatric disorders. Many of these tasks use generic stimuli such as letters, numbers, or neutral images to assess global deficits in these constructs. Because an imbalance in reward and control systems has been implicated in other psychopathologies (e.g., [31–34]), use of these general tasks in binge-eating populations is especially useful for identifying transdiagnostic neurocognitive mechanisms. However, improving targeted interventions for binge eating requires an understanding of the unique alterations in interacting reward and control-related processes that specifically underlie dysregulated eating behavior. Tasks that use symptom-specific (i.e., food- or eating-related) stimuli can isolate the distinct or perhaps more pronounced

neurocognitive deficits that predispose individuals to or result from eating problems. Several neuroimaging studies in the fields of substance and alcohol use disorders have used both general and substance-specific versions of tasks or stimuli within the same sample of individuals to isolate substance-specific behavioral and neural circuit alterations [35–37]. Few studies focused on binge eating pathology have followed a similar model, much less combined these two task types with neuroimaging. A thorough understanding of these symptom-specific tasks is necessary to adequately interpret alterations within binge-eating populations.

Moreover, results of existing investigations in binge-eating populations have been somewhat mixed. Some studies have and others have not detected altered behavioral task performance in individuals with binge eating compared with controls, and both hyper- and hypoactivation in reward and self-regulatory control circuits have been documented in binge-eating populations relative to controls (e.g., [38,39–42]). Given these inconsistent findings, optimal methods for reliably assessing the contributions of these reward and control-related processes and their neural correlates to binge eating are unclear.

Prior meta-analyses and reviews provide helpful syntheses of the results of neuroimaging and neurocognitive studies that assess general and symptom-specific impulsivity, reward responsivity, inhibitory control deficits, and reward-related decision-making alterations in binge-eating populations [25,43–46]. Several reviews also provide comprehensive summaries of the ventral and dorsal fronto-striatal reward and control-related circuit abnormalities hypothesized to be integral to binge eating and other behaviors [29,44,47–49]. However, to date, no paper has focused primarily on tasks that assess food-specific alterations in these processes among individuals with binge eating.

The purpose of the current review is to summarize and discuss the advantages and disadvantages of food-specific adaptations of well-established tasks designed to assess reward-related processes and self-regulatory control, with or without concurrent neuroimaging. The current review focuses on reward responsivity, reward-related decision-making, and inhibitory control assessments because of robust research findings implicating these constructs in binge eating [25,27,42,43,45,50] and the proliferation of food-adapted measures for these constructs. Of note, food-specific versions of tasks that assess and train other cognitive constructs, such as attentional bias to food cues [51–54] and food-specific memory bias [55], have been applied to binge-eating populations, and new studies are constantly emerging in this nascent field. However, the overwhelming majority of the literature has focused on the roles of self-regulation and reward-based processes in binge eating. Thus, we highlight these areas in detail to provide an in-depth analysis of food-specific tasks designed to investigate these constructs.

A summary of the food-specific tasks that have been used to study binge-eating populations are presented in Table 1 for reference, but we include example findings only for illustrative purposes. Additional food-specific task adaptations have only been studied in healthy or over-weight populations, but because their application to binge eating may significantly improve our understanding of this transdiagnostic behavior, we describe these tasks as well. We discuss the advantages and limitations of food-specific approaches and outline directions

for future research. By focusing on design considerations, this review is meant to serve as a methodological reference for researchers and a guide for future investigations within this population.

### 2. Reward responsivity, valuation, and learning

Reward abnormalities have been implicated consistently in binge eating. Literature dedicated to the understanding of reward has parsed this construct into three distinct components: liking, wanting, and learning [56]. In this model, liking refers to the hedonic valuation of a rewarding stimulus; wanting refers to the incentive salience of a stimulus; learning refers to associations formed between a predictive cue and the likelihood of a subsequent reward. Theoretical models posit that individuals who engage in binge eating are hyper-responsive to anticipatory food cues ("wanting") and are hypo-responsive to actual food consumption ("liking") and that this may interfere with effective subsequent estimation of the value of food and use of that valuation to guide behavior (learning). High levels of motivation to consume food coupled with diminishing returns upon actual consumption could contribute to the development and maintenance of binge eating behaviors [57]. Utilization of tasks with both non-food (e.g., money, pleasant non-food images) and food-specific stimuli may help to characterize the role of reward in binge eating more precisely.

#### 2.1. Traditional tasks that assess reward processes

Neurocognitive paradigms used to assess reward processes usually involve presentation of a rewarding stimulus and often require a behavioral response that serves as a proxy for sensitivity to the reward. Although most reward-based neuroimaging studies only require passive viewing or experiencing of rewarding stimuli (e.g., pleasant images), neurocognitive tasks have been developed to assess subcomponents of reward and integrate behavioral measurement with reward delivery.

**2.1.1. Progressive ratio tasks**—Progressive ratio tasks, adapted from the animal literature [58], require participants to "work," typically via repeated button pressing, for a rewarding stimulus. Participants must work longer and/or harder for each subsequent rewarding stimulus (i.e., under a progressive instead of fixed ratio schedule). The ratio of response to reinforcement increases until the participant stops responding a sufficient number of times to earn the reinforcer within an allotted time period. In this way, the task can assess a "breakpoint" that indicates the threshold at which an individual is no longer willing to work for the reward (e.g., [59,60]). Progressive ratio tasks are thought to measure reward motivation specifically and the reinforcing value or, if multiple rewards are used, relative reinforcing value, of a stimulus. The exact reinforcement schedule may vary considerably across studies, but all progressive ratio tasks assess the potency of the specific reinforcer using the "breakpoint" metric, with higher breakpoints representing higher reinforcing values [61].

**2.1.2. Reward-based learning tasks**—The monetary incentive delay task (MID) is a commonly used reward task that, combined with neuroimaging, can assess anticipation of, response to, and learning from monetary wins and/or losses [62]. The task, based on theories

of decision-making and instrumental conditioning, was designed to examine how reinforcement expectations guide behavior [63]. The MID assess reward-based learning via differential neural responses to "win" trials, "loss" trials, and trials on which there are no consequences. Neural circuitry implicated in the MID is comprehensively reviewed elsewhere [64]. Over the course of the task, participants implicitly learn the contingencies between cue stimuli and outcomes. With each "win" or "reward" the subjective value of the corresponding predictive cue is increased and the value of "non-winning" cues is decreased. Slight variations in MID design have been used to assess different stages of the reward process from reward or loss anticipation to processing [64]. In general, participants are typically first presented with a cue indicating the amount of money they can win or lose on the trial. Participants are then instructed to behaviorally respond to a target stimulus (during the "delay" period), and a subsequent reward or loss outcome is visually presented. Depending on the task version, the outcome may depend on the accuracy of the participant's response, which allows for additional assessment of operant conditioning. In addition, the magnitudes and probabilities of rewarding and aversive outcomes vary across versions of the MID.

#### 2.2. Food-specific adaptations of reward-related tasks

**2.2.1. Progressive ratio tasks**—Several variations of progressive ratio tasks have been adapted to measure the absolute or relative reinforcing value of food [61] and have demonstrated strong psychometric properties in children and adults [65–67]. These tasks require participants to press a keyboard button an increasing number of times to receive food rewards that they are then asked to consume. One adaptation of this task was used to compare how hard adult women with BN worked for chocolate candies (food reinforcers) relative to controls [68]. Participants could earn 10 candies per trial for 10 total trials. The first trial required 50 keyboard presses, and each subsequent trial increased by 200 presses. Chocolate candies were delivered as they were earned to provide immediate, direct reinforcement, and participants were instructed to consume all of the candies before moving on to the next trial.

Another study used a variant of this food-specific progressive ratio task to examine the reinforcing value of food when women with BN and healthy controls were instructed to consume a "comfortable" amount of food versus binge eat (or overeat, in the case of healthy controls). In this version of the task, the food reinforcer was not immediately delivered and consumed after it was earned, but button presses earned points that could be cashed in for the reinforcer (aliquots of a palatable yogurt shake) at the end of the task [69]. This task included 12 total trials. The first trial required 50 keyboard presses for one portion of shake (175 mL) and each subsequent trial required 200 more taps than the prior trial for a total of 13,800 taps for 2100 mL of the shake. Progress was visually depicted as an image of a container filled with shake on the screen, but the reinforcer was not consumed until after task completion.

These tasks used the same reinforcement schedule, but the tasks differed on the total number of trials (10 vs. 12), instructions (i.e., explicit instructions to binge eat or not), and reinforcer delivery timing (i.e., immediately or after the task is completed). The progressive ratio task

version that requires individuals to immediately consume the candies earned [68] may more closely mimic original progressive ratio tasks used to measure food and drug reinforcement in rodent models, and may measure the reinforcing value of continuing to consume food over time after eating has commenced. The version of the task with delayed consumption [69] more closely resembles human drug studies in which participants sample the drug on the study day and earn tokens to self-administer the drug on the days following the experiment [70]. This progressive ratio task may specifically assess the reinforcing value of delayed food rewards, instead of more purely assessing the immediate reinforcing value of food. In addition, the tasks used different food reinforcers. One used a food reinforcer that required chewing (chocolate candies), and the other used a liquid reinforcer (palatable shake). Evidence suggests that chewing impacts satiety [71]. Just as substance use literature indicates that route of administration affects the reinforcing value of drugs of abuse [70], whether foods are consumed as liquids or chewable solids in eating-adapted tasks may impact the rewarding properties of stimuli.

In addition to tasks that assess the reinforcing value of food, tasks that assess the relative reinforcing value of food stimuli have also been developed. Epstein and colleagues developed the most widely used progressive ratio food task designed for this purpose, the Food Reinforcement Task. Similar to tasks that assess the reinforcing value of a drug relative to money [72], various iterations of this task assess breakpoints for responses to food versus an alternative reinforcer (money or a pleasant sedentary activity) on a progressive ratio schedule [66,67,73–79]. This task has been used predominantly to study obese individuals, but a version of the task comparing the reinforcing value of snack foods and "healthy foods" (fruits and vegetables) was used to study women with and without binge eating who reported low and high levels of stress reactivity [80]. In this task, participants pressed a button to receive points toward their chosen reinforcer (either fruits/vegetables or snack food provided after the study). Participants could repeatedly change which reinforcer to work for, but over time, the reinforcement schedule for the snack food increased, while the fruit/vegetable reinforcement schedule stayed consistent. Total point values earned for the snack foods versus fruits and vegetables served as a proxy for the overall relative reinforcing value of snack food. This version of the task innovatively compared the value of "unhealthy" foods more likely to be included in binge episodes to that of "healthy" foods less likely to be included in binge episodes. However, because the task only used "healthy foods" as a comparison condition, rather than money or another non-food reinforcer, the task cannot be used to assess whether potential exaggerated reinforcement motivation among individuals who binge eat is food-specific or more generalized. Additionally, unlike tasks that assess the relative reinforcing value of a drug [72], the reinforcement schedule increased only for snack foods, rather than for the chosen reinforcer irrespective of its class. As a result, the relative reinforcing value of "healthy foods" cannot be directly compared with that of snack foods, because only snack foods became more difficult to earn.

Finally, one eating-specific progressive ratio task was adapted directly from those widely used in the animal literature [58,81,82] and required participants to suck on a straw for progressively longer time intervals to obtain a pre-determined amount of palatable liquid [83]. This task may be more ecologically valid than other adaptations of the progressive ratio

task, as the work required is eating-related and results in immediate food availability and consumption, but it has not yet been used to study binge-eating populations.

Overall, given food-specific progressive ratio task variations in pre-assessment instructions, food stimuli, relative reinforcers, and reinforcement schedules, comparing results across tasks is challenging.

2.2.2. Food reward-based learning tasks-The standard MID has been used to examine neural responses during reward anticipation and reward delivery or "receipt" in individuals with BED compared with controls [84], however, to our knowledge, only one study has utilized a combination monetary incentive delay/food-specific incentive delay task (MID/FID) to study individuals with binge eating, specifically those with BN or BED [85]. This task mirrored the traditional MID, but it included monetary-reinforcement blocks alternating with food-specific blocks during which, instead of winning money, participants could win "snack points". Upon completion of functional magnetic resonance imaging (fMRI), those points could be exchanged for a maximum of six sweet and salty snack foods, fruits, or beverages. The authors note that "snack points" make food abstract and closely represent a secondary reinforcer for food that is more directly comparable to monetary reward. However, there are important differences between traditional MID tasks and the MID/FID combination task: Traditional MID tasks typically include the potential to win or lose money, whereas the MID/FID assesses only the potential to win, and the traditional MID typically requires participants to press one button in response to a cue [86] whereas the MID/FID requires left or right button presses. These subtle differences in both content and display of information may substantially impact working memory and response selection demands, slightly altering the constructs each task is measuring.

Most of the other paradigms that have been adapted to assess the neural substrates of appetitive reward-related processes involve passive processing or anticipation and receipt of food-specific stimuli. These tasks can be used to study food-specific, reward-based learning by delivering rewards that are either predicted or not predicted by a preceding stimulus and assessing learning and appetitive prediction error signaling via computational modeling [87]. A version of such a paradigm was used to study reward learning differences in women with BN compared to matched healthy controls [88]. Participants were provided with one of three taste stimuli-sucrose solution, no solution, or artificial saliva-in association with three different paired visual stimuli [88]. Over the course of the task, participants learned to associate the visual stimulus with subsequent receipt of a taste, however, in 20% of the trials, the learned association was violated. In these cases, participants either received an unexpected sweet taste or the taste was unexpectedly omitted. The paradigm disentangles several reward related constructs by evaluating responses to a) taste anticipation, b) expected taste receipt, c) unexpected taste receipt, and d) unexpected taste omission. This permits comprehensive assessment of the neural correlates of associative learning in the taste domain.

To date, this task design has been used to study responses only to sweet and neutral tastes in individuals with binge eating. A temporal difference learning task that uses sweet, neutral, and aversive tastants has identified distinct neural substrates of appetitive and aversive

learning in healthy individuals [89], but this task has not yet been applied to the study of binge eating. Moreover, although binge eating episodes typically include sweet and high-fat tastes [90], few studies have investigated fat taste processing in binge-eating populations [91]. Temporal difference learning paradigms that use a variety of tastes to study reward-based learning in binge-eating populations would be beneficial.

Another version of this task uses intermixed food stimulus trials (juice or neutral tastes) and monetary image trials [92], but to our knowledge, this task has not been used to study bingeeating populations. In this task, participants do not passively view or receive stimuli, but are asked to initiate button-pressing responses to select a potential reward [92]. Because this task measures behavioral responses, it advantageously permits the inclusion of reaction time in computational models. Future applications of this combined taste and money task could distinguish food-specific from more generalized temporal difference learning alterations in individuals with binge eating.

# 3. Self-regulatory control

The broad construct of self-regulatory control encompasses attention, decision-making, response selection, action execution, conflict monitoring, response inhibition, and the ability to regulate emotional responses [93]. Difficulties with motor inhibitory control may be particularly relevant to understanding binge eating, since a sense of "loss of control" over eating is a defining, key element of binge eating episodes [94]. This sense of loss of control has, in fact, been suggested to be a more essential aspect of binge eating than the amount of food consumed during the episode [94–96]. In addition to difficulty controlling eating, high rates of other dysregulated behaviors, including, for example, impulsive shoplifting and non-suicidal self-injury [14,97–100], suggest more generalized impairment in control-related processes across multiple domains among individuals with binge eating. Thus, assessment of both generalized and eating-specific deficits in control-related processes may best inform understanding of the pathophysiology of binge eating.

#### 3.1. Traditional neurocognitive tasks that assess cognitive and behavioral control

Neurocognitive paradigms used to study cognitive and behavioral control typically require inhibition of a pre-potent response or ignoring interfering information in favor of some other response.

**3.1.1. Go/no-go tasks**—Go/no-go tasks require execution (i.e., go responses, usually button pressing) and inhibition of a prepotent motor response (i.e., no-go responses). These tasks are designed to measure action restraint, but also require attention, decision-making, and response selection capacities [101]. Button presses on no-go trials, or "commission errors" serve as in index of inhibitory failure.

**3.1.2. Stop-signal tasks**—Stop-signal tasks (SSTs) measure action cancellation by requiring a button-pressing response to go stimuli but withholding of that response when a rare auditory or visual stop signal sounds or appears [93,102]. The delay between the go stimulus and the stop signal varies across the task, resulting in easy (short stop-signal delay) and more difficult (long stop-signal delay) trials. Some SSTs are adaptive and become

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increasingly easy or difficult based on participant performance in the previous block. The main SST outcome measure is stop signal reaction time (SSRT), which accounts for both reaction time and response accuracy and is used as a proxy for inhibitory control. Unlike the go/no-go task, which measures preparational inhibition in an early activation sequence, the SST requires individuals to decide how long to wait before responding or inhibiting a response to the go stimulus [103]. Action restraint and action cancellation involve overlapping and distinct neural circuitry, further supporting the notion that go/no-go tasks and SSTs assess shared and unique inhibitory constructs [104,105].

**3.1.3. Stroop tasks**—Another commonly used inhibitory control task, the Stroop task [106], is a test of color-word interference and measures conflict monitoring, behavioral inhibition, and attentional interference [107,108] by requiring individuals to override a prepotent, word-reading response and instead name the color in which each word is printed. The Simon Spatial Incompatibility task [109], which like the Stroop task assesses interference inhibition, has been used to study individuals with BN [39,110], but this task has not yet been adapted for food-specific stimuli.

The abilities tapped by go/no-go tasks, SSTs, and Stroop tasks (action restraint, action cancellation, and conflict monitoring) may all interact and play a role in binge eating. To date, studies have used these standard neurocognitive tasks to document control-related neural alterations in individuals with binge eating [38,39,111,112,170].

#### 3.2. Food-specific adaptations of control-related tasks

**3.2.1. Go/no-go tasks**—One food-specific go/no-go adaptation was used to examine reaction times and task performance in women with and without binge eating and isolate food-specific inhibitory control deficits [113]. This task included images of both low and high-calorie foods and images of household items. Across six runs, the task examined go responses to high-calorie foods compared with no-go responses to household items, go responses to household items compared with no-go responses to high-calorie foods, go responses to household items compared with no-go responses to low-calorie foods, go responses to household items compared with no-go responses to low-calorie foods, go responses to high-calorie foods compared with no-go responses to low-calorie foods, and go responses to high-calorie foods compared with no-go responses to low-calorie foods, and go responses to low-calorie foods compared with no-go responses to low-calorie foods [113]. Teslovich and colleagues [114] developed a similar go/no-go task that included control images of toys instead of household items and did not include the two runs that directly contrasted high-calorie go cues versus low-calorie no-go cues and vice-versa. This four-run task was tested in healthy children and adults, but to our knowledge, the task has not yet been used to study binge-eating populations.

In both tasks, 25% of stimuli were no-go trials, creating a pre-potent response tendency, and image stimuli were carefully selected. All food and non-food images used in the first study were matched on a variety of dimensions, including brightness, complexity, and color composition [113], but whether participants perceived the food and non-food images as equally pleasant or rewarding is unknown. In the second study, an independent sample rated all stimuli for valence and arousal to ensure food and non-food stimuli were matched on

these parameters [114], but the images were not matched on visual characteristics like complexity and color. Other food-specific adaptations of the go/no-go task use neutral object and food words as stimuli [115–117], which may be less arousing, rewarding, or emotionally evocative than images. Because subjective stimulus features could impact accuracy and reaction time, future studies implementing these tasks should consider collecting participant ratings of all of these features.

Only two published studies of binge-eating populations have used neuroimaging in combination with go/no-go tasks that included both food-specific and neutral stimuli [118,119]. In one task, one block included high-calorie food images as go stimuli and toy images as no-go stimuli, and in the other block this was reversed [118]. The task was used with concurrent magnetoencephalography (MEG) to determine the specific nature of inhibitory control deficits in individuals with BED relative to weight-matched controls [118]. The second task was used with fMRI to compare women with BN to controls [119] and was a variant of a paradigm that had been previously adapted to study substance-use specific inhibition in alcohol use disorders [35]. This task included only neutral shapes as go and no-go stimuli, or neutral object images as go stimuli and food images as no-go stimuli [119]. Thus, these tasks used different combinations of image types paired differently with go or no-go instructions. Furthermore, in the task used to study women with BN, food images were never paired with a go response, preventing assessment of whether approach responses to food and action restraint in response to food are both altered in BN.

In two out of the three food-specific go/no-go tasks that have been tested in binge-eating populations thus far, pairings of go and no-go instructions with food and non-food stimuli change throughout the task. This requires continuous updating of associations between stimulus types and responses, which may increase the working memory load required for successful performance [120]. As a result, findings from "simple" go/no-go tasks in which the go and no-go stimuli remain constant may not be directly comparable to findings from more "complex" go/no-go tasks in which stimulus-response pairs change across alternating task runs.

**3.2.2. Stop-signal tasks**—Food-specific SSTs using both visual and auditory stop signals have been developed. One food-specific SST included mixed food picture and neutral object picture stimuli within the same run, requiring participants to inhibit responses when a blue frame appeared around the target image [121]. This task was used to study individuals with BED relative to weight-matched controls, and performance on the task was examined in relation to self-reported measures of disinhibited eating [121]. A similar adaptation of the SST has been applied to the study of individuals with binge eating relative to weight-matched controls and assessed inhibition in response to neutral images (2 runs), pleasant images (2 runs), and highly palatable food images (2 runs) using an auditory, instead of visual, stop signal [122]. These tasks differed on whether stimuli were separated or intermixed within runs, whether a pleasant image control condition was included, and the sensory modality of the stop signal. The relative advantages of the auditory SST [122] are 1) the separation of stimulus types across runs, which reduces the risk of potential arousal-based carry-over effects from trial to trial that could affect responses to neutral stimuli; and

2) the inclusion of a pleasant image control condition, which permits isolation of unique food-specific deficits from general difficulties inhibiting responses to pleasant stimuli.

Some studies have administered separate and complete general and food-specific versions of SSTs in the same sample of participants to examine general and food-specific action cancellation. For example, Houben, Nederkoorn, and Jansen [123] administered a traditional SST and a food-specific SST in a sample of healthy individuals. The tasks were parametermatched, except that the traditional task used two letters as go stimuli (Xs and Os), and the food-specific task used pictures of chips, chocolate, nuts, and cookies. The administration of two full tasks disadvantageously increases overall assessment time, but it reduces working memory load by avoiding intermixing blocks with different stimulus-response associations. This repeated measures SST design that completely isolates non-food and food stimuli has not, to our knowledge, been used to study individuals who engage in binge eating.

**3.2.3. Stroop tasks**—Stroop tasks that use food and eating related words are the beststudied food-related variants of self-regulatory control tasks. In these tasks, participants must name the color in which words related to food or eating (e.g., "cookies," "cake," "diet," "butter," and "cream") are printed [124,125]. Reaction time and interference effects are calculated, and faster reaction times and lower interference scores on incongruent trials suggest greater inhibitory control. Like SST adaptions, while some food-specific Stroop tasks use block designs, others use mixed designs in which both food- and non-food stimulus types are included in the same block and response time to each individual trial is assessed [126]. Results of a meta-analysis including mostly Stroop task studies suggest that deficits in cognitive control, or "interference inhibition," in individuals with binge/purge eating disorders were more pronounced when food-specific stimuli were used [25]. However, as previously noted, food words are likely less arousing and emotionally evocative than food images or actual food stimuli, reducing the ecological validity of food-specific Stroop tasks. In addition, the Stroop task simultaneously assesses inhibitory and attentional control and conflict monitoring. Therefore, it is unclear whether altered performance on food-specific versions of the Stroop task is indicative of deficits in one or all of these dimensions of self-regulatory control.

### 4. Food-specific task challenges and considerations

Results of studies using food-specific adaptations of classic paradigms have helped to identify differences between individuals with and without binge eating [25,29,44–49]. However, these paradigms have several limitations. Slight differences in task designs translate to the assessment of slightly different constructs using the same task type, and the extent to which increased food-specific reward responsivity and impaired control over eating-related responses uniquely contributes to binge-eating pathophysiology remains unclear.

#### 4.1. Ecological and construct validity challenges

Most food-specific task adaptations have used images of food (of varying palatability and caloric content) or food- or eating-related words. These visual stimulus tasks may assess responsivity to the anticipation of eating, or "wanting" [127–129]. Because reward

anticipation may predict actual food intake better than does the reward experienced once food is consumed [67,130], results from these tasks are very valuable; however, these tasks do not measure eating-specific behaviors, nor do they involve any consummatory component. Similarly, examination of neural response to liquid tastes—small and investigator-controlled quantities of "food"—elucidate the neural correlates of "liking," but may ultimately tell us little about reward responses associated with actual eating or the neural mechanisms of the sense of the loss-of-control over eating essential for a "binge episode."

Moreover, the extensive variety of foods included in binge-eating episodes [90] and the wide range of possible image stimuli that could be included in a food-specific task present a challenge for paradigm development and the study of binge-eating populations. Specific foods and quantities of food may be more novel, arousing, or salient to some individuals than to others. Using the same stimuli across participants reduces variability in data and facilitates between-group comparisons. Nevertheless, some research suggests that personalizing task stimuli may be beneficial. For example, in recent tasks and computer-based training programs used to study anxiety disorder populations, stimuli are individually-selected words or pictures that each participant rates as particularly salient to their unique fears [131,132]. Further research is needed to evaluate the potential advantages of personalized food stimuli in neurocognitive task adaptations. Assessing participant ratings of attractiveness, familiarity, frequency of consumption (in regular eating and binge eating episodes), liking, and wanting of each stimulus used in paradigms permits examination of the potentially confounding effects of these individual differences.

In addition to unique challenges to task design, food stimulus properties also present challenges to results interpretation. Findings from food-specific task adaptations, like drug-specific task adaptations, may be difficult to compare to those from traditional neurocognitive tasks because of differences between primary and secondary reinforcers. Most notably, neural activation associated with primary and secondary reinforcers differs [133]. Some tasks have attempted to minimize this difference by using secondary reinforcers or "points" toward later food to be consumed and money to be awarded [85]. However, secondary reinforcers, like money, are relatively less vulnerable to saturation and more likely to hold stable values, whereas after a certain point, earning and consuming more food is no longer rewarding. These differences need to be considered in tasks that aim to directly compare food and non-food rewards.

The extent to which various task adaptations assess overlapping or unique processes also remains unclear. Food-specific adaptations of standard neurocognitive and monetary tasks may simultaneously assess food reward anticipation, food reward receipt, food-related decision-making and response selection, food reward valuation, and behavioral inhibition in response to food.

#### 4.2. Timing challenges

Results of studies examining responses to visual food stimuli or palatable tastes are also difficult to compare because of differences in participant hunger level and metabolic state before task completion. Some studies have opted for fasted study designs, while others

require participants to be recently fed. Among female study participants, menstrual phase also may affect task performance and neural response [134–138]. In addition, just as time of last use may confound results in the substance use literature, recency of eating and binge eating, recency of weight gain or loss, duration of binge eating, and treatment status may contribute to heterogeneity in food-specific task findings in healthy and binge-eating populations.

#### 4.3. Population validity challenges

Inconsistencies across sample characteristics also preclude definitive conclusions about mechanisms that contribute to binge eating behaviors. Studies with stringent inclusion and exclusion criteria may beneficially isolate characteristics unique to binge eating disorders, but results of these studies may not apply to the majority of individuals who struggle with these behaviors. Given that binge eating is highly comorbid with other disorders and behaviors associated with neurocognitive deficits, including substance use and personality, mood, and anxiety disorders [2], balancing internal and external validity in studies of binge eating is particularly challenging. There is also considerable heterogeneity among individuals who binge eat across eating disorder-specific variables, including binge-eating episode size and typical content, weight status, weight history, and the presence, type, and frequency of compensatory behaviors.

Further, different studies of binge eating measure clinical features differently. Some use dichotomous presence or absence of a diagnosis, while others use interviews or questionnaires to characterize binge eating severity or establish a "clinical cutoff" to characterize binge eating and non-binge eating groups (e.g., [80]). Comparison groups also differ across studies, with some using matched controls, others using another clinical sample and matched healthy controls for comparison (e.g., [139]), and others still including no control group. Further, individuals with BED or BN may be overweight, obese, or at a healthy weight [1,140], but healthy-weight and overweight individuals with and without binge eating are not consistently included across studies. Without weight-matched controls and non-weight-matched controls, deficits that may contribute to loss-of-control eating, those that may contribute to overeating without a sense of loss of control, and those that may be a consequence of overweight or obesity cannot be distinguished.

# 5. Future directions

Review of existing food-specific task adaptations and consideration of the methodological challenges unique to these tasks and the study of binge-eating populations suggest several directions for future study. First, future research should replicate previous designs. The exact same food-specific task is rarely used twice, and traditional and food-specific tasks are rarely matched on all parameters, thereby making it difficult to isolate consistent correlates and/or predictors of binge eating. Future research should also assess time-related factors, including time since last eating and binge eating episode, weight change, and duration of illness or age of binge eating onset, to control for any potential variance in study outcomes.

In addition, tasks that include pleasant, non-food control stimuli are required to tease apart the relative contributions of general and eating-specific increased "approach" or appetitive

"go" drives to dysregulated eating. To our knowledge, only one self-regulatory control task used in a binge-eating population has attempted to include such a control condition [122]. Alternative, non-food control stimuli that are primary reinforcers (e.g., sexual or romantic images [141]) and food-related secondary reinforcers, as used by Simon and colleagues [85] may reduce potential confounds. Studies including non-food, reinforcing stimuli may also have implications for treatment. For example, results of future studies using progressive ratio tasks with non-food alternative reinforcers may indicate that, as with substance use in addicted populations [142], alternative reinforcers can reduce binge food consumption in binge-eating populations. This would suggest that treatments focusing on increasing alternative reinforcers could be particularly effective for binge eating.

Very few studies, especially neuroimaging studies, include a priori a full, separate group of participants who engage in binge eating while at a low weight (e.g., [143]). Administration of the same tasks that have been used to document deficits in individuals with binge eating should be tested across the full weight spectrum, and in threshold BN, AN-BP, and clinical control (e.g., substance use disorder) groups. This could identify deficits that may be better explained by low-weight or compensatory behaviors and characterize cross-diagnostic general and food-specific mechanisms of binge eating and other impulsive behaviors.

In addition, future research should assess and investigate potential moderating factors, such as dietary restraint, that could impact performance on neurocognitive tasks. Individuals high on measures of dietary restraint show differences in inhibitory control at both a behavioral and neural level [144–147], particularly in relation to food-specific stimuli. Given that dietary restraint is associated with binge eating [148,149], future studies should examine dietary restraint as a potential moderator of task performance in binge-eating populations. Assessment and investigation of this and other similar self-report variables could further delineate the role individual differences may play in food-specific deficits among individuals with binge eating.

Only two published studies of individuals with binge eating have used both general and food-specific task blocks in the same sample with concurrent fMRI [85,119]. Precisely matching parameters of food-specific task adaptations to standard, non-food-specific tasks that have been previously tested in binge-eating samples would likely improve cross-study comparison. Studies that integrate standard and eating-related tasks in the same samples permit within- and between-subjects analyses that help delineate unique mechanisms of eating pathology.

Additional research should also investigate food-specific tasks as both treatment and assessment tools for binge eating. For example, food-specific go/no-go tasks that pair pictures of food (e.g., chocolate) with no-go stimuli also have been developed to test whether food-specific inhibition can be enhanced among individuals without binge eating (e.g., [123,150]). In these neurocognitive training interventions, food pictures are paired with "no-go" instructions. A recent meta-analysis reported a medium effect size for food-specific inhibition training programs on reducing food consumption among healthy individuals without disordered eating [151]. Thus, interventions designed to train action restraint in response to highly-caloric, palatable food stimuli may also provide added benefit to current

binge eating treatments. Like the go/no-go task, food-specific adaptations of the SST that pair pictures of palatable food or neutral images with no-go signals have also been developed to test whether food-specific inhibition can be enhanced [153] and thus implemented in novel treatment protocols. Although such tasks may hold promise for the treatment of disordered eating [154], no published investigations have used food-specific SST training interventions to study individuals with binge eating. Initial results in BN and BED have been promising [152], but large, randomized controlled trials are needed to determine the utility of these cognitive training programs in reducing binge eating symptomatology.

Of note, recent advances in the understanding of inhibitory control mechanisms suggest that both reactive control (i.e., stopping a response based on an external cue, as in all of the tasks described above) and proactive control (i.e., goal-focused preparation to stop a future impending response) should be considered in the context of psychiatric disorders [155]. Both types of inhibition may be relevant for binge eating, as the hallmark characteristic of a binge episode – the sense of loss of control over eating - is defined as difficulty stopping eating once one has started, and individuals with binge eating report difficulty preventing binge episodes from occurring [1]. Despite the potential relevance of proactive and reactive control to binge eating, tasks that permit explicit separate assessment of these abilities have not yet been tested in eating disorder samples [156]. Application of these tasks to bingeeating populations represents an important direction for future study.

In addition, further study of food-specific versions of tasks that assess the complex interplay of reward and inhibition is warranted. For example, the delay-discounting task assesses the ability to delay gratification by measuring the relative value of short-term smaller rewards compared with longer-term larger rewards. Both monetary and food-specific versions of the task have been used to study a number of populations, including individuals with binge eating [157–160]. However, the task concurrently assesses constructs related to reward anticipation, inhibition, decision-making and future orientation [161], and food-specific delay discounting tasks have thus far yielded inconsistent findings [47]. Because the task simultaneously assesses multiple complex cognitive abilities, delay discounting results in isolation may be less informative in delineating the processes contributing to binge eating. Delay discounting task data may be especially useful in combination with data from tasks that separately assess self-regulation and reward, and future research using food-specific delay discounting tasks in binge-eating populations is needed.

Despite the need for replication using existing tasks, future studies would also benefit from novel paradigms and technologies to improve ecological validity. Tasks that permit concurrent neuroimaging as participants become progressively more sated may clarify how neural reward valuation and responsivity change with changing metabolic state in individuals with eating pathology. In addition, tasks that assess reward response during eating and inhibitory control over eating behavior are needed. These capacities may be most relevant to the neurocognitive dysfunction that contributes specifically to binge eating. Because naturalistic eating creates problematic movement artifacts in fMRI, other imaging methodologies, including EEG, magnetoencephalography (MEG), and functional near-infrared spectroscopy (fNIR), alone or in combination with fMRI, may be required in

conjunction with food-specific adaptations of neurocognitive tasks to further elucidate the eating-specific neurocognitive processes in binge-eating populations [79]. Recently, fNIR was used to assess prefrontal activation in women with BN during a standard go/no-go task and a novel eating-go/no-go task that required inhibition of a prepotent tendency to inhibit sipping and swallowing of a palatable shake [162,163]. Future studies of individuals with binge eating would benefit from using a similar combination of methods that permits differentiation of the neural correlates of eating behavior-specific deficits.

Finally, as has been highlighted in the substance use disorder literature [164], future studies should focus on characterizing how general and food-specific reward and control-related processes change over time and whether alterations in these processes recover after symptom abstinence. Longitudinal designs are needed to determine whether behavioral and neural performance on food-specific paradigms relates to clinical outcomes in binge eating patients. Administration of neurocognitive tasks before and after non-invasive neuromodulatory interventions is another important direction for future research. Thus far, craving tasks have been used before and after repetitive transcranial magnetic stimulation (rTMS) and transcranial direct current stimulation (tDCS) to assess eating or appetite-related changes in healthy individuals [165–167]. Only one of these studies also included a monetary delay discounting task, but no changes were detected on this task or in food consumption following tDCS administration [167]. Studies of rtMS administration in individuals with "bulimic-type" eating disorders have similarly used self-reported craving or binge-purge frequencies to assess change [168,169]. Future research combining general and food-specific neurocognitive tasks with neuroimaging before and after neuromodulation will improve characterization of the neurocognitive processes and circuits that are directly and causally implicated in binge eating.

# 6. Conclusion

Methodological inconsistencies and the limited ecological validity of existing paradigms make it difficult to draw definitive conclusions about the reward- and control-related alterations that may contribute to binge eating. However, replication, continued development of food-specific adaptations of traditional neurocognitive tasks, and creative combinations of these tasks with neuroimaging and the study of eating behavior have potential to reveal the precise mechanisms of this behavior. Distinguishing food-specific from more global deficits will inform targeted, novel treatments for this transdiagnostic symptom.

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# References

- 1. American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders. fifth. American Psychiatric Association; Washington. D.C.: 2013. DSM-5
- Hudson J, Hiripi E, Pope H, Kessler R. The prevalence and correlates of eating disorders in the National Comorbidity Survey Replication. Biol Psychol. 2007; 61:348–358.

- Kessler R, Berglund P, Chiu W, Deitz A, Hudson J, Shahly V, et al. The prevalence and correlates of binge eating disorder in the World Health Organization World Mental Health Surveys. Biol Psychiatry. 2013; 73:904–914. [PubMed: 23290497]
- 4. de Zwaan M. Binge eating disorder and obesity. Int J Obes. 2001; 25(Suppl. 1):S51–S55.
- 5. Spitzer R, Yanovski S, Wadden T. Binge eating disorder: its further validation in a multisite study. Int J Eat Disord. 1993; 13:137–153. [PubMed: 8477283]
- 6. Spitzer R, Devlin M, Walsh B, Hasin D, Wing R, Marcus M, et al. Binge eating disorder: a multisite field trial of the diagnostic criteria. Int J Eat Disord. 1992; 11:191–203.
- 7. Mitchell J. Medical comorbidity and medical complications associated with binge-eating disorder. Int J Eat Disord. 2016; 49:319–323. [PubMed: 26311499]
- Mitchell J, King W, Courcoulas A, Dakin G, Elder K, Engel S, et al. Eating behavior and eating disorders in adults before bariatric surgery. Int J Eat Disord. 2015; 48:215–222. [PubMed: 24719222]
- 9. Bulik C, Reichborn-Kjennerud T. Medical morbidity in binge eating disorder. Int J Eat Disord. 2003; 34:S39–S46. [PubMed: 12900985]
- Agras W. The consequences and costs of the eating disorders. Psychiatr Clin N Am. 2001; 24:371– 379.
- 11. Grucza R, Pryzybeck T, CR C. Prevalence and correlates of binge eating disorder in a community sample. Compr Psychiatry. 2007; 48:124–131. [PubMed: 17292702]
- Javaras K, Pope H, Lalonde J, Roberts J, Nillni Y, Laird N, et al. Co-occurrence of binge eating disorder with psychiatric and medical disorders. J Clin Psychiatry. 2008; 69:266–273. [PubMed: 18348600]
- Telch C, Stice E. Psychiatric comorbidity in women with binge eating disorder: prevalence rates from a non-treatment-seeking sample. J Consult Clin Psychol. 1998; 66:768–776. [PubMed: 9803695]
- Yanovski S, Nelson J, Dubbert B, Spitzer R. Association of binge eating disorder and psychiatric comorbidity in obese subjects. Am J Psychiatry. 1993; 150:1472–1479. [PubMed: 8379549]
- Masheb R, Grilo C. Quality of life in patients with binge eating disorder. Eat Weight Disord. 2004; 9:194–199. [PubMed: 15656013]
- Rieger E, Wilfley D, Stein R, Marino V, Crow S. A comparison of quality of life in obese individuals with and without binge eating disorder. Int J Eat Disord. 2005; 37:234–240. [PubMed: 15822089]
- Erskine H, Whiteford H, Pike K. The global burden of eating disorders. Curr Open Psychiatry. 2016; 29:346–353.
- Wonderlich SA, Mitchell JE. Eating disorders and comorbidity: empirical, conceptual, and clinical implications. Psychopharmacol Bull. 1997; 33:381–390. [PubMed: 9550882]
- Eddy K, Dorer D, Franko D, Tahilani K, Thompson-Brenner H, Herzog D. Diagnostic crossover in anorexia nervosa and bulimia nervosa: implications for DSM-V. Am J Psychiatry. 2008; 165:245– 250. [PubMed: 18198267]
- 20. Grilo C, Masheb R, Wilson G, Gueorguieva R, White M. Cognitive-behavioral therapy, behavioral weight loss, and sequential treatment for obese patients with binge-eating disorder: a randomized controlled trial. J Consult Clin Psychol. 2011; 79:675–685. [PubMed: 21859185]
- 21. Mitchell J, Agras S, Wonderlich S. Treatment of bulimia nervosa: where are we and where are we going? Int J Eat Disord. 2007; 40:95–101. [PubMed: 17080448]
- Wilson G, Grilo C, Vitousek K. Psychological treatment of eating disorders. Am Psychol. 2007; 62:199–216. [PubMed: 17469898]
- 23. Wilson G, Wilfley D, Agras W, Bryson S. Psychological treatments of binge eating disorder. Arch Gen Psychiatry. 2010; 67:94–101. [PubMed: 20048227]
- Insel T, Cuthbert B, Garvey M, Heinssen R, Pine DS, Quinn K, et al. Research domain criteria (RDoC): toward a new classification framework for research on mental disorders. Am J Psychiatry. 2010; 167:748–751. [PubMed: 20595427]

- Wu M, Hartmann M, Skunde M, Herzog W, Friederich H. Inhibitory control in bulimic-type eating disorders: a systematic review and meta-analysis. PLoS One. 2013; 8:e83412. [PubMed: 24391763]
- 26. Haedt-Matt AA, Keel PK. Revisiting the affect regulation model of binge eating: a meta-analysis of studies using ecological momentary assessment. Psychol Bull. 2011; 137:660–681. [PubMed: 21574678]
- Van den Eynde F, Guillaume S, Broadbent H, Stahl D, Campbell IC, Schmidt U, et al. Neurocognition in bulimic eating disorders: a systematic review. Acta Psychiatr Scand. 2011; 124:120–140. [PubMed: 21477100]
- Appelhans B. Neurobehavioral inhibition of reward-driven feeding: implications for dieting and obesity. Obesity (Silver Spring). 2009; 17:640–647. [PubMed: 19165160]
- 29. Berner L, Marsh R. Frontostriatal circuits and the development of bulimia nervosa. Front Behav Neurosci. 2014; 17:395.
- Wierenga C, Ely A, Bischoff-Grethe A, Bailer U, Simmons A, Kaye W. Are extremes of consumption in eating disorders related to an altered balance between reward and inhibition? Front Behav Neurosci. 2014; 9:410.
- Dawe S, Loxton N. The role of impulsivity in the development of substance use and eating disorders. Neurosci Biobehav Rev. 2004; 29:343–351.
- Goldstein R, Volkow ND. Drug addiction and its underlying neurobiological basis: neuroimaging evidence for the involvement of the frontal cortex. Am J Psychiatry. 2002; 159:1642–1652. [PubMed: 12359667]
- Volkow N, Wang G, Baler R. Reward, dopamine and the control of food intake: implications for obesity. Trends Cogn Sci. 2011; 15:37–46. [PubMed: 21109477]
- Volkow ND, Wang G, Telang F, Fowler JS, Logan J, Jayne M, et al. Profound decreases in dopamine release in striatum in detoxified alcoholics: possible orbitofrontal involvement. J Neurosci. 2007; 27:12700–12706. [PubMed: 18003850]
- 35. Czapla M, Simon J, Frederich HC, Herpertz S, Zimmermann P, Loeber S. Is binge drinking in young adults associated with an alcohol-specific impairment of response inhibition? Eur Addict Res. 2014; 21:105–113. [PubMed: 25428114]
- Garavan H, Pankiewicz J, Bloom A, Cho JK, Sperry L, Ross T, et al. Cue-induced cocaine craving: neuroanatomical specificity for drug users and drug stimuli. Am J Psychiatry. 2000; 157:1789– 1798. [PubMed: 11058476]
- Goldstein R, Romasi D, Rajaram S, Cottone L, Zhang L, Maloney T, et al. Role of the anterior cingulate and medial orbitofrontal cortex in processing drug cues in cocaine addiction. Neuroscience. 2007; 144:1153–1159. [PubMed: 17197102]
- Lock J, Garrett A, Beenhakker J, Reiss A. Aberrant brain activation during a response inhibition task in adolescent eating disorder subtypes. Am J Psychiatry. 2011; 168:55–64. [PubMed: 21123315]
- Marsh R, Horga G, Wang Z, Wang P, Klahr K, Berner L, et al. An FMRI study of self-regulatory control and conflict resolution in adolescents with bulimia nervosa. Am J Psychiatry. 2011; 168:1210–1220. [PubMed: 21676991]
- 40. Bohon C, Stice E. Reward abnormalities among women with full and subthreshold bulimia nervosa: a functional magnetic resonance imaging study. Int J Eat Disord. 2010
- 41. Brooks S, O'Daly O, Uher R, Frederich HC, Giampietro V, Brammer M, et al. Differential neural responses to food images in women with bulimia versus anorexia nervosa. PLoS One. 2011; 6:1–8.
- 42. Schienle A, Schafer A, Hermann A, Vaitl D. Binge-eating disorder: reward sensitivity and brain action to images of food. Biol Psychol. 2008; 65:654–661.
- 43. Schag K, Schonleber J, Teufel M, Zipfel S, Giel K. Food-related impulsivity in obesity and binge eating disorder—a systematic review. Obes Rev. 2013; 14:477–495. [PubMed: 23331770]
- Lavagnino L, Arnone D, Cao B, Soares JC, Selvaraj S. Inhibitory control in obesity and binge eating disorder: a systematic review and meta-analysis of neurocognitive and neuroimaging studies. Neurosci Biobehav Rev. 2016; 68:714–726. [PubMed: 27381956]
- 45. Wu M, Brockmeyer T, Hartmann M, Skunde M, Herzog W, Friederich HC. Reward-related decision making in eating and weight disorders: a systematic review and meta-analysis of the

evidence from neuropsychological studies. Neurosci Biobehav Rev. 2016; 61:177–196. [PubMed: 26698021]

- 46. García-García I, Narberhaus A, Marqués-Iturria I, Garolera M, R doi A, Segura B, et al. Neural responses to visual food cues: insights from functional magnetic resonance imaging. Eur Eat Disord Rev. 2013; 21:89–98. [PubMed: 23348964]
- McClelland J, Dalton B, Kekic M, Bartholdy S, Campbell IC, Schmidt U. A systematic review of temporal discounting in eating disorders and obesity: behavioural and neuroimaging findings. Neurosci Biobehav Rev. 2016; 71:506–528. [PubMed: 27693228]
- Frank GKW. Altered brain reward circuits in eating disorders: chicken or egg? Curr Psychiatry Rep. 2013; 15:396. [PubMed: 23963630]
- 49. Kessler RM, Hutson PH, Herman BK, Potenza MN. The neurobiological basis of binge-eating disorder. Neurosci Biobehav Rev. 2016; 63:223–238. [PubMed: 26850211]
- 50. Waxman S. A systematic review of impulsivity in eating disorders. Eur Eat Disord Rev. 2009; 17:408–425. [PubMed: 19548249]
- Boutelle KN, Monreal T, Strong DR, Amir N. An open trial evaluating an attention bias modification program for overweight adults who binge eat. J Behav Ther Exp Psychiatry. 2016; 52:138–146. [PubMed: 27116704]
- 52. Shank LM, Tanofsky-Kraff M, Nelson EE, Shomaker LB, Ranzenhofer LM, Hannallah LM, et al. Attentional bias to food cues in youth with loss of control eating. Appetite. 2015; 87:68–75. [PubMed: 25435490]
- 53. Giel KE, Schag K, Plewnia C, Zipfel S. Antisaccadic training to improve impulsivity in binge eating disorder. Eur Eat Disord Rev. 2013; 21:488–492. [PubMed: 23893405]
- Schag K, Teufel M, Junne F, Preissl H, Hautzinger M, Zipfel S, et al. Impulsivity in binge eating disorder: food cues elicit increased reward responses and disinhibition. PLoS One. 2013; 8:e76542. [PubMed: 24146885]
- 55. Svaldi J, Schmitz F, Trentowska M, Tuschen-Caffier B, Berking M, Naumann E. Cognitive interference and a food-related memory bias in binge eating disorder. Appetite. 2014; 72:28–36. [PubMed: 24076410]
- 56. Berridge K, Robinson T, Aldridge J. Dissecting components of reward: 'liking', 'wanting', and learning. Curr Opin Pharmacol. 2009; 9:65–73. [PubMed: 19162544]
- 57. Davis C, Carter J. Compulsive overeating as an addiction disorder. A review of theory and evidence. Appetite. 2009; 53:1–8. [PubMed: 19500625]
- 58. Hodos W. Progressive ratio as a measure of reward strength. Science. 1961; 134:943–944. [PubMed: 13714876]
- 59. Chelonis J, Gravelin C, Paule M. Assessing motivation in children using a progressive ratio task. Behav Process. 2011; 87:203–209.
- Wolf D, Satterthwaite T, Kantrowitz J, Katchmar N, Vandekar L, Elliott M, et al. Amotivation in schizophrenia: integrated assessment with behavioral, clinical, and imaging measures. Schizophr Bull. 2014; 40:1328–1337. [PubMed: 24657876]
- Roane H. On the applied use of progressive-ratio schedules of reinforcement. J Appl Behav Anal. 2008; 41:155–161. [PubMed: 18595280]
- Knutson B, Fong GW, Adams CM, Varner JL, Hommer D. Dissociation of reward anticipation and outcome with event-related fMRI. Neuroreport. 2001; 12:3683–3687. [PubMed: 11726774]
- Knutson B, Westdorp A, Kaiser E, Hommer D. FMRI visualization of brain activity during a monetary incentive delay task. NeuroImage. 2000; 12:20–27. [PubMed: 10875899]
- 64. Lutz K, Widmer M. What can the monetary incentive delay task tell us about the neural processing of reward and punishment. Neurosci Neuroecon. 2014; 3:33–45.
- 65. Hill C, Saxton J, Webber L, Blundell J, Wardle J. The relative reinforcing value of food predicts weight gain in a longitudinal study of 7—10-y-old children. Am J Clin Nutr. 2009; 90:276–291. [PubMed: 19535428]
- 66. Saelens B, Epstein L. Reinforcing value of food in obese and non-obese women. Appetite. 1996; 27:41–50. [PubMed: 8879418]

- Epstein LH, Temple JL, Neaderhiser BJ, Salis RJ, Erbe RW, Leddy JJ. Food reinforcement, the dopamine D(2) receptor genotype, and energy intake in obese and nonobese humans. Behav Neurosci. 2007; 121:877–886. [PubMed: 17907820]
- Bodell L, Keel P. Weight suppression in bulimia nervosa: associations with biology and behavior. J Abnorm Psychol. 2015; 124:994–1002. [PubMed: 26191637]
- 69. Schebendach J, Broft A, Foltin R, Walsh B. Can the reinforcing value of food be measured in bulimia nervosa? Appetite. 2013; 62:70–75. [PubMed: 23178173]
- Stoops WW. Reinforcing effects of stimulants in humans: sensitivity of progressive-ratio schedules. Exp Clin Psychopharmacol. 2008; 16:503–512. [PubMed: 19086771]
- Miquel-Kergoat S, Azais-Braesco V, Burton-Freeman B, Hetherington MM. Effects of chewing on appetite, food intake and gut hormones: a systematic review and meta-analysis. Physiol Behav. 2015; 151:88–96. [PubMed: 26188140]
- Haney M, Foltin RW, Fischman MW. Effects of pergolide on intravenous cocaine selfadministration in men and women. Psychopharmacology (Berl). 1998; 137:15–24. [PubMed: 9631952]
- Epstein L, Bulik C, Perkins K, Caggiula A, Rodefer J. Behavioral economic analysis of smoking: money and food as alternatives. Pharmacol Biochem Behav. 1991; 38:715–721. [PubMed: 1871188]
- 74. Epstein L, Truesdale R, Wojcik A, Paluch R, Raynor H. Effects of deprivation on hedonics and reinforcing value of food. Physiol Behav. 2003; 78:221–227. [PubMed: 12576119]
- 75. Epstein L, Yokum S, Freda D, Stice E. Food reinforcement and parental obesity predict future weight gain in non-obese adolescents. Appetite. 2014; 82:138–142. [PubMed: 25045864]
- 76. Epstein L, Carr K, Lin H, Fletcher K. Food reinforcement, energy intake, and macronutrient choice. Am J Clin Nutr. 2011; 94:12–18. [PubMed: 21543545]
- 77. Temple J, Bulkley A, Badawy R, Krause N, McCann S, Epstein L. Differential effects of daily snack food intake on the reinforcing value of food in obese and nonobese women. Am J Clin Nutr. 2009; 90:304–313. [PubMed: 19458018]
- Carr K, Lin H, Fletcher K, Epstein L. Food reinforcement, dietary disinhibition and weight gain in nonobese adults. Obesity (Silver Spring). 2014; 22:254–259. [PubMed: 23512958]
- Val-Laillet D, Aarts E, Weber B, Ferrari M, Quaresima V, Stoeckel L, et al. Neuroimaging and neuromodulation approaches to study eating behavior and prevent and treat eating disorders and obesity. Neuroimage Clin. 2015; 24:1–31.
- 80. Goldfield G, Adamo K, Rutheford J, Legg C. Stress and the relative reinforcing value of food in female binge eaters. Physiol Behav. 2008; 93:579–587. [PubMed: 18158166]
- Richardson N, Roberts D. Progressive ratio schedules in drug self-administration studies in rats: a method to evaluate reinforcing efficacy. J Neurosci Methods. 1996; 66:1–11. [PubMed: 8794935]
- Meisch R. Oral drug self-administration: an overview of laboratory animal studies. Alcohol. 2001; 24:117–128. [PubMed: 11522433]
- Nally S, Gordon R, Gondek-Brown M, Farkas J, Sclafani A. Measuring food reward value in humans. Appetite. 2007; 49:304.
- 84. Balodis I, Kober H, Worhunsky P, White M, Stevens M, Pearlson G, et al. Monetary reward processing in obese individuals with and without binge eating disorder. Biol Psychiatry. 2013; 73:877–886. [PubMed: 23462319]
- Simon JJ, Skunde M, Walther S, Bendszus M, Herzog W, Friederich HC. Neural signature of food reward processing in bulimic-type eating disorders. Soc Cogn Affect Neurosci. 2016; 11:1393– 1401. [PubMed: 27056455]
- Wrase J, Kahnt T, Schlagenhauf F, Beck A, Cohen MX, Knutson B, et al. Different neural systems adjust motor behavior in response to reward and punishment. NeuroImage. 2007; 36:1253–1262. [PubMed: 17521924]
- O'Doherty JP, Dayan P, Friston K, Critchley H, Dolan RJ. Temporal difference models and rewardrelated learning in the human brain. Neuron. 2003; 38:329–337. [PubMed: 12718865]
- Frank G, Reynolds J, Shott M, O'Reilly R. Altered temporal difference learning in bulimia nervosa. Biol Psychol. 2011; 70:728–735.

- Pauli W, Larsen T, Collette S, Tyszka J, Seymour B, O'Doherty J. Distinct contributions of ventromedial and dorsolateral subregions of the human substantia nigra to appetitive and aversive learning. J Neurosci. 2015; 35:14220–14233. [PubMed: 26490862]
- 90. Allison S, Timmerman G. Anatomy of a binge: food environment and characteristics of nonpurge binge episodes. Eat Behav. 2007; 8:31–38. [PubMed: 17174849]
- 91. Radeloff D, Willmann K, Otto L, Lindner M, Putnam K, Leeuwen SV, et al. High-fat taste challenge reveals altered striatal response in women recovered from bulimia nervosa: a pilot study. World J Biol Psychiatry. 2014; 15:307–316. [PubMed: 22540408]
- Valentin V, O'Doherty J. Overlapping prediction errors in dorsal striatum during instrumental learning with juice and money reward in the human brain. J Neurophysiol. 2009; 102:3384–3391. [PubMed: 19793875]
- Eagle D, Bari A, Robbins T. The neuropsychopharmacology of action inhibition: cross-species translation of the stop-signal and go/no-go tasks. Psychopharmacology. 2008; 199:439–456. [PubMed: 18542931]
- 94. Wolfe B, Baker C, Smith A, Kelly-Weeder S. Validity and utility of the current definition of binge eating. Int J Eat Disord. 2009; 42:674–686. [PubMed: 19610126]
- 95. Mond J, Latner J, Hay P, Owen C, Rodgers B. Objective and subjective bulimic episodes in the classification of bulimic-type eating disorders: another nail in the coffin of a problematic distinction. Behav Brain Res Ther. 2010; 48:661–669.
- 96. Shoemaker L, Tanofsky-Kraff M, Elliott C, Wolkoff L, Columbo K, Ranzenhofer L, et al. Salience of loss of control for pediatric binge episodes: Does size really matter? 2010; 43:8.
- 97. Fischer S, Smith GT, Anderson KG. Clarifying the role of impulsivity in bulimia nervosa. Int J Eat Disord. 2003; 33:406–411. [PubMed: 12658670]
- Nasser J, Gluck M, Geliebter A. Impulsivity and test meal intake in obese binge eating women. Appetite. 2004; 43:303–307. [PubMed: 15527933]
- Rosval L, Steiger H, Bruce K, Israel M, Richardson J, Aubut M. Impulsivity in women with eating disorders: problem of response inhibition, planning, or attention? Int J Eat Disord. 2006; 39:590– 593. [PubMed: 16826575]
- 100. Steiger H, Lehoux P, Gauvin L. Impulsivity, dietary control and the urge to binge in bulimic syndromes. Int J Eat Disord. 1999; 26:261–274. [PubMed: 10441241]
- 101. Rubia K, Russelol T, Overmeyer S, Brammer M, Bullmore E, Sharma T, et al. Mapping motor inhibition: conjunctive brain activations across different versions of go/no-go and stop tasks. NeuroImage. 2001; 13:250–261. [PubMed: 11162266]
- 102. Logan G, Schachar R, Tannock R. Impulsivity and inhibitory control. Psychol Sci. 1997; 8:60-64.
- 103. Rubia K, Russell T, Overmeyer S, Brammer MJ, Bullmore ET, Sharma T, et al. Mapping motor inhibition: conjunctive brain activations across different versions of go/no-go and stop tasks. NeuroImage. 2001; 13:250–261. [PubMed: 11162266]
- 104. Mostofsky SH, Schafer JG, Abrams MT, Goldberg MC, Flower AA, Boyce A, et al. fMRI evidence that the neural basis of response inhibition is task-dependent. Cogn Brain Res. 2003; 17:419–430.
- 105. Swick D, Ashley V, Turken U. Are the neural correlates of stopping and not going identical? Quantitative meta-analysis of two response inhibition tasks. NeuroImage. 2011; 56:1655–1665. [PubMed: 21376819]
- 106. Stroop J. Studies of interference in serial verbal reactions. J Exp Psychol. 1935; 18
- 107. Stroop JR. Studies of interference in serial verbal reactions. J Exp Psychol. 1935; 18:643-662.
- Botvinick MM, Braver TS, Barch DM, Carter CS, Cohen JD. Conflict monitoring and cognitive control. Psychol Rev. 2001; 108:624. [PubMed: 11488380]
- 109. Simon JR. Reactions toward the source of stimulation. J Exp Psychol. 1969; 81:174–176. [PubMed: 5812172]
- 110. Marsh R, Steinglass JE, Gerber AJ, O'Leary KG, Walsh BT, Peterson BS. Deficient activity in the neural systems that mediate self-regulatory control in bulimia nervosa. Arch Gen Psychiatry. 2009; 66:1–13.

- 111. Balodis I, Molina N, Kober H, Worhunsky P, White M, Rajita S, et al. Divergent neural substrates of inhibitory control in binge eating disorder relative to other manifestations of obesity. Obesity (Silver Spring). 2013; 21:367–377. [PubMed: 23404820]
- 112. Marsh R, Steinglass J, Gerber A, O'Leary K Graziano, Wang Z, Murphy D, et al. Deficient activity in the neural systems that mediate self-regulatory control in bulimia nervosa. Arch Gen Psychiatry. 2009; 66:51–63. [PubMed: 19124688]
- 113. Lyu Z, Zheng P, Chen H, Jackson T. Approach and inhibition responses to external food cues among average-weight women who binge eat and weight-matched controls. Appetite. 2017; 108:367–374. [PubMed: 27789376]
- 114. Teslovich T, Friedl E, Kostro K, Weigel J, Davidow J, Riddle M, et al. Probing behavioral responses to food: development of a food-specific go/no-go task. Psychiatry Res. 2014; 219:166– 170. [PubMed: 24909971]
- 115. Loeber S, Grosshans M, Herpertz S, Kiefer F, Herpertz S. Hunger modulates behavioral disinhibition and attention allocation to food-associated cues in normal-weight controls. Appetite. 2013; 71:32–39. [PubMed: 23899903]
- 116. Loeber S, Grosshans M, Korucuoglu O, Vollmert C, Vollstadt-Klein S, Schneider S, et al. Impairment of inhibitory control in response to food-associated cues and attentional bias of obese participants and normal-weight controls. Int J Obes (Lond). 2012; 36:1334–1339. [PubMed: 21986703]
- 117. Mobbs O, Iglesias K, Golay A, Van der Linden M. Cognitive deficits in obese persons with and without binge eating disorder. Investigation using a mental flexibility task. Appetite. 2011; 57:263–271. [PubMed: 21600255]
- 118. Hege M, Stingl K, Kullmann S, Schag K, Giel K, Zipfel S, et al. Attentional impulsivity in binge eating disorder modulates response inhibition performance and frontal brain networks. Int J Obes. 2015; 39:353–360.
- 119. Skunde M, Walther S, Simon J, Wu M, Bendszus M, Herzog W, et al. Neural signature of behavioural inhibition in women with bulimia nervosa. J Psychiatry Neurosci. 2016; 41:E69– E78. [PubMed: 27575858]
- Simmonds DJ, Pekar JJ, Mostofsky SH. Meta-analysis of go/no-go tasks demonstrating that fMRI activation associated with response inhibition is task-dependent. Neuropsychologia. 2008; 46:224–232. [PubMed: 17850833]
- 121. Svaldi J, Naumann E, Trentowska M, Schmitz F. General and food-specific inhibitory deficits in binge eating disorder. Int J Eat Disord. 2014; 47:534–542. [PubMed: 24573740]
- 122. Manasse S, Goldstein S, Wyckoff E, Forman E, Juarascio A, Butryn M, et al. Slowing down and taking a second look: inhibitory deficits associated with binge eating are not food-specific. Appetite. 2016; 96:555–559. [PubMed: 26522509]
- 123. Houbon K, Jansen A. Training inhibitory control. A recipe for resisting sweet temptations. Appetite. 2011; 56:345–349. [PubMed: 21185896]
- 124. Ben-Tovin D, Walker M, Fok D, Yap E. An adaptation of the stroop test for measuring shape and food concerns in eating disorders: a quantitative measure of psychopathology? Int J Eat Disord. 1989; 8:681–687.
- 125. Black C, Wilson G, Labouvie E, Heffernan K. Selective processing of eating disorder relevant stimuli: does the stroop test provide an objective measure of bulimia nervosa? Int J Eat Disord. 1997; 22:329–333. [PubMed: 9285271]
- 126. Jones-Chesters M, Monsell S, Cooper P. The disorder-salient stroop effect as a measure of psychopathology in eating disorders. Int J Eat Disord. 1998; 24:65–82. [PubMed: 9589312]
- 127. Stice E, Spoor S. Relation of reward from food intake and anticipated food intake to obesity: a functional magnetic resonance imaging study. J Abnorm Psychol. 2008; 117:924–935. [PubMed: 19025237]
- 128. van der Laan L, de Ridder D, Viergever M, Smeets P. The first taste is always with the eyes: a meta-analysis on the neural correlates of processing visual food cues. NeuroImage. 2011; 55:296–303. [PubMed: 21111829]
- 129. Stice E, Spoor S, Ng J, Zald D. Relation of obesity to consummatory and anticipatory food reward. Physiol Behav. 2009; 97:551–560. [PubMed: 19328819]

- 130. Epstein LH, Wright SM, Paluch RA, Leddy J, Hawk LW, Jaroni JL, et al. Food hedonics and reinforcement as determinants of laboratory food intake in smokers. Physiol Behav. 2004; 81:511–517. [PubMed: 15135024]
- Amir N, Kuckertz JM, Najmi S, Conley SL. Preliminary evidence for the enhancement of selfconducted exposures for OCD using cognitive bias modification. Cogn Ther Res. 2015; 39:424– 440.
- 132. Amir N, Beard C, Cobb M, Bomyea J. Attention modification program in individuals with generalized anxiety disorder. J Abnorm Psychol. 2009; 118:28–33. [PubMed: 19222311]
- 133. Sescousse G, Caldu X, Segura B, Dreher JC. Processing of primary and secondary rewards: a quantitative meta-analysis and review of human functional neuroimaging studies. Neurosci Biobehav Rev. 2013; 37:681–696. [PubMed: 23415703]
- 134. Alonso-Alonso M, Ziemke F, Magkos F, Barrios FA, Brinkoetter M, Boyd I, et al. Brain responses to food images during the early and late follicular phase of the menstrual cycle in healthy young women: relation to fasting and feeding. Am J Clin Nutr. 2011; 94:377–384. [PubMed: 21593494]
- 135. Dreher JC, Schmidt PJ, Kohn P, Furman D, Rubinow D, Berman KF. Menstrual cycle phase modulates reward-related neural function in women. Proc Natl Acad Sci U S A. 2007; 104:2465– 2470. [PubMed: 17267613]
- 136. Gladis MM, Walsh BT. Premenstrual exacerbation of binge eating in bulimia. Am J Psychiatry. 1987; 144:1592–1595. [PubMed: 3688285]
- 137. Lester NA, Keel PK, Lipson SF. Symptom fluctuation in bulimia nervosa: relation to menstrualcycle phase and cortisol levels. Psychol Med. 2003; 33:51–60. [PubMed: 12537036]
- 138. Ossewaarde L, van Wingen GA, Kooijman SC, Backstrom T, Fernandez G, Hermans EJ. Changes in functioning of mesolimbic incentive processing circuits during the premenstrual phase. Soc Cogn Affect Neurosci. 2011; 6:612–620. [PubMed: 20817665]
- 139. Schienle A, Schafer A, Hermann A, Vaitl D. Binge-eating disorder: reward sensitivity and brain activation to images of food. Biol Psychol. 2009; 65:654–661.
- 140. Striegel-Moore RH, Franko DL. Should binge eating disorder be included in the DSM-V? A critical review of the state of the evidence. Annu Rev Clin Psychol. 2008; 4:305–324. [PubMed: 18370619]
- 141. Demos KE, Heatherton TF, Kelley WM. Individual differences in nucleus accumbens activity to food and sexual images predict weight gain and sexual behavior. J Neurosci. 2012; 32:5549– 5552. [PubMed: 22514316]
- 142. Donny EC, Bigelow GE, Walsh SL. Choosing to take cocaine in the human laboratory: effects of cocaine dose, inter-choice interval, and magnitude of alternative reinforcement. Drug Alcohol Depend. 2003; 69:289–301. [PubMed: 12633915]
- 143. Brooks S, O'Daly O, Uher R, Friederich H, Giampietro V, Brammer M, et al. Thinking about eating food activates visual cortex with reduced bilateral cerebellar activation in females with anorexia nervosa: an fMRI study. PLoS One. 2012; 7:e34000. [PubMed: 22479499]
- 144. Johnson F, Pratt M, Wardle J. Dietary restraint and self-regulation in eating behavior. Int J Obes. 2012; 36:665–674.
- 145. Watson TD, Garvey KT. Neurocognitive correlates of processing food-related stimuli in a Go/Nogo paradigm. Appetite. 2013; 71:40–47. [PubMed: 23892319]
- 146. Meule A, Lukito S, Vögele C, Kübler A. Enhanced behavioral inhibition in restrained eaters. Eat Behav. 2011; 12:152–155. [PubMed: 21385646]
- Kemmotsu N, Murphy C. Restrained eaters show altered brain response to food odor. Physiol Behav. 2006; 87:323–329. [PubMed: 16403540]
- 148. Spurrell EB, Wilfley DE, Tanofsky MB, Brownell KD. Age of onset for binge eating: are there different pathways to binge eating? Int J Eat Disord. 1997; 21:55–65. [PubMed: 8986518]
- Tuschl RJ. From dietary restraint to binge eating: some theoretical considerations. Appetite. 1990; 14:105–109. [PubMed: 2186700]
- 150. Veling H, Aarts H, Stroebe W. Using stop signals to reduce impulsive choices for palatable unhealthy foods. Br J Psychol. 2013; 18:354–368.

- 151. Turton R, Bruidegom K, Cardi V, Hirsch CR, Treasure J. Novel methods to help develop healthier eating habits for eating and weight disorders: a systematic review and meta-analysis. Neurosci Biobehav Rev. 2016; 61:132–155. [PubMed: 26695383]
- 152. Turton R, Nazar BP, Burgess EE, Lawrence NS, Cardi V, Treasure J, et al. To go or not to go: a pilot study of food-specific inhibition training for women with bulimia nervosa and binge eating disorder. preparation.
- 153. Lawrence N, Verbreggen F, Morrison S, Adams R, Chambers C. Stopping to food can reduce intake. Effects of stimulus-specificity and individual differences in dietary restraint. Appetite. 2015; 85:91–103. [PubMed: 25447023]
- 154. Juarascio AS, Manasse SM, Espel HM, Kerrigan SG, Forman EM. Could training executive function improve treatment outcomes for eating disorders? Appetite. 2015; 90:187–193. [PubMed: 25777264]
- 155. Aron AR. From reactive to proactive and selective control: developing a richer model for stopping inappropriate responses. Biol Psychiatry. 2011; 69:e55–e68. [PubMed: 20932513]
- 156. Bartholdy S, Campbell IC, Schmidt U, O'Daly OG. Proactive inhibition: an element of inhibitory control in eating disorders. Neurosci Biobehav Rev. 2016; 71:1–6. [PubMed: 27565516]
- 157. Davis C, Patte K, Curtis C, Reid C. Immediate pleasures and future consequences. A neuropsychological study of binge eating and obesity. Appetite. 2010; 54:208–213. [PubMed: 19896515]
- 158. Manwaring JL, Green L, Myerson J, Strube MJ, Wilfley DE. Discounting of various types of rewards by women with and without binge eating disorder: evidence for general rather than specific differences. Psychol Rec. 2011; 61:561–582. [PubMed: 24039301]
- 159. Reynolds B. A review of delay-discounting research with humans: relations to drug use and gambling. Behav Pharmacol. 2006; 17:651–667. [PubMed: 17110792]
- 160. Odum AL. Delay discounting: trait variable? Behav Process. 2011; 87:1-9.
- 161. McClure S, Laibson D, Loewenstein G, Cohen J. Separate neural systems value immediate and delayed monetary rewards. Science. 2004; 306:503–507. [PubMed: 15486304]
- 162. Berner, L., Winter, S., Ayaz, H., Shewokis, P., Izzetoglu, M., Marsh, R., et al. Reduced Prefrontal Activation during the Inhibition of Eating in Bulimia Nervosa: Potential Neural Mechanism Underlying the Sense of Loss of Control; Annual Meeting of the Eating Disorders Research Society, New York; 2016.
- 163. Berner, L., Witer, S., Ayaz, H., Shewokis, P., Izzetoglu, M., Marsh, R., et al. Eating-specific Inhibitory Deficits and Reduced Prefrontal Activation May Contribute to Loss-of-control Eating in Bulimia Nervosa; Annual Meeting of the Society for the Study of Ingestive Behavior, Porto; 2016.
- 164. Balodis I, Potenza M. Anticipatory reward processing in addicted populations: a focus on the monetary incentive delay task. Biol Psychiatry. 2015; 77:434–444. [PubMed: 25481621]
- 165. Fregni F, Orsati F, Pedrosa W, Fecteau S, Tome F, Nitsche M, et al. Transcranial direct current stimulation of the prefrontal cortex modulates the desire for specific foods. Appetite. 2008; 51:34–41. [PubMed: 18243412]
- 166. Goldman R, Borckardt J, Frohman H, O'Neil P, Madan A, Campbell L, et al. Prefrontal cortex transcranial direct current stimulation (tDCS) temporarily reduces food cravings and increases the self-reported ability to resist food in adults with frequent food craving. Appetite. 2011; 56:741–746. [PubMed: 21352881]
- 167. Kekic M, McClelland J, Campbell I, Nestler S, Rubia K, David A, et al. The effects of prefrontal cortex transcranial direct current stimulation (tDCS) on food craving and temporal discounting in women with frequent food cravings. Appetite. 2014; 78:55–62. [PubMed: 24656950]
- 168. Dunlop K, Woodside B, Lam E, Olmsted M, Colton P, Giacobbe P, et al. Increases in frontostriatal connectivity are associated with response to dorsomedial repetitive transcranial magnetic stimulation in refractory binge/purge behaviors. Neuroimage Clin. 2015; 8:611–618. [PubMed: 26199873]
- 169. Van den Eynde F, Claudino A, Mogg A, Horrell L, Stahl D, Ribeiro W, et al. Repetitive transcranial magnetic stimulation reduces cue-induced food craving in bulimic disorders. Biol Psychiatry. 2010; 67:793–795. [PubMed: 20060105]

170. Wu M, Giel KE, Skunde M, Schag K, Rudofsky G, de Zwaan M, et al. Inhibitory control and decision making under risk in bulimia nervosa and binge-eating disorder. Int J Eat Disord. 2013; 46:721–728. [PubMed: 23729277]

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# Table 1

Studies using food-specific adaptations of standard reward and inhibitory control tasks to assess binge-eating populations.

Authors	Participants	Task	Food and non-food specific task design	Stimulus type	Concurrent neuroimaging	Main findings
Bodell & Keel 2015	BN ( $N$ = 32); control ( $N$ = 32)	Reinforcing value of food	Progressive ratio food-specific reinforcement schedule to earn chocolate candies	Consumed food	None	BN > controls RVF breakpoint
Schebendach et al. 2013	BN ( $N$ = 10); control ( $N$ = 10)	Reinforcing value of food	Progressive ratio food-specific reinforcement schedule to earn strawberry yogurt shake	Consumed food	None	Under binge instructions: BN > control RVF breakpoint and work performance Under non-binge instruction: No group differences
Goldfield et al. 2008	BE/low stress $(N = 12)$ ; BE/high stress $(N = 12)$ ; BE/high stress $(N = 0)$ ; non-BE/low stress (N = 6); non-BE/ high stress $(N = 9)$	Relative reinforcing value of "unhealthy" vs. "healthy" food	Progressive ratio food-specific reinforcement schedule; participant choice between snack food or fruit/ vegetable	Consumed food	None	Stress vs. control condition: BE/ high stress earned more snack food points and non-BE/high stress earned less snack food points Stress condition: BE/high stress > non-BE high stress snack food points
Simon et al. 2016	BED ( <i>N</i> = 27); BN ( <i>N</i> = 29); control ( <i>N</i> = 55)	Monetary incentive delay/ food incentive delay task (MID/ FID)	Event-related design of modified MID including snack food-specific (S) and monetary (M) blocks in the order of SMSM or MSMS, counterbalanced across participants	Visual graphic cue, triangle target, and pictures of money with monetary amounts or pictures of snack foods with snack point values	fMRI	Expectation of food: BED/BN < control activation in PCC Receipt of food: BED/BN > control activation in mOFC, anterior mPFC and PCC anterior mPFC and PCC No group differences related to monetary reward
Frank et al. 2011	BN ( $N$ = 20); control ( $N$ = 23)	Taste temporal difference learning task	Event-related cued delivery of sucrose, no solution and artificial saliva in randomized order, 20% of trials had no presentation following sucrose cue and 20% of trials had sucrose presentation following the no solution cue; allocation of stimuli counterbalanced across subjects	Tastants	fMRI	BN < control activation in the insula, ventral putamen, amygdala and OFC for unexpected receipt BN: omission of taste and brain regression response to TD model
Lyu et al. 2017	BE ( <i>N</i> = 31); control ( <i>N</i> = 31)	Go/No-Go task	Runs including high-calorie food, low- calorie food and non-food household item stimult; each stimulus type served as a "go" or "no-go" in a block with each of the other stimulus types to total six blocks; order was counterbalanced across subjects	Pictures	None	BE > non-BE faster RTs and greater accuracy on high-calorie food "Go" trials
Mobbs et al. 2011	BED ( $N$ = 16); non- BED OB ( $N$ = 16); control ( $N$ = 16)	Go/No-Go task	"Food/body-mental flexibility task"; made up of two sections: (1) food and objects (e.g. pencil), (2) body-related words and objects; "No-go" target shifts within each section	Words	None	BED > non-BED errors and omissions in both sections of the task

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Authors	Participants	Task	Food and non-food specific task design	Stimulus type	Concurrent neuroimaging	Main findings
Hege et al. 2015	BED ( $N=17$ ); non-BED ( $N=17$ )	Go/No-Go task	One Go/no-go task of high-calorie food and nonfood toy stimuli, each stimulus type served as a "go" or "no-go" in one of two blocks; blocks presented in pseudo-randomized order and counterbalanced across groups	Pictures	MEG	BED: trend for decrease in accuracy for "No-go" of food stimuli BED > control activation in pre- and postcentral gyri associated with activity differences for "Go" in both stimulus types Food vs. toy stimuli in BED and toy vs. food stimuli in control: increased activity in BED and toy vs. food stimuli in control: acceptal gyrus control (not BED): accesstul "No-go" of food associated with activity in right dIPFC
Skunde et al. 2016	BN ( $N$ = 28); control ( $N$ = 29)	Go/No-Go task	Event-related Go/no-go task made up of general (G; square "go '/circle "no-go") and food (F; household item "go"/food "no-go") specific blocks, ordered GFFGGFFG for one run and FGGFFGGF for the other run, runs were counterbalanced across participants	Visual shapes and Pictures	fMRI	General task: controls > BN inhibition to no-go stimuli control > BN participants with most frequent BE acivation in right pre- and postcentral gyrus, right caudate, and right putamen No differences for food specific task
Svaldi et al. 2014	BED $(N = 31)$ ; non- BED $(N = 29)$	Stop-signal task	Modified SST including highly appetitive food items and neutral, non- palatable household items, balanced stimuli types	Pictures	None	BED > non-BED stop signal reaction time, and more difficulty inhibiting responses to food stimuli
Manasse et al. 2016	BE ( <i>N</i> = 25); non-BE ( <i>N</i> = 65)	Stop-Signal task	Modified SST including even number of blocks of neutral non-food stimuli (e.g. scissors), pleasant non-food stimuli (e.g. flowers) and highly palatable food stimuli; block order was counter-balanced across subjects	Pictures	None	BE > controls SSRT across stimuli types

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BN, bulimia nervosa; BED, binge eating disorder; OB, obese; BE, individuals with binge eating; ED, eating disorder; RVF, reinforcing value of food; OFC, orbitofrontal cortex; mOFC, medial orbitofrontal cortex; MID, Monetary Incentive Delay Task; RT, reaction time; fMRI, functional magnetic resonance imaging; MEG, magnetoencephalography.