



Original Article

Associations Between Neural Reward Processing and Binge Eating Among Adolescent Girls

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A B S T R A C T

Purpose: Neuroimaging studies suggest that altered brain responses to food-related cues in reward-sensitive regions characterize individuals who experience binge-eating episodes. However, the absence of longitudinal data limits the understanding of whether reward-system alterations increase vulnerability to binge eating, as theorized in models of the development of this behavior.

Methods: Adolescent girls (N = 122) completed a functional magnetic resonance imaging monetary reward task at age 16 years as part of an ongoing longitudinal study. Self-report of binge eating was assessed using the Eating Attitudes Test at ages 16 and 18 years. Regression analyses examined concurrent and longitudinal associations between the blood-oxygenation-level-dependent response to anticipating and winning monetary rewards and the severity of binge eating while controlling for age 16 depressive symptoms and socioeconomic status.

Results: Greater ventromedial prefrontal cortex and caudate responses to winning money were correlated with greater severity of binge eating concurrently but not prospectively.

Conclusions: This study is the first to examine longitudinal associations between reward responding and binge eating in community-based, mostly low-socioeconomic status adolescent girls. Ventromedial prefrontal cortex response to reward outcome—possibly reflecting an enhanced subjective reward value—appears to be a state marker of binge-eating severity rather than a predictor of future severity.

IMPLICATIONS AND CONTRIBUTION

Binge eating is a prevalent behavior in youth and is associated with negative health outcomes. This study contributes new information about the role of neural reward processing in binge eating during adolescence and has implications for future research on this topic.

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Binge eating is the consumption of an unambiguously large amount of food while simultaneously feeling a loss of control. This behavior typically emerges during adolescence and is associated with negative health outcomes and psychosocial impairment [1]. Thus, adolescence is an important developmental period for understanding binge-eating risk.

Given the characteristic overconsumption of palatable food during binge-eating episodes, disturbances in reward processing have been implicated in the etiology of binge eating [2]. The incentive sensitization theory posits that a heightened neural response to food receipt may influence the initial overconsumption of palatable foods, and repeated consumption leads to heightened neural response to food cues via conditioning [3]. Consummatory reward (i.e., reward from consuming palatable foods) decreases, whereas anticipatory reward (e.g., reward from cues associated with consumption) increases over time, which may exacerbate overeating episodes. Despite the support for this theory, key aspects remain to be examined, including the role of anticipatory and consummatory reward responses in predicting binge eating.

Neural correlates of reward anticipation and receipt

Findings from functional magnetic resonance imaging (fMRI) studies support heightened brain activation in response to viewing palatable food pictures in individuals with bulimia nervosa (BN) and binge-eating disorder (BED). Participants with eating disorders (EDs) have demonstrated increased activation in the ventromedial prefrontal cortex (vmPFC) [4,5] or the medial orbital frontal cortex (OFC) [5,6], the insula [6], the anterior cingulate cortex (ACC) [6,7], the posterior cingulate cortex [5], and the middle frontal gyrus [8]. Many of these regions have been implicated in processing emotional and motivational information, including reward valuation [9,10], suggesting an increased food reward sensitivity in individuals who experience binge-eating episodes. However, some studies show no differences between individuals with or without binge-eating behavior on neural response to an anticipatory food reward [11–13].

In contrast, several neuroimaging studies have found decreased brain activation in response to food reward outcomes in individuals with binge-type eating disorders. When given food, women with full or subthreshold BN have exhibited decreased activation in the insula [11,14], the precentral gyrus [11], the middle frontal gyrus [11], the thalamus [11], the lateral OFC [14], and the amygdala [14]. These regions have specific roles in reward-based learning and attention, gustatory sensations, and/or taste processing [9,15]. Taken together, reduced activation in response to food may underlie the need to overconsume in order to experience the desired reward [11].

The degree to which alterations in reward circuitry are specific to food or reflect general reward disruptions is less clear. In support of general disruptions, individuals who engage in binge eating exhibit other reward-related or impulsive behaviors [16] and greater self-reported reward sensitivity compared with controls [17]. Behavioral evidence suggests an increased overall reward valuation in individuals with BED [18] and a greater sensitivity to monetary gains in individuals with BN [19], supporting increased response to food and nonfood rewards in these populations.

Few studies have examined neural reward function using non-food reward cues in individuals who engage in binge eating. Compared with both overweight and healthy-weight nonbinge eaters, adults with BED demonstrated diminished activity in several prefrontal and insular regions in response to monetary reward outcome, suggesting broad alterations in reward responding in adults with BED [20]. However, in another study [5], individuals with BN or BED did not differ from controls on neural response to anticipating or receiving a monetary reward.

Additional studies using generic reward cues are necessary to elucidate the influence of neural reward processing on binge eating.

Neural mechanisms in the development of binge eating

Longitudinal studies also are required to test hypotheses about the predictive role of neural response to reward in the development of binge eating. In particular, studies that focus on (1) community-based populations that have broader variability in symptoms than clinical groups in traditional case-control studies and on (2) adolescents who are most vulnerable to eating pathology and experiencing ongoing neurodevelopment of reward systems [21] are needed.

The current study examined whether alterations in reward-related neural circuitry are concurrently and prospectively associated with binge eating in a community-based sample of adolescent girls. Consistent with the incentive sensitization theory regarding processing of rewards during the *development* of EDs, we hypothesized that greater activation in reward-related regions to anticipation and receipt of nonfood rewards would be positively associated with binge-eating severity at baseline and 2 years later.

Methods

Participants

Participants were 122 adolescent girls from the Pittsburgh Girls Study—Emotions Substudy (PGS-E) [22], recruited from the Pittsburgh Girls Study (PGS, $N = 2,450$) [23]. The PGS used a stratified random household sampling procedure with oversampling in low-income neighborhoods to enroll girls into four age cohorts (ages 5–8 years in wave 1). Girls and their mothers from the youngest PGS cohort were recruited at age 9 for PGS-E ($N = 232$) [22], and both completed annual assessments from ages 5 to 18 years. Girls completed an fMRI scan for the first time at age 16. Of the 232 PGS-E girls, 148 completed an fMRI monetary reward task at age 16, and 122 had useable data for the current analyses.¹

Study protocols were approved by the University of Pittsburgh Human Research Protection Office. Mothers provided written informed consent, and girls provided assent at age 16 and verbal consent at age 18.

Measures

The Eating Attitudes Test (EAT) [24] is a 26-item questionnaire that assesses ED cognitions and behaviors. Binge eating was assessed by one item: “I have gone on eating binges where I feel that I may not be able to stop,” which was scored on a six-point scale ranging from 1 (never) to 6 (always).

Kiddie Schedule for Affective Disorders and Schizophrenia—Present and Lifetime Version (KSADS-PL) [25] is a semistructured interview that assessed depressive symptoms at age 16. “Skip rules” were not enforced, so all nine depression items were assessed. Consistent with previous studies [26], responses were rated using a three-point scale (1 = not present, 2 = subthreshold, and

¹ Reasons for exclusion included <80% striatum coverage ($n = 13$), >25% of volumes with movement of >3 standard deviations from the participant's mean, >.5-mm scan-to-scan translation or >.01° of scan-to-scan rotation ($n = 5$), poor quality scan ($n = 1$), incidental findings ($n = 1$), or failure to respond on >80% of the trials, incomplete data, or inappropriate response timing ($n = 6$).

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