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The Impact of Sleep Restriction on Food-Related Inhibitory Control and Food Reward in Adolescents: Physical Activity and Weight Status as Potential Moderators

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A dissertation submitted to the faculty of
Brigham Young University
in partial fulfillment of the requirements for the degree of

Doctor of Philosophy

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ABSTRACT

The Impact of Sleep Restriction on Food-Related Inhibitory Control and Food Reward in Adolescents: Physical Activity and Weight Status as Potential Moderators

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Doctor of Philosophy

The present study aimed to evaluate associations between sleep duration and food-related inhibitory control and food reward in adolescents aged 12-18. Potential moderating effects of physical activity and weight status on the association between sleep, inhibitory control, and food reward were also examined. To evaluate these associations, the study employed a two-phase crossover design in which participants spent either 5 hours per night (restricted sleep) or 9 hours per night (habitual sleep) in bed for 5 nights. Participants completed a food-related inhibitory control task and a questionnaire assessing for food reward on the 6th day of each study phase. Repeated measures analyses of variance examined the effect of sleep restriction on food-related inhibitory control and food reward, and explored the moderating impact of weight status and physical activity. Adolescents performed more poorly on a food-related inhibitory control task and have heightened food reward following sleep restriction. Though no differences were noted across weight status in performance of a food inhibitory control task, adolescents with overweight/obesity demonstrated heightened food reward. An interaction between sleep duration and weight status predicted food reward, indicated that normal-weight adolescents are more susceptible to heightened food reward following sleep restriction compared to overweight/obese adolescents. Conversely, overweight/obese adolescents showed consistently high food reward with no effect of sleep duration, suggesting that they consistently view food as rewarding. These study findings may suggest that shortened sleep duration increased food reward for normal weight individuals, potentially putting them at risk for development of overweight/obesity.

Keywords: sleep restriction, inhibitory control, food reward, obesity, physical activity

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The Impact of Sleep Restriction on Food-Related Inhibitory Control and Food Reward in Adolescents: Physical Activity and Weight Status as Potential Moderators

The prevalence of obesity among children and adolescents in the United States has increased drastically over the past twenty years, and currently 20.5% of adolescents meet criteria for obesity (Ogden et al., 2016). Pediatric overweight and obesity are associated with numerous negative physical and mental health outcomes (Sahoo et al., 2015; Vivier & Tompkins, 2008; Zeller & Modi, 2008). The likelihood of obesity being maintained into adulthood increases with age of the child (Guo, Roche, Chumlea, Gardner, & Siervogel, 1994; Whitaker, Wright, Pepe, Seidel, & Dietz, 1997). Thus, adolescence is a critical period for the prevention of obesity in adulthood (Lawlor & Chaturvedi, 2006). Understanding the influencing factors behind eating behaviors and physical activity in adolescents is essential in discovering the possible mechanisms of weight gain.

Sleep and Obesity

The National Sleep Foundation suggests that children obtain 9-11 hours of sleep per night, and that adolescents obtain 8-10 hours of sleep (Hirshkowitz et al., 2015). Short sleep duration predicts overweight and obesity in children and adolescents (Cappuccio et al., 2008; X. Chen, Beydoun, & Wang, 2008; Ekstedt, Nyberg, Ingre, Ekblom, & Marcus, 2013; Miller, Kruisbrink, Wallace, Ji, & Cappuccio, 2018; Patel & Hu, 2008). Children and adolescents who report poorer sleep quality and more sleep disturbances have higher body mass index (BMI) and more total body fat than those who report better sleep quality (Jarrin, McGrath, & Drake, 2013; Lumeng et al., 2007). However, the relationship between sleep and BMI is often argued to be bidirectional. Several longitudinal studies have been conducted to examine the impact of sleep on a child's later BMI. Two-year-old children who slept less than 11 hours per night were more

likely to be obese at age 7 than children who slept 12 hours or more per night (Agras, Hammer, McNicholas, & Kraemer, 2004). Additionally, children who were overweight at age nine reportedly slept about 30 minutes less on average at ages 3-5 (Agras et al., 2004). Bell and Zimmerman (2010) examined sleep duration in young children (ages 0-4 years) and older children (ages 5-13 years) and their weight status five years later found that short sleep duration was associated with increased risk of overweight and obesity in young children.

Several studies have examined the relationship between sleep and obesity within an adolescent population, though findings are somewhat discrepant. In a longitudinal study that examined nearly 15,000 adolescents from 1994-2009, Krueger and colleagues (2015) found that adolescents who slept .5 standard deviations or less than the recommended sleep duration had significantly greater risk of having greater waist circumference compared to their peers who were not sleep restricted. A similar longitudinal study of nearly 10,000 adolescents found that adolescents who slept less than 6 hours per night had a significantly higher risk of developing obesity than adolescents who slept 8 hours or more per night (Suglia, Kara, & Robinson, 2014). However, Roberts and Duong (2015) did not observe a relationship between short sleep and obesity status one year later in a sample of over 4,000 youth. Despite these discrepant findings, a recent meta-analysis aggregating these study findings suggested that shortened sleep duration does increase risk for obesity in an adolescent population (Miller et al., 2018).

One explanation for why restricted sleep leads to increase risk for childhood and adolescent obesity is that youth with short sleep duration will be less likely to engage in physical activity due to feelings of tiredness or exhaustion. Children and adolescents who have efficient sleep patterns are more likely to engage in physical activity during the day (Stone, Stevens, & Faulkner, 2013). Conversely, adolescents with shorter sleep duration engage in greater sedentary

behaviors during the day (Garaulet et al., 2011). Decreased sleep also may lead to increased food intake, either as a function of spending more time awake during which more calories may be consumed or due to decreased inhibitory control and overconsumption of food. Each of these explanations has been confirmed within the adult literature, with findings suggesting that adults who sleep less are more likely to consume a greater amount of total calories throughout the day (Brondel, Romer, Nougues, Touyarou, & Davenne, 2010). Additionally, adults with less sleep have been found to have a decreased inhibitory response when viewing high calorie food images than adults who are well rested (St-Onge et al., 2012; St-Onge, Wolfe, Sy, Shechter, & Hirsch, 2014). The relationship between sleep and eating behaviors in adolescents has been less explored, though Beebe and colleagues (2013) demonstrated that adolescents who underwent sleep restriction (i.e., 6.5 hours in bed for 5 nights) consumed more calories and foods of higher glycemic index than when they obtained habitual sleep (i.e., 10 hours in bed for 5 nights).

Executive Function and Inhibitory Control

One neurocognitive mechanism that may be involved with obesity and food intake is executive function (Nakata et al., 2008). Executive function is an umbrella term that encompasses the higher order cognitive processes responsible for orchestrating thought and action in goal-directed behavior (Banich, 2009; Blaire & Ursache, 2011). Executive functions are called upon when the brain cannot run on automatic processes; executive function includes holding information in one's mind, managing and organizing that information, and resolving conflict between response options (Blaire & Ursache, 2011). There are several working components that are theorized to make up executive function, but the most prominent theoretical framework suggests that executive function is made up of working memory, cognitive flexibility/shifting, and inhibitory control (Diamond, 2006; Miyake et al., 2000).

Inhibitory control is one component of executive function theorized to be a critical cognitive mechanism involved in food intake. Inhibitory control can be defined as one's ability to withhold a dominant response to an external cue in order to correctly respond to one's goals (Ko & Miller, 2013). The inability to withhold the automatic response to eat in the presence of high calorie foods may yield to weight gain (Pauli-Pott, Albayrak, Hebebrand, & Pott, 2010b). Multiple adult studies have demonstrated that weakened inhibitory control results in greater food intake (Guerrieri, Nederkoorn, Schrooten, Martijn, & Jansen, 2009; St-Onge et al., 2012; St-Onge et al., 2014).

Neural circuitry within the prefrontal cortex has been associated with inhibitory control abilities (Shimamura, 2000). Inhibitory control develops in concert with development of white matter in the prefrontal cortex (Verburgh, Königs, Scherder, & Oosterlaan, 2014), with the prefrontal cortex maturing by late adolescence or early adulthood. Similarly, inhibitory control typically matures at some point between adolescence and early adulthood (Best, Miller, & Jones, 2009). As children and adolescents age, they develop greater competence on tasks that assess inhibitory control (Diamond, 2006). Thus, a commonly held belief is that the late development of inhibitory control in adolescence is the result of late maturation of the prefrontal cortex (Verbeken, Braet, Goossens, & van der Oord, 2013). As inhibitory control skills and prefrontal neural circuitry are not always fully developed in adolescents, it is critical to study inhibitory control in relation to health-related behaviors in adolescents.

Body Mass and Inhibitory Control

Body mass index (BMI) in adults has also been shown to correlate negatively with behavioral measures of inhibitory control (Appelhans et al., 2011; Nederkoorn, Smulders, Havermans, Roefs, & Jansen, 2006; Vainik, Dagher, Dubé, & Fellows, 2013). Adults with lower

inhibitory control have been shown to have increased risk of gaining future weight (Anzman & Birch, 2009; Nederkoorn, Houben, Hofmann, Roefs, & Jansen, 2010). These results suggest that low inhibitory control may predispose adults to engage in unhealthy dietary decision making, and that as individuals gain weight inhibitory control continues to decrease.

The relationship between BMI and inhibitory control has only recently begun to be examined in adolescents. Adolescents with overweight/obesity demonstrated poorer performance in behavioral measures of inhibitory control (e.g., decreased accuracy, shorter reaction times), such as in the go/no-go task (Nederkoorn, Braet, Van Eijs, Tanghe, & Jansen, 2006; Pauli-Pott, Albayrak, Hebebrand, & Pott, 2010a). The go/no-go task is a task where participants are asked to respond to the majority of stimuli but required to refrain from responding to a select group of stimuli. For example, the participant may be instructed to press a button when they see the color green (which would account for the majority of trials), but withhold their response when they see the color blue (a small portion of the overall number of trials). Adolescents with overweight/obesity also demonstrate poorer performance in a variety of other inhibitory control tasks (e.g., shifting five digit test, stop task; Verbeken, Braet, Claus, Nederkoorn, & Oosterlaan, 2009; Verdejo-García et al., 2010). Adolescents with overweight/obesity also demonstrated lower inhibitory control when viewing highly palatable foods compared to normal weight adolescents (Batterink, Yokum, & Stice, 2010; Black et al., 2014), as evidenced by shorter reaction times and increased errors in a food go/no-go task. Further, evidence suggests that improvement in inhibitory control facilitates greater reductions in BMI following a multicomponent behavioral weight loss intervention (Delgado-Rico et al., 2012).

Beyond behavioral measurements, functional imaging studies have elucidated key brain alterations in adolescents with overweight/obesity. For example, obese children and adolescents

have shown alterations in activation in brain regions associated with inhibitory control when viewing high calorie, appetizing food images (Batterink et al., 2010; Bruce et al., 2010; Davids et al., 2010; Yokum, Ng, & Stice, 2011), food logos (Bruce et al., 2013), and following the consumption of foods (Bruce et al., 2010; Burger & Stice, 2011; Stice, Yokum, Burger, Epstein, & Small, 2011). Further, alterations in inhibitory brain regions in the presence of food have been shown to predict future BMI gain (Yokum et al., 2011). Similarly, adolescents who have successfully lost weight show increased activation to high-calorie foods in inhibitory regions, perhaps suggesting successful weight losing adolescents have an increase in inhibitory control following weight loss (Jensen & Kirwan, 2015). These findings suggest that overweight and obese children/adolescents have decreased inhibitory control, as measured by both behavioral and functional imaging methodologies, and therefore may be more vulnerable to palatable food cues than normal weight children/adolescents.

Sleep and Inhibitory Control

Sleep deficits may significantly impact higher-order cognitive skills, including inhibitory control. Specifically, adults experiencing 1-2 nights of complete sleep deprivation demonstrated poorer performance during a go/no-go inhibitory task as characterized by having increased button presses, longer reaction times, and poorer overall accuracy (Anderson & Platten, 2011; Chuah, Venkatraman, Dinges, & Chee, 2006; Drummond, Paulus, & Tapert, 2006). Further, adults with obstructive sleep apnea who experience heightened arousal at night have significantly slower reaction times, as compared to adults with obstructive sleep apnea without nighttime arousals (Ayalon, Ancoli-Israel, Aka, McKenna, & Drummond, 2009). Interestingly, inhibitory control (e.g., accuracy of task, reaction times) is found to return to baseline following a night of sleep recovery (Drummond et al., 2006), demonstrating the immediate effect of sleep restriction

on inhibitory control in adults. Furthermore, functional neuroimaging studies confirm that sleep restriction affects prefrontal cortical brain activity in adults, specifically in regions associated with inhibitory control (Durmer & Dinges, 2005; St-Onge et al., 2012; St-Onge et al., 2014). These results suggest that adults who experience sleep deprivation demonstrate difficulty in withholding inappropriate responses. However, little research has examined how prolonged sleep restriction across an extended period of time (as compared to total sleep deprivation over one or two nights) impacts inhibitory control, and no studies to date have examined how sleep restriction impacts inhibitory control around food.

The association between sleep restriction and inhibitory control has been explored in only a few studies involving adolescents. While it has been reported that adolescent inhibitory control does not differ following single night of sleep restriction (four hours of sleep) as compared to a night of full sleep (10 hours of sleep; Fallone, Acebo, Arnedt, Seifer, & Carskadon, 2001), this study did not examine the impact of prolonged sleep restriction on inhibitory control processes. Examining sleep restriction over an extended period of time more closely resembles current sleep habits exhibited by adolescents, with the majority of adolescents obtaining 7 or fewer hours of sleep per night (National Sleep Foundation, 2014). Only two studies have examined the impact of extended sleep restriction on executive functioning in adolescents and young adults. In one study, Beebe and colleagues (2008) reported that adolescents who spent 6.5 hours a night in bed for 5 nights (compared to spending 10 hours in bed for 5 nights) were less attentive and had executive function deficits in areas such as organization, planning, self-monitoring, and self-initiation (Beebe et al., 2008). While the impact of sleep restriction on inhibitory control was not specifically examined, adolescents who slept 6.5 hours a night for 5 nights were more likely to eat foods that were higher in their glycemic content, suggesting a decrease in overall food related

inhibitory control (Beebe et al., 2008). In a more recent neuroimaging study, Demos and colleagues (2017) found that young adults who spent six hours in bed for four consecutive nights demonstrated more neural activation in brain regions associated with inhibitory control while viewing food images (compared to spending nine hours in bed for four consecutive nights).

Physical Activity and Inhibitory Control

Adolescents are encouraged to be physically active for at least 60 minutes every day, according to the recommendations provided by the Centers for Disease Control and Prevention (Centers for Disease Control and Prevention, 2015). Physical activity has numerous physical and psychological health benefits, such as controlling weight, reducing risk for cardiovascular disease, and improving mental health. Additionally, aerobic physical activity has been shown to promote children's inhibitory control (Best, 2010). These positive changes have been demonstrated following single bouts of physical activity (Budde, Voelcker-Rehage, Pietraßyk-Kendziorra, Ribeiro, & Tidow, 2008; Hillman, Buck, Themanson, Pontifex, & Castelli, 2009) as well as after routine training (Davis, Tomporowski, et al., 2011).

Several studies demonstrate that routine physical activity improves inhibitory control. A recent study conducted by Davis, Tomporowski, and colleagues (2011) provided evidence that indicated inhibitory control was sensitive to routine physical activity in children. Overweight children (ages 7-11) receiving an extended physical activity intervention showed significant improvement in tasks requiring inhibition of a prepotent response and demonstrated increased activation in the prefrontal cortex (using an fMRI anti-saccade paradigm task) as compared to the no physical activity condition. Similar findings were observed after a 9-month physical activity intervention in preadolescent children (ages 7-9; Kamijo et al., 2011). A recent study examining the impact of routine physical activity on inhibitory control found that children ages 8-11 who

underwent an 8-month physical activity intervention demonstrated improved performance on an inhibitory task (Krafft et al., 2014). These findings suggest that participating in routine aerobic physical activity positively influences inhibitory control as well as the underlying neural networks associated with inhibitory control.

In addition to routine physical activity, acute bouts of physical activity can produce short-term increases in executive function. Although some researchers have reported that acute treadmill walking had no effect on adolescent's ability to utilize inhibitory control (Soga, Shishido, & Nagatomi, 2015), others demonstrated that acute treadmill walking did have a positive effect on children's inhibitory control (Hillman et al., 2009). Best (2010) suggested that more complex physical activity would have a stronger effect on a child's inhibitory abilities than simpler physical activity. One study that examined this principle was conducted by Budde and colleagues (2008), who demonstrated that adolescents assigned to a 10 minute bout of coordinated physical activity performed better on a response inhibitory control task than adolescents who engaged in uncoordinated physical activity (simply engaging in repetitive movements). The authors suggested that participating in coordinated physical activity called upon greater executive function skills, which enhanced the adolescent's prefrontal neural functioning.

A meta-analysis conducted by Verburgh and colleagues (2014) outlined several theories as to why physical activity may positively influence inhibitory control. One theory states that cerebral blood flow is elevated in the brain during physical activity, which may positively impact cognitive functioning. Another suggests that increases in physical activity are accompanied by increases in lactate threshold levels, which increases certain hormones in the brain that are thought to reflect increased neurotransmitter secretion in the central nervous system. A third

theory is that neural growth and neural survival increase with physical activity; additionally, physical activity is found to promote new blood vessel formation, thus improving the perfusion capacity of the brain. It has been found that children who are physically active frequently have larger brain volumes, suggesting that brain growth may be related to physical activity habits (Chaddock et al., 2010).

Moderating Effect of Physical Activity

Because physical activity appears to positively impact inhibitory control, it is possible that physical activity may prevent the decline in inhibitory control normally seen in individuals who are sleep restricted. While no studies to date have examined the moderating effect of physical activity in the association between sleep duration and inhibitory control, one study evaluated the moderating effect of physical activity on broader executive function outcomes. Lambiase, Gabriel, Kuller, and Matthews (2014) found that women who had greater sleep efficiency performed better on tests of executive function than those with poorer sleep efficiency. However, the authors also found that there was a significant interaction between physical activity and sleep efficiency. Specifically, women with poorer sleep efficiency performed poorly on tests of executive function when they had engaged in low levels of physical activity, but not when they had engaged in higher levels of physical activity. These findings suggest that more physical activity engagement may attenuate the negative impact of poor sleep on executive functioning in older women.

The moderating effect of physical activity on the association between sleep duration and inhibitory control has not been examined in the adolescent population. As executive functioning skills are still being developed in adolescents, understanding the effect of sleep and physical activity on inhibitory control is critical. Furthermore, most adolescents are not meeting the

National Sleep Foundation recommended 9 hours per night (Hirshkowitz et al., 2015), with 58% obtaining less than 7 hours of sleep a night. If the majority of adolescents are failing to obtain sufficient sleep, and as such are likely experiencing inhibitory control deficits, it is essential to understand whether physical activity can counteract the potential decreases in inhibitory control associated with sleep restriction.

Sleep and Food Reward

Although much of the literature examining effects of cognitive processes on weight and dietary decision making examines inhibitory control, a separate body of literature examines food reward processes. If inhibitory control is conceptualized as one's ability to withhold the response to eat a desirable food, food reward is the cognitive process related to how one determines a food to be desirable in the first place. Several adult neuroimaging studies have examined the impact of sleep on food reward. Benedict and colleagues (2012) established that adults who experienced a single night of sleep deprivation had increases in brain activation associated with food reward. To examine the impact of sleep restriction on food reward, St-Onge and colleagues (2012) demonstrated that normal-weight adults who had spent four hours in bed for six nights exhibited heightened neural activity in brain regions associated with food reward when viewing food images compared to when the participants spent nine hours a night in bed. St-Onge and colleagues (2014) went on to demonstrate that adults following the same sleep restriction paradigm demonstrated even greater increases of neural activation in brain regions associated with food reward when viewing unhealthy food images relative to healthy food images. Demos and colleagues (2017) recently replicated these study findings, demonstrating that adults who had undergone sleep restriction (i.e., four nights of six hours in bed) had increases in activation in brain regions associated with food reward, as compared to when the participants were well

rested (i.e., four nights of nine hours in bed). Evidence suggests that shortened sleep duration not only reduces activation in brain regions involved in appetite evaluation, but increases activation in brain regions involved in determining the salience of foods during dietary decision making (Greer, Goldstein, & Walker, 2013). Foods that are perceived to be more salient or rewarding are more likely to be consumed (Wansink, 2004), so assessing for food reward provides information that may help understand dietary decision making. Though several studies have been conducted in the adult literature examining how food reward varies based on sleep length, no study to date has examined the impact of shortened sleep on food reward in adolescents.

One validated measure that has been created to determine the reward-properties of food is the Power of Food Scale (PFS; Lowe et al., 2009). The PFS specifically measures the drive to consume foods that are highly palatable, and individuals who score highly on the PFS have consistently demonstrated higher potential to overeat (Cappelleri et al., 2009; Davis, Curtis, et al., 2011; Forman et al., 2007; Lowe et al., 2009; Ochner, Green, van Steenburgh, Kounios, & Lowe, 2009). Research also has demonstrated that adults with higher PFS scores have increased cravings and decreased feelings of control following a fasting state, as compared to individuals with lower PFS scores (Rejeski et al., 2012). Adults with obesity who demonstrate neural markers associated with overeating (i.e., asymmetrical prefrontal cortex activation) have also been found to have elevated PFS scores (Ochner et al., 2009). Further, adolescents who score higher on the PFS have been shown to have decreased activation in brain regions associated with inhibitory control (Jensen, Duraccio, Barnett, & Stevens, 2016). These study findings suggest that the PFS is related to reward-related neural activation (which has been associated with overeating), and that higher scores on the PFS increase likelihood of overeating. As the PFS has

consistently been related to overeating, the PFS is a useful measure of food reward. No research to date has examined how sleep duration is related to PFS scores.

Body Mass Index and Food Reward

Some evidence also suggests that body mass index (BMI) may also influence food reward. Children with obesity appear to have greater functional brain connectivity from reward regions to various other regions throughout the brain (Black et al., 2014), and obese children have been shown to have increased neural activation in reward regions when viewing food logos, as compared to healthy-weight children (Bruce et al., 2010). Further, Yokum and colleagues (2011) demonstrated that within a population of adolescent females, BMI was positively correlated with activation in regions of the brain associated with food reward. Similarly, Stice and colleagues (2011) demonstrated that youth at risk for developing obesity (i.e., a child of two overweight/obese parents) have increased neural activation in brain regions associated with food reward in response to food intake (compared to youth who were at low risk for developing obesity). While no studies have examined the association between food reward (measured using the PFS) and BMI in an adolescent population, a study conducted in adults found a weak but positive linear relationship with food reward (as measured by the PFS) and BMI (Cappelleri et al., 2009). Further, a study conducted by Appelhans and colleagues (2011) examined how food reward and self-control predicted dietary decision making in overweight adult women. The authors found that high food reward sensitivity (as measured by the PFS) predicted palatable food intake, particularly for individuals with lower levels of self-control. These study findings suggest that food reward varies based on weight status, with individuals who are overweight/obese demonstrating higher food reward than individuals of a normal weight. No

study has examined how food reward (as measured by the PFS) differs based on weight status in an adolescent population.

Current Study

The purpose of this study was to examine the effects of sleep restriction on food-related inhibitory control and food reward in an adolescent population, as well as to explore potential moderating effects of weight status and physical activity on these processes. This study has the potential to address several unexplored topics within the adolescent obesity, sleep, and cognition literatures. Specifically, no study to date has examined the impact of prolonged sleep restriction on adolescent's food-related inhibitory control or food reward. Further, no study has determined if adolescents with overweight/obesity demonstrate differential inhibitory or food reward processes when sleep restricted compared to healthy weight individuals. Finally, no study has examined whether physical activity mitigates the negative impact of sleep restriction on inhibitory control and food reward within an adolescent population. This study has potential to advance knowledge within the obesity literature, with particular relevance to changes in inhibitory control and food reward that may result from sleep duration, physical activity, or weight status. Information from this study will help elucidate mechanisms influential in adolescent decision making around food. Specific study aims and hypotheses are listed below.

Aim 1: The first study aim was to examine the impact of sleep restriction on food-related inhibitory control and food reward.

Hypothesis 1: We hypothesized that sleep restricted adolescents would perform more poorly on a food-related inhibitory control task (Food Go/No-go) compared to their performance following habitual sleep, as demonstrated by poorer accuracy and longer reaction times.

Hypothesis 2: We hypothesized that, when sleep restricted, adolescents would have increased food reward as measured by the PFS, compared to following habitual sleep.

Aim 2: Our second study aim was to examine the moderating effect of weight status (overweight/obese vs. healthy weight) and physical activity on the association between sleep and inhibitory control.

Hypothesis 3: We hypothesized that there would be a moderating effect of weight status on the relationship between sleep condition and inhibitory control, such that adolescents with overweight/obesity would experience greater deficits in a food-related inhibitory control task (Food Go/No-go) following sleep restriction compared to normal weight adolescents.

Hypothesis 4: We hypothesized that there would be a moderating effect of physical activity on the association between sleep condition (restricted vs habitual) and inhibitory control (Food Go/No-go), such that adolescents who engaged in more physical activity would demonstrate better inhibitory control when sleep restricted compared to adolescents who engaged in less physical activity.

Aim 3: Our third study aim was to examine the moderating effect of weight status (overweight/obese vs. healthy weight) and physical activity on the association between sleep and food reward.

Hypothesis 5: We hypothesized that there would be a moderating effect of weight status on the relationship between sleep condition (restricted vs. habitual) and food reward, such that adolescents with overweight/obesity would report higher PFS scores following sleep restriction compared to normal weight adolescents.

Hypothesis 6: We hypothesized that there would be a moderating effect of physical activity on the association between sleep condition (restricted vs habitual) and food reward (PFS), such that

adolescents who engaged in more physical activity would demonstrate lower PFS scores when sleep restricted compared to adolescents who engaged in less physical activity.

Methods

Participants

Thirty-two normal weight (body mass index percentile ≥ 5 and ≤ 85) and thirty-three overweight/obese (body mass index percentile ≥85) adolescent participants (ages 12-18; *M* age = 16.25, *SD* = 1.78) were recruited for this study (see Table 1 for demographic information). One participant with overweight/obesity withdrew from the study following consent procedures. Adolescents with overweight/obesity had significantly higher socioeconomic status (SES) than did adolescents of normal weight ($t(56) = -2.81$, $p < .01$; see Table 1 for SES means by group).

Table 1 *Summary of Demographic and Anthropometric Data, by Weight Class*

	All Participants	Normal Weight	Overweight/Obese
N	64	32	32
Average Age (*SD*)	16.25 (1.78)	16.80 (1.49)	15.74 (1.89)
BMI Percentile (*SD*)	73.01 (26.84)	55.97 (24.64)	91.77 (10.99)
zBMI (*SD*)	.90 (1.01)	.15 (0.74)	1.64 (0.62)
Race (% of Total)			
Caucasian	49 (76.6%)	25 (78.1%)	24 (75.0%)
Hispanic	4 (6.3%)	2 (6.3%)	2 (6.3%)
Native American	1 (1.6%)	1 (3.1%)	-
Asian American	5 (7.8%)	4 (12.5%)	1 (3.1%)
African American	1 (1.6%)	-	1 (3.1%)
Multiracial & Other	4 (6.3%)	-	4 (12.5%)
Yearly Gross Income (*SD*)	7.14 (2.59)	6.27 (2.73)	8.07 (2.09)

Note. Yearly Gross Income was measured in approximately $1,000 increments such that 1 = $0 - $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 +

Participants were recruited using fliers in public locations (e.g., recreation facilities, schools, pediatrician's offices). Exclusion criteria included several variables that have been shown to influence food reward and inhibitory control, including: use of weight loss medication,

history of bariatric surgery, use of medications that affect salivation (e.g., antihistamines, antidepressants), history of an eating disorder, and food allergies. Additionally, as data was collected as part of a larger study that included neuroimaging, exclusion criteria also included left handedness, psychiatric conditions (e.g., epilepsy, traumatic brain injury, schizophrenia, bipolar disorder), and MRI contraindications (e.g., pregnancy, braces, metal implants). Participants with a history of sleep disorders were also excluded, and we ensured that adolescents were engaging in relatively normal sleep patterns, sleeping at least five hours a night and no more than 11 hours per night. We interviewed both the adolescent and the adolescent's primary caregiver via telephone to determine eligibility.

Study Design

The research design consisted of a two-phase crossover study in which participants spent either 5 hours per night (restricted sleep) or 9 hours per night (habitual sleep) in bed. Multi-night, at home sleep manipulations have been shown to be feasible with adolescents (Beebe et al., 2008). Each sleep phase lasted for 6 days, and participants underwent a food inhibitory control task and a food reward task on the 6th day of each period after having fasted for 4 hours. Participants were randomly assigned to either the restricted or habitual sleep condition for the first phase. Wake time for each participant was set as the time it would take the participant to arise to attend a 9:00 am meeting. Bedtimes varied based on what the participants selected as their wake-time, a strategy which has been employed successfully in other studies (Beebe, DiFrancesco, Tlustos, McNally, & Holland, 2009). After a 3-week washout period, participants completed the second phase of the study, alternating to the opposite sleep protocol (i.e. those completing restricted sleep engaged in habitual sleep). The three-week interval between assessments allowed for females to be tested in the similar menstrual cycle phase during both

assessments, assuming a regular 28-day menstrual cycle, as well as allowed for recovery from the previous sleep condition.

Participants received a phone call from study staff each evening of both sleep phases reminding them to adhere to the sleep protocol. Additionally, participants received a text message in the morning after their assigned wake time, instructing participants to respond to this text with their bedtime and wake time. These daily texts served as a sleep diary. Additionally, participants provided a self-report of daily physical activity within these daily texts. Furthermore, participants wore a waist-worn accelerometer (Actigraph GT3X+) during the two 6-day sleep modification periods as a validity check for sleep duration and as a measure of daily physical activity ("Actigraph accelerometers," 2014). Participants completed several online questionnaires throughout both phases of the study.

The first visit with participants took place on the first day in the first phase of the study. Participants and their parents attended this appointment to complete informed consent/assent and to determine randomization of study protocols. Participants were informed of study requirements (e.g., adhering to sleep protocol, not napping during the day, refraining from consuming caffeine, texting study staff each morning), were measured and weighed, completed select questionnaires, and had the accelerometer placed on their dominant hip. Sleep and wake times were determined, and a calendar was provided to each participant outlining study responsibilities to increase compliance across each phase of the study. Following the six-day sleep phase, participants completed a Food Go/No-go task developed by Batterink and colleagues (2010), a task for examining inhibitory control while viewing food images, as well as the PFS (Lowe et al., 2009).

Participants were compensated $25 after each assessment occasion (up to $50 total for imaging). Additionally, they were compensated $3 for every complete day of wearing the

accelerometer, with an additional $7 bonus for wearing the accelerometer successfully for all days at each time point (up to $50 total for accelerometer wearing). An additional bonus of $50 was given for wearing the accelerometer for all study days, adhering to the sleep protocol, and completing both assessments. Participants received a maximum total compensation of $150. The accelerometer bonuses and the final bonus were created to encourage adolescents to wear accelerometers for all days, to adhere to the sleep protocol, and to complete both phases of the study. All study procedures were approved by the BYU Institutional Review Board for human subjects.

Measures

Physical activity measures. Participants wore the Actigraph GT3X+ on the right hip, fastened with an adjustable belt for the six days in each phase of the study. Actigraphs are reported to be well tolerated, comfortable to wear, and not to hinder the adolescent's activities (de Vries, Bakker, Hopman-Rock, Hirasing, & van Mechelen, 2006). The ActiGraph GT3X+ uses a solid-state triaxial accelerometer to measure motion on three separate axes ("Actigraph accelerometers," 2014). Waist-worn accelerometers have been found to yield reliable and valid data of physical activity in a variety of laboratory and free-living settings; additionally, Actigraph accelerometers are able to successfully discriminate the intensity of certain physical activity (Berlin, Storti, & Brach, 2006). Intrainstrument reliability for Actigraph accelerometers ranges from 0.31 (for 1 day of monitoring) to 0.71 (for 4 days of monitoring) to 0.87 (for 7 days of monitoring; Berlin et al., 2006). Additionally, actigraphy data has been shown to have good convergent validity with other measures of physical activity, such as heart rate monitors (0.50 - 0.74), indirect calorimetry (0.87), and direct observation (0.50 - 0.87; Berlin et al., 2006).

Accelerometers were initialized to save the movement data in 15-second intervals (epochs; Pate, Almeida, Pfeiffer, & Dowda, 2006).

Sleep. Data collected from Actigraph GT3X+ accelerometers were used as behavioral measurement of sleep across each phase of the study. Waist-worn accelerometers (as compared to wrist-worn accelerometers) are good at detecting total sleep time and sleep efficiency (with a sensitivity of 98.8-99.7%), but are less sensitive to sleep disturbances than wrist-worn accelerometers (with a specificity of 29.8-46.9%; Hjorth et al., 2012; Takeshima, Echizenya, Inomata, Shimizu, & Shimizu, 2014). For the purpose of this study we only analyzed total sleep duration (i.e., total time spent asleep) and sleep efficiency (i.e., calculated as total time spent asleep divided by total time spent in bed). Additionally, participants were instructed to text study staff each morning to report their bedtime and wake time. These texts were used as a self-report sleep diary that will guide accelerometry cleaning and scoring procedures ("Actigraph accelerometers," 2014).

Inhibitory Control. Adolescents completed a Food Go/No-go task to assess inhibitory control while viewing food images (Batterink et al., 2010). Adolescents completed this task two times, following each sleep modification paradigm. Two runs consisting of 48 trials per run were conducted. For each trial, an image of a healthy food (e.g., broccoli, asparagus, carrots; go trial, 70% occurrence) or an unhealthy food (e.g., cheesecake, cookie, pie; no-go trial, 30% occurrence) was presented for 500 milliseconds. Trials were separated by a fixation cross presented for intervals between 7 and 19 seconds. Participants were instructed to respond with a button press to all healthy foods and to avoid pressing the button when viewing unhealthy foods. Participants were also instructed to respond as quickly and accurately as possible. Reaction times were measured using a fiber-optic response system. Trials were presented in random order.

Response accuracy for both no-go and go trials were calculated (i.e., percentage of correct responses) as well as reaction time for go trials (i.e., time it took the participant to respond to the stimulus). For the purpose of this study, we used response accuracy for go and no-go trails (e.g., the percentage of trials in which the participant correctly responded to a healthy food or inhibited to an unhealthy food) as well as reaction times for the go trials (e.g., the length of time transpired before the participant correctly responded to healthy foods) as the primary outcomes for the study analyses regarding hypotheses 1, 3, and 4.

The go/no-go task has been shown to be associated with the inhibitory regions of the brain, such as the rostral superior medial wall, middle/inferior frontal gyrus, and the bilateral inferior parietal regions (Simmonds, Pekar, & Mostofsky, 2008). Furthermore, go-no/go tasks in general have been found to have strong test-retest reliability (r = .83) and moderate convergent validity with other inhibitory control tasks (Langenecker, Zubieta, Young, Akil, & Nielson, 2007), though the reliability and validity of the Food Go/no-go task developed by Batterink and colleagues (2010) has yet to be established. Data from the Food Go/No-go task will provide indications of how well adolescents are able to inhibit prepotent responses when faced with unhealthy foods.

Weight Status. Weight and height were measured at the initial appointment using a Seca scale and stadiometer. From these estimates, the z-score of Body Mass Index for age and sex (zBMI) and the age and sex corrected body mass index percentile (BMI%) was calculated for each participant. zBMI measurements have been accepted as a reliable indicator of overweight and obesity in children and adolescents (Himes, 2009). zBMI is a moderately reliable indicator of body fat percentage (Mei et al., 2002), and has a moderate inverse relationship with measures of cardiovascular fitness, such that individuals with a higher zBMI are less likely to be

aerobically fit (J. L. Chen, Unnithan, Kennedy, & Yeh, 2008; Joshi, Bryan, & Howat, 2012). BMI was calculated using a standardized formula (BMI = [weight (kg)]/[height (m)]2; Keys, Fidanza, Karvonen, Kimura, & Taylor, 1972), which was then converted to an age- and sex-adjusted z-score and BMI% using the Center for Disease Control and Prevention (CDC) zBMI calculator (Centers for Disease Control and Prevention, 2010). We then used zBMI and BMI% to classify participants into weight status groups, including normal weight (BMI% between ≥ 5 and ≤ 85; zBMI < 1) and overweight/obese (BMI% ≥ 85, zBMI > 1; Centers for Disease Control and Prevention, 2016; Onis et al., 2007). For the analyses outlined below, weight status was used as the moderating variable for hypotheses 3 and 5.

Baseline Executive Function. The Behavioral Rating Inventory of Executive Function – self-report (BRIEF) was completed at the initial appointment prior to sleep modification as a baseline control measure for broad executive function abilities. The BRIEF is an 80-item self-report inventory that assesses executive functioning in children and adolescents (Guy, Isquith, & Gioia, 1996). It contains a list of statements that describe behaviors and then asks if they have had any "problems" with these behaviors in the last six months, rating from 1 ("never a problem") to 3 ("often a problem") (Guy et al., 1996). There are eight clinical scales derived from the BRIEF: inhibition, shift, emotional control, monitor, working memory, plan/organize, organization of materials, and task completion. These scales are grouped into either a Behavioral Regulation Index (BRI) or a metacognition index (MI) which together form the Global Executive Composite (GEC; Guy et al., 1996). Internal consistency ranges from 0.72 to 0.87, with the BRI at 0.93, the MI at 0.95, and the GEC at 0.96. The GEC of the BRIEF was utilized to determine whether broad executive functioning was related to food-related inhibitory control or food reward following sleep modification.

Food Reward. We administered the Power of Food Scale (PFS) to each participant following each sleep modification phase. The PFS is a measure of the psychological influences of the food environment (Lowe et al., 2009). Specifically, this is a measure of the appetite-related thoughts, feelings, and motivations in the environments where palatable foods are available. Based off of exploratory and confirmatory factor analyses, three primary factors exist within the measure: food available, food present, and food tasted (Cappelleri et al., 2009; Lowe et al., 2009). Questions include "If I see or smell a food I like, I get a very strong desire to have some" or "Just before I taste a favorite food, I get very excited" (See Appendix A for a complete list of items included in the PFS). Questions are rated on a 5-point likert scale, with answers ranging from "I don't agree at all" to "I strongly agree." The PFS has excellent internal consistency ($\alpha = 0.91$), and a four-month test-retest reliability was adequate ($r = 0.77$; Cappelleri et al., 2009). The PFS has adequate convergent validity with other measures of eating behaviors, such as measures that tap into cognitive restraint around food and hunger (correlations ranging from 0.54 - 0.66; Cappelleri et al., 2009). The PFS has also been recently validated in an adolescent sample (Mitchell, Cushing, & Amaro, 2016). Additionally, a recent study conducted by Jensen and colleagues (Jensen, Duraccio, Carbine, Barnett, & Kirwan, 2016) found that adolescents with higher PFS food-available scores demonstrated decreased neural activation in brain regions involving behavioral inhibitory control after viewing images of high-calorie foods. As such, the PFS completed following the habitual sleep condition was included as a potential covariate to determine if food perception is related to food-related inhibitory control. Additionally, the PFS total score was used as primary outcomes for hypotheses 2, 5, and 6.

Analytic Plan

Physical Activity Accelerometry Cleaning and Scoring. To obtain physical activity measurements from actigraphy data, data were downloaded in 15-second epochs and converted to a .csv format through ActiLife5. MeterPlus4.3 was used to both clean and score the data. Participants needed to have at least 10 valid hours of recorded physical activity to be considered a valid wear day (60 consecutive minutes of non-movement was defined as an invalid hour; "Actigraph accelerometers," 2014). Physical activity was downloaded in an hourly format. Cut points for adolescents ages 12-18 for sedentary, light, moderate, and vigorous physical activity were set at 0-25 counts/15s epoch, 26-555 counts/15s epoch, 556-1034 counts/15s epoch, and 1035+ counts/60s epoch, respectively (Freedson, Pober, & Janz, 2005). For this study, daily values of moderate and vigorous activity were added together to create a daily physical activity variable. These daily physical activity variables were then averaged across each condition week to create a "physical activity" variable for each condition.

Sleep Accelerometry Cleaning and Scoring. To obtain total sleep duration measurement from actigraphy data, data was initialized in 60-second epoch intervals utilizing the ActiLife5 software. Consistent with the ActiLife5 manual instructions, sleep time was established by utilizing adolescent report of bedtime and wake times (as reported via text sleep diaries). For instances in which the adolescent failed to provide bed/wake times, research staff marked bedtime as when physical activity nearly ceased in the evening (downward decline of physical activity) and marked wake time by visually marking when a participant engaged in a noticeable amount of physical activity in the morning ("Actigraph accelerometers," 2014). If no movement was recorded during the night, it was assumed that the accelerometer was removed prior to bedtime and the data were excluded. A sleep report was generated for each participant

using the sleep algorithm developed by Sadeh and colleagues (Sadeh, Sharkey, & Carskadon, 1994). The report included sleep latency, sleep duration, sleep efficiency, minutes awake after sleep onset, the number of awakenings, and the average awakening length. Consistent with previous research, we used the sleep duration variable to assess whether the adolescent was adherent to the sleep protocol. Furthermore, we compared sleep efficiency across weight status, as obesity has been shown to be related to poorer sleep efficiency in children (Chamorro et al., 2014).

Compliance with Statistical Assumptions. Before conducting our statistical analyses, we ensured that certain statistical assumptions are met. An *a priori* power analyses conducted using G*Power 3 revealed that for our primary hypotheses (i.e., examining how sleep duration impacts food-related inhibitory control and food reward, with no interactions included) a sample of 54 individuals allowed for .95 power and a .05 alpha to detect a small effect size of .25, suggesting that our sample size is adequate to evaluate this study hypotheses. Power analyses also revealed that a sample size of 54 was appropriate for examining between-subjects (i.e., weight status) and within-subjects (i.e. sleep condition) interactions. In addition to ensuring that we have an adequate sample size, we checked for normality in our dependent variables across groups (i.e., go/no-go response accuracy, go reaction times, PFS) using Shapiro-Wilk test for normality. Accuracy of no-go trials in the sleep restricted condition was slightly right skewed and the accuracy of go-trials in the habitual sleep condition was also slightly right skewed. Furthermore, RT during both the sleep restricted and the habitual sleep condition was slightly left skewed. We did not transform the data, as transforming the data did not improve skewness, and the skewness observed appeared representative of the data. All other dependent variables

were found to be normally distributed. No kurtosis was observed for dependent variables. Further, all dependent variables were relatively linear and homoscedastic.

Because our statistical models are sensitive to outliers, we ensured that there are no significant univariate or multivariate outliers within our data. We observed three low outliers in the accuracy of go trials in the sleep restricted condition, three low outliers in the accuracy of go trials in the habitual sleep condition, three low outliers in the accuracy of no-go trials in the habitual sleep condition, three high outliers in the reaction time during the sleep restricted condition, and six high outliers in the reaction time during the habitual sleep condition. No other significant outliers were observed. All outliers detected were fenced to fall within two standard deviations above or below the median. We conducted all study analyses outlined before and after fencing outliers, and no study findings were significantly different following the fencing of outliers. As such, we maintained the fencing of the outliers. No multivariate outliers were observed.

Participant compliance also varied by task. One participant in the sleep-restricted condition and a separate participant in the habitual sleep condition did not complete the PFS. We were unable to gather physical activity from four participants during the sleep-restriction condition and from five participants from the habitual sleep condition. We were also unable to determine sleep efficiency from six participants in the sleep restricted condition, and from eleven participants in the habitual sleep condition. Finally, we were unable to download data from eleven participants for the go/no-go task.

Analyses for Hypotheses. The first study hypothesis examining how sleep restriction impacts food-related inhibitory control was tested by utilizing two trial (no-go, go) x two sleep condition (restricted, habitual) repeated measures analysis of variance (ANOVA). Repeated

measures ANOVAs allows for independent variables to be categorical or continuous, can test the main effects between and within subjects (as well as the interactions between main effects), and test for covariate effects and the interactions between covariates and between subject factors. Two repeated measures ANOVAs were conducted, one with the dependent variable as go/no-go response accuracy and the other with the dependent variable as go reaction time.

The second study hypothesis examining how sleep restriction impacts food-related reward processes was tested using a two-sleep condition (restricted, habitual) repeated measures ANOVA. Further, to examine the moderating impact of weight status and physical activity as outlined in the second and third study aims (for hypotheses 3-6), weight status (normal weight, overweight/obese) was included as a between-subject independent variable and physical activity was included as a within-subjects independent variable in each ANOVA outlined above. A series of bivariate regressions were conducted to determine which covariates will be included in the repeated measures ANOVAs; specifically, age, baseline executive functioning, and gender were all regressed onto the dependent variables of go/no-go response accuracy, go response time, and PFS scores. A bivariate regression was also conducted to determine if baseline PFS scores significantly regressed onto the dependent variable of go/no-go response accuracy and go response time. Any significant regressors were included as covariates in our final repeated-measures ANOVAs.

Finally, we conducted a two-sleep condition (restricted, habitual) by two-weight status (normal-weight, overweight/obese) repeated measures ANOVA to determine how sleep condition and weight status influenced physical activity levels. We also conducted a two-sleep condition (restricted, habitual) by two-weight status (normal-weight, overweight/obese) repeated

measures ANOVA to determine how weight status and sleep condition influenced sleep efficiency. Please refer to Table 2 for study hypothesis and corresponding statistical analyses.

Table 2 *Hypotheses and Corresponding Statistical Analyses*

Hypothesis	Statistical Analysis	Dependent Variable
Hypothesis #1: Inhibitory control will decrease following sleep restriction	Two trial (no-go, go) x two sleep condition (restricted, habitual) repeated measures analysis of variance (ANOVA)	Go/No-Go Accuracy Go Reaction Time
Hypothesis #2: Food reward will increase following sleep restriction	Two-sleep condition (restricted, habitual) repeated measures ANOVA	Power of Food Scale Total Score
Hypothesis #3: Weight status will moderate the relationship between sleep condition and inhibitory control	Two-trial (go, no-go) by two-sleep condition (restricted, normal) by two-weight status (normal-weight, overweight/obese) repeated measures ANOVA with a within-subjects factor of physical activity	Go/No-Go Accuracy Go Reaction Time
Hypothesis #4: Physical activity levels will moderate the relationship between sleep condition and inhibitory control	Two-trial (go, no-go) by two-sleep condition (restricted, normal) by two-weight status (normal-weight, overweight/obese) repeated measures ANOVA with a within-subjects factor of physical activity	Go/No-Go Accuracy Go Reaction Time
Hypothesis #5: Weight status will moderate the relationship between sleep condition and food reward	Two-sleep condition (restricted, normal) by two-weight status (normal-weight, overweight/obese) repeated measures ANOVA with a within-subjects factor of physical activity	Power of Food Scale Total Score
Hypothesis 6: Physical activity levels will moderate the relationship between sleep condition and food reward	Two-sleep condition (restricted, normal) by two-weight status (normal-weight, overweight/obese) repeated measures ANOVA with a within-subjects factor of physical activity	Power of Food Scale Total Score

Results

Adherence to Sleep Protocol

Accelerometry data demonstrated that participants were generally adherent to the study protocol. Participants spent an average of 5.10 hours (*SD* = 0.35; range = 4.63 – 7.40) in bed

during the sleep restriction condition and spent an average of 8.99 hours in bed (*SD* = 0.37; range = 8.34-11.39) for the habitual sleep condition. Of this time spent in bed, participants in the sleep restriction condition slept an average of 4.92 hours (*SD* = 0.26; range = 4.43 – 6.16) and participants in the habitual sleep condition slept an average of 8.5 hours (*SD* = 0.46; range = 7.58 – 10.96) All participants adhered to the sleep protocol within an hour of expected duration (e.g., 4-6 hours in the sleep restricted condition, 7-9 hours in the habitual sleep condition). There were no differences in total time in bed or total sleep time across either condition by weight group.

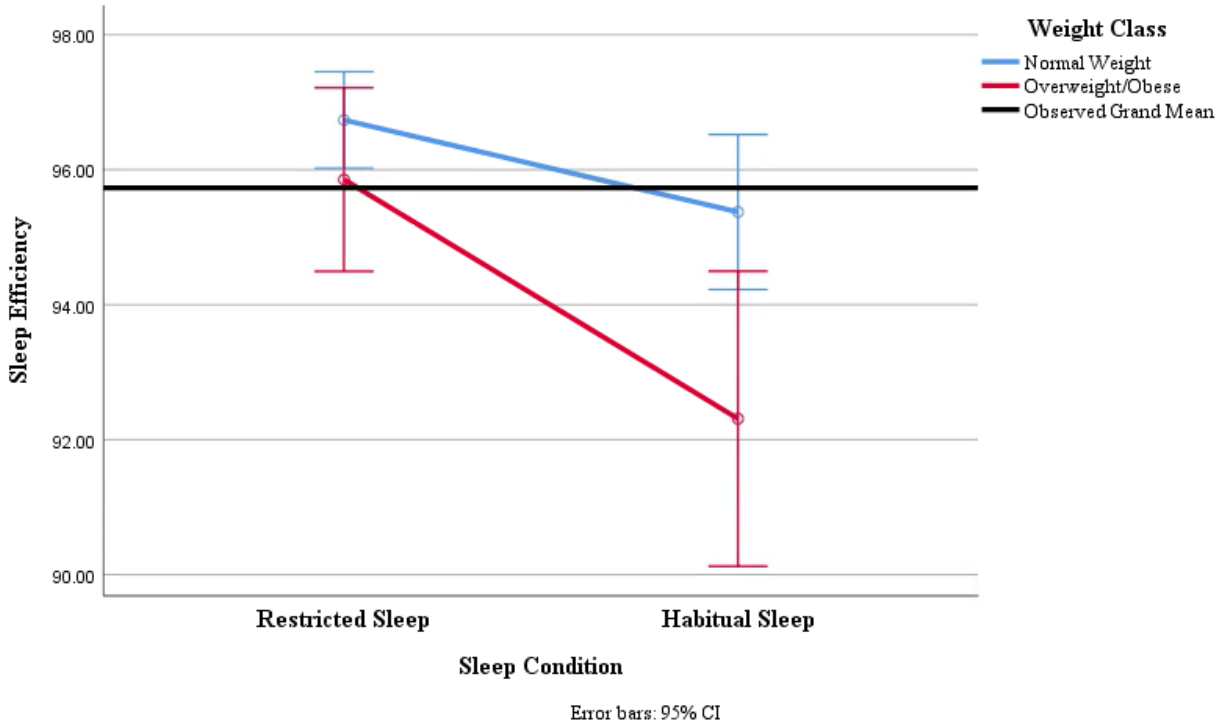
Physical Activity Descriptive Statistics

A 2-sleep condition (restricted, habitual) by 2-weight status (normal-weight, overweight/obese) repeated measures ANOVA was conducted to determine how sleep condition and weight status influenced physical activity levels. During the sleep restricted condition, participants engaged in an average of 0.57 hours of moderate and/or vigorous activity per day (*SD* = 0.49). During the habitual sleep condition, participants engaged in an average of 0.55 hours of moderate and/or vigorous activity per day (*SD* = 0.42). Physical activity levels were not significantly different based on sleep condition ($p = .24$). There was a main effect of weight class observed ($F [1, 53] = 38.20$, $p < .001$, $\eta_p^2 = .42$). Specifically, overweight individuals engaged in significantly less physical activity than healthy weight participants during both the sleep restricted (normal weight $M = 0.87$, $SD = 0.47$, overweight/obese $M = 0.26$, $SD = 0.24$), and habitual sleep conditions ($t(56) = 6.59$, $p < .001$; normal weight $M = 0.81$, $SD = 0.40$, overweight/obese $M = 0.25$, $SD = 0.19$). No significant interaction of sleep condition and weight class was observed on physical activity levels ($p = .39$).

Sleep Efficiency

A 2-sleep condition (restricted, habitual) by 2-weight status (normal-weight, overweight/obese) repeated measures ANOVA with a between factor effect of physical activity was conducted to determine how weight status and sleep condition influenced sleep efficiency. There was a main effect of sleep (F [1, 46] = 18.25 $p < .001$, $\eta_p^2 = .28$), with higher sleep efficiency during the restricted sleep condition (M = 96.82, SD = 1.85) relative to the habitual sleep condition (M = 94.64, SD = 3.05). There was also a main effect of weight status (F [1, 46] = 5.46 $p < .05$, $\eta_p^2 = .11$), with overweight/obese having a lower sleep efficiency (M = 95.47, SD = 2.06) than normal weight individuals (M = 95.92, SD = 2.21). The interaction between weight status and sleep condition was not significant, (F [1, 46] = 3.61 $p < .06$, $\eta_p^2 = .07$), with normal weight maintaining higher sleep efficiency scores across sleep conditions (M = 96.70, SD = 1.94; M = 95.14, SD = 2.47, respectively). Overweight/obese individuals had an increased discrepancy between sleep efficiency scores when sleep restricted than when well-rested (M = 96.99, SD = 1.75; M = 93.95, SD = 3.65; *see figure 1*). No main effect of physical activity on sleep efficiency was observed, and no interaction of physical activity with weight class or physical activity with sleep condition on sleep efficiency were observed.

Figure 1 Interaction of Weight Class and Sleep Condition on Sleep Efficiency



Covariates

A series of bivariate regressions were conducted to determine whether age, gender, or BRIEF-GEC significantly predicted go accuracy, no-go accuracy, go reaction time, and PFS scores. The PFS total score gathered from the habitual sleep condition was also used as a covariate in analyses in which go/no-go accuracy and go reaction time were the primary outcome. Age, gender, PFS total score during habitual sleep condition, or BRIEF-GEC were not found to significantly predict any of the go/no-go primary outcomes (see table 3). Neither age, gender, or BRIEF-GEC was found to significantly predict PFS outcomes. As such, no covariates were included in the final statistical models.

Table 3 *Standardized Regression Coefficients Predicting Inhibitory Control and Food Reward*

Outcome	B	Std. Error	*B*	*t*	*p*
Go-Accuracy					
Age	.00	.01	.05	.37	.71
Gender	-.01	.02	-.04	-.25	.80
Power of Food Scale (Habitual)	.00	.00	-.08	-.58	.56
BRIEF – GEC	.00	.00	.24	1.74	.09
No-Go Accuracy					
Age	.00	.01	.05	.32	.75
Gender	-.01	.03	-.04	-.28	.78
Power of Food Scale (Habitual)	.00	.00	-.22	-1.59	.12
BRIEF – GEC	.00	.00	.13	.92	.36
Reaction Time					
Age	.07	7.57	.00	.01	.99
Gender	12.68	23.97	.07	.53	.60
Power of Food Scale (Habitual)	-.05	.69	-.01	-.07	.94
BRIEF – GEC	-.59	1.37	-.06	-.43	.67
Food Reward					
Age	1.03	1.07	.13	.96	.34
Gender	2.47	3.96	.08	.62	.54
BRIEF – GEC	-.25	.20	-.16	-1.26	.21

Hypothesis 1: The Effect of Sleep on Go/No-Go Accuracy

To test our first hypotheses that sleep restriction would result in decreased accuracy during a food inhibitory control task, a 2-sleep condition (restricted, normal) by 2 trial (go, no-go) repeated measures ANOVA revealed a main effect of trial on accuracy (F [1, 52] = 38.34, $p < .001$, $\eta_p^2 = .42$). Participants were more accurate on go-trails ($M = 93.7$, $SD = 0.03$) than no-go trials ($M = 86.6$, $SD = 0.08$), which suggests that our behavioral task operated as expected, as participants demonstrated more difficulty inhibiting behavioral responses in the no-go trials (as evidenced by decreased accuracy). There was also a main effect of sleep on accuracy (F [1, 52] = 8.69, $p < .01$, $\eta_p^2 = .14$). Specifically, participants were more accurate in the habitual sleep condition ($M = 91.1$, $SD = 0.05$) than the restricted sleep condition ($M = 89.1$, $SD = 0.07$), which suggests that participants had decreased accuracy when sleep restricted as compared to well-

rested. No significant interaction between sleep condition and trial type was noted (p = .227; Table 4).

Table 4 *Repeated Measures ANOVA – Go/No-Go Accuracy*

Trial			Sleep			Trial x Sleep		
F	*p*	η_p^2	*F*	*p*	η_p^2	*F*	*p*	η_p^2
8.69	.005	.14	38.34	.00	.42	1.49	.23	.028

Hypothesis #1: The Effect of Sleep on Go Reaction Time

To test our first hypotheses that sleep restriction would also result in longer reaction time during a food inhibitory control task, a two-sleep condition repeated measures ANOVA revealed that there was not a significant main effect of sleep on Go reaction times (F [1, 52] = 3.59, p = .06, η_p^2 = 0.07). Those in the sleep restricted condition took somewhat longer to react to the Go trials (M = 605.33, SD = 142.09) than those in the habitual sleep condition (M = 582.89, SD = 128.87).

Hypothesis #2: The Effect of Sleep on Food Reward

To test our second hypotheses that sleep restriction would result in increased levels of food reward, a two-sleep condition repeated measures ANOVA revealed that a main effect of sleep on PFS scores (F [1, 61] 9.48, p < .01, η_p^2 = .14). Specifically, participants had higher PFS scores (or higher food reward) following the sleep restriction condition (M = 37.26, SD = 12.31) than following the habitual sleep condition (M = 31.24, SD = 17.65).

Hypothesis #3 and #4: Moderating Effect of Weight Status and Physical Activity on Go/No-Go Accuracy

To test our hypotheses that weight status (hypothesis #3) and physical activity (hypothesis #4) would moderate the association between sleep condition and accuracy of a food inhibitory control task, a 2-trial (go, no-go) by 2-sleep condition (restricted, normal) by 2-weight

status (normal-weight, overweight/obese) repeated measures ANOVA with a within-subject factor of physical activity was conducted for go/no-go accuracy. Similar to previously reported, there was a main effect of trial, (F [1,51] = 38.02, $p < .001$, $\eta_p^2 = .43$), with accuracy remaining higher in the go trials ($M = 93.7$, $SD = 0.03$) than the no-go trials, ($M = 86.6$, $SD = 0.08$). There was also a main effect of sleep (F [1, 52] = 8.61, $p < .01$, $\eta_p^2 = .14$), with participants having higher accuracy in the habitual sleep condition ($M = 91.1$, $SD = 0.05$) than the restricted sleep condition ($M = 89.1$, $SD = 0.07$). There was no main effect for weight status or for physical activity, and no significant interacts were noted (ps > .21; see Table 5).

Table 5 *Repeated Measures ANOVA – Go/No-Go Accuracy with Weight and Physical Activity (PA) Moderators*

Trial			Sleep			Weight			PA			Trial x Weight			Sleep x Weight		
F	*p*	η_p^2	*F*	*p*	η_p^2	*F*	*p*	η_p^2	*F*	*p*	η_p^2	*F*	*p*	η_p^2	*F*	*p*	η_p^2
38.02	.00	.43	8.61	.01	.14	.59	.45	.01	.18	.67	.01	.88	.35	.02	1.31	.257	.025
Trial x Sleep			Trial x PA			Sleep x PA			Weight x PA			Sleep x Weight x PA					
F	*p*	η_p^2	*F*	*p*	η_p^2	*F*	*p*	η_p^2	*F*	*p*	η_p^2	*F*	*p*	η_p^2			
1.45	.23	.03	.72	.36	.02	1.57	.21	.03	.15	.70	.01	.23	.64	.01			

Hypothesis #3 and #4: Moderating Effect of Weight Status and Physical Activity on Reaction Time

To test our hypotheses that weight status (hypothesis #3) and physical activity (hypothesis #4) would moderate the association between sleep condition and reaction time of the food reward task, a 2-sleep condition (restricted, normal) by 2-weight status (normal-weight, overweight/obese) repeated measures ANOVA with a within-subject factor of physical activity was conducted for reaction time. With the interactions included, there was a main effect of sleep (F [1, 49] = 5.80, $p < .05$, $\eta_p^2 = .11$), with those in the sleep restricted condition demonstrating a slower reaction time ($M = 605.80$, $SD = 143.44$) than those in the normal sleep condition ($M =$ 584.24, SD = 129.75). There was no main effect of weight status or physical activity levels on

reaction time, nor were there any significant interactions of weight status, physical activity, and sleep (*p*s < .09; see Table 6).

Table 6 *Repeated Measures ANOVA – Go RT with Weight and Physical Activity (PA) Moderators*

Sleep			Weight			PA			Sleep x Weight			Sleep x PA			Weight x PA		
F	*p*	η_p^2	*F*	*p*	η_p^2	*F*	*p*	η_p^2	*F*	*p*	η_p^2	*F*	*p*	η_p^2	*F*	*p*	η_p^2
5.80	.02	.11	2.27	.13	.06	.04	.85	.00	.38	.54	.01	2.72	.11	.05	.01	.91	.00

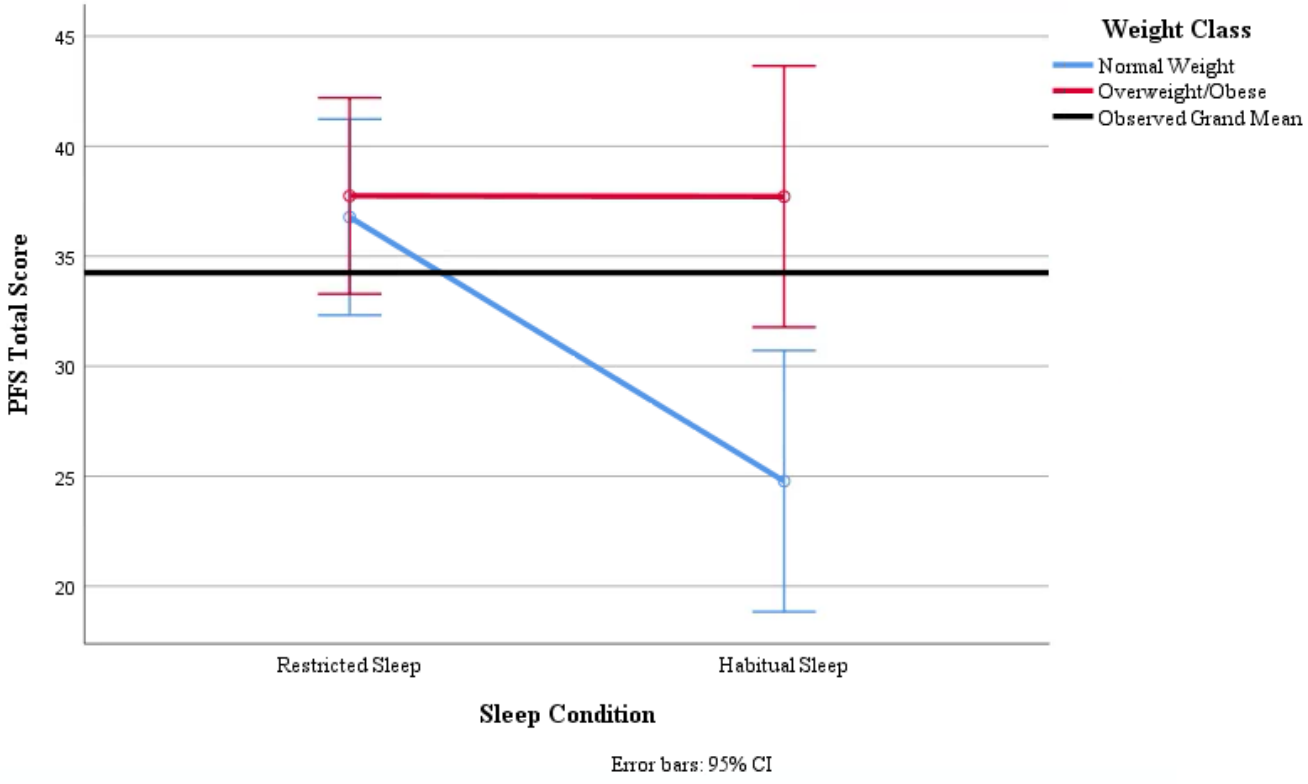
Sleep x Weight x PA		
F	*p*	η_p^2
.788	.37	.02

Hypothesis #5 and #6: Moderating Effect of Weight Status and Physical Activity on Food Reward

To test our hypotheses that weight status (hypothesis #5) and physical activity (hypothesis #6) would moderate the association between sleep condition and score of the food reward task, a 2-sleep condition (restricted, normal) by 2-weight status (normal-weight, overweight/obese) repeated measures ANOVA with a within-subject factor of physical activity was conducted for PFS total scores. There was a main effect of sleep (F [1, 60] = 11.02, $p < .01$, $\eta_p^2 = .156$), with those in the sleep restricted condition having a higher PFS total score (M = 37.26, SD = 12.31) than those in the habitual sleep condition (M = 31.24, SD = 17.65). There was also a main effect of weight status (F [1, 60] = 4.60, $p < .05$, $\eta_p^2 = .07$), with overweight individuals having a higher PFS total score (M = 37.73, SD = 13.49) than normal weight individuals (M = 30.77, SD = 15.36). A significant interaction of sleep and weight status was also observed, (F [1, 60] = 10.90, $p < .01$, $\eta_p^2 = .154$). Overweight individuals had relatively high PFS total scores, regardless of whether in the sleep restriction or habitual sleep condition (M = 37.74, SD = 12.22; M = 37.71, SD = 14.77, respectively). However, normal weight individuals

only had elevated PFS total scores when sleep restricted compared to habitual sleep (M = 36.77, SD = 12.85; M = 24.77, SD = 18.13; *see figure 2*).

Figure 2 Interaction of Weight Class and Sleep Condition on Power of Food Scale (PFS) Total Score



There was no main effect of physical activity, or significant interactions of physical activity, weight status, or sleep (ps < .21; see table 7).

Table 7 *Repeated Measures ANOVA – Food Reward with Weight and Physical Activity (PA) Moderators*

Sleep			Weight			PA			Sleep x Weight			Sleep x PA			Weight x PA		
F	p	η_p^2	F	p	η_p^2	F	p	η_p^2	F	p	η_p^2	F	p	η_p^2	F	p	η_p^2
11.02	.001	.16	4.60	.04	.07	.0	.95	.00	10.90	.002	.15	.00	.97	.00	.04	.85	.01

Sleep x PA x Weight		
F	p	η_p^2
.40	.53	.02

Discussion

The purpose of this study was to examine how prolonged sleep restriction impacted food-related inhibitory control and food reward in a sample of normal weight and overweight/obese adolescents. As more than half of all US adolescents habitually fail to attain recommended sleep duration (Hirshkowitz et al., 2015), it is critical to understand the implications of insufficient sleep in adolescents. Further, given that shortened sleep duration has been implicated as a risk factor for developing obesity in adolescents (Krueger et al., 2015; Suglia et al., 2014), it is important to uncover the underlying mechanisms that are driving the relationship between shortened sleep duration and obesity. Furthermore, this study aimed to explore whether moderating influences such as physical activity or weight status would attenuate the possible risk for poorer food-related inhibitory control and heightened food reward following sleep restriction, as such findings could inform current weight-loss or preventative weight-gain efforts.

Sleep Restriction and Inhibitory Control

For our first study hypothesis, we expected that adolescents experiencing sleep restriction would have decreased inhibitory control, as compared to when they were well-rested. Our study findings were consistent with our hypothesis, and showed that adolescents demonstrated decreased accuracy and increased reaction times on a food-inhibition task following sleep restriction (compared to habitual sleep). This indicates that adolescents are less accurate in inhibiting prepotent cognitive responses when viewing food images under sleep restriction, and that they take longer to classify foods as healthy or unhealthy when sleep restricted. These study findings are similar to Beebe and colleagues' (2008), who demonstrated that adolescents who underwent a similar sleep restriction paradigm exhibited impairments in executive functioning (specifically in attention, organization, planning, self-monitoring, and self-initiation). However,

as Beebe and colleagues did not directly examine inhibitory control as an outcome, our study findings help to clarify an additional area of executive functioning that can be impaired by prolonged sleep restriction. Though Demos and colleagues (2017) used neuroimaging to examine inhibitory control, they also found that young adults undergoing sleep restriction in a similar fashion to our study design had increased activation in brain regions implicated in inhibitory control.

As we had hypothesized, adolescents in our study took significantly longer to respond to the food inhibitory control task when sleep restricted. This aligns with previous studies conducted in adult populations that has found that sleep deprivation or nighttime arousals lengthen reaction time to a go/no-go task (Ayalon et al., 2009; Drummond et al., 2006). However, others have argued that shortened reaction time on a go/no-go task reflects poorer inhibitory control (Batterink et al., 2010). We interpret our study findings to imply that adolescents take significantly longer to determine if a food is healthy or unhealthy when sleep restricted, but still commit more errors in classifying foods as healthy or unhealthy when sleep restricted.

Sleep Restriction and Food Reward

For our second study hypothesis, we hypothesized that adolescents would have increased food reward following the sleep restriction condition, as compared to the habitual sleep condition. Consistent with this hypothesis, we found that sleep restriction increases food reward (as measured by the PFS) in an adolescent population. This study finding is in line with several neuroimaging studies that have shown that individuals who are sleep restricted have increased neural activation in regions associated with reward following viewing food images (compared to habitual sleep; Demos et al., 2017; Greer et al., 2013; St-Onge et al., 2012; St-Onge et al., 2014).

However, the challenge with understanding food reward using neuroimaging methodology is that it can be unclear if neural activation in reward regions align with perceptions of food reward. Therefore, examining food reward from a self-report perspective (i.e., the PFS) following sleep restriction adds novel insight into understanding how sleep restriction impacts food reward. As this is first study to examine how sleep restriction impacts perceptions of food reward (as measured through the PFS), and is also among the first to use the PFS in an adolescent population, our study provides insight into how sleep restriction increases food reward in an adolescent population.

Taken together, these study findings suggest that when adolescents are sleep restricted, not only do they find foods to be more rewarding, but they also experience difficulty with inhibiting cognitive responses in the presence of high calorie foods. While previous studies have demonstrated that sleep restricted individuals are more likely to overeat and eat foods of a higher glycemic content (Beebe et al., 2013; Haghighatdoost, Karimi, Esmaillzadeh, & Azadbakht, 2012), findings from this study help explain why the relationship between shortened sleep duration and overeating exists. Specifically, our study findings show that adolescents who are sleep restricted perceive foods to be more appetizing, which may increase their susceptibility to overeat.

Moderating Impact of Weight

Inhibitory Control. For our third and fourth hypotheses, we explored whether weight status moderated the impact of sleep restriction on food-related inhibitory control and food reward. For our third study hypothesis, we expected overweight individuals to have poorer accuracy and longer reaction times on an inhibitory control task as compared to their normal weight peers, and that overweight adolescents would experience an even greater decline of

inhibitory control following the sleep restriction period (compared to their normal weight peers). Contrary to our hypotheses, we did not observe a main effect of weight status on the performance of a food inhibitory control task, nor did we observe an interaction of weight class and sleep condition on inhibitory control performance. While Black and colleagues (2014) have suggested that overweight/obese children have altered inhibitory control neural networks as compared to normal weight individuals, our study population demonstrated similar inhibitory control outcomes across weight status. This study finding differs from previous literature examining weight class and inhibitory control, which has found that overweight adolescents perform more poorly in inhibitory control tasks than normal weight adolescents (Nederkoorn, Braet, et al., 2006; Pauli-Pott et al., 2010a). Further, our study findings contrast those observed by Batterink and colleagues, (2010) which reported a positive correlation between BMI and number of errors and a negative correlation with BMI and reaction times on the food go/no-go task utilized in our study. However, differences in study findings may be attributable to Batterink and colleagues examining an exclusively female adolescent population. Adolescent sex differences have been observed in how adolescents perceive food images (Jensen, Duraccio, Carbine, et al., 2016), and so it is possible that there is a greater weight interaction in a female population than in a population of both sexes. However, given that several study findings have found a main effect of weight class on food inhibitory control, future research on this topic in an adolescent population is warranted.

Food Reward. For our fourth study hypothesis, the moderating impact of weight status on the relationship between sleep condition and food reward was explored. Specifically, we hypothesized that overweight adolescents would have increased food reward (compared to normal-weight adolescents), and that overweight adolescents would have significantly greater

increases of food reward following the sleep restriction condition (compared to normal weight peers). In line with our study hypotheses, we observed a main effect of weight, with overweight/obese adolescents exhibiting greater food reward than normal weight adolescents. This is comparable to findings in the adult literature that have shown a positive correlation between food reward and BMI (Cappelleri et al., 2009) and that overweight women are more sensitive to food reward than normal weight women (Davis, Strachan, & Berkson, 2004). However, a previous study examining food reward in a child population did not observe a correlation between child and adolescent BMI and PFS scores (Mitchell et al., 2016). Despite this discrepant finding, our study findings align with neuroimaging study findings in children and adolescents, which have observed that obese children to have increased activation to food images and food logos in brain regions associated with food reward, as compared to healthy weight children (Black et al., 2014; Bruce et al., 2010; Bruce et al., 2013; Yokum et al., 2011). Our study findings suggest that overweight/obese adolescents differ from normal weight adolescents on the way they report they perceive food reward, finding foods to be more rewarding than their normal weight peers.

Also in line with our fourth study hypothesis, we observed a significant sleep condition by weight class interaction on food reward. Specifically, we demonstrated that overweight adolescents tend to find foods to be highly rewarding at all times, regardless of sleep condition. It is possible that since overweight/obese adolescents are more likely to be chronically sleep restricted (Khan, Chu, Kirk, & Veugelers, 2015), the overweight/obese adolescents in our study may have been less sensitive to the sleep-restriction paradigm as compared to the normal weight adolescents in our study. Our normal weight adolescents demonstrated a spike of food reward following sleep restriction, bringing their food reward levels to a level indistinguishable from

their overweight/obese peers. When well-rested, normal weight adolescents' food reward levels are significantly lower than their overweight/obese peers. These study findings suggest that normal weight adolescents are at increased risk of increasing food reward following a sleep restriction condition. This is the first study to examine the interaction of sleep length and weight status on food reward. These study findings help elucidate the link between shortened sleep duration and increased risk for later development of obesity. If normal-weight adolescents are regularly sleep restricted, our study findings would suggest that they will subsequently increase their food reward, a process which has been associated with the likelihood of overeating and gaining weight (Davis et al., 2004).

Moderating Impact of Physical Activity

For our fifth and sixth study hypotheses, we also aimed to explore whether physical activity would moderate the relationship between sleep condition and food-related inhibitory control and food reward. We hypothesized that physical activity would attenuate the decreases in inhibitory control and increases in food reward exhibited in adolescents who experienced sleep restriction. However, we did not observe a main effect of physical activity on food-related inhibitory control or food reward or an interaction between physical activity levels and sleep condition on food-related inhibitory control or food reward. Previous studies have demonstrated that increased physical activity resulted in increased inhibitory reward in children and adolescents (Budde et al., 2008; Davis, Tomporowski, et al., 2011; Hillman et al., 2009); however, we did not observe that adolescents who had higher levels of physical activity had increased inhibitory control scores. Further, Lambiase and colleagues (2014) found that in an sample of older adult women, physical activity helped prevent the declines in executive functioning observed following obtaining poor sleep efficiency. While our study findings differ

from that of Lambiase and colleagues', study finding differences may be attributable in part to different outcomes (executive functioning broadly, compared to inhibitory control) as well as sample characteristic differences (older adult women compared to adolescents of both sexes).

We did observe that adolescents who were overweight/obese were significantly less physically active than normal weight individuals across both sleep conditions, engaging in 15 minutes of physical activity per day on average. This falls far below the 60 minutes of physical activity recommended by the CDC (Centers for Disease Control and Prevention, 2015). Physical activity dose alone has shown to predict dietary behavior within adult population. Specifically, sedentary adults are found to eat more total fat and eat less fiber, fruits, and vegetables than moderately active and highly active adults (Eaton et al., 1995). Higher physical activity has also been shown to predict higher quality diets in university students (Moreno-Gómez et al., 2012). No studies have examined the relationship between physical activity and dietary decision making in an adolescent population, so future studies on this topic is warranted.

Sleep Efficiency

We also explored the impact of weight and physical activity on sleep efficiency across each sleep condition. We found that adolescents who were sleep restricted had better sleep efficiency. Increases in sleep efficiency following sleep restriction is a phenomenon well documented in the child and adolescent literature (Astill, Verhoeven, Vijzelaar, & Van Someren, 2013; Levine, Lumley, Roehrs, Zorick, & Roth, 1988; Sadeh, Gruber, & Raviv, 2003). We also expected a main effect of physical activity on sleep efficiency, as increases in physical activity have been associated with increases in sleep efficiency in the general population (Gubelmann et al., 2018) as well as in an adolescent population (Lang et al., 2013). However, we did not observe a main effect of physical activity levels on sleep efficiency in our sample. It is possible

that the waist-worn accelerometers that we used to gather sleep efficiency data were not sensitive enough to detect changes in sleep efficiency caused by physical activity.

We also observed a main effect of weight class on sleep efficiency, and observed an interaction between weight class and sleep duration that trended towards significance. Adolescents with overweight/obesity are more likely to have difficulties with breathing (e.g., obstructive sleep apnea) that can directly influence sleep efficiency (Kang, Lee, Weng, & Hsu, 2012). While we screened for sleep disorders over the phone (as part of the recruitment processes), we did not administer any measures to assess for baseline sleep quality. Therefore, it is possible that some of our overweight/obese adolescent participants may unknowingly have symptoms of a sleep disorder that could have influenced their sleep efficiency in this study.

Strengths of the Current Study

Our study possesses several salient strengths. First, our study possessed high internal validity, due to our high selection criteria and within-subject design, which strengthens our power to detect the effects outlined in our study hypotheses. Second, our study examined the full spectrum of weight status, allowing for the observation of weight interactions. Third, our study was among the first to examine both food-related inhibitory control and food reward processes, rather than examining a single construct in isolation. This allowed for greater conclusions to be made regarding the effects of sleep restriction on food processing. Finally, our study was also among one of the first to demonstrate the feasibility of a sleep restriction paradigm in adolescents.

Weaknesses of the Current Study

Despite these study strengths, our study was not without limitations. First, though we used a novel method for assessing food-related inhibitory control (i.e., Food Go/No-go task), the

task used food images as proxies for real food, which limits several of the sensory experiences that accompany being in the presence of food (e.g., olfactory, tactile). Second, the reliability of the adapted Food Go/No-go has yet to be established. Third, we did not directly measure dietary decision making using methods such as dietary recall or food buffet; instead, we used the PFS as a marker for increased risk of overeating. Limited research has been conducted examining how PFS relates to dietary decision making. Therefore, our measures of food-related inhibitory control and food reward only serve as predictors of dietary decision making, rather than measuring dietary decision making directly. Fourth, we did not assess for perceived hunger before participants completed the study tasks, which may have influenced the way that adolescents perceived food images. However, we did require participants to fast for four hours before each study visit as an attempt to standardize hunger levels.

Fifth, because we did not require participants to sleep inside a laboratory, we noted some deviations in the sleep protocol. We were also unable to assess for what behaviors the participants engaged in prior to sleep, which may have influenced sleep efficiency. Fortunately, we observed that all participants adhered to the sleep requirements within one hour of the instructed bedtime/waketime. Sixth, the actigraphy GT3x+ worn on the waist tends to underestimate nighttime disturbances. This lack of sensitivity to nighttime awakenings may have overestimated our study findings regarding sleep efficiency. Seventh, because we did not administer any baseline measures of sleep behavior or quality, we were unable to examine how baseline sleep influenced study findings. Eighth, our analyses examining food inhibition via the food go/no-go task are slightly underpowered, as eleven go/no-go behavioral files were unable to be downloaded. Ninth, our ability to generalize results is limited, due to restrictive inclusion criteria and due to our sample being predominantly Caucasian. Finally, our two weight groups

differed significantly by SES; it is possible that factors relating to SES may have influenced analysis comparing weight groups.

Research Implications and Future Directions

Our study was among one of the first to demonstrate the feasibility of conducting a repeated-measures sleep restriction paradigm in an adolescent population under free-living conditions. We found that our participants were generally adherent to the sleep requirements, with all participants sleeping within a one-hour window of what was assigned. We also only had one participant drop out of the study, and had no participants fail to show at a study appointment. We believe that our success in maintaining high adherence in this study was due to our frequent contact with study participants (e.g., nightly calls, morning texts, reminder calls), as well as providing several small monetary bonuses for adhering to the study protocol. Additionally, by only running participants during the summer months (as to not impair academic performance with sleep restriction), we also demonstrated that a sleep restriction paradigm can be conducted without significantly disturbing an adolescents day-to-day functioning.

Our study is also the first to use the PFS as an outcome of sleep restriction, which provides for an alternate way to measure how sleep restriction impacts food reward. As this is the first study to examine the relationship between sleep and the PFS, replication of this current study is warranted. Of note, our study findings demonstrated that overweight/obese adolescents did not experiences changes in their PFS scores across sleep conditions, with their PFS scores remaining relatively high across both sleep conditions. This may implicate that overweight/obese adolescents demonstrate a ceiling effect in food reward (as measured by the PFS), meaning that external factors are less likely to influence PFS scores in overweight/obese adolescents. Future replication studies that examine food reward (as measured by the PFS) in overweight/obese

adolescents should include additional methods of measuring food reward (e.g., neuroimaging, dietary recall) to accurately examine changes in food reward following sleep manipulation.

Our study is also among one of the first to use the Food Go/No-Go task as an outcome of sleep restriction. Based on several study findings, we encourage replication of this current study, particularly due to our discrepant findings from previous research in which we found that there was no interaction between weight status and sleep on food-related inhibitory control. Additionally, while our Food Go/No-Go task was designed to approximate the participant having real experiences with food, some researchers have begun to examine more ecologically valid stimuli (including olfaction and ingestion of actual food; Bohon & Stice, 2011; Boswell & Kober, 2016; Stice, Yokum, & Burger, 2013) to measure both food-related inhibitory control and food reward. Additionally, as our study uses inhibitory control and food reward to approximate dietary decision making, future studies should examine the impact of a sleep restriction paradigm on direct dietary outcomes, perhaps using dietary recall or food buffets to measure dietary behavior. Inhibitory control and food reward could then be examined as moderating factors between sleep length and dietary decision making.

As overweight/obese adolescents are already at increased risk for having poorer sleep quality and shortened sleep duration (Khan et al., 2015), it is possible that our "habitual sleep condition" represented more of a "recovery sleep condition" for some of our overweight/obese participants. However, as we did not measure for baseline sleep behaviors, we were unable to control for baseline sleep behaviors in our analyses, or objectively rule out any sleep disorders that the overweight/obese participants may be at increased risk for (e.g., obstructive sleep apnea). Therefore, we recommend that future studies include a baseline assessment in which they measure for baseline sleep behaviors. Beebe and colleagues (2013) have structured their sleep

restriction studies in this format, having participants wear a wrist-worn actigraph for a week to assess for baseline sleep behaviors (as well as complete questionnaires regarding sleep behaviors to rule out sleep disorders) prior to being randomized into an experimental sleep condition.

Additional future directions may include modifying the sleep restriction paradigm. Given that adolescents are sleeping closer to 7 hours on average (instead of the recommended 9 hours of sleep; Hirshkowitz et al., 2015), replicating this study by extending the sleep restricted condition to be 7 hours per night may provide insight to the real-world consequences of the current sleep habits exhibited by adolescents. We also encourage future studies to use wrist-worn (as compared to waist-worn) accelerometers, as they demonstrate greater reliability in generating sleep outcomes (Paavonen, Fjällberg, Steenari, & Aronen, 2002; Takeshima et al., 2014). Finally, replication of this study is needed with a more diverse sample with less stringent inclusion criteria.

Clinical Implications

Study findings suggest that sleep restriction in an adolescent population increases the adolescent's sensitivity to food by heightening the reward properties of food, while simultaneously decreasing the ability to inhibit impulses in the presence of food. These study findings help elucidate one mechanism explaining why sleep restricted individuals overeat. Furthermore, these findings may help explain the link between shortened sleep duration and risk for developing obesity in normal weight adolescents. We also found that adolescents who are overweight tend to find foods more rewarding, and are therefore less susceptible to the effects of sleep restriction on heightened food reward. On the other hand, normal-weight individuals are particularly susceptible to increases in food reward following sleep restriction. As adolescents are regularly sleep restricted, these study findings may suggest that they are increased risk for

overeating and subsequent weight gain. Study findings may inform current intervention recommendations aimed at managing weight outcomes. Specifically, encouraging healthy sleep habits and providing sleep recommendations as part of weight loss interventions should be encouraged. Research in the adult literature suggests that there is added utility in intervening at the level of sleep to promote weight loss efforts (Coughlin & Smith, 2014), and our study findings would suggest that promoting healthy sleeping habits would impact the perception of foods in adolescents. Further, given that normal weight individuals are susceptible to increased food reward following sleep restriction, education about proper sleep should be provided to all adolescents to help prevent future weight gain. In summary, results from this study may be used to inform health interventions that foster sleep health in addition to promoting weight loss, weight maintenance, and healthy eating habits.

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APPENDIX A: THE POWER OF FOOD SCALE.

1. I find myself thinking about food even when I am not physically hungry.
2. I get more pleasure from eating than I do from almost anything else.
3. If I see or smell a food I like, I get a powerful urge to have some.
4. When I am around a fattening food I love, it is hard to stop myself from at least tasting it.
5. It is scary to think of the power that food has over me.
6. When I know a delicious food is available, I can't help myself from thinking about having some.
7. I love the taste of certain foods so much that I cannot avoid eating them even if they are bad for me.
8. Just before I taste a favorite food, I feel intense anticipation.
9. When I eat delicious food I focus a lot on how good it tastes.
10. Sometimes, when I am doing everyday activities, I get an urge to eat "out of the blue" (for no apparent reason).
11. I think I enjoy eating a lot more than most other people.
12. Hearing someone describe a great meal makes me really want to have something to eat.
13. It seems like I have food on my mind a lot.
14. It is very important to me that the foods I eat are as delicious as possible.
15. Before I eat a favorite food, my mouth tends to flood with saliva.

Respondents are instructed to indicate the extent to which each statement describes them. Response options are on a 5-point Likert scale ranging from (1) *don't agree at all* to (5) *strongly agree.*