Monetary Reward Processing in Obese Individuals With and Without Binge Eating Disorder

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Background: An important step in obesity research involves identifying neurobiological underpinnings of nonfood reward processing unique to specific subgroups of obese individuals.

Methods: Nineteen obese individuals seeking treatment for binge eating disorder (BED) were compared with 19 non-BED obese individuals (OB) and 19 lean control subjects (LC) while performing a monetary reward/loss task that parses anticipatory and outcome components during functional magnetic resonance imaging. Differences in regional activation were investigated in BED, OB, and LC groups during reward/loss prospect, anticipation, and notification.

Results: Relative to the LC group, the OB group demonstrated increased ventral striatal and ventromedial prefrontal cortex activity during anticipatory phases. In contrast, the BED group relative to the OB group demonstrated diminished bilateral ventral striatal activity during anticipatory reward/loss processing. No differences were observed between the BED and LC groups in the ventral striatum.

Conclusions: Heterogeneity exists among obese individuals with respect to the neural correlates of reward/loss processing. Neural differences in separable groups with obesity suggest that multiple, varying interventions might be important in optimizing prevention and treatment strategies for obesity.

Key Words: Binge eating disorder, fMRI, inferior frontal gyrus, insula, obesity, reward, ventral striatum

Neural reward systems—through their regulation of appetite, weight regulation, and treatment response—have been implicated in obesity (1–3). However, studies in obese populations have demonstrated both hyper- and hyporesponsivity reward neurocircuitry in response to food cues (4–8). These seemingly discordant findings might relate to heterogeneities among obese individuals (9). Obesity is associated with different forms of disordered eating behaviors. For example, groups with obesity and binge eating disorder (BED) differ from those with non-binge-related obesity on numerous behavioral and psychological dimensions (10). A current debate exists with regard to the applicability of "food addiction" to eating behaviors; although some investigators argue a lack of evidence (11), others propose that the construct seems particularly relevant to certain obese subgroups, such as BED (12,13).

Seemingly discordant findings might also reflect failures to adequately disambiguate phases relating to anticipatory and outcome processing (14). Reward anticipation is linked with ventral striatal (VS) activity, whereas greater medial prefrontal cortex activity is associated with reward notification or the outcome phase of reward processing (15–18). Food-cue studies making anticipatory-consummatory distinctions report greater anticipatory responsiveness in the VS, midbrain, amygdala, and thalamus relative to consummatory phases of reward processing in healthy individuals (19,20). Palatable food consumption is associated with greater activity in the orbitofrontal cortex (OFC) and insula, with increased responsiveness observed in obese individuals (19,21,22). In obesity, the anticipatory-consummatory distinction is particularly important, because energy intake seems strongly influenced by anticipatory signaling rather than actual food consumption (23). Heightened anticipation of food reward is posited as a trigger for overeating in obese individuals (20,24).

To date, neuroimaging studies distinguishing anticipatory/consummatory processing in populations with disordered eating provide complex findings. Obese, relative to lean, individuals show increased activity in the insula and inferior frontal gyrus (IFG) during food anticipation (22). However, in bulimia nervosa, a disorder characterized by binge eating, food anticipation is associated with diminished prefrontal and insula activity, relative to nonbinge-eating individuals (25). Striatal activity is associated with reward processing tasks (15–18,26,27), and altered striatal responses are associated with obesity and weight gain; however, although some studies demonstrate diminished activity after palatable food intake in obese individuals, others report increased striatal responding (6,22,28,29).

Similarly, the addiction literature includes seemingly ambiguous findings in reward processing, even when distinguishing anticipatory/consummatory components. For example, increased striatal activity has been reported in cocaine dependence during anticipatory processing (30), whereas diminished anticipatory VS responses have been noted in alcohol dependence (31) and pathological gambling (32). These differences might relate to specific disorders, methodological/analytical considerations, treatment-seeking status, or anatomical delineations of the VS; additional differences might relate to types of reinforcers (e.g., addiction-related/unrelated).

Although many neuroimaging studies examine reward processes related to food cue paradigms in obese populations, there is a dearth of investigations into non-food reward processing in obesity (33,34). Understanding generalized reward processing in obesity is important, because alterations in reward circuitry might

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represent vulnerabilities for disordered eating. The current study used functional magnetic resonance imaging (fMRI) to examine monetary reward processing during the anticipation and receipt of wins/losses in obese individuals with and without BED and a lean comparison (LC) group. Binge eating disorder differs significantly from other forms of obesity and eating disorders in numerous behavioral, body-image, psychological, and psychiatric markers (10,35,36). However, to date, only two neuroimaging studies have examined the bio-behavioral correlates of this disorder relative to other obese conditions. The first observed differences in overweight BED participants relative to overweight and lean groups without BED in responses of the ventromedial prefrontal cortex (vmPFC) to food cues (37). Recently, we observed brain activation differences between obese individuals with and without BED during a cognitive control task, with the BED group demonstrating relatively diminished activation in the IFG, vmPFC, and insula (38).

To investigate further differences in obese individuals with and without BED, we employed a widely used monetary incentive delay task (MIDT) to examine reward/loss processing (16,17,32,39,40). We hypothesized that the BED group would show diminished responding in the VS during anticipatory phases, whereas the OB group would demonstrate increased VS activity relative to the LC group. We hypothesized that, consistent with fMRI studies in bulimia (25), during the outcome phase the BED group would demonstrate decreased vmPFC, insula, thalamus, and IFG activity relative to the non-BED groups. Similarities in BED and OB groups were examined, given potential similarities between obese individuals in the neural correlates of reward processing.

Methods and Materials

Participants

Participants included 57 adults 19–64 years of age (mean age: 38.9, 34 female), where 64.9% ($n = 37$) identified as Caucasian, 29.0% ($n = 17$) identified as African American, 5.3% ($n = 3$) identified as Native American, and 1.8% ($n = 1$) identified as Asian American; 5.3% ($n = 3$) identified themselves as Hispanic, and 94.7% ($n = 54$) identified as non-Hispanic. Demographic information is in Table 1 and Supplement 1. Age was included as covariates in all group contrast analyses, given group differences in age and to control for potential age-related effects. Body mass index (BMI) in the BED group ranged from 30.1 to 44.1. The OB group included 19 individuals with a BMI ranging from 30.4 to 41.6 and the LC group consisted of 19 individuals with BMIs ranging from 20.4 to 24.6. The BED and OB groups did not differ on mean BMI, and as expected, these groups had higher BMIs than the LC group.

The obese BED group consisted of 19 treatment-seeking participants enrolled in a randomized placebo-controlled trial testing 4-month treatments of sibutramine and cognitive-behavioral-self-help interventions, alone or in combination. Following baseline measures described here, participants underwent the fMRI protocol before starting the treatments, which were delivered for 4 months. The proposed DSM-5 criteria for BED (www.dsm5.org) was used to verify that all individuals in the BED group met criteria, but no individuals in the OB or LC groups had a history or current expression of binge eating or other disordered eating behaviors.

Measures

MIDT. All participants completed the MIDT; the task and experimental methods are described elsewhere (32,39) and in the Methods section of Supplement 1.

fMRI Acquisition and Analysis. Images were obtained with Siemens TIM Trio 3T MRI systems (Siemens, Malvern, Pennsylvania). Image acquisition and analysis methods are detailed in Supplement 1. Functional images were preprocessed with SPM5 (Welcome Functional Imaging Laboratory, London, UK), normalized to the Montreal-Neurological-Institute template and smoothed with a 6-mm kernel full-width-at-half-maximum.

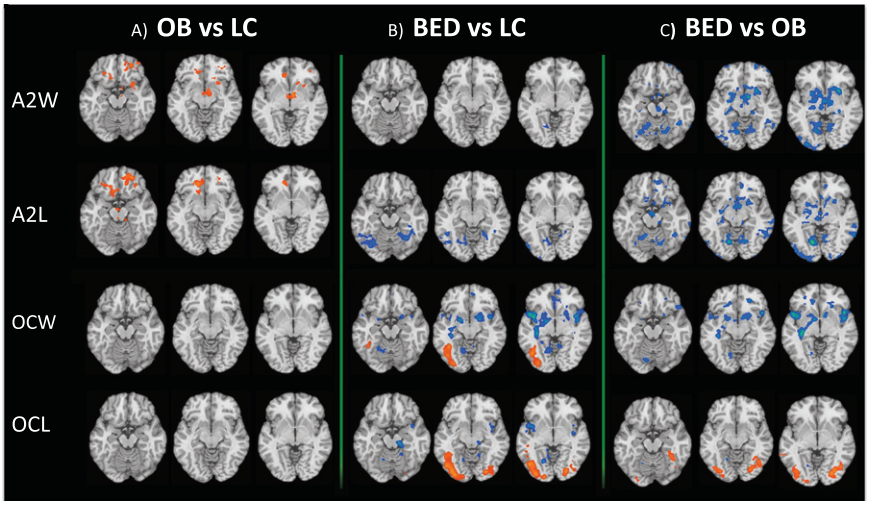

Figure 1. Group differences on the Monetary Incentive Delay Task in ventral fronto-striatal areas in obese individuals with binge eating disorder (BED) ($n = 19$), obese individuals without BED (OB) ($n = 19$), and a lean comparison (LC) ($n = 19$) group at $z = -17, -11, -6$. Brain activation maps demonstrate differences in the A2 winning phase (A2W) (associated with the anticipation of potentially winning money), the A2 losing phase (A2L) (associated with the anticipation of potentially losing money), the outcome winning phase (OCW) (associated with the receipt of a monetary reward), and the outcome losing phase (OCL) (associated with the loss of money). All contrast maps are thresholded at an uncorrected level of $p < .05$ two-tailed and family-wise-error-corrected at $p < .05$. Blue color demonstrates areas where subjects show relatively less activation, and red color indicates where participants show relatively greater activation. The right side of the brain is on the right.