Neural mechanisms that promote food consumption following sleep loss and social stress: An fMRI study in adolescent girls with overweight/obesity

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Abstract

Study Objectives: Insufficient sleep and social stress are associated with weight gain and obesity development in adolescent girls. Functional magnetic resonance imaging (fMRI) research suggests that altered engagement of emotion-related neural networks may explain overeating when under stress. The purpose of this study is to explore the effects of acute sleep restriction on female adolescents' neural responding during social evaluative stress and their subsequent eating behavior.

Methods: Forty-two adolescent females (ages 15-18 years) with overweight or obesity completed a social stress induction task in which they were told they would be rated by peers based on their photograph and profile. Participants were randomly assigned to one night of sleep deprivation or 9 hours of sleep the night before undergoing fMRI while receiving positive and negative evaluations from their peers. After which, subjects participated in an *ad libitum* buffet.

Results: Sleep deprived, relative to non-deprived girls had distinct patterns of neural engagement to positive and negative evaluation in anterior, mid, and posterior aspects of midline brain structures. Moreover, a sleep deprivation-by-evaluation valence-by-caloric intake interaction emerged in bilateral dorsal anterior cingulate. Among sleep deprived girls, greater engagement during negative, but not positive, feedback was associated with lower caloric intake. This was not observed for non-sleep deprived girls.

Conclusions: Results suggest an interaction between acute sleep loss and social evaluation that predicts emotion-related neural activation and caloric intake in adolescents. This research helps to elucidate the relationship between sleep loss, social stress, and weight status using a novel health neuroscience model.

Key words: Sleep Duration, Food, Stress, Adolescents, Brain, Obesity

Statement of Significance

This study is the first to examine the relationship between sleep loss, social evaluation, and weight status in adolescents using functional magnetic resonance imaging (fMRI). We found that among sleep-deprived adolescent girls with overweight or obesity, social evaluation impacts emotion-related neural activation and subsequent caloric intake. Thus, body image concerns and sleep loss, both of which prevalent are in adolescent girls, should be considered interactively to understand the relation between social evaluation and food consumption. Our results underscore that the critical, yet nuanced, association between weight status and sleep should be considered in the design of interventions tailored for this at-risk population.

Introduction

Sleep is an important behavior that affects numerous domains of adolescent health and development, including weight status, mood, attention processes, and neural development[1]. Sleep is particularly important in adolescence because it influences health risk- and reward-related behavior[2]. The majority of adolescents do not obtain sufficient sleep[3]. Furthermore, research suggests that insufficient sleep is associated with obesity development in adolescent girls[4], an association which may result from alterations in eating behavior[5]. Indeed, some studies have shown that sleep restriction is correlated with increased caloric consumption[6,7].

Social stress and perceived social rejection can also increase risk for overeating and preference for high-calorie foods[8,9]. A potential mechanism driving these changes in eating may be that interpersonal distress overrides homeostatic processes that regulate appetite and food consumption[10]. Several studies have suggested that altered engagement of anxiety- and stress-related neural pathways during social provocation may partially explain interpersonal distress-related overeating[11-12]. For example, aberrant activity in striatal and corticolimbic circuits during social evaluation in adolescents has often been linked to poor regulation of emotions, attention, and behavioral responding[13]. Cortical regions along the brain's midline, and specifically the cingulate cortex, are commonly implicated in social evaluation. For example, engagement of aspects of the posterior cingulate cortex (PCC)[14] and anterior cingulate cortex (ACC) have been linked to processing negative social experiences[15]. While greater engagement of the mid ACC during social rejection is associated with heightened distress, greater perigenual ACC engagement has been implicated in regulating behavioral and physiological reactivity to psychosocial stress[16]. Thus, alterations in anxiety- and stress-related neural pathways during social interactions may allow for more emotional response and poorer regulation of emotions,

which may, in turn, lead to overeating to cope with such feelings. In this way, interpersonal distress may contribute to the positive energy balance that promotes weight gain in overweight and obese adolescents.

A social stress model of overeating suggests that maladaptive neural processes limit an individual's ability to regulate distress associated with social evaluation[17]. Overweight and obesity in adolescence may, in turn, promote social stress, which potentiates patterns of eating that maintain elevated weight. For example, greater body mass increases risk for social stressors such as bullying-based victimization, which is associated with weight gain[18] and social consequences of failure to meet expectations of traditional standards of beauty[19,20]. While both males and females can be impacted by weight-related bullying, older adolescence is a critical period for bullying in females that puts them at risk for weight gain and obesity[21]. Bi-directional associations between social stress-related cognitive processes and risk for negative social interactions related to weight status are important in conceptualizing the social contextual factors leading to development and maintainance of obesity.

Finally, sleep has also been implicated in emotion processing and regulation. Poor sleep co-occurs with nearly all mood- and anxiety-related disorders[22], and is a critical target behavior for physical and emotional health. Acute and chronic sleep loss also modulate one's capacity to respond to life stressors, including social stressors such as negative evaluation and rejection[23]. For example, brain regions implicated in memory and emotion systems, such as the amygdala, striatum, and hippocampus, can be hypersensitive to positive or negative stimuli after sleep deprivation[24–26]. Thus, the lack of sleep is associated with both heightened emotional saliency and differential brain processes. To this point, the dorsal ACC and thalamus, which are associated with emotional processing and physiologic responding, respectively, have also shown

altered engagement after sleep restriction[27]. This suggests that sleep can alter neural systems and thereby impacting emotion processing, which could make someone more susceptible to emotional distress.

Our study aims to merge these three lines of evidence to explore the effects of sleep restriction on adolescent girls' emotion-related neural response to social evaluative threat and subsequent caloric intake. We studied female adolescents with overweight or obesity, as this is a high-risk group for stress related to social evaluation. Social evaluation was simulated using the "chatroom task"[28], where participants receive unpredictable positive and negative feedback from purported peers who had rated the participant based on their photograph and a personal profile – a context that engenders social evaluative stress in youth. This enabled us to examine how sleep impacts neural activation while experiencing peer evaluation.

To our knowledge, the association between sleep deprivation and neural responding during social evaluative stress has yet to be explored in adolescents, and neither have the combined elements of sleep duration, neural responses during social evaluative stress, and caloric intake has not been previously examined. Therefore, this study enabled us to test several novel research questions relevant to the aforementioned social stress model of overeating. First, we hypothesized that adolescent girls who were deprived of sleep, versus those who slept for 9 hours the night prior to fMRI, would demonstrate more activity to the social evaluative context in brain regions implicated in emotion processing and physiological responding (i.e., bilateral amygdala, thalamus, cingulate cortex, hippocampus, and striatum)[29]. Next, we hypothesized that adolescent girls with sleep deprivation, relative to habitual sleep, would demonstrate greater engagement in the same emotion processing network during negative compared to positive feedback from peers. Finally, we hypothesized that adolescent girls with sleep deprivation,

relative to habitual sleep, would demonstrate a stronger association between neural responding during negative social evaluation (i.e., rejection) and subsequent caloric consumption.

Methods

Participants

Forty-two adolescent girls ages 14 to 18 years with overweight or obesity (*M* age = 16.48, *SD* = 4.73; see Table 1 for demographic summary) were included in our analyses. Seven participants had to be excluded from our analyses due to excessive movement during the scan where the data could not be appropriately analyzed. Participants were recruited using advertisements in local schools. Exclusion criteria included use of weight-loss or sleep medication, history of bariatric surgery, use of medications that affect salivation, previously diagnosed eating disorders, left-handedness, history of mental or psychiatric conditions, food allergies, and standard fMRI contraindications. Verification of overweight or obese status (BMI ≥ 25) was accomplished by measuring height with a standard stadiometer (Seca model number 217) and weight using a digital scale (Seca model number 813) then calculating body mass index percentile for age and sex following United States Centers for Disease Control and Prevention guidelines [30]

Experimental Design and Procedure

The Institutional Review Board of the first author's academic institution approved all study procedures. The data underlying this article will be shared on reasonable request to the corresponding author. At the first study visit, written parental permission and adolescent assent were obtained. Parents were aware of deception (i.e., participants would not truly be chatting with anyone online) from the onset of the study. Next, the first phase of a two-visit well-established chatroom task developed by Guyer and colleagues[28] which has been validated in

adolescent girls with overweight or obesity[31] was conducted. In this task, teens were told that they would be making judgments about their peers, and that their peers would do the same about them; this approach was taken to increase the salience of purported social evaluation. Participants viewed pictures of 60 purported male and female peers and identified 30 they were interested in chatting with and the 30 they were not interested in chatting with. Next, each participant was photographed and completed a personal interest profile and were led to believe that the same 60 peers they just rated would judge their own desirability, and that in instances in which both parties were "interested," the adolescent would have the opportunity to chat with the other teen.

Additionally, participants were randomly assigned to either one night of total sleep deprivation (no sleep) or nine hours in bed (habitual sleep) on the night immediately preceding the second lab visit. Nine hours was determined as the control/habitual condition based on previous research demonstrating that this is the ideal number of sleep hours for adolescents[32]. Participants received a phone call from study staff on the night preceding the second lab visit to remind them to adhere to the sleep protocol. Participants received an accelerometer (Actigraph GT3x+) at the first study visit and were instructed to wear the device around their waist for 24 hours preceding the second assessment as a validity check for sleep duration. Parents and participants were also informed that the participant was expected to fast for 4 hours prior to the second visit, to control for hunger levels across participants before administration of the food buffet. Moreover, because sleep duration alters appetitive hormones that regulate morning food intake[33] this second visit was scheduled between 7:00 AM and 12:00 PM. Lastly, participants were asked to refrain from consumption of caffeine after 2:00 PM the day before their scanning appointment.

The second study visit occurred 4 to 7 days after the initial lab visit. Participants first underwent a structural MRI scan (7 mins), and then completed the chatroom task while undergoing fMRI. In the first run of the task (~9 mins), participants were shown photographs of the previously rated peers and were asked to rate how interested they thought the peers were in chatting with them. During the second run of the task (~12 mins), teens were reminded if they were "Interested" or "Not Interested" in each peer prior to receiving "Interested" or "Not Interested" feedback from each purported peer. There was a variable duration jitter (0-8000 ms) during which a fixation cross was displayed in between each feedback trial. Feedback was standardized across participants (i.e., 50% "Interested", 50% "Not Interested" for peers that participants wanted to chat with and the same for those they did not). Because we were interested in the response to social evaluative stress generated by the chatroom task, analyses for the current study focus on the second functional run obtained during purported social evaluation (see Figure 1). Total scanning time was approximately 30 minutes.

Immediately after their scan, an *ad libitum* test breakfast was provided to participants in a room occupied by only the participant, devoid of any phones or distracting items. Each participant was presented with a large array of breakfast foods and beverages (e.g., muffins, yogurt, fresh fruit, bagels, granola, cereal, milk, and orange juice) and were invited to eat or drink as much as they desired. The researcher returned after 20 minutes. Food was weighed to the nearest gram before and after the meal and total caloric intake was determined using the food packaging nutritional information. *Ad libitum* food buffets have been shown to be reliable and valid assessments of eating behavior[34].

Participants completed a debriefing questionnaire at the end of the experiment that asked: "While you were doing the chatroom task, did you believe that you would really be chatting with

one of these people on the computer after your scan?" and were asked to respond by circling "yes" or "no". Finally, each participant was debriefed by a research assistant regarding the deception involved in the chatroom task using a script. Seventy-six percent of the participants responded "yes" they did believe they would actually be chatting with a peer. Results remained largely the same when non-deceived participants were excluded from analyses.

Sleep Adherence. Accelerometry was used to corroborate time in bed for participants in the habitual sleep group. Waist-worn accelerometers accurately detect total sleep time and total time in bed (with a sensitivity of 98.8-99.7%), but are less sensitive to sleep disturbances than wrist-worn accelerometers (with a specificity of 29.8-46.9%)[35,36]. Waist-worn accelerometers have shown to have poorer concurrent validity with polysomnography relative to wrist-worn accelerometers[37]. Because of these limitations, we only examined the parameter "total time in bed" as a measure of adherence. If no movement was recorded during the night, it was assumed that the accelerometer was removed prior to bedtime and the data were excluded. Total time spent in bed was determined with ActiLife5 software using Sadeh and colleague's[38] sleep scoring algorithm. Participants in the no sleep group were considered adherent if there was sufficient movement during the night and no total sleep time was recorded. All participants were also asked to verify sleep protocol adherence verbally before scanning procedures began.

Two research assistants coded the accelerometry data and separately determined whether or not the participant was adherent. Total sleep time for the habitual sleep group was averaged across the two coders and there was no disagreement between coders on the overall adherence of participants. In the no sleep group, one participant's accelerometry data were corrupted and could not be analyzed but this participant's data were still included based on self-reported adherence. The other participants were deemed adherent. In the habitual sleep condition, one

participant's accelerometry data were corrupted and could not be analyzed but this participant's data were still included based on self-reported bedtime and wake time.

Food Reward Sensitivity. The Power of Food Scale (PFS) assesses general trait sensitivity to food reward[39] as a potential covariate. This measure was administered to all participants at the baseline appointment. The PFS is a measure of appetite-related thoughts, feelings, and motivations toward palatable foods. The PFS comprises three factors: food tasted, food available, and food present[39,40]. Questions include "If I see or smell a food, I get a very strong desire to have some," or "Just before I taste a favorite food, I get very excited." The PFS has been shown to have excellent internal consistency in adult (α = .91; [40]) and adolescent (α = .86–.95; [41]; α = .93-.95; [42]) samples. Four-month test–retest reliability is good (r = .77; [40]). Total scores on the PFS between healthy and no-sleep groups were significantly different from each other, in that those in the healthy sleep condition had significantly higher scores than those in the no-sleep condition (p = .02). Thus, the total PFS score was included as a covariate of no interest in all fMRI analyses.

MRI Data Acquisition. All neuroimaging was conducted using a Siemens TIM Trio 3T MRI scanner utilizing a 12-channel head coil. We collected T2*-weighted functional data during the chatroom task using an echo planar imaging sequence with the following parameters: slices = 48 interleaved, TE = 25 ms, TR = 2500 ms, field of view = 240 × 240 mm^2, acquisition matrix = 80 × 80, slice thickness = 3 mm, voxel size = 3 × 3 × 3 mm^3, flip = 90°. Additionally, we collected a T1-weighted structural brain scan for functional localization using a magnetization-prepared rapid acquisition with gradient echo (MP-RAGE) sequence with the following parameters: slices = 62 interleaved, TE = 2.26 ms, TR = 1900 ms, field of view= 218 × 250 mm^2,

acquisition matrix = 215 × 256, slice thickness = 1 mm, voxel size = 0.97 × 0.97 × 1 mm^3, flip = 9°.

MRI Data Processing and Analysis

DICOM data were converted to NIfTI format using dcm2niix (Li et a., 2006). All other processing of structural and functional MRI data was conducted using Analysis of Functional NeuroImages (AFNI[43]). The functional data were transformed into Montreal Neurological Institute (MNI) standardized space as the function of three sets of calculations. First, the single volume of each participant with the least number of outlier voxels was used as a registration base, and rigid body transformations for each volume to the registration base were computed. Second, the rigid body transformation of the T1-weighted structural scan to the registration base, using a lpc+ZZ cost function, was calculated. Third, the non-linear, diffeomorphic transformation of the structural scan in native space to the MNI template was computed and stored. Concatenating these three sets of calculations, then, allowed the functional data to be moved from native space into MNI using a single interpolation. All subsequent processing of fMRI data occurred in MNI space.

The amount of signal at each voxel for both T1- and T2*-weighted data was assessed with respect to the mean signal, and this assessment was used to construct an extent mask which indicated which voxels had sufficient signal for processing in both structural and functional domains. These subject-specific extent masks were used to constrain analyses, in order to account for signal dropout, and were also applied in group-level analysis. Further, as per AFNI's standard censoring protocols, volumes where >10% of the voxels had outlier signal and/or motion events relative to the previous volume were removed, as was the immediately previous volume. Motion events were defined as volumes with a derivative value greater or less than a

Euclidean Norm of 0.3 were censored, as was the preceding volume given that motion resulting in detectable frame displacement is unlikely to begin at the start of the volume. Participants with >10% of TRs censored were excluded from further analyses. Finally, the functional data were scaled by the mean signal. The number of TRs censored for the no sleep (M = 32.95, SD = 34.60) and habitual sleep groups (M = 38.00, SD = 32.70) did not differ t(40)= -.485, p=0.63. On average, less than 10% of TRs were censored (M = 9.23%, SD = 8.88; min = 0% max =29.35%), there was no difference in the number of TRs censored across any of the six event types (t's < 1.0, p's > .40), and all participants had at least 66 non-censored TRs for any event type.

Individual-Level fMRI Analyses. As is standard for AFNI, single-subject analyses were performed using a regression matrix that included timing regressors as well as the censoring vector. Single-subject regression was accomplished using a Generalized Least Squares (GLS) fit that utilized a nonlinear REML estimate in order to determine the best-fitting autoregressive-moving average (ARMA) model. Nuisance regressors included motion regressors for six degrees of freedom as well as a noise regressor that was indicative of the mean white matter timeseries[44]. Values for white matter time series were derived from the template used for image registration was segmented for tissue class via the Atropos N4 method (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3297199/), generating gray matter, white matter, and CSF masks. The white matter mask was then resampled into functional dimensions and eroded (as the data were already in template space, no warping of the mask occurred). For regressors of interest, all six possible event types were used to model the BOLD signal using a boxcar convolution with the canonical HRF. The six event types included: two separate regressors for Anticipation Period events (Subject Interest: interested/not interested) and four regressors for Peer Feedback events (Subject Interest: interested/not interested; Peer Feedback:

positive/negative). The latter four regressors were used to address the current study aims, with the fixation period used as baseline. Resulting parameter estimates (β–coefficients) were blurred with a 6 mm FWHM Gaussian blur prior to group analyses.

For individual level GLS analyses, there was a standard number of degrees of freedom (DFs) used to account for stimulus events (N=6), slow wave drift (N=8), and motion (N=6). Because there was a variable number of TRs censored for motion, there was a variable number of DFs utilized for censoring (M = 35.17, SD = 33.38). A sufficient number of DFs remained for parameter estimation in the habitual sleep (M = 323.85, SD = 39.38) and no sleep groups (M = 327.82, SD = 37.08). The minimum number of available DFs across participants was 221, and there was no difference in DFs across groups (t(40)=-.34, p=.74). Variability in DFs for individual level GLS analyses is expected, thus each GLS matrix will differ. However, this does not mean that hypotheses were tested with uneven matrices. Indeed, we are following current recommendations for analysis[45], in that we remove varying amounts of noise from the signal at the individual level, and then test different aspects of the signal in subsequent group level analyses as described below.

Group-Level fMRI Analyses.

This paper focuses on neural response elicited by peer evaluation. Because of this, group level analyses only included events that occurred during Peer Feedback. Additionally, we are specifically interested in the neural response associated with peer feedback and the valence of that feedback, not in interactive effects between peer feedback and participant interest on peers. However, some prior studies have demonstrated that participant interest in peers can have an influence on neural responding during peer feedback[30]. Thus, participant interest was included in group level analyses to control for these potential effects. To test our main hypotheses, we

performed a repeated measure ANOVA with the AFNI tool 3dMVM. Specifically, we performed a social evaluation (positive/negative) × sleep condition (no sleep/habitual sleep) × caloric consumption (continuous measure from the *ad libitum* breakfast) with participant interest (interested/not interested) and PFS (continuous measure) included as covariates of no interest.

Group level analyses were constrained to a combined mask of *a priori* regions of interest (ROI) implicated in emotion processing that included the bilateral thalamus, caudate, putamen, nucleus accumbens, hippocampus, amygdala, and cingulate (see Appendix A). This 3,996 voxel mask was constructed in atlas space according to the Desikan-Killiany-Tourville segmentation protocol[46]. Criteria for significance was determined via Monte Carlo simulations generated by 3dClustSim (AFNI), simulations which included parameter estimates of the autocorrelation function derived from single-subject model residuals. These simulations determined that a cluster size (k) of 13 contiguous voxels (NN=1) with a voxel-wise threshold of $p < .005$ resulted in an overall family-wise error rate of $p < .05$ within our ROI mask. To assist in interpreting these results, a single β–coefficient, which reflects the average of values across each surviving cluster, was extracted for each individual. These values were plotted to aid in the interpretation and visualization of main and interaction effects. Secondary descriptive analyses were likewise performed with IBM SPSS Statistics (Version 27.0) to facilitation in the interpretation of results. Specifically, independent sample t-tests were perfomed to assess group differences, single sample t-tests were used to assess differences in activation relative to zero, and paired sample t-tests were used to assess differences across experimental conditions.

To minimize the need to correct for multiple comparisons across numerous analyses, all three hypotheses were assessed within the context of lower order interactions or main effects from the single repeated measure ANOVA, described above. Hypothesis 1 was interrogated via

the main effect of sleep group. Hypothesis 2 was interrogated via the sleep group × social evaluation interaction. Hypothesis 3 was interrogated via the sleep group × social evaluation × caloric consumption interaction.

Results

Adherence to Sleep Condition

For habitual sleep participants, the average total sleep time was 8.56 hours (SD = .98). While the accelerometry data appeared to show variance from the assigned 9 hours (6.42 to 10.41) of total sleep, most participants got at least 8 hours which is within the range of healthy sleep for adolescents[47]. Taking this into consideration along with verbal confirmation of adherence from participants, no participants were excluded from the habitual sleep group on grounds of non-adherence.

Group-Level fMRI Analyses

Main Effect of Sleep Condition (No Sleep/Habitual Sleep) on Brain Activation During Social Evaluation. There was a main effect of sleep condition in the left putamen and in the right hippocampal tail (see Figure 2). Descriptive statistics confirmed a significant difference between groups in the left putamen ($t(40) = 3.17$, $p = .003$) and a difference that trended toward significance in the right hippocampal tail ($t(40) = 2.01$, $p = .052$). Activation of both of these clusters was significantly different from 0 in the no sleep ($t(21) = 5.34$, $p < .001$; $t(21) = 4.58$, $p < .001$, respectively), but not habitual sleep group.

Sleep Condition (No Sleep/Habitual Sleep) x Social Evaluation (Positive/Negative) Interaction. Significant interactions emerged along the medial wall in the left posterior cingulate gyrus, right medial frontal gyrus, and left perigenual ACC (see Figure 3). To determine what was driving these effects, interactions were decomposed and plotted. Independent samples t-tests

revealed a significant difference between groups for activation of the left posterior cingulate gyrus during positive feedback ($t(40) = 4.05$, $p < .001$), such that the no sleep group showed greater activation than the habitual sleep group. A paired samples t-test also revealed that within the no sleep group, there was greater activation to positive compared to negative feedback ($t(21) = 3.17$, $p = .003$). Although decomposition of the other clusters failed to meet traditional statistical significance, they demonstrated the identical pattern as observed in the posterior cingulate.

Sleep Condition (No Sleep/Habitual Sleep) x Social Evaluation (Positive/Negative) x Caloric Intake Interaction. Two significant clusters in bilateral ACC emerged. Decompositions of these interactions revealed they were primarily driven by differences in the no sleep group. Specifically, among adolescent girls in the no sleep group, decreased engagement of the ACC during negative social evaluation was associated with greater subsequent caloric intake. This effect emerged significant in the right midcingulate gyrus ($r = -0.523$, $p = .013$) and trended toward significance in the left midcingulate gyrus ($r = -0.412$, $p = .057$; see Figure 4).

Discussion

This study explored potential differences in neural response to social evaluation among overweight/obese adolescent girls with acute sleep deprivation relative to girls that were fully rested. This study is the first to explore the neural pathways between social evaluation, acute sleep loss, and subsequent caloric consumption. Our study focused on brain regions that are implicated in emotion processing and regulation. We chose these regions to evaluate the neural response while undergoing social evaluation and how this interacted with subsequent eating. Sleep disturbance and stress are positively associated with weight status and eating behaviors in adolescents and adults[6,48], with poor sleep (i.e., short sleep duration) being predictive of

increased risk for overeating and susceptibility to stress. Moreover, poor sleep may exacerbate the imbalance between affective and cognitive control systems in adolescents, which can impact emotion regulation and decision making[2]. Our study merged three critical lines of research (i.e., sleep, social stress, caloric intake) and generally supported the idea that sleep loss is related to differential neural responses to social evaluation as well as eating behavior.

Consistent with our first hypothesis, we found increased neural response to the social evaluative context among sleep deprived, relative to fully rested, adolescent girls. Specifically, greater activity was observed in the left putamen and right hippocampal tail during social evaluation. This supports our hypothesis that sleep duration alone, regardless of the valence of the social evaluation, significantly impacts brain functioning. This finding implies that adolescent girls who are not obtaining sufficient sleep are attending more to social feedback from peers. This is consistent with past literature that has found the putamen, a brain region implicated in motivation and emotion regulation, to be highly sensitive to sleep loss[24]. Additionally, research shows that the hippocampus is related to maladaptive emotional regulation after sleep deprivation[49]. The hippocampus compared to other brain structures implicated in emotion processing may be the most vulnerable to the negative effects of sleep loss[50]. Subsections of the hippocampus have also been found to be more activated while undergoing social stress[51].

Contrary to our second hypothesis, we found that sleep deprivation was associated with greater neural response to positive, rather than negative feedback, compared with habitual sleep. While this effect was most robust in posterior cingulate cortex, a similar pattern emerged in the middle cingulate and subgenual ACC. Generally speaking, sleep loss results in increased activation to both positive and negative stimuli in brain regions implicated in reward and emotionprocessing (e.g., striatum, amygdala, and insula,[24]). As our study is the first to examine

the impact of sleep loss on neural processes in overweight adolescents during social evaluation, it is difficult to interpret these unexpected findings within the context of existing literature. Given research suggesting that activation in the posterior cingulate gyrus is associated with mind wandering and future-oriented thinking, it is possible that sleep loss increased proneness to distraction or thoughts of future interactions with interested peers[52]. The ACC is engaged during positive and negative emotional responses and is more active after sleep deprivation[53,54]. We had originally hypothesized that the amygdala would be more active in response to negative emotion, but negative peer feedback may not have been salient enough to create a significant response for sleep deprived participants in this brain region[55].

Lastly, there was partial support for our third hypothesis. We posited that greater activity to negative feedback among adolescents in the sleep deprivation group would be associated with greater caloric consumption during an *ad libitum* test breakfast. While we did find the relation between brain function and caloric consumption was primarily associated with response to negative feedback among adolescents in the no sleep group, the direction was opposite of what was predicted. Specifically, in the bilateral ACC, greater caloric consumption was associated with less activation during negative purported social evaluation. While no previous research has examined the interaction of sleep, caloric consumption, and social evaluation, this finding is not what we would have expected given the extant literature in these different domains. For example, past research has shown that the ACC is more activated after sleep deprivation[54] and this region has been linked to increased sensitivity to peer rejection among adolescents[56]. Again, this is contrary to what we hypothesized and to the literature more broadly. There may be something unique to sleep deprivation that leads to varied and unpredictable responses to social evaluation.

Furthermore, in contrast to our finding that a weaker response to negative evaluation is associated with increased caloric consumption, past research has found heightened neural responses during this task to be associated with increased eating[31]. While this discrepancy from previous research may simply be an anomaly, it could also result from the sleep manipulation component of our study which has not been used in previous research. Sleep loss is linked to deficits in emotion regulation[57], which could present as either increased or decreased activation in the involved brain regions. A well-rested teen may be able to better use their emotional faculties to respond to social stressors, while a sleep-deprived teen might be more sensitive to evaluation or might generally struggle to manage their emotional responses, resulting in dysregulated neural responses. Simon and colleagues[58] posits that sleep deprivation leads to a loss of emotional neutrality and adolescents are a population known to already have heightened emotional reactivity[59], suggesting that the impact of sleep deprivation on emotion regulation could be even more pronounced.

This study should be interpreted within context of several study limitations. First, our naturalistic experimental design made adherence to our prescribed sleep modification protocol difficult, producing some variability in sleep duration in the habitual sleep group. Relatedly, our sleep manipulation examined acute sleep loss which is not generalizable to adolescents who experience chronic sleep debt, a common presentation in this population[60]. Furthermore, we used total time in bed as a proxy for sleep duration and this measure may not accurately reflect actual sleep duration. Similarly, we focused on sleep duration but did not assess sleep quality or perceived sleep adequacy. Next, because our study design required deception and immediate debriefing, we were not able to use a within-subjects design which would have improved interpretability of fMRI findings. Additionally, despite random assignment to condition, we

observed a main effect of sleep duration group on PFS score, a difference which may have resulted from our relatively low sample size. There were also several potential confounds for which we did not control including menstrual cycle phase, subjective hunger before completing the MRI scan (although fast duration was standardized), and time of day effects (although all scans were completed in the morning). Finally, there is potential for observer effects with our experimental food consumption buffet.

Despite these limitations, our study also offers a number of strengths to the existing body of research, including a research design that assessed effects of interpersonal distress on neural and behavioral outcomes in overweight/obese adolescent females, a population with particular sensitivity to social threat. The study also included a relatively large sample size relative to comparable research and we employed rigorous methods for assessing important study variables (i.e.,accelerometry for determination of sleep adherence, fMRI to calculate neural response, and food buffet to estimate caloric consumption). Furthermore, our free-living research design enhances ecological validity and generalizability.

Findings from this study suggest that sleep restriction significantly increases neural responding associated with emotion dysregulation in adolescent girls with overweight or obesity. Our study implies that one mechanism through which insufficient sleep may influence weight gain is greater neural responsiveness associated with emotional dysregulation when experiencing social stress. Because social stress is common among this population, the neuroprotective effects of sleep may be particularly salient for adolescent females with overweight/obesity. These findings support intervention and prevention efforts aimed at improving sleep duration in this population. Future research examining whether neural processes associated with stress and sleep correspond with psychological or behavioral measures of emotion regulation is recommended.

Furthermore, extending this research to examine longitudinal effects of neural processes associated with insufficient sleep on habitual eating behavior is warranted.

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Figure Captions

Figure 1. Visual depiction of the second run of the Chatroom Task conducted during the scanning session, used for analysis in the current study

Figure 2. Main Effect of Sleep

Figure 3. Interaction of Group and Social Evaluation

Figure 4. Interaction of Group, Social Evaluation, and Caloric Intake

Table 1

Summary of Demographic and Anthropometric Data, by Sleep Condition

	Total Sample	Habitual Sleep Group	No Sleep Group	*p*-value	T-test/Chi-square
N	42	20	22		
Age (SD)	16.48 (1.01)	16.57 (.87)	16.41 (1.2)	0.61	-0.52
BMI %ile (SD)	94.57 (4.4)	95.87 (3.6)	93.39 (4.8)	0.07	-1.9
Calories (SD)	724.1 (389.3)	728.2 (101.5)	720.4 (70.6)	0.95	0.06
PFS (SD)	32.4 (11.1)	36.5 (13.2)	28.6 (7.1)	0.02	-2.4
Ethnicity *N*				0.44	2.7
White	71%	70%	73%		
Hispanic	9%	5%	9%		
Black	5%	0%	9%		
Other	14%	2%	9%		

Table 2

Task effects from ROI analyses

	MNI Coordinates			Cluster Size	F	Effect Size
	x	*y*	*z*	*Voxels*		*Partial eta-squared*
Sleep						
Left Putamen	-26	7	1	17	19.348	.33
Right Hippocampal Tail	22	-35	-2	16	29.353	.42
Sleep x Social Evaluation						
Left Posterior Cingulate Gyrus	-2	-29	43	79	11.18	.22
Right Medial Frontal Gyrus	4	4	49	18	9.49	.19
Left Perigenual Anterior Cingulate	-5	43	-5	17	8.97	.18
Sleep x Social Evaluation x Caloric Intake						
Left Mid Cingulate Gyrus	-2	13	37	28	7.304	.15
Right Mid Cingulate Gyrus	7	1	43	20	11.36	.22

Note. Activation clusters reflect Sleep (No Sleep/Habitual Sleep), Social Evaluation (Positive/Negative), and Caloric Intake interactions or main effects. Small volume analysis, $p < .005$, cluster size > 13.

Figure 1

Visual depiction of the second run of the Chatroom Task conducted during the scanning session, used for analysis in the current study

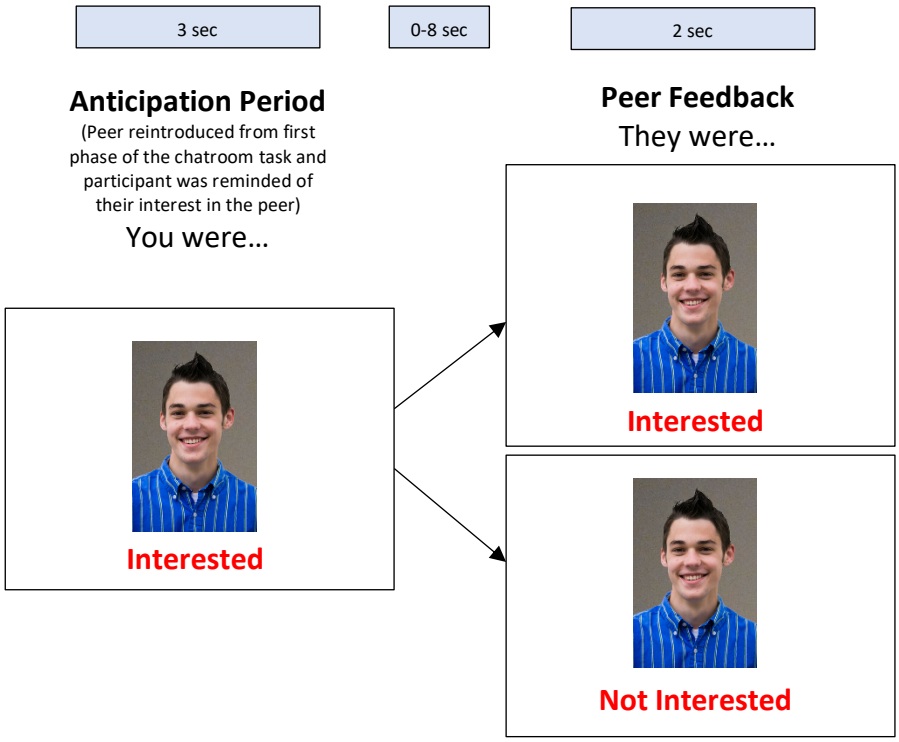
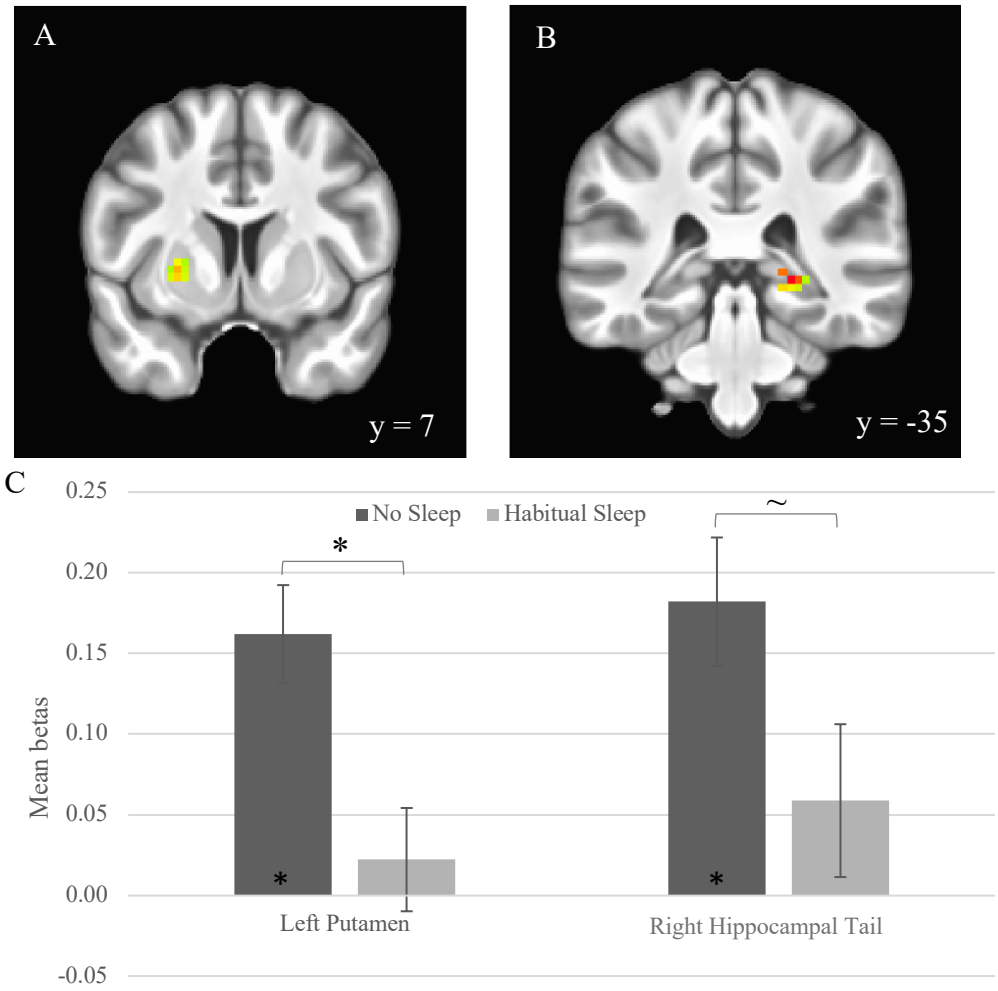


Figure 2

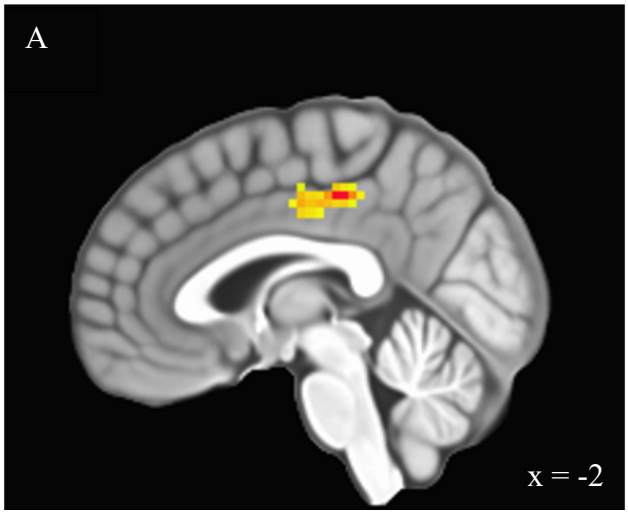
Main Effect of Sleep

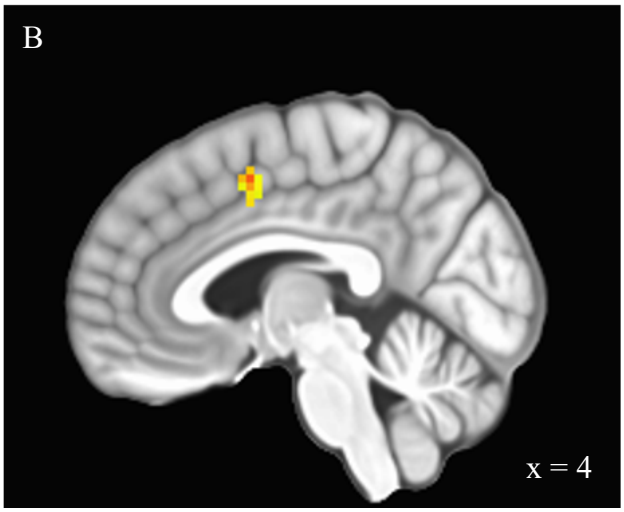


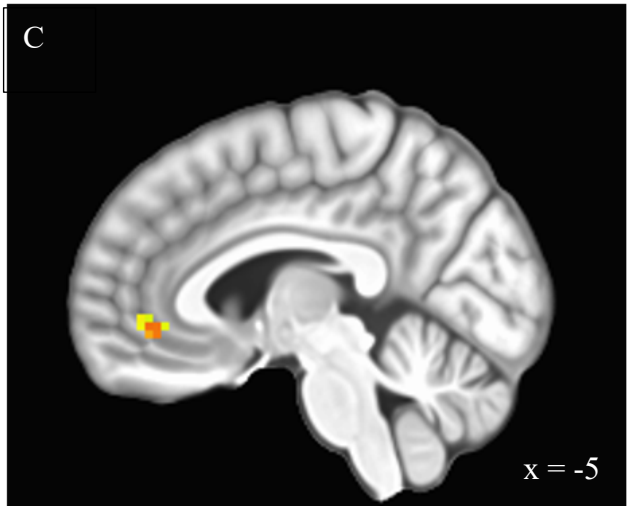
Note. Significant clusters for the main effect of sleep group (no sleep-habitual sleep) on brain activation during social evaluation. A. Left putamen (-26, 7, 1); and B. Right hippocampal tail (22, -35, -2). C. Decompositions describe differences in engagement between the "no sleep" and "habitual sleep" group, with heightened engagement in the "no sleep"
*p < .05, ~p = .052.

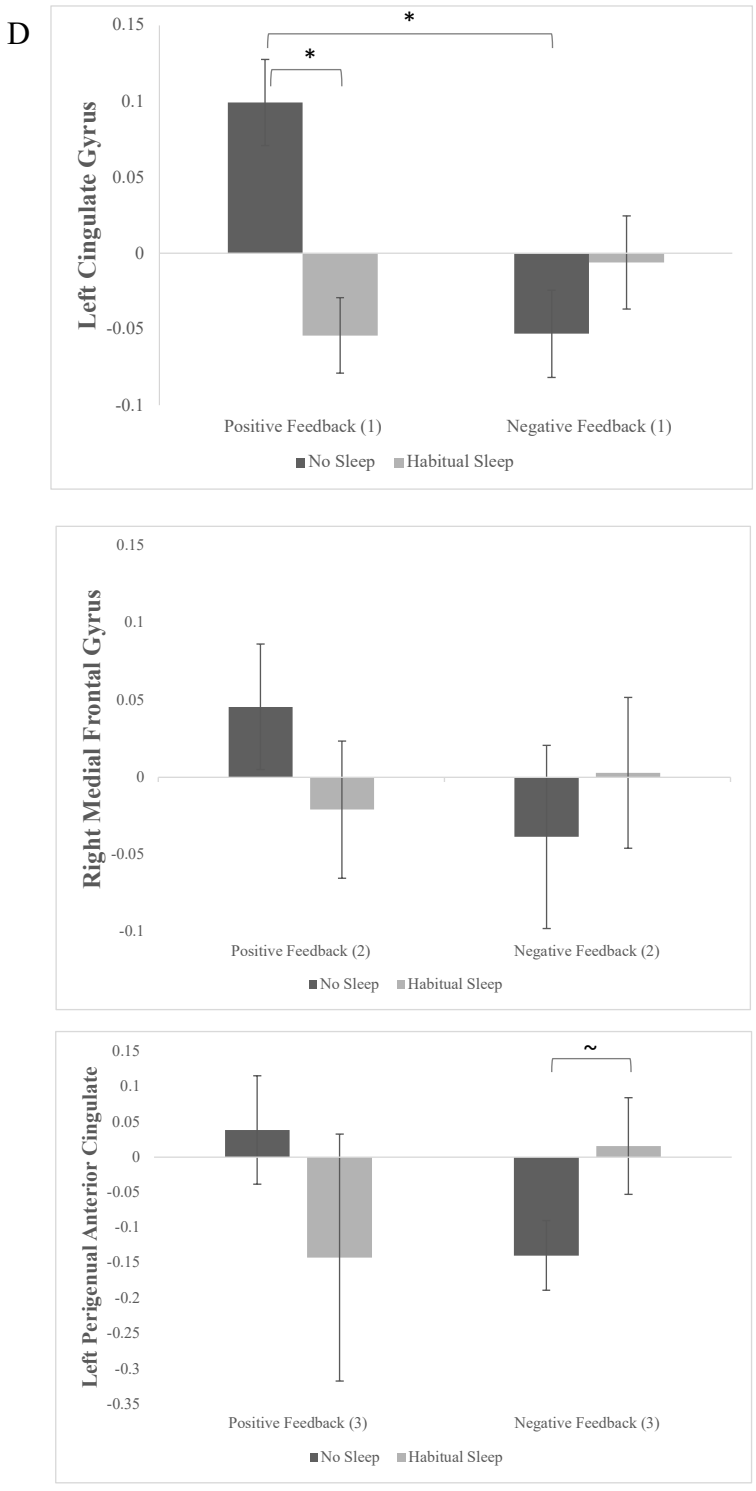
Figure 3

Interaction of Group and Social Evaluation





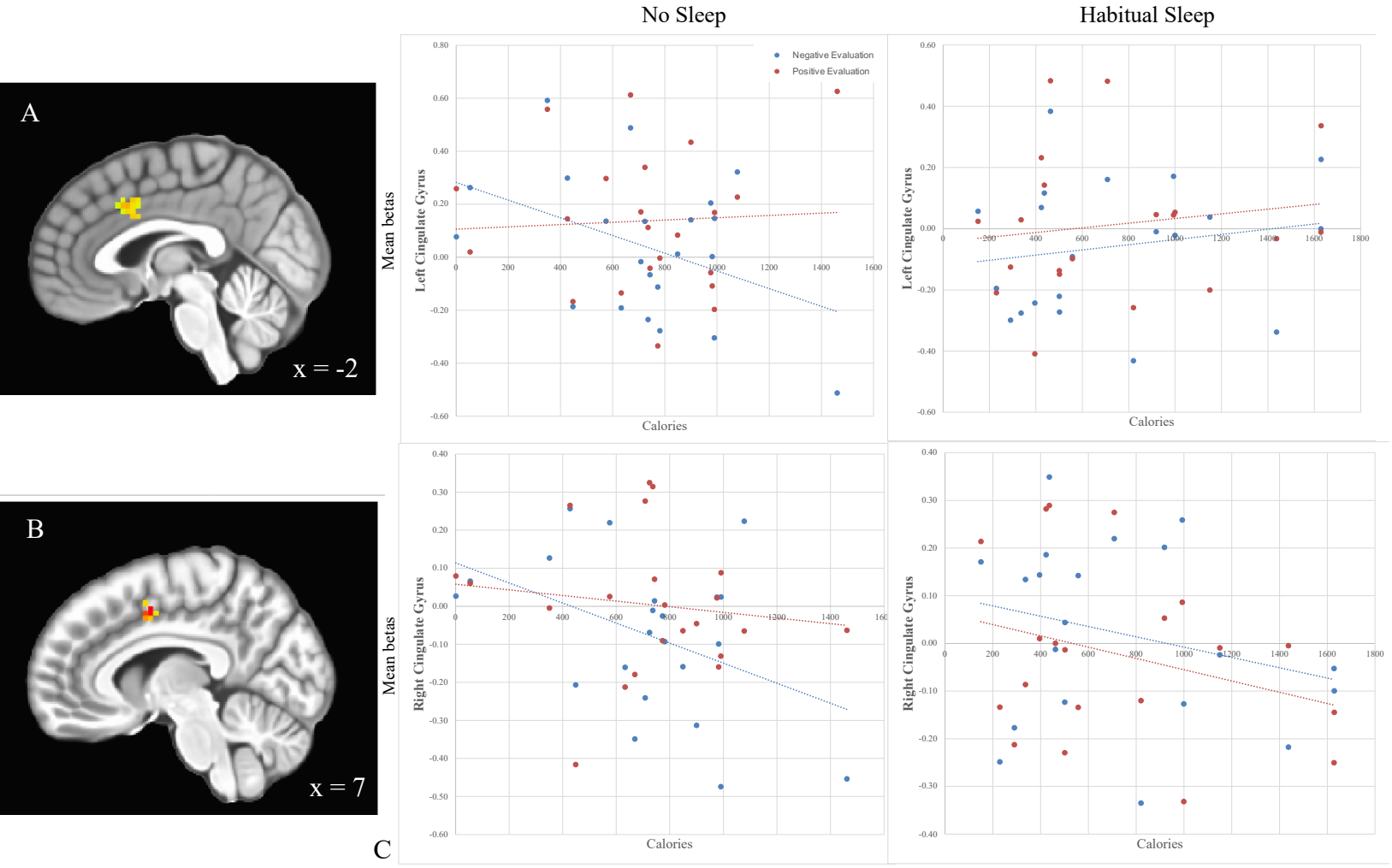




Note. Significant clusters for the interaction between group and social evaluation. A. Left cingulate gyrus (-2, -29, 43). B. Right medial frontal gyrus (4, 4, 49). C. Left perigenual anterior cingulate (-5, 43, -5). D. Decompositions describe differences in engagement between the "no sleep" and "habitual sleep" groups, broken down by positive and negative feedback.
*p < .05, ~p = .07.

Figure 4

Interaction of Group, Social Evaluation, and Caloric Intake



Note. Significant clusters for the interaction between group, social evaluation, and caloric intake. A. Left cingulate gyrus (-2, 13, 37); B. Right cingulate gyrus (7, 1, 43); C. Decompositions describe the interaction between social feedback and caloric intake at *ad libitum* breakfast buffet, broken by "no sleep" and "habitual sleep" groups.

Neural mechanisms that promote food consumption following sleep loss and social stress: An fMRI study in adolescent girls with overweight/obesity

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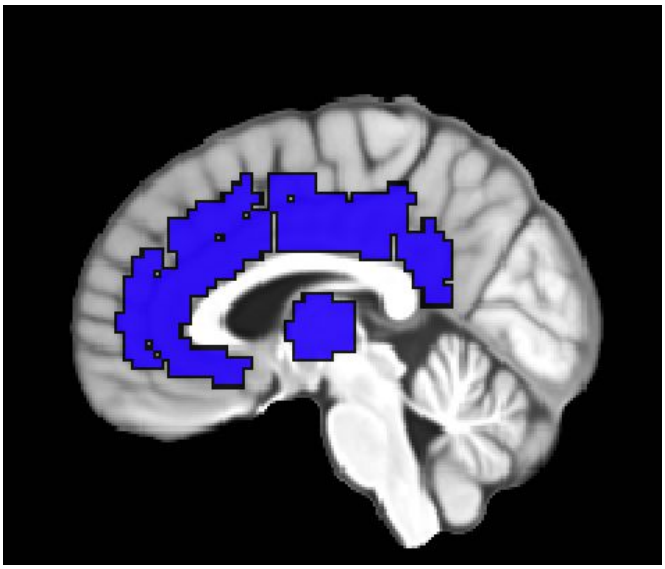
Appendix A

Small Volume Mask



Appendix A. Small volume mask (3,996 voxels) used in analyses comprised of brain regions involved in emotion processing (bilateral thalamus, caudate, putamen, nucleus accumbens, hippocampus, amygdala, and cingulate).
The script that made this mask can be found at: https://github.com/nmuncy/INI/blob/master/Task_step4_grpAnalysis.sh
Lines 106,7 have the label values and names used in the mask construction. The section "Create Masks" starting on line 238 is where the construction occurs.