Sleep and Eating Behavior Among Adolescent Females with Overweight or Obesity: The Role of Appetite-Related Cognitive Processes

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Sleep and Eating Behavior Among Adolescent Females with Overweight or Obesity:

The Role of Appetite-Related Cognitive Processes

Kirandeep Kaur

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

Sleep and Eating Behavior Among Adolescent Females with Overweight or Obesity: The Role of Appetite-Related Cognitive Processes

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Doctor of Philosophy

Insufficient sleep duration and poor sleep quality can potentiate weight gain and obesity in adolescents. Furthermore, overweight and obese females are at unique risk for insufficient sleep and associated health complications. We examined self-reported sleep duration and self-reported adequacy of sleep duration as potential moderators of the relationship between eating behavior and several cognitive processes including hedonic hunger, executive dysfunction, and self-control. We used a multisystemic conceptual framework to highlight the pathways that may explain the relationship between sleep behaviors and the Healthy Eating Index (HEI). The study employed a cross-sectional design. Participants completed baseline measures of height and weight, self-control, executive functioning, hedonic hunger, and sleep functioning. Self-report of poor sleep adequacy directly influenced executive dysfunction which consequently explained a decrease in self-control functioning. Moreover, we evaluated whether sleep deprivation and extension influences caloric intake. We offer novel yet promising evidence that 9 hrs of sleep fostered greater self-control functioning which promoted intake of 484.69 fewer calories per day compared to sleep deprivation. Our study is well-positioned to improve understanding of individual cognitive subsystems and the mechanism that underlies the influence of sleep behavior on weight-related behaviors among overweight and obese females. Findings from this study have the potential to inform health interventions that promote healthy eating and sleep behaviors.

Keywords: sleep extension, sleep deprivation, executive dysfunction, self-control, hedonic hunger, inadequate sleep
ACKNOWLEDGEMENTS

There are many whose great effort has made this work possible. I would like thank Dr. Chad Jensen for his guidance, support, and encouragement. I am grateful that he continually pushed me to make my work better. I also wish to thank my committee members for their efforts and insightful suggestions.

Profound and enduring gratitude to my husband, Arpit Sharma, not only for his relentless and unparalleled support, but also for believing in me. I would like to acknowledge my indebtedness to my parents, because nobody has been more important to me in the pursuit of this project than my parents. Lastly, the completion of my Ph. D coursework would not have been possible without the nurturance of my dog, Mango.
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Sleep and Eating Behavior Among Adolescent Females with Overweight or Obesity:

The Role of Appetite-Related Cognitive Processes

Adequate sleep is critical for adolescent’s health and health-related behaviors because sleep influences physical and emotional well-being (e.g., brain-maturation, biological and psychological changes in puberty (Chaput & St-Onge, 2014). Healthy sleep behavior can be characterized by quantity, continuity, and timing (Hall M. et al., 2008). Optimal sleep requires adequate duration, good quality, appropriate timing and regularity, and the absence of sleep disturbance or disorders (Ohayon et al., 2017). In a comprehensive review of specific dimensions of sleep health, Buysee (2014) noted five salient sleep features, (1) Sleep duration, i.e., the total amount of sleep obtained per 24 hours; (2) Sleep continuity or efficiency, i.e., the ease of falling asleep and returning to sleep; (3) Timing, i.e., the placement of sleep within the 24- hour day; (4) Alertness/sleepiness, i.e., the ability to maintain attentive wakefulness, and (5) Satisfaction/quality, i.e., the subjective assessment of “good” or “poor” sleep.

According to the National Sleep Foundation’s recommendations, adolescents aged 14-17 years should sleep between 8 and 10 hours per night to maximize overall health and well-being. However, recent estimates from more than 20 countries suggest a steep decline in adolescent’s sleep duration over the past decade compared with young children or adults. With a secular decline of 0.75 minutes/night/year in sleep duration over the last 100 years (Chaput & Dutil, 2016), only 33% of teens are attaining the recommended 9 hours of sleep per night (Weiss et al., 2010). These statistics are concerning because an accumulating body of evidence indicates that chronic sleep deprivation poses a serious threat to the weight-related health of adolescents. Specifically, sleep debt impacts three hormonal mechanisms in humans that affect metabolic and
endocrine function which affects energy balance: 1) Glucose metabolism, 2) serotonin release, and 3) hypocretins (Gupta et al., 2002).

Sleep is intricately connected to hormonal and metabolic functioning and is important to maintain metabolic homeostasis (Sharma & Kavuru, 2010), the lack of is known to promote multifactorial metabolic dysregulation. In one such study, when subjects who simulated shift work showed alterations in postprandial glucose and metabolism. Moreover, the same group took at least 2 days to adapt to eating meals on a simulated night shift (Hampton et al., 1998).

In addition, sympathetic stimulation has been shown to occur with sleep deprivation and is likely to promote metabolic dysregulation (Speigel et al., 2004). Lastly, sleep deprivation contributes to inflammation, as noted in one experimental sleep deprivation study that noted alerted immune response and increase in proinflammatory markers (Vgontzas et al., 2004).

Sleep is more complex than duration alone. Timing, quality, and satisfaction are important components of healthy sleep (Buysse, 2014). An examination of the association between sleep quality and sleep quantity among adolescents (55% females) reported a weak correlation (range -0.02 to -0.17; Pilcher et al., 1997). Therefore, adequate sleep is also a critical factor for adolescent sleep. Sleep adequacy can be defined as a combination of sufficient sleep duration and sleep quality (Buxton et al., 2009). Researchers have begun to understand the role of adequate sleep in health-prevention and weight status. In one such examination, adolescents’ subjective rating of sleep adequacy (rarely, sometimes, usually, and always) and objective measure of sleep duration indicated a significant association between adequate sleep and health-related behaviors (OR = 1.98), nutrition (OR= 2.99), exercise (OR= 2.15), non-obesity (OR= 1.74; Chen et al., 2006). Poor sleep quality can initiate a metabolic response with wide-ranging consequences that can influence appetite fatigue and fat mass. In one such study with healthy
adults, sleep fragmentation explained reduction in insulin sensitivity and glucose effectiveness controlling for total sleep time (Stamatakis & Punjabi, 2010). Taken together, inadequate sleep explains a wide range of negative health outcomes including obesity, type 2 diabetes, heart diseases, and some cancers (Kruger et al., 2014).

**Sleep Behavior, Eating Behavior, and Obesity**

There is mounting evidence that insufficient sleep is associated with obesity and increased adiposity (Chen et al., 2008). This relationship was initially explained by Spiegel and colleagues (Spiegel & Leproult et al., 2004) in their landmark study which demonstrated that short sleep in young healthy males was associated with decreased leptin levels, increased ghrelin levels, and increased hunger and appetite. There is growing evidence that short sleep duration is an independent risk factor for obesity and poor dietary functioning in the younger population (Bel et al., 2013). In an analysis of 11 longitudinal studies of children and adolescents, short sleepers had twice the risk of developing overweight/obesity compared with those sleeping for a long duration (OR= 2.15; 95 % CI, 1.64 – 2.81; Fatima et al., 2015). In another meta-analysis, a decrease of 1-hour of sleep per night in children was associated with a BMI that was 0.35kg/m² greater (Cappuccio et al., 2008). Whereas, each 1-hour increase in sleep duration results in a 9% decrease in obesity risk (Chen et al., 2008; Graef et al., 2014). While there is accumulating evidence that shorter sleep duration is a risk factor for adiposity, many studies report inconsistent findings within the pediatric population. For example, Chen and colleagues (2008) examined the association between standardized sleep duration (≥ 11h for children aged <5 years, ≥ 10h for children between 5 and 10 years, and ≥ 9h for age 10 and above), gender, and BMI. They noted a clear association between short sleep duration and increased risk of obesity only among children but not among adolescents.
However, research is limited on the influence of subjective sleep variables (e.g., sleep quality, sleep adequacy) on obesity and obesity-related behavior. Nonetheless, some clinical and epidemiological studies established a relationship between sleep fragmentation (poor sleep or sleep quality) and glucose metabolism independent of sleep duration (Punjabi et al., 2004). Therefore, it seems that not only inadequate sleep duration, but subjective sleep quality has the potential to influence pathways promoting weight gain. In a meta-analysis examining the role of sleep quality in overweight/obesity among children and adolescents, Fatima and colleagues (2016) noted considerable variations across studies in defining inadequate sleep, however, they described three categories: (1) Inadequate sleep predominantly measured as insufficient sleep duration; (2) Inadequate sleep predominantly measured as poor quality irrespective of sleep duration (e.g., problems associated with sleep initiation and maintenance), and (3) Inadequate sleep as an overall measurement of sleep including both duration and quality. While they noted research studies that examined various dimensions of sleep to assess sleep quality and sleep quantity (e.g., sleep disturbance, sleep duration, sleep problems, sleep timing). However, the association between poor sleep quality and overweight/obesity did not change for cross-sectional studies conducted with adolescents. Overall, they argue that relying on only sleep duration to define inadequate sleep may not offer insight into the complex relationship between sleep and weight status.

When it comes to the mechanism that explains the resultant increase in body weight, eating behavior has received the most attention. Multiple studies support that energy consumption is a key mediator of the association between insufficient sleep and weight gain (Bel et al., 2013; Chaput et al., 2010; Chapman et al., 2012; Chaput & St-Onge, 2014). Eating is known to influence the relationship between sleep duration and obesity; an inadequate sleep
duration modifies eating behavior which likely influences the relationship between sleep duration and obesity (Bel et al., 2013; Kim et al., 2011). Multiple studies conducted with adults have reported that restricting sleep in normal sleepers increases food intake, mostly by increasing the intake of snacks (St-Onge et al., 2011; Nedeltcheva et al., 2008) and fat (St-Onge et al., 2011).

In one of the first studies with college-age young adults, habitual short sleepers (average 6h/night) ate more often (i.e., > 3 meals/day with more frequent nibbling) than did long sleepers (Hicks et al., 1986). Similarly, the National Longitudinal Study of Adolescent Health (1994-2008) report that short sleep duration (<7h/night) is associated with 25% decreased odds of adequate vegetable and fruit consumption and a 20% increase in odds of fast-food consumption (Kruger et al., 2014). Adolescents may spend their increased awake time participating in sedentary activities, which typically involve eating highly caloric foods, to manage the nocturnal energy deficit resulting from restricted sleep (Chaput & Dutil, 2016). In fact, shortening the sleep time by just 1.5h/night for 2 weeks has been shown to produce a marked increase in appetite and caloric intake among normal-weight adults (Nedeltcheva et al., 2008).

Furthermore, consistent results are noted from international studies that strengthen the argument that inadequate sleep influences dietary behavior. For example, an observational study from ten European cities with over 3300 adolescents (1748 females) found that adolescents with short sleep-duration (<8h/night) had a lower intake of fruits and vegetables. In a similar study, adolescents who slept < 8h/night consumed a higher percentage of energy from fat than those who slept > 8h/night (Garaulet et al., 2011). Moreover, adolescents with a shorter sleep duration had an inadequate amount of fruits, vegetables, fish, fat-free milk, and breakfast cereals. In addition, they had increased consumption of junk food, such as pizza, hamburger, and pasta
snacks products. Similarly, in a study with Iranian young females, short sleep duration was associated with poor diet quality (e.g., more dietary energy and carbohydrates but a lower amount of fiber and fruits), despite similar energy density across groups with differing sleep duration (Haghighatdoost et al., 2012). Moreover, short-sleepers had lower diet quality indices and higher values of BMI, waist circumference, and abdominal adiposity compared to long sleepers (>8 h/night). In a large population study from Canada, short sleep duration was associated with intake of fewer fruits and vegetables and more calorie-dense food, such as sugar-sweetened beverages or fast food (Tatone-Tokuda et al., 2012). On the other hand, consistent sleep duration has been found to have incremental beneficial effects; studies with young adults support an association between sleep duration and subsequent weight loss such that for every 1h/day increment in sleep duration, the annual body mass index gain reduced by 0.05 kg/m² (β = −0.05; 95 % CI −0.09, −0.01) (Ruan et al., 2015). Even though an impressive number of studies have suggested that sleep restriction is associated with poorer dietary quality in adolescents.

However, not all laboratory studies have demonstrated increased food intake following sleep loss. For example, a recent meta-analysis indicated only small to moderate effects of sleep deprivation on acute energy intake (Chapman et al., 2012). In an examination of the longitudinal relationship between sleep duration and obesity in a large sample of adolescents from 12 to 18 years old, sleep duration was not a significant predictor of obesity adjusting for age, race, gender, and SES (Calamaro et al., 2010). Similarly, in a review of 15 studies that directly investigated the effects of short sleep duration on overweight and obesity, there was inconclusive evidence of shortened sleep duration influencing weight status among adolescents (Guidolin & Gradisar, 2012). However, some argue that the disparity in findings is likely a result of the amount of food
provided (portion size), so offering a large amount of foods to sleep-deprived participants may stimulate eating beyond satiety, which might mask the modest effects of sleep deprivation on food intake (Rolls, 2009). Taken together, adolescents who get less sleep are noted to consume more calories in the form of fast foods, sweets, and snacks, and fewer micronutrients than adolescents who get more sleep.

While decrease in sleep duration parallels the prevalence of chronic health issues and obesity, and while sleep is a modifiable risk factor for obesity (Chaput, 2014). Turning the relation other way around, researchers have begun to examine the impact of sleep extension on health functioning. For example, sleep extension in young adults with habitually restricted sleep was associated with beneficial impacts on glucose metabolism and insulin sensitivity (Leproult et al., 2015). At-home sleep extension was associated with a reduction in intake of fat, salt, carbohydrates, and sugar among adults who extended their daily sleep by 1-1.5h/night for a month (Al Khatib et al., 2018). In a study with overweight young adults, sleep extension to 8.5 h/night for 3-weeks predicted significant decrease in carbohydrate intake. Increasing total sleep time is associated with reduced sleepiness and fatigue and improved attention, reaction time, and mood (Kamdar et al., 2004). In an experimental manipulation of 5-week home-based sleep manipulation that focused on sleep extension in healthy habitually short-sleeping adolescents explained a decrease in anger, fatigue, and confusion (Van Dyk et al., 2017).

Research also suggests that extending sleep may ameliorate the increase in weight and energy intake associated with sleep restriction (Moreno-Friás et al., 2020). Moreover, this reduction is associated with greater weight loss than diet and exercise alone (Logue et al., 2012). However, research has yet to examine the influence of sleep extension on dietary behavior. In one such study, adolescents with elevated weight increased their sleep by 5 minutes each night to
reach a target of 1 hour of additional sleep. Which demonstrated a decrease in weight, waist circumference, and energy consumption with sex-based differences (weight reduction was higher in men than in women). Similarly, among the adult population, weekend sleep extension (weekend sleep duration longer than weekdays sleep) demonstrated protective effects in preventing sleep-restriction related obesity (Im et al., 2017). These findings suggest that sleep extension is conducive to the maintenance of healthy weight among adolescents and young adults.

The aforementioned studies contribute to our understanding of the relationship between inadequate sleep and eating behavior. However, this relationship may not be linear in nature as research suggests that alterations in multiple systems may also influence the compounding effect of sleep impairment on dietary behavior. On those lines, we propose a multisystemic perspective model to (Figure 1) illustrate the relationship between inadequate sleep behavior and increased food intake.

**Sleep Impairments and Eating Behaviors in Overweight and Obese Adolescents**

Altogether, the accumulating body of evidence in this research area suggests that sleep impairments have a potential negative implication on adolescent’s health and eating behaviors. However, this relationship is more pronounced among overweight or obese adolescents. In an examination of the association between sleep quality and caloric intake among adolescents a higher percentage of obese subjects (both males and females) had poor sleep quality than did non-obese subjects (Sagala et al., 2017). Moreover, this study found a significant association between poor sleep quality and caloric intake contributing to obesity. Research indicates that obese individuals are more responsive to environmental food cues when sleep deprived (Braet et al., 2008), overweight/obese adolescents may be particularly susceptible to overeating when
sleep deprived. For example, young adults with increased adiposity rated high caloric food as
being 24% more appetizing after a night of no sleep relative to well-rested (Benedict et al.,
2012). The relationship between sleep and weight gain is further complicated by the fact that
obesity itself can cause somatic problems, some of which may affect sleep behavior and
potentially exacerbate weight, sleep, and other general medical problems, making the
directionality of this relationship between sleep and obesity difficult to ascertain (Liu et al.,
2012). Multiple studies note that sleep problems predict higher odds of overweight and obesity
after adjusting for sleep duration (Liu et al., 2011).

Furthermore, sex differences in the association between sleep duration, body
composition, and eating behavior are also commonly observed. Chaput, and colleagues (Chaput
et al., 2006) found a significant association between sleep duration and body weight, for boys
and not for girls. Similarly, Knutson (2005) analyzed data from the National Longitudinal Study
of Adolescent Health and noted sex-related differences in the association between sleep duration
and BMI. Sleep duration was significantly associated with body mass index among males only,
such that every hour increase in sleep duration was associated with 10% reduction in risk of
being overweight only in males. In a longitudinal study, sleep restriction did not predict an
increase in waist circumference in young females, unlike in males (Eisenmann et al., 2006;
Gupta et al., 2002). Conversely, females report poorer sleep quality relative to males (Fatima et
al., 2016). In terms of food choices, males have been found to eat fewer fruits, vegetables, and
low-fat foods compared to females (Li & Kenrick, 2006). Similarly, in an examination of
changes in sleep duration among adolescents, Keyes and colleagues (Keyes et al., 2015) found
that females, racial/minorities, and students of low SES reported obtaining fewer total hours of
sleep compared to male, non-Hispanic, white, and students belonging to high SES. In an
examination of sleep quality among obese adolescents, the risk of obesity in males was greater than in females (Sagala et al., 2017) due to changes in hormonal functioning. Testosterone decreases serum leptin secretion by 62% and low leptin levels are known to increase appetite (Park, 2011). Results from a study with a representative sample of adolescents (77% females and 33% with clinically elevated adiposity) showed that sleep fragmentation explained BMI whereas sleep duration did not (Vargas et al., 2014).

While accumulating evidence suggests a pronounced influence of sleep behavior on dietary functioning among overweight and obese adolescents, a recent meta-analysis reported inconsistent findings (Krietsch et al., 2019). They investigated studies (between 2011 and 2017), examining the pathways linking pediatric sleep (including duration, timing, variability, and quality) and weight-related factors (including dietary intake, altered eating behavior/patterns, physical or sedentary activities, and hormones). They noted that while experimental studies showed that restricting time in bed increased caloric intake, but no such relationship was reported in cross-sectional studies. Moreover, a handful of studies reported an association between sleep quality and altered eating behavior. Taken together, findings suggest that sleep behavior affects eating behavior and that this relationship is likely to be influenced by sex. Thus, it is important to consider how sex differences might influence the relationship between sleep restriction and weight status. However, despite meta-analytic evidence of an association between pediatric sleep and obesity, the literature is more mixed than consistent on the mechanism linking inadequate sleep to obesity (Krietsch et al., 2019).

**Sleep Behavior and Hedonic Eating**

A greater desire for energy-dense food among short-sleepers has been established (Spiegel & Leproult et al., 2004). A large body of evidence (Chaput, 2016) suggests that
SLEEP AND EATING BEHAVIOR AMONG ADOLESCENT FEMALES

consuming large quantities of food after restricted sleep results from hedonic rather than hormonal factors; in an environment where energy-dense foods are highly palatable and readily available, caloric intake may be directly proportional to the time spent awake and snacking. Chaput and St-Onge (2014) document that sleep enhances vulnerability in the current obesogenic environment (an environment that promotes weight gain). They proposed a pathway arguing that insufficient sleep increases caloric consumption because excess energy intake is associated with inadequate sleep, explained by hedonic factors in the environment where hedonic aspects of food intake override hormonal factors. This relationship has been explained by conditioning theories (Chapman et al., 2012); repeated consumption of rewarding food results in the formation of new linked memories that condition the individual to anticipate reward in response to any environmental stimulus paired with the reward. This theory is supported by recent data where sleep duration restriction did not alter leptin and ghrelin levels but predicted increased snacking, indicating a predominantly hedonic rather than homeostatic drive for food intake (Nedeltcheva et al., 2008).

Specifically, sleep deprivation is associated with an increase in hedonic hunger and appetite ratings, with the most pronounced effects for energy-dense foods high in carbohydrates (Spiegel & Tasali et al., 2004). Findings from adult studies suggest that acute sleep deprivation influences hedonic rather than homeostatic pathways to food consumption (Chaput et al., 2012) more so because these eating behaviors result from the presence of food cues rather than due to impaired satiety processes (Chaput, 2014). Habitually short sleep duration is associated with increased food responsiveness in children (McDonald et al., 2015). In one such study, controlled curtailment of sleep in the context of ad libitum energy intake increased preference and consumption of energy-dense foods, without any evidence of change in metabolic signals.
(Markwald et al., 2013). Similarly, an experimental sleep restriction study showed increased consumption of desserts and sweets (foods with a high glycemic index) in adolescents (Beebe et al., 2013). In a similar study with adolescents, sleep-restricted adolescents (5 consecutive nights of 6.5 hours in bed) had 11% higher caloric intake and 52% greater consumption of sweet/dessert servings than healthy sleepers. Moreover, sleep restrictors rated pictures of sweets/dessert food to be more appealing (Simon et al., 2015). Likewise, adolescents with short sleep consume higher calorie fat food and have a 2-fold increased risk of consuming $\geq 475$ kcal/d from snacks (Weiss et al., 2010).

**Sleep Behavior, Hedonic Hunger, and Dietary Behavior**

Findings from pediatric and adult literature indicate that sleep restriction may increase the salience of palatable and consequently increased the drive to consume high caloric food within a permissive environment (Cappelleri et al., 2009). Thus, one potential pathway to explain the relationship between inadequate sleep behavior and poor dietary quality is via an increase in hedonic hunger among the overweight and obese population. The reward value of food can be amplified in genetically vulnerable individuals (genetic predisposition to obesity), it can also be increased by environmental factors (Chapman et al., 2012). As pointed out by Lowe and Butryn (2007), humans often consume food for pleasure in the absence of hunger or caloric need, a concept referred to as hedonic hunger. Eating in the absence of hunger is sustained by rewarding properties and easy access to palatable or hyper-palatable (e.g., high-fat, sugar-rich, and often salty) food. Reward learning and pleasurable incentives associated with palatable food consumption further exacerbate energy imbalance and vulnerability to hedonic experience (Coccorello & Maccarrone, 2018).
Overweight or obese individuals are at increased risk for hedonic hunger because body mass index influences the preferences for and consumption of highly palatable (often, high-fat) food (Lowe & Butryn, 2007). Moreover, people with high adiposity ingest more total food energy and fat than normal-weight persons (Lundahl & Nelson, 2015). Similar patterns are noted in young adults. In an investigation of food intake patterns and activity levels of weight gaining and weight-stable young adults, participants in the weight-gaining group consumed 1600 kcal more food energy (increased consumption of carbohydrates and fat) per day than their weight-stable counterparts which resulted in a gain of 1kg adipose tissue every 3 weeks. Moreover, the weight-gaining group ate larger meals and had more food in their stomach after each meal than the weight stable group (Pearcey & De Castro, 2002). Thus, overweight or obese young adults are at increased risk of eating even in the absence of hunger. Regardless of the precise mechanism, the forgoing arguments suggest that exaggerated hedonic response to food constitutes a psychological risk factor that promotes excessive energy intake in overweight and obese youth.

While BMI is related to energy intake, caution that this relationship may not be linear in nature (Finlayson et al., 2007). For instance, they argue that the combination of biological dispositions (behavioral risk factors) and the presence of a conducive food environment (obesogenic environment) leads to patterns of consumption characterized by the size, frequency, and availability of food. Moreover, there is increasing evidence that short sleep duration may affect food intake, appetite, satiety, and energy imbalance through modification of hormonal responses (Vgontzas et al., 2003). Taken together, these research findings suggest that inadequate sleep behavior in adolescents with elevated body weight contributes to increased caloric intake in the absence of hunger promoting obesogenic eating behavior. However, no
known study has investigated the plausible role of sleep behavior as a potential moderator of the associations between hedonic hunger and dietary behavior among overweight and obese young females.

**Sleep Behavior and Executive Dysfunctions**

The term executive functioning (EF) describes a broad category of complex cognitive processes involved in the control and coordination of willful action towards future goals (Goel et al., 2009). These processes include the ability to focus attention while ignoring irrelevant information, planning and sequencing thoughts and behaviors, updating information as contingencies change, inhibiting inappropriate thoughts or actions, forming abstract concepts, shifting mental set as appropriate, and thinking flexibly, divergently, and innovatively (Killgore, 2010).

A significant body of literature demonstrates that sleep deprivation degrades many aspects of neurocognitive performance (Durmer & Dinges, 2005). The prefrontal cortex (PFC) associated network areas are most sensitive to individual differences in sleep. Thus, cognitive processes including working memory, inhibition, and controlled memory processes tend to show the greatest deficits post-sleep deprivation among young adults and older adults (Wilckens et al., 2014). In a meta-analysis of the effects of short-term sleep deprivation (<48 hr) on various cognitive domains, Lim and Dinges (2010) presented three mutually compatible hypotheses. First, according to the controlled attention hypothesis, tasks that are monotonous or intrinsically less engaging are more severely affected by sleep deprivation (SD) because greater top-down control is needed to sustain optimal performance on these tests. Second, the neuropsychological hypothesis argues that sleep deprivation has domain-specific effects on cognition, specifically
tasks mediated by prefrontal cortex (PFC) function. Third, the vigilance hypothesis proposes that sleep deprivation impairs simple attention and vigilance abilities.

Moreover, research support that disrupted sleep patterns impair executive functions even independent of mental fatigue, stress, effort, test monotony, length, and difficulty (Nilsson et al., 2005). For example, restricting adolescents’ sleep to 5h for 7 nights showed degradation of sustained attention, working memory, executive function, and speed of processing even in academically strong students (Lo et al., 2016). Similarly, in an experimentally controlled laboratory sleep restriction study (14 days) with healthy young adults, sleep period chronically limited to 4h and 6 h night progressively eroded the psychomotor vigilance, working memory, and cognitive throughout the performance. Furthermore, the cognitive deficits were not accounted for by boredom, monotony, non-compliance, this provides convergent evidence for the adverse effects of chronic sleep restriction on cognitive functions (Van Dongen et al., 2003).

On the other hand, studies on the effect of sleep extension (1hr) on cognitive performance are scarce. In one such study, researchers investigated the effects of gradual sleep extension on children’s cognitive performance and noted significant improvement in continuous performance and memory tasks (Sadeh et al., 2003). A meta-analysis noted that longer sleep duration is positively associated with cognitive performance, specifically executive functions and cognitive performance involving multiple cognitive domains among the pediatric population (Astill et al., 2012).

Taken together, findings from numerous studies suggest that inadequate sleep impacts simple attention, complex attention, processing speed, working memory, short-term memory, and reasoning almost equally (effect size from -0.125 to -0.762), with tasks requiring greater
complexity being least impaired after a total sleep deprivation and simple attention and vigilance tasks with the most significant impairment (Lim & Dinges, 2010).

**Sleep Behavior, Executive Dysfunction, and Dietary Behavior**

In both adult and pediatric populations, impaired executive functioning has been linked to increased food intake (Riggs et al., 2012). A research study by Dohle and colleagues (2018) offered multiple pathways to explain the relationship between executive function (EF) and eating behavior: (1) Eating behavior may be a predictor of executive functioning; (2) There could be a bidirectional relationship between the two; (3) Executive functioning as a moderator may determine the strength between dietary intentions and actual eating behavior; (4) Lastly, the executive function could be influenced by a situational variable and in those cases, EF could be seen as a mediator. Taken together, it is plausible that impaired executive functioning as a result of sleep impairment could potentiate poor dietary behavior.

Inhibitory control is one of the components of executive function theorized to be a critical cognitive mechanism involved in food intake (Chuah et al., 2006). Inhibitory control can be defined as one’s ability to withhold a dominant response to an external cue to correctly respond to one’s goals (Ko & Miller, 2013). Greater inhibitory control is associated with healthy eating behaviors including lower saturated fat intake (Allom & Mullan, 2014), greater intake of fruits and vegetables (Hall P. et al., 2008), and less consumption of high fat foods (Limbers & Young, 2015). Whereas, poor inhibition and attention functioning predict obesity (Applehans, 2009). Several studies have employed tasks that measure dysregulated eating, reward sensitivity, and response inhibition (Naets et al., 2018) and most document that impaired inhibitory control predicts dysregulated eating (Limbers & Young, 2015). In one such study with college females, weaker executive control was related to greater caloric consumption, especially when the
reinforcing value of food was high (Rollins et al., 2010). Likewise, sleep-deprived healthy young men made more commission errors in a go/no-go task when food-related words were presented irrespective of their blood glucose levels (Cedernaes et al., 2014). Therefore, short sleep duration can impair executive functioning because it reduces cognitive control and inhibition, which then reduces the ability to resist temptation and exert self-control in the presence of palatable food (Burt et al., 2014).

There is a lack of literature on how inadequate sleep and its cognitive manifestations influence eating behaviors among overweight or obese adolescents. Studies with individuals with clinically elevated body weight have found reduced gray matter density in the hippocampus and cerebellum, regions that are important for EF (Mueller et al., 2012). It is an important area to study because frontal systems and executive functions are not well developed in adolescence (Steinberg et al., 2008), which can influence poor health decision making (Reynolds et al., 2019). Moreover, research support that neurobiological processes important in executive functioning appear to be altered in overweight/obese adolescents, potentially increasing their risk for suboptimal eating behavior. For example, overweight teens struggle to withhold the automatic response to eat in presence of palatable foods (Guerrieri et al., 2009). Furthermore, impaired executive functioning is associated with inappropriate or maladaptive eating behavior (Gowey et al., 2017). Poor planning and greater impulsivity are associated with binge eating and subsequent excess weight gain in overweight and obese females who attend college (Goldschmidt et al., 2015). Overall, studies support that overweight/obesity is associated with lower executive functioning, even after adjusting for age, sex, and education difference (Sabia et al., 2008).

Poor executive function predicts a significant increase in BMI due to suboptimal eating behaviors via multiple neurocognitive pathways, such as difficulties with self-regulating or
Inhibiting one’s eating behavior (Gowey et al., 2017). Moreover, impaired executive functioning abilities potentiate or maintain obesity via less optimal dietary behavior. Furthermore, living in an obesogenic environment exerts increased strain on planning in order to avoid overeating (Fitzpatrick et al., 2013). In a longitudinal investigation of relations between BMI, executive functioning, and binge eating in a sample of females (both normal weight and overweight/obese), poorer planning ability at age 10 predicted a greater increase in BMI from age 10 to 16. In addition, self-reported binge-eating tendencies mediated the relation between impulsivity and change in BMI. Such that for every 1 unit increase in impulsivity, the change in BMI from age 10 to 16 increased by 0.06 to 0.08 (Goldschmidt et al., 2015). However, studies that report an association between obesity and impaired executive functioning need to be interpreted with caution because the direction of the relationship remains unclear. Relatedly, few weight-control intervention studies have supported the inverse association between body weight and cognitive functioning (Chaddock et al., 2012).

Taken together, executive functioning plays a crucial role in translating intention into health behavior. Moreover, impaired sleep potentiates reduced executive functioning. However, no known study has examined the influence of inadequate sleep behavior on executive functioning and its result on dietary behavior. This is an important area of research because it is likely that the interaction between body weight, sleep deprivation, and executive functioning may be particularly detrimental to eating behaviors.

**Sleep Behavior and Self-control**

Sleep is part of an intricate physiological mechanism that restores the nervous system and contributes to long-term health, and poor sleep habits including inconsistent sleep times and inadequate sleep quantity negatively impact self-control (Barber et al., 2010; Hagger et al., 2010;
Pilcher, 2015). The potential interaction between impaired sleep parameters and self-control is not well understood and researchers have begun to examine the extent to which sleep behaviors influence self-control functioning.

Self-control represents one’s ability to alter, maintain or cease one’s current behavior, thoughts, or emotions (Baumeister & Heatherton, 1996; Muraven & Baumeister, 2000). Some studies suggest a close resemblance between executive function and self-control; however, executive functions reflect a range of processes that allow the individual to flexibly regulate attention and behavior (Saunders et al., 2018). Self-control involves the management of desired and undesired behavior by either doing the positive (desired behavior) or avoiding the negative (undesired behavior; de Ridder et al., 2011) or as good and bad; good self-control comprises of planfulness whereas poor self-control refers to impulsiveness (Wills et al., 2007). Although self-control focuses on people’s efforts to make a desirable response and inhibit undesirable responses, (Muraven & Baumeister, 2000) however, this exertion is influenced by whether it is state self-control or dispositional self-control (Tangney et al., 2004). State self-control varies across situations and time, whereas trait self-control is assumed to be relatively stable across situations over time. People who score higher in trait self-control are at the advantage of being able to resist problematic impulses, whereas low trait self-control is often equated with trait impulsiveness (Tangney et al., 2004). However, trait self-control likely interacts with situational depletion of self-control resources and making one more susceptible to depletion (Baumeister et al., 2007; Schmeichel & Zell, 2007).

A meta-analysis on behavioral concomitants of self-control noted three prominent theories of self-control (de Ridder et al., 2012). The discounting model of impulsiveness (Ainslie, 1975) asserts that self-control involves deciding between delayed but a more valuable
outcome over a more immediate outcome of less value. In the second model hot/cool system approach to self-regulation (Loewenstein, 2005), the cool system is associated with high self-control and lack of impulsive decision-making. In contrast, the hot system is associated with low-self-control and impulsiveness. The model that has received a decade of research and evidence is the strength model of self-control. According to Baumeister (2002) self-control derives from a limited resource and becomes exhausted after repeated acts of self-control. This exhaustion of internal resources that allow self-control is referred to as ego-depletion. A major tenant of this model is that engaging in acts of self-control draws from a limited “reservoir” of self-control that when depleted results in a reduced capacity for further self-regulation (Hagger et al., 2010). As a consequence, after people have engaged in acts of self-control, their capacity to exercise self-control further is exhausted. This likely results from a decrease in blood glucose and the glucose supplementation attenuate ego-depletion effects (DeWall et al., 2008). Meta-analysis report medium-to-large (d= 0.62) effect size of ego-depletion on self-control (Hagger et al., 2010).

Sleep deprivation creates a strong physiological need for sleep and can potentiate ego depletion and thus influence one’s ability to monitor and manage internal resources (Pilcher et al., 2015). It influences one’s capacity for performance as well as their access to physiological energy resources (Engel- Friedman, 2004). For example, sleep-deprived medical residents exhibited impaired cognitive energy for self-control (Zohar et al., 2005). Research suggests that sleep-deprived individuals select less challenging tasks than when fully rested (Engel-Friedman & Riela, 2004), signifying that sleep-deprived individuals could attempt to compensate by choosing easier tasks (Pilcher et al., 2015). On the other hand, sleep may replenish self-control
functioning, which is required to monitor and control thoughts, emotions, and behavior (Muraven & Baumeister, 2000).

In addition to the ego depletion pathway, sleep deprivation impairs metabolism and endocrine function thereby reducing responsivity to stimuli and resulting in decreased attention (Spiegel & Leproult et al., 2004). In a landmark study, Spiegel and colleagues (1999) examined the effects of sleep curtailment to 4 hours for 6 days, with baseline and recovery sleep opportunities and noted that compared to baseline, glucose tolerance decreased by 40%, glucose disposal decreased by 30%, acute insulin response decreased by 30%, and insulin sensitivity trended lower. Therefore, it is likely that self-control is influenced by inadequate levels of blood glucose and glucose utilization resulting from restricted sleep (Froy, 2007).

Several researchers have proposed that sleep is a method of replenishing and enhancing self-regulatory resources (Lim & Dinges, 2010; Zohar et al., 2005), and healthy sleep influence the successful management of internal resources (Froy, 2007). On those lines, poor sleep practices disrupt the self-regulatory replenishment and recovery process, which contributes to self-regulatory resource depletion (Barber et al., 2012). This argument is further augmented by studies that found that individuals with good sleep habits have a lower psychological strain and better self-control, suggesting that sufficient sleep is necessary for replenishing self-regulatory energy (Barber et al., 2010). As resources are sufficiently exhausted; the person may not be able to execute the necessary self-control until the physiological resources are restored. Thus, it is plausible to argue that sleep and self-control could create a feedback loop where good sleep habits could be an important component of the person’s capacity for self-control, and exhausting energy sources could result from poor sleep habits.
Sleep Behavior, Self-control, and Dietary Behavior

Poor sleep is associated with diminished self-regulation of appetite and an increased risk of overeating and obesity (Burt et al., 2014). Multiple studies have unequivocally noted that failure of self-control is associated with high caloric intake (e.g., Patton et al., 1995), weight gain, and obesity (Blundell & Gillett, 2001).

In a longitudinal study, children who exhibited low self-regulation had significantly higher BMI because of failures in the ability to delay gratification for a food item (Francis & Susman, 2009). Individuals scoring high in dispositional self-control are more likely than lower scores to report healthy dietary behaviors such as regular breakfast and avoiding unhealthy sweets (Junger & Van Kampen, 2010). On the other hand, Tsukayama and colleagues (2019) found that children who were rated as possessing higher self-control (by teachers and parents) were less likely to become overweight by age 15 years of age. Similarly, multiple weight-control studies illustrate that control over the pattern of food intake and food items selected, together with self-monitoring contribute to weight reduction and weight loss maintenance (Klem et al., 1997).

Research on self-control with individuals with elevated body weight argues that long-term demand on self-control may impair self-control, especially among dieters who are frequently faced with a situation requiring resisting to tempting food (Hagger et al., 2013). This is likely because engaging in acts of self-control (e.g., dietary restraint, emotion regulation) exhausts one’s capacity to exercise self-control, resulting in overeating to restore the depleted resources (Loth et al., 2016). In an examination of the interacting effect of BMI and ego-depletion on food consumed, Hagger and colleagues (2013) found that individuals with high BMI and high restraint demonstrated depletion of self-control and consumed more food
compared to those with high BMI but no restraint. However, they noted that the trait self-control did not moderate the effect of eating restraint and urged researchers to examine self-control in conjunction with executive functioning. In another study, a weight loss program with young adults found that participant’s level of self-control did not predict baseline percentage of fat consumption but predicted eating behavior such that high self-control was associated with fewer calories than lower self-control (Will et al., 2011). Taken together, an impressive amount of evidence suggests that self-control influences dietary behavior.

Even though several lines of evidence suggest that inadequate sleep is associated with diminished self-regulation of appetite and increase risk of overeating and obesity (Burt et al., 2014) however, little is known about the mechanism. According to Hagger (2014), sleep quality could influence self-control resources, or it may also moderate the association between self-control and health-behavior. Similarly, it is also likely that it may mediate the relationship, such that individuals with better sleep quality will be more likely to be better at engaging in health behavior since sleep quality would provide them with sufficient cognitive resources for effective planning. In another similar study, Hofmann (2014) presented a similar model arguing that trait self-control may mediate health behavior indirectly via intentions and goal-directed action or it may moderate effect on the intention-health behavior, such that people with high self-control are likely to form concrete, clear, and realistic intentions. Lastly, some researchers suggest an interaction between executive functioning and self-control predicting dietary functioning (Wyckoff et al., 2017). Research applying executive functioning to health behavior suggests self-regulation (self-control) could be a moderator of the intention-behavior relationship (Hall P. et al., 2008). For example, planning abilities predict variance in breakfast consumption (Wong & Mullan, 2009) whereas inhibition moderates sleep behavior (Kor & Mullan, 2011). Taken
together various researchers have suggested potential pathways explaining the association between self-control and impaired sleep functioning and how it can potentiate poor dietary behavior.

**Current Study**

The present study was designed to explore potential mechanisms driving the relationship between sleep (self-reported sleep duration and self-reported adequacy of sleep duration) and eating behaviors measured using HEI within a multisystemic conceptual framework (Figure 1).

The study assessed whether self-reported sleep duration (Sleep habit survey (SHS)—*figure out how long you usually sleep on a normal school night and weekends*) moderated the association between executive dysfunction and 24 hr caloric intake, self-control and, 24 hr caloric intake, and hedonic hunger and 24 hr caloric intake. Next, our study aimed to fill the gaps in research regarding the role of the self-reported adequacy of sleep duration and its influence on eating behavior. Thus, we assessed whether self-reported adequacy of sleep duration (SHS—*how often do you think you get enough sleep?*) moderated the association between executive dysfunction and 24 hr caloric intake, self-control and 24 hr caloric intake, and hedonic hunger and 24 hr caloric intake.

In addition to examining sleep behavior, the current investigation was designed to explore the conditional influence of sleep deprivation (0 hr) and sleep extension (9 hrs) on the relationship between executive dysfunction and caloric intake mediated by self-control (*Figure 3*). Using moderated mediation, we examined whether self-control mediated the association between executive dysfunction and caloric intake and whether experimental sleep condition moderating this association.
This study is unique because no published research has examined the effects of self-reported sleep duration and experimentally manipulated sleep on multiple eating behavior-related cognitive processes (hedonic hunger, executive functioning, and self-control). Moreover, these associations have never been explored among overweight/obese adolescents. Furthermore, studies on sleep behavior and eating are limited to the examination of caloric intake using dietary recall indices and have failed to examine the overall dietary quality. Dietary indices such as the Healthy eating index (HEI) represent overall dietary quality and are thus best suited to examine eating behavior. Findings from this study have the potential to inform health interventions that promote sleep management, weight loss, weight maintenance, and healthy eating habits.

**Hypotheses**

**Research Question 1**

To determine whether self-reported sleep duration (measured using the Sleep Habits Survey For Adolescents) and self-reported adequacy of sleep duration (measured using the Sleep Habits Survey For Adolescents; how often do you think you get enough sleep?) moderated the association between hedonic hunger (measured using the Power of Food scale) and caloric intake (measured using ASA-24 diet recall).

**Hypothesis 1a.** Given the existing research, we predicted a positive association between hedonic hunger and caloric intake moderated by sleep duration such that this association would be stronger for adolescents with shorter sleep duration controlling for self-reported adequacy of sleep duration.

**Hypothesis 1b.** Given the existing research, we predicted a positive association between hedonic hunger and caloric intake moderated by the self-reported adequacy of sleep duration
such that this association would be stronger for adolescents with inadequate sleep controlling for self-reported sleep duration.

**Research Question 2**

To determine whether self-reported sleep duration (measured using the Sleep habit survey) and self-reported adequacy of sleep duration (measured using the SHS; *how often do you think you get enough sleep?*) moderated the association between executive dysfunction (Global Executive Composite (GEC) measured using BRIEF-2) and caloric intake (measured using ASA-24 diet recall).

**Hypothesis 2a.** Given the existing research, we predicted a positive association between executive dysfunction and caloric intake moderated by self-reported sleep duration, with short sleep increasing the magnitude of this association controlling the self-reported adequacy of sleep duration.

**Hypothesis 2b.** Given the existing research, we predicted a positive association between executive dysfunction and caloric intake moderated by the self-reported adequacy of sleep duration such that this association would be stronger for adolescents with inadequate sleep controlling for self-reported sleep duration.

**Research Question 3**

The third aim of this study was to determine whether self-reported sleep duration (measured using the Sleep habit survey) and self-reported adequacy of sleep duration (measured using SHS; *how often do you think you get enough sleep?*) moderated the association between self-control (measured using the Self-control scale) and caloric intake behavior (measured using ASA-24 diet recall).
Hypothesis 3a.: Given the current research, we predicted an inverse relationship between self-control with caloric intake moderated by self-reported sleep duration, with short sleep increasing the magnitude of this association controlling for self-reported adequacy of sleep duration.

Hypothesis 3b. Given the existing research, we predicted an inverse association between self-control and caloric intake moderated by the self-reported adequacy of sleep duration such that this association would be stronger for adolescents with inadequate sleep controlling for self-reported sleep duration.

Research Question 4

The fourth aim of the study was to explore whether self-control mediate the association between executive dysfunction and ad libitum caloric intake.

Hypothesis 4a. We predicted that the indirect effect of self-control would be a function of sleep condition such that individuals with restricted sleep would report increased caloric intake due to ego-depletion when compared to individuals with sleep extension.

Method

Data for this study were collected as part of a larger investigation examining cognitive and behavioral effects of sleep deprivation. The details of the larger investigation are described in Figure 2, only the measures in bold were used for the purpose of the current investigation.

Measures

Body Mass

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) calculated for each participant. zBMI measurements have been accepted as a reliable indicator of
overweight and obesity in the children and adolescents population (Himes, 2009). zBMI is a moderately reliable indicator of body fat percentage (Mei et al., 2002). BMI was calculated using a standardized formula (\( \text{BMI} = \frac{\text{weight (kg)}}{\text{height (m)}^2} \); Keys et al., 1972) which was then be converted to an age- and sex-adjusted z-score using the Center for Disease Control and Prevention (CDC) zBMI calculator.

**Hedonic Hunger**

Participants completed the Power of Food Scale (PFS) at baseline assessment (Lowe et al., 2009). This measure is a 15-item questionnaire assessing appetite for and motivation to consume palatable food on a five-point Likert-type scale ranging from 1 (do not agree at all) to 5 (strongly agree) in three domains: (1) when food is available but not physically present; (2) when food is physically present but has not been tasted; and (3) when food has been tasted but not yet consumed. The PFS has been shown to predict problematic eating behavior in field and laboratory settings (Lowe et al., 2009). These three domain scores are aggregated to create a total score, which will be the variable of interest in this study. The scale has good internal consistency (Cronbach’s alpha from 0.81 to 0.91) and test-retest reliability (Cappelleri et al., 2009). With a community sample of preadolescents and adolescents, Cronbach’s alpha for the total scale and three subscales ranged from 0.86 to 0.95 (Mitchell et al., 2016). Research has also shown that PFS scores are not substantially affected by daily variations in hunger (Witt et al., 2014). PFS scores are not significantly affected by respondents’ hunger state when completing the measure suggesting that the hedonic hunger construct remains stable in the short term, regardless of one’s physiological need for food (Cappelleri et al., 2009). The PFS intentionally does not measure the quantity or frequency of actual palatable food consumed. Thus, it allows distinguishing motivation to consume from actual food consumption (Espel-Huynh et al., 2018).
**Executive Dysfunction**

The Behavioral Rating Inventory of Executive Functions 2 (BRIEF-2) was completed at the initial appointment prior to sleep modification as a baseline measure of broad executive dysfunction. The BRIEF-2 is an 80-item self-report inventory that assesses executive functioning in children and adolescents. 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”)(Gioia et al., 2000). There are eight clinical scales derived from the BRIEF-2: 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; Gioia et al., 2000). The scale has demonstrated reliability, validity, and clinical utility as an ecologically valid assessment of executive functions across a range of conditions. The BRIEF-2 self-report scales have demonstrated high internal consistency for Global Executive Composite (GEC) (α= 0.96) and moderate to high for the clinical scales (0.72- 0.96). The measure also has strong temporal stability (r= 0.89) for GEC for over a period of approximately five weeks (Gioia et al., 2000). Moreover, we know that sleep impairment in the pediatric population has shown to have deficits in a variety of executive functions including, inhibition and planning. Therefore, the global executive composite scale was used to assess executive functioning in this study. Taken together there is strong evidence of convergent and divergent validity of BRIEF-2 self-report.

**Self-Control**

The Self-Control Scale (SCS) is based on the theory of self-control as a limited mental resource (Tangney et al., 2004). It is a 36-items trait measure to assess individual differences in
the ability to inhibit impulses and control their emotions and behaviors. It has Likert-type response format ranging from 1 to 5 with higher scores representing better self-control. The scale measures five domains: achievement and task performance, impulse control behaviors, interpersonal relationships, and moral emotions. The scale has high internal consistency with alpha estimates between 0.83 and 0.85. Over a three-week interval, test-retest reliability was 0.87 (Tangney et al., 2004). A series of confirmatory factor analysis demonstrated that SCS consists of two factors: inhibitory self-control and initiatory self-control such that behaviors which required stopping a response were predicted by inhibitory self-control (e.g., smoking), whereas behaviors that required starting a response such as studying were predicted by initiatory self-control (Allom & Mullan, 2014). Items on SCS examines people’s ability to interrupt undesired behavioral tendencies and refrain from acting on them (e.g., “I am good at resisting temptations”) and initiating wanted behavior (e.g., “I am able to work effectively toward long-term goals.”) with an alpha of 0.65 for initiatory self-control and 0.68 for inhibitory self-control (de Ridder et al., 2011). Self-control measured by self-control scale on average has a small to medium effect on behavior, regardless of the type of behavior assessed. However, it has shown relatively stronger effects on performance at work and school \( r = 0.36 \), but relatively smaller effects on regulated eating \( r = 0.17 \) (de Ridder et al., 2012).

**Self-reported Sleep Duration**

The Sleep Habit Survey (SHS) was used to measure self-reported sleep duration using the following items: “during weekdays: How many hours (and minutes) do you usually sleep?”; and “during weekend days: How many hours (and minutes) do you usually sleep?” The validity of the sleep habit survey with adolescents has been supported by multiple studies (Wolfson & Carskadon, 1998). Moreover, it has been found to be valid and comparable to sleep diary and
actigraphy among adolescents (Wolfson et al., 2003). The subscales have good reliability (Lewandowski et al., 2011; sleepiness (alpha = .70), sleep/wake behavior scale (alpha = .75). Habitual weekly sleep score was calculated as: \((\text{min Weekdays} \times 5) + (\text{min Weekend days} \times 2))/7\). Multiple studies have examined total sleep duration in adolescents using this formula (Bel et al., 2013; Garaulet et al., 2011).

**Self-reported Adequacy of Sleep Duration**

The Sleep Habits Survey was used to assess the self-reported adequacy of sleep duration using the question “How often do you think you get enough sleep?” This is a Likert-type question with five possible options 1= always, 2= usually, 3= sometimes, 4= rarely, and 5= Never. Self-reported adequacy of sleep duration (how often do you think you get enough sleep) was measured using sleep adequacy ratings (adequate sleep [always and usually] inadequate [sometimes, rarely, never]). Multiple studies have examined subjective sleep quality/adequacy using a single item. For example, in an examination of the association between sleep quality, cardiovascular fitness, and BMI among adolescent girls, Mota and Vale (2010) asked participants to report the quality of their sleep by responding to the question “In general how is your sleeping time?” Items were scored on a Likert scale with 1= poor and 5= excellent. The researchers dichotomized the variable into good and poor sleep quality. Similarly, Buxton and colleagues (2009) examined the association of sleep adequacy and food choices among adults. They assessed sleep adequacy with the item: “How often during the past 4 weeks did you get enough sleep to feel rested upon waking up?” The response option ranged from never to very often and for analyses they combined often and very often in one category and never, rarely, and sometimes as a second category. In addition, Dorofaeff and Denny (2006) examined sleep patterns of secondary school students using single item. “If they (participants) thought that they
get enough sleep”, with the possible responses being: most of the time, half of the time, very little of the time, and I get too much sleep. Furthermore, in an examination of sleep duration and quality among obese adolescents, Bawazeer and colleagues (2009) asked participants to report sleep quality as either continuous or disturbed.

**Sleep Extension/Deprivation**

Participants were randomly assigned to one of the two sleep conditions sleep deprivation (0 hours of sleep) and sleep extension (9 hours of sleep). They were asked to adhere to the prescribed sleep duration during the experimental manipulation. Adherence to sleep protocol was measured using Actigraph GT3X+ accelerometers. 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, 2014). Total time in bed was determined with ActiLife5 software using Sadeh and colleagues (1994) sleep scoring algorithm. For the purposes of this study, we only examined the variable “total time in bed” as a measure of adherence to the assigned sleep protocol. 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); if adolescents failed to provide bed/wake times, research staff marked when physical activity nearly ceased in the evening (downward decline of physical activity) and wake time was established by visually marking when a participant engaged in a noticeable amount of physical activity in the morning. If no movement was recorded during the night, it was assumed that the accelerometer was removed prior to bedtime and the data was excluded.
**Caloric Intake**

The Automated Self-Administered 24-hour Dietary Assessment Tool for Children (ASA-24 Kids) was used to assess baseline dietary behavior. The ASA-24 is an Internet-based automated 24-hour diet recall assessment developed to understand the influences of nutritional status and assessment of dietary behavior change on health. Concordance rates suggest 47.8% match in recall between interviewer-administered and self-administered 24-hour diet recall (Baranowski et al., 2014). The literature suggests comparable report quality of self-administered dietary recall to the interviewer-administered dietary recall using ASA-24 (Hughes, et al., 2017). ASA-24 provides a valid estimate for intakes of a wide variety of dietary variables (Yuan, 2017).

**Diet Quality**

A healthy eating index (HEI-2015) was calculated as per the guidelines from the National Cancer Control and Population Sciences. ASA data was transformed using a SAS algorithm code available on the HEI website. The Healthy Eating Index (HEI) is a diet quality index that measures conformance with federal dietary guidelines. It is a measure of diet quality that assesses the adequacy of diet, including 1) total fruit; 2) whole fruit; 3) total vegetables; 4) greens and beans; 5) whole grains; 6) dairy; 7) total protein food; 8) seafood and plant proteins; and 9) Fatty acids (FAs). The remaining three, measure refined grains, sodium, and empty calories (i.e., calories from solid fats, alcohol, and added sugars). The score of the 12 components is summed to yield a total score, which has a maximum value of 100. Higher scores indicate better diet quality; HEI > 80 indicates a “good” diet, scores ranging from 51 to 80 reflect a diet that “needs improvement,” and HEI <51 implies a “poor” diet (Hurley et al., 2009). The index has demonstrated high concurrent validity with the values ranging from 86.4 - 98.1. The standardized Cronbach alpha coefficient of 0.68 indicates moderate reliability. Overall, research
support that the index has the ability to distinguish between nutritional quality among the pediatric and adult population (Guenther et al., 2014).

**Breakfast Buffet**

Methods such as 24-h dietary recall and food frequency recall are often used to measure energy and macronutrient intake. However, these methods are limited by forgetfulness and in some cases, the mere act of record keeping can cause a change in eating habits (Venti et al., 2010). Laboratory methods to assess energy intake such as buffet tables or *ad libitum* course meals are gaining attention and studies reported its reproducibility (Arvaniti et al., 2000; Gregersen et al., 2008; Nair, 2009). In our study, a standardized breakfast buffet was provided to participants to directly assess dietary behavior. The buffet consisted of the Frosted flakes, Frosted mini-wheats, Fruit loops, Chocolate muffin, Blueberry muffin, hardboiled eggs, Apple, Grapefruit cup, Vanilla yogurt, Strawberry yogurt, Berry horn pastry, Cheese horn pastry, Breakfast claw pastry, Apple granola bar, Blueberry granola bar, Orange juice, Milk, and water. Similar designs have been used in other studies; Broussard (2016) demonstrated that *ad libitum* feeding (buffet meals and snacks) corresponded well with daily diaries. In another study, McNeil (2017) used *ad libitum* breakfast buffet and instructed participants to consume “as much or as little as you want” from the foods that they selected from the menu (whole-wheat toast, strawberry jam, peanut butter, cheddar cheese, and orange juice). Energy and macronutrient intakes were assessed by weighing each food item before and after lunch consumption. Similarly, in our study participants were given 20-minutes to finish their meal. Once finished, the food was weighed again.
Analytic Plan

Data were analyzed using the Statistical Package for the Social Sciences (SPSS), version 21.0 (SPSS Inc). The predictor and outcome variables were examined for systematic patterns of missing values using Little’s Missing Completely at Random Test (MCAR). The MCAR test was not significant for any predictor or outcome variables ($p > .05$) suggesting that data were missing completely at random. The percentage of missing data in dependent variables [5% (HEI) and 3% (caloric intake *ad libitum*)] and independent variables [1.8% (hedonic hunger), 6% (executive dysfunction) and 18% (self-control)] was small. Statistical guidelines suggest that bias is likely in analyses with more than 40% missing data and thus warrants use of hypothesis generating methods (Madley-Dowd et al., 2019). MCAR is a special case of missing at random in which missingness does not depend on the observed data which results in a loss of statistical power. Although, MCAR data yields unbiased parameter estimates using maximum likelihood estimates. There are two prominent ways to handle missing data using listwise/pairwise deletion or imputation. Previous research has shown that one can remedy up to 20% - 30% of missing data with an imputation technique and still have good parameter estimates (Eekhout, 2014). To examine direct, indirect, and total effects and use bootstrapping, a regression imputation was used. The method of imputation for MCAR data involves placing estimates scores into the data set in the location of the missing data. In regression imputation, the model was fitted using maximum likelihood. In the next step, model parameters were equal to their maximum likelihood estimates and linear regression was used to predict the unobserved values for each case as a linear combination of the observed values for that same case, lastly, the predicted values were then plugged in for missing values (Arbuckle, 2011). Overall, regression imputation takes into consideration by predicting a score for each subject by using multiple regression based on their
non-missing scores for other variables. Research suggests that when the data are missing completely at random there is little difference in the estimation bias for listwise deletion, pairwise deletion, and maximum likelihood (Carter, 2006). In an examination of various kinds of imputation methods, Zhang (2016) found that mean, median, mode imputation methods are simple and thus tend to underestimate the variance. Whereas regression imputation can preserve their correlation with other variables because an estimated regression equation is used to generate predicted values for the cases with missing data. Moreover, there is little evidence of biased results using regression imputation. For example, Little and colleagues (2014) argue that regression imputation if used for MAR instead of MCAR could influence correlation and group differences.

In addition to ensuring we have an adequate sample size, we checked for normality in our dependent variable using Shapiro-Wilk test for normality. No kurtosis was observed, and dependent variables were relatively linear and homoscedastic. Because our statistical models are sensitive to outliers, we ensured that there were no significant univariate or multivariate outliers within our data.

SEM path analysis was done using the AMOS full information likelihood method (FIML) to handle missing data as described by Collier (2020). This method allows model parameters and standard errors to be estimated directly from the missing data. However, AMOS does not support advanced analysis options like bootstrapping and modification indices with missing data that is addressed by FIML. Furthermore, under small size ML estimators may be biased although they are asymptotically unbiased (Pituch & Stevens, 2015).

Examination of moderation was done using the PROCESS macro v3.2 model 1 (Hayes, 2015) for SPSS, and moderated mediation was examined using PROCESS macro v3.2 (Model
James and Brett (1984) coined the term moderated mediation implying that mediation models involving relations that require the addition of a moderator. According to Hayes (2015) moderated mediation models attempt to explain both how and when a given effect occurs. This kind of conditional process model examines how the indirect effect of X on Y through M could be contingent on a fourth variable if that fourth variable W moderates one or more of the relationships in a three-variable causal system. It is said to occur when the strength of an indirect effect depends on the level of some variable i.e., mediation relation is contingent on the level of a moderator. This model provides an opportunity to simultaneously investigate contingent and indirect effects. Similarly, such models are particularly useful to understand both why and under what conditions variables are related to each other. In addition to ensuring that data is MAR or MCAR, Preacher and colleagues (Preacher et al., 2007) recommended that the model should be specified to a reasonable degree (i.e., relationships are linear, disturbances are normally distributed, and homoscedastic). The variables that define the interaction terms, were mean centered prior to analyses using mean-centering function in the PROCESS macro.

A bias-corrected bootstrapping technique was used for analysis. It is based upon resampling procedures that determines the statistical significance and confidence intervals for the indirect effect. Bootstrapping determines the degree to which the sampling distribution of the research sample reflects that of the population (Mallinckrodt et al., 2006). This procedure is suggested to be superior in power and type 1 error rate than other methods. The bias-corrected bootstrapping procedure entails four steps: (a) creating a sample of $N$ cases to determine a bootstrap sample using random sampling with replacements, (b) calculating the path from the predictor variable to the mediator, the mediator to the outcome variable, and the relationship between each of the paths of the bootstrapping samples, and (c) re-run the number of bootstraps
specified in the procedure (Shrout & Bolger, 2002). This procedure improves statistical power and estimates confidence intervals. Moreover, research suggests that the resampling distribution is a superior method for testing the indirect effect (MacKinnon et al., 2002).

Results

Fifty-five overweight or obese (BMI % ile ≥ 85) female participants (ages 12-17; \(M \text{ age} = 16.48, \text{SD}=1.01\)) were recruited for the study (Table 1). Average family yearly income (M =$50, 000; SD=$5,254) and the sample was primarily White (not Hispanic, 66%). Participants’ BMI (\(M = 94.57; \text{SD} 4.4\)), self-reported sleep duration (M= 6.98; SD= 1.21), about self-reported adequacy of sleep duration, 58.5% reported adequate sleep whereas 41.5 % reported inadequate sleep. Participant reported healthy eating index (M= 43.59; SD= 14.5), ASA- 24 (M= 1692.8; \text{SD}= 647.9), Self-control (M= 122.7; SD= 15.9), Executive dysfunction (M= 86.21; SD= 15.9), and Hedonic hunger (M= 33.02; SD= 10.9). About sleep manipulation, 28 participants were randomized to sleep deprivation and 25 to sleep extension condition. Analyses of Pearson correlation noted significant positive correlation between executive dysfunction and self-reported adequacy of sleep duration (\(r = .356, p < .05\)), and a negative correlation between self-reported adequacy of sleep duration and self-control (\(r = -.370, p < .05\)), and negative correlation between executive dysfunction and self-control (\(r = -.752, p < .01\)).

Path Analysis

The model included two exogenous variables (self-reported sleep duration and self-reported adequacy of sleep duration) and four endogenous variables (executive dysfunction, self-control, hedonic hunger, and HEI). The chi-square test of model fit tested the null hypothesis that the population covariance matrix is equal to the implied matrix in the population after estimating the parameters using our sample data. Our model indicated a good fit with the data: \(\chi^2= 1.50\),
df = 7, p = 0.982. The non-significant chi-square test provides support for our hypothesized model. However, because chi-square statistics are highly sensitive to sample size (Kline, 2005), alternative fit statistics were also used. Root Mean-Square Error of Approximation (RMSEA) values < .05 are considered indicative of close fit whereas values up to .08 are considered acceptable fit (Pituch & Stevens, 2015). The pclose is a test of whether the model departs significantly from one that is close fit to the data. Overall, RMSEA value of .000 and pclose of .988 suggest a near-perfect model fit to the data. The Comparative Fit Index (CFI) ranges from 0-1. Our model with a CFI of 1.00 indicates a very good fit (values above .90 is the recommended cut-off).

Direct Effects

The proposed hypotheses were examined using bias-corrected bootstrapping methods (Figure 5). The hypothesized direct effect of self-reported sleep duration on executive dysfunction was not supported (B = -0.133, 95% [CI = -.298, .052], p = .164, SE = .0017). Whereas the standardized direct effect of self-reported adequacy of sleep duration on executive dysfunction was significant (B = 0.420, 95% [CI = 0.187, 0.632], p < .01, SE = 0.125). Support was demonstrated for the hypothesized direct influence of executive dysfunction on self-control (B = -.778, 95% [CI = -0.831, -.644], p < .001, SE = .052). Whereas the direct effect of executive dysfunction on hedonic hunger was not supported (B = -.13, 95% [CI = -0.295, .066], p = .109, SE = 0.109). Support was not demonstrated for the hypothesized direct influence of self-control on healthy eating index (B = -0.192, 95% [CI = -0.381, .054], p = 0.130, SE = 0.130). Similarly, the hypothesized direct influence of hedonic hunger on healthy eating index was not supported (B = 0.194, 95% [CI = -.381, .054], p = 0.152, SE = 0.147).
**Indirect Effects**

Support was not demonstrated for the hypothesized indirect relationship between self-reported sleep duration and hedonic hunger via executive dysfunction (B = .017, 95% [CI = -.002, 0.106], p = 0.146, SE = .023). The hypothesized relationship between self-reported sleep duration and self-control via executive dysfunction was not supported (B = -0.017, 95% [CI = -.029, .238], p = 0.172, SE = 0.172). Similarly, the proposed indirect influence of sleep perception on hedonic hunger via executive dysfunction was not supported (B = -.054, 95% [CI = -0.161, .012], p =.173, SE = .053). However, support was demonstrated for the indirect relation between sleep perception and self-control. Specifically, self-reported adequacy of sleep duration influenced self-control via executive dysfunction. The indirect effect was statistically significant (B = -.327, 95% [CI = -0.456, -0.102], p < .05, SE = 0.103). Thus, self-report of inadequate duration of sleep when compared to adequate sleep explained significant decrease in self-control functioning via executive dysfunction. Cohen’s d (Cohen, 1992) of this association was 0.33, corresponding to a small-to-medium effect size. The indirect influence of executive dysfunction on healthy eating index via self-control and hedonic hunger was not supported (B = 0.124, 95% [CI = -0.084, .259], p = 0.447, SE = 0.106). Similarly, indirect influence of sleep perception on healthy eating index was not supported (B = 0.052, 95% [CI = -0.019, 0.140], p = 0.287, SE = 0.287). Lastly, the indirect influence of self-reported sleep duration on healthy eating index was not supported (B = -.017, 95% [CI = -0.081, .002], p = 0.185, SE = .024).

**Moderation Analyses**

Moderation analyses were analyzed using the Preacher and Hayes SPSS Process macro created by Andrew Hayes (Preacher et al., 2007). This macro facilitates the implementation of
the recommended bootstrapping methods and provides a method for probing the significance of conditional indirect effect at different values of the moderator variable.

**Moderating Influence of Self-reported Sleep Duration on Hedonic hunger and Caloric Intake**

To test for the hypothesis that self-reported sleep duration moderates the association between hedonic hunger and caloric intake (ASA-24) we examined simple slopes and interaction effect. The overall model was not significant, $R^2 = .079$, $F (4, 44) = 0.784$, $p = 0.447$. Hedonic hunger did not influence caloric intake (ASA-24) ($B = -.867$, 95% CI [-2.5, .787], $t = -1.19$, $p = 0.237$). Similarly, sleep duration did not predict caloric intake (ASA-24) ($B = .002$, 95% CI [-.243, 0.248], $t = .021$, $p = 0.983$). Finally, the interaction effect of self-reported sleep with hedonic hunger was also not supported ($B = 0.102$, 95% CI [-.137, 0.343], $t = 0.862$, $p = 0.393$). Thus, self-reported sleep duration did not moderate the relationship between hedonic hunger and caloric intake (ASA-24).

**Moderating Influence of Self-reported adequacy of Sleep Duration on Hedonic hunger and Caloric Intake**

The test for the hypothesis whether self-reported adequacy of sleep duration moderate the association between hedonic hunger and caloric intake (ASA-24) was not supported, $R^2 = .066$, $F (4, 44) = 0.77$, $p = 0.545$. Hedonic hunger did not influence caloric intake (ASA-24) ($B = -.216$, 95% CI [-.605, 0.171], $t = -1.1$, $p = 0.224$). Similarly, self-reported adequacy of sleep duration did not influence caloric intake (ASA-24) ($B = 0.327$, 95% CI [-.313, 0.968], $t = 1.12$, $p = 0.308$). An interaction of self-reported adequacy of sleep duration and hedonic hunger did not predict caloric intake (ASA-24) ($B = .101$, 95% CI [-.484, 0.688], $t = 0.350$, $p = 0.727$). The results indicate that self-reported adequacy of sleep duration did not moderate relationship between hedonic hunger and caloric intake.
Moderating Influence of Self-reported Sleep Duration on Executive Dysfunction and Caloric Intake

The test for the hypothesis whether self-reported sleep duration moderate the association between executive dysfunction and caloric intake (ASA-24) was not supported, $R^2 = .058$, $F (4, 39) = .608$, $p = 0.652$. Executive dysfunction did not influence caloric intake (ASA-24) ($B = 1.19$, 95% CI [-.898, 3.29], $t =1.15$, $p = 0.255$). Similarly, self-reported sleep duration did not influence caloric intake (ASA-24), ($B = -.037$ 95% CI [-.314, 0.242], $t = -.265$, $p = 0.757$). Finally, the interaction of executive dysfunction and self-reported sleep duration did not explain variance in caloric intake (ASA-24), ($B = -.168$, 95% CI [-.447, .110], $t = -1.2$, $p = 0.298$). Thus, self-reported sleep duration did not moderate the relationship between executive dysfunction and caloric intake (ASA-24).

Moderating Influence of Self-reported Adequacy of Sleep Duration on Executive Dysfunction and Caloric Intake

The test for hypothesis whether self-reported adequacy of sleep duration moderate relationship between executive dysfunction and caloric intake (ASA-24) was not supported, $R^2 = .047$, $F (4, 40) = 1.16$, $p = 0.342$. Executive dysfunction did not influence caloric intake (ASA-24), ($B = -129.89$, 95% CI [-384.2, 124.4], $t = -1.03$, $p = 0.308$). Similarly, self-reported adequacy of sleep duration did not influence caloric intake (ASA-24), ($B = -172.7$, 95% CI [-417, 71.7], $t = -1.4$, $p = 0.161$). Finally, the interaction between executive dysfunction and self-reported adequacy of sleep duration did not explain change in caloric intake (ASA-24), ($B = 116.6$, 95% CI [-167.9, 401.2], $t = 0.518$, $p =.607$). Thus, self-reported adequacy of sleep duration did not moderate the association between executive dysfunction and caloric intake (ASA-24).
Moderating Influence of Self-reported Sleep Duration on Self-control and Caloric Intake

The test for hypothesis whether self-reported sleep duration moderate the relationship between self-control and caloric intake (ASA-24) was not supported, $R^2 = .224$, $F (4, 33) = 2.83$, $p = .072$. Self-control did not explain variance in caloric intake (ASA-24), ($B = -1.64$, 95% CI [-3.68, 0.383], $t = -1.65$, $p = 0.108$). Similarly, self-reported sleep duration did not influence caloric intake (ASA-24), ($B = -.027$, 95% CI [-.298, 0.244], $t = -.202$, $p = 0.841$). Finally, interaction between self-reported sleep duration and self-control did not influence caloric intake (ASA-24), ($B = 0.265$, 95% CI [-.009, 0.540], $t = 1.96$, $p =.0579$). Thus, self-reported sleep duration did not influence relationship between self-control and caloric intake (ASA-24).

Moderating Influence of Self-reported Adequacy of Sleep Duration on Self-control and Caloric Intake

The test for hypothesis whether self-reported adequacy of sleep duration moderate the association between self-control and caloric intake (ASA-24) was not supported, $R^2 = .07$, $F (4, 33) = 2.15$, $p = .091$. Self-control did not influence caloric intake ($B = -.804$, 95% CI [-.628, 0.467] $t = -.298$, $p = 0.767$). Self-reported adequacy of sleep duration did not influence caloric intake (ASA-24), ($B = 0.691$, 95% CI [-.040, 1.42], $t = 1.92$, $p = 0.063$). However, we noted significant conditional effects (Table 7) of self-reported adequacy of sleep duration. Specifically, self-report of inadequate sleep duration and self-control explained significant variance in caloric intake (ASA-24), ($B = .5300$, 95% CI [.084, .975], $t = 2.4$, $p <.05$) An interaction of self-control and self-reported adequacy of sleep duration did not explain variance in caloric intake, ($B = -3.21$, 95% CI [-21.1, 14.6], $t = -.325$, $p = 0.746$). Overall, self-report of inadequate sleep duration explained 343.38 kcal. Thus, those with inadequate sleep with self-control showed significantly higher caloric intake.
Conditional Effects of Sleep Deprivation and Sleep Extension

In examination of direct effects (Figure 3), executive dysfunction explained 0.729 units decrease in self-control functioning (b = -0.729, p < .001, 95% CI [-1.09, -.384]) controlling for other variables. Whereas sleep condition did not influence self-control (b = -0.047, p = 0.990, 95% CI [-7.95, 7.86]). Similarly, interaction of executive dysfunction and sleep condition did not explain variance in self-control (b = 0.160, p = 0.497, 95% CI [-.314, 0.635]). We did not find support for the influence of executive dysfunction on caloric intake ad libitum (b = -1.64, p = 0.825, 95% CI [-16.7, 13.5]). Similarly, sleep condition (b = 83.1, p = 0.492, 95% CI [-161, 328]) and interaction of executive dysfunction with sleep condition (b = 1.77, p = 0.801, 95% CI [-4.06, 38.0]) did not explain variance in caloric intake ad libitum. Whereas interaction of self-control and sleep condition explained significant variance in caloric intake ad libitum (b = 30.5, p < .01, 95% CI [7.10, 53.9]) holding other variables constant.

Conditional process modeling examined the conditional direct and indirect association between executive dysfunction and caloric intake transmitted through self-control contingent upon sleep condition (sleep deprivation vs sleep extension). For those under sleep extension condition, executive dysfunction explained 15.32 kcal increase in caloric intake (b = 15.32, p < .05, 95% CI [.440, 30.2]). Which implies that average change in caloric intake when executive dysfunction increases by 1 unit is 15.32 kcal. In terms of clinical implication, we examined standardized scores, and noted that an increase in executive dysfunction raw score by 15 points (1SD) explains an increase in caloric intake by 314.61 kcal. Examination of the conditional indirect effect of sleep conditions (deprivation vs extension) explained a significant difference in the indirect effects in the extension condition, whereas no association was noted in the deprivation condition. Sleep extension (Table 8) explained 18.4 units lower caloric intake via
self-control (b = -18.4, 95% CI -39.5, -7.02]). Specifically, the indirect relationship explained 484.69 kcal lower caloric intake. Which suggests that self-control mediated the relationship between executive dysfunction and caloric intake ad libitum for those who slept for 9 hrs. Further explained by a significant index of moderated mediation (-17.1, 95% CI - 40.4 to -2.27). *When the moderator W is dichotomous* (similar to our case), *the index of moderated mediation corresponds to the difference between the indirect effects in the two groups* (Hayes, 2015).

Specifically, for participants under sleep extension condition, 9 hrs of sleep, on average accounted for 484.69 kcal lower in caloric intake via self-control. For the examination of effect size (Cohen, 1992), standardized beta coefficients were used. This association explained a large effect size (*Cohens ’d* = 0.85). This supports the conditional nature of our presented relationship suggesting that self-control mediated the association between executive dysfunction and caloric intake for those with extended sleep compared to sleep deprivation. Taken together, significant conditional direct and indirect effects offer evidence that sleep condition influences the strength of the relationship between executive dysfunction and caloric intake ad libitum via self-control functioning.

**Discussion**

**Moderating Influence of Self-reported Sleep Duration**

Research suggests that sleep deprivation can contribute to obesity by promoting higher caloric intake that causes weight gain (Magee et al., 2010). A greater desire for energy-dense food among short sleepers (Chaput, 2016; Spiegel & Leproult et al., 2004) is widely noted. Moreover, previous studies have demonstrated that overweight and obese teens are more responsive to food cues in the environment, and short sleepers could be more susceptible to weight gain because they have more time to eat (Magee et al., 2010). Our first research question
(Hypothesis 1a) examined the role of self-reported sleep duration as a potential moderator of how hedonic hunger influences caloric intake. However, we noted no such association between hedonic hunger, sleep duration, and caloric intake. Although BMI is related to energy intake (Blundell & Finlayson, 2017), and sleep duration may affect food intake (Vgontzas et al., 2003), this relationship may not be linear in nature. For example, Spiegel and colleagues (Spiegel & Tasali et al., 2004) noted that “chronic” sleep deprivation is associated with an increase in hedonic hunger and appetite ratings. Similarly, among adults, “acute” sleep deprivation influences hedonic pathways to food consumption. Taken together, we noted no influence of sleep duration on hedonic hunger and caloric intake. It is possible that this finding is attributable to the fact that, on average, our participants did not have chronic sleep debt.

A significant body of literature demonstrates that sleep deprivation degrades many aspects of neurocognitive performance (Durmer & Dinges, 2005) and in both adult and pediatric populations, impaired executive functioning has been linked to increased food intake (Riggs et al., 2012.) However, our examination of whether sleep duration moderate association between executive dysfunction and caloric intake did not yield significant findings (Hypothesis 2a). No known study has examined this specific moderation hypothesis. Thus, while our findings are novel, but we addressed multiple factors that promote inadequate caloric intake among overweight and obese adolescent females.

Research has begun to explore the influence of sleep deprivation on self-control functioning and whether it potentiates caloric intake. Sleep deprivation creates a strong physiological need for sleep and can potentiate ego depletion and thus influence one’s ability to monitor and manage internal resources (Pilcher et al., 2015). Some argue that exhaustion of self-control resources could promote higher caloric intake. However, our examination of whether
self-reported sleep moderates the association between self-control and caloric intake (Hypothesis 3a) did not yield significant results. Specifically, we found no direct effect of hedonic hunger, executive dysfunction, or self-control on caloric intake nor a moderating effect of sleep duration on the association between hedonic hunger and caloric intake, executive dysfunction and caloric intake and, self-control and caloric intake.

Several potential explanations for why our hypotheses were not confirmed are possible. First, no known study has examined the aforementioned hypotheses. Rather, most previous studies have examined the association between sleep duration and risk of overweight and obesity (Fatima et al., 2015), whereas research on how sleep duration influences eating behaviors among females with clinically elevated adiposity is nonexistent. Therefore, our novel findings should be understood in the larger context of research on the relationship between self-reported sleep duration, eating behaviors, and obesity among the obese and overweight adolescent population. Literature on pediatric obesity relating to the influence of sleep on caloric intake is inconclusive (Chapman et al., 2012).

Next, our non-significant results may be attributable to sex-related differences in sleep characteristics (Mong & Cusmano, 2016) of our female-only sample. Mounting research suggests sex-related differences in the association between sleep duration and BMI, with previous research indicating that an hour increase in sleep duration explains a 10% reduction in risk of being overweight for males only (Lytle et al., 2011). Similarly, adolescent boys with < 8 hrs of sleep are at 3.1 times greater odds of developing obesity compared to females irrespective of hours of sleep duration. Furthermore, research suggests an OR of developing obesity of 4.85 for males who sleep less than 8 hours and 0.81 for females (Eisenmann et al., 2006). Our argument of significant sex differences is further augmented by a meta-analysis that reported that
boys with short sleep had an odds ratio for obesity (2.50) twice that of girls (1.24; Chen et al., 2008). These differences are attributed to variations in endocrinology during puberty; thus, metabolism may influence weight gain differently among males and females (Knutson, 2005). After adjusting for age, race, gender, and SES, an individual’s self-reported sleep duration was not a significant predictor of obesity among adolescents in two previous studies. (Calamaro et al., 2010; Guidolin & Gradisar, 2012). In another large investigation (74% overweight and obese adolescent females), male adolescents were significantly more likely to develop overweight and obesity compared to females due to sleep loss (Ferranti et al., 2016).

Second, a growing body of evidence on the association between short sleep and body mass in the pediatric population shows equivocal findings compared to the adult population (Patel & Hu, 2008). This inconsistency could result from the use of varying classifications of daily sleep duration across studies. For example, Gupta and colleagues (2002) noted that a short sleep (range 5-7 hrs; avg =7 hr) explained weight status among adolescents with elevated BMI. Whereas, a meta-analysis on the association between obesity and sleep among children and adolescents, found that most studies categorized short sleep as sleep less 10 hrs/night; Cappuccio et al., 2008). Furthermore, females with increased adiposity and short habitual sleep (<5 hrs) showed a significantly higher percentage of carbohydrate intake compared to 7 hrs of sleep (Al-Disi et al., 2010). In a similar study with a large sample of adolescent females (healthy and unhealthy weight), short sleep i.e., 8 hrs or less (≤6 - 8) showed association with higher BMI, body fat, lower quality food intake than those who slept 8 hrs or more (Garaulet et al., 2011). While our participants reported an average habitual sleep duration of 6.98 hrs (range 5.77- 8.19 hrs) which is in the upper range of short sleep, but it failed to capture the change in caloric intake. Similar results were reported in a large study with a nationally representative sample
(adolescents; 50% females). Mid-range sleep (7-8 h/night) did not significantly decrease the odds of consuming vegetables and fruit or increased odds of consuming fast food compared with the recommended sleepers (>8 hrs; Kruger et al., 2014).

Third, the examination of moderating effect of self-reported sleep duration could have limited our understanding of the influence of other plausible variables potentially contributing to sleep behavior and caloric intake. For example, we did not examine factors such as the timing of the sleep, recurrence of sleep deprivation, chronicity of sleep deprivation, and circadian functioning which are likely to have contributed to our non-significant findings. Cross-sectional data on sleep functioning in adults report that chronic sleep deprivation or recurrent bedtime curtailment promotes overeating due to change in peripheral concentrations of ghrelin and leptin (Nedeltcheva et al., 2008). Moreover, emerging research suggests that habitual sleep variation of more than an hour instead of habitual sleep is related to increased snack consumption, implying that maintaining a regular sleep pattern may decrease the risk of obesity among adolescents (He et al., 2015). Whereas Kjeldsen and colleagues (2014) identified the independent influence of short sleep duration and large variability in sleep duration as risk factors for obesity among school children. Furthermore, the timing of the sleep is also known to influence eating behavior (Baron et al., 2011). For example, in an investigation of sleep timing, diet, and BMI in healthy and overweight adolescents, those who had a late bed and late wake-up times reported a higher intake of energy-dense food, independent of the influence of sleep duration (Fleig & Randler, 2009; Golley et al., 2013). In a similar investigation, Beebe and colleagues (2015) noted that healthy weight adolescents (61 % females) with a later circadian preference, when shifted to “early to bed” sleep condition, showed improved diet intake.
Fourth what entails “insufficient sleep” is not certain. In a review of how lack of sleep contributes to obesity in adolescents, Chaput and Dutil (2016) echoed the same concerns. They argue that although the National Sleep Foundation recommends adolescents (14-17yrs) sleep between 8 to 10 hrs, however, there is a clear need for sleep deprivation/extension interventions in adolescents to determine the upper and lower limits of healthy sleep duration to help us gauge a dose-response relationship. Therefore, research is needed to understand the threshold value instead of range to decipher the complex underpinnings of the potential mechanism.

Taken together, based upon our current knowledge, the examination of insufficient sleep can cause weight gain (Chen et al., 2008) due to increased caloric intake (Chaput, 2014). However, recent studies are presenting a different picture. In a recent large systematic review (86 studies, between 2012 and 2017), Krietsch and colleagues (2019) investigated the relationship between sleep and dietary intake, altered eating behavior, physical/sedentary activity, or hormone-regulating hunger/satiety. The findings revealed highly variable results with a high risk of bias. Specifically, some consistent patterns were showing no cross-sectional association between sleep duration and caloric intake (despite experimental evidence). Thus, our results reflect the current status of research which has contradictory findings on sleep duration and eating behavior and its risk of obesity among adolescents. Overall, while self-reported sleep duration did not influence cognitive subsystems and caloric intake. But it is important to highlight that since no known study has examined this phenomenon in a female-only sample thus our findings will have a basis to draw comparison and generalization from the current literature once research with overweight and obese females is well established.
Moderating Influence of Self-reported Adequacy of Sleep Duration

The results of our study support a significant association between self-report of inadequate sleep duration and caloric intake. Research has begun to examine subjective sleep and its influence on weight status among adolescents and most suggest an inverse relationship (Vargas et al., 2014). Examination of subjective sleep behavior is pivotal to understand pediatric sleep behavior because it offers an understanding of the inter-individual variability in sleep needs (Chaput & Dutil, 2016). Moreover, it is increasingly recognized that self-reported sleep duration among adolescents is prone to error due to overestimation of actual sleep and social desirability bias (Girschik et al., 2012). Furthermore, eating practices are regulated in response to the sleep-wake cycle in line with individual’s circadian clock (Beebe et al., 2015) and adolescents with sleep debt struggle with metabolic functioning due to hormonal imbalance (Gupta et al., 2002). In a meta-analysis on sleep quality and obesity in the pediatric population, Fatima and colleagues (2016) urged researchers that sleep duration cannot be used as a proxy for sleep, and studies assessing sleep-obesity relationship should look for the cumulative effect of both sleep quality and sleep duration to understand the influence of inadequate sleep. On those lines, we examined the influence of self-reported adequacy of sleep duration and how it moderates the association between hedonic hunger and caloric intake ad libitum (Hypothesis 1b), executive dysfunction and caloric intake ad libitum (Hypothesis 2b), and self-control functioning and caloric intake ad libitum (Hypothesis 3b).

Growing research suggests an association between sleep quality and weight status but there is a discrepancy in results depending upon the criteria used to define sleep quality (Fatima et al., 2016). Nonetheless, mounting evidence support that sleeps quality seems to have a considerable role in overweight and obesity (Liu et al., 2011), perhaps independent to sleep
duration (Fatima et al., 2016). We classified adequate and inadequate sleep depending on participant’s subjective reports of how often they get enough sleep. No known study has explored how subjective sleep functioning i.e., self-reported adequacy of sleep duration relates to caloric intake among the overweight pediatric population. While a few have analyzed sleep adequacy by categorizing it into adequate and inadequate using the metric of recommended sleep for that age group. Such objective categorization may fail to reflect individual’s perception of their sleep requirements. For example, Bawazeer and colleagues (2009), examined sleep quality (measures using a subjective rating of intermittent vs continuous sleep) and noted a significant influence of sleep quality as a risk factor for the development of obesity among adolescents. Similarly, Valrie and colleagues (2015) examined the effect of sleep quality [using the Adolescent Sleep-Wake Scale (LeBourgeois et al., 2005)] on weight status and weight-loss among adolescent females and found that lower subjective sleep quality was related to higher weight status.

Self-reported adequacy of sleep duration did not influence the association between hedonic hunger and caloric intake ASA-24 and executive dysfunction and caloric intake ASA-24. However, self-reported adequacy of sleep duration moderated the association between self-control and caloric intake (Hypothesis 3a). Specifically, we noted conditional moderation effects, such that inadequate sleep with self-control explained significantly higher caloric intake. While no known study has explored the influence of self-control and how it relates with subjective sleep to influence caloric intake. Peripherally, our results align with Al-Disi and colleagues (2010) examination with overweight and obese adolescent females. Those with interrupted sleep (subjective sleep duration) showed a higher carbohydrate intake than those who reported uninterrupted sleep. Poor sleep quality can increase vulnerability to overeating in an obesogenic
environment partly due to changes in cognitive functions involved in reward saliency and inhibitory control (Chapman et al., 2012). A similar examination from Korea, augments our findings (Min et al., 2018). They asked participants (55% females) to report their sleep quality as good, moderate, and poor and found a significant association between poor sleep quality and increased intake of fast food, confectionaries, and soda. Another study with adolescents (74% overweight and obese females) reported a significant relationship between poor sleep quality (classified as sleepiness on a Likert scale) and dietary behavior (Ferranti et al., 2016).

It is interesting to note that we found a conditional influence of self-control and self-reported adequacy of sleep duration on caloric intake and this finding could have resulted due to multiple factors. For example, although the importance of self-control for well-being is well established in research (de Ridder et al., 2012), in recent years a growing body of evidence has found a small-to-zero correlations between trait self-report measures of self-control and behavioral inhibition (Wennerhold et al., 2020). The self-control measure we used in our study (Tangney et al., 2004) is limited when it comes to the examination of “inhibition” because the items are more globally reflective of self-discipline and goal directed actions (Saunders et al., 2018). Moreover, it is pivotal to understand that the Self-Control Scale asks about an individual’s typical behavior rather than their optimal ability. Therefore, the measure may not be well-positioned to measure stable life habits in particular situations (Wennerhold et al., 2020).

Our results can also be explained in the light of how practicing self-control in typical situations is easier than in situations that pose dilemmas. We know that adolescents with elevated adiposity are more responsive to environmental food cues when sleep deprived (Braet et al., 2008) and that they are more susceptible to overeating when sleep deprived (Bel et al., 2013). Thus, it is possible that inadequate sleep functioning counteracts self-control functioning.
Furthermore, we know that individuals with adequate self-control do not practice adequate self-control all the time because of the issues that arise from the person-situation debate (Fleeson & Jayawickreme, 2015). Specifically, a person shapes how situations impact behavior, and situations shape how a person’s attributes impact behavior (Furr, 2018). Research suggests that an individual’s behavior is variable across situations due to strong intraindividual variability (Fleeson, 2004). Along these lines, a person’s momentary behavior can vary widely, therefore prediction of how a person might act in a given situation is incomplete without examining psychological processes involved during interaction with situation. While Fleeson (2004), did not examine self-control specifically, they found wide variability in other traits like agreeableness, extraversion, or conscientiousness across context. Therefore, it is likely that a moderately self-controlled individual may not behave in a moderately self-controlled manner all the time due to situational demands (Wennerhold et al., 2020). Lastly, our findings reflect Hagger’s (2014) argument that sleep quality can moderate the link between self-control and health-behavior because, although the availability of self-control is a limiting factor on task performance, however, rest (good sleep quality) can assist in greater capacity to commit to behavioral engagement.

Overall, evidence from multiple studies strengthens our results suggesting that inadequate sleep can contribute to higher caloric intake. Furthermore, chronic inadequate sleep can pose significant challenges (Pilcher et al., 2015) because it can limit one’s practice of self-control and, over an extended period of time, is likely to influence dietary behavior. Limited self-control practice has long-lasting effects, because research suggests that self-control is associated with desirable behaviors. However, it results from “doing the right thing” most of the time over extended periods of time and not in single situation (Wennerhold et al., 2020). Furthermore,
among individuals with elevated weight status, optimal caloric intake requires additional self control that exceeds that required for normal weight individuals (Hagger et al., 2013). While self-control promotes desirable behavior in “typical” situations, poor sleepers with elevated weight are at uniquely at risk of self control failures and subsequent caloric overconsumption (Wennerhold et al., 2020). Our finding indicate a non-linear relationship between self-control, sleep, and caloric intake. Research on sleep and self-control suggests that poor sleep can disrupt replenishment and recovery processes (Barber et al., 2012). It is therefore logical to conclude that, among adolescents’ with overweight and obesity, there is a plausible feedback loop that could prevent exhaustion of energy required to practice self-control among adequate sleepers.

It is interesting to note that although the objective measure of sleep did not influence caloric intake, self-reported adequacy of sleep duration was a significant predictor of caloric intake. These results could arise from a low correlation between objective sleep and sleep quality. We know that subjective sleep ratings and objective measures of sleep quality often show a relatively modest or non-existent relationship (Baker et al., 1999). Thus, it is likely that our examination of objective sleep duration failed to capture the influence of sleep on caloric intake. Whereas self-reported adequacy of sleep duration grasped individual differences in sleep behavior and its consequences on dietary behavior. Thus, taken together from the standpoint of research on pediatric sleep behavior and obesity, we offer preliminary yet compelling evidence that inadequate sleep is a significant predictor of caloric intake.

Path Analysis

The mechanism by which inadequate sleep is associated with negative health outcomes, including obesity among adolescents is not well understood. Mounting evidence suggests that short sleep duration may be associated with negative health outcomes via poor regulation of
energy balance (Kruger et al., 2014). Moreover, change in sleep influences decision making and dietary choices in terms of both the quality and quantity of food consumed (St-Onge et al., 2011). For example, short sleep duration stimulates hunger thereby raising hunger rating and consequently resulting in elevated food consumption (Hogenkamp et al., 2013). On those lines, we examined the influence of self-reported sleep duration and self-reported adequacy of sleep duration on three cognitive systems (hedonic hunger, executive dysfunction, and self-control) and their consequent influence on the healthy eating index (HEI). It is a comprehensive measure of dietary functioning and is noted as preferable to an index of single nutrient or food in the area of dietary assessment (Radwan et al., 2015).

Self-reported sleep duration did not predict a change in cognitive systems, but self-reported adequacy of sleep duration supported our hypotheses. In our examination, we noted a direct influence of self-report of inadequate sleep duration on executive dysfunction. Few studies support that sleep problems and short sleep duration contribute to fatigue which in turn lowers energy levels thereby impairing the ability to implement executive function skills and self-regulation (Turnbull et al., 2013). While there has been relatively little attention to the relationship between sleep quality and executive dysfunction among overweight and obese adolescents. Measures of poor sleep quality have been found to be related to degraded performance on a number of cognitive tasks drawing on global executive functioning (Stormark et al., 2019). As per our understanding, research has not examined the influence of sleep quality on executive functioning and its consequent influence on eating behavior among overweight and obese adolescent females. In one related study, adolescents who had better sleep quality reported better self-perceived executive functioning and demonstrated better academic performance (Boschloo, 2013). Our results find their basis in studies that examined the influence of
inadequate sleep on working memory and reasoning (Lim & Dinges, 2010). Similarly, sleepiness (sleep deprivation) is associated with decreased performance on neuropsychological tasks, specifically executive functioning, needed for behavioral planning and self-control among adolescents (Anderson et al., 2009). This finding is consonant with our results because the self-reported adequacy of sleep duration, specifically self-report of inadequate sleep duration explained an increase in executive dysfunction.

Mounting evidence suggests that inadequate sleep is associated with decrements in executive function and self-regulation (Daviaux et al., 2014). Sleep reduction is likely to put constraints on an individual’s capacity to function, such as engaging in behaviors that require active control over impulses (Hagger, 2014). On the other hand, better sleep quality provides sufficient cognitive resources for more effective planning (Mullan et al., 2011). Our results suggest that impaired executive function explained a significant decrease in self-control functioning. We know that disruption of EF skills, such as working memory explains poor self-regulation of behavior among children (Turnbull et al., 2013). Our findings are consistent with other studies that support an association between executive functioning and its influence on the ability to suppress impulses and exert self-control (Clark et al., 2014). Finally, we noted that self-report of inadequate sleep duration was associated with executive dysfunction which consequently explained a decrease in self-control functioning. This association corresponded to a small-to-medium effect size. To contextualize these results, Dewald and colleagues (2010) noted a small effect with sleep quality predicting children and adolescents’ school performance. Limited studies have examined this mechanism, but research support that both short sleep duration and sleep difficulties predict impulsive behaviors and poor response inhibition in adolescents (Lundahl & Nelson, 2015). While we did not examine daytime sleepiness, but
inadequate sleep can result in daytime sleepiness and could influence self-regulation (Owens et al., 2016).

We proposed a multisystem mechanistic model specific to adolescents to examine several pathways by which inadequate sleep might influence dietary quality. However, we did not find support for the complete model. The self-reported sleep duration did not influence cognitive systems and did not explain dietary quality either. We have detailed potential factors that could have influenced our findings in the initial paragraphs on the moderating influence of sleep duration. Furthermore, it is important to understand that our model did not examine between-subject variables such as, family functioning, economic, and socio-cultural factors that could influence sleep and eating behavior (Duraccio et al., 2019). In addition, we did not examine physical functioning and lack of which could have influenced our findings. Research suggests that shorter sleep duration also causes tiredness and daytime drowsiness, which in turn can lead to decreased physical activity and a preference for watching television (Taheri, 2006).

Overall, combining findings from research on the association between inadequate sleep and executive dysfunction and with those on the association between executive dysfunction and its consequences on self-control functioning, our findings imply an indirect relationship between self-report of inadequate sleep duration and decrement in self-control functioning due to executive dysfunction. Our examination of the mechanism although did not offer substantial information but it suggests possible underlying causes of how sleep behavior could influence executive functioning and self-control.

**Conditional Effects of Sleep Deprivation and Sleep Extension**

Sleep researchers have begun to examine the impact of sleep extension on health functioning. In order to understand the conditional effects of sleep deprivation and sleep
extension on caloric intake we explored a moderated mediation model to examine the relationship between executive dysfunction and caloric intake via the indirect influence of self-control functioning. We found a direct linear relationship between executive dysfunction and caloric intake. Our results align with Dohle and colleagues (2018) assertion that executive dysfunction may predict eating behavior, or it could mediate the association between dietary intentions and eating behavior. Moreover, self-control mediated the association between executive dysfunction and caloric intake under the sleep extension condition. To our knowledge, no known study has examined self-control functioning as a mediator of influence of executive functioning and caloric intake. However, researchers suggest that adequate sleep may moderate the association between self-control and health-behavior, particularly behaviors that have been identified as having regulatory problems such as participating in physical activity, eating healthily, reducing alcohol intake, and quitting smoking (Hagger, 2014). Moreover, healthy sleep helps in maintenance of cognitive for effective planning (Hofmann, 2014). Taken together we found that 9 hrs of sleep fostered greater self-control functioning which promoted fewer caloric intake.

Our examination of whether sleep deprivation explains a decrease in self-control thereby increasing caloric intake produced non-significant results. There are several possible explanations for the lack of association. First, only a few studies have reported such an interaction and this association is not well established. For example, an experimental sleep loss of one night explained deficits in self-control such as impaired decision making, response inhibition, and attention control (Chuah et al., 2006). It is important to mention that researchers used go/no go task only. Whereas few studies report a lack of association between sleep restriction and dietary behavior. For example, an experimental study of adolescent males’ short-
term sleep restriction (4 d) demonstrated a decrease in ad libitum energy intake and decreased motivation to eat (Klingenberg et al., 2012). Second, acute sleep deprivation has shown a consistent influence on caloric intake. It is possible that one night of sleep deprivation perhaps failed to influence caloric intake. Previous studies that reported significant findings included longer periods of sleep restriction. For example, Beebe and colleagues (2013) found that five nights of sleep restriction in adolescents explained increased consumption of sweets, thereby potentially causing an elevated risk of obesity among healthy-weight adolescents. Similarly, in an acute partial sleep deprivation study, Brondel and colleagues (2010) reported that reduced sleep significantly increased ad libitum food intake in a laboratory setting in healthy men. Similarly, St-Onge and colleagues (2011), noted that 5 nights of short sleep showed an increase in energy and fat intake among normal-weight men and women. Lastly, an experimental sleep restriction over 8 days with young adults noted that sleep restriction explained a 599 kcal/d increase in caloric intake (Calvin et al., 2013). Third, it is possible that the association of sleep deprivation with dietary intake is not straightforward. An observational study with school children reported that short sleep duration was associated with overweight and obesity, but neither energy intake nor snacking mediated the association (Chaput et al., 2010). Similarly, a 3-week sleep manipulation among normal BMI adolescents did not influence food appeal and ratings of food items (Simon et al., 2015). Whereas a night of wakefulness was associated with increase in morning plasma concentrations of the hunger-producing hormones ghrelin, but it did not alter taste preference (sweet taste) in a sample of male young adults (Hogenkamp et al., 2013).

There is a small body of literature that supports beneficial effects of sleep extension on health (Mantua et al., 2019) and only limited studies have examined the influence of sleep
extension on caloric intake. In our examination the conditional direct effect in the sleep extension condition demonstrated that an increase in executive dysfunction was associated with an increase in caloric intake. Whereas indirect effect via self-control showed an inverse relationship between executive dysfunction and caloric intake. Specifically, we noted mediating influence of self-control, such that higher self-control functioning explained a decrease in caloric intake for those who slept for 9 hours of sleep. Although, our period of sleep extension is too short to influence long-term self-control functioning but research on resource replenishment suggests that people can build their self-regulatory capacity (Muraven & Baumeister, 2000). We found evidence of significant difference in the caloric intake ad libitum between sleep deprivation and extension. Specifically, the difference between the two sleep conditions accounted for 454.26 kcal fewer calories due to the indirect influence on self-control. This association constitutes a large effect size (Cohen’s $d = 0.85$). In terms of clinical implications of our findings, Benjamins and colleagues (2021) noted that, under sleep deprivation conditions, participants consumed 134.5 kcal more compared to normal sleep conditions. Whereas normal sleepers consumed 102 kcal among normal weight females. Moreover, a daily energy deficit of 500-600 kcal can promote satiety and provide a great variety of beneficial micronutrients and lead to a modest weight loss of 0.5 kg per week or 2 kg per month among adults with elevated body weight (Koliaki et al., 2018). Our findings find its basis in limited and novel pediatric literature on sleep extension. First, it is possible since our participants reported short habitual sleep duration, thus an extension of sleep provided resource replenishment. Numerous studies have established sleep as a restorative mechanism influencing performance in cognition-demanding activities (Barber et al., 2012; Tyler & Burns, 2008). Growing evidence argues that sleep can serve to replenish self’s depleted resources, in part, due to relaxation (Baumeister,
2002), because it has been shown to counteract the negative effects typically associated with resource depletion (Smith, 2002). Furthermore, while the association of sleep extension and cognitive functioning is still under investigation. One such study examined the effects of gradual sleep extension adolescents with chronic sleep reduction (85.5 % females) and noted that sleep extension (advancing bedtime by 55 minutes) and following a personalized sleep schedule explained faster reaction times and correct responses on visuospatial processing (Dewald-Kaufmann et al., 2013). Moreover, while insufficient sleep impairs self-regulatory capacities, several studies suggest that consistently sufficient sleep may improve people’s ability to self-regulate (Nauts & Kroese, 2017). For example, increasing total sleep time is associated with reduced sleepiness and fatigue and improved attention, reaction time, and mood (Kamdar et al., 2004). Second, self-control functioning was more pronounced among individuals with 9 hrs of sleep, possibly because sleep extension is conducive to optimal glucose functioning. Short-term sleep studies suggest that sleep extension influences metabolism and energy expenditure, diminishing the desire for high energy density food, and increasing insulin sensitivity (Tasali et al., 2014). Moreover, impaired glucose has been linked with impaired performance on complex cognitive tasks and limits effortful processes (Fairclough & Houston, 2004). While depletion in self-control capacity influences blood glucose levels (Baumeister et al., 2007), sleep extension likely helped in the maintenance of glucose levels conducive to healthy caloric intake. Thus, it is likely 9 hrs of sleep helped maintain glucose metabolism in the sleep extension condition and, perhaps as a result, individuals did not seek food for fast-acting glucose contained in many unhealthy sweets or drinks (Gailliot et al., 2007). Third, we argue that 9 hours of sleep fostered self-control because, according to the strength model of self-control (Baumeister et al., 2007) adequate sleep maintained self-control resources and thus promoted lower caloric intake. Our
results align with Moreno-Frías and colleagues' (2020) study, where they noted that sleep extension of 1.1 hours for 4 weeks facilitated caloric restriction and weight reduction of .89 kg among overweight and obese adolescents. Taken together, while novel, but our examination of experimental manipulation of sleep contributes to the understanding of the potential mechanism that directly and indirectly explains the influence of sleep on caloric intake in lab settings. Our findings suggest that the resource-replenishment approach to adequate sleep deserves further investigation. Taken together, we found evidence that 9 hours of sleep may bolster cognitive resources to ameliorate executive dysfunction’s influence on caloric intake.

**Limitations**

Our study was not without limitations. First, although our female-only sample is a strength of our study, it limits generalizability to individuals who are not female. Future studies should address sex differences in the outcome measures associated with sleep functioning. Additionally, our sample was mostly white, high socioeconomic status overweight and obese females. The relationship between sleep and weight gain is further complicated in the overweight and obese pediatric population because weight status is a predictor of sleep problems, therefore, the directionality of this relationship is not certain (Liu et al., 2012). Thus, we highlight that there may be sample effects that emerged from our unique sample. A greater degree of racial/ethnic and socioeconomic diversity would allow us to generalize findings more broadly.

Second, while our examination of the mechanistic pathway explored the influence of potential pathways it is by no means comprehensive enough because we did not assess the influence of energy expenditure on that relationship. Thus, long-term studies are needed to unleash the role of sleep behaviors on eating behaviors. For example, we did not examine how sleep deprivation impacts hormonal functioning. It is a pivotal area to examine because sleep and
disorders influence metabolic functioning and sleep deprivation may have profound metabolic and cardiovascular implications (Sharma & Kavuru, 2010).

Third, considering the cross-sectional nature of this study, causality and direction of influence may only be inferred with limitations due to the lack of temporal precedence between variables of interest within this study. Additional studies should continue to evaluate the causal relationship assumed in the present examination while attempting to rule out plausible alternative models through randomized experimental design.

Fourth, self-reported diary recalls, such as ASA24, tend to underestimate total food intake, particularly for individuals with overweight and obesity, and are less reliable than other methods of food intake (Kupis et al., 2019). While we examined dietary quality using Healthy Eating Index (HEI), we only had a single recall for each participant and thus what we have is a snapshot of overall diet quality.

Fifth, while both the BRIEF scale and Self-control scale have good psychometric properties, our reliance on a single measure and a single reporter could have introduced mono-method measurement bias. Future research could address this limitation through direct behavioral measurement and/or the use of neurophysiological measures. Similarly, it is important to note that sleep quality and self-reported adequacy of sleep duration are subjective concepts and interpretation may vary from individual to individual. While we examined self-reported adequacy of sleep duration using the Sleep Habit Survey, it only contains one question that explicitly relates to sub-constructs of sleep behavior. Thus, standardization of sleep quality and subjective components could lead to improved clinical implications.

Sixth, we did not control or examine participant’s daily routines. Research suggests that energy expenditure (Markwald et al., 2013), sedentary activity (Chaput et al., 2011), and screen
time (Duraccio et al., 2019) can influence adolescent’s sleep behavior. Similarly, while participants fasted prior to coming to the appointment, but we were unable to assess for other behaviors they engaged in before sleep manipulation, which may have influenced caloric intake (ad libitum).

Seventh, while we instructed participants in both sleep conditions to refrain from strenuous activities. However, we did not examine for what did participants in the sleep deprivation condition do while they maintained sleep deprivation. For example, caffeine use interferes with sleep duration and quality, and we did not account for whether participants consumed caffeinated beverages to energy drinks (ED) to combat sleepiness (Moore & Meltzer, 2008). Moreover, consuming one or more ED a day is associated with elevated problems in self-reported behavioral regulation among young adolescents (Batenburg-Eddes et al., 2014).

Eighth, participants finished a chatroom task prior to buffet which could have introduced observer effects. Although we left participants alone with the ad libitum buffet and placed them facing away from any cameras to avoid changing their behavior due to being monitored, participants still may have felt they were being observed during the study and altered their food consumption due to observer effects.

Lastly, some bias might have been created because of the self-selection process that occurs in voluntary research. Individuals who participated in this study might have been more interested in learning about sleep behavior and brain functioning. This might lead to a sample that possesses more motivation to improve their sleep behavior.

**Clinical Implications**

Mounting evidence suggests that inadequate sleep is a risk factor for overweight and obesity. However, limited research has explored the underpinnings of this process in overweight
and obese adolescent females. At this time there is mixed research on the influence of self-reported habitual sleep duration on caloric intake, partly due to the use of the varied classification of inadequate sleep duration in pediatric research. We addressed several gaps in this literature by examining self-reported habitual sleep duration as a moderator of the association between cognitive systems (hedonic functioning, executive dysfunction, and self-control) and caloric intake. Sleep duration did not influence caloric intake, supporting previous research which suggests that sleep duration is not a salient predictor of caloric intake. In our understanding, researchers should examine “sleep behavior” rather than sleep duration, because sleep is not a regular, singular, and static activity. Thus, examination of potential factors such as sleep quality, the timing of the sleep, and chronicity of sleep deprivation is likely to inform our understanding of the underpinnings of how sleep behavior influences dietary behavior.

Prior research efforts have not directly examined the perception of sleep and our study is first to address the influence of self-reported adequacy of sleep duration. We examined the influence of perception of sleep adequacy on three cognitive systems (hedonic hunger, executive dysfunction, and self-control) and its influence on caloric intake.

We noted that self-report of inadequate sleep duration along with optimal self-control explained significantly higher caloric intake. These findings are concerning because poor sleep impacts self-control which may lead to caloric overconsumption for youth with overweight or obesity. Moreover, researchers have begun to argue that sleep and self-control can create a feedback loop where good sleep habits could improve self-control and vice versa. (Pilcher et al., 2015). Thus, clinical intervention studies geared towards improving subjective sleep functioning are warranted to promote adequate caloric intake.
Our findings revealed some interesting nuances in the relationship between sleep behavior, cognitive systems, caloric intake, and dietary quality. The perception of inadequate sleep influenced executive dysfunction which further explained the decrease in self-control functioning. Poor sleep quality is detrimental to adolescents’ overall functioning but more so in overweight and obese adolescents. Chronic sleep restriction puts an individual at an increased risk of poor sleep quality, influencing self-control and thereby resulting in higher caloric intake (Pilcher et al., 2015). Whereas better sleep quality provides individuals with sufficient cognitive resources for more effective planning and engagement in health behavior (Hagger, 2014). Moreover, previous investigations focusing on the association between subjective sleep quality and various aspects of cognitive performance have noted a potential relationship between procedural learning/memory including perceptual-motor skills, habits, grammar, and social skills (Zavecz et al., 2020). Our results further support the need to examine and improve sleep functioning in adolescents due to its detrimental influence on cognitive functioning.

Although self-control functioning and hedonic hunger did not influence dietary quality in our study, these findings have significant clinical implications. We recommend the use of a multi-method assessment of self-control functioning including the use of behavioral measures to assess the influence of self-control on dietary quality. This approach will improve understanding of how sleep quality can influence self-control functioning and how sleep quality relates to dietary quality, because previous research suggest that trait measures of self-control are limited when it comes to assessing self-control in demanding situations (Wennerhold et al., 2020) such as deciding between sweets and vegetables after a night of inadequate sleep. Our data are well-positioned to recommend clinicians and researchers to incorporate strategies to improve sleep quality to promote adequate dietary behavior and maintain healthy weight functioning. For
example, sleep loss can modify energy intake and expenditure. In an experimental sleep restriction study sleep curtailment among overweight adults’ sleep deprivation was associated with a decrease in fraction of weight loss by 55%, moreover it explained increased body mass by 60% (Nedeltcheva et al., 2010). Thus, insufficient sleep may inhibit the maintenance of fat-free body mass and curtail weight loss. We believe that our findings have direct relevance for weight-management programs that aim to improve sleep behavior as an important component. In a 6-month weight-loss program with overweight/obese adult females, better subjective sleep quality increased the likelihood of weight loss by 33% as did sleeping more than 7 hrs/night (Thomson et al., 2012). Moreover, our data align with studies that recommend efforts targeting improvement in sleep behavior along with executive and self-control functioning due to its beneficial effects on adolescent’s dietary behavior (Dassen et al., 2018).

Lastly, our examination of the conditional influence of executive dysfunction on caloric intake via self-control functioning between sleep extension and deprivation adds to the limited pediatric literature on sleep extension interventions. A brief period of sleep extension demonstrated that executive dysfunction had a direct effect on higher caloric intake ad libitum. However, the inclusion of self-control as a mediator indicated significantly lower caloric intake. Specifically, in terms of clinical implications, our result suggests adequate self-control functioning can mitigate executive dysfunction and promote adequate caloric intake (lower by 484.69 kcal) among individuals with 9hrs of sleep. These findings imply that adequate sleep may protect against executive dysfunction failures via self-control and promote adequate caloric consumption.

We conclude that a significantly high percentage of overweight and obese girls are not meeting daily recommendations for sleep and are thus struggling with poor sleep quality
resulting in limited executive and self-control functioning consequently leading to higher caloric intake. We offer novel yet promising evidence that augments the benefits of sleep extension. Overall, our findings can inform parents, families, educators, clinicians, and policymakers. First, because there is a low agreement between objective and subjective sleep reports (Zavec et al., 2020), both should be assessed before planning a treatment. Second, adequate sleep is key to self-regulation of behavior because it maintains health-behavior change and replenishes self-control resources (Barber et al., 2012). Therefore, examination of self-control and executive skills in weight management interventions will improve understanding of factors that promote poor sleep and cognitive functioning to improve weight status among adolescents with elevated adiposity. Third, it is useful for parents and clinicians to explore factors that encourage poor sleeping behavior, including screen use, to promote sleep hygiene among adolescents. Last, we recommend that researchers examine social, cognitive, and motivational factors to predict an individual’s motivation and intentions to engage in behaviors that promote adequate sleep (Hagger, 2014).
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### Table 1
**Demographic and Anthropometric Variable Means and Standard Deviations**

<table>
<thead>
<tr>
<th>Variable</th>
<th>Mean (SD)</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (SD)</td>
<td>16.48 (1.01)</td>
<td>4.73</td>
</tr>
<tr>
<td>BMI (SD)</td>
<td>94.57 (4.4)</td>
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<td>Ethnicity</td>
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<tr>
<td>Other</td>
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<td></td>
</tr>
<tr>
<td>Habitual Sleep Duration</td>
<td>6.98 (1.21)</td>
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</tr>
<tr>
<td>Executive Dysfunction (GEC)</td>
<td>86.21 (15.9)</td>
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<tr>
<td>Self-control</td>
<td>122.7 (15.9)</td>
<td></td>
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<tr>
<td>Caloric Intake (ASA-24)</td>
<td>1692.8 (647.9)</td>
<td></td>
</tr>
<tr>
<td>Caloric Intake ad libitum</td>
<td>739.4 (533.8)</td>
<td></td>
</tr>
<tr>
<td>Hedonic hunger (PFS)</td>
<td>33.02 (10.9)</td>
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<tr>
<td>Healthy Eating Index (HEI)</td>
<td>43.59 (14.5)</td>
<td></td>
</tr>
</tbody>
</table>

N observations = 53

### Table 2
**Summary of Pearson Correlations for Habitual sleep, Executive dysfunction, Hedonic hunger, Self-control, Caloric intake (ASA-24), Healthy Eating Index (HEI), and Caloric intake Ad libitum**

<table>
<thead>
<tr>
<th>Measure</th>
<th>HS</th>
<th>SP</th>
<th>GEC</th>
<th>PFS</th>
<th>SC</th>
<th>ASA-24</th>
<th>HEI</th>
<th>Ad libitum</th>
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<tbody>
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<td>SP</td>
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<td>GEC</td>
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<td>PFS</td>
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<tr>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ASA-24</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HEI</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Ad libitum</td>
<td></td>
<td></td>
<td></td>
<td></td>
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</tbody>
</table>

Note: HS= habitual sleep. SP= Perception of sleep adequacy. GEC = executive dysfunction. PFS = hedonic hunger. SC = self-control. HEI= healthy eating index. ASA 24= Caloric intake. Ad libitum = buffet caloric intake

***P < .001, **p < .01, *p < .05
Table 3
Standardized Direct Effects Using Path Analysis

<table>
<thead>
<tr>
<th>Variables</th>
<th>β</th>
<th>p</th>
<th>Bootstrap SE</th>
<th>BBLL</th>
<th>BBUL</th>
</tr>
</thead>
<tbody>
<tr>
<td>HS</td>
<td>-133</td>
<td>.164</td>
<td>.104</td>
<td>-.298</td>
<td>.052</td>
</tr>
<tr>
<td>SP</td>
<td>.420***</td>
<td>.001</td>
<td>.125</td>
<td>.116</td>
<td>.566</td>
</tr>
<tr>
<td>GEC</td>
<td>-.129</td>
<td>.348</td>
<td>.109</td>
<td>-.295</td>
<td>.044</td>
</tr>
<tr>
<td>PFS</td>
<td>-.778***</td>
<td>.001</td>
<td>.052</td>
<td>-.831</td>
<td>-.644</td>
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<tr>
<td>SC</td>
<td>-.192</td>
<td>.152</td>
<td>.130</td>
<td>-.381</td>
<td>.054</td>
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</tbody>
</table>

Note: HS= Habitual sleep. SP= Perception of sleep adequacy. GEC = Executive dysfunction. PFS = Hedonic hunger. SC = Self-control. HEI= Healthy eating index. ASA 24= Caloric intake. Ad libitum = buffet caloric intake. S= Sleep condition

***P < .001, **p < .01, *p < .05
N observations = 53
Model fit: χ²= 1.50, df= 7, p=.982
CFI= 1.00; RMSEA=.00

Table 4
Standardized Indirect Effects Using Path Analysis

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<tr>
<th>Variables</th>
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<td>.053</td>
<td>-.161</td>
<td>.012</td>
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<tr>
<td>HS → GEC → PFS</td>
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<td>.146</td>
<td>.023</td>
<td>-.002</td>
<td>.106</td>
</tr>
<tr>
<td>SP → GEC → SC</td>
<td>-.327*</td>
<td>.026</td>
<td>.103</td>
<td>-.456</td>
<td>-.102</td>
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<tr>
<td>HS → GEC → SC</td>
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<td>.172</td>
<td>.083</td>
<td>-.029</td>
<td>.238</td>
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<tr>
<td>SP → GEC → SC → HEI</td>
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<td>.287</td>
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<tr>
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<tr>
<td>GEC → HEI</td>
<td>.124</td>
<td>.447</td>
<td>.106</td>
<td>-.084</td>
<td>.259</td>
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</tbody>
</table>

Note: HS= Habitual sleep. SP= Perception of sleep adequacy. GEC = Executive dysfunction. PFS = Hedonic hunger. SC = Self-control. HEI= Healthy eating index. ASA 24= Caloric intake. Ad libitum = buffet caloric intake. S= Sleep condition
Note ***P < .001, **p < .01, *p < .05
### Table 5
*Standardized Total Effects Using Path Analysis*

<table>
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<tr>
<th>Variables</th>
<th>β</th>
<th>p</th>
<th>Bootstrap SE</th>
<th>BBLL</th>
<th>BBUL</th>
</tr>
</thead>
<tbody>
<tr>
<td>SP</td>
<td>.420***</td>
<td>.001</td>
<td>.125</td>
<td>.187</td>
<td>.633</td>
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<tr>
<td>HS</td>
<td>-.133</td>
<td>.164</td>
<td>.104</td>
<td>-.298</td>
<td>.052</td>
</tr>
<tr>
<td>SP</td>
<td>-.054</td>
<td>.173</td>
<td>.053</td>
<td>-.153</td>
<td>.026</td>
</tr>
<tr>
<td>GEC</td>
<td>-.129</td>
<td>.226</td>
<td>.109</td>
<td>-.295</td>
<td>.066</td>
</tr>
<tr>
<td>HS</td>
<td>.017</td>
<td>.146</td>
<td>.023</td>
<td>-.012</td>
<td>.065</td>
</tr>
<tr>
<td>SP</td>
<td>-.327*</td>
<td>.026</td>
<td>.103</td>
<td>-.498</td>
<td>-.149</td>
</tr>
<tr>
<td>HS</td>
<td>-.017</td>
<td>.172</td>
<td>.083</td>
<td>-.029</td>
<td>.238</td>
</tr>
<tr>
<td>GEC</td>
<td>-.778***</td>
<td>.001</td>
<td>.052</td>
<td>-.831</td>
<td>-.644</td>
</tr>
<tr>
<td>SP</td>
<td>.052</td>
<td>.287</td>
<td>.053</td>
<td>-.011</td>
<td>.155</td>
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<tr>
<td>HS</td>
<td>-.017</td>
<td>.185</td>
<td>.024</td>
<td>-.066</td>
<td>.007</td>
</tr>
<tr>
<td>GEC</td>
<td>.012</td>
<td>.447</td>
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<tr>
<td>PFS</td>
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<td>.218</td>
<td>.130</td>
<td>-.402</td>
<td>.031</td>
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</tbody>
</table>

*Note: HS= Habitual sleep. SP= Perception of sleep adequacy. GEC = Executive dysfunction. PFS = Hedonic hunger. SC = Self-control. HEI= Healthy eating index. ASA 24= Caloric intake. Ad libitum = buffet caloric intake. S= Sleep condition*

Note ***P < .001, **p < .01, *p < .05

### Table 6
*Moderation Analysis Summary for Executive dysfunction, Hedonic Hunger and Self-control Predicting Caloric Intake (ASA-24) Moderated by Habitual Sleep Duration.*

<table>
<thead>
<tr>
<th>Variable</th>
<th>B</th>
<th>t</th>
<th>p</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Executive Dysfunction (GEC)</td>
<td>1.19</td>
<td>1.15</td>
<td>.255</td>
<td>-.898, 3.29</td>
</tr>
<tr>
<td>Habitual Sleep</td>
<td>-.037</td>
<td>-.265</td>
<td>.757</td>
<td>-.314, .242</td>
</tr>
<tr>
<td>GEC X Habitual Sleep</td>
<td>-.168</td>
<td>-.12</td>
<td>.298</td>
<td>-.447, .110</td>
</tr>
<tr>
<td>Hedonic Hunger (PFS)</td>
<td>-.867</td>
<td>-1.19</td>
<td>.237</td>
<td>-.52, 7.87</td>
</tr>
<tr>
<td>Habitual Sleep</td>
<td>.002</td>
<td>.021</td>
<td>.983</td>
<td>-.243, .248</td>
</tr>
<tr>
<td>PFS X Habitual Sleep</td>
<td>.102</td>
<td>.862</td>
<td>.393</td>
<td>-.137, .343</td>
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<tr>
<td>Self-control (SCS)</td>
<td>-1.64</td>
<td>-1.65</td>
<td>.108</td>
<td>-3.68, .383</td>
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<tr>
<td>Habitual Sleep</td>
<td>-.027</td>
<td>-.202</td>
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<td>-.298, .244</td>
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<tr>
<td>SCS X Habitual Sleep</td>
<td>.265</td>
<td>1.96</td>
<td>.057</td>
<td>-.009, .540</td>
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</table>

Note ***P < .001, **p < .01, *p < .05
Table 7
Moderation Analysis Summary for Executive dysfunction, Hedonic Hunger and Self-control Predicting Caloric Intake (ASA-24) Moderated by Perception of Sleep Adequacy

<table>
<thead>
<tr>
<th>Variable</th>
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<th>t</th>
<th>p</th>
<th>95% CI</th>
</tr>
</thead>
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<tr>
<td>Executive Dysfunction</td>
<td>129.8</td>
<td>1.03</td>
<td>.308</td>
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<tr>
<td>Sleep Perception</td>
<td>-172.7</td>
<td>-1.4</td>
<td>.161</td>
<td>-417, 71.7</td>
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<tr>
<td>Executive Dysfunction X Sleep Perception</td>
<td>116.6</td>
<td>.518</td>
<td>.607</td>
<td>-167.9, 401.2</td>
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</tbody>
</table>

<table>
<thead>
<tr>
<th>Variable</th>
<th>B</th>
<th>t</th>
<th>p</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Self-control</td>
<td>-.804</td>
<td>-.298</td>
<td>.767</td>
<td>-.628, .467</td>
</tr>
<tr>
<td>Sleep Perception</td>
<td>.691</td>
<td>1.92</td>
<td>.063</td>
<td>-.040, 1.42</td>
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<tr>
<td>Self-control X Sleep Perception</td>
<td>-3.21</td>
<td>.325</td>
<td>.746</td>
<td>-21.1, 14.6</td>
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</table>

Conditional Effects of Self-Control and Perception of Sleep Adequacy on Caloric Intake

<table>
<thead>
<tr>
<th>Variable</th>
<th>B</th>
<th>t</th>
<th>p</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adequate Sleep</td>
<td>-.084</td>
<td>-.298</td>
<td>.767</td>
<td>-.628, .467</td>
</tr>
<tr>
<td>Inadequate Sleep</td>
<td>.530*</td>
<td>2.4</td>
<td>.031</td>
<td>.084, .975</td>
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</table>

<table>
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<th>95% CI</th>
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<tr>
<td>Hedonic Hunger</td>
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<td>.224</td>
<td>-.605, .171</td>
</tr>
<tr>
<td>Sleep Perception</td>
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<td>1.12</td>
<td>.308</td>
<td>-.313, .968</td>
</tr>
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<td>Hedonic Hunger X Sleep Perception</td>
<td>.101</td>
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<td>.727</td>
<td>-.484, .688</td>
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Note: ***P < .001, **p < .01, *p < .05
### Table 8

**Moderated Mediation Analysis Summary for Executive dysfunction, Hedonic Hunger and Self-control Predicting Caloric Intake ad libitum Moderated by Sleep condition**

<table>
<thead>
<tr>
<th>Variable</th>
<th>Self-control β</th>
<th>p</th>
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<th>Caloric Intake ad libitum β</th>
<th>p</th>
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<tr>
<td>GEC</td>
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<td>.000</td>
<td>-1.09, -.384</td>
<td>-1.64</td>
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<td>-16.7, 13.5</td>
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<tr>
<td>S</td>
<td>-.047</td>
<td>.990</td>
<td>-7.95, 7.86</td>
<td>83.1</td>
<td>.492</td>
<td>-161.6, 328</td>
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<tr>
<td>SCS</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>1.71</td>
<td>.801</td>
<td>-12.9, 16.3</td>
</tr>
<tr>
<td>GEC X S</td>
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<td>-314, .635</td>
<td>1.77</td>
<td>.801</td>
<td>-4.06, 38.0</td>
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<tr>
<td>SCS X S</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>30.5**</td>
<td>.012</td>
<td>7.10, 53.9</td>
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<tr>
<td>⊥ SP</td>
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<td>.693</td>
<td>-10.0, 6.78</td>
<td>-159.2</td>
<td>.224</td>
<td>-421.5, 102.9</td>
</tr>
</tbody>
</table>

*Note:* HS = Habitual sleep, SP = Perception of sleep adequacy, GEC = Executive dysfunction, PFS = Hedonic hunger, SC = Self-control, HEI = Healthy eating index, ASA 24 = Caloric intake. Ad libitum = buffet caloric intake. S = Sleep condition

***P < .001, **p < .01, *p < .05

⊥ covariate

### Table 9

**Conditional Direct Effect (GEC → Calories ad libitum)**

<table>
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<th>Sleep Condition</th>
<th>β</th>
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</thead>
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<tr>
<td>0 hr</td>
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<td>.825</td>
<td>-16.7, 13.5</td>
</tr>
<tr>
<td>9 hrs</td>
<td>15.32*</td>
<td>.044</td>
<td>.440, 30.2</td>
</tr>
</tbody>
</table>

*Note***P < .001, **p < .01, *p < .05

### Table 10

**Conditional Indirect Effect (GEC → SCS → Calories ad libitum)**

<table>
<thead>
<tr>
<th>Sleep Condition</th>
<th>β</th>
<th>BootSE</th>
<th>BootLLCI</th>
<th>BootULCI</th>
</tr>
</thead>
<tbody>
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<td>0 hr</td>
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<td>4.93</td>
<td>-11.7</td>
<td>8.00</td>
</tr>
<tr>
<td>9 hrs</td>
<td>-18.4***</td>
<td>8.65</td>
<td>-39.5</td>
<td>-7.02</td>
</tr>
</tbody>
</table>

Index of Moderated Mediation

Index = -17.1; SE (Boot) = 9.7; BootLLCI = -40.4; BootULCI = -2.27

*Note***P < .001, **p < .01, *p < .05
Figure 1
Plausible Mechanism of Sleep Behavior Influencing Healthy Eating Index

Sleep Behavior

Homeostatic Eating
1) Reduction in natural levels of serotonin (resultant need of carbohydrates)
2) Impaired glucose utilization

Hedonic Eating
1) Increased susceptibility to obesogenic food environment
2) Increased awake time resulting in increased snacking

Dysfunction of Executive Functioning
1) Decreased Inhibitory control
2) Increased pleasurable reward sensitivity
3) Impaired planning

Self-control
1) Self-regulation resources depletion (resultant need to replenish lost resources)
2) Impaired self-regulatory control results in impaired effortful control and impaired self-monitoring and decision making

Dietary Behavior
Dietary Quality
Figure 2
Proposed Conceptual Model of Habitual Sleep, Perception of sleep adequacy, Executive Functioning, Self-control, Hedonic Hunger, Caloric Intake (ASA-24)
Figure 3
Proposed Conceptual and Statistical Model of Global Executive Composite (GEC) Executive Functioning, Self-control, and Caloric Intake ad libitum for 0 vs 9 hours of Sleep.
### Baseline
- A brief description of the study was provided to the interested participants who contacted the laboratory via telephone or text message. Participants were recruited using fliers in public locations (e.g., recreation facilities, schools, pediatrician’s office). Exclusion criteria included use of weight loss medication, history of bariatric surgery, use of medication that affect salivation (e.g., antihistamines, antidepressants), history of an eating disorder, history of sleep disorders, left handedness, psychiatric conditions (e.g., epilepsy, traumatic brain injury, schizophrenia, bipolar disorder) food allergies and standard fMRI contraindications (e.g., metallic implants, pregnancy, claustrophobia). Once, participants met inclusion criteria, parental consent was taken.
- A Qualtrics survey was sent to gather measures on demographics, pubertal development, hedonic eating (Power of Food Scale) and self-reported sleep duration (Adolescent Sleep Habit Survey).

### Time 1 In Person Meeting
- **Measures of weight and height were taken to calculate BMI percentile and eligibility criteria was reassessed. Participants provided written assent and parents signed informed consent.**
- 1st phase of chatroom task by Guyer and colleagues (2008) was conducted. In this, participants were asked to rate a series of images of peers as high vs low value and they categorized 60 images of boys and girls as either 1) high-value peers with whom they are interested in chatting, or 2) low-value peers with whom they are not interested in chatting.
- Each participant was photographed and they were informed that the 60 purported peers will be shown their photographs.
- Each participant was randomly assigned to either extended sleep (9 hours) or no sleep.
- Waist accelerators were provided as a validity check for sleep duration.
- **Measures of executive functioning (BRIEF- SR) and self-control (Self-control Scale) were taken.**

### Time 2 In Person Meeting (One week after Time 1)
- Each participant completed the MRI screening form.
- Measures of height and weight were taken.
- **Hunger rating on a scale of 0-10 was taken.**
- A E prime MRI task was conducted: participants were shown photographs of the previously rated peers and each participant provided a rating of how interested the peer was in chatting with the participant. In second task, each participant was reminded of their ratings of high vs low value for each peer. Afterwards, they received a purported feedback from the high and low value peers (words interested or not interested appeared below each photograph; standardized across participants). Later, participants rated how much they expect or did not expect the peer’s ratings of their desirability (from 0-100).
- Trails A and B were administered
- Participant was directed to the buffet room and each participant was offered a large array of breakfast items (similar across participants). The items were weighed to the nearest grams before and after consumption.
- **After 20-minutes, participant was requested to answer the ASA-24 and record all the food in the last 24 hours minus the breakfast buffet.**
- Each participant and parent was debriefed about the nature of the study and the computerized induced social stress on the chatroom task and each received $75 for completing the study.
Figure 5
Path Analysis of Plausible Mechanism of Relationship Between Sleep Behavior and Healthy Eating Index
Figure 6
*Conditional Influence of Perception of Sleep Adequacy and Self-control on Caloric Intake*
Figure 7
*Moderated Mediation Direct Effect of Executive Dysfunction and Indirect Effect of Self-control on Caloric Intake ad libitum under Sleep Manipulation*

![Graph showing the effects of sleep manipulation on caloric intake.]

<table>
<thead>
<tr>
<th></th>
<th>Restriction</th>
<th>Extension</th>
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</thead>
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<tr>
<td>Direct effect</td>
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<td>15.32</td>
</tr>
<tr>
<td>Indirect effect</td>
<td>-1.29</td>
<td>-18.40</td>
</tr>
</tbody>
</table>
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.
Appendix B:

Adolescent Sleep Habit Survey

There are no right or wrong answers. Be careful to choose the one answer that best describes the way your sleep has been in the last two school weeks (unless otherwise instructed).

The next set of questions has to do with your usual schedule on days when you have school.

22. What time do you usually go to bed on school days?
List ONE time, not a range.

__________________________
A.M.  
P.M.

23. There are many reasons for doing things at one time or another. What is the main reason you usually go to bed at this time on school days? (mark one)
○ My parents have set my bedtime
○ I feel sleepy
○ I finish my homework
○ My TV shows are over
○ My brother(s) or sister(s) go to bed
○ I finish socializing
○ I get home from my job
○ Other:

24. What time do you usually wake up on school days?

__________________________
A.M.  
P.M.

25. What is the main reason you usually wake up at this time on school days? (choose one)
○ Noises or my pet wakes me up
○ My alarm clock wakes me up
○ My parents or other family members wake me up
○ I need to go to the bathroom
○ I don’t know, I just wake up
○ Other:

26. What time do you usually leave home on school days?

__________________________
A.M.  
P.M.

27. How do you usually get to school?
○ Walk
○ Take the bus
○ Get a ride with friend(s)
○ Drive my car
○ Get a ride with parent

28. Figure out how long you usually sleep on a normal school night and fill it in here. [Do not include time you spend awake in bed. Remember to mark hours and minutes, even if minutes are zero.]

29. On school days, after you go to bed at night, about how long does it usually take you to fall asleep?

__________________________ minutes

The next set of questions has to do with your usual schedule on days when you do not have school, such as on the weekend.

30. What time do you usually go to bed on weekends?

__________________________
A.M.  
P.M.

31. There are many reasons for doing things at one time or another. What is the main reason you usually go to bed at this time on weekends? (choose one)
○ My parents have set my bedtime
○ My brother(s) or sister(s) go to bed then
○ I feel sleepy
○ I finish socializing
○ I finish my homework
○ My TV shows are over
○ I get home from my job
○ Other:

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DO NOT WRITE IN THIS AREA
32. What time do you usually wake up on weekends?
   ○ A.M.  ○ P.M.

33. What is the main reason you usually wake up at this time on weekends? (choose one)
   ○ Noises or my pet wakes me up
   ○ My alarm clock wakes me up
   ○ My parents wake me up
   ○ I need to go to the bathroom
   ○ I don’t know, I just wake up
   ○ Other: _______________________

34. Figure out how long you usually sleep on a night when you do not have school the next day (such as a weekend night) and fill it in here. [Do not include time you spend awake in bed. Remember to mark hours and minutes, even if minutes are zero.]
   _________ hours _________ minutes

35. On weekends, after you go to bed at night, about how long does it usually take you to fall asleep?
   _________ minutes

36. Some people wake up during the night. Others never do. How many times do you usually wake up at night?
   ○ Never
   ○ Once
   ○ 2 or 3 times
   ○ More than 3 times
   ○ I have no idea

37. People sometimes feel sleepy during the daytime. During your daytime activities, how much of a problem do you have with sleepiness (feeling sleepy, struggling to stay awake)?
   ○ No problem at all
   ○ A little problem
   ○ More than a little problem
   ○ A big problem
   ○ A very big problem

38. Some people take naps in the daytime every day, others never do. When do you nap? (mark all that apply:)
   ○ I never nap.
   ○ I sometimes nap on school days.
   ○ I sometimes nap on weekends.
   ○ I never nap unless I am sick.

39. Can you figure out how much sleep you need? Fill out below how much sleep you think you would need each night to feel your best every day. [Remember to mark hours and minutes, even if minutes are zero.]
   _________ hours _________ minutes

40. In general, do you feel you usually get...
   ○ too much sleep?
   ○ enough sleep?
   ○ too little sleep?

41. Do you consider yourself to be...
   ○ a good sleeper?
   ○ a poor sleeper?

42. How often do you think that you get enough sleep?
   ○ Always
   ○ Usually
   ○ Sometimes
   ○ Rarely
   ○ Never
Questions 43 to 46 are about things that have happened in the last two weeks.

43. During the last two weeks, have you struggled to stay awake (fought sleep) or fallen asleep in the following situations? (Mark one answer for every item.)

Both struggled to stay awake and fallen asleep
Fallen asleep
Struggled to stay awake

- in a face-to-face conversation with another person? ........................................... O O O O O
- traveling in a bus, train, plane or car? ................................................................. O O O O O
- attending a performance (movie, concert, play)? .................................................. O O O O O
- watching television or listening to the radio or stereo? ........................................... O O O O O
- reading, studying or doing homework? ................................................................. O O O O O
- during a test? ........................................................................................................ O O O O O
- in a class at school? ............................................................................................... O O O O O
- while doing work on a computer or typewriter? ..................................................... O O O O O
- playing video games? .......................................................................................... O O O O O
- driving a car? ........................................................................................................ O O O O O

Do you drive?  O Yes  O No

44. During the last two weeks, how often did you...
(Mark one answer for every item.)

Every day
Several times every day
Once or twice a day
Never

a. drink soda with caffeine [like Coke, Pepsi; not like root beer, orange soda or Sprite]? ........................................................................ O O O O O
b. drink coffee or tea with caffeine? ........................................................................ O O O O O
c. use tobacco? [cigarettes, cigar, chewing tobacco, etc.]? ........................................ O O O O O
d. drink alcohol [beer, wine, liquor]? ........................................................................ O O O O O
e. use drugs [like marijuana, cocaine]? ................................................................... O O O O O

If yes, please specify type:

45. In the last two weeks, how often have you...
(Mark one answer for every item.)

Never
Once
Twice
Several times
Everyday/night

a. felt satisfied with your sleep? ................................................................. O O O O O
b. arrived late to class because you overslept? .................................................. O O O O O
c. fallen asleep in a morning class? ................................................................. O O O O O
d. fallen asleep in an afternoon class? ............................................................... O O O O O
e. awakened too early in the morning and couldn't get back to sleep? ................. O O O O O
f. stayed up until at least 3 a.m.? ........................................................................ O O O O O
g. stayed up all night? .......................................................................................... O O O O O
h. slept in past noon? ........................................................................................... O O O O O
i. felt tired, dragged out, or sleepy during the day? ........................................... O O O O O
j. needed more than one reminder to get up in the morning? ............................. O O O O O
k. had an extremely hard time falling asleep? .................................................... O O O O O
l. had nightmares or bad dreams during the night? ............................................. O O O O O
m. gone to bed because you just could not stay awake any longer? ................. O O O O O
n. done dangerous things without thinking? ....................................................... O O O O O
o. had a good night's sleep? ................................................................................ O O O O O

46. During the last two weeks, how often were you bothered or trouble by the following?

Much
Somewhat
Not at all

a. Feeling too tired to do things ............................................................................. O O O
b. Having trouble going to sleep or staying asleep ............................................. O O O

c. Feeling unhappy, sad, or depressed .................................................................. O O O

d. Feeling hopeless about the future ..................................................................... O O O

e. Feeling nervous or tense .................................................................................. O O O

f. Worrying too much about things ...................................................................... O O O
Questions 47 - 56 have to do with how you might organize the timing of various activities if you were free to plan your day according to when you feel your best. Please answer the questions based on your body’s “feeling best” times.

47. Imagine: School is cancelled! You can get up whenever you want to. When would you get out of bed? Between:
   - 5:00 and 6:30 a.m.
   - 6:30 and 7:45 a.m.
   - 7:45 and 9:45 a.m.
   - 9:45 and 11:00 a.m.
   - 11:00 a.m. and noon

48. Is it easy for you to get up in the morning?
   - No way!
   - Sort of.
   - Pretty easy.
   - It’s a cinch!

49. Gym class is set for 7:00 in the morning. How do you think you’ll do?
   - My best!
   - Okay.
   - Worse than usual.
   - Awful!

50. The bad news: You have to take a two-hour test. The good news: You can take it when you think you’ll do your best. What time is that?
   - 8:00 to 10:00 a.m.
   - 11:00 a.m. to 1:00 p.m.
   - 2:00 p.m. to 5:00 p.m.
   - 7:00 p.m. to 9:00 p.m.

51. When do you have the most energy to do your favorite things?
   - Morning! I am tired in the evening.
   - Morning more than evening.
   - Evening more than morning.
   - Evening! I am tired in the morning.

52. Your parents have decided to let you set your own bed time. What time would you pick? Between:
   - 8:00 and 9:00 p.m.
   - 9:00 and 10:15 p.m.
   - 10:15 p.m. and 12:30 a.m.
   - 12:30 and 1:45 a.m.
   - 1:45 and 3:00 a.m.

53. How alert are you in the first half hour you’re up?
   - Out of it.
   - A little dazed.
   - Okay.
   - Ready to take on the world.

54. When does your body start to tell you it’s time for bed (even if you ignore it)? Between:
   - 8:00 and 9:00 p.m.
   - 9:00 and 10:15 p.m.
   - 10:15 p.m. and 12:30 a.m.
   - 12:30 and 1:45 a.m.
   - 1:45 and 3:00 a.m.

55. Say you had to get up at 6:00 a.m. every morning. What would it be like?
   - Awful!
   - Not so great.
   - Okay (if I have to).
   - Fine, no problem!

56. When you wake up in the morning how long does it take for you to be totally "with it"?
   - 0 to 10 minutes
   - 11 to 20 minutes
   - 21 to 40 minutes
   - More than 40 minutes

57. Would you say that your growth in height:
   - Has not begun to spurt ("spurt" means faster growth than usual)
   - Has barely started
   - Is definitely underway
   - Seems complete
   - I don’t know

58. Would you say that your other signs of physical maturation:
   - Have not yet started to show
   - Have barely started to show
   - Are definitely underway
   - Seem complete
   - I don’t know
59. During the last week, did you work at a job for pay? (If no, skip to number 60.)

- Yes    - No

What kind of job?

How many days did you work at the following times?
- in the morning before school: 0 1 2 3 4 5
- in the afternoon after school: 0 1 2 3 4 5
- in the evening on days that you have school: 0 1 2 3 4 5
- on the weekend: 0 1 2

How many hours did you work at your paying job this week?
- during the school week: ________ hours
- during the weekend: ________ hours

During the last two weeks, have you struggled to stay awake (fought sleep) or fallen asleep at your job?
- no    - struggled to stay awake
- fallen asleep - both struggled to stay awake and fallen asleep

If you did not have your job, would you go to bed:
- earlier than you do.    - the same as you do.    - later than you do.

If you did not have your job, would you wake up:
- earlier than you do.    - the same as you do.    - later than you do.

60. During the last week, did you engage in organized sports or a regularly scheduled physical activity? (If no, skip to number 61.)

- Yes    - No

What kind of sport?

How many days did you practice at the following times?
- in the morning before school: 0 1 2 3 4 5
- in the afternoon after school: 0 1 2 3 4 5
- in the evening on days that you have school: 0 1 2 3 4 5
- on the weekend: 0 1 2

How many hours did you practice this week?
- during the school week: ________ hours
- during the weekend: ________ hours

During the last two weeks, have you struggled to stay awake (fought sleep) or fallen asleep during practice?
- no    - struggled to stay awake
- fallen asleep - both struggled to stay awake and fallen asleep

If you did not have your sports activity, would you go to bed:
- earlier than you do.    - the same as you do.    - later than you do.

If you did not have your sports activity, would you wake up:
- earlier than you do.    - the same as you do.    - later than you do.

61. During the last week, did you participate in organized extracurricular activities? (For example, committees, clubs, volunteer work, musical groups, church groups, etc.) (If no, skip to number 62.)

- Yes    - No

What kind of activity?

How many days did you participate at the following times?
- in the morning before school: 0 1 2 3 4 5
- in the afternoon after school: 0 1 2 3 4 5
- in the evening on days that you have school: 0 1 2 3 4 5
- on the weekend: 0 1

How many hours did you participate this week?
- during the school week: ________ hours
- during the weekend: ________ hours

During the last two weeks, have you struggled to stay awake (fought sleep) or fallen asleep during this participation?
- no    - struggled to stay awake
- fallen asleep - both struggled to stay awake and fallen asleep

If you did not have your organized activity, would you go to bed:
- earlier than you do.    - the same as you do.    - later than you do.

If you did not have your organized activity, would you wake up:
- earlier than you do.    - the same as you do.    - later than you do.

62. During the last week, did you study/do homework?

- Yes    - No (If no, skip to number 63.)

How many days did you study at the following times?
- in the morning before school: 0 1 2 3 4 5
- in the afternoon after school: 0 1 2 3 4 5
- in the evening on days that you have school: 0 1 2 3 4 5
- on the weekend: 0 1 2

How many hours did you study this week?
- during the school week: ________ hours
- during the weekend: ________ hours

During the last two weeks, have you struggled to stay awake (fought sleep) or fallen asleep during studying?
- no    - struggled to stay awake
- fallen asleep - both struggled to stay awake and fallen asleep

If you did not have your homework, would you go to bed:
- earlier than you do.    - the same as you do.    - later than you do.

If you did not have your homework, would you wake up:
- earlier than you do.    - the same as you do.    - later than you do.