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Is Variability in Inhibition-Related Neural Activation After Sleep Restriction Associated with Eating Behavior in Adolescents?

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Doctor of Philosophy

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ABSTRACT

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The primary aim of the present study was to evaluate whether intra-individual variability in inhibition-related neural activation in response to sleep restriction is associated with eating behavior in adolescents aged 12-18 years. In addition, the potential moderating effects of sex and body mass index on the association between sleep and variability in neural activation were examined. This study employed a within-subjects crossover design that randomized subjects to both a 5 hours per night (sleep restricted) and 9 hours per night (well-rested) sleep condition for 5 nights, with experimental conditions separated by four weeks. On the 6th day of each study phase participants completed a 24-hour diet recall and a food-related inhibitory go/no-go task while undergoing functional magnetic resonance imaging. Repeated measures multilevel models examined individual differences attributable to sleep duration and a series of separate multivariate analysis of variance models examined the effect that vulnerability to sleep restriction has on eating behavior as well as the moderating impact of sex and weight status. Findings suggest that adolescents who exhibited greater efficiency in inhibitory and reward-related neural activation when sleep restricted demonstrated less pronounced decrements in neural activation when sleep restricted relative to when they were well-rested. These findings suggest that the effect of sleep restriction on inhibitory control may differ between individuals such that there are individuals who appear able to sustain inhibitory control comparable to when they are well-rested while other individuals show marked declines in executive functioning-related neural activation when sleep restricted. Results from separate exploratory models including regions of interest associated with reward and across the whole brain were consistent with these findings. We also found that the effect of vulnerability to sleep restriction on inhibitory efficiency in the right inferior parietal lobule (R – IPL) and right middle frontal gyrus (R – MFG) differed by sex and was predictive of differences in overall eating behavior and sugar intake, respectively, when sleep restricted compared to well-rested. In addition, vulnerability in the inhibitory network was predictive of differences in individual eating behavior (i.e., total calories, added sugar, sugar, and total fat) for males and females across conditions. This finding demonstrates there is significant variability in the impact that sleep restriction has on inhibitory efficiency in adolescence relative to when they are well-rested, and vulnerability to inhibitory efficiency appears to effect male and female adolescent's dietary behaviors differently when they obtain insufficient sleep. Vulnerability to inhibitory efficiency when sleep restricted compared to well-rested may cause males and females to consume more energy dense foods when they obtain insufficient sleep and also differs for males and females irrespective of their sleep duration. Given the pervasiveness of chronic sleep restriction in adolescence, males who are unable to counter the effect that insufficient sleep has on palatable foods may be at greatest risk of obesity.

Keywords: individual variability, sleep restriction, inhibition-related neural activation, eating behavior

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Table of Contents

LIST OF TABLES

LIST OF FIGURES

Is Variability in Inhibition-Related Neural Activation After Sleep Restriction Associated with Eating Behavior in Adolescents?

Sleep is a fundamental physiological process that is essential for maintaining physical and mental health. Sleep is especially vital in adolescence given that sleep-dependent growth and developmental changes occur within the brain during these formative years (Telzer et al., 2015). In addition, adolescence is characterized by increased independence, responsibility, and an overall predilection to engage in risk taking and novelty seeking behavior (Arain et al., 2013). According to the National Sleep Foundation and the American Academy of Pediatrics, adolescents between the ages of 13 and 18 should obtain 8-10 hours of sleep per night (American Academy of Pediatrics, 2016; Owens et al., 2014; Paruthi et al., 2016). Prevalence estimates from a nationally representative survey conducted by the Center for Disease Control and Prevention indicate that 60-80% of adolescents regularly fail to meet these recommendations (Wheaton et al., 2016), which has since been corroborated by the American Academy of Pediatrics in a recent technical report. Inherent in this literature is the pervasive finding that adolescents in middle and high school consistently do not get enough sleep (Owens et al., 2014). Poor sleep in adolescence results in a number of negative consequences, including increased risk of accidental injury, poor school performance, cognitive deficits, obesity, sedentary activity, substance use, and mental health issues (Baum et al., 2014; Crowley et al., 2018; Lo et al., 2016; Moran & Everhart, 2012; Owens et al., 2014). Sleep also plays an important role in the maintenance of neurocognitive skills in adolescents (Beebe, 2011) and is necessary for maintaining cognitive function and attention processes (Crowley et al., 2018). However, recent findings from experimental studies and meta-analyses suggest that optimal sleep duration for adolescents is 9 – 9.25 hours of sleep, although teens obtain approximately 7 hours of sleep per

night during the week on average (Crowley et al., 2018). In fact, behavioral outcomes suggest that 8.16 – 9.3 hours are necessary for sustaining waking vigilance and alertness (Short et al., 2018), while findings from a study using self-reported outcomes of sleep and externalizing and internalizing symptoms indicate that approximately 9 hours of sleep are necessary for maintaining optimal mood (Fuligni et al., 2019). Given the effects of sleep on adolescent functioning, sleep represents a crucial factor that may influence brain maturation and neurobehavioral outcomes during adolescence (Telzer et al., 2015).

Effects of Insufficient Sleep on Cognitive Performance

One of the most profound consequences of sleep restriction in adolescence is its impact on a broad array of executive function skills that involve inhibitory control, including attention, reaction time, working memory, decision-making, and emotion regulation (Chuah et al., 2006). Several studies within the adolescent literature highlight the relationship between insufficient sleep and neurocognition. For example, Beebe and colleagues (2008) explored behavioral consequences of experimentally restricted sleep in adolescents, reporting that, relative to normal sleep (10-hours), sleep restriction (6.5-hours) induced greater deficits in attention, oppositionality/irritability, behavior regulation, and metacognition. Similarly, a recent experimental study that examined the impact of sleep duration on cognitive functioning in children (ages 8-12) demonstrated that shortened sleep (1-hour later bed-time) produced impaired functioning on measures of affect, emotion regulation, memory, and attention (Vriend et al., 2013); a finding which has been corroborated by recent systematic reviews (Owens et al., 2014). In addition, Gruber and colleagues noted that relatively modest levels of sleep restriction (sleep restricted by 1-hour) produced appreciable deterioration on measures of sustained attention, and vigilance (Gruber et al., 2011). Likewise, findings from a study in children (ages

10-11) found that a single night of sleep restriction produced slower reaction times and more lapses in attention versus control sleep (Peters et al., 2009). Taken together, these findings suggest that the effects of insufficient sleep on adolescents induce deleterious effects on an array of executive functions that involve inhibitory processes.

Under the umbrella of executive functions lies the construct of inhibition. Inhibition refers to a specific sub-set of abilities ranging from vigilance, attention, and perception, that are designed to selectively regulate automatic attentional and behavioral responses (Lowe et al., 2017; Miyake et al., 2000). Inhibition is important for preventing impulsive action, and impulsive decision-making (Demos et al., 2016). Impulsive action requires an individual to inhibit automatic responses, whereas, impulsive decision-making requires an individual to assess risk and rewards (Demos et al., 2016; Evenden, 1999). A recent meta-analysis investigating the neurocognitive consequences of sleep restriction suggests that sleep restriction negatively affects multiple domains of cognition, with the largest effects observed on attentional lapses and behavioral inhibition (Lowe et al., 2017). Findings from a within-subjects study in adults that investigated the effects of short (6-hours) compared to long sleep (9-hours) duration on impulsivity via behavioral inhibitions indicated that short sleep produced more inhibitory errors compared to long sleep (Demos et al., 2016). In addition, a study examining the impact that experimentally manipulated sleep has on go/no-go accuracy during a food inhibitory control task in a sample of obese/overweight and normal weight adolescents, provides evidence that short sleep negatively impacted reaction time and accuracy (Duraccio, Zaugg, et al., 2019).

However, the effects of sleep restriction on cognitive performance vary substantially by age, sex, and individual vulnerability to sleep restriction (Alhola & Polo-Kantola, 2007; Durmer & Dinges, 2005). A study conducted by Louca and colleagues, demonstrated significant

individual differences in adolescents' performance on objective measures of sustained attention, reaction time, and cognitive processing, following one night of total sleep deprivation (Louca & Short, 2014). Results showed increased inter-individual variance in lapses, fastest reaction times, and correct responses on a measure of cognitive processing speed ($p < .008$). In addition, they observed large between-subjects variance in performance on measures of sustained attention (e.g., errors of commission, errors of omission). Specifically, they observed that some subjects were able to sustain performance, while others had marked deficits in response to sleep deprivation. These findings mirror those reported by Demos and colleagues (2016), which found that the effect of sleep on inhibition was greater for those reporting longer habitual time in bed. Moreover, findings from this body of research provide evidence that the differential effects of chronic sleep restriction on neurocognitive performance are stable and trait-like, suggesting that insufficient sleep affect sensitive individuals consistently and across behavioral domains (Goel & Dinges, 2011; Krause et al., 2017; Rupp et al., 2012; Tkachenko & Dinges, 2018; Van Dongen et al., 2004).

Furthermore, evidence from studies conducted in adolescents suggest that alterations in executive processes that involve inhibition may moderate the relationship between sleep and adolescent risky behavior. Specifically, a recent study found that adolescents most vulnerable to attentional decline after sleep restriction had poorer lateral vehicle control and reduced driving speeds (Garner et al., 2017). In addition, a study conducted by Telzer and colleagues (2013) that investigated the association between sleep quality and cognitive control, demonstrated adolescents who reported insufficient sleep displayed greater risk-taking behavior which corresponded to reduced activation in the dorsolateral prefrontal cortex, and greater insula activation. Furthermore, Mayes and colleagues reported that the mechanism responsible for the

relationship between sleep insufficiency and learning problems in children is symptoms of inattention (Mayes et al., 2008). Collectively, these findings provide evidence that cognitive control processes may explain the link between insufficient sleep and behavioral dysregulation in adolescents (Beebe, 2011).

Although the biological basis for executive function deficits associated with sleep restriction has yet to be determined, preliminary research using functional magnetic resonance imaging (fMRI) has shown evidence that neural activation corresponds to neurocognitive performance. These findings indicate that the cognitive impairments following sleep restriction may be at least partially explained by its influence on neural structures and functions within frontal parietal regions of the brain (Chee et al., 2006; Epstein et al., 2009). fMRI studies have consistently shown that sleep restriction is associated with task-related reductions in activation in dorsolateral prefrontal cortex, and intraparietal sulcus (Krause et al., 2017). In addition, there is substantial evidence within the adult literature indicating that the degree of cognitive impairment and deficits in brain function associated with sleep restriction varies substantially between individuals (Krause et al., 2017; Van Dongen et al., 2004). However, the differential effects of sleep restriction on underlying brain functions associated with inhibition in adolescents is unknown (Beebe, 2011).

Neural Consequences of Insufficient Sleep are not Uniform

Research also suggests that sensitivity to insufficient sleep varies across individuals such that individuals with greater vulnerability to sleep restriction have decreased activation in brain regions associated with inhibition and other executive function processes. Conversely, less vulnerable individuals have increased activation in regions associated with executive functions including response inhibition (e.g., right inferior frontal region) and working memory (e.g.,

prefrontal cortex, supplementary motor area, parietal cortex) after experimentally induced sleep restriction, suggesting a compensatory response in resistant individuals (Chee & Tan, 2010; Chuah et al., 2006; Mu et al., 2005). In addition, findings from Chee & Chuah (2008) suggest that the degree of task-related functional activation when well-rested predicts the extent of performance decline when sleep restricted. In an experimental research study that examined 33 healthy young adult men, Mu and colleagues (2005) found that individual vulnerability to sleep restriction was predictive of neural activation during a working memory task when well-rested and following 30-hours of total sleep deprivation. Specifically, in this study they demonstrated that individuals resistant to sleep deprivation had more brain activation when both well-rested and sleep deprived compared to those that were most vulnerable to the effects of sleep deprivation. In addition, sleep deprivation also produced both within and between group differences in subject's neural circuitry. Those subjects deemed most vulnerable to the effects of sleep deprivation exhibited reduced activation in circuits involved in executive function and working memory. Similarly, results from a study conducted by Chua and colleagues (2014) using electroencephalogram (EEG) measures found that variability in behavioral and physiologic measures at baseline was related to a decline in performance on sustained attention tasks when sleep deprived. Specifically, they found that vulnerability to sleep deprivation was predictive of slower reaction times and increased variability in response times as well as increased variability in EEG theta frequency. In addition, a recent study in adults showed that greater specificity in functional connectivity in cortical networks associated with externally focused attention when rested may predict vulnerability to sleep deprivation (Yeo et al., 2015). However, there is substantial variability in the effects of insufficient sleep on task specific prefrontal cortical

activation (Chee et al., 2006), indicating that individual differences in vulnerability to sleep restriction may vary by task.

While the extent of individual differences in cognitive impairment and brain function from insufficient sleep is not as clearly established in adolescents, preliminary findings suggest that adolescents also differ in their response to sleep restriction (Louca & Short, 2014). Garner and colleagues (2017) illustrated this phenomenon in a recent experimental study that examined the impact of sleep restriction on adolescent drivers. In this study, they found that an adolescent's vulnerability, measured using raw score change in attention ratings between sleep restriction and healthy sleep, to attention deficits following sleep restriction, moderated the association between inadequate sleep and lateral vehicle control. This line of research provides additional evidence for variability in the effect of sleep restriction on health behaviors in adolescents.

Short Sleep Increases Dietary Consumption and Obesity Risk

Inadequate sleep is significantly associated with an increased risk of obesity (Sluggett et al., 2019; Wu et al., 2017). Evidence from cross-sectional research suggests that for every hour of increased sleep time, the odds for obesity in adolescents decreases by 80% (Gupta et al., 2002). Findings from recent meta-analyses of longitudinal, cross sectional, and prospective studies suggests that short sleep duration in children and adolescents confers substantial risk for developing overweight/obesity (OR: 2.15; 95% confidence interval; Fatima et al., 2015). Taken together, this body of research suggests, sleep duration is significantly associated with changes in weight status such that longer sleep duration during childhood is associated with decreased weight gain as one ages (Miller et al., 2018). In fact, Chen and colleagues published evidence in a meta-analysis that suggests youth who fail to meet sleep duration guidelines are at increased

risk for developing overweight and obesity (Chen et al., 2008). Based on findings from this literature, there is evidence that the obesity risk conferred by insufficient sleep is commensurate with if not greater than other risk factors for developing obesity, including parental obesity and screen time (Liou et al., 2010; Owens et al., 2014). Intervention studies have furthered this body of knowledge about the link between sleep and obesity, demonstrating that improvements in sleep duration led to positive changes in body mass index (Valrie et al., 2015) and food choices (Asarnow et al., 2017) in adolescents following participation in an intervention program (Sluggett et al., 2019).

Previous research suggests that diet may play an important role in this relationship (Chaput & Dutil, 2016; Miller et al., 2015). Findings from literature investigating the effects of sleep on diet have shown that insufficient sleep is associated with increased caloric intake, higher energy-dense snack consumption, irregular eating patterns, decreased dietary quality, and increased consumption of highly palatable foods (Chaput & Dutil, 2016; Dashti et al., 2015; Miller et al., 2015; Sluggett et al., 2019). A recent systematic review of this scholarship noted that with regard to the impact that sleep duration has on caloric intake, findings are mixed. Authors of this review note that there is sufficient evidence from correlational research to conclude that simple counts of macronutrient intake have a weak or negligible association with pediatric sleep. Further they suggest that investigating the relationship between total sleep duration and macronutrient intake may not provide further insight into the link between shortened sleep and increased obesity risk; however, experimental paradigms that manipulate sleep duration may elucidate the causal relationship between sleep and obesity (Krietsch et al., 2019).

Furthermore, research indicates that sleep deprivation also results in alterations in appetite regulating hormones (e.g., leptin, ghrelin) and increased insulin resistance (Leproult & Van Cauter, 2010; Matthews et al., 2012), which leads to greater caloric intake of sweet (Beebe et al., 2013), salty, and high calorie foods (Matthews et al., 2012; Moran & Everhart, 2012). Findings from an experimental study in young men found that, compared to 10 hours of sleep, sleep restriction of 4 hours per night over two days resulted in an 18% decrease in leptin, a 28% increase in ghrelin, and a 24% increase in hunger (Spiegel et al., 2004). Moreover, findings from cross-sectional research in adolescents suggests that short sleep duration (< 8 hours) is associated with alterations in the proportion of calories consumed daily from fat and carbohydrates, relative to longer sleep duration (> 8-hours; Weiss et al., 2010). Beebe and colleagues (2013) corroborated these findings in a randomized cross-over design study that explored the relationship between sleep and dietary intake in adolescents (ages 14-16). In this study, Beebe and colleagues found that sleep restricted adolescents consumed significantly more calories from foods high in sugar following 5-nights of sleep restriction (5-hours) compared to 5-nights of healthy sleep (10-hours). This same research group also found that adolescents rated pictures of sweet/dessert foods as more appealing during sleep restriction relative to healthy sleep (Simon et al., 2015). Furthermore, preliminary research in adults provides evidence that the effects of sleep deprivation on weight gain, late-night eating, and caloric and fat intake vary considerably between individuals, and are also stable over time (Spaeth et al., 2015). Evidence from longitudinal and cross-sectional findings, suggests that the relationship between sleep and obesity risk may be greatest for adolescent males, indicating that the strength of the relationship may vary by sex (Knutson, 2005). The effects of sleep on diet propose that sleep restriction may confer an increased risk of developing obesity by disrupting healthy eating habits and the

negative effects of sleep restriction on eating behavior are variable across individuals (Weiss et al., 2010). It is also possible that commonalities exist across individuals with greater sensitivity to sleep restriction and identifying subclasses of individuals with heightened sensitivity may be an important research aim.

Sleep Restriction Alters Food-Related Reward and Inhibition Processes

One mechanism of action that might underlie the relationship between sleep, diet, and weight gain is altered recruitment of brain regions involved in inhibitory control. Inhibition is a central element of self-control and subsequent eating behavior that modulates appetitive drive and motivation (Hall, 2016). A previous study using a food-related go/no-go task and a self-report measure of food reward found that sleep restriction resulted in impairments in food-related inhibitory control and increased reward sensitivity relative to normal sleep in both normal and overweight/obese adolescents (Duraccio, Zaugg, et al., 2019), which corroborates findings from similar studies that sleep restriction in adolescents has a negative impact on executive function (Beebe et al., 2008). Prior research conducted in obese and healthy weight adults using fMRI further suggests that activation in the medial prefrontal cortex (a brain region associated with inhibitory control) is negatively correlated with disinhibition when viewing food relative to non-food images (Martin et al., 2010). In addition, fMRI studies have shown that sleep restricted adults and adolescents have greater activation in brain regions associated with food-related behaviors (e.g., superior and middle temporal gyri, middle and superior frontal gyri, left inferior parietal lobule, orbital frontal cortex, and right insula) when viewing food images (Demos et al., 2017; Jensen et al., 2019; St-Onge et al., 2014). Similarly, neuroimaging studies in adolescents suggests that relative to lean subjects, overweight subjects exhibit reductions in fMRI signal in regions associated with inhibition, including superior frontal gyrus, middle frontal gyrus,

ventrolateral prefrontal cortex, medial prefrontal cortex, and orbitofrontal cortex, while performing a food-related attentional task (Batterink et al., 2010). Interestingly, although research findings in young adults and adolescents suggest that sleep restriction has a negative impact on food-related inhibitory control and reward valuation (Benedict et al., 2012), findings are mixed as to whether weight status affects this relationship (Duraccio, Zaugg, et al., 2019). For example, a study in obese and normal weight children found that relative to healthy weight subjects, obese children demonstrated increased inhibitory activation in response to food images (Davids et al., 2010). Davids and colleagues propose that the discrepancy in findings may be due to the fact that obese children have less mature, less focused patterns of brain activation (Davids et al., 2010). In addition, Black and colleagues (2014) illustrated differences in functional connectivity in brain regions associated with reward and self-control in obese and healthy weight children while viewing food-associated prompts. Specifically, they found that obese children had greater connectivity between self-control and reward regions of the brain, including the left middle frontal gyrus, left ventromedial prefrontal cortex, and left lateral orbitofrontal cortex when viewing food-related stimuli. Taken together, previous research provides evidence that alterations in neural activation in brain regions associated with reward processing and inhibition may underlie increased dietary consumption in sleep restricted adolescents.

Inhibition Affects Dietary Behavior

A recent study conducted in adults that examined the causal effects of inhibition on dietary cravings and eating behavior using active cortical modulation techniques found that temporary suppression of cortical activation in the dorsolateral prefrontal cortex resulted in greater cravings in response to food images, and increased consumption of high calorie foods. Furthermore, they found that the effects of cortical suppression on food intake were mediated by

alterations in Stroop performance (Lowe et al., 2014), an objective measure of executive function (Golden et al., 2003). These findings provide evidentiary support that inhibitory control directly influences appetitive motivation and subsequent dietary intake.

Given the effects of sleep restriction on inhibitory processes, and the relationship between inhibitory control associated with food reward and dietary behavior, alterations in inhibition may mediate the relationship between sleep and dietary behavior. Preliminary findings from St-Onge and colleagues (2014) supports this theory. In this study, they found that reduced brain oxygenation level-dependent (BOLD) activity in the insula, a brain region associated with compulsive and impulsive behavior, corresponded to increased food intake when sleep restricted. Thus, it is hypothesized that sleep restriction alters recruitment of inhibitory control, which reduces an individual's ability to resist high-calorie foods (Lowe et al., 2017). A recent study by Jensen and colleagues supports such a theory, which found that normal weight adolescents demonstrated significantly greater neural activation in brain regions associated with inhibition in response to food images when sleep restricted relative to adolescents with overweight/obesity, and greater reward activation when sleep restricted collapsing across weight groups (Jensen et al., 2019). Taken together inhibition-related neural responding may be one characteristic that distinguishes individuals at greater risk for increased consumption of high-calorie foods following sleep restriction from those with less risk for suboptimal eating behavior.

Relatedly, although a substantial body of research has investigated whether differences in cognitive abilities, including inhibition and impulsivity, between adolescents of different weight statuses exist, the effects sizes have been small. A central criticism of this literature is that it relies on group level differences and has failed to account for variability in cognitive functions, suggesting that overweight/obese adolescents are not consistently impaired. In a recent study

conducted comparing obese and normal weight adolescent's cognitive ability using intra-subject variability, they found that relative to normal weight females those whose weight was in the obese category had greater variability in reaction times during a go/no-go task (Bauer & Houston, 2017).

Primary Study Aims and Hypotheses

Given the lack of research investigating neural activation following sleep restriction and its association with eating behavior in adolescents, the primary aim of this study is to determine whether intra-individual variability in inhibition-related neural activation in regions of interest (ROIs) as defined by automated meta-analyses, including the right middle frontal gyrus (R – MFG), right anterior insula (R – AI), right anterior cingulate cortex (R – ACC), and right inferior parietal lobule (R – IPL) when sleep restricted can be useful in predicting eating behavior (see Figure 1). These specific ROIs were chosen based on previously published research showing differences in adolescent neural activation while completing a food-related attentional task (Batterink et al., 2010). Specifically, we hypothesize that individuals with more marked reductions in intra-individual variability in inhibition-related neural activation after sleep restriction (relative to well-rested) will demonstrate increased consumption of dietary fat and sugar, in addition to higher total caloric consumption. By exploring potential risk profiles, this study may provide guidance regarding individual characteristics that make one particularly susceptible to dietary overconsumption following sleep restriction, which could have important clinical implications. Finally, we conducted an exploratory whole brain analysis to compare intra-individual differences in neural activation in response to food images under restricted sleep and habitual sleep conditions in order to evaluate whether differences in neural activation exist in brain regions outside of the specified ROIs.

Method

Subjects

Data for this study were collected as part of a larger investigation which examined normal and overweight adolescents' brain responses to high and low-energy food images under restricted and habitual sleep conditions. Fifty-three adolescent subjects were recruited for this study using fliers in public locations in the community. Subjects were excluded if they met any of the following criteria: history of bariatric surgery, binge eating, or psychiatric conditions (e.g., traumatic brain injury, schizophrenia), used weight loss medications, or medications that may affect salivation (e.g., antihistamines, antidepressants), were left-handed, or had food allergies. Subjects were also screened for standard MRI contraindications, such as ferrous implants, pregnancy, etc. Subjects received a prorated compensation of $150 for completion of all study procedures. One parent/guardian provided written permission for their child to participate, and all subjects provided written informed consent/assent.

Procedures

This study consisted of a two-phase within-subjects cross over design that randomly assigned subjects to complete two separate sleep conditions (5-hour; sleep restriction, 9-hour; habitual sleep). Each phase lasted 6 days/5 nights and took place three weeks apart from each other to ensure that females were in the same phase of their menstrual cycle for both assessments. Subjects were instructed to wake up prior to 9 am and establish a bedtime 5 or 9 hours before their established wake time, depending on experimental condition. Adherence to the sleep protocol was determined through self-reported sleep and wake times and accelerometry data (Actigraph GT3x+). During the final day of each sleep phase, subjects completed an assessment which included a 45-minute MRI protocol, 24-hour dietary recall, and self-report

questionnaires. During the 45-minute MRI protocol, participants completed a food-related go/no-go task (Batterink et al., 2010) where they were instructed to respond using a button press to images of healthy foods and withhold their response when viewing unhealthy food images. Prior to the assessment, subjects were instructed to fast for four hours and avoid caffeine consumption for 24 hours. Data collection occurred over the course of two consecutive years between the months of May – August. All study related procedures and measures were approved by the institutional review board.

As previously reported by Duraccio and colleagues (2019), adherence to the experimental sleep protocol was assessed using accelerometry. Based on analyses of subject's total time in bed, subjects were generally adherent to the sleep conditions and deviated from their expected sleep duration on average by less than 1 hour (Duraccio, Zaugg, et al., 2019).

MRI Data Acquisition. Neuroimaging data were obtained using a Siemens TIM Trio 3T MRI scanner using a 12-channel head coil. Functional data were collected during the go/no-go tasks using an echo planar imaging sequence with the following parameters: repetition time (TR; 28ms), echo time (TE; 28ms), field of view (192 × 192mm), acquisition matrix (64 × 64), voxel size (3× 3 × 3mm), and slice thickness (3mm). A high-resolution T1-weighted structural brain scan used for functional localization was acquired with the following parameters: TE = 2.26ms, TR = 1900ms, field of view = 218 × 250mm, acquisition matrix = 215 × 256, slice thickness = 1mm, voxel size = 0.98 × 0.98 × 1mm.

MRI Data Processing and Analysis. All MRI data were processed using the Analysis of Functional Images (AFNI) suite of software applications (Cox, 1996). Slice-time correction was applied to the functional data as part of the preprocessing procedures to account for differences between slices within a single TR. In addition, motion correction procedures were applied to

align the data with the 50th acquisition volume. The structural scans and adjacent functional scans in the session were co-registered. We created a single subjects' regression model for the go/no-go task that included six motion regressors and 10 polynomial regressors (5 per run accounting for the two runs and for scanner drift within a run). Three behavioral regressors coding for correct no-go trials, correct go trials, and fixation periods were also included. A model was created for the no-go trials by convolving the canonical hemodynamic response with a 1-second boxcar function. Fixation crosses were presented for a duration that varied randomly and ranged from 7 to 11 seconds, and subsequent events' durations were fashioned accordingly. TRs with significant motion events (> .6 mm translation or > .3° rotation; Jensen et al., 2017) were excluded from the analysis. Since an a priori ROI analysis was conducted, we did not blur the fMRI data as part of the preprocessing procedures.

We utilized Advanced Normalization Tools (ANTs; Avants et al., 2008) to accomplish spatial normalization. A nonlinear diffeomorphic spatial transformation was processed from the individual subject's structural scan to the study specific MNI template using *ants*.sh and applied to all functional data. A definition for the ROIs was obtained from a meta-analysis using the term "Inhibition" in the neurosynth.org database. The search for "Inhibition" produced 482 papers. We created ROI maps by identifying the regions with the strongest correlations using association test maps with a spatial extent threshold of k>20 contiguous voxels for the Inhibition maps. Four ROIs were associated with inhibition (R – MFG, R – AI, R – ACC, and R – IPL). We extracted and analyzed the mean activity within significant ROIs using SPSS Statistics (IBM, 2012).

For the exploratory whole-brain analysis, anatomical ROIs for reward regions were defined based on meta-analyses conducted using neurosynth.org database for the general terms "Reward," which yielded 671 papers. Using association test maps, six ROIs associated with

reward were identified (right striatum (R – STM), left striatum (L – STM), ACC, right orbitofrontal cortex (R – OFC), left orbitofrontal cortex (L – OFC), and midbrain (MDB)). Similarly to the process used for ROIs associated with inhibition, the mean activity within significant ROIs associated with reward were extracted and analyzed using SPSS Statistics (IBM, 2012). In addition, as reported by Jensen and colleagues (2019), an exploratory whole-brain analysis was conducted using 3dMVM in AFNI. The following a priori criterion: voxel-wise threshold of $p < .02$ and a spatial-extent threshold of 24 voxels, and a template brain mask created with a 5-mm FWHM spatial blur yielded a family-wise error rate $p < .05$. The mean coefficients for no-go and go trials within the significant clusters identified were extracted and analyzed using SPSS Statistics (IBM, 2012).

Voxel specific data gathered during go- versus no-go trials were utilized to calculate an index of intra-individual coefficient of variation $(ICV)^2$ in neural activation during no-go vs. go trials in specified ROIs when sleep restricted (5-hour condition) relative to well-rested (9-hour condition). The coefficient of variation is a measure of relative variability that for a defined variable is calculated by dividing the standard deviation (SD) of the variable by the mean of the variable. The ICV value for each sleep condition was created by calculating the SD/Mean for the no-go beta value and go beta value, which were derived using the mean activation during the trial type and fixation cross for each specified ROI. Therefore, an ICV value that is greater would suggest that there was increased activation in inhibitory regions (i.e., the activation relative to baseline for no-go trials was greater than the activation relative to baseline for go trials) and is a proxy for inhibitory control or efficiency (no-go greater than go). ICV when sleep restricted and well-rested was then used to determine if the variability in inhibitory control (i.e., coefficient of

variation) when sleep restricted is greater than when well-rested. Thus, the independent variable is operationalized as the variability in neural activation for no-go vs. go trials within subjects.

Measures

Demographics. Demographic information including subject's height, weight, race, ethnicity, age, and sex were collected from subjects during both assessments.

Dietary Recall. Subjects recorded their food intake for the 24-hours prior to each scanning appointment using the Automated Self-Administered 24-hour Dietary Assessment Tool for Children (ASA24-Kids-2014), 2014 version. The ASA24 is an online tool used to determine portion sizes and energy, nutrient, and total food consumption in the last 24-hours (National Cancer Institute). Study staff provided verbal instructions and were available to assist subjects as they completed the assessment. The ASA24 – Kids – 2014 has been shown to be a reliable and valid measure of adolescent dietary intake comparable to that of interviewer administered assessments and other dietary intake questionnaires (Hewawitharana et al., 2018; Hughes et al., 2017).

Go/No-Go Task. During each MRI scan, subjects completed a food-based go/no-go task to determine food-related inhibitory control (Batterink et al., 2010). For this task, subjects were shown pictures of food, and instructed to respond using a button press when they saw pictures of healthy foods and withhold their response when they saw a picture of an unhealthy food. Subject's responses and reaction times were recorded using a fiber-optic response system. Two blocks of 48 food images (75% go trials, 25% no-go) were presented for 500 milliseconds separated by a fixation cross that was presented for 7-11 seconds. Pictures were randomly chosen from a standardized database of food images (FoodPics; Blechert et al., 2014), displayed using E-Prime software (Psychology Software Tools, 2012), and synchronized with MRI image

acquisition. Previous studies utilizing a similar go/no-go paradigm have demonstrated increased neural activation in regions associated with response inhibition for no-go/go contrasts, and increased inhibition-related neural activation (Blechert et al., 2014).

Data Analytic Procedure Overview

Specific Aim 1. To evaluate the extent of individual differences in inhibitory control following sleep restriction, we calculated the ICV in neural activation during no-go vs. go trials in the specified ROIs for each sleep condition. To evaluate individual change in inhibitory efficiency in the specified ROIs when sleep restricted relative to well-rested, we performed a repeated measures multilevel mixed model. This analysis is most suitable for examining data that include nested or repeated measurements where subjects are nested within conditions (Heck et al., 2013) and is appropriate for modeling complex hierarchical structures. Given the study design, the data were treated as a type of two level-data where ROIs and condition were crossed within-subject factors such that measurements were made for the same ROIs and the same conditions within each subject. A model was constructed to examine changes in neural activation in four regions associated with inhibitory control (R – MFG, R – AI, R – ACC, and R – IPL) for go versus no-go fixation trials (Durmer & Dinges, 2005).

Next, we conducted an exploratory whole brain analysis to compare intra-individual differences in neural activation in response to food images under restricted sleep and habitual sleep conditions in order to evaluate whether differences in neural activation exist in brain regions outside of the specified ROIs. We again calculated an ICV in neural activation during no-go vs. go trials in each specified ROI for each sleep condition and performed a series of two separate repeated measures multilevel mixed model where subjects are nested within condition

(Heck et al., 2013). Of which one model included ROIs previously identified as coupled with food reward and another with the remaining ROIs across the brain.

Specific Aim 2. Based on the extent of an individual's change in inhibitory efficiency in neural activation during the go/no-go task when sleep restricted, we created a variable to estimate vulnerability to sleep restriction that accounted for inhibitory activation when they were well-rested at the individual level, using the following equation ($ICV_{\text{Sleep Restricted (SR)}} - ICV_{\text{Well-rested (WR)}}$) × ICV_{WR}. Using this value, we were able to quantify the magnitude of an individual's change in inhibitory efficiency on the go/no-go task when sleep restricted relative to well-rested in each ROI specified. Consistent with previous literature, to delineate subjects based on the extent of change in inhibitory efficiency when sleep restricted, we divided subjects into tertiles based on individual vulnerability to sleep restriction (Chuah et al., 2006). To evaluate the effect individual vulnerability to sleep loss has on caloric intake a series of repeated-measure MANOVAs were conducted to examine whether vulnerability in each of the ROIs is useful in predicting eating behavior. Due to interest in determining how efficiency in neural activation in ROIs across the brain affects vulnerability to sleep restriction and whether vulnerability to sleep restriction is useful in predicting eating behavior, we chose to divide subjects into tertiles on an ROI by ROI basis to elucidate the relationship between vulnerability to sleep restriction in specific ROIs and eating behavior. We hypothesized that individuals belonging to tertiles with increased susceptibility to sleep restriction in terms of neural responding will demonstrate increased dietary consumption (e.g., total calories, sugar, added sugar, carbohydrate, protein, total fat) relative to resistant individuals.

Consistent with specific aim 2 we created a variable to quantify vulnerability to sleep restriction at the individual level for an exploratory whole-brain analysis using the following

equation $(ICV_{SR} - ICV_{WR}) \times ICV_{WR}$ and divided subjects into tertiles based on their individual vulnerability to sleep restriction (Chuah et al., 2006). To evaluate the effect individual vulnerability to sleep loss has on caloric intake a series of 19 repeated-measure MANOVAs were conducted to examine whether vulnerability in each of the ROIs included as part of an exploratory whole brain analysis is useful in predicting eating behavior.

Specific Aims 3 & 4. For the exploratory moderator analyses we conducted a similar series of repeated-measure MANOVA models, adding the between-subject potential moderators of sex, and weight category in separate analyses. Due to the inclusion of ROIs across the whole brain and moderator variables an additional 57 exploratory analyses were conducted, therefore we used the Bonferroni correction to set the significant value threshold to $p = .001$ to reduce the likelihood of obtaining false positives.

Analysis Steps Aim 1

For aim 1 a top-down modeling strategy was used with repeated measure variables included at level 1 (Sleep condition, brain region, index of intra-individual coefficient of variation $(ICV)^2$ in neural activation during no-go vs. go trials) and individual subject variables at level 2.

Step 1. The primary aim of step 1 was to fit a model with a loaded mean structure and random subject-specific intercept, which included fixed effects associated with specified ROIs, sleep condition, and the interaction between ROIs and sleep condition. This model included a single random effect associated with the intercept for each subject and a residual associated with each observation. The residual variance associated with each observation is assumed to be independent and to have the same variance across all ROIs and conditions.

Step 2. A second model was fit by adding a random subject specific effect for sleep condition, which allowed the marginal variance of observations for the well-rested condition to differ from that of the sleep restricted condition such that there are two random intercepts for each subject. In Step 2, we assume that the residual variance is constant across all levels of region and sleep condition. We tested the hypothesis that the variance of the residuals is constant across both sleep conditions using a REML-based likelihood ratio test, which is calculated by subtracting the -2 REML log-likelihood value for Model 2 (the reference model) from that for Model 1 (the nested model).

Step 3. We fit a third model to explore whether there is heterogeneity in the residual variance by specifying heterogenous residual variances for each sleep condition to decide whether the model should have homogenous or heterogenous residual variances. In this model we assume that the variance of the residuals is different across levels of condition. We test the hypothesis that the variance of the residuals is constant across both sleep conditions and select a structure for the random effect using a REML-based likelihood ratio test, which is calculated by subtracting the -2 REML log-likelihood value for Model 3 (the reference model) from that for Model 2 (the nested model).

Step 4. For the fourth and final step, we decide whether to keep the fixed effects of the ROI by sleep condition interaction in Model 3. A final model (Model 4) was reduced by removing nonsignificant fixed effects and model diagnostics were assessed. To test the hypothesis that the fixed effects associated with the ROI by sleep condition interaction can be omitted we use an *F*-test, based on the REML estimation of the parameters in Model 3.

In addition, a similar stepwise approach was utilized to conduct an exploratory whole brain analysis to determine whether intra-individual differences in neural activation exists in

brain regions outside of the specified ROIs. We ran separate models, one that included ROIs that previous research suggests are involved in reward seeking behavior and reward valuation, including the PCC, R – OFC, L – OFC, MDB, ACC, R – STM, and L – STM. In addition, a final series of models were conducted which included fitting a final model with all remaining ROIs, including the right supplementary motor area (R – SMA), right lingual gyrus (R – LG), right triangularis (R – TG), inferior lateral operculum (ILO), right angular gyrus (R – AG), left inferior occipital gyrus (L – IOG), left precentral gyrus (L – PcG), left medial operculum (L – MO), right hippocampus (R – Hip), right caudate (R – Cau), superior temporal gyrus (STG), and right temporal pole (R – TP).

Results

Data Screening

Data were screened prior to conducting analyses to identify significant outliers and missing data. ICV outliers for go/no-go activation varied by ROI. For ROIs associated with inhibitory control we observed two very high outliers in ICV within the R – ACC (> 250,000), for reward valuation we observed two very high outliers in ICV within the R – OFC (>1,000,000), and for the whole brain analyses, we observed six very high outliers within the ILO (n = 1), L – PcG (n = 2), R – AG (n = 1), R – Hip (n = 1), and R – LG (>100,000). All outliers identified came from 3 subjects, with one subject containing 7/10 outliers. Given the large discrepancy in the outlier values relative to the overall study sample, as well as the localization of the outlier data, we chose to exclude outlier data within ROIs by subject from the study analyses. Such that for a given subject whose data contained an outlier within a specific ROI, only that ROI data was excluded from the analyses.

In order to examine how the presence of outliers affected the statistical analyses, we ran a series of mixed models that included outliers and one without. When we examined the model

that included outliers, it is notable that we received the following error message: “Iteration was terminated but convergence has not been achieved. The MIXED procedure continues despite this warning. Subsequent results produced are based on the last iteration. Validity of the model fit is uncertain.” This suggests that the parameter estimates, and standard errors may be invalid and should be interpreted with caution. Results from this model do not appear to be redundant; however, based on the model’s fixed effect estimations the outliers observed appear to have implications on the model results. Specifically, we observe that the fixed effects estimates are only significant within ROIs that contain the significant outliers. The influence of the outliers on our results, provides further support for removing outlier data from our primary analyses.

Data missingness was also assessed. With regard to fMRI data, all data were complete. We were unable to collect dietary data from three subjects. Two subjects were missing dietary data for the 5- hour condition and one subject was missing dietary data for the 9-hour condition.

Data Analytics

We divided subjects into tertiles based on their individual vulnerability to sleep restriction within each ROI. Individual vulnerability was determined using the following equation $(ICV_{SR} - ICV_{WR}) \times ICV_{WR.}$ Using this value, we were able to quantify the magnitude of an individual’s change in inhibitory efficiency on the go/no-go task when sleep restricted relative to well-rested. While the range and standard deviation of vulnerability scores for each vulnerability group varied by ROI, we observed a consistent pattern when examining the relationship between ICV values and vulnerability group categorization. Specifically, individuals in Group 1 generally had greater ICV values when well-rested relative to sleep restricted and were categorized as those that were most vulnerable to sleep restriction. Group 2 exhibited generally consistent ICV values for both conditions, and Group 3 had greater ICV values when

sleep restricted and were deemed least vulnerable to the effects of sleep restriction (see Table 1 – 3). Of note, within the R – SMA vulnerability Groups 1 and 3 exhibited increased activation when sleep restricted relative to well-rested with Group 2 exhibiting relatively consistent activation across conditions.

Subjects

Fifty-three subjects (45.3% female; 79.2% Caucasian) between the ages of 12-18 (M_{age} = 16.51, *SD* = 1.65) were included in the final analyses. The final sample included twenty-seven normal weight adolescents (body mass index percentile ≥ 5 and ≤ 85; $M_{BMI\%}$ = 57.19, *SD* = 23.94), and twenty-six overweight/obese adolescents (body mass index percentile ≥ 85; $M_{BMI\%}$ = 91.35, *SD* = 11.92). See Table 4 for detailed demographic information.

Specific Aim 1

Model 1. To test whether the random effects associated with condition for each subject can be omitted from subsequent models, we first fit a model with a loaded mean structure and random-subject intercept. Results from the first model suggest there was a main effect for sleep condition in each of the specified ROIs, indicating that the average difference in inhibitory efficiency, as measured by the intraindividual coefficient of variation in beta activation for the No-go versus Go trials, between the sleep restricted and well-rested condition was significant (F = 5.88, p = .02). We also observed a significant main effect for variability in inhibitory efficiency differences by condition in each of the ROIs, suggesting that there were significant differences in the average variability in each of the specified ROIs when well-rested versus sleep restricted, (F = 4.16, p = .01). We also observed a significant interaction between condition and ROI, suggesting that condition was a significant predictor of variability in inhibitory efficiency, (F = 2.58, p = .05). In addition, with respect to the random effects estimate, the residual variance

associated with the intercept for each subject (Wald Z = 19.76, p = .00) and the residual associated with each observation were statistically significant (Wald Z = 3.87, p = .00), suggesting that there is statistically significant variability at the between-subject and within-subject level. Based on the estimates of the variance components, 18.4% of variability in inhibitory efficiency occurred between subjects.

Model 2. In our second step, a random condition effect for each subject was added, allowing the effect of the sleep restricted condition vs. well-rested to vary from subject to subject. In the parameterization of the model, we assume that fixed effects associated with the ROI, R – IPL, and sleep restriction are set to zero representing the reference categories throughout the results. Consistent with the first model, there was a significant condition by ROI interaction effect on inhibitory efficiency, (F = 3.47, p = .02), suggesting that inhibitory efficiency in ROIs differed by condition. There was also a significant main effect of ROI on inhibitory efficiency, (F = 5.24, p = .00). The fixed effects estimate from this model indicate that the effect of inhibitory efficiency significantly differed between the R – IPL vs. the R – ACC (β = -523.79, p = .00), R – MFG (β = -585.40, p = .00), and R – AI (β = -564.38, p = .00) when sleep restricted. The parameters for the fixed effects associated with the ROI by condition interactions suggest there were significant changes in the ROI effects for the sleep restricted condition relative to the well-rested condition in the R – ACC (β = 548.27, p = .03), R – MFG (β = 743.24, p = .00), R – AI (β = 571.29, p = .02), and the R – IPL (β = -767.19, p = .01). In regard to the level 2 variance components, findings suggest that there was significant variability in individual ROI's inhibitory efficiency around the individual regression lines for each subject (Wald Z = 18.81, p = .00), variability in the condition-inhibitory efficiency slope across subjects (Wald Z = 4.35, p = .00), and variance in the intercepts across subjects (Wald Z = 4.64, p = .00;

see Table 5). To determine whether the random condition effects can be omitted from Model 1, we performed a likelihood ratio test (see Table 6). Based on a significant result in this test (p = .00), we decided to retain the random condition effects as a result of this significant test and reject the null hypothesis; thus, model 2 is deemed the preferred model at this stage in the analysis.

Model 3. In this step of the analysis, we fit model 3 to allow the residual variances to vary for each level of condition by including separate residual variances for the sleep restricted and well-rested condition. This process allows a more flexible specification of the residual variance by allowing observations at different levels of the condition on the same subject to have different residual variances. Similar to model 2, we observed a significant main effect for ROI (F = 2.75, p = .04) on inhibitory efficiency as well as a significant ROI by condition interaction (F =3.50, p = .02). The fixed effects estimate from this model indicate that the effect of inhibitory efficiency significantly differed between the R – IPL vs. the R – ACC (β = -519.63, p = .03), R – MFG (β = -585.40, p = .01), and R – AI (β = -564.38, p = .02) when sleep restricted. In all ROIs the coefficients suggest decreased variability in inhibitory efficiency when well-rested relative to sleep restricted. The parameters for the fixed effects associated with the ROI by condition interactions suggest there were significant changes in the ROI effects for the sleep restricted condition relative to the well-rested condition in the R – ACC (β = 540.90, p = .03), R – MFG (β = 743.24, p = .00), R – AI (β = 571.29, p = .02), and the R – IPL (β = 767.19, p = .01). Again, we observe all variance components are significant, including the random intercept, random linear slope, and an estimate of the covariance between them. These data suggest that there is significant variability in the random intercept to be explained between individuals (Wald Z = 4.42, p = .00). The linear time slope also varies significantly across individuals (Wald Z = 4.41, p

= .00). In this equation we define the parameterization of the heterogeneous residual variances by estimating two parameters which define the variance as a function of condition. Based on this parameterization we observed the residual variance for the sleep restricted condition (3004774.23; corresponds to observations for the four ROIs in the sleep restricted condition) to be greater than the residual variance in the well-rested condition (132925.81; corresponds to observations for the four ROIs in the well-rested condition), which contradicted the hypothesis of within-group homogenization. In addition, the significant negative covariance between the random slopes and intercepts (Wald Z = -4.41, p = .00), suggests that those who exhibited greater inhibitory efficiency when sleep restricted exhibited less change in terms of inhibitory efficiency compared to when well-rested. To verify that different residual variances be estimated for the residuals we performed a likelihood ratio test to determine whether the model should have homogenous residual variances or heterogeneous residual variances. Based on the results of this test, we reject the null hypothesis that the residual variance is equal for the sleep restricted and well-rested conditions, p = .00, and retain the heterogenous residual variances as our preferred model.

Model 4. For the fourth final model, we decide whether to keep the fixed effects of the ROI by condition interaction in Model 3 by assessing the F-test based on the results of the REML estimation of Model 3. As noted previously, the Type III F-test were significant at p = .02, which indicates that the fixed effect of condition on inhibitory efficiency differs by ROI and we retain the fixed effects associated with the ROI by condition; thus, selecting model 3 as our final model.

Model Diagnostics. We checked the assumptions underlying of our final model. We conducted a Kolmogorov-Smirnov test for normality of the conditional residuals. This test was

significant, D(844) = 0.40, p = 0.00, suggesting that the conditional residuals from this analysis do not appear to follow a normal distribution. We then examined normality using Q-Q plots of the residual and found that most of the data followed a normal distribution fairly well with only a few outliers, suggesting that the residuals followed a fairly normal distribution. In addition, a scatterplot of the conditional residuals against the conditional predicted values showed some asymmetry within each sleep condition group, with the well-rested condition exhibiting less variability than the sleep restricted condition, suggesting that the variance within each group likely differed. Of note the DF is representative of the total "N" included in the model. Given that our study design is within subjects, and the use of a nested model in our analyses, the DF is representative of ICV nested within ROI (n = 4), nested within sleep condition (n = 2) for each subject ID (n = 53). In the dataset, the ICV value is calculated from the Go versus No/Go trials when sleep deprived versus well-rested, therefore the ICV value is repeated within the dataset (n = 2) and trial type is included in the analysis to uniquely identify each ICV value. Based on model diagnostics, the conditional residuals from this analysis appear to follow a normal distribution fairly well and the assumptions of the model were reasonably met.

Exploratory Analyses

For the exploratory whole brain analysis, a series of repeated measure multilevel mixed models were conducted to examine intra-individual differences in neural activation. Areas associated with reward valuation (e.g., PCC, R – OFC, L – OFC, MDB, ACC, R – STM, and L – STM)) were analyzed together in the same model. We then analyzed the remaining ROIs in a final repeated measures multilevel mixed model. Of note during an initial evaluation of the subject data, there were two subject ROIs whose ICV values were significantly greater than other subject ICV values. As such, we chose to exclude the two subject ROIs from the next series of mixed modeling.

Reward Regions Exploratory Analysis

Model 2.1. To test our first hypothesis that the random effects associated with condition for each subject can be omitted from subsequent models, we first fit a model with a loaded mean structure and random-subject intercept. Results from the first model suggest the main effect for condition in each of the specified reward ROIs was not significant, indicating that the average difference in reward sensitivity, as measured by the intraindividual coefficient of variation in beta activation in ROIs associated with reward for the No-go versus Go trials, between the sleep restricted and well-rested condition was not significant, ($F = 2.40$, $p = .12$). We also observed a non-significant main effect for variability in reward activation, ($F = 1.56$, $p = .16$). In addition, the interaction between condition and reward ROIs was not significant, suggesting that condition was not a significant predictor of variability in reward sensitivity, ($F = .65$, $p = .69$). In addition, with respect to the random effects estimate, the residual variance associated with the intercept for each subject (Wald Z = 18.54, $p = .00$) was statistically significant; however, the residual associated with each observation was not statistically significant (Wald Z = 1.54, $p = .06$), suggesting that there is statistically significant variability at the between-subjects level but not the within-subject level. Based on the estimates of the variance components, 3.02% of variability in activation in reward ROIs occurred between subjects (see Table 7).

Model 2.2. In our second step, a random condition effect for each subject was added, allowing the effect of the sleep restricted condition vs. well-rested to vary from subject to subject. In the parameterization of the model, we assume that fixed effects associated with the ROI, ACC, and sleep restriction are set to zero. When we attempt to fit this model, the following warning message appeared in the output: “The final Hessian matrix is not positive definite although all convergence criteria are satisfied. The mixed procedure continues despite this

warning. Validity of subsequent results cannot be ascertained." Although, this warning message is not an indication of a critical error, investigation of the estimates of covariance parameters suggests that the variance of the random effects associated with condition for each subject is redundant. However, due to the inclusion of multiple ROIs per subject in the analysis we chose to retain the random effects associated with condition in the subsequent model, which includes a repeated measures effect that specifies that repeated measures uniquely indexed by levels of condition are collected for each combination of the subject and ROI variables.

Model 2.3. A repeated measures effect that specified repeated measures collected for each combination of the subject and ROI variables were uniquely indexed by levels of condition was added to model 2.2 and convergence was achieved. Consistent with the first model, in model 2.3 the interaction between condition and ROIs associated with reward was not significant, (F = .68, p = .67), suggesting that activation in ROIs associated with reward did not differ by condition. In addition, the main effects associated with condition and ROI were not significant (F = 1.64, p = .21, F = .82, p = .56, respectively). The fixed effects estimates from this model indicate that the effect of reward sensitivity in the R – STM was significantly greater when sleep restricted relative to well-rested (β = 3207.92, p = .03); however, activation in the PCC, R – OFC, L – OFC, L – STM, MDB, and ACC did not differ between conditions. The parameters for the fixed effects associated with the ROI by condition interactions suggest there were not significant changes in the ROI effects for the sleep restricted condition relative to the well-rested condition (all ps > .05).

In this model, the variance components suggest there is significant variation in activation in ROIs associated with reward across subjects (Wald Z = 0.08, p = .05), variation in the influence of sleep condition on reward sensitivity across ROIs associated with reward (Wald Z =

1.71, p = .04), and covariance between the random intercepts and slopes (Wald Z = -1.70, p = .04). We observed residual variance for the sleep restricted condition (100523868.65) was again higher than the residual variance for the well-rested condition (1336457.01), which is in contradiction to the hypothesis of within-group homogenization. We performed a likelihood ratio test to determine whether the model should have homogenous residual variances or heterogeneous residual variances. Based on the results of this test, we reject the null hypothesis that the residual variance is equal for the sleep restricted and well-rested conditions, p = .00, and retain the heterogenous residual variances as our preferred model (see Table 8).

Model 2.4. For the fourth model, we decide whether to keep the fixed effects of the ROI by condition interaction in Model 2.3 by assessing the *F*-test. As noted previously, the Type III *F*-test was not significant at p = .67, which indicates that the fixed effects of condition on reward sensitivity does not differ by ROI. However, we also performed a likelihood ratio test to determine whether the model fit improved after eliminating the interactions. Based on the non-significant results of this test, we retain the interaction effects in the final model and keep model 2.3 as our final model, p = .67 (see Table 8).

Model Diagnostics. We checked the assumptions underlying the final model. We conducted a Kolmogorov-Smirnov test for normality of the conditional residuals, which was significant, D(740) = 0.44, p = 0.00, suggesting that the conditional residuals from this analysis do not appear to follow a normal distribution. We then examined normality using Q-Q plots of the residual and found that most of the data followed a normal distribution fairly well with only a few outliers, suggesting that the residuals followed a fairly normal distribution. A scatterplot of the conditional residuals against the conditional predicted values showed some asymmetry within each sleep condition, with the well-rested condition exhibiting less variability than the sleep restricted

condition, suggesting that the variance within each group likely differed. Based on the model diagnostics, the conditional residuals from this analysis appear to follow a normal distribution fairly well and the assumptions of the model were reasonably met.

Whole Brain Exploratory Analysis

Model 3.1. To test our first hypothesis that the random effects associated with condition for each subject can be omitted from subsequent models, we first fit a model with a loaded mean structure and random-subject intercept. Results from model 3.1 yielded a non-significant *F* ratio for condition ($F = .73$, $p = .40$), suggesting that condition may not be an important predictor of neural activation. Similarly, the *F* ratio for ROI ($F = 1.48$, $p = .13$) and the condition by ROI interaction ($F = 1.27$, $p = .23$) were also not significant. In addition, with respect to the random effects estimate, the residual variance associated with the intercept for each subject was significant (Wald Z = 24.27, $p = .00$). In contrast, the residual associated with each observation did not reach statistical significance (Wald Z = 1.52, $p = .06$). These findings suggest that there is statistically significant variability at the within subject level; however, at the between-subject level there is not significant variability (see Table 9).

Model 3.2. In our second step, a random condition effect for each subject was added, allowing the effect of the sleep restricted vs. well-rested condition to vary from subject to subject. In the parameterization of the model, we assume that fixed effects associated with the ROI, R – LG, and sleep restriction are set to zero. Consistent with the first model, there was no significant condition by ROI interaction effect on neural efficiency, ($F = 1.41$, $p = .16$), nor a significant main effect of condition on neural efficiency ($F = .60$, $p = .44$). In addition, although we observed a significant *F* ratio for ROI in model 1, it did not appear to remain significant in this model ($F = 1.60$, $p = .09$). Regarding the level 2 variance components, findings suggest that

there was significant variability in the random intercept to be explained between individuals (Wald Z = 3.47, p = .00), variability in the linear time slope across individuals (Wald Z = 2.95, p = .00), and covariance between the well-rested intercept and growth status (Wald Z = -3.41, p = .00). The significant negative covariance estimate suggests that those subjects who had lower neural activation experienced higher rates of growth and vice versus such that those who exhibited greater efficiency when sleep restricted exhibited less change in terms of efficiency compared to when well-rested. To test and determine whether the random condition effects can be omitted from Model 3.1, we performed a likelihood ratio test. Based on the significant results (p = .00), we decide to retain the random condition effects as a result of this significant test and reject the null hypothesis; thus, model 3.2 is deemed the preferred model at this stage in the analysis (see Table 10).

Model 3.3. In this step of the analysis, we fit model 3.3 to allow the residual variances to vary for each level of condition by including separate residual variances for the sleep restricted and well-rested condition. This specification allows a more flexible specification of the residual variance by allowing observations at different levels of the condition on the same subject to have different residual variances. Like model 3.2, the main effect for ROI (F = 1.11, p = .35), condition (F =.57, p = .45), and the condition by ROI interaction (F =1.36, p = .19) were not significant. Again, we observe all variance components are significant, including the random intercept, random linear slope, and an estimate of the covariance between them. This data suggests that there is significant variability in the random intercept to be explained between individuals (Wald Z = 2.72, p = .01), the linear time slope across individuals (Wald Z = 2.53, p = .01), and the covariance between the random intercept and slope (Wald Z = -2.68, p = .01). We also observed that the residual variance for the sleep restricted condition (15289804.92) was

higher than the residual variance in the well-rested condition (6561798.46), which is in contradiction to the hypothesis of within-group homogenization. To verify that different residual variances be estimated for the residuals we performed a likelihood ratio test to determine whether the model should have homogenous residual variances or heterogeneous residual variances. Based on the results of this test, we reject the null hypothesis that the residual variance is equal for the sleep restricted and well-rested conditions, $p = .00$, and retain the heterogenous residual variances as our preferred model (see Table 10).

Model 3.4. For the fourth model, we decide whether to keep the fixed effects of the ROI by condition interaction in Model 3.3 by assessing the *F*-test based on the results of the REML estimation of Model 3.3. As noted previously, the Type III *F*-test was not significant at $p = .19$ and we reduce the model by removing the fixed effects associated with the ROI by condition interaction. We performed a likelihood ratio test to determine whether the model fit improved after eliminating the interactions. Based on the significant results of this test, we chose to keep the reduced model, 3.4, as our final model, $p = .00$ (see Table 10).

Model Diagnostics. A Kolmogorov-Smirnov test for normality of the conditional residuals was significant, $D(1266) = 0.43$, $p = 0.00$, suggesting that the conditional residuals do not appear to follow a normal distribution. Q-Q plots of the residual found that most of the data followed a normal distribution fairly well with a few outliers, suggesting that the residuals followed a fairly normal distribution. A scatterplot of the conditional residuals against the conditional predicted values showed some asymmetry within each sleep condition, with the well-rested condition exhibiting less variability than the sleep restricted condition, suggesting that the variance within each group likely differed. Based on model diagnostics, the conditional residuals from this analysis

appear to follow a normal distribution fairly well and the assumptions of the model were reasonably met.

Specific Aim 2: Effects of Vulnerability to Sleep Restriction on Eating Behavior

A series of repeated measures MANOVAs were conducted to test the effect that individual vulnerability to sleep restriction within each ROI has on eating behavior. In addition, given the subjects included in this study were recruited as part of a larger study investigating the effect that sleep restriction has on neural processes associated with inhibitory control and reward in normal weight and overweight/obese adolescents, we ran separate MANOVAs that also investigated whether BMI as well as sex moderated the relationship between vulnerability to sleep restriction and eating behavior.

Consistent with the primary aims of this study a series of repeated measures MANOVA were conducted to test the effect that individual vulnerability to sleep restriction within each reward ROI (e.g., PCC, R – OFC, L – OFC, MDB, R – STM, L – STM, ACC) and each whole brain ROI (e.g., R – SMA, R – LG, R – TG, ILO, R – AG, L – IOG, L – PcG, L – MO, R – Hip, R – Cau, STG, R – TP) has on eating behavior. For additional details, including outcomes from the exploratory analyses and null findings please see Table 12 – 13.

Inhibitory ROIs. The results showed there was no difference between vulnerability groups due to sleep restriction in the R – IPL, R – ACC, R – MFG, and R – AI on overall eating behavior when sleep restricted relative to well-rested. Univariate tests also indicated that there was no vulnerability group effect on individual eating behavior when sleep restricted versus well-rested (see Table 11).

Reward ROIs. Although the interactions between condition and vulnerability group within the L-STM, MDB, ACC, and PCC, respectively, approached significance, no interaction

effects between sleep condition and vulnerability group within ROIs associated with reward met the corrected threshold for significance to predict differences in caloric intake when sleep restricted relative to well-rested (see Table 12).

Whole Brain ROIs. Univariate tests approached significance suggesting that there was a condition by vulnerability group effect within the R – TG on individual eating behavior for protein consumption. In addition, the vulnerability group within the R – Hip effect also approached significance suggesting there were vulnerability group differences in individual eating behavior across conditions for added sugar intake; however, they did not meet the corrected threshold for significance. All other findings within the ILO, R – SMA, R – AG, L – IOG, L – PcG, L – MO, R – Cau, STG, R – TP, and R – LG, as well as the vulnerability group by condition interaction effect on caloric intake were not significant (see Table 13).

Specific Aim 3: Vulnerability to Sleep Restriction and the Moderating Effect of Sex on Eating Behavior

Inhibitory ROIs. In contrast, there was a significant three-way interaction effect between condition, vulnerability group in the R – IPL, and sex on overall eating behavior (F = 2.14, p = .02, η_p^2 = .25). Results from the univariate tests, indicated that there was no sex by vulnerability group effect on individual eating behavior between the two conditions. In addition, we observed a significant interaction between sex and group vulnerability that was predictive of individual eating behavior across the two conditions for total calories (F = 3.37, p = .04, η_p^2 = .13; see Figure 2), and total fat (F = 5.15, p = .01, η_p^2 = .19; see Figure 3) (all ps < .05, see Table 11). Specifically, males that demonstrated increased vulnerability to inhibitory efficiency when sleep restricted relative to well-rested had the greatest intake across all nutritional domains. Conversely, females demonstrated an opposite effect across conditions; those with greater variability in go versus no-

go activation when well-rested relative to sleep restricted consumed the most calories across dietary domains.

In addition, we observed a significant three-way interaction between sex, vulnerability group within the R – MFG, and condition on individual eating behavior when sleep restricted relative to well-rested for sugar intake (F = 3.51, p = .04, η_p^2 = .14; see Figure 4). Inspection of the estimated means suggest that the impact of vulnerability group varied by sex. Males with decreased activation when sleep restricted relative to well-rested consumed more sugar when sleep restricted, with those in the vulnerability group with the least amount of change in activation between conditions consuming the most calories from sugar when sleep restricted and demonstrated the greatest difference in sugar intake. In contrast, females who demonstrated a decrease in vulnerability to inhibitory efficiency when sleep restricted consumed more calories from sugar overall; however, those with increased inhibitory efficiency when sleep restricted had the greatest difference in sugar intake between conditions. Furthermore, results indicated that the interaction between vulnerability group and sex was predictive of differences in eating behavior across conditions for added sugar (F = 3.31, p = .04, η_p^2 = .13; see Figure 5). Females in Group 1, who demonstrated a decrease in vulnerability to inhibitory efficiency when sleep restricted relative to well-rested consumed the most calories from added sugar, followed by females in Group 3, and Group 2. In contrast, males in Group 1 and 3, who demonstrated a change vulnerability to inhibitory efficiency between conditions consumed a similar amount of added sugar across conditions, with males who exhibited relatively consistent inhibitory efficiency between conditions consuming the most calories across conditions.

In contrast, there were no significant interactions between the effect of vulnerability group in the R – ACC and R – AI, and sex on overall caloric intake when sleep restricted relative to well-rested (see Table 11).

Reward ROIs. In separate MANOVAs investigating the moderating effect sex has on the relationship between vulnerability to sleep restriction in bilateral regions of the OFC and STM, as well as MDB, PCC, and ACC and eating behavior when well-rested relative to sleep restricted, no interaction effects met the corrected threshold of significance (see Table 12).

Whole Brain ROIs. Furthermore, although the three-way interaction between sex, vulnerability group within the R – TP, and condition on individual eating behavior when sleep restricted relative to well-rested approached significance for protein intake, they did not meet the corrected threshold for significance. Similar findings were observed suggesting that the interaction between sex and vulnerability group within the R – TP, L – PcG, L – MO approached significance for predicting sugar, added sugar, and total caloric intake, respectively across conditions, but did not meet the corrected threshold for significance. In addition, all results from MANOVAs investigating the moderating effect of sex on the relationship between vulnerability to sleep restriction in the ILO, R – SMA, R – TG, R – AG, L – IOG, R – Hip, R – Cau, STG, and R – LG, and eating behavior were not significant (see Table 13).

Specific Aim 4: Vulnerability to Sleep Restriction and the Moderating Effect of BMI on Eating Behavior

Inhibitory ROIs. Findings from a separate MANOVA suggested that the BMI by vulnerability group within the R – ACC interaction was predictive of differences in overall eating behavior across conditions for total calories (F = 3.14, p = .05, η_p^2 = .13; see Figure 6), carbohydrate (F = 5.33, p = .01, η_p^2 = .20; see Figure 7), sugar (F = 4.26, p = .02, η_p^2 = .16; see

Figure 8), and added sugar intake (F = 4.41, p = .02, η_p^2 = .17; see Figure 9). Inspection of the results suggested that adolescents in the overweight/obese weight category who fell in the vulnerability group (Group 2) demonstrating the least amount of change in inhibitory efficiency consumed the most calories across dietary domains.

All findings from MANOVAs investigating the effect that BMI and vulnerability to sleep restriction in the R – IPL, R – MFG, and R – AI have on eating behavior were not significant (see Table 11).

Reward ROIs. In addition, although the interaction between vulnerability group within the R – STM and BMI approached significance for predicting differences in caloric intake across conditions, no other interaction effects including vulnerability group within bilateral regions of the OFC and STM, as well as MDB, PCC, and ACC and BMI met the corrected threshold for significance. See Table 12 for complete results.

Whole Brain ROIs. Results from a MANOVA investigating the effect that BMI has on the relationship between vulnerability to sleep restriction in the R – SMA and R – TG, and caloric intake approached significance suggesting there was a significant three-way interaction effect between condition, vulnerability group, and BMI on individual eating behavior for total fat consumption; however, they did not meet the corrected threshold for significance. In addition, all results from MANOVAs investigating the effect BMI has on the relationship between vulnerability to sleep restriction in the ILO, R – AG, L – IOG, L – PcG, L – MO, R – Hip, R – Cau, STG, R – TP, and R – LG and eating behavior were not significant. See Table 13 for all results.

Discussion

Findings from research examining associations between sleep restriction and eating behavior in adolescents are equivocal, highlighting the need for experimental studies to

investigate mechanistic factors that may elucidate this relationship. In addition, there is a notable gap in this body of research regarding our understanding of individual differences in this relationship as well as the factors that may cause an individual to be more susceptible to the negative consequences sleep restriction has on weight-related behavior (Krietsch et al., 2019). As such, the aims of this study were two-fold. First, this study examined whether intra-individual variability in neural activation associated with inhibitory control in response to sleep restriction occurs in adolescents. Second, the study evaluated whether the extent of variability in inhibitory efficiency predicted dietary behavior. The study examined ROIs associated with inhibitory control in addition to an exploratory whole brain analysis.

Aim 1: Sleep Duration and Neural Activation

Given the pervasiveness of insufficient sleep in adolescents, with more than half of adolescents regularly sleeping less than the recommended guidelines for sleep, there is a critical need to understand the consequences of insufficient sleep. Further, previous research suggests that inadequate sleep impacts cognitive processes (e.g., inhibitory control, reward valuation) and brain function, and that these consequences vary substantially between individuals. However, much of this research has been conducted in adult samples, demonstrating a visible gap in the adolescent literature. In addition, inadequate sleep is significantly associated with an increased risk of obesity. Alterations in inhibitory control have further been shown to impact food reward and dietary behavior. Therefore, individual variability in inhibitory control attributable to insufficient sleep may be an important mechanism explaining individual differences in sleep-related changes in eating behavior.

Inhibitory ROIs. For the first aim, we sought to investigate the extent to which intra-individual variability in inhibitory efficiency occurs in response to sleep restriction in a sample

of adolescents. Within ROIs associated with inhibition, including the R – ACC, R – MFG, R – AI, and R – IPL, we observed decreased variability in inhibitory efficiency when viewing food images under habitual sleep conditions relative to restricted sleep. In addition, there was a significant ROI by condition interaction effect, suggesting that there were significant changes in all the inhibition-related ROI effects when sleep restricted relative to well-rested. Results from this study help illuminate the effect of sleep restriction on neural mechanisms associated with inhibitory control in adolescents. Adolescence is a crucial stage of development that is characterized by brain maturation and myelination of neurons within the prefrontal cortex (Arain et al., 2013). The prefrontal cortex plays a central role in executive function processes, including inhibitory control (Aron et al., 2014). Successful maintenance of sustained executive function processes is contingent upon obtaining sufficient sleep (Krause et al., 2017; Lo & Chee, 2020). A significant body of literature indicates that chronic sleep restriction impairs function in the prefrontal cortex, and contributes to neurocognitive deficits (Krause et al., 2017). Specifically, neuroimaging research demonstrates that insufficient sleep negatively impacts outcomes during cognitive function tasks that corresponds to alterations in neural activity (Lowe et al., 2017). Findings from previous neuroimaging research in adolescents are comparable. These study findings show that adolescents who obtain short sleep exhibit impaired inhibitory control or suboptimal neural activation in brain regions associated with inhibitory control (Jensen et al., 2019) and have impaired performance on inhibition-related tasks (Duraccio, Zaugg, et al., 2019). Furthermore, studies that include both neuroimaging and cognitive measures have shown that poor performance on cognitive outcome measures following sleep loss corresponds to insufficient recruitment of neural processes within brain regions involved in inhibitory control and executive function such as the prefrontal cortex (Krause et al., 2017; Lowe et al., 2017).

Outcomes from this scholarship suggest that in order to maintain cognitive control similar to when well-rested, adolescents who are sleep restricted may need to recruit additional neural resources to maintain optimal cognitive functioning (Beebe et al., 2009; Demos et al., 2017). These findings are particularly salient given the proportion of adolescents that regularly obtain insufficient sleep (Wheaton et al., 2016). However, the extent of deterioration in neurocognitive functions due to insufficient sleep varies considerably (Krause et al., 2017; Louca & Short, 2014).

Furthermore, we also found significant variability in inhibitory efficiency in beta activation in ROIs associated with inhibition across subjects, variation in the effect of sleep condition on inhibitory efficiency across subjects, and variation in the effect of sleep condition on inhibitory efficiency across ROIs. These findings extend beyond the existing literature to provide evidentiary support that the effect of sleep restriction on inhibitory activation in adolescents varies between and within individuals. Research in this area suggests that sensitivity to sleep restriction varies across individuals, such that individuals with greater vulnerability to sleep restriction have decreased activation in brain regions associated with inhibition (Chuah et al., 2006). These study findings are corroborated by adult studies, which demonstrated a trait-like, phenotypic vulnerability to insufficient sleep on neurobehavioral outcomes and cognitive deficits highlighting evidence for interindividual differences (Dennis et al., 2017; Goel et al., 2015; Goel & Dinges, 2011; Lowe et al., 2017; Van Dongen et al., 2004). Within the adolescent literature, there is considerable evidence illustrating how sleep negatively impacts cognitive functions, including inhibitory control, working memory, and sustained attention (Lo & Chee, 2020; Short & Chee, 2019). However, to our knowledge very few studies have investigated interindividual differences in the impact that insufficient sleep has on inhibition-related neural

activation in adolescents. Findings from the limited existing literature investigating vulnerability to sleep restriction, demonstrate significant individual variability in neurobehavioral measures of sustained attention, processing speed, and reaction time following one night of total sleep deprivation. Specifically, these studies demonstrated that the consequences of inadequate sleep on cognitive performance in adolescents varies between individuals and the extent of impairment on specific cognitive domains varies within individuals (Goel et al., 2015; Louca & Short, 2014). The current study corroborates these findings by demonstrating that, while sleep restriction appears to impair inhibitory efficiency in adolescents, the degree to which sleep restriction impacted neural activation relative to neural activation when well-rested varied considerable between adolescents.

In this study, individuals who exhibited greater inhibitory efficiency when sleep restricted exhibited less change in inhibitory efficiency relative to when they were well-rested. Consistent with previous research these findings suggest that individuals resistant to the effects of sleep restriction are better able to buffer the effects through sustained recruitment of attentional processes in the prefrontal cortex, or other neural mechanisms involved in maintaining attention (Chee & Chuah, 2008). This finding supports the theory that the effect of sleep restriction on inhibitory control may differ between individuals such that there is a subgroup of individuals who appear able to sustain inhibitory control when sleep restricted to a similar degree as when they achieve optimal levels of sleep. In contrast, there also appears to be evidence that a subgroup of individuals are unable to recruit inhibitory processes in the same manner, suggesting they may be more susceptible to the negative effects of sleep restriction on inhibitory control (Nofzinger et al., 2013; Nofzinger, 2006; Stern, 2002). This is one of the first studies conducted with adolescents to demonstrate that the effect of sleep restriction on neural activation associated

with inhibitory control varies substantially between and within individuals and that shifts in neural activation following inadequate sleep may be a useful indicator for identifying subgroups of adolescents most vulnerable to sleep restriction.

Preliminary data from a neuroimaging study in adults suggests that individuals most vulnerable to the effects of insufficient sleep, exhibit less brain activation during a working memory task, both when well-rested and sleep deprived, compared to resilient individuals (Mu et al., 2005). Similarly, findings from Chua and colleagues, demonstrated that variability in behavioral and physiological measures including electrocardiogram, electroencephalogram when well-rested are associated with interindividual differences in sustained attention when sleep deprived (Chua et al., 2014).Therefore, changes in neural activation in response to sleep loss appear to align with neurocognitive outcomes which may provide insight into the neurobiological underpinnings associated with interindividual differences in response to sleep loss (Lowe et al., 2017; Whitney et al., 2019). Although less is understood in terms of potential predictors, there is some evidence that potential biomarkers, including differences in genetics, sleep homeostasis, and circadian rhythm may differentiate individuals most susceptible to the negative effects of sleep restriction; thus the inability to maintain inhibitory efficiency in response to sleep loss may be a useful indicator linking the biological mechanisms that correspond to impairments in neurobehavioral outcomes (Goel et al., 2015; Goel & Dinges, 2011; Lowe et al., 2017; Satterfield et al., 2019; Sletten et al., 2015; Song et al., 2019; Van Dongen et al., 2012; Whitney et al., 2019).

Reward ROIs. Interestingly, we also observed significant variation in activation in ROIs associated with reward valuation (e.g., PCC, R – OFC, L – OFC, MDB, R – STM, L – STM, ACC) across subjects, and variation in the influence of condition on reward sensitivity across

ROIs associated with reward. These findings indicate that insufficient sleep does indeed exert an effect on neural activation in regions associated with reward in response to food images, which aligns with previous findings from neuroimaging studies in adults and adolescents (Demos et al., 2017; Jensen et al., 2019; St-Onge et al., 2012). Specifically, previous studies have shown that brain regions associated with reward processing and valuation demonstrate increased reactivity when sleep deprived relative to well-rested (Krause et al., 2017). In addition results from experimental studies that included a similar sleep paradigm suggest that sleep restricted adolescents not only demonstrate greater sensitivity to food reward (Duraccio, Zaugg, et al., 2019; Jensen et al., 2019), but also consume more calories from foods with a high glycemic index relative to when they are well-rested (Beebe et al., 2013). These findings imply that adolescents who fail to meet sleep recommendations may be at risk for increased consumption of high calorie foods due to increased reward sensitivity, suggesting that increased neural activation in regions associated with food reward may be an important factor responsible for the link between short sleep and obesity risk (Benedict et al., 2012; Demos et al., 2017; Duraccio, Krietsch, et al., 2019; Duraccio, Zaugg, et al., 2019; Lundahl & Nelson, 2015).

Consistent with our primary aim, we also observed a higher residual variance for the sleep restricted condition relative to the well-rested condition. Similarly, we found that adolescents who exhibited greater reward sensitivity when sleep restricted exhibited less change relative to when they were well-rested. This finding supports the hypothesis that the effect of sleep restriction on reward salience may also differ between individuals, highlighting that there are adolescents who appear more sensitive to the effects that sleep restriction has on reward valuation in a manner that is inconsistent to when they achieve optimal levels of sleep. To our knowledge this is one of the first studies in adolescents to illustrate a trait-like response in reward

activation when sleep restricted. Within the limited existing literature, a recent study in adults found that individual differences in reward-related neural processing when well-rested was predictive of differences in eating behavior when sleep restricted, suggesting that variability in reward processes may be a critical component to understand the link between insufficient sleep and obesity risk (Satterfield et al., 2018). In addition, previous research in adolescents and adults suggests that hyper-responsivity of reward-related neural responding in response to a rewarding stimulus is associated with increased risk of subsequent weight gain (Stice et al., 2011; Winter et al., 2017). Specifically, Stice and colleagues, demonstrated that differences in reward activation in response to palatable foods differentiated adolescents at greatest risk for developing obesity (Stice et al., 2011). Although Stice and colleagues did not evaluate sleep restriction in their study, these findings suggest that variability in reward-related neural activation in response to a rewarding stimulus occurs in adolescent populations and may be a useful indicator for identifying sub-groups of individuals that are most vulnerable to maladaptive behaviors (Satterfield et al., 2018; Stice et al., 2011).

Given that much of the previous literature has focused on the effect that vulnerability to restricted sleep has on attention and inhibitory processes, the additional finding that reward-related neural responding is also sensitive to insufficient sleep and exhibits significant variability between and within individuals is of importance. Nevertheless, current findings indicate that insufficient sleep does indeed exert an effect on neural activation in regions associated with reward. Specifically, findings suggest that brain regions associated with reward processing and valuation demonstrate increased reactivity when sleep deprived relative to well-rested (Krause et al., 2017) and, reward sensitivity has been shown to vary between individuals and predict activation in brain regions associated with reward when viewing food images (Beaver et al.,

2006). Furthermore, evidence from adult studies suggests that individual biological factors including sex and trait genetics may influence the effect of insufficient sleep on reward processing (Ahrens & Ahmed, 2020; Greer et al., 2016; Krause et al., 2017; Tkachenko & Dinges, 2018).

Whole Brain ROIs. Regarding our final model, results that included the remaining ROIs included in the whole brain analysis (e.g., R – SMA, R – LG, R – TG, ILO, R – AG, L – IOG, L – PcG, L – MO, R – Hip, R – Cau, STG, R – TP) were consistent with regard to the random intercept, random slope, and the estimate of covariance between them. Consistent with findings from the inhibitory and reward models, our findings demonstrated that the residual variance for the sleep restricted condition was greater than that of the well-rested condition. Specifically, we observed significant variation in activation in the remaining ROIs across subjects, variation in the influence of condition on ROI sensitivity across ROIs, and the covariance between the random intercept and slope. The covariance estimate was again negative, suggesting that adolescents who exhibited greater efficiency when sleep restricted exhibited less change in reward activation relative to when they were well-rested. Thus, these adolescents were able to sustain neural activation in a manner that closely resembles neural activation when they obtain optimal sleep.

Taken together these findings are consistent with previous work from fMRI studies that have shown individuals who exhibit greater activation across the brain when well-rested, are less susceptible to the negative effect that sleep deprivation has on cognitive outcome measures. Findings from this study support the cognitive reserve theory (Nofzinger, 2006), which surmises that individuals are resilient to the effects of insufficient sleep because they have greater neural resources at rested baseline. In other words resilient individuals may have more cognitive

resources readily available at any given time or have the ability to recruit additional resources to counter the impact sleep restriction has on neural processing (Nofzinger, 2006). Interestingly, outcomes and the interpretation of findings from fMRI research investigating vulnerability to sleep restriction vary in terms of how they operationalize neural activation changes that constitute efficiency versus vulnerability. Even so, consistent with previous research, we observed that the extent of deterioration in neurocognitive processes due to insufficient sleep varies considerably and help elucidate the neural underpinnings associated with vulnerability to sleep loss in adolescents.

Aim 2: Vulnerability to Sleep Restriction and Eating Behavior

Relatedly, short sleep duration in adolescents is associated with increased food intake and an increased risk for developing overweight/obesity (Lundahl & Nelson, 2015; Wu et al., 2017). Previous research suggests that the connection between short sleep and obesity may be at least partially explained by the effects of insufficient sleep on dietary behavior (Chaput & Tremblay, 2012; Miller et al., 2015). Therefore, another aim of this study is to determine whether the extent of decline in inhibitory efficiency predicts dietary behavior.

Inhibitory ROIs. For the second hypothesis, we expected that adolescents with greater vulnerability in inhibitory efficiency in neural activation in response to sleep restriction would demonstrate increased dietary intake. Contrary to our hypothesis, we found that vulnerability group within inhibitory ROIs alone was not predictive of differences in dietary consumption. These findings suggest that despite subjects exhibiting differences in the degree of impairment sleep restriction produced in neural activation in regions associated with inhibitory control, there were no differences in caloric intake between conditions. This finding provides evidence for how neural activation in inhibitory regions aligns with eating behavior and suggests other factors

including motivation, sensitivity to hedonic food stimuli, and access to high calorie foods may be useful to better understand how sleep restriction and inhibition-related neural activation contribute to increased caloric intake and risk of subsequent weight gain. In addition, although we observed significant differences in inhibitory efficiency when sleep restricted, based on our sample size we may have not been adequately powered to detect between group differences in caloric intake.

While inhibitory control is an important component to understand the relationship between insufficient sleep and increased obesity risk, it is important to recognize that these neural processes are multifaceted and likely interact with other neural, biological, and environmental factors to influence eating behavior. Given neural processes are not singularly responsible for any one behavioral outcome, it is possible that our analyses, which only considered activation within each region separately, did not capture the complex processes or co-activation involved in dietary decision-making and eating behavior, which may be useful for understanding the relationship between short sleep and obesity risk. With regard to eating behavior, inhibition and reward-related neurocircuitry are tightly coupled and work in concert during dietary decision-making and are both affected by insufficient sleep. Specifically, previous research has shown a significant association between activation in brain regions associated with food reward when attending to palatable food images and increased caloric intake when sleep restricted, suggesting that increased responsivity to appetizing foods in brain regions associated with reward and inhibition in response to poor sleep may be a risk factor for future weight gain (Duraccio, Krietsch, et al., 2019; Lundahl & Nelson, 2015; St-Onge et al., 2012; Yokum et al., 2011). Therefore, it is possible that vulnerability in inhibitory efficiency when sleep restricted may only exert a negative impact on dietary intake in the presence of co-occurring increased

reward salience. Our findings suggest that vulnerability to sleep restriction in inhibitory activation may be a potential mechanism that mediates the relationship between weight status and increased dietary intake that places an individual at increased risk for weight gain.

Although our findings did not meet the threshold for significance, they are relevant and consistent with previous findings in adults and adolescents which demonstrate that sleep loss differentially impacts neurocognitive processes both within and between individuals which has implications on eating behavior. Specifically, previous research in this area suggests that individual variability in neurobehavioral outcomes is predictive of differences in snacking behavior (Powell et al., 2017). Furthermore, individuals demonstrate a stable phenotypic response in eating behavior when sleep deprived that varies substantially between individuals and is stable overtime (Spaeth et al., 2015). Given that restricted sleep has deleterious effects on inhibition and reward-related neural processes, which influence dietary decision-making and eating behavior, and the effect insufficient sleep has on neurobehavioral processes and eating behavior is trait-like and stable overtime, individual variability in neural activation when sleep restricted and well-rested may be a key factor in determining individuals most susceptible for increased caloric intake when sleep deprived.

Aim 3: Vulnerability to Sleep Restriction and the Moderating Effect of Sex on Eating Behavior

Inhibitory ROIs. When considering the moderating effect of sex on the relationship between vulnerability group in the R – IPL, R – ACC, R – MFG, and R – AI, and dietary intake, respectively, there is evidence that sex alone predicted differences in caloric intake across conditions, which is important to consider when drawing conclusions about subsequent interactions. In addition, it is also important to acknowledge limitations with regard to our

sample size prior to discussing the findings from the moderator analyses. Specifically, given the implications a small sample size has on reducing power and increasing the margin of error, the sample size contained within the exploratory analyses presents a considerable limitation in our ability to interpret the findings from the moderator analyses and draw a meaningful conclusion. As such it is important to preface that results from the moderator analyses should be interpreted with caution and subsequent interpretation of results contained in this section are speculative at best. Nevertheless, we observed a significant three-way interaction between condition, vulnerability group in the R – IPL, and sex on overall eating behavior. In addition, we observed a significant interaction effect between sex, vulnerability group within the R – IPL, and condition on overall eating behavior as well as a significant three-way interaction between sex, vulnerability group within the R – MFG, and condition on individual eating behavior when sleep restricted relative to well-rested for sugar intake. Findings also included a significant interaction between sex and vulnerability group within the inhibitory network (i.e., R – IPL, R – MFG), suggesting that the interaction was predictive of individual eating behavior (i.e., total calories, added sugar, sugar, and total fat) across the two conditions. This indicates that the effect of vulnerability to sleep restriction on inhibitory efficiency in the R – IPL and R – MFG differs by sex and is predictive of differences in caloric intake when sleep restricted compared with caloric intake when well-rested. In addition, these finding imply that the effect that vulnerability to inhibitory efficiency has on caloric intake may differ for males and females irrespective of their sleep duration.

In sum, we observed that adolescent males consumed more calories than females irrespective of sleep condition and vulnerability group. With regard to vulnerability, differences in dietary intake between sleep conditions were observed for males and females based on their

ability to maintain inhibitory efficiency. Although we were unable to identify a specific pattern in the effect that vulnerability group and sex had on dietary domains due to being under powered, based on the significant findings from this study it is reasonable to conclude that vulnerability to sleep restriction on inhibition-related neural responding may differentially affect dietary outcomes for males and females. As such an important next step to distill the implications of this interaction on dietary outcomes will be conducting a follow-up study in a larger sample of adolescents.

Findings from this study demonstrate male and female adolescent's dietary intake may differ as a result of their ability to maintain inhibitory efficiency when they obtain insufficient sleep. Several studies in adults have demonstrated that dietary intake when sleep deprived differs for men and women. Specifically, Spaeth and colleagues found that sex was a significant predictor of differences in weight gain when sleep restricted in two separate studies. Following a 5-night laboratory stay where subjects were allowed to sleep 4 hours per night, they found that sleep restricted subjects relative to well-rested controls gained more weight, with sleep deprived males gaining more weight relative to sleep deprived females (Spaeth et al., 2013). In addition, they demonstrated that regardless of sleep condition men not only consumed more calories relative to women but also exhibited the greatest increase in caloric intake when sleep restricted especially during late night hours (Spaeth et al., 2014). Furthermore, a study conducted by this same group of researchers that required participants to complete two separate sleep restriction protocols in a laboratory setting approximately 60 – 2132 days apart demonstrated men who gained a substantial amount of weight during consecutive bouts of sleep restriction also consumed more calories during both sleep restricted conditions. Comparatively, men who either maintained or lost weight also consumed a relatively consistent number of calories during both

sleep restricted conditions. Collectively, these findings provide evidence that men may be more susceptible to the negative consequences that sleep restriction has on caloric intake and subsequent weight gain relative to women. Furthermore, men who exhibit significant changes in weight status and calorie consumption when sleep deprived appear to be most vulnerability to the effects of insufficient sleep (Spaeth et al., 2015). Moreover, the negative consequences of insufficient sleep on eating behavior are phenotypic and stable overtime and differ based on an individual's biological sex.

In addition to potential differences in sex hormones, our study findings add to the existing literature and suggest that sex differences in caloric intake during sleep restriction may be partially driven by differences in the impact that insufficient sleep has on inhibitory processes that drive over consumption and subsequent weight gain. Findings from a recent meta-analysis aimed investigating the effect that sleep restriction has on cognitive functioning, highlights biological sex as an important attenuating factor (Lowe et al., 2017). In this review, they found that men appeared most vulnerable to the negative impact that sleep restriction has on overall cognitive functioning and sustained attention relative to women. Conceptually, the ability to maintain inhibitory efficiency when sleep restricted may be a potential mechanism that is moderated by sex and useful in identifying sub-groups of individuals that may be at risk for weight gain as a result of increased caloric intake when sleep restricted.

Aim 4: Vulnerability to Sleep Restriction and the Moderating Effect of BMI on Eating Behavior

Inhibitory ROIs. We also found the BMI by vulnerability group interaction within the R – ACC was predictive of differences in overall eating behavior (e.g., total calories, carbohydrate, sugar, and added sugar intake) across conditions. Specifically, findings suggested that adolescents

in the overweight/obese weight category who fell in the vulnerability group demonstrating the least amount of change in inhibitory efficiency consumed the most calories across dietary domains. These findings are consistent with previous research which demonstrates that inhibitory efficiency is an important component for differentiating subjects most susceptible to the negative impact that sleep has on food reward and may be integral to understanding how sleep restriction confers increased risk for developing obesity in adolescents. Specifically, our findings corroborate results reported by Jensen and colleagues which suggest that overweight/obese and normal weight subjects exhibit differential activation in inhibition-related brain regions. In this study they found that overweight/obese subjects also exhibited increased activity in reward regions when sleep restricted; however, unlike normal weight subjects they did not demonstrate a co-occurring increase in inhibitory processing (Jensen et al., 2019). Findings from this study and previous literature suggest that overweight/obese individuals who are unable to recruit inhibitory neural processing in response, to counterbalance increased hedonic salience of food when sleep restricted may be most susceptible to alterations in dietary consumption when they obtain insufficient sleep (Jensen et al., 2019).

While research consistently demonstrates that poor sleep is associated with increased responsivity in brain regions associated with food reward and weight status, and may be a risk factor for future weight gain (Demos et al., 2017; Jensen et al., 2019; Stice et al., 2011; Winter et al., 2017; Yokum et al., 2011), the ability to maintain inhibitory control when sleep deprived may be especially important in determining subjects most vulnerable to the negative consequences insufficient sleep has on dietary intake and subsequent weight gain. Taken together, vulnerability to sleep restriction may be a potential mechanism that mediates the relationship between weight status and increased dietary intake that places an individual at increased risk for weight gain.

Strengths of the Current Study

This study has several notable strengths which provide support for the legitimacy of the results discussed. Namely, this study used a within subjects' cross-over design which previous research has shown to be an advantageous approach for increasing the power to detect a true effect. Similarly, in terms of the study design, this study demonstrates the feasibility and acceptability of conducting a within subjects' cross-over design sleep paradigm in adolescents within the home environment. Furthermore, although we did not include weight status as a covariate in our primary analyses, our study sample included subjects of both normal and overweight/obese weight status. While previous research demonstrates a relationship between inadequate sleep and obesity, considerably less research has focused on understanding the mechanisms that may influence this relationship. Similarly, this is one of the first studies in adolescents to investigate how the impact of sleep restriction may differ between and within individuals, which fills a gap in the existing literature much of which has focused on group level analyses. A recent review concluded that the majority of the existing research investigating the relationship between health behaviors and dietary intake are based on cross-section and group level analyses, which fail to account for how these two factors vary individually from day to day (Krietsch et al., 2019). Although previous research in adults has investigated individual vulnerability to sleep loss, there is a relative paucity of research examining variability in neural changes associated with sleep restriction in adolescents (Beebe, 2011). Among the few studies that have investigated this phenomenon in adolescents, the majority of studies utilize indirect measures of executive function, rather than more direct measures, such as functional magnetic resonance imaging (Garner et al., 2017; Louca & Short, 2014).To our knowledge, this study is one of the first to examine intra-individual variability in neural activation during a go/no-go task

using novel fMRI techniques in a sample of adolescents, and the extent to which intra-individual variation in neural activation effects dietary behavior, which provides evidence to help clarify the mechanisms that may confer an increased risk for developing overweight/obesity. Thus, findings from this study help elucidate the neural underpinnings associated with vulnerability to sleep loss in adolescents and how differences in vulnerability to sleep loss for male and female adolescents may be useful in predicting caloric intake.

Limitations of the Current Study

Several limitations to this study should be noted. First, although our sample size is reasonably large relative to other fMRI studies, our sample size is modest for conducting mixture modeling. In addition, subjects for this study were drawn from a larger study that recruited subjects from a community located near a university. Thus, the results of this study may not be generalizable to adolescents from diverse socioeconomic or racial/ethnic backgrounds. There are also limitations associated with the measurement of the variables of interest. First, neural activation in the specified ROIs is only a proxy for inhibition, and these brain regions are responsible for other cognitive and behavioral processes, limiting our ability to conclude that activation in these regions are solely inhibition-related. In addition, limitations associated with the study design were present. Data for this study were collected during two measurement occasions both of which occurred in the morning, which limits our interpretation of whether our findings directly capture intra-individual variability related to time of day. Further, given the impact that circadian rhythms have on alertness timing and the delayed shift in adolescence, it is possible that the timing of our measurement occasions may have impacted alertness and subsequent inhibitory and neural activation (Crowley et al., 2018). While subjects were randomly assigned to condition and the images presented during the food-related go/no-go task

were randomly selected from a pool of images, we did not assess whether subjects were able to recognize each food image or whether randomization order of the sleep conditions affected the results of this study. Lastly, prior to coming into the lab for each study visit and fMRI scan, subjects were instructed to fast except for drinking water. However, we did not assess subjects' subjective hunger prior to being scanned and completing the food-related go/no-go task, which may have influenced their perception and co-occurring neural activation when viewing food images.

Future Directions

The primary focus of this study was on investigating variability in neural activation in response to sleep restriction and its usefulness in predicting caloric intake. Considerable attention in recent research investigating the link between short sleep and obesity points to a need for additional studies to include variability in sleep quality, timing, and circadian rhythm to better understand this relationship as well as data above and beyond caloric intake including meal timing and frequency (Krietsch et al., 2019). In addition, future studies that assess variability in the neural consequences of insufficient sleep using fMRI would benefit from the inclusion of multiple measurement occasions including a baseline evaluation, multiple measurement occasions when sleep restricted and well-rested, and overtime to more thoroughly determine the extent of impact that achieving the recommend number of hours of sleep relative to inadequate sleep has on intra- and inter- individual variability in neural activation. Similarly, much of the research in this area has focused on the adverse effects of acute sleep loss; therefore, an important direction for future direction will be investigating how chronic sleep restriction affects individuals most vulnerable to sleep restriction (Goel & Dinges, 2011). In this same vein, the inclusion of collecting multiple 24-hour dietary recall could provide utility for understanding the

impact that sleep restriction has on diet overtime and has promise to further elucidate the relationship between sleep and obesity risk (Krietsch et al., 2019). fMRI studies in this area may also benefit by including additional objective and self-reported measurements to corroborate underlying neurocognitive processes and advance our understanding of the functions that underlie neural activation; thus, verifying the construct validity of fMRI measurements. The inclusion of such measures may also help identify a clinically useful screening tool to identify individuals at the greatest risk for experiencing negative consequences associated with insufficient sleep. Given the lack of convergence when analyzing ROIs according to their associated functions it may be important to analyze all ROIs within subject in a single model, which may further our understanding of how ROI activation across the brain fluctuates during specific tasks. Lastly, previous research suggests that over activation in inhibitory regions relative to reward regions of the brain may be an important component necessary to counter the impact insufficient sleep has in enhancing the hedonic properties of palatable, nutrient dense foods. As such, future studies that include both inhibitory and reward regions in one model may provide evidence to further elucidate the impact that sleep has on eating behavior and obesity risk.

Study Implications

Study findings suggest that sensitivity to sleep restriction in adolescents varies substantially across individuals such that individuals resistant to the effects of sleep restriction appear better able to maintain sufficient neural activation necessary to support cognitive function. Given the pervasiveness of insufficient sleep in adolescents, and the impact that insufficient sleep has in maintaining neural processes these findings elucidate one potential mechanism that may be useful in identifying a sub-group of adolescents that are particularly

susceptible to the detrimental effect that inadequate sleep has on cognition. Identifying adolescents who are unable to maintain efficient cognitive function when they achieve suboptimal sleep may inform clinical intervention recommendations aimed at improving sleep hygiene. Research in the adult literature suggests that while some individuals are able to sustain cognitive function when sleep restricted similar to when they are well-rested this may only be feasible when sleep restricted for short periods of time (Lowe et al., 2017; Van Dongen et al., 2004). As most adolescents consistently fail to meet the recommended guidelines for sleep duration (Whitney et al., 2019), all adolescents would benefit from receiving sleep hygiene education to promote healthy sleep habits and optimal neural function, particularly in the presence of increased academic pressure and workload.

In addition, the impact that vulnerability to sleep restriction has on caloric intake when sleep restricted versus well-rested varied for males and females. Specifically, male adolescents who appeared unable to maintain inhibitory efficiency appeared most susceptible to overeating and subsequent weight gain when they were sleep restricted. Thus, vulnerability to sleep restriction on inhibition-related neural responding may help to identify adolescents particularly susceptible to dietary overconsumption and subsequent weight gain when sleep restricted. The current findings extend previous research by identifying vulnerability to inhibitory efficiency in neural activation as a mechanism that underlies sex differences in caloric intake and subsequent weight gain. Therefore, individuals sensitive to inhibition-related neural changes resulting from sleep restriction may benefit from interventions to improve sleep as part of weight control interventions.

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Table 1 *ICV by Sleep Condition and Vulnerability Group Descriptives for Inhibitory ROIs*

		ICV_{RW}		ICV_{SR}		Vulnerability Score	
ROI	N	M (SD)	Range	M (SD)	Range	M (SD)	Range
R-ACC							
Group 1	16	88.01(139.27)	1.42 – 461.69	14.39(51.15)	.003 – 205.82	-21170.55(53052.59)	-213010.39 - -2.02
Group 2	18	.54(.64)	.00 – 1.81	.17(.30)	.002 – 1.15	-.47(.64)	-1.88 – .00
Group 3	17	.78(1.79)	.03 – 7.16	35.08(118.85)	.18 – 493.74	39.48 (107.22)	.01 – 331.98
R-IPL							
Group 1	17	28.89(85.40)	.90 – 358.07	1.25(3.16)	.01 – 13.27	-7670.27(30544.59)	-128028.98 - -.59
Group 2	18	.31(.36)	.00 – 1.29	1.44(3.44)	.00 – 14.36	-.08(.21)	-.52 – .25
Group 3	18	4.51(10.90)	.15 – 46.60	2256.34(7507.79)	2.96 – 31378.60	2248.28(6611.25)	1.17 – 28611.10
R-MFG							
Group 1	18	494.66(1300.22)	2.54 – 5198.56	3.37(10.17)	.00 – 43.84	-1836737.20(6269748.93)	-27011469 - -6.20
Group 2	17	.45(.75)	.00 – 2.64	.27(.52)	.00 – 2.00	-.38(.98)	-3.88 – .01
Group 3	18	1.77(4.98)	.01 – 21.58	505.24(1391.61)	1.09 – 4527.92	1662.63(5031.94)	.07 – 20693.81
R-AI							
Group 1	18	51.26(98.13)	1.06 – 336.36	.82(1.98)	.00 – 8.48	-11697.72(29085.99)	-112937.07 - -1.08
Group 2	17	.40(.36)	.12(.13) .00 – 1.02	.18(.13)	.00 – .50	-.22(.29)	-1.01 – .00
Group 3	18	.82(1.49)	.01 – 5.89	596.24(2460.43)	.40 – 10453.96	216.53(795.85)	.01 – 3436.95

Table 2 *ICV by Sleep Condition and Vulnerability Group Descriptive Statistics for Reward ROIs*

		ICV_{RW}		ICV_{SR}		Vulnerability Score	
ROI	N	M (SD)	Range	M (SD)	Range	M (SD)	Range
ACC							
Group 1	18	115.83(237.46)	4.93 – 952.21	4.91(10.83)	.03 – 44.99	-66243.51(212142.78)	-906684.79 - -21.64
Group 2	17	1.75(1.73)	.01 – 6.46	.71(1.34)	.00 – 4.61	-2.84(4.67)	-17.62 – .00
Group 3	18	.73(.98)	.01 – 3.53	635.05(1614.90)	.07 – 6646.00	438.30(973.65)	.00 – 3152.23
PCC							
Group 1	17	733.04(2615.72)	1.57 – 10853.50	1.04(3.22)	.00 – 13.45	-6976414.4(28121844.1)	-1.18E + 8 - -2.46
Group 2	18	.64(.68)	.00 – 1.92	.25(.30)	.00 – 1.10	-.59(.83)	-2.43 – .03
Group 3	18	.56(.62)	.07 – 2.40	39.33(113.77)	.51 – 470.75	74.21(260.97)	.04 – 1124.96

R – OFC							
Group 1	16	28.49(75.83)	.94 – 302.72	.97(1.64)	.00 – 5.60	-6155.31(22354.80)	-91222.53 - -.89
Group 2	18	.49(.47)	.00 – 1.65	.59(1.49)	.00 – 6.34	-.18(.20)	-.56 - .02
Group 3	17	.91(1.32)	.01 – 4.86	1993.67(8176.91)	.28 – 33724.67	9655.83(39156.18)	.02 – 163960.01
L – OFC							
Group 1	17	415.93(782.35)	8.56 – 2924.65	3.35(6.26)	.00 – 19.58	-748149.32(2064434.37)	-8551415.3 - -73.26
Group 2	18	2.10(2.70)	.00 – 9.34	1.13(2.03)	.00 – 8.02	-8.32(16.73)	-57.57 – .01
Group 3	18	4.07(9.37)	.00 – 35.64	6479.06(26061.47)	.26 – 110833.14	220874.95(917077.02)	.01 – 3949138.40
R – STM							
Group 1	17	1347.41(4633.13)	.97 – 18989.09	.51(1.01)	.00 – 3.93	-22017582(85975666.2)	-3.61E+8 - -.93
Group 2	18	.35(.48)	.00 – 2.06	.38(.81)	.00 – 3.30	-.11(.24)	-.86 – .02
Group 3	18	5.93(21.31)	.00 – 91.26	9444.65(38522.61)	.58 – 163702.86	829937.01(3468377.91)	.05 – 14930406.7
L – STM							
Group 1	17	40.43(118.97)	1.96 – 498.28	20.65(85.24)	.00 – 3.92	-14928.17(59180.25)	-248112.52 - -2.98
Group 2	18	.46(.46)	.00 – 1.36	.533(1.34)	.00 – 7.03	-.31(.54)	-1.86 – .00
Group 3	18	.55(.75)	.01 – 3.10	25.25(62.40)	.21 – 248.73	11.84(34.28)	.00 – 149.35
MDB							
Group 1	17	127.50(469.42)	.57 – 1945.88	1.02(1.45)	.00 – 4.27	-223605.60(904071.57)	-3786295.2 - -.31
Group 2	18	.28(.28)	.00 – 1.06	.17(.22)	.00 – .77	-.07(.10)	-.30 – .00
Group 3	18	3.4(8.97)	.04 – 38.16	1227.44(4970.65)	.14 – 21135.39	3573.92(10579.57)	.00 – 40697.43

Table 3 *ICV by Sleep Condition and Vulnerability Group Descriptives for Whole Brain ROIs*

		ICV_{RW}		ICV_{SR}		Vulnerability Score	
ROI	N	M (SD)	Range	M (SD)	Range	M (SD)	Range
ILO							
Group 1	17	123.32(333.27)	1.11 – 1370.82	2.51(3.81)	.03 – 13.50	-118679.96(444266.22)	-1866272.1 - -.53
Group 2	18	.28(.25)	.00 – .72	1.68(2.32)	.00 – 7.72	.06(.19)	-.28 – .36
Group 3	17	6.45(19.59)	.06 – 82.19	435.34(1177.31)	1.23 – 4629.29	2652.77(10243.67)	.37 – 42999.93
R – SMA							
Group 1	23	124.35(573.03)	.01 – 2752.26	1033.82(3473.65)	.00 – 13935.26	-328608.81(1561280.40)	-7571605.6 – 17789.34
Group 2	21	.18(.30)	.00 – 1.30	2.15(4.24)	.00 – 17.77	.07(.18)	-.15 – .69

Group 3	9	1.07(1.84)	.02 – 5.92	304.50(941.50)	1.20 – 2545.86	225.90(617.12)	.27 – 1920.95
R – TG							
Group 1	21	255.49(1118.78)	.26 – 5136.46	2.79(6.54)	.00 – 29.15	-1256770.6(5684797.92)	-26375475 – 9.24
Group 2	19	.22(.23)	.00 – .78	.53(.49)	.00 – 1.66	.05(.10)	-.06 – .31
Group 3	13	.60(.48)	.00 – 1.88	723.93(1638.42)	.00 – 4568.52	285.02(824.51)	.70 – 3036.61
R – AG							
Group 1	17	161.65(560.83)	1.43 – 2332.59	23.58(94.62)	.00 – 390.74	-268532.37(1081283.16)	-4529539.5 - -1.54
Group 2	18	.54(.64)	.00 – 1.94	.36(.51)	.00 – 1.65	-.24(.40)	-1.45 – .01
Group 3	17	.23(.35)	.01 – 1.40	5.91(10.09)	.14 – 41.08	1.64(3.59)	.01 – 13.95
L – IOG							
Group 1	18	7.79(24.86)	.05 – 106.39	.09(.22)	.00 – .96	-643.24(2622.46)	-11303.34 - -.00
Group 2	17	.01(.01)	.00 – .04	.19(.39)	.00 – 1.34	.00(.00)	-.00 – .00
Group 3	18	.41(.94)	.01 – 3.90	97.60(402.98)	.20 – 1712.24	10.77(43.29)	.00 – 186.74
L – PcG							
Group 1	19	2274.52(8499.57)	.87 – 37158.80	1.95(5.04)	.03 – 22.11	-73605904(312233422)	-1.3807E+9 – .18
Group 2	17	.31(.31)	.00 – .82	3.42(12.55)	.00 – 52.06	-.13(.21)	-.64 – .11
Group 3	15	.70(.92)	.02 – 3.19	207.06(758.79)	1.74 – 2949.47	631.98(2384.78)	.40 – 9404.94
L – MO							
Group 1	17	1186.38(4498.31)	1.19 – 18610.19	.85(1.57)	.00 – 6.04	-20451113(82694372.4)	-3.4633E+8 - -1.20
Group 2	19	.60(.81)	.00 – 2.99	.59(.93)	.01(3.07)	-.25(.37)	-1.10 – .09
Group 3	17	2.01(2.53)	.01 – 7.91	1460.66(5851.72)	1.29 – 24167.22	1487.27(5348.42)	.12 – 22506.01
R – Hip							
Group 1	18	18.07(70.70)	.10 – 301.28	.07(.10)	.00 – .43	-5047.49(21085.33)	-90768.56 - -.01
Group 2	17	.03(.03)	.00 – .11	.25(.30)	.01 – .98	.00(.01)	-.00 – .03
Group 3	17	1.81(3.81)	.00 – 15.31	571.06(1856.18)	.90 – 7594.36	2009.28(8031.58)	.122 – 33658.64
R – Cau							
Group 1	18	2374.32(9998.19)	1.54 – 42436.14	.31(.44)	.00 – 1.36	-1.0005E+8(418350457)	-1.8008E+9 - -1.51
Group 2	17	.37(.50)	.00 – 1.84	1.29(2.63)	.01 – 11.10	-.12(.28)	-1.09 – .07
Group 3	18	1.83(2.41)	.08 – 8.69	147.34(428.91)	.08 – 1820.17	925.09(3647.21)	.13 – 15738.75
STG							

Group 1	17	14.89(26.62)	.87 – 113.01	1.12(2.06)	.04 – 8.22	-878.44(3030.09)	-12761.75 - -.72
Group 2	18	.49(.68)	.00 – 2.38	1.07(1.43)	.02 – 5.95	.05(.33)	-.62 – .74
Group 3	18	1.91(2.66)	.01 – 10.02	5852.37(20116.31)	2.76 – 84998.67	1121.60(3430.01)	.75 – 14821.67
R – TP							
Group 1	18	1687.50(4017.48)	.00 – 23621.96	2.68(5.49)	.00 – 20.80	-32810439(129397149)	-5.5799E+8 - -.80
Group 2	17	.25(.45)	.00 – 1.77	6.32(24.76)	.01 – 102.40	-.07(.17)	-.62 – .02
Group 3	18	5.13(14.16)	.01 – 58.69	195.01(675.50)	.90 – 2890.76	294.06(736.35)	.04 – 3002.01
R – LG							
Group 1	18	35.30(111.09)	.29 – 455.15	1.11(3.12)	.00 – 12.88	-12676.64(47645.52)	-205162.67 - -.07
Group 2	17	.04(.05)	.00 – .18	.10(.19)	.00 – .66	-.00(.01)	-.03 – .00
Group 3	17	.39(.84)	.00 – 3.51	2954.52(12126.23)	.15 – 50011.11	1170.53(4681.18)	.00 – 19616.09

Table 4 *Subject Demographic Information*

N	53	(29 Males)
Mean Age (*SD*)	16.51	(1.65)
Grade (% of Total)	28.3	(15 College Freshman)
BMI percentile (*SD*)	53.16	(29.11)
Race (*%* of Total)		
Caucasian	42	(79.2%)
Hispanic	3	(5.7%)
Native American	1	(1.9%)
Asian or Asian American	4	(7.5%)
Other	3	(5.7%)
Gross Annual Income (*SD*)	4.65	(3.21)

Note. Monthly Gross Income was measured in the following <$10,000 increments: 1 = <$9,999, 2 = $10,000 – 19,999, 3 = $20,000 – 29,999, 4 = $30,000 – 39,999, 5 = $40,000 – 49,999, 6 = $50,000 – 59,999, 7 = $60,000 – 69,999, 8 = $70,000 – 79,999, 9 = >$80,000.

Table 5 *Summary of Model 1 - 3*

	Model 1	Model 2	Model 3
Estimation Method	RE/ML	RE/ML	RE/ML

Fixed-Effect Parameter	*Estimate (SE)*	*Estimate (SE)*	*Estimate (SE)*
β_0 (Intercept)	767.19 (180.19)	767.19 (267.73)	767.19 (300.19)
β_1 (R-ACC vs. R-IPL)	-664.69 (212.64)	-523.79 (174.00)	-519.63 (239.58)
β_2 (R-MFG vs. R-IPL)	-585.40 (211.52)	-585.79 (147.00)	-585.40 (238.58)
β_3 (R-AI vs. R-IPL)	-564.38 (211.52)	-564.38 (172.94)	-564.38 (238.10)
β_4 (Condition)	-756.29 (211.52)	-756.29 (290.13)	-756.29 (304.06)
β_5 (R-ACC × Condition)	681.88 (300.73)	548.27 (246.09)	540.90 (244.81)
β_6 (R-MFG × Condition)	743.24 (299.13)	743.26 (244.57)	743.24 (243.31)
β_7 (R-AI × Condition)	571.29 (299.13)	571.29 (244.57)	571.29 (243.31)
Covariance Parameter	*Estimate (SE)*	*Estimate (SE)*	*Estimate (SE)*
σ^2 (Residual variance)	2371236.07 (119996.25)	15851 28.53 (84284.30)	
σ^2_{int}	535296.11 (138362.00	3006541.34 (647796.29)	3273553.10 (741271.13)
$\sigma^2_{int,\ condition}$		2876054.48 (661934.24)	-3291209.1 (746524.97)
$\sigma^2_{condition}$			3331054.93 (755491.03)
$\sigma^2_{sleep\ restricted}$			3004774.23 (223190.23)
$\sigma^2_{well\text{-}rested}$			132925.81 (9837.44)
Model Information Criteria			
-2RE/ML log-likelihood	14760.60	14634.46	13737.70
AIC	14764.60	14640.46	13747.70
BIC	14774.05	14654.65	13771.34
Tests for Fixed Effects	Type III *F*-Tests	Type III *F*-Tests	Type III *F*-Tests
Intercept	$F(1, 72.76) = 6.25$, $p < .01$	$F(1, 49.10) = 2.01$, $p = .16$	$F(1, 48.69) = 1.78$, $p = .19$
Region	$F(3, 781.25) = 4.16$, $p < .01$	$F(3, 707.77) = 5.24$, $p < .01$	$F(3, 362.65) = 2.75$, $p < .05$
Condition	$F(1, 781.26) = 5.88$, $p < .05$	$F(1, 49.19) = 1.37$, $p = .25$	$F(1, 48.79) = 1.22$, $p = .27$
Region × Condition	$F(3, 781.25) = 2.58$, $p < .05$	$F(3, 707.69= 3.47$, $p < .05$	$F(3, 394.66) = 3.50$, $p < .05$

Note. ROI abbreviations and labels contained in this table include: right anterior cingulate cortex (R – ACC), right inferior parietal lobule (R – IPL), right middle frontal gyrus (R – MFG), right anterior insula (R – AI).

Table 6 *Summary of Hypothesis Test Results*

Hypothesis Label	Test	Estimation Method	Models Compared (Nested vs. Reference)	Test Statistic Values (Calculations)	*p*-Value

1	LRT[a]	REML	1 vs. 2	$\chi^2(1:2) = 126.13$ (14760.60 – 14634.46)	.00
2	LRT	REML	2 vs. 3	$\chi^2(2:3) = 677.1$ (14634.46 – 13957.35)	.00
3	Type-III *F*-test	REML	3[b]	$F(3, 394.66) = 3.43$	.02

Note. [a]Liklihood ratio test; the test statistic by subtracting the -2 REML log-liklihood for the reference model from that of the nested model. [b]The use of an *F*-test does not require fitting a nested model.

Table 7 *Model 2.1 - 2.4*

	Model 2.1	Model 2.2	Model 2.3	Model 2.4
Estimation Method	RE/ML	RE/ML	RE/ML	RE/ML
Fixed-Effect Parameter	*Estimate (SE)*	*Estimate (SE)*	*Estimate (SE)*	*Estimate (SE)*
β_0 (Intercept)	217.57 (1015.33)	217.57 (1015.33)	217.57 (1427.02)	861.10 (657.84)
β_1 (PCC vs. ACC)	-203.80 (1414.03)	-203.80 (1414.03)	-203.80 (1947.32)	190.21 (223.13)
β_2 (L-OFC vs. ACC)	1984.32 (1414.03)	1984.32 (1414.03)	1984.32 (1947.65)	119.84 (223.13)
β_3 (L-STM vs. ACC)	-208.53 (1414.03)	-208.53 (1414.03)	-208.53 (1947.65)	-29.20 (223.13)
β_4 (MDB vs. ACC)	199.68 (1414.03)	199.68 (1414.03)	199.68 (1947.65)	4.56 (223.13)
β_5 (R-OFC vs. ACC)	432.89 (1420.97)	432.89 (1420.97)	432.68 (1957.47)	-23.29 (224.21)
β_6 (R-STM vs. ACC)	2990.35 (1414.03)	2990.35 (1414.03)	2990.35 (1947.65)	427.82 (223.13)
β_7 (Condition)	-177.42 (1414.03)	-177.42 (1414.03)	-177.42 (1437.32)	-829.40 (647.560
β_8 (PCC × Condition)	399.18 (1999.75)	399.18 (1999.75)	399.18 (1960.56)	
β_9 (L-OFC × Condition)	-1888.97 (1999.75)	-1888.97 (1999.75)	-1888.97 (1960.56)	
β_{10} (L-STM × Condition)	181.69 (1999.75)	181.69 (1999.75)	181.69 (1960.56)	
β_{11} (MDB × Condition)	-197.68 (1999.75)	-197.68 (1999.75)	-197.68 (1960.56)	
β_{12} (R-OFC × Condition)	-458.50 (2596.55)	-458.50 (2009.55)	-457.89 (1970.43)	
β_{13} (R-STM × Condition)	2596.18 (1999.75)	2596.18 (1999.75)	-2596.18 (1960.56)	
Covariance Parameter	*Estimate (SE)*	*Estimate (SE)*	*Estimate (SE)*	*Estimate (SE)*

σ^2 (Residual variance)	52986603.97 (2858551.72)	52986603.97 (2858551.72)		
σ^2_{int}	1650819.62 (1075613.09)	1650819.62 (1075613.09)	7404372.41 (4379056.53)	
$\sigma^2_{int,\ condition}$		.000000 (.000000)	-7515136.00 (4409048.15)	
$\sigma^2_{condition}$			7632495.44 (4457888.99)	
$\sigma^2_{sleep\ restricted}$			100523868.65 (7982339.23)	
$s^2_{well\text{-}rested}$			1336457.01 (108446.83)	
Model Information Criteria				
-2RE/ML log-likelihood	15280.48	15280.48	14160.29	14164.32
AIC	15312.48	15314.48	14198.29	14190.32
BIC	15386.18	15392.79	14285.82	14250.20
Tests for Fixed Effects	Type III *F*-Tests	Type III *F*-Tests	Type III *F*-Tests	Type III *F*-Tests
Intercept	$F(1, 147.48) = 5.28$, $p < .01$	$F(1, 147.48) = 5.28$, $p < .01$	$F(1, 53.16) = 2.24$, $p = .14$	$F(1, 53.16) = 2.24$, $p = .14$
Region	$F(6, 687.37) = 1.56$, $p = .16$	$F(6, 687.37) = 1.56$, $p = .16$	$F(6, 317.36) = .82$, $p = .56$	$F(6, 311.88) = 1.12$, $p = .35$
Condition	$F(1, 687.38) = 2.40$, $p = .12$	$F(1, 687.38) = 2.40$, $p = .12$	$F(1, 53.16) = 1.64$, $p = .21$	$F(1, 53.16) = 1.64$, $p = .21$
Region × Condition	$F(6, 687.38) = .65$, $p = .69$	$F(6, 687.38) = .65$, $p = .69$	$F(6, 325.79) = .68$, $p = .67$	

Note. ROI abbreviations and labels contained in this table: posterior cingulate cortex (PCC), anterior cingulate cortex (ACC), left orbitofrontal cortex (L – OFC), left striatum (L – OFC), midbrain (MDB), right striatum (R – STM), right orbitofrontal cortex (R – OFC).

Table 8 *Summary of Hypothesis Test Results for Model 2.1 – 2.4*

Hypothesis Label	Test	Estimation Method	Models Compared (Nested vs. Reference)	Test Statistic Values (Calculations)	*p*-Value
1	LRT[a]	RE/ML	2.1 vs. 2.2	$\chi^2(1:2) = 0$	

				(15280.48 – 15280.48)	
				$\chi^2(2:3) = 1120.18$	
2	LRT	RE/ML	2.2 vs. 2.3	(15280.48 – 14160.29)	.00
	Type-III *F*-				
3	test	RE/ML	2.3[b]	$F(6, 325.79) = .68$	.56
				$\chi^2(3:4) = 4.02$	
4	LRT	RE/ML	2.3 vs. 2.4	(14164.32 – 14160.29)	.67

Note. [a]Liklihood ratio test; the test statistic by subtracting the -2 REML log-liklihood for the reference model from that of the nested model. [b]The use of an *F*-test does not require fitting a nested model.

Table 9 *Model 3.1 – 3.4*

	Model 3.1	Model 3.2	Model 3.3	Model 3.4
Estimation Method	RE/ML	RE/ML	RE/ML	RE/ML
Fixed-Effect				
Parameter	*Estimate (SE)*	*Estimate (SE)*	*Estimate (SE)*	*Estimate (SE)*
β_0 (Intercept)	998.62 (479.26)	1186.97 (523.50)	1137.75 (586.90)	501.05 (392.78)
β_1 (R-SMA vs. R-LG)	-497.42 (667.60)	-685.77 (685.22)	-636.55 (763.52)	-157.33 (417.48)
β_2 (R- TG vs. R-LG)	-819.76 (667.60)	-1008.11 (636.59)	-958.89 (763.52)	-220.87 (417.48)
β_3 (ILO vs.-LG)	-822.59 (670.72)	-822.59 (639.31)	-822.59 (766.86)	-222.37 (418.03)
β_4 (R-AG vs. R-LG)	-956.55 (670.72)	-956.55 (639.31)	-956.55 (766.86)	-254.72 (418.03)
β_5 (L-IOG vs. R-LG)	-965.38 (667.60)	-1153.73 (636.59)	-1104.52 (763.52)	-333.61 (417.48)
β_6 (L-PcG vs. R-LG)	-903.96 (674.05)	-901.77 (642.66)	-901.85 (770.83)	302.65 (418.66)
β_7 (L-MO vs. R-LG)	-529.62 (667.60)	-717.98 (636.59)	-668.76 (763.52)	62.13 (417.48)
β_8 (R-Hip vs. R-LG)	-779.52 (670.72)	-779.52 (639.31)	-779.52 (766.86)	-234.32 (418.03)
β_9 (R-Cau vs. R-LG)	-948.06 (667.60)	-1136.41 (636.59)	-1087.19 (763.52)	234.84 (417.48)
β_{10} (STG vs. R-LG)	989.70 (667.60)	801.35 (636.59)	850.57 (763.52)	254.33 (417.48)
β_{11} (R-TP vs. R-LG)	-929.45 (667.60)	-1117.80 (636.59)	-1068.59 (763.52)	77.83 (417.48)
β_{12} (Condition)	-986.49 (667.60)	-1174.84 (685.22)	-1125.63 (685.30)	-222.10 (292.73)
β_{13} (R-SMA × Condition)	539.51 (941.84)	727.86 (897.92)	678.64 (911.36)	

β_{14} (R-TG × Condition)	909.09 (941.84)	1097.44 (897.92)	1048.22 (911.36)	
β_{15} (ILO × Condition)	852.26 (944.06)	852.26 (899.85)	852.26 (914.16)	
β_{16} (R-AG × Condition)	996.54 (944.06)	996.54 (899.85)	996.54 (914.16)	
β_{17} (L-IOG × Condition)	956.04 (941.84)	1144.39 (897.92)	1095.17 (911.36)	
β_{18} (L-PcG × Condition)	1707.54 (946.43)	1705.35 (902.23)	1705.43 (917.49)	
β_{19} (L-MO) × Condition)	898.89 (941.84)	1807.25 (897.92)	1038.03 (911.36)	
β_{20} (R-Hip × Condition)	774.13 (944.06)	774.13 (899.85)	774.13 (914.16)	
b_{21} (R-Cau × Condition)	1743.04 (941.84)	1931.40 (897.92)	1882.18 (911.36)	
β_{22} (STG × Condition)	-996.24 (941.84)	-807.88 (897.92)	-857.10 (911.36)	
β_{23} (R-TP × Condition)	1492.26 (941.84)	1680.61 (897.92)	1631.39 (911.36)	
Covariance Parameter	*Estimate (SE)*	*Estimate (SE)*	*Estimate (SE)*	*Estimate (SE)*
σ^2 (Residual variance)	11696585.024 (481997.99)	10626683.07 (437307.27)		
σ^2_{int}	248550.82 (163262.24)	3673072.02 (1059734.00)	2649277.24 (972717.22)	2625169.47 (967887.90)
$\sigma^2_{int, condition}$		-3537170.65 (1036986.32)	-2685411.02 (1000344.16)	-2660932.24 (995438.44)
$\sigma^2_{condition}$		3406297.56 (1153640.63)23854.47	2722037.64 (1076856.62)	2697182.21 (1072079.20)
$\sigma^2_{sleep restricted}$			15289804.92 (921665.20)	15363071.67 (922426.25)

$\sigma^2_{well\text{-}rested}$			6561798.46 (387190.85)	6575277.72 (388222.46)
Model Information Criteria				
-2RE/ML log-likelihood	23854.47	23804.89	23698.57	23878.52
AIC	23858.47	23812.89	23708.57	23888.52
BIC	23868.72	23833.38	23734.19	23914.18
Tests for Fixed Effects	Type III *F*-Tests	Type III *F*-Tests	Type III *F*-Tests	Type III *F*-Tests
Intercept	$F(1, 116.24) = 6.94$, $p < .01$	$F(1, 37.36) = 2.68$, $p < .11$	$F(1,33.62) = 2.85$, $p = .10$	$F(1,33.65) = 2.86$, $p = .10$
Region	$F(11, 1178.36) = 1.48$, $p = .13$	$F(11, 1181.64) = 1.60$, $p = .09$	$F(11, 550.92) = 1.11$, $p = .35$	$F(1,33.62) = 2.85$, $p = .10$
Condition	$F(1, 1179.68) = .73$, $p = .40$	$F(1, 56.14) = .60$, $p = .44$	$F(1, 42.59) = .57$, $p = .45$	$F(11, 986.00) = .58$, $p = .85$
Region × Condition	$F(11, 1178.06) = 1.27$, $p = .23$	$F(11, 1181.33) = 1.41$, $p = .16$	$F(11, 953.43) = 1.36$, $p = .19$	

Note. ROI abbreviation and labels contained in this table: right supplementary motor area (R – SMA), right lingual gyrus (R – LG), right triangularis (R – TG), inferior lateral operculum (ILO), right angular gyrus (R – AG), left inferior occipital gyrus (L – IOG), left precentral gyrus (L – PcG), left medial operculum (L – MO), right hippocampus (R-Hip), right caudate (R – Cau), superior temporal gyrus (STG), right temporal pole (R – TP).

Table 10 *Summary of Hypothesis Test Results for Model 3.1 – 3.4*

Hypothesis Label	Test	Estimation Method	Models Compared (Nested vs. Reference)	Test Statistic Values (Calculations)	*p*-Value
1	LRT[a]	RE/ML	3.1 vs. 3.2	$\chi^2(1:2) = 49.58$ (23854.47– 23804.89)	.00
2	LRT	RE/ML	3.2 vs. 3.3	$\chi^2(2:3) = 106.31$ (23804.89 – 23698.57)	.00
3	Type-III *F*-test	RE/ML	3.3[b]	$F(11, 953.43) = 1.36$	.19
4	LRT	RE/ML	3.3 vs. 3.4	$\chi^2(3:4) = 179.95$	.00

(23878.52 – 23698.57)

Note. [a]Liklihood ratio test; the test statistic by subtracting the -2 REML log-liklihood for the reference model from that of the nested model. [b]The use of an *F*-test does not require fitting a nested model.

Table 11 *Repeated Measures MANOVA Vulnerability Group within Inhibitory ROIs Predicting Caloric Intake*

	Condition			Condition x Vulnerability Group		
Within Subject Effects	*F*	*p*	η_p^2	*F*	*p*	η_p^2
R – IPL	.84	.55	.11	.80	.65	.10
R – ACC	.76	.67	.10	.15	.99	.02
R – MFG	.81	.57	.10	.79	.66	.10
R – AI	.78	.59	.10	1.57	.12	.18
	Condition x Sex			Condition x Sex x Vulnerability Group		
Within Subject Effects	*F*	*p*	η_p^2	*F*	*p*	η_p^2
R – IPL	1.84	.12	.22	2.14	.02	.25
R – ACC	1.65	.16	.20	.64	.80	.09
R – MFG	1.57	.18	.20	1.70	.08	.21
R – AI	2.82	.02	.30	.84	.61	.12
	Condition x BMI			Condition x BMI x Vulnerability Group		
Within Subject Effects	*F*	*p*	η_p^2	*F*	*p*	η_p^2
R – IPL	.39	.88	.06	.76	.69	.10
R – ACC	.51	.80	.07	.81	.64	.11
R – MFG	.61	.72	.09	1.25	.27	.16
R – AI	.54	.77	.08	1.21	.29	.16

Table 12 *Repeated Measures MANOVA Vulnerability Group within Reward ROIs Predicting Caloric Intake*

	Condition			Condition x Vulnerability Group		
Within Subject Effects	*F*	*p*	η_p^2	*F*	*p*	η_p^2
R – OFC	.76	.61	.10	.78	.67	.10
L – OFC	.79	.59	.10	.75	.70	.10
MDB	.86	.53	.11	.84	.61	.11
R – STM	.84	.55	.11	.90	.55	.11
L – STM	.82	.56	.10	1.35	.21	.16
PCC	.80	.57	.10	2.18	.02	.24

ACC	.76	.60	.10	1.75	.07	.20
	Condition x Sex			Condition x Sex x Vulnerability Group		
Within Subject Effects	*F*	*p*	η_p^2	*F*	*p*	η_p^2
R – OFC	1.51	.20	.19	.63	.81	.09
L – OFC	1.54	.19	.19	.86	.59	.12
MDB	1.75	.14	.21	1.27	.26	.16
R – STM	1.69	.15	.21	.62	.82	.09
L – STM	1.62	.17	.20	.91	.54	.12
PCC	1.74	.14	.21	.70	.74	.10
ACC	2.32	.05	.26	.97	.49	.13
	Condition x BMI			Condition x BMI x Vulnerability Group		
Within Subject Effects	*F*	*p*	η_p^2	*F*	*p*	η_p^2
R – OFC	.64	.69	.09	.72	.73	.10
L – OFC	.68	.67	.10	.67	.78	.09
MDB	.68	.67	.09	1.03	.43	.14
R – STM	.59	.74	.08	.32	.98	.05
L – STM	.59	.74	.08	.91	.54	.12
PCC	.54	.78	.08	.49	.91	.07
ACC	.62	.71	.09	.78	.67	.11

Table 13 *Repeated Measures MANOVA Vulnerability Group within Exploratory ROIs Predicting Caloric Intake*

	Condition			Condition x Vulnerability Group		
Within Subject Effects	*F*	*p*	η_p^2	*F*	*p*	η_p^2
ILO	.76	.61	.10	.93	.52	.12
R – SMA	.63	.71	.08	1.44	.16	.17
R – TG	.89	.51	.11	1.60	.11	.19
R – AG	.79	.58	.10	.55	.88	.07
L – IOG	.77	.60	.10	1.04	.42	.13
L – PcG	.81	.57	.10	1.29	.24	.16
L – MO	.78	.59	.10	.23	.99	.03
R – Hip	.77	.60	.10	.48	.92	.06
R – Cau	.76	.60	.10	.68	.76	.09
STG	.76	.61	.10	.81	.64	.10

R – TP	.75	.61	.10	1.08	.39	.13
R - LG	.79	.58	.10	.43	.95	.06
	Condition x Sex			Condition x Sex x Vulnerability Group		
Within Subject Effects	*F*	*p*	η_p^2	*F*	*p*	η_p^2
ILO	1.58	.18	.20	.77	.68	.11
R - SMA	.90	.50	.12	.29	.99	.04
R – TG	1.91	.10	.23	1.07	.39	.14
R – AG	1.62	.17	.20	.81	.64	.11
L – IOG	2.12	.07	.25	.48	.92	.07
L – PcG	1.90	.11	.23	.64	.80	.09
L – MO	1.53	.19	.19	.51	.91	.07
R – Hip	1.46	.22	.18	1.48	.15	.19
R – Cau	1.58	.18	.20	.62	.82	.09
STG	1.61	.17	.20	1.24	.27	.16
R – TP	2.85	.02	.31	1.33	.22	.17
R – LG	1.54	.19	.19	.56	.87	.08
	Condition x BMI			Condition x BMI x Vulnerability Group		
Within Subject Effects	*F*	*p*	η_p^2	*F*	*p*	η_p^2
ILO	.38	.87	.06	.74	.71	.10
R - SMA	1.18	.34	.15	1.44	.17	.18
R – TG	.25	.96	.04	1.65	.09	.20
R – AG	.54	.78	.08	.83	.62	.11
L – IOG	.57	.75	.08	1.07	.39	.14
L – PcG	.78	.59	.11	1.44	.17	.18
L – MO	.52	.79	.07	.80	.65	.11
R – Hip	.66	.69	.09	.69	.76	.10
R – Cau	.68	.67	.10	.72	.73	.10
STG	.57	.76	.08	1.28	.25	.16
R – TP	.78	.59	.11	1.48	.15	.19
R – LG	.53	.78	.08	.45	.94	.06

Figure 1 A Priori Regions of Interest as Defined by Automated Meta-Analyses

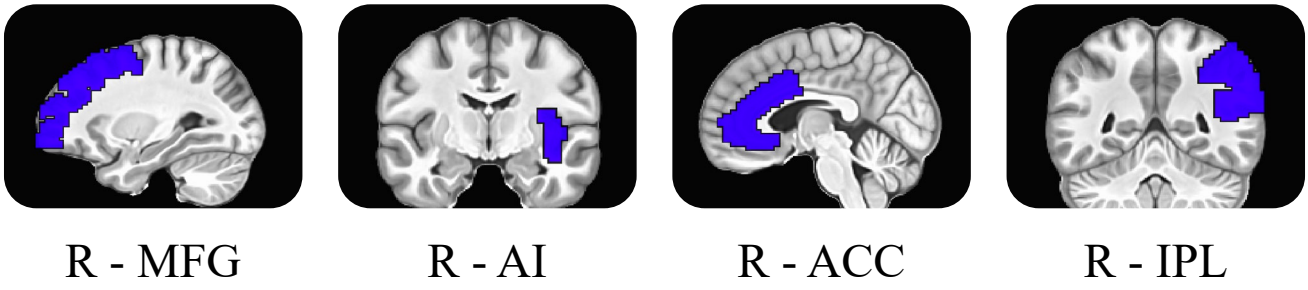

Figure 2 Clustered Bar Mean of Total Fat Intake by Vulnerability Group in R – IPL by Sex Across Conditions

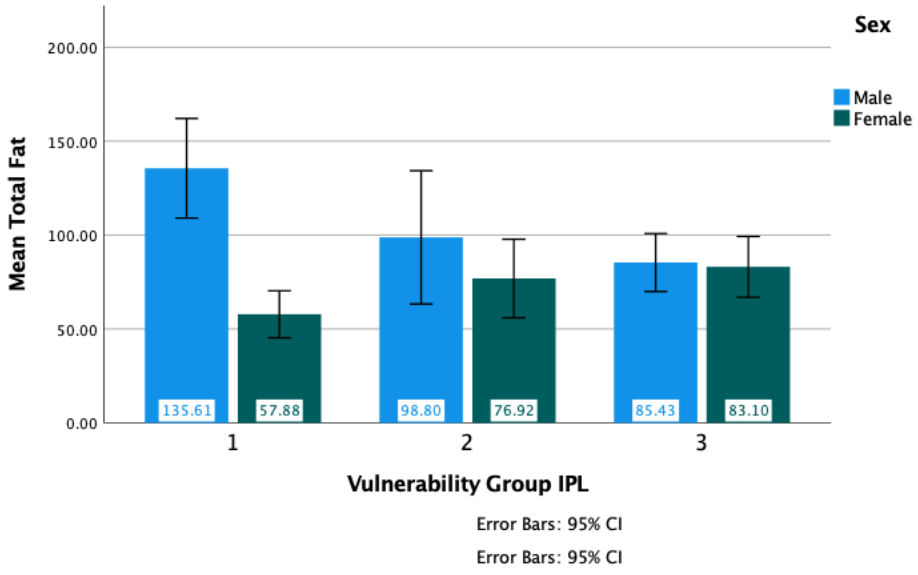

Figure 3 Clustered Bar Mean of Sugar Intake by Vulnerability Group in R – IPL by Sex Across Sleep Conditions

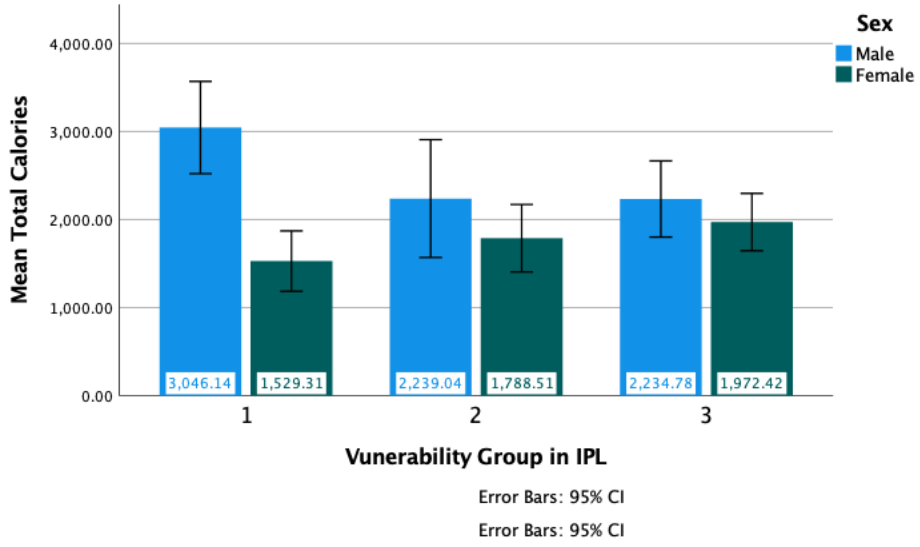

Figure 4 Clustered Bar Mean of Sugar Intake by Vulnerability Group in R – MFG by Sex and Sleep Condition

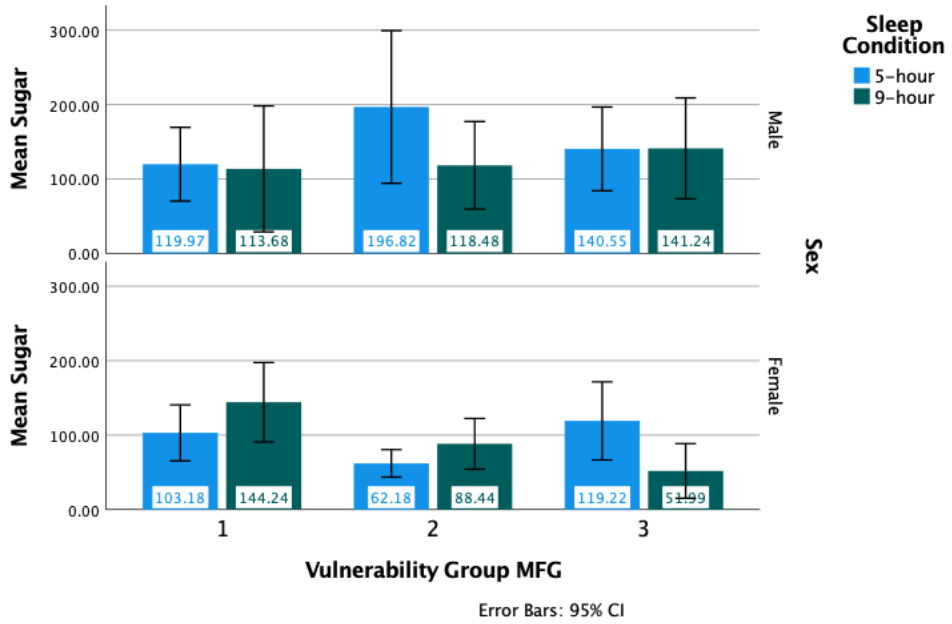

Figure 5 Clustered Bar Mean of Added Sugar Intake by Vulnerability Group in R – MFG by Sex Across Sleep Conditions

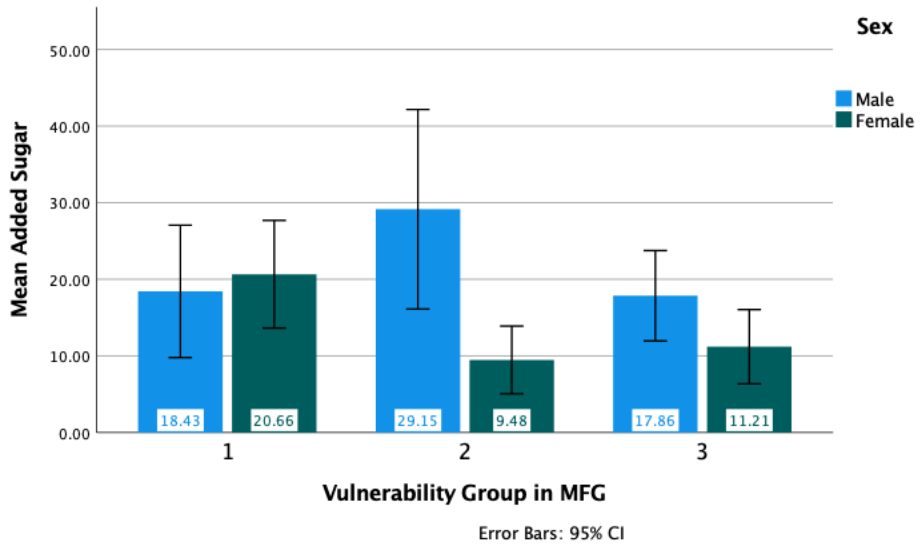

Figure 6 Clustered Bar Mean of Total Calorie Intake by Vulnerability Group in R – ACC by BMI Weight Category Across Sleep Condition

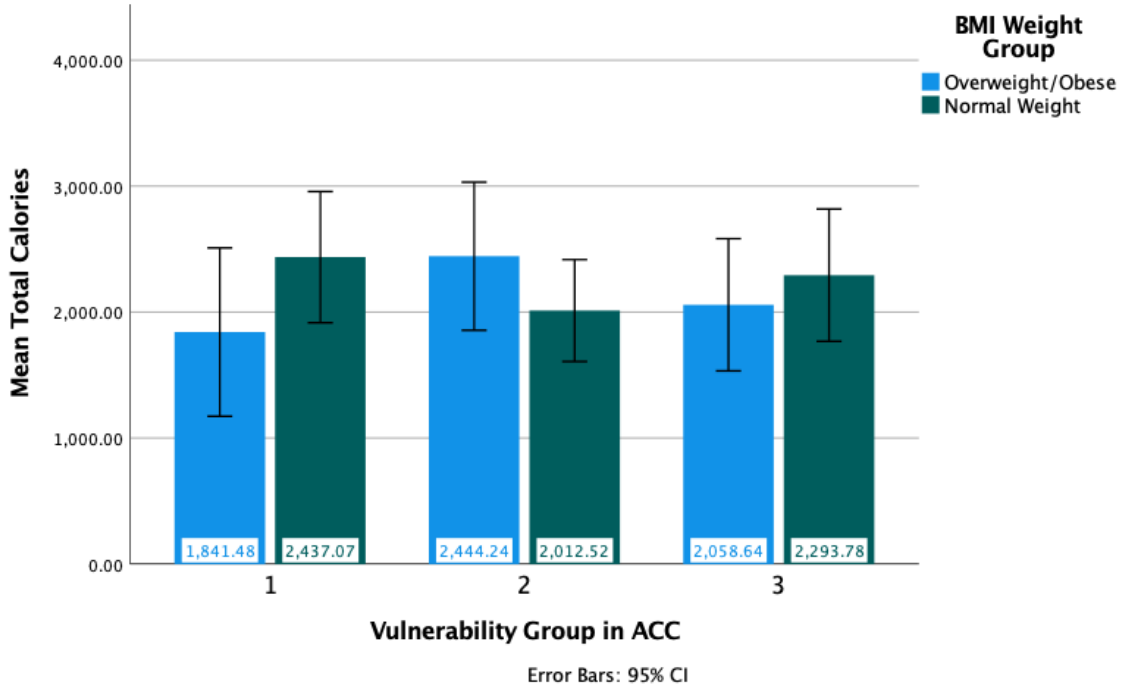

Figure 7 Clustered Bar Mean of Carbohydrate Intake by Vulnerability Group in R – ACC by BMI Weight Group Across Sleep Conditions

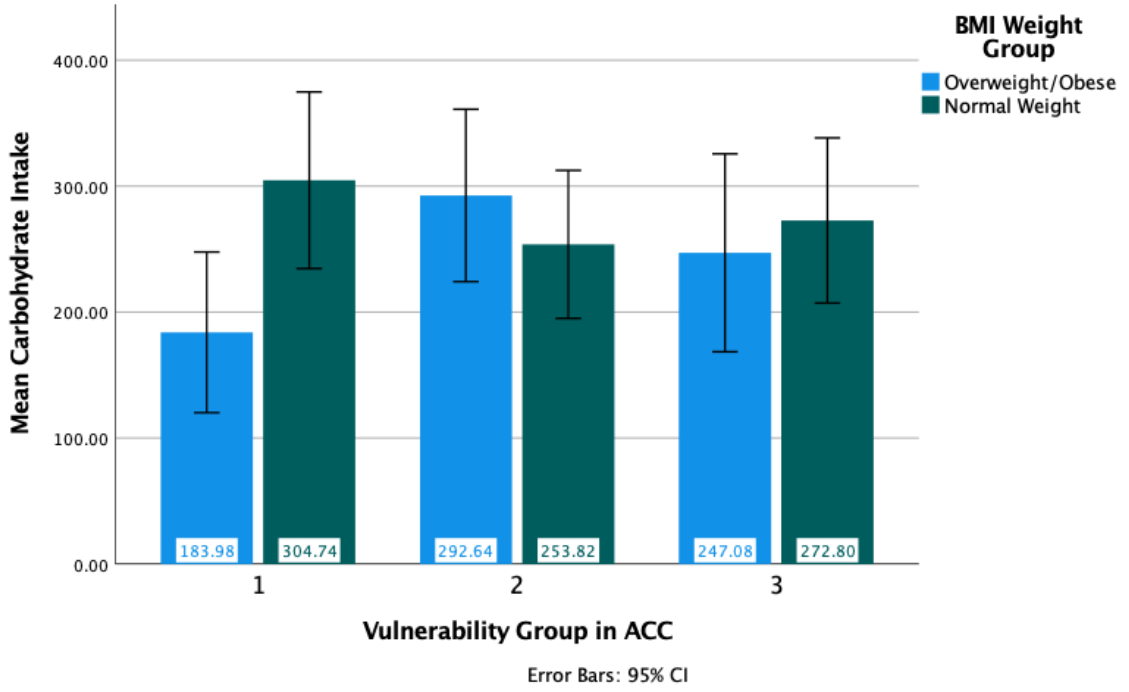

Figure 8 Clustered Bar Mean of Sugar Intake by Vulnerability Group in R – ACC by BMI Weight Category Across Conditions

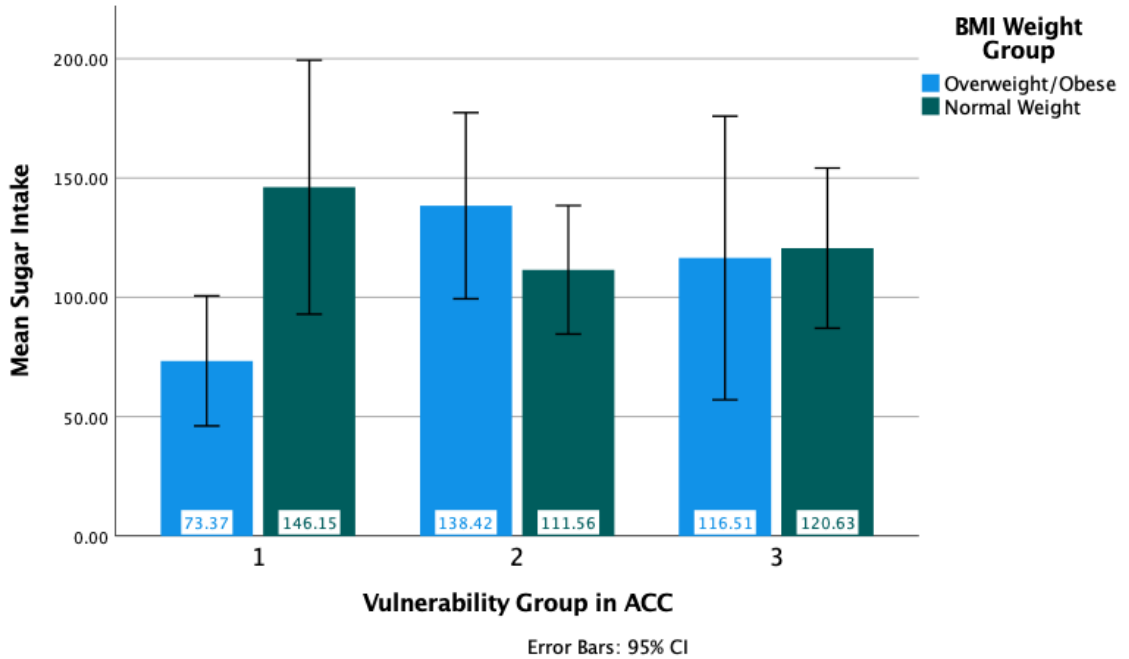

Figure 9 Clustered Bar Mean of Added Sugar Intake by Vulnerability Group in R – ACC by BMI Weight Group Across Sleep Conditions