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Title

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ISSN

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Publication Date

DOI

Associations Between Neural Reward Processing and Binge Eating Among Adolescent Girls

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Abstract

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 understanding of whether reward-system alterations increase vulnerability to binge eating, as theorized in models of the development of this behavior.

Method—Adolescent girls (n=122) completed an fMRI 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 BOLD response to anticipating and winning monetary rewards and severity of binge eating while controlling for age 16 depressive symptoms and socioeconomic status (SES).

Results—Greater ventromedial prefrontal cortex (PFC) and caudate response 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-SES adolescent girls. Ventromedial PFC response to reward outcome—possibly reflecting enhanced subjective reward value—appears to be a state marker of binge eating severity rather than a predictor of future severity.

Keywords

adolescents; functional magnetic resonance imaging; binge eating; reward; disordered eating

Binge eating is the consumption of an unambiguously large amount of food while simultaneously feeling 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 heightened neural response to food receipt may influence 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 while anticipatory reward (e.g., reward from *cues* associated with consumption) increases over time, which may exacerbate overeating episodes. Despite 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 (ED) have demonstrated increased activation in the ventromedial prefrontal cortex (vmPFC)[4,5] or medial orbital frontal cortex (OFC),[5,6] insula,[6] anterior cingulate cortex (ACC),[6,7] posterior cingulate cortex,[5] and middle frontal gyrus (MFG).[8] Many of these regions have been implicated in processing emotional and motivational information, including reward valuation,[9,10] suggesting increased food reward sensitivity in individuals with binge eating. However, some studies show no differences between individuals with or without binge eating on neural response to 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 eating. When given food, women with full or subthreshold BN have exhibited decreased activation in the insula,[11,14] precentral gyrus,[11] MFG,[11] thalamus,[11] lateral OFC,[14] and 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 with binge eating exhibit other reward-related or impulsive behaviors[16] and greater self-reported reward sensitivity compared to controls.[17] Behavioral evidence suggests increased overall reward valuation in individuals with BED[18] and greater sensitivity to monetary gains in individuals with BN,[19] supporting increased response to food and non-food rewards in these populations.

Few studies have examined neural reward function using non-food reward cues in individuals with binge eating. Compared to both overweight and healthy weight non-binge 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 binge eating.[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, who have broader variability in symptoms than clinical groups in traditional case-control studies and (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 non-food rewards would be positively associated with binge eating severity at baseline and two years later.

Method

Participants

Participants were 122 adolescent girls from the Pittsburgh Girls Study -- Emotions Substudy (PGS-E),[22] recruited from the Pittsburgh Girls Study (PGS; $N = 2450$).[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 nine for PGS-E ($N = 232$),[22] and both completed annual assessments from ages five to 18 years. Girls completed an fMRI scan for the first time at age 16. Of 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.

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[1]Reasons for exclusion included < 80% striatum coverage ($n = 13$), > 25% of volumes with movement > 3 standard deviations from the participant's mean, > 0.5 mm scan-to-scan translation or > 0.01 degrees of scan-to-scan rotation ($n = 5$), poor quality scan ($n = 1$), incidental findings ($n = 1$), or failure to respond on >80% of trials, incomplete data, or inappropriate response timing ($n = 6$).

Measures

Eating Attitudes Test (EAT)[24] is a 26-item questionnaire that assesses eating disorder 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" that 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 semi-structured 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, 3=threshold). The interview was administered to the girls and their mothers separately, and a symptom was considered present if endorsed at threshold by either informant. The number of symptoms endorsed was summed to create a depressive symptoms score. Symptoms were included as a covariate, given associations with binge eating and reward.

Body mass index (BMI) was assessed via interviewer-measured height and weight and calculated as weight (kg)/height (m^2). Age 16 BMI was considered as a covariate, given associations with binge eating and reward.[3]

Receipt of public assistance (i.e., Women, Infants, and Children [WIC], food stamps, or welfare) was used to determine low socioeconomic status (SES) and considered as a covariate given PGS sampling procedures and associations with psychopathology.

Reward-Guessing fMRI Task

Neural response to reward was assessed using an event-related reward-guessing task during an fMRI scan.[27] Participants were presented with an image of a blank card and instructed to guess whether the card value (possible values=1–9) was greater or less than five. At each trial, a blank card was presented and participants had 4 seconds to make their guess (via button press). Next, hands shuffling cards were displayed for 6 seconds along with the trial type; an upward arrow indicated potential reward ($1) for a correct guess and a downward arrow indicated potential loss (50 cents) for an incorrect guess. If participants "guessed incorrectly" on a potential reward trial or "guessed correctly" on a potential loss trial then the outcome was neutral (no win or loss of money). The trial-type cue was followed by the "actual" card value (500 msec) and feedback on the outcome of the trial (an upward green arrow for win, downward red arrow for loss, and yellow circle for no win/loss) (500 msec). The trial ended when a crosshair appeared (9 seconds). The task was eight minutes long, with 24 trials (20 seconds each). Trial order was pseudorandom and outcomes were predetermined with 12 potential reward and 12 potential loss trials resulting in 6 "wins," 6 "losses," and 12 "no win/loss" outcomes. Participants were told that their research compensation would be determined by the outcomes of their guesses, and all believed this deception. Participants received $10 for participation.

Neuroimaging Acquisition, Processing, and Analysis

Neuroimaging was conducted on a Siemens 3T TIM Trio scanner. Functional images were acquired using a gradient echo planar imaging (EPI) sequence that included 39 axial slices (3.1 mm wide) beginning at the cerebral vertex and extending across the entire cerebrum and most of the cerebellum (TR/TE=2000/28 ms, field of view=20 cm, matrix=64×64). Scanning parameters were selected to optimize blood-oxygenation-level dependent (BOLD) signal quality while maximizing whole brain coverage. A reference EPI scan was conducted before fMRI data collection to inspect for artifacts and ensure adequate signaling across the whole brain. A 160-slice high-resolution sagittally acquired T1-weighted anatomical image also was collected for co-registration and normalization of functional images (TR/TE=2300/2.98 ms, field of view=20 cm, matrix=256×240).

Preprocessing and imaging analyses were conducted with Statistical Parametric Mapping software (SPM8; http://www.fil.ion.ucl.ac.uk/spm). Preprocessing included: motion correction, slice timing correction, co-registration, spatial normalization (Montreal Neurological Institute space), and smoothing with Gaussian filter (6 mm full-width half-maximum). Artifact Detection Toolbox (http://www.nitrc.org/projects/artifact_detect/) was used to censor functional volumes with movement > 3 standard deviations from the participant's mean, > 0.5 mm scan-to-scan translation, or > 0.01 degrees of scan-to-scan rotation. Preprocessed data were inspected prior to second-level analysis to ensure all participants had good whole brain coverage, 80% coverage of ventral striatum, and < 2 mm or 2 degrees average movement in any direction during the scan.

Second-level random effects models were used to estimate neural response to reward anticipation and outcome, accounting for scan-to-scan and between-participant variability. For each participant, condition effects were calculated at each voxel using paired t-tests for reward anticipation > baseline and reward outcome > baseline.[2] Reward anticipation was defined as the 12 potential-win intervals that included the 6-second potential-win arrow cue. Reward outcome was defined as the intervals that included the presentation of the "actual" card value (500 msec), arrow feedback (500 msec), and the first 6 seconds of fixation. Baseline was defined as the last 3 seconds of all 24 trials (i.e., during fixation).

Based on prior studies utilizing this task and emphasis on reward circuitry in the current study, analysis of imaging data focused on a composite region-of-interest (ROI) mask of eight (bilateral) reward-related regions, including the striatum, mPFC, OFC, and amygdala.[26–28] This ROI was defined using PickAtlas 3.0.3 (http://fmri.wfubmc.edu/software/PickAtlas) and contained 23,190 voxels. The striatum was defined as a sphere with 20 mm radius centered on the Talairach coordinates x=0, y=10, z=−10. The mPFC was defined as a sphere with 25mm radius centered on the Talairach coordinates x=0, y=42, z=18, which primarily included BA32, dorsal/rostral BA24, medial regions of BA9 and BA10, and medial frontal gyrus. The OFC was defined as BA47 and BA11 with a dilation=2, and the amygdala was defined using the human PickAtlas label of the amygdala. To control for type I error, $p < .001$ was used in SPM, and $p_{FWE} < .05$ was used to evaluate

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[2]"No win/loss" outcomes reflect disappointment and relief, respectively, and are not likely to be affectively neutral. Thus, reward anticipation and outcome were contrasted with "baseline" as opposed to the no-win no-loss outcomes.

significant clusters.[29] GingerALE[30] was used to convert coordinates from MNI to Talairach spaces.

Average mean BOLD response beta values across each significant cluster were extracted using the "eigenvariate" tool in SPM. Statistical analyses were performed in SPSS. Independent samples t-tests were conducted to compare individuals with binge eating versus those without on neural response to reward. Correlational analyses were conducted to examine associations between neural response to reward and binge eating severity in the full sample and subsample of individuals who endorsed binge eating.

Results

Participant Characteristics

Descriptive statistics appear in Table 1. Approximately 3.3% of the sample at age 16 (n=4) and 5.7% of the sample at age 18 (n=7) were considered high-risk for clinically significant eating disorder symptoms,[31] which is lower than some community-based samples.[32] About 23% of the sample at ages 16 (n=28) or 18 (n=29) endorsed binge eating. There was moderate-high stability in binge eating over the two years (r=.45, p< .001). Fifteen participants endorsed binge eating at both time points. Of those who denied binge eating at baseline (n=92), 13 endorsed binge eating at age 18.

Approximately 5.7% of the sample (n=7) endorsed subthreshold or clinical levels of depressive symptoms (i.e., 4 or more symptoms) at age 16. Compared to those without, individuals with baseline binge eating had more depressive symptoms (t=2.17; p<.04). Baseline depressive symptoms were associated with greater binge severity at ages 16 and 18 (Table 2); however, the prospective association was not significant after accounting for baseline binge eating (β=.07, p=.44). Baseline public assistance, but not race or BMI, was associated with binge eating (Table 2). Thus, baseline depressive symptoms and public assistance were included as covariates. Baseline binge eating was added as a covariate in prospective analyses to account for symptom stability.

Neural Response to Reward Anticipation and Outcome

ROI results with family-wise-error corrected significant clusters appear in Table 3. Within the full sample, reward anticipation elicited response in the dorsolateral PFC, ventrolateral PFC, dorsomedial PFC, rostral ACC, caudate, and VS. Reward outcome elicited response in the ventrolateral PFC, dorsomedial PFC, vmPFC, and caudate.

Neural Response to Reward Anticipation and Associations with Binge Eating

No differences were found on neural response to reward anticipation between individuals with or without binge eating (all ps > .08).

Among the full sample, greater response in the caudate during reward anticipation was associated with binge eating severity both concurrently and prospectively (Table 3). Neither concurrent (β=.13, t=1.44, p=.15) nor prospective (β=.11, t=1.19, p=.24) associations remained significant after controlling for covariates.

Among the binge eating subsample, greater caudate response during reward anticipation was associated with binge eating severity at age 18, but not after accounting for covariates (β=. 32, t=1.64, p=.11).

Neural Response to Reward Outcome and Associations with Binge Eating

Compared to participants without binge eating, those with binge eating demonstrated greater caudate response to winning money (t=2.10; p=.04). No other group differences were found in neural response to reward receipt.

In correlational analyses, greater vmPFC response during reward receipt was concurrently associated with binge eating severity in the full and binge eating subsample (Table 3; Figure 1). These associations remained significant after controlling for depressive symptoms and public assistance (β=.26, t=2.96, p=.004 [full sample]; β=.60, t=3.71, p=.001 [binge sample]). Among the binge eating subsample, greater caudate response to reward receipt also was concurrently associated with binge eating severity after accounting for covariates (β=.39, t=2.09, p< .05) (Table 3).

There were no significant prospective associations between neural response to reward receipt and binge eating in the full or subsample.

Discussion

This study is the first to examine concurrent and prospective associations between neural correlates of reward processing and binge eating in a large sample of community adolescents, which is essential for understanding processes involved in ED development. Greater BOLD response in the vmPFC to winning money was concurrently associated with binge eating severity, even after accounting for depression and public assistance. We also found concurrent and prospective correlations between binge eating severity and VS and caudate response to anticipating a reward, but not after controlling for covariates. Findings suggest that alterations in vmPFC response to reward receipt may impact binge eating during adolescence, but co-morbidity or symptom stability play greater roles in the persistence of binge eating over time.

The vmPFC has been implicated in emotion regulation as well as response to reward delivery and events that predict reward outcomes, particularly value-related information.[33] Damage to the vmPFC in humans has been linked to deficits in value-based decision making,[34] and this region has been implicated in other psychiatric disorders comorbid with binge eating.[35,36] Taken together, our finding that greater vmPFC response to winning money was associated with binge eating suggests that subjective appraisal of rewards may influence binge eating in adolescents. To our knowledge, no studies have noted increased activation in this region in response to reward outcome in individuals with binge eating. However, the significant cluster in the current study overlapped with portions of the ACC (i.e., BA 32), and one prior study observed heightened ACC response to milkshake consumption during a negative mood state in emotional overeaters.[37] Perhaps adolescents who are more sensitive to pleasant experiences are more likely to engage these regions when experiencing reward, reflecting difficulty managing excessive reward-seeking behavior

related to eating. Indeed, behavioral studies show greater reinforcing value of food in individuals with BN compared to controls,[38] but the current study extends previous research to non-food rewards. No prospective associations between vmPFC activation and binge eating were found, suggesting that greater reward value of stimuli may be a correlate of binge eating, but not a predictor of worsening symptoms over time.

This concurrent association between increased vmPFC response to winning money and binge eating is inconsistent with the incentive sensitization theory and prior studies in adult clinical samples demonstrating decreased activation in response to food consumption. These discrepancies are likely related to differences in the developmental timing (adolescents) and clinical severity of the samples. Adolescents have heightened reward responding compared to children and adults, resulting from an imbalance between early maturing motivational and emotional neural systems (e.g., reward network) and slower maturing inhibitory control systems.[39] Thus, high sensitivity to reward receipts may be associated with binge eating during adolescence, with a shift to anticipatory processing as the behavior becomes more ingrained in adulthood. Moreover, stronger correlations between binge eating severity and vmPFC and caudate response to reward receipt within the binge eating subsample in the current study suggest that heightened sensitivity to reward outcomes may be a particularly relevant correlate of binge eating during adolescence.

Furthermore, use of a generic monetary reward task versus a food-specific task may have contributed to our pattern of findings. Although we found associations between binge eating severity and increased vmPFC activation in response to monetary reward receipt, prior neuroimaging studies in BN and BED found increased activation in the mOFC in response to food pictures,[4–6] which typically reflect anticipatory processing (versus reward outcome processing). The hypothetical nature of winning money in the current study may be more similar to viewing food pictures than to consuming palatable food. Additionally, individuals with EDs may have differential response to food versus other rewards; findings of altered reward response are stronger for food rewards than for generic rewards (e.g., monetary).[2,5] Simon and colleagues (2016) found significant differences between individuals with BN or BED and healthy controls in neural response to food cues but not monetary reward. Similarly, a meta-analysis on behavioral performance on inhibitory control tasks in binge-type EDs noted large effect sizes for inhibitory control impairments to food versus small effects for inhibitory control impairments to general stimuli.[40] The use of secondary rewards has key benefits, including greater transdiagnostic relevance that could inform mechanisms underlying comorbidity and the reduction of confounding symptoms (e.g., anxiety) potentially elicited by food-related stimuli. Given benefits of using general stimuli to examine reward processing but larger effects found for disease-salient stimuli, future studies should contrast food and non-food rewards to improve understanding of associations between reward processing and binge eating in adolescents.

Strengths and Limitations

Strengths of the study include the large sample size, longitudinal design, and focus on community adolescents, who are within the peak age of BN onset and have a range of severity. Use of a well-validated fMRI measure of reward anticipation and receipt also was a

strength. However, the hypothetical nature of winning money makes it difficult to compare current findings to those from studies that examined actual receipt of palatable food; future studies would benefit from inclusion of both food and non-food reward cue conditions. Given the task design, we were unable to compare responses of reward outcome to a neutral outcome condition. Furthermore, the original study did not control for menstrual status and did not assess the hunger state of participants prior to the imaging scan, which could influence brain response to reward. Our use of the EAT versus an interview measure for the assessment of binge eating is another limitation. A more objective measure of binge eating, including binge eating frequency is ideal. Finally, most individuals high in binge eating at age 18 already were exhibiting binge eating by age 16, which likely decreased our power to predict changes in symptoms over time.

Future Directions

In sum, this study adds to the growing literature on reward in binge eating and alludes to age-related differences in associations between reward responding and binge eating. Improved understanding of the nuances of both food and non-food reward processing in the development of binge eating has the potential to inform early intervention; future studies, especially in younger adolescents, are warranted.

Acknowledgments

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Implications and Contribution

Binge eating is a prevalent behavior in youth and 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.
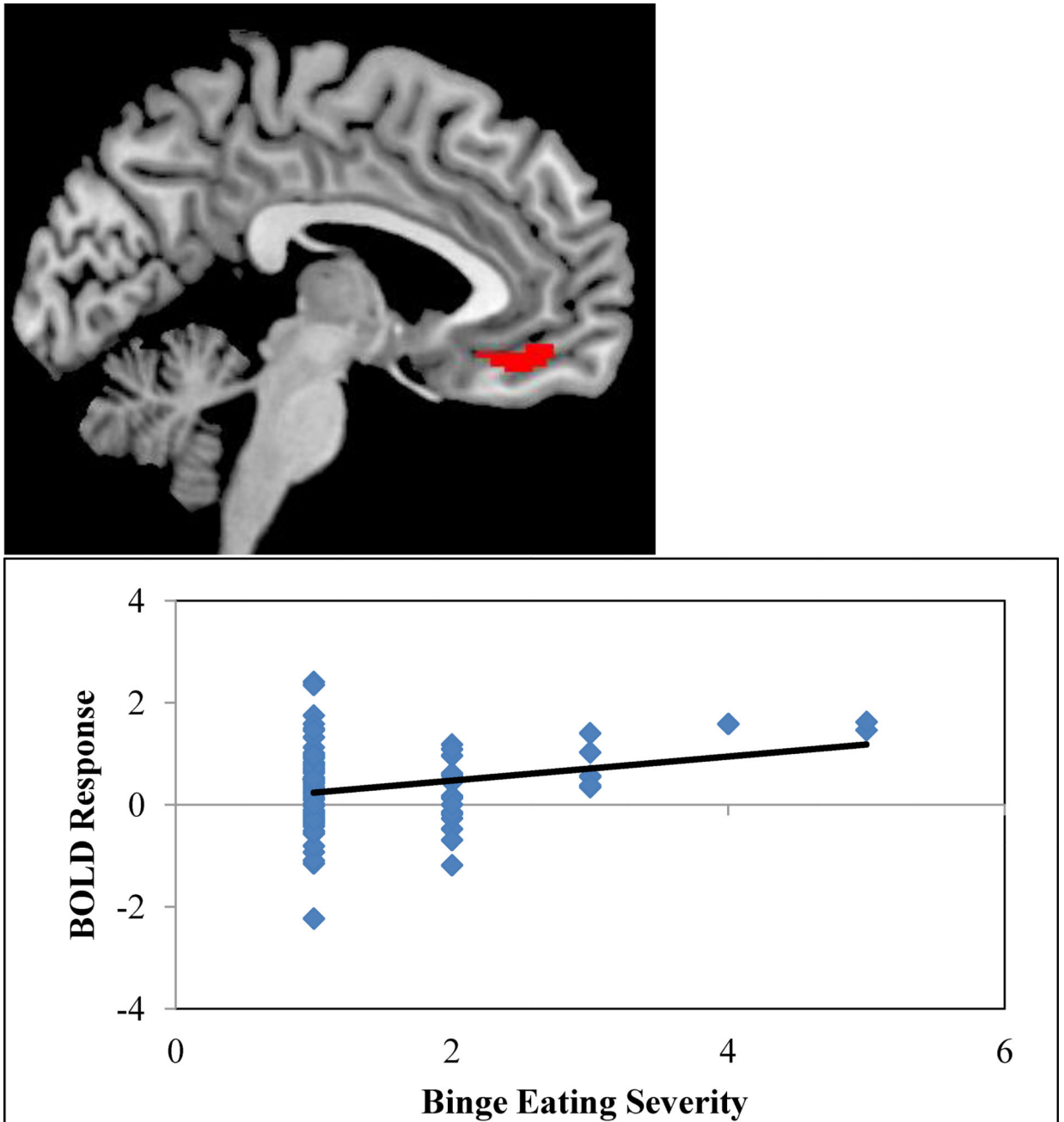
Figure 1.
Positive associations between age 16 binge eating and ventromedial prefrontal cortex blood-oxygenation-level-dependent (BOLD) signal response to winning money in the full sample ($r = .3$; $p < .01$).

Table 1

Descriptive statistics

Variable	Mean	SD	Range
Binge Eating Age 16	1.35	0.76	1–5
Binge Eating Age 18	1.48	1.06	1–6
Body Mass Index Age 16	26.45	6.67	15.34–47.37
Body Mass Index Age 18	27.27	7.09	16.36–49.42
Depressive Symptoms Age 16	1.17	1.35	0–8
	Frequency % (n)		
Receipt of Public Assistance Age 16	43.1% (53)		
Race			
Black/African-American	65% (80)		
White/Caucasian	26.8% (33)		
Multi-Race	8.1% (10)		

Note. SD=standard deviation; receipt of public assistance reported as percentage of families receiving public assistance (number of participants)

Table 2

Intercorrelations between study variables

Variable	1	2	3	4	5	6
1. Binge Eating Age 16	1.00	--	--	--	--	--
2. Binge Eating Age 18	.45 **	1.00	--	--	--	--
3. Body Mass Index Age 16	-.01	-.04	1.00	--	--	--
4. Body Mass Index Age 18	.01	-.10	.92 **	1.00	--	--
5. Depressive Symptoms Age 16	.25 **	.23 *	.12	.14	1.00	--
6. Public Assistance	.19 *	.17	.11	.10	.13	1.00

Note.

*
$p<.05$;

**
$p<.01$

Table 3

Reward anticipation- and outcome-related positive neural activity clusters across full sample and correlations with binge eating frequency at ages 16 and 18 years within full and binge-eating subsample

Region	Hemisphere	Talairach Coordinates			Cluster size	t	Full Sample r		Binge Eating Sample r	
		x	y	z			Age 16	Age 18	Age 16	Age 18
Reward Anticipation										
dlPFC	Left	−45	38	4	720	10.82	0.10	0.07	0.26	0.14
vlPFC	Right	42	43	4	758	9.88	0.10	0.10	0.24	0.03
dmPFC, rostral ACC	Left	−3	32	43	1280	8.52	0.02	0.06	−0.02	−0.03
Caudate Body, Caudate Head, VS	Right	6	13	9	191	6.95	0.20*	0.25**	0.22	0.33*
Reward Outcome										
vlPFC	Left	−47	17	2	601	9.12	0.02	−0.07	−0.09	−0.24
vlPFC, Anterior Insula	Right	36	18	−8	448	7.78	0.13	0.07	0.14	−0.05
dmPFC	Left	−3	34	40	3260	7.69	0.01	0.05	0.03	0.13
vmPFC, BA32	Right	3	35	−3	205	5.30	0.25**±	0.12	0.61**±	0.23
Caudate Body, Caudate Head	Left	−10	2	19	170	5.51	0.18*	0.15	0.40*±	0.09

Note. ACC=anterior cingulate cortex, BA= Brodmann's Area, dlPFC = Dorsolateral Prefrontal Cortex, dmPFC = Dorsomedial Prefrontal Cortex, vlPFC = Ventrolateral Prefrontal Cortex, vmPFC=Ventromedial Prefrontal Cortex, VS=ventral striatum;

*
p<.05; r age 16= bivariate correlation with binge eating severity at age 16; r age 18= bivariate correlation with binge eating severity at age 18;

±
= association remained significant after accounting for age 16 depressive symptoms and receipt of public assistance (coded as 0=no assistance, 1=received public assistance).