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**The Relationships Among Facets of Sleep, Eating Behavior, Energy Intake, and
Adiposity in Youth**

By:

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Uniformed Services University of the Health Sciences

**Dissertation submitted to the Faculty of the
Medical and Clinical Psychology Graduate Program
Uniformed Services University of the Health Sciences
In partial fulfillment of the requirements for the degree of
Doctor of Philosophy**



UNIFORMED SERVICES UNIVERSITY OF THE HEALTH SCIENCES

SCHOOL OF MEDICINE GRADUATE PROGRAMS

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June 9, 2021

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SUBJECT: Appointment to the Final Doctoral Examination Committee for Sarah LeMay-Russell

In accordance with the School of Medicine Graduate Program Guidelines, you are formally appointed to the Final Examination Committee of Sarah LeMay-Russell, doctoral student in the Department of Medical and Clinical Psychology.

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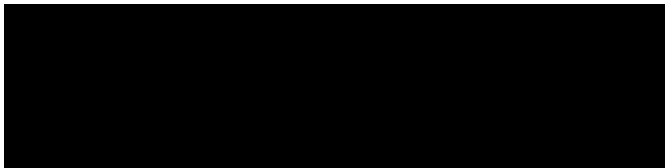
In addition, the Committee will conduct an Oral Examination of the student's scientific knowledge, as well as the knowledge and understanding of the research project and Dissertation. This examination constitutes the Private Defense.

The Private Defense for Sarah LeMay-Russell will take place on Tuesday, June 8, 2021 at 9:00 AM via Zoom meeting.

Dr. Tracy Sbrocco is appointed Chair of the Examination Committee. Dr. Sbrocco will oversee the examination. Passage of the Private Defense will be by majority vote. If corrections to the Dissertation are needed, the Chair may oversee this process and sign-off after the corrections are made.

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Saibal Dey, Ph.D.
Associate Dean for Graduate Education



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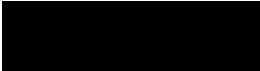
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DECISION OF EXAMINATION COMMITTEE MEMBERS:

PASS

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APPROVAL OF THE DOCTORAL DISSERTATION IN THE DEPARTMENT OF MEDICAL AND CLINICAL PSYCHOLOGY

Title of Dissertation: "The Relationships Among Facets of Sleep, Eating Behavior, Energy Intake, and Adiposity in Youth"

Name of Candidate: Sarah LeMay-Russell
Doctor of Philosophy Degree
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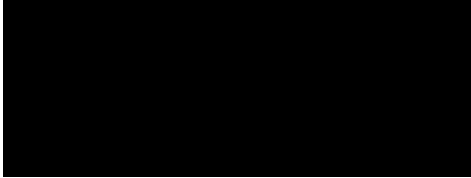
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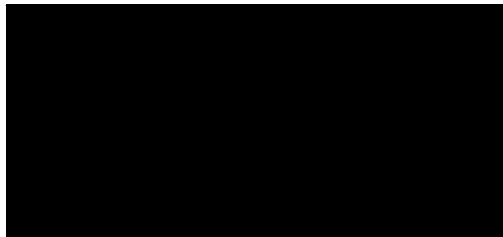
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04/1/2022

ABSTRACT

Title of Dissertation: The Relationships Among Facets of Sleep, Eating Behavior, Energy Intake, and Adiposity in Youth

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Thesis directed by: Marian Tanofsky-Kraff, Ph.D., Professor, Department of Medical and Clinical Psychology and Natasha A. Schvey, Ph.D., Assistant Professor, Department of Medical and Clinical Psychology

Some studies suggest that there are characteristics of sleep beyond insufficient duration that are associated with obesity risk in youth. Recent data suggest that, among pre-adolescents, variables beyond duration alone, such as sleep quality, timing, and variability are associated with weight status. Facets of sleep variability generally fall into two broad categories: those that include measures of duration, and those that include behavioral estimations of circadian rhythm shift. The latter may be particularly salient to consider in its relation to dietary intake, disinhibited eating, and obesity risk in a broad pediatric age range, as sleep needs and timing change significantly after puberty onset. Sleep timing (i.e., the time one falls asleep and/or wakes up) may also play an important role. Preliminary data indicate that greater sleep variability and later bed and wake timing is linked with greater energy intake, specifically from fat, carbohydrates, and added sugar; higher body mass index; and greater fat mass. Further, sleep variability has been associated with disinhibited eating and cravings, which in some cases have been linked to greater overall energy consumption, carbohydrates, snacks, and dessert-type food intake, and

high weight in youth. However, there is a relative paucity of objectively measured data examining the links between facets of sleep variability and timing and obesity risk in youth. Additionally, there is a lack of consensus on which facets of sleep warrant investigative effort. Given that available data are primarily cross-sectional, more longitudinal data are needed to better understand which facets of sleep may be linked with obesogenic behaviors over time. Therefore, the following three studies utilized actigraphy-measured sleep among youth to examine: 1) the associations of weekend catch-up sleep with eating in the absence of hunger and adiposity; 2) the temporal and cumulative associations of facets of sleep, food cravings, and loss of control-eating severity; and 3) the longitudinal associations of facets of sleep and adiposity at 1-year follow up. Study 1 results showed that, among 123 participants (52.0% female; 12.7 ± 2.6 y; 29.3% Non-Hispanic Black or African American; 30.9% with overweight/obesity), there was no significant association between average weekly sleep duration and eating in the absence of hunger. There was also no significant association among average weekly sleep duration or eating in the absence of hunger and fat mass. However, average weekday sleep was negatively associated, and average weekend sleep was positively associated, with eating in the absence of hunger ($p < 0.02$). Further, weekend “catch-up” sleep (the difference between weekend and weekday sleep) was positively associated with eating in the absence of hunger ($p < 0.01$). For study 2, among 48 youths (68.8% female; 12.8 ± 2.7 y; 14.6% Non-Hispanic Black or African American; 33.3% with overweight/obesity), neither weekly nor nightly facets of sleep were significantly associated with food cravings ($p = .08-.93$). However, youths with shorter weekly sleep duration (est. $\beta = -0.31$, $p = .004$), earlier weekly midpoints (est. $\beta = -0.47$, $p = .01$) and later weekly waketimes (est. $\beta = 0.49$, $p = .01$) reported greater loss of control-eating severity. Finally, for study 3, which included 137 youths (54.0% female; 12.5 ± 2.6 y; 28.4% Non-Hispanic Black

or African American; 28.5% with overweight/obesity at baseline), findings suggested that both earlier waketime ($p=.03$) and earlier sleep midpoint ($p=.02$) were associated with greater 1-year fat mass. No other facet of sleep was significantly associated with 1-year fat mass ($ps>.09$).

Taken together, results from these studies provide a more nuanced understanding of the links between facets of sleep and obesity risk in youth, with the broad goal of informing prevention and intervention strategies.

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CHAPTER 1: Introduction

Preliminary data indicate that nuanced characteristics of sleep may be associated with risk for obesity among youth. Therefore, this dissertation will investigate these associations through three empirical studies. The dissertation will begin with a broad overview of the literature on pediatric obesity and then describe the observed links between specific components of sleep and potentially obesogenic behaviors, such as disinhibited eating and excess energy intake. Importantly, sleep is complex and involves many characteristics beyond duration (hours per night); for instance, sleep variability may be particularly salient among youth and includes several nuanced facets. Thus, an in-depth description of the multiple facets of sleep variability and timing will be provided. A description of relevant disinhibited eating behaviors, as well as an examination of how specific facets of sleep are linked to these behaviors and energy intake broadly will also be discussed. Additionally, self-control depletion theory will provide a theoretical framework for understanding the link between facets of sleep, disinhibited eating behaviors, increased energy intake, and subsequent weight gain in youth. The review of the literature and the theoretical basis of self-control depletion theory will lay the framework for the three empirical studies, which, collectively, build on previous research and aim to further elucidate the link between sleep and obesity risk in youth.

OVERWEIGHT AND OBESITY

Adult Overweight/Obesity

Rates of overweight and obesity in adulthood continue to rise in the United States, with recent estimates suggesting prevalence has nearly tripled in the last forty years (1). Over two thirds (68%) of adults in the United States have overweight, which is defined as having a body

mass index (BMI; kg/m^2) between 25.0-29.9 (1). Further, over forty percent of adults have obesity [$\text{BMI} \geq 30$; (2, 3)]. Given the increasing prevalence of overweight and obesity, significant attention has been given to understanding associated health outcomes (4).

Adult overweight and obesity are associated with a number of physiological comorbidities including stroke, hypertensive heart disease, diabetes mellitus, and several types of cancer (4). Further, high body weight has been identified as a significant risk factor for mortality, with approximately 280,000 deaths annually that are attributable to obesity-related diseases (4, 5). Of particular current concern, preliminary data suggest that individuals with obesity are at increased risk of severe complications associated with COVID-19 (6-8), although the mechanisms linking this relationship warrant study. There are also associated psychological sequelae including depression, stress, low self-esteem, poor quality of life, and eating disorders (9). Consequently, individuals with obesity have medical costs that are approximately thirty percent greater than those of their lower-weight peers (10). Given rising rates of adult obesity and related physical and mental health comorbidities, in conjunction with limited effective long-term treatments (1, 11), it is important to focus on prevention among children and adolescents, as youth with high weight are five times as likely to have obesity in adulthood (12-14).

Pediatric Overweight/Obesity

Prevalence of Pediatric Obesity

Over one-third of adolescents in the United States have overweight and nearly twenty percent have obesity (2, 15). Among youth (2-18 years), overweight is defined as having an age-, and sex-specific BMI at or above the 85th percentile but less than the 95th percentile, and obesity is defined as having a BMI at or above the 95th percentile (15). Additionally, an “extreme” or “severe” obesity category was included in 2009 to better quantify individuals on the highest end

of the weight strata, and is defined as having a BMI $\geq$ 120% of the 95th percentile for age and sex (16). Recent estimates suggested that nearly six percent of youth have extreme obesity and the prevalence of this category has increased the most in recent decades (17). Such statistics are concerning because youth with overweight or obesity are at increased risk for numerous physiological and psychosocial consequences (18-21).

Correlates and Outcomes of Pediatric Obesity

High weight in childhood is associated with several physiological comorbidities including asthma, gastroesophageal reflux, nonalcoholic fatty liver disease, type 2 diabetes, and orthopedic problems (22-26). Pediatric obesity is also associated with a cluster of physiological abnormalities known as metabolic syndrome [MetS; (20, 27, 28)]. MetS variables include abdominal obesity, elevated blood pressure, high triglyceride concentrations, low high-density lipoprotein cholesterol (HDL-C), and elevated glucose, and are known to increase risk for heart disease and type 2 diabetes (29). Pediatric obesity has also been shown to be predictive of increased risk for lifetime cardiovascular disease, type 2 diabetes, and adult obesity (30, 31).

High weight in youth is also linked with weight-based victimization, poorer self-esteem, and depression (32), and is predictive of depression and poorer self-esteem in adulthood (21). Given the long-term consequences of childhood obesity and limited effectiveness of current treatment options, it is important to understand the etiology of high weight in childhood more fully, and to elucidate early, modifiable risk factors for excess weight gain that could improve the effectiveness of obesity prevention and treatment in children and adolescents.

Etiology of Pediatric Obesity

The etiology of pediatric obesity is complex and multi-factorial, involving an interaction of genetic, environmental, and behavioral components (33). While understanding each of these

facets is important to the prevention and treatment of obesity, much attention has been drawn to obesogenic behaviors, as these are thought to be the most immediately modifiable (34). Indeed, there are substantial data demonstrating that excess energy intake, specifically from sugar-sweetened beverages and energy-dense foods, and physical inactivity lead to high weight in children and adolescents (35). However, the limited long-term efficacy of interventions targeting diet and exercise in youth (36), has illuminated the complex nature of childhood obesity which is not entirely explained by behavioral factors associated with energy imbalance. Therefore, elucidation of additional early, modifiable risk factors for excess weight gain is necessary to improve the effectiveness of obesity prevention and treatment in children and adolescents. Two such factors that have been associated with excess weight and adiposity and increased energy intake, are disinhibited eating behaviors [i.e. eating in response to cues other than hunger (37, 38)], and inadequate sleep (39-41).

DISINHIBITED EATING

Disinhibited eating is characterized by eating involving a lack of healthy restraint (37) and energy consumption for reasons other than physiological hunger (38). These behaviors include, but are not limited to, emotional eating, binge-eating, eating in the absence of hunger, and loss of control eating (37). Disinhibited eating behaviors are associated with greater energy consumption at laboratory test meals as well as greater consumption of carbohydrates, snacks, and dessert-type foods (42, 43), each of which can promote high weight (44-46). Additionally, specific disinhibited eating behaviors, such as loss of control eating and binge-eating, have been shown to be associated with greater adiposity and predictive of the development of overweight and obesity in youth (47-51). Given that disinhibited eating behaviors are associated with greater obesity risk in youth, it is important to more fully understand the mechanisms that may be driving these

behaviors. Two disinhibited eating behaviors that are highly prevalent among youth across the weight strata and potentially linked with sleep are eating in the absence of hunger and loss of control eating (52-55). Therefore, this dissertation examines these two specific behaviors in relation to sleep and adiposity in youth.

Eating in the Absence of Hunger (EAH)

EAH is characterized by eating despite lack of physical hunger, or past satiation in response to negative affect, fatigue, or when cued by external circumstances such as the smell of food or seeing others eat (56, 57). EAH is highly prevalent among children and adolescents, with some studies estimating that as many as two-thirds of youth report this behavior to some degree (37, 57). Cross-sectionally, EAH (measured either objectively with laboratory test meals or by self-report), is associated with greater consumption of energy dense snack-foods, higher fat mass and greater BMIz (56-58). However, longitudinal data have not shown EAH to predict increases in BMI, BMIz, or fat mass over time in youth (56, 58, 59). Regardless, the relationship between EAH and high weight track together such that youth with overweight or obesity have greater EAH than those without high weight, which persists over time (60). While the etiology of EAH remains unclear, it is possible that EAH may be related to decreased cognitive control and increased reward sensitivity, each of which could be impacted by poor sleep (55, 61, 62). However, there are relatively few studies examining the association of sleep and eating in the absence of hunger in youth, and more data are needed to better understand this relationship.

Binge Eating and Loss of Control (LOC)-Eating

Binge eating is defined as the consumption of an objectively large amount of food accompanied by the subjective feeling that one cannot stop eating and is the hallmark diagnostic criterion of binge-eating disorder [BED; (63)]. LOC-eating is a related but distinct construct,

which is defined as a sense that one cannot control how much, or what one eats, irrespective of the amount of food consumed (49). Full-syndrome BED is defined by recurrent distressing binge eating episodes in the absence of compensatory behaviors. However, full-syndrome BED is relatively uncommon in childhood (63). This may be due to the difficulty determining what constitutes an objectively large amount of food across a broad age-range of developing youth with varying caloric needs, or the lack of access to large amounts of food among youth given their limited autonomy (53). Whereas full-syndrome BED is infrequently reported, youth may still experience a subjective sense of loss of control overeating, even in the absence of a large amount of food. Additionally, some data support that the experience of LOC-eating may be more salient marker for eating disturbances and high weight than the amount consumed among children and adolescents (64). Therefore, LOC-eating is considered preferable to full-syndrome BED when examining disinhibiting eating in youth (65).

Youth across weight strata report LOC-eating, with 16-23% of community-based adolescents and 33% of youth with overweight or obesity engaging in the behavior (53, 66-68). LOC-eating has been associated with increased energy intake, specifically from snack and dessert-type foods (43), and is prospectively predictive of excess weight and adiposity in youth (49, 50). Further, preliminary data suggest that LOC-eating may be impacted by decreased sleep duration or variable sleep patterns (54, 55, 69, 70), but data in youth are limited. More objectively measured data are needed to better understand this relationship.

Food Craving

An appetitive drive that may be related to LOC-eating is food craving (71, 72). Food craving is described as an intense desire to eat specific food(s), irrespective of actual energy intake (73). Regardless, experiencing an intense desire for food may increase one's risk for engaging in

LOC-eating. Preliminary data suggest a positive relationship between food cravings and LOC eating in youth (71, 72). Additionally, food cravings have been associated with greater food intake and high weight, both cross-sectionally and prospectively (73-80). However, to date, all studies linking LOC-eating and cravings in children and adolescents have been among youth with overweight or obesity. Therefore, additional data are needed from samples across the weight strata as this relationship may be important in identifying youth at risk for overweight or disinhibited eating.

Theoretical Models that Support Relationships among Disinhibited Eating, Obesity and Sleep

There are several theories hypothesizing the drivers of disinhibited eating, including interpersonal theory, dietary restraint theory, and dual process theory, among which dual process theory appears to have the clearest potential link with sleep characteristics. The interpersonal model of LOC-eating draws on adult depression literature (81), and posits that conflictual or unsupportive close relationships may result in increased negative emotions (82). Individuals may then engage in LOC-eating as a means of coping with these negative emotions (83). There are data to support the link between interpersonal social stressors, negative affect, and LOC-eating in youth and young adults (84-86). For example, in a study of healthy adolescent girls at risk for high weight, the link between a recent social stressor and LOC-eating was mediated by pre-meal state anxiety (84). This study supports the theory that negative affect, and potentially more specifically state anxiety, resulting from interpersonal stressors, may contribute to increased disinhibited eating among adolescent girls.

The dietary restraint model is another theoretical framework for understanding the onset and maintenance of disinhibited eating. This theory suggests that dieting, defined as altering what or

how much one eats in an attempt to reduce weight or change body shape, may trigger disinhibited eating (37, 38, 87). Herman and Polivy (1975) suggested that individuals who restrict their diets could be at greater risk for overeating due to increased feelings of hunger (87). Herman and Polivy (1975) additionally theorized that cognitive factors play an important role in restraint theory. For example, if an individual holds rigid cognitions regarding dieting rules, violating even a small rule could result in “all or nothing thinking” that one has failed. This type of thinking could then lead to disinhibited eating [e.g. “*I’ve already blown my diet for today, I might as well go all out*” (88)]. As a result, despite the rather contradictory notion that restricting intake might ameliorate risk for obesity, data support that self-reported dieting is associated with greater disinhibited eating and weight gain among youth (48, 89, 90). These data are further complicated by a general lack of consensus regarding how to define dieting constructs (91). Regardless, reducing dietary restraint is a cornerstone of cognitive-behavioral interventions targeting disinhibited eating and excess weight (92-95).

Another relevant theory for understanding disinhibited eating is the dual-process model. Strack and Deutsch (2004) proposed that there are two processes at play in the regulation of eating behaviors: 1) automatic processes (i.e. fast, implicit, automatic attention toward food stimuli), and 2) inhibitory control (i.e. the ability to restrain one’s behavioral response in order to maintain progress toward a goal) (96). Automatic processes include attentional biases toward a salient stimuli that occur quickly and effortlessly (97). Some studies have demonstrated that attentional bias toward highly palatable foods may be related to disinhibited eating and high weight in children and adolescents (98, 99). For instance, Shank et al. (2015) found that among youth with LOC-eating, attentional bias toward energy-dense foods was associated with higher BMIz (98). Inhibitory control, which is the other component of the dual-process model, is

defined as the ability to stop oneself or suppress behavioral responses that are not in line with current goals, such as weight loss (100). Decreased inhibitory control has been associated with high weight (101-103) and disinhibited eating (62, 104-106) in youth and young adulthood. A study among college undergraduates found that decreased inhibitory control was associated with greater eating in response to negative affective states and in response to external food cues (62). Further, a prospective study by Anzman & Birch (2009) found that girls with lower inhibitory control at age 7 had higher BMIs and excess weight gain in adolescence (104).

These data suggest that inhibitory control may play a particularly important role in the onset of disinhibited eating and high weight in youth. Importantly, insufficient and inconsistent sleep has been associated with poorer inhibitory control and disinhibited eating in youth, as described in greater detail below (52, 55, 107), and may highlight a key target for obesity prevention efforts.

Self-Control Depletion Theory

Self-control depletion theory may provide a framework for better understanding how sleep is associated with poorer inhibitory control and disinhibited eating in youth (108). Self-control, similar to inhibitory control, refers to one's ability to inhibit maladaptive cognitions, emotions, and behaviors to persist toward goals (109). Theoretically, self-control resources become depleted after a person attempts repeatedly to regulate thoughts, emotions, desires to eat, or eating behaviors. This depletion is often compared to a muscle which experiences short-term weakness after repeated use (110). When self-control resources are diminished, individuals are more likely to engage in disinhibited eating behaviors. Importantly, adequate sleep may replenish self-control resources, and poor sleep may result in a more limited capacity to engage in acts of self-restraint (111, 112). Indeed, in a study among adolescents, sleep deprivation was associated

with low self-reported self-control, as assessed by the Weinberger Adjustment Inventory [WAI; (112, 113)]. It is therefore possible that insufficient and/or inconsistent sleep may lead to decreased self-control resources which contributes to disinhibited eating and excess energy intake.

Supporting this notion, a study found that bed- and waketimes (specific facets of sleep which are explained in more detail below) were associated with decreased eating-related self-efficacy, such that participants who went to bed later on weekends than weekdays had greater self-reported difficulty making healthy food choices in response to negative affect (55). In the same study, later weekend waketime was also associated with increased preoccupation with food. These results suggest that facets of sleep may be associated with a decreased ability to modulate food intake (i.e., decreased self-control).

While the self-control resource model is beyond the scope of the current studies, it provides a helpful framework for understanding how sleep is associated with disinhibited eating, food cravings, energy intake, and weight in youth. This model may also help explain the poorly understood link between food cravings and LOC-eating. It is possible that decreased self-control mediates the relationship between food cravings and LOC-eating such that if an individual experiences food craving and does not have the self-control to overcome that drive, they may be at heightened risk for engaging in LOC-eating. Though the three papers do not directly assess self-control depletion theory, it will be utilized as a conceptual framework to elucidate how facets of sleep are associated with disinhibited eating behaviors, energy intake, and adiposity in youth (Figure 6).

GENERAL INTRODUCTION TO PEDIATRIC SLEEP

Sleep plays an important role in healthy child and adolescent development (114). Generally, more hours of nightly sleep are linked with better psychological and physiological outcomes (114). Research has demonstrated that longer sleep duration is associated with better emotion regulation (115), cognitive performance (116), and cardiometabolic health (117). Conversely, shorter sleep and sleep disorders have been linked with poorer physical and mental health in youth (118, 119). Data support that sleep disorders are highly prevalent among children and adolescents (119, 120) with one study finding that 16.9% of participants age 2-18 reported (via self and/or caregiver report) habitual snoring, 15.1% having sleep disordered breathing, and 13.2% having insomnia (121). These statistics are concerning as sleep disorders have been associated with obesity risk in youth (122). For instance, in a large nationally representative sample of US adolescents, 13-18 years, youth with insomnia symptoms (defined as having difficulty initiating or maintaining sleep, and/or early morning awakening nearly every day for at least 2 weeks) had greater odds of having obesity than individuals without insomnia (69). Further, Beebe et al., (2011) found that among youth (10-17 years) with obesity, obstructive sleep apnea was associated greater consumption of highly palatable, energy-dense foods, even after adjusting for BMIz (123). Data also support the finding that obstructive sleep apnea, restless legs syndrome, and nocturnal enuresis are comorbidities of obesity in children and adolescents (122, 124). Finally, studies have linked sleep disorders with inattention, cognitive disorders, emotional regulation difficulties, depression, and increased risk for eating disorders (120, 125, 126). Therefore, it is important to understand the development and maintenance of normative and problematic sleep patterns in youth.

Sleep is developmentally patterned such that throughout childhood, sleep duration and timing change in a predictable manner (127, 128). Most data support that younger children require more hours of sleep while older children and adolescents need fewer (128). As such, the American Academy of Sleep Medicine recommends the following guidelines for sleep for every 24 hours by age: infants (4 months to 12 months) should sleep 12-16 hours including naps, children ages 1-2 years should sleep 11-14 hours including naps, children ages 3-5 years should sleep 10-13 hours including naps, children 6-12 years should sleep 9-12 hours, and adolescents age 13-18 should sleep 8-10 hours (129).

Additionally, emerging literature suggests that as children age, there are changes in sleep timing needs, or chronotype (130). Chronotype refers to biologically driven timing preferences for daily activities including exercising, eating, and sleeping (131). Typically, individuals are classified as having a morning type (indicating a preference for activities in the morning), neither morning nor evening, or an evening type (indicating a preference for activities in the evening). As children age, their chronotypes progress such that there is a shift to later sleep onset and waketimes, with a peak evening-preference typically occurring around 19 years (127, 132).

While studies support that adequate sleep duration and timing are linked with better physical and mental health outcomes (114), recent data suggest that only 5% of youth receive the recommended amount of sleep (133), and there is an increasing trend toward shorter sleep duration among youth (134). While the drivers of these decrements are not entirely clear, data indicate that increased screen time and increased educational and extra-curricular demands may be contributing (135, 136). Additionally, data support that there are differences in sleep duration associated with race and/or ethnicity. For example, a recent meta-analysis found that generally individuals who identify as Black or African American, Hispanic, or members of another

minority group obtain significantly fewer hours of sleep per night than individuals who identify as White (137). While the mechanisms underlying racial and ethnic disparities in sleep remain largely unclear, emerging data suggest it may be due, in part, to stress associated with racial discrimination (138). For instance, Yip et al. (2020) found that among adolescents (13-15 years) identifying as an ethnic/racial minority, self-reported daily discrimination based on race was associated with worse sleep [e.g. more reported sleep disturbance and greater next day sleepiness (138)]. Cultural factors, such as acceptability of co-sleeping, earlier/later bed timing, or daytime napping also plays a role in sleep practices that may lead to differences in sleep duration and timing (139, 140). For instance, one study found that among 40,000 European children, ages 11-16, there were significant self-reported differences in sleep duration and timing based on country and presumably culture [e.g., children from Switzerland slept longest, between 9-10 hours nightly, while children from Israel between 8.3-9 hours nightly (141)]. While authors of this study did not directly assess cultural differences, they hypothesized that variations in sleep patterns could potentially be driven by differences in school start/stop time, television programming availability, and cultural philosophies regarding leisure/exercise time (141). Further, while adolescents are known to have later chronotypes (130), school start times remain early. This incongruity between physiologically driven sleep timing and academic demands may lead to circadian rhythm misalignment (142-144). These demonstrated decreases in sleep length and changes in timing are concerning, as insufficient sleep duration and circadian rhythm misalignment has been associated with increased obesity risk in youth (39-41, 145).

SPECIFIC FACETS OF PEDIATRIC SLEEP AND OBESITY RISK IN YOUTH

Average Weekly Sleep Duration

Studies generally support a link between short nightly sleep and greater risk for higher BMI, overweight, and obesity in youth (39, 130, 146-150). In a recent systematic review examining the associations between multiple dimensions of sleep and obesity in children (40), eighty-six articles reported an inverse association between sleep duration and weight. This relationship has been demonstrated both cross-sectionally, as well as prospectively (39, 40, 145). For example, Martinez et al. (2014) found that among 303 Mexican-American children, less average sleep duration, as measured by actigraphy, was associated with higher BMIz [i.e., age and sex-adjusted BMI percentiles based on 2000 CDC growth reference (11, 151, 152)]. Prospectively, Silva et al. (2011) found that children who slept 7.5 hours or fewer nightly, as assessed by polysomnography, a complex medical procedure considered the gold standard for sleep evaluation (153), had a three times greater risk (OR=3.3) of having obesity five years later than those who slept 9 hours or more nightly (146). Similarly, another study examining multiple facets of objectively measured sleep longitudinally, found that a higher score for short sleep duration (i.e. children who had habitually shorter sleep from ages 2-6) was associated with greater increases in BMIz scores (154). These data highlight that age-appropriate sufficient sleep may be a key target of prevention and treatment efforts for childhood obesity.

It is possible the link between sleep duration and high weight is at least partially due to increased energy intake and poor diet quality (41). Indeed, experimental studies have found restricted nightly sleep, as assessed by actigraphy, to be associated with increased caloric intake in youth (155-158). Observational studies, using both objective (159, 160) and self-report (55) measures of sleep, have also reported an association between insufficient sleep and poorer diet quality, such as greater intake of added sugar, fats, and carbohydrates (55, 159, 160). For example, Mi et al. found that among children and adolescents, individuals who slept less than

seven hours nightly, as assessed by actigraphy, consumed a higher percentage of calories from carbohydrates at a buffet test meal than those who slept 7 hours or more, potentially increasing their risk for weight gain (160). Data support that excessive intake of energy-dense foods, such as added sugar, carbohydrates, and fat, promote weight gain (44-46). Conversely, diets that are high in vegetables, fruits, and whole grains are protective against excess weight (45, 161, 162). Therefore, it is possible that shorter sleep leading to higher intake from energy-dense foods may increase obesity risk in youth.

However, the data are not entirely consistent regarding the associations of sleep duration and obesity risk in youth (148, 163-165). For example, McNeil et al. (2015) reported that, after adjusting for relevant covariates (e.g. age, sex, ethnicity) objectively measured sleep duration was not associated with adiposity, waist circumference, or BMIz (165). Similarly, another study found no significant association between parent-reported sleep duration and intake of fruits and vegetables in either boys or girls (164). These mixed results suggest that sleep duration alone may not fully account for the associations between sleep and BMI, and that other aspects of sleep may play a relevant role (for list of sleep constructs and definitions, see Table 1).

One characteristic of sleep that may be particularly salient for obesity risk in youth is sleep variability (55, 159, 166, 167). There are several facets of sleep variability (Table 1) that generally can be divided into two broad categories: measures of the variability of sleep duration (i.e. measures based on hours per night of sleep, e.g. weekend catch-up sleep, or the difference between weekend sleep and weekday sleep (168)) and behavioral estimations of circadian rhythm shifts (i.e. metrics of sleep timing; e.g. bed- and waketime shift or difference in weekend and weekday bed/waketime (55)). Data generally support that sleep variability may play an

important role in energy intake, disinhibited eating, BMI, and adiposity (40, 41) beyond what is attributable to sleep duration alone.

Table 1. Sleep constructs

	Construct	Definition/calculation
General Sleep Constructs	Average weekly sleep duration	Average hours per week of sleep (52)
	Bedtime	The time one falls asleep. When using actigraphy data, defined as the first minute of a 5-minute period of no actigraphy-detected activity (160).
	Chronotype	Subjectively reported preference for time of day waking and activity (typically more morning or more evening) (130).
	Circadian rhythm misalignment	Sleep and wake cycles which are not appropriately related to biologically-driven sleep and wake preferences (169).
	In-bed-time	The duration (hours/night) one lies in bed (160).
	Waketime	The time one awakens. When using actigraphy data, defined as the first minute of a 5-minute period of actigraphy-detected activity (160).
	Weekday sleep	Average sleep Sunday night through Thursday nights (160, 170).
	Weekend sleep	Average sleep across Friday and Saturday nights (160, 170).
Sleep Duration Variability	Weekend catch-up sleep (Figure 2)	The difference between weekend sleep and weekday sleep [i.e. average weekend sleep (Friday-Saturday nights)-average weekday sleep (Sunday-Thursday nights)] (170).
	Within-subject sleep duration variability (Figure 1)	Night to night hours of variation from the mean and is calculated as the intra-subject standard deviation of average sleep duration across a multi-day period (167).
Circadian Rhythm Variability	Bed- and waketime shift (Figures 3 and 4)	The difference in weekend and weekday bed/waketime, calculated as average weekend bed/waketime minus average weekday bed/waketime (55).
	Social jet lag (Figure 5)	The difference between midpoint of sleep on the weekends and midpoint of sleep on weekdays (143)

Sleep Duration Variability

Within-Subject Sleep Duration Variability

Within-subject sleep duration variability is defined as night-to-night hours of variation from the mean and is calculated as the intra-subject standard deviation of average sleep duration across a multi-day period [Figure 1; (167, 171)]. Though data are limited, findings indicate that greater objectively measured within-subject sleep duration variability is associated with more abdominal obesity (171), increased energy intake, specifically from fats and carbohydrates (167), and increased sugar-sweetened beverage intake (159). These data suggest that variability in night-to-night hours of sleep could play a role in the development of high weight in youth; however, to date, all reported associations have been from cross-sectional data. Therefore, longitudinal data are needed to understand these relationships more fully.

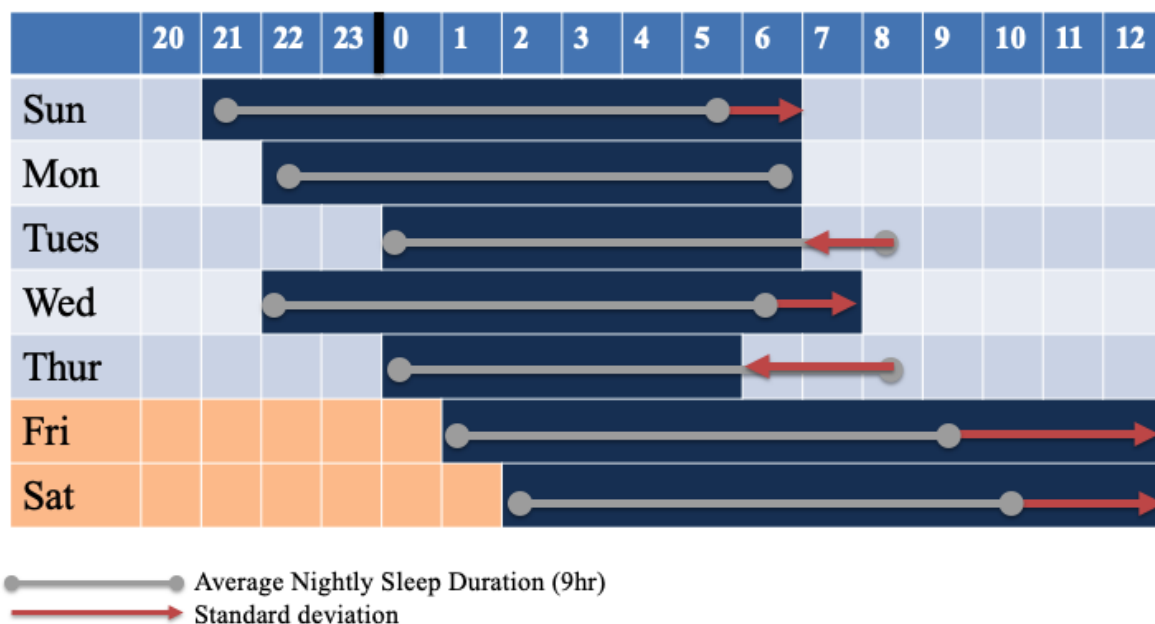


Figure 1. Within-subject sleep duration variability. Note: Hours of the night/morning are listed across the top with a black bar denoting the separation from night and morning. Abbreviated days of the week are listed in the left-most column. Friday and Saturday nights are highlighted as these are considered weekend nights. Within-subject sleep duration variability is calculated by taking the average standard deviation across a multi-day period.

Weekend Catch-Up Sleep

Weekend catch-up sleep is the difference in hours per night of sleep on the weekends, and weekdays [calculated as average hours/night of weekend sleep (Friday-Saturday nights) minus average hours/night weekday sleep (Sunday-Thursday nights; Figure 2; (170)]. Some data support a link between weekend catch-up sleep and risk factors for obesity in youth. However, the data on the association between weekend catch-up sleep and risk for high weight are mixed, with some self-report studies reporting inverse associations (172, 173) and a study using objective-measures of sleep reporting null findings (52). For example, one study using self-report measures of sleep found differences among children who compensated for their sleep on the weekend (i.e. had greater weekend catch-up sleep) and those who had persistently short sleep duration [≤ 8 hours during weekdays and weekends(173)]. As compared to the “compensated group,” those with persistently short sleep had over two and a half times greater likelihood of overweight/obesity, thus highlighting that catch-up sleep may be protective against high weight (173). Similarly, another study found no association between self-reported weekend catch-up sleep and weight or abdominal obesity in children and adolescents (174). While these findings highlight an inconsistent link between weekend catch-up sleep and obesity risk in youth, few studies have examined this association.

These mixed results suggest that while sleep duration variability may be an important factor in the development of high BMIz, and adiposity, other variables related to circadian rhythm alignment may also be important to consider. Indeed, recent studies have found that circadian rhythm variability may be particularly important in the development of high weight among youth (142, 143, 148, 167, 175).

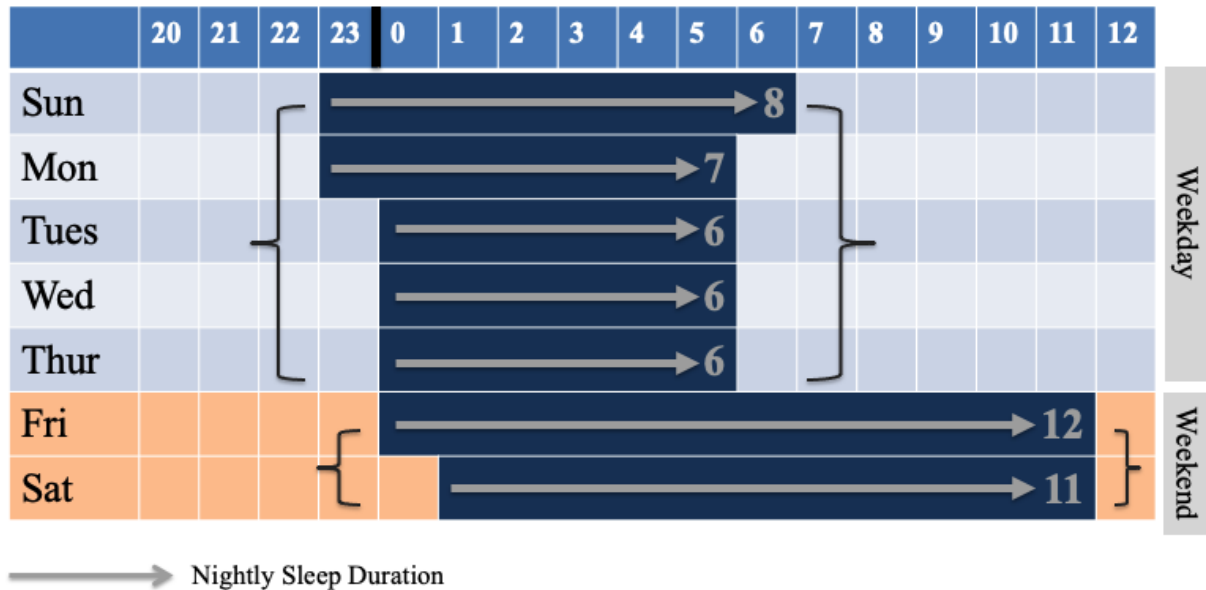


Figure 2. Weekend catch-up sleep. Note: Hours of the night/morning are listed across the top with a black bar denoting the separation from night and morning. Abbreviated days of the week are listed in the left-most column. Friday and Saturday nights are highlighted as these are considered weekend nights. Weekend catch-up sleep is calculated by subtracting average weekday sleep duration from average weekend sleep duration.

Bed- and Waketime Shift

Bed- and waketime shift are defined as the difference in average bed/waketimes on weekends and weekdays, calculated by subtracting weekday bed- and waketimes from weekend bed- and waketimes [Figures 3 and 4 (55)]. Data support a link between greater parent/self-reported bed- and waketime shift and excess weight and poorer diet quality (55, 174, 175). For example, one study among a large community sample of children and adolescents found that differences of two hours or more between weekend and weekday self-reported bedtimes (using the Pittsburgh Sleep Quality Index) were associated with two and a half times greater risk of overweight or obesity (174).

Bed- and waketime shift has also been associated with disinhibited eating behaviors. One study reported that later weekend bedtime compared to weekday bedtime was associated with decreased eating-related self-efficacy such that participants had greater self-reported difficulty

regulating negative affect in order to make healthy food choices [e.g., consuming more whole grains and fruits and vegetables than saturated fats and sugary beverages (55)]. It is possible that shift in sleep timing leads to a decreased ability to engage in self-regulation which could lead to disinhibited eating behaviors. This has been supported in other studies showing that poorer inhibitory control is associated with greater eating in response to negative affective states and in response to external food cues (62).

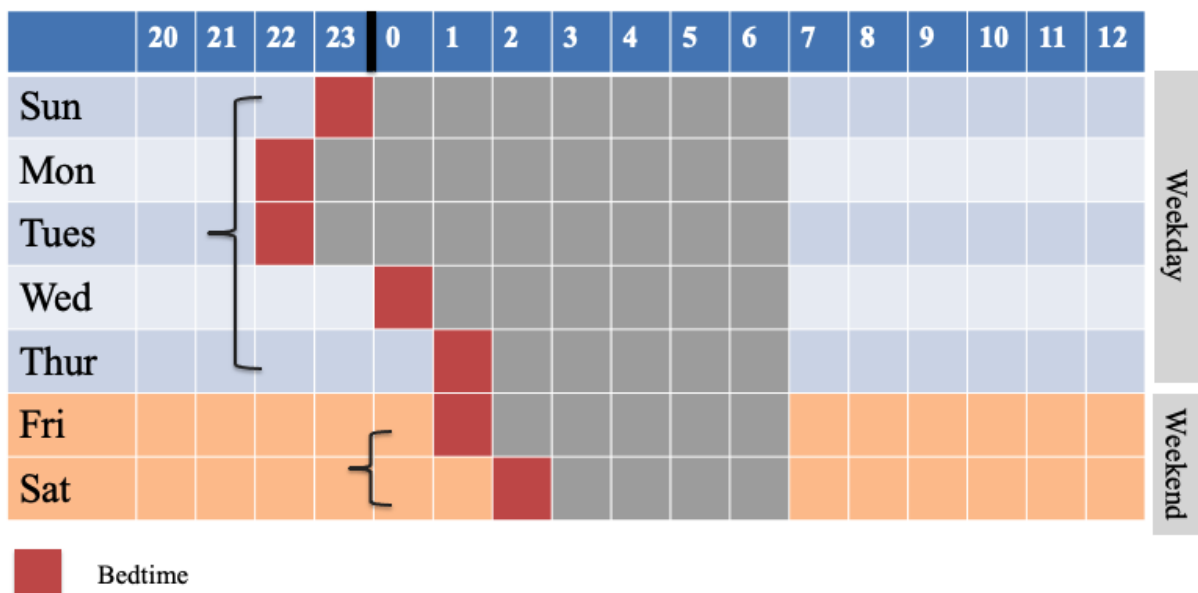


Figure 3. Bedtime shift. Note: Hours of the night/morning are listed across the top with a black bar denoting the separation from night and morning. Abbreviated days of the week are listed in the left-most column. Friday and Saturday nights are highlighted as these are considered weekend nights. Bedtime shift calculated by subtracting average weekday bedtime from average weekend sleep bedtime.

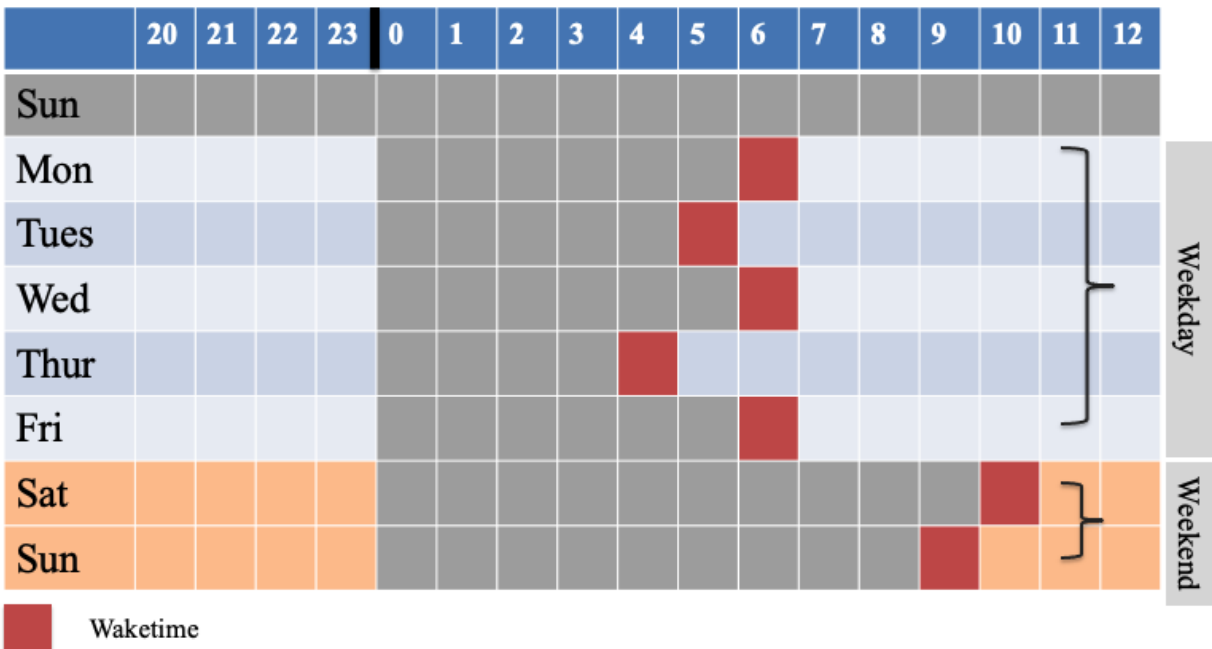


Figure 4. Waketime shift. Note: Hours of the night/morning are listed across the top with a black bar denoting the separation from night and morning. Abbreviated days of the week are listed in the left-most column. Saturday and Sunday mornings are highlighted as these are considered weekend mornings. Waketime shift calculated by subtracting average weekday waketime from average weekend waketime bedtime.

Social Jet Lag

Among circadian variables, social jet lag (a conflict between chronotype and environmental demands) may be particularly salient among adolescents (142, 143, 176). Social jet lag is calculated as the difference between midpoint of sleep on the weekends and midpoint of sleep on weekdays (Figure 5). This reflects an overall sleep timing difference, including bed and waketimes, between weekend and weekdays (143). As noted previously, as children age, there is a biologically based circadian shift to later sleep onset and waketimes [i.e. evening chronotypes, (127)], yet school start times remain early. This incongruity between physiologically driven sleep cycles and academic demands may lead to increased social jet lag among adolescents (142-144). Preliminary data in youth suggest that objectively measured (142, 143) and parent-reported (176) social jet lag is associated with greater energy intake and higher BMIz, waist-to-height ratio, waist circumference, and fat mass. However, there is a relative paucity of data in child and adolescent samples, and to date, only 1 study using a longitudinal design (177). This study

demonstrated that among a sample of 83 16-17-year-olds, self-reported social jet lag was not associated with gains in BMI, body fat percentage, or waist circumference at 1-year follow up (177). Though this study was in a relatively small sample, these data may suggest that other facets of sleep beyond social jet lag contribute to the long-term association between sleep and obesity risk in youth. More longitudinal data are needed to better understand this relationship

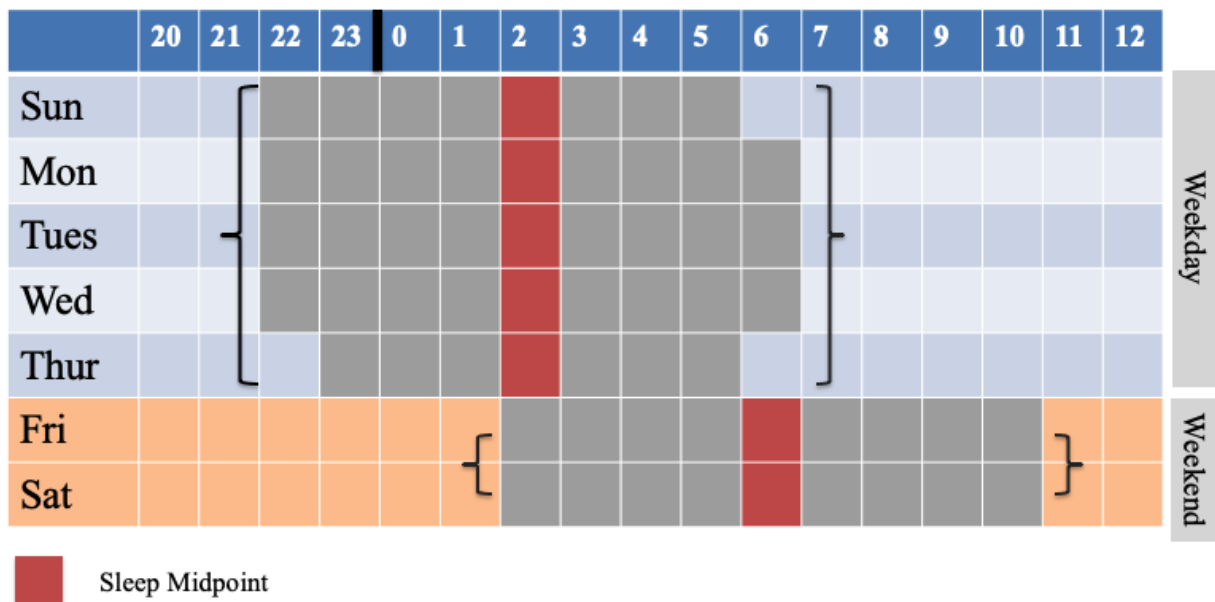


Figure 5. Social jet lag. Note: Hours of the night/morning are listed across the top with a black bar denoting the separation from night and morning. Abbreviated days of the week are listed in the left-most column. Friday and Saturday are highlighted as these are considered weekend days. Social jet lag is calculated by subtracting average weekday midpoint from average weekend midpoint.

Sleep Timing

Bed- and Waketime

Bedtime [i.e. the time one falls asleep (160)] and waketime (i.e. the time one awakens) have been associated with weight in youth (40), with data generally supporting a relationship between later self/parent-reported bed- and waketimes and higher weight (178-180). For example, in a recent 5-year longitudinal study examining multiple facets of actigraphy measured sleep in children from ages 2-6 years, data indicated that, independent of sleep duration, higher “late

sleep” score (i.e. children who consistently fell asleep later than 9pm across the study period) was associated with greater increases in BMIz scores and waist circumference (154). Later self-reported bed- and waketimes have also been associated with higher intake from sugar-sweetened beverages and nutrient-poor, energy-dense foods (181, 182). While data suggest that bed- and waketimes may be important to consider in relation to BMIz, adiposity, and obesity risk, other studies suggest that the shift in bed- and waketimes, indicating a circadian rhythm misalignment, may also be at play.

CONCLUSIONS AND PROPOSED THEORETICAL MODEL

Together these data highlight that there are several facets of sleep (including average weekly sleep duration, within-subject sleep duration variability, weekend catch-up sleep, bedtime and waketime shift, social jet lag, bed- and waketimes, and sleep midpoint) which may play an important role in the development and maintenance of high weight in youth. It is possible, that insufficient and inconsistent sleep could lead to depleted self-control resources and an inability to modulate one’s actions. This decreased self-regulatory capacity may then lead to disinhibited eating, food cravings, and/or greater energy consumption from energy-dense foods. Disinhibited eating behaviors, food cravings, and/or greater consumption of energy from fat, carbohydrates, and added sugar could then lead to greater adiposity in youth (Figure 6). Therefore, the series of studies examined these links.

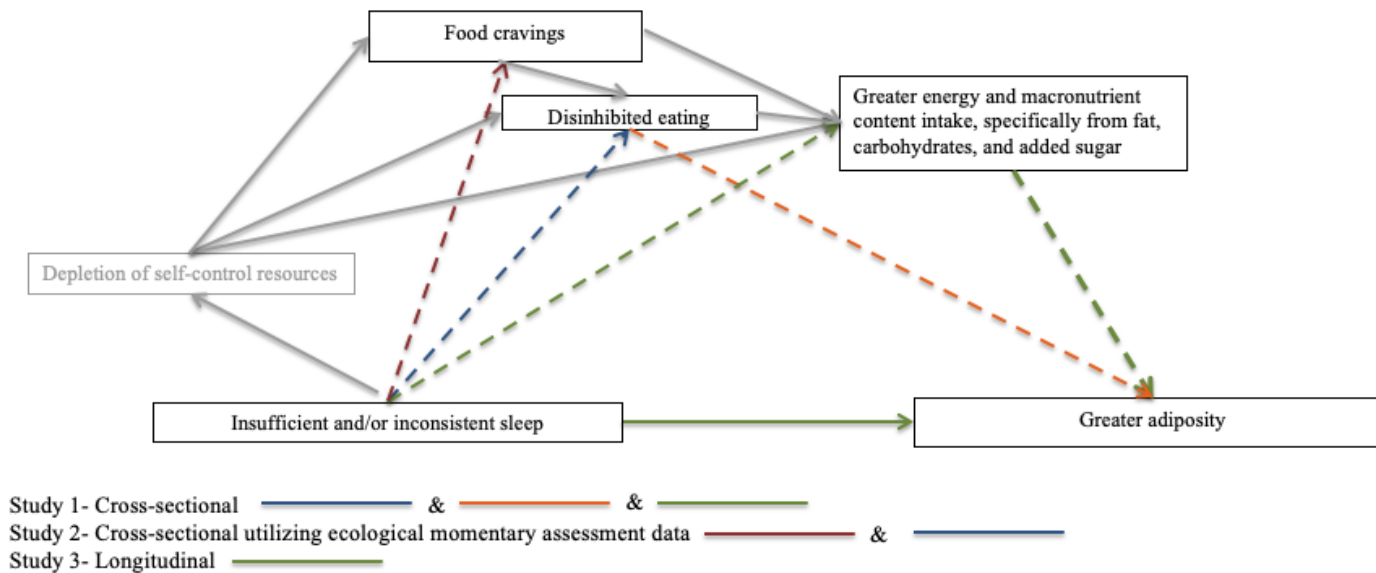


Figure 6. Conceptual model of the link between facets of sleep, disinhibited eating, food cravings, energy consumption, and risk for overweight/obesity among youth. Note: grey lines indicate associations that will not be formally tested in the current series of studies; greyed-out boxes indicate constructs that will not formally be tested in the current series of studies; dashed lines indicate potential mediators. Study 1 examined the cross-sectional link between facets of sleep, EAH, and adiposity as well as explores a potential mediation model. Study 2 utilized EMA data to examine the cross-sectional link between facets of sleep, LOC-eating, and food cravings. Study 3 utilized longitudinal data to study the link between facets of sleep and adiposity through energy and macronutrient content intake at a laboratory test meal.

IDENTIFIED GAPS

There are several identified gaps in the literature exploring the relationships between facets of sleep and obesity risk in youth that will be addressed in the series of studies. Primarily, there is a relative paucity of objectively measured sleep data in youth, with meta-analyses and systematic reviews finding that most studies relying on parent or self-report (39, 41, 145). Of concern, several studies rely on one or two self-reported items on questionnaires. Considering some research has found as few as 15 minutes of social jet lag to be associated with increased energy intake in young children (176), it is critical to use objective measurements which are more precise and can assess sleep more incrementally than self-report. Further, there is a relative lack of consensus on where to focus investigative efforts, and more data are needed to more fully understand which facets of sleep may be specifically linked to obesogenic behaviors in a broad age-range of children and adolescents. Outlined below are three specific gaps in the literature which the current studies seek to address.

Some studies suggest that a potential mechanism linking sleep and weight in youth may be increased disinhibited eating behaviors resulting from sleep-related decreased self-regulatory resources (55, 108). However, there are limited objectively measured sleep data examining this association. Therefore, the aim of Study 1 was to examine the relationships between average weekly sleep duration across all seven nights, weekday versus weekend sleep, and weekend catch-up sleep, using an objective measure of sleep, with self-reported eating in the absence of hunger and adiposity among a sample of non-treatment seeking youth.

There are very few studies examining temporal or within-subjects relationships between facets of sleep and eating behaviors. To date, there is only one study examining the association of at-home nightly sleep and next day eating behaviors in youth (183). In a relatively small sample of

29 youth with overweight or obesity, results indicated that longer total sleep time was associated with consumption of fewer calories from solid fats, alcohol, and added sugars. Study 2 aimed to build on these results by utilizing actigraphy and ecological momentary assessment to determine both the acute and cumulative associations of several facets of sleep with the intensity of food cravings and LOC-eating in one's natural environment during a 14-day period.

Finally, while there is a significant body of cross-sectional literature supporting the link between fewer hours of nightly sleep and high weight in youth, more longitudinal data are needed. There are even fewer longitudinal studies that utilize objective measures of sleep. Further, there are no longitudinal data examining multiple facets of sleep, including metrics of sleep duration, variability, or timing in a broad pediatric age range. Therefore, Study 3 sought to examine how multiple facets of sleep (including sleep duration, within-subject sleep duration variability, weekend catch-up sleep, bed-and waketime shift, social jet lag, bed- and waketimes, and sleep midpoint) are associated with 1-year change in adiposity in children and adolescents, and to explore potential mediation by energy intake.

CHAPTER 2: Study One- Associations of Weekday and Weekend Sleep with Children's Reported Eating in the Absence of Hunger

ABSTRACT

Insufficient average sleep duration has been inconsistently associated with poor diet and obesity risk in youth. Inconsistencies in findings across studies may be due to a general failure to examine associations by weekday versus weekend sleep. We hypothesized that greater variations in weekday and weekend sleep duration would be associated with more disinhibited eating behaviors which in turn, might be involved in the relationship between sleep and weight. We therefore examined, among healthy, non-treatment seeking youth, the associations of average weekly, weekend, and weekday sleep duration with eating in the absence of hunger (EAH), a disinhibited eating behavior associated with disordered eating and obesity. Sleep was assessed via actigraphy for 14 days. Participants completed a self-report measure of EAH. Adiposity was measured by dual-energy X-ray absorptiometry. Linear regressions were used to test the associations of sleep duration with EAH and the associations of sleep duration and EAH, with fat mass. Among 123 participants (8-17y; 52.0% female; 30.9% with overweight), there was no significant association between average weekly sleep and EAH. Further, there was no significant association among average weekly sleep duration or EAH and fat mass. However, average weekday sleep was negatively associated, and average weekend sleep was positively associated, with EAH ($p < 0.02$). Weekend “catch-up” sleep (the difference between weekend and weekday sleep) was positively associated with EAH ($p < 0.01$). Findings indicate that shorter weekday sleep and greater weekend “catch-up” sleep are associated with EAH, which may place youth at risk for the development of excess weight gain over time.

INTRODUCTION

In the United States, over one third of children and adolescents currently have overweight and nearly 20% have obesity (2, 15). Given the negative long-term health impacts of overweight and obesity (184-186), elucidation of early, modifiable risk factors for excess weight gain is necessary to enhance the effectiveness of obesity prevention and treatment in children and adolescents. Two such factors that have been associated with excess weight and adiposity among youth are insufficient sleep (39, 145) and disinhibited eating behaviors [eating behaviors characterized by a lack of healthy restraint (48-51, 187)].

Sleep plays a critical role in healthy child and adolescent development (114). Throughout childhood, sleep is developmentally patterned such that as children grow and mature, their sleep needs vary in a predictable manner (128). Specifically, most research indicates that younger children require more sleep while older children and adolescents need less (128). As such, the American Academy of Sleep Medicine recommends the following guidelines for sleep for every 24 hours by age: infants (4 months to 12 months) should sleep 12-16 hours including naps, children ages 1-2 years should sleep 11-14 hours including naps, children ages 3-5 years should sleep 10-13 hours including naps, children 6-12 years should sleep 9-12 hours, and adolescents age 13-18 should sleep 8-10 hours (129). Recent data indicate that only 5% of youth get the recommended amount of sleep (133) and that insufficient sleep has been an increasing trend (134). Children's sleep duration across all ages is shorter than it was in the past (134), such that overall duration among youth in the last 100 years has decreased by approximately one hour per night (134).

Reduced sleep duration has frequently been found to be associated with obesity in childhood and adolescence. Studies generally support a relationship between fewer than eight reported

hours of sleep nightly and greater risk for developing higher body mass index (BMI), overweight and obesity (39, 130, 146-149). However, the data are not entirely consistent (148, 163). For example, Jarrin et al. (148) reported that when relevant covariates (such as age, sex, pubertal status, physical activity, screen time, and parental education) were included in their model, the negative association between fewer hours of self-reported sleep and obesity was attenuated and no longer significant. Likewise, Lytle et al. (163) reported no statistically significant prospective relationship between decreases in self-reported sleep and increases in BMI or percent body fat in either adolescent boys or girls.

These mixed results suggest there may be other factors contributing to the association between children's sleep duration and BMI. One possible consideration is that there are many dimensions of sleep beyond the average number of hours spent in bed (148). For example, specific characteristics of sleep such as bedtime shift (i.e. going to bed at different times on weekdays versus weekends), and weekend (Friday and Saturday nights) versus weekday (Sunday through Thursday nights) sleep duration have been associated with greater severity of overweight and increased intake of sugar-sweetened beverages (55, 159, 178). Again, data are somewhat mixed. For example, Wing et al. found that self-reported weekend and holiday "catch up" sleep to be associated with a lower risk of overweight and obesity in children (173).

Given these mixed results, other studies have attempted to elucidate the associations of weekend versus weekday sleep and the neurobiological and behavioral underpinnings that may influence eating and weight (55, 61). For example, Hasler et al. (61) reported that greater weekend mid-sleep shift, or shifting bedtimes to either earlier or later on the weekend than bedtimes on weekdays leading to circadian rhythm misalignment, was associated with decreased medial prefrontal cortex and striatal reactivity to reward, suggesting decreased regulatory

responses and increased reward sensitivity. It is possible that such a decreased ability to self-regulate could lead to disinhibited eating behaviors, as other studies have shown poor inhibitory control associated with eating in response to external food cues and in response to negative emotional states (62). Supporting this notion, Ievers-Landis et al. (55) found that later weekend bedtime compared to weekday bedtime was associated with decreased eating-related self-efficacy, such that participants had greater self-reported difficulty regulating negative affect in order to make healthy food choices (e.g. consuming more whole grains and fruits and vegetables than saturated fats and sugary beverages). These results underscore the need to investigate not only average sleep duration, but also specific sleep characteristics, such as weekday versus weekend sleep and weekend “catch-up” sleep, with subsequent reward-based disinhibited eating behaviors known to be associated with obesity (60).

One disinhibited eating pattern that may play a role in obesity risk is eating in the absence of hunger (EAH), which is characterized by eating when not hungry, or past satiation in response to fatigue, negative affect, or when cued by external circumstances such as sight or smell of food (58, 188). Among children, EAH has been shown to be a stable trait (56, 57), appears to increase with age (56, 189), and cross-sectional data have shown the behavior (both measured in the laboratory and by self-report) is positively correlated with fat mass and BMIz (58). Yet, prospectively, EAH has not been shown to predict increases in BMI, BMIz or fat mass over a 1-year period among youth (56, 58, 59). Nevertheless, overweight and obesity track with EAH such that children with overweight and obesity have higher levels of EAH than their counterparts without overweight or obesity, and this relationship persists over time (60). Although EAH has been shown to be stable, its etiology remains unclear and it is possible that EAH may initially vary in response to poorer cognitive control and increased reward sensitivity. Given the mixed

findings, eating in the absence of hunger (EAH) may put certain individuals at increased risk of excess weight.

Taken together, extant studies suggest that both sleep behavior and EAH have associations with excess weight, however, some studies report inconsistent results and the relationships among these variables are not fully understood. Further data indicate that facets of sleep, such as weekday versus weekend sleep and weekend “catch-up” sleep, are associated with decreased eating-related cognitive control and increased reward sensitivity (55, 62, 171, 175) that could lead to poorer dietary decisions, including disinhibited eating patterns such as EAH. There are some data among adult samples to suggest that sleep duration is related to disinhibited eating behaviors and that short sleep may interact with these behaviors to promote excess weight (190, 191). However, although disinhibited eating is related to EAH, to date, there are no data among youth that examine the relationship between sleep and EAH specifically.

An important first step in understanding this potential relationship involves examining the association between overall average weekly sleep duration and facets of sleep (weekday, weekend, and weekend-“catch-up” sleep) with EAH. Therefore, we examined the association of: self-reported EAH with average weekly sleep duration, weekday (Sunday through Thursday nights) versus weekend sleep (Friday and Saturday nights), and weekend “catch-up” sleep [the difference between weekend and weekday sleep (170)] using an objective measure of sleep behavior among a sample of non-treatment seeking youth. We hypothesized that, after adjusting for relevant covariates (age, sex, race, height, total fat mass, and depressive symptoms), individuals with 1) less average weekly sleep 2) fewer hours of weekday sleep, 3) more hours of weekend sleep, and 4) greater weekend “catch-up” sleep would report greater EAH. Further, we expected that, after adjusting for relevant covariates (age, sex, race, height, and depressive

symptoms), individuals with 1) less average weekly sleep and 2) fewer hours of weekday sleep, 3) more hours of weekend sleep, 4) greater “catch-up” sleep, and 5) more reported EAH would have greater fat mass. Finally, in an exploratory analysis, we hypothesized that EAH would mediate the relationship between facets of sleep and fat mass such that fewer hours of average weekly or weekday sleep, and greater “catch-up” sleep would lead to greater EAH and greater fat mass.

MATERIALS AND METHODS

Participants

Healthy, non-treatment-seeking boys and girls (8-17 years old) were recruited to participate in the Children’s Growth and Behavior Study (Clinical Trials Identifier: NCT02390765), which was designed to understand eating behaviors in relation to body weight in children longitudinally. The protocol was approved by the Institutional Review Boards at the *Eunice Kennedy Shriver* National Institute of Child Health and Human Development and the Uniformed Services University. All participants were in good general health, with the exception of minor, well-controlled ailments. Exclusion criteria included: 1) history of major illness, brain injury, or pregnancy; 2) use of medication known to affect body weight or energy intake; 3) significant and recent weight loss (>5% body weight); 4) BMI < 5th percentile for age and sex; 5) current and regular use of illicit substances; 6) presence of a full-threshold psychiatric disorder; and 7) full scale intelligence quotient score < 70 (192). Signed informed consent and assent were obtained from parents/caregivers and children, respectively.

Procedure

Participants were seen at the outpatient Pediatric Clinic at the National Institutes of Health Hatfield Clinical Research Center. In order to evaluate overall health, a health history was taken, and children were examined by a physician or nurse practitioner. Following an overnight fast, height (cm) was measured in triplicate to the nearest millimeter by a calibrated stadiometer, and weight (kg) was measured using a calibrated scale after participants removed shoes and heavy clothing. BMI percentiles normed for age and sex were computed according to the Centers of Disease Control and Prevention growth standards, with overweight being defined as a BMI percentile 85th - <95th, and obesity defined as BMI percentile $\geq 95^{\text{th}}$ (151). Given that BMI is known to inconsistently capture differences in adiposity (193), we directly measured fat mass. Total lean and fat mass were measured using dual-energy X-ray absorptiometry (DEXA; iDEXA system, GE Healthcare, Madison, WI). As detailed below, participants completed questionnaires and were given wrist-mounted triaxial accelerometers to wear for a 14-day period on their non-dominant arm.

Measures

Sleep. Wrist-mounted triaxial accelerometers (ActiGraph LLC, Pensacola, FL, model GT3X) were configured [based on height (cm), weight (kg), race, age, and handedness] to collect information in 60 second epochs. Participants were instructed to wear accelerometers throughout 14 days and nights. They were asked to complete sleep diaries during this period, as well as to make notes of anything that might have significantly influenced their sleep (e.g., staying over at a friend's house). To be included in analyses, participants were required to have valid data from ≥ 3 weekday nights of sleep and ≥ 1 weekend night of sleep. As described in Mi et al., 2019 (160), sleep data were downloaded from devices using Actilife software version 6.13.3 (ActiGraph, Pensacola, FL). The Sedeh sleep detection algorithm, which has been previously

validated for adolescents (194), was applied to data in order to detect periods of sleep. Data were also visually inspected and confirmed by cross-referencing sleep diaries. In-bed time was defined as the first of a five-minute period in which activity counts were all zero and the subsequent activity counts were determined to be sleep by the Sedeh algorithm. Wake up time was similarly defined as the first of a five-minute period in which activity counts were above zero and the subsequent activity was determined by the Sedeh algorithm to be awake time. Average nightly sleep (min/night; total time in bed minus sleep onset minus minutes of wakeful activity after sleep onset) was also calculated. If a nap occurred less than 60 minutes from the wake-up time, the nap was included as part of the total nightly sleep. Naps (i.e., sleep that did not occur during part of total nightly sleep) were not included in analyses. Average weekly sleep duration (hour/night) of all nights (Sunday-Saturday) was calculated. Additionally, the average durations (hour/night) of weeknights (Sunday-Thursday), and weekend nights (Friday-Saturday) were calculated. Finally, weekend “catch-up sleep” was calculated by subtracting average weekday sleep from average weekend sleep (hour/night) (170). The term “catch-up sleep” is used in the literature for this weekend-weekday difference even though there are participants who sleep less on the weekends (170). Actigraphy has been previously validated for objective sleep duration measurement, with an overall agreement rate of >90% when compared to polysomnography (PSG) in normal adult subjects (195, 196). In a study done in healthy youth (5-12y), actigraphy also demonstrated acceptable validity for average weekly sleep when compared to PSG (197).

Eating in the absence of hunger. EAH was measured with the 14-item Eating in the Absence of Hunger Questionnaire for Children and Adolescents [EAH-C (188)]. Items assessed eating when not hungry or past satiation in response to external cues (e.g., “how often do you keep eating because others are still eating?”); fatigue and boredom (e.g., “how often do you keep

eating because you are feeling bored”); and negative affect (e.g., “how often do you keep eating because you are feeling anxious or nervous?”). Items are rated on a 5-point Likert scale ranging from 0= “never” to 4= “always.” The average total score of all items on the EAH-C was used, with higher scores indicating greater EAH. The EAH-C has demonstrated convergent validity and temporal stability for all scales (188), and good internal consistency in the current sample (Cronbach’s $\alpha=0.85$).

Depressive symptoms. Depressive symptoms were assessed using the Children’s Depression Inventory- Second Edition (198-200). This measure asks respondents to indicate to what degree they have experienced depressive symptoms in the last two weeks. Each item response ranges in order from 0-2 and total scores range between 0 and 52. Higher total scores indicate more depressive symptoms. The Children’s Depression Inventory- Second Edition is a widely used measure with good validity (201), and good internal consistency for the present sample (Cronbach’s $\alpha=0.86$).

Statistical Analyses

All analyses were conducted using SPSS for Windows version 25 (IBM Corp. Armonk, NY). Data were screened for outliers and normality. Neither EAH-C total scores nor depressive symptom total scores were normally distributed; therefore, scores were log base 10 transformed to achieve normality.

Three multiple linear regressions were conducted to examine the associations of 1) average weekly sleep (hour/night), 2) average weekday and weekend sleep time, (entered into the same model; hour/night), and 3) weekend “catch-up” sleep (hour/night) with EAH-C total score. Four additional multiple linear regressions were conducted to examine the associations of: 1) average weekly sleep (hour/night), 2) average weekday and weekend sleep time, (entered into the same

model; hour/night), 3) weekend “catch-up” sleep (hour/night), and 4) EAH-C total score in relation to fat mass (kg). Because sleep timing is known to have associations with excess weight (181), eating related cognitive control (55), and reward sensitivity (61), in exploratory analyses, all tests were re-analyzed using weekday versus weekend in-bed time and wake times rather than sleep duration. For all models, given the links of depressive symptoms with sleep in children (202, 203), depressive symptoms score entered as a covariate. Additionally, all models were adjusted for age, sex (0=male, 1=female), race (1=Non-Hispanic white, 0=other), height (cm). Finally, total fat mass (kg) was entered as a covariate in models that included EAH as a dependent variable. All tests were two-tailed and, $p < 0.05$ was considered significant. Using G*Power software version 3.1 (204), a power analysis was conducted to determine the sample size required for a small effect size ($r^2 = 0.14$) with $\alpha = 0.05$, power = 0.80, and the number of predictors as 7. The sample size needed was determined to be 96, therefore, our sample would be predicted to be adequately powered for such analyses.

Understanding that sleep differs across the age spectrum (114, 128), secondary analyses were conducted to examine if patterns of results differed by age (child <12 years; adolescent ≥ 12 years). Although sample sizes were not adequately powered for statistical interpretation by age group, patterns of results did not differ from the total sample; therefore, all subsequent results are represented with the age groups combined.

An exploratory mediation model was conducted using Preacher and Hayes Indirect Mediation macro for SPSS (205). The model examined whether EAH mediated the relationship between facets of sleep and fat mass. Bootstrapping with 1000 resamples was used to estimate the 95% confidence interval for indirect effects.

RESULTS

One-hundred-forty-eight children participated. Twelve youths were excluded from analyses due to non-compliance with the actigraphy-wearing protocol and ten were excluded due to technological issues downloading their actigraphy data. Those with missing actigraphy data did not significantly differ from the studied sample with regard to sex, race, age, or BMIz ($p>.22$). One participant was excluded for missing depressive symptom data, and two participants were excluded due to missing DEXA data. Therefore, a total of 123 participants (52.0% female; 12.7 ± 2.6 y; 43.9% Non-Hispanic White; 30.9% with overweight/obesity) were included in the final analyses (Table 1). On average, from the 14 days of data collection, data were available for $13.3 (\pm 2.2)$ days of average weekly sleep, $9.5 (\pm 1.6)$ days of weekday sleep, and $3.8 (\pm .9)$ days of weekend sleep. Average weekly sleep was 7.3 ± 0.8 hour/night, average weekday sleep was 7.2 ± 0.8 hour/night, average weekend sleep was 7.5 ± 1.0 hour/night, and average weekend “catch-up” sleep was 0.3 ± 1.0 hour/night. Mean values were significantly different between sample with overweight and sample without overweight for race/ethnicity, fat mass and BMIz scores, as expected.

Table 1. Sample characteristics.

Characteristic ^a	Sample <i>with</i> Overweight (N=38)	Sample <i>without</i> Overweight (N= 85)	Total Sample (N=123)	Total Sample Range
Age (years)	12.4 ± 2.5	12.9 ± 2.6	12.7 ± 2.6	8.0 – 17.9
Sex (% female)	58.0	49.0	52.0	
Race/Ethnicity (%)*				
Hispanic or Latino	7.9	7.1	7.3	
Asian	0.0	16.5	11.4	
Black or African American	47.4	21.1	29.3	
Multiple Races	10.5	5.9	7.3	
White	31.6	49.4	43.9	
Unknown	2.6	0.0	0.8	
Fat Mass (kg) *	24.9 ± 9.4	11.2 ± 4.4	15.5 ± 9.0	4.0 – 54
Overweight/Obesity (≥ 85 th percentile, %)			30.9	-1.6 – 2.8
BMIZ Score*	1.7 ± 0.4	0.0 ± 0.7	0.5 ± 1.0	-1.6 – 2.8
Average Weekly Sleep Duration (hour/night)	7.1 ± 0.8	7.3 ± 0.7	7.3 ± 0.7	4.9 – 8.8
Weekday Sleep Duration (hour/night)	7.0 ± 0.8	7.2 ± 0.8	7.2 ± 0.8	4.6 – 8.7
Weekend Sleep Duration (hour/night)	7.3 ± 1.0	7.6 ± 1.0	7.5 ± 1.0	5.3 – 9.6
Weekend “Catch-Up” Sleep Duration (hour/night)	0.2 ± 0.9	0.3 ± 1.0	0.3 ± 1.0	-1.5 – 4.5
Eating in the Absence of Hunger	1.1 ± 0.5	1.1 ± 0.5	1.1 ± 0.5	0.5 – 3.0

^aValues presented are mean ± standard deviation, unless otherwise noted as percentage. Results are presented for individuals

with/without overweight to examine if there are any significant differences in outcome variables between the two groups.

*Mean values were significantly different between the sample with overweight and the sample without overweight for race/ethnicity, fat mass and BMIZ scores, as expected; $p < 0.05$.

EAH

Adjusting for covariates, average weekly sleep duration (hour/night for the entire week) was not significantly associated with EAH ($F(7,115)=1.57$, $\beta=-0.09$, $p=0.37$, $R^2=0.09$, Figure 1A). However, weekday average sleep time (hour/night) was inversely associated with EAH ($F(8,114)=2.46$, $\beta=-0.25$, $p=0.01$, $R^2=0.15$; Figure 1B) such that participants with greater weekday sleep reported lower EAH. Conversely, weekend average sleep time (hour/night) was positively associated with EAH ($F(8,114)=2.46$, $\beta=0.23$, $p=0.02$, $R^2=.15$; Figure 1C) such that participants with more weekend sleep reported greater EAH. Finally, weekend “catch-up” sleep (hour/night) was positively associated with EAH-C total score ($F(7,115)=2.79$, $\beta=0.26$, $p=0.004$, $R^2=0.15$, Figure 1D) such that individuals with greater weekend “catch-up” sleep reported greater EAH. In exploratory analyses, neither weekday [$F(8,114)=1.61$, $\beta=-0.09$, $p=0.38$, $R^2=0.10$] nor weekend [$F(8,114)=1.61$, $\beta=0.18$, $p=0.11$, $R^2=0.10$] in-bed time was significantly associated with EAH. Similarly, neither weekday [$F(8,114)=1.36$, $\beta=-0.06$, $p=0.61$, $R^2=0.30$] nor weekend [$F(8,114)=1.36$, $\beta=-0.04$, $p=0.71$, $R^2=0.30$] wake time was associated with EAH.

Fat Mass

Adjusting for age, sex, height, race, and depressive symptoms, none of the facets of sleep [average weekly $F(6,116)=3.76$, $\beta=-0.07$, $p=0.42$, $R^2=0.16$; weekday $F(7,115)=3.76$, $\beta=0.001$, $p=0.99$, $R^2=0.17$; weekend $F(7,115)=3.79$, $\beta=-0.12$, $p=0.22$, $R^2=0.18$, or weekend “catch-up” $F(6,116)=3.79$, $\beta=-0.08$, $p=0.38$, $R^2=0.16$] were significantly associated with fat mass, respectively. Moreover, EAH-C total score was not significantly associated with fat mass ($F(6,116)=3.74$, $\beta=-0.06$, $p=0.47$, $R^2=0.16$). In exploratory analyses, neither weekday [$F(7,115)=3.55$, $\beta=-0.17$, $p=0.12$, $R^2=0.18$] nor weekend in-bed time [$F(7,115)=3.55$, $\beta=0.005$, $p=0.68$,

$R^2=0.18$] was significantly associated with fat mass. Similarly, neither weekday [$F(7,115)= 3.12$, $\beta=0.05$, $p=0.66$, $R^2=0.16$] nor weekend [$F(7,115)= 3.12$, $\beta=-0.01$, $p=0.91$, $R^2=0.16$] wake time was associated with fat mass.

Figure 1.

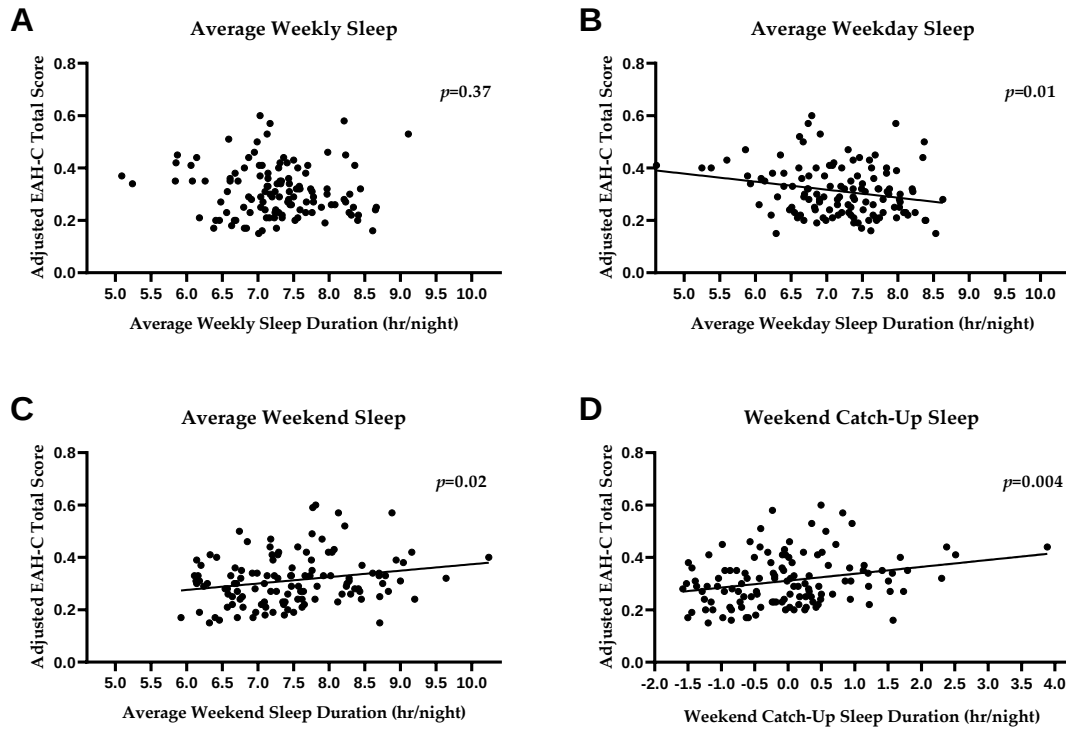


Figure 1: Associations between sleep and eating in the absence of hunger (EAH). There was no association between average weekly sleep and EAH total score ($p=0.37$; Figure 1A). There was a significant inverse association between EAH and average weekday sleep ($p=0.01$; Figure 1B), and a significant positive association between average weekend sleep and EAH ($p=0.02$, Figure 1C). There was a significant positive association between weekend “catch-up” sleep and EAH ($p=0.004$, Figure 1D). All analyses were adjusted for age, sex, race, height, total fat mass, and depressive symptoms.

Exploratory Mediation Analyses

EAH did not mediate the relationship between any facet of sleep [average weekly ($R^2 = 0.01$; 95% bootstrap CI: (-0.004 to 0.009); SE= 0.003), weekday ($R^2 = 0.01$; 95% bootstrap CI: (-0.005 to 0.010); SE= 0.004), weekend ($R^2 = 0.007$; 95% bootstrap CI: (-0.005 to 0.004); SE= 0.002), weekend “catch-up” ($R^2 = 0.004$; 95% bootstrap CI: (-0.009 to 0.007); SE= 0.004] and fat mass (Table 2).

Table 2. Results of mediation model

Facet of Sleep	Direct Effect (c' path)			Indirect Effect (ab path)		
	t- value	SE	p -value	95% CI	R^2	SE
Average Weekly Sleep	-1.33	0.02	0.19	-0.004-	0.01	0.003
Weekday Sleep	-1.30	0.02	0.20	0.009	0.01	0.004
Weekend Sleep	-0.90	0.01	0.37	-0.005-	0.007	0.002
Weekend Catch-up Sleep	0.02	0.01	0.85	0.01	0.004	0.004
				-0.005-		
				0.004		
				-0.009-		
				0.007		

DISCUSSION

Using actigraphy, an objective measure of sleep duration, we found no significant association between average weekly sleep duration and self-reported EAH. However, when sleep duration was examined separately for weekdays and weekends, significant associations emerged such that weekday sleep was negatively associated with EAH, weekend sleep was positively associated with EAH, and, correspondingly, weekend “catch-up” sleep was positively associated with EAH.

The finding that there was no significant association between average weekly sleep and EAH was contrary to our hypothesis. It is possible that examining sleep as a continuous variable rather than categorically by sufficiency attenuated the strength of this association. Indeed, previous literature often has categorized youth sleep duration into “inadequate” or “adequate” when examining it in relation to weight outcomes (145). Studies suggest that sleeping too little (less than eight hours per night) or too much (twelve or more hours per night) may be more robustly associated with eating behaviors and obesity risk than average sleep duration alone (206-209). However, given the lack of specified hours of nightly sleep recommended for certain age ranges (145), and the lack of individuals in the present sample with 12 hours or more of sleep, the present study analyzed the sleep data continuously rather than categorized by sleep sufficiency and adjusted for age.

Notably, both weekday and weekend sleep were significantly associated with EAH. EAH was inversely associated with weekday sleep, but positively associated with weekend sleep and weekend “catch-up” sleep. It appears that examining group means obscures the individual differences in weekday and weekend sleep, which are reflected in the wide range of Weekend Catch-Up Sleep Duration in Table 1 (-1.5 to +4.5 hours). These findings highlight the potential importance of sleep variability. A study by He et al. (171) made use of objective measures of

sleep and found that habitual sleep variability (intra-subject standard deviation of average sleep duration across all 7 nights of sleep) was significantly associated with abdominal adiposity among adolescents. Notably, however, He et al. found no significant association between total average sleep duration and abdominal adiposity. Such findings are consistent with other reports indicating that sleep variability may be associated with eating behaviors and excess weight over and above sleep duration alone (55, 159, 171, 175, 178). Our results complement and build on this literature in that even small differences in sleep duration between weekday and weekend may have unique associations with disinhibited eating behaviors.

Similarly, our findings indicated that children with greater weekend “catch-up” sleep reported greater EAH. This supports previous literature, which suggests that weekend “catch-up” sleep may be uniquely associated with reward-based disinhibited behaviors due to circadian rhythm misalignment (173). Although there are few studies to date examining the link between circadian rhythm shift and eating behaviors among youth, a recent experimental study found that adults who worked night shifts are more likely to choose a high-fat breakfast option than those who worked regular daytime hours (210). Interestingly, Depner et al. (211) recently reported that ad libitum weekend recovery sleep could not prevent metabolic dysregulation caused by experimentally induced insufficient sleep in young adults. These findings highlight the potential impact of circadian rhythm shift not only on disinhibition, but also on dietary intake and the metabolic consequences of short sleep. Data further suggest that sleep restriction and circadian misalignment are associated with decreased concentrations of leptin, an appetite hormone important for satiety, and with increased concentrations of ghrelin, an appetite-stimulating hormone (156, 212-215). It is possible that youth experiencing a shift in their biological circadian rhythm by engaging in weekend “catch-up” sleep (216) may create hormonal changes

associated with increases in consummatory behaviors. Future research is warranted to assess the impact of weekday versus weekend sleep, weekend “catch-up” sleep, and circadian misalignment on these hormones.

We did not find a significant association for any sleep variable or EAH with fat mass. The extant literature is mixed regarding the association of sleep and EAH with adiposity (54, 60, 130, 156, 159, 171, 217). Although neither sleep nor EAH was associated with fat mass, the significant association of weekend and weekday sleep with EAH may highlight an important risk factor for the development of excess weight gain over time. In exploratory analyses, EAH also did not significantly mediate the relationship between any of the facets of sleep examined and fat mass. This may be due to the relatively small sample size, as previous studies that have observed a relationship between objectively measured sleep and weight have had larger samples (130, 159). Further, data support that other dimensions of sleep such as poor sleep quality and evening chronotype (preference for later bedtime) are associated with increased risk of excess weight in youth (130, 217-219). It is possible these factors, which were not measured in the present study, are more robustly associated with fat mass than nightly hours of sleep. Longitudinal data from large samples are needed to investigate these relationships further.

Study strengths include the use of actigraphy to analyze participants’ sleep, which provided an objective measure of a difficult variable to quantify by self-report. Additionally, our study included a racially and ethnically diverse sample of healthy youth with a broad range of ages and weights. Finally, we utilized a widely used self-report measure of EAH (188).

Limitations of this study include use of cross-sectional data, and thus, no causal or mechanistic conclusions can be drawn from findings. An additional limitation is that our sample was not large enough to examine the variability of sleep needs across development (128) and the

range of sleep was limited. Similarly, our sample did not have enough participants with overweight or obesity to analyze results by weight status. Given the known associations between sleep duration and EAH with weight status (39, 58, 60, 130, 146, 148, 149), future studies should recruit individuals specifically with overweight and obesity in order to better elucidate any potential differences among groups. Additionally, given the nature of the EAH-C as a self-report measure, it may not reflect actual energy intake (220, 221); however, the measure is positively associated with daily energy intake among obesity-prone youth (220) and therefore may highlight a disinhibited eating behavior that places youth at risk for excess weight. Future studies should investigate sleep variability in relation to EAH assessed in the laboratory. Additional research should also assess these associations among samples that specifically recruited individuals with overweight and obesity. Finally, future studies should assess chronotype in order to effectively examine the associations of chronotype and circadian misalignment with EAH and fat mass.

In conclusion, this study builds on previous literature to suggest that sleep characteristics such as weekday versus weekend sleep and weekend “catch-up” sleep may be associated with higher self-reported disinhibited eating behavior (55, 159, 178). Prospective data are needed to determine if this association contributes to the relationship between sleep and excess weight gain risk, as EAH and sleep variability may be modifiable targets for reducing and preventing the high rates of pediatric obesity.

For the first page of the published version, refer to Appendix A

CHAPTER 3: Study 2 Associations of Sleep with Food Cravings and Loss-of-Control Eating in Youth: An Ecological Momentary Assessment Study

ABSTRACT

Background: Inconsistent sleep patterns may promote excess weight gain by increasing food cravings and loss-of-control (LOC)-eating; however, these relationships have not been elucidated in youth. **Objective:** We tested whether sleep duration and timing were associated with food cravings and LOC-eating. **Method:** For 14 days, youths wore actigraphy monitors to assess sleep and reported severity of food cravings and LOC-eating using ecological momentary assessment. Generalized linear mixed models tested the associations between weekly and nightly shifts in facets of sleep (i.e., duration, onset, midpoint, and waketime) and next day food cravings and LOC-eating. Models were re-run adjusting for relevant covariates (e.g., age, sex, adiposity). **Results:** Among 48 youths (12.88±2.69 years, 68.8% female, 33.3% with overweight/obesity), neither weekly nor nightly facets of sleep were significantly associated with food cravings (p s=.08-.93). Youths with shorter weekly sleep duration (est. β =-0.31, p =.004), earlier weekly midpoints (est. β =-0.47, p =.01) and later weekly waketimes (est. β =0.49, p =.01) reported greater LOC-eating severity; findings persisted in adjusted models. **Conclusions:** In youth, weekly, but not nightly, shifts in multiple facets of sleep were associated with LOC-eating severity; associations were not significant for food cravings. Sleep should be assessed as a potentially modifiable target in pediatric LOC-eating and obesity prevention programs.

Keywords: food cravings, loss-of-control eating, youth, ecological momentary assessment, sleep, actigraphy

INTRODUCTION

Loss-of-control (LOC)-eating, and the factors that promote LOC-eating, may be modifiable targets for the prevention of obesity. LOC-eating, a subjective sense of feeling out of control while eating, is prospectively predictive of excessive gain in BMI, the development of overweight and obesity, and worsening of components of metabolic syndrome in youth (49, 50, 222). Food cravings [i.e., intense desires to eat specific food(s) or type of food (223)] are positively linked to the severity of LOC-eating (72) as well as greater energy intake, weight gain, and obesity in adult and pediatric samples(73). One preliminary study suggests food cravings are modifiable among youth (71); therefore, the identification of factors that promote food cravings among youth across a wide weight range could inform the enhancement of obesity prevention programs.

One modifiable factor that may promote or exacerbate food cravings and subsequent LOC-eating is sleep. Sleep patterns are comprised of several facets that have been linked to obesity risk in youth. Specifically, fewer hours of nightly sleep [sleep duration (39, 150)] greater within-person sleep duration variability [the within-subject standard deviation of average sleep duration across a multi-day period (167, 171)] and later sleep onset and wake timing [falling asleep and waking up at a time that is later than one's average fall asleep or wake time (154, 178, 179)] have been associated with poorer dietary habits, aberrant eating behaviors, and increased obesity risk in youth (41). While the mechanisms linking sleep to pediatric obesity are largely unknown, acute changes in sleep duration and timing may indirectly contribute to obesity through a positive association with LOC-eating and with the desire to eat. However, data exploring the link between these facets of sleep and food cravings and LOC-eating are sparse.

Findings from the few studies examining the association between sleep and food cravings, or food craving-related constructs (e.g., desire for food and food appeal) in youth are inconsistent. For example, Kracht et al. (2019) cross-sectionally analyzed multiple facets of sleep and frequency of food cravings and found that sleep efficiency (i.e., the proportion of time one is asleep while lying in bed), but not sleep duration or variability, was related to cravings for foods high in fat and carbohydrates (224). Yet, preliminary experimental studies among adolescents have linked earlier bedtimes to lower desires for sweet foods (225) and shorter sleep duration to increased desires for sweets (158). Although there is no consensus regarding which facets of sleep are associated with food cravings, preliminary research suggests a link between sleep duration/timing and food cravings.

The only two studies among children and adolescents examining the link between sleep duration and LOC-eating did not find a significant association. Kelly et al. (2016) studied adolescent girls with overweight/obesity at high-risk for type 2 diabetes and found that self-reported sleep duration was not linked to LOC-eating (54). Using ecological momentary assessment (EMA), Goldschmidt et al. (2020) failed to detect between-subject associations among characteristics of prior night's sleep (e.g., sleep duration, sleep efficiency, wake after sleep onset, number of nighttime awakenings) and odds of reporting LOC-eating the following day among 40 youth with overweight and obesity (183). However, given the paucity of studies testing the associations of sleep with LOC-eating among youth across the weight strata, examination of the link between sleep and LOC-eating in more heterogeneous samples of youth is needed. Such data would elucidate whether insufficient and inconsistent sleep are relevant for the development of pediatric obesity.

Inconsistent and statistically nonsignificant findings across previous studies testing the associations among sleep, food cravings and LOC-eating may be due, in part, to multiple methodological and statistical limitations. First, studies that rely solely on retrospective self-reports of sleep duration and timing (54, 225) are limited due to potential bias and inaccuracy in measurement of sleep (41). Second, there are distinct yet interrelated facets of sleep linked to pediatric obesity (e.g., timing of sleep onset, midpoint, and waketime) that may impact food cravings and LOC-eating that have not been consistently examined in previous studies. This is potentially problematic because going to bed later than usual but arising at one's typical waketime constitutes a shift or deviation from one's average sleep onset and duration. Yet, previous studies (54, 158, 183, 224, 225) have examined the effects of independent facets of sleep on food cravings/LOC-eating without accounting for how individual facets of sleep duration and timing relate to one-another – thereby insufficiently considering this interdependence conceptually and statistically.

Lastly, measures of sleep duration and timing are often averaged over a one- or two-week period, but it is possible that daily shifts in sleep acutely impact eating drives and behaviors. Indeed, an experimental study using a randomized and balanced within-subject design found that, among young adult men, just one night of total sleep deprivation was associated with greater portion size intake the next day compared to one night of adequate sleep (226). However, to date, the effects of acute shifts in sleep on next day eating drives and behaviors have not been examined in youth using a within-subject design. Thus, methodologically and conceptually rigorous studies that address these limitations are necessary to clarify previous discrepant findings.

Given the possibility that variations in sleep patterns may promote greater food cravings, which may in turn increase LOC-eating severity, further exploration of these relationships is warranted. We therefore utilized concurrent actigraphy (an objective measure of sleep) and EMA (repeated measurement of current behaviors and experience in real time) to examine associations between weekly and nightly facets of sleep duration and timing and intensity of food cravings and LOC-eating in the natural environment during a 14-day period. We hypothesized that after adjusting for all facets of sleep 1) youth with, on average, fewer hours of weekly sleep and later sleep onset, midpoint, and waketimes would report greater food cravings and LOC-eating severity, and 2) greater shifts in previous night's sleep duration, onset, midpoint, and waketime would be associated with greater next day food cravings and LOC-eating severity.

METHOD

Participants

A secondary analysis of data collected as part of the Children's Growth and Behavior Study (Clinical Trials Identifier: NCT02390765) was conducted. Some findings related to sleep and eating behaviors have been previously reported (52, 160). Youth in good general health were recruited to participate in a study designed to understand the relationships between eating behaviors and body weight in children. Youth were eligible if they were 8-17 years old, cognitively capable of completing study procedures, and had a BMI $\geq$ 5th percentile for age and sex according to the Centers for Disease Control and Prevention 2000 US standards (151). Exclusion criteria included: history of brain injuries, major medical or psychiatric illness, current or past pregnancy, current and regular use of illicit drugs, greater than 5% body weight reduction

during the three months prior to recruitment, or regular use of medications known to impact eating behavior, weight, autonomic functioning, or endocrine functioning. In-person study visits were completed at the outpatient Pediatric Clinic at the National Institutes of Health Hatfield Clinical Research Center. The study procedures were approved by the *Eunice Kennedy Shriver* National Institute of Child Health and Human Development Institutional Review Board. Signed informed consent and assent were obtained from parents/guardians and children, respectively. All data were collected prior to the onset of the COVID-19 pandemic.

Measures

Ecological Momentary Assessment

Participants were given the option to complete a two-week EMA protocol while concurrently wearing an actigraphy watch, which was an adjunctive study to the primary protocol. Those who opted to complete the EMA protocol were provided with a smartphone and trained on how to complete the EMA surveys. The EMA surveys assessed multiple cognitions and behaviors, including food cravings and LOC-eating, and were collected using the Real Time Assessment in the Natural Environment system (227). Consistent with prior studies in youth, (72, 228) the EMA protocol contained three types of surveys: signal-, interval-, and event-contingent. Randomly, around stratified daily intervals (i.e., at approximately 11:10am, 1:50pm, 3:30pm, 5:40pm, and 8:20pm), participants received a notification instructing them to complete a survey (signal-contingent). On weekends, participants received five signal-prompted surveys between the hours of 11am and 9pm. On weekdays, participants only received three signal-prompted surveys between the hours of 3pm and 9pm, to minimize interference with school. Participants were also asked to complete a survey before bed and, on weekdays, immediately after the end of school (interval-contingent), and following every time they ate or drank a meal or snack (event-

contingent). The day after their in-person visit, participants practiced completing the EMA surveys. If compliance on this day was poor (completed $\leq 80\%$ of signal- and interval-contingent surveys) youth were contacted by study personnel to solve barriers to compliance and completed another practice day. When compliance on the practice day was satisfactory, participants began the 14-day protocol. Participants received additional compensation for completion of the EMA protocol.

Food Cravings. Food cravings were assessed in all interval and signal contingent surveys, regardless of whether an eating episode occurred. Items from the Intense Desire to Eat subscale of the Food Cravings Questionnaire-state version (229) were used to assess current cravings for food. A Likert type scale from 1= ‘not at all’ to 5= ‘extremely’ was used to measure the following items: “How strong is your desire to eat one or more specific foods?”; “How much do you crave one or more specific foods?”; “How strongly do you want to eat one or more specific foods?”; and “How much does your desire or craving to eat have power over you?” All items were averaged to obtain a composite score of food cravings. Internal consistency was excellent (Cronbach’s $\alpha = .96$).

LOC-Eating Severity. When an eating episode was reported, participants rated the degree to which they experienced LOC-eating during that meal/snack, using items adapted from the *Eating Disorder Examination* (230). Questions in the EMA surveys were similar to previous studies (72, 228) and included the following items: “How much did you lose control during this eating episode?”; “Did you feel that you could not keep yourself from eating?”; “Did you feel that you could not stop eating once you started?”; “During the eating episode you just finished, how much did you feel a sense of loss of control?”; “How upset or distressed are you about how much you just ate?”; and “How much did you feel driven to eat?”. Participants indicated their responses on

a Likert scale ranging from 1= ‘not at all’ to 5= ‘extremely’. All items were averaged to obtain a composite score of LOC-eating severity at each eating episode. Internal consistency was good (Cronbach’s $\alpha = .87$).

Sleep

Wrist-worn actigraphy was used to assess sleep behavior in youth for the 14 days following their in-person visit. Participants were given an ActiGraph GT3X+ activity monitor (ActiGraph, Pensacola, FL), which records 24-hour physical activity and sleep/wake measurements. In young adults, the GT3X+ is a valid and reliable device for detecting sleep/wake diurnal patterns and has good concordance with polysomnography (195). The GT3X+ has been used in previous studies of youth ages 8-18 years (143, 231). To be included in analyses, participants were required to have valid data from ≥ 3 weekday nights of sleep and ≥ 1 weekend night of sleep. Sleep data were downloaded from devices using Actilife software version 6.13.3 (ActiGraph, Pensacola, FL). The Sedeh sleep detection algorithm, which has been previously validated for children and adolescents, (194, 232) was applied to data to detect periods of sleep. Data were also visually inspected, and sleep periods were confirmed by cross-referencing self-reported sleep logs.

Facets of sleep examined included sleep duration, onset, midpoint, and waketime. Sleep duration was defined as the amount of time between sleep onset and wake time. Sleep onset time was defined as the first 5-minute period in which activity counts were all zero and the subsequent activity counts were determined to be sleep by the Sedeh algorithm. Sleep midpoint was defined as halfway between sleep onset and wake time. Waketime was similarly defined as the first 5-minute period in which activity counts were above zero and the subsequent activity was determined by the Sedeh algorithm to be awake time.

Race and Ethnicity

Race and ethnicity were reported by the child's parents.

Snoring

Presence or absence of participant's snoring/known sleep apnea was reported by parents during the physical examination.

Socioeconomic status

Socioeconomic status was determined by parents' reported educational and occupational status via the Hollingshead Two Factor Index of Socioeconomic Status. Possible scores range from 8-66 with higher scores indicating higher family socioeconomic status (233).

Anthropometrics

Fasting weight was measured to the nearest 0.1 kg using a calibrated scale. Height (to the nearest 0.1 cm) was measured in triplicate by stadiometer. Body mass index (BMI, kg/m²) was calculated and standard deviation BMI scores (BMI_z) were calculated following Center of Disease Control and Prevention growth standards for age and sex (151) and used to determine weight status. Given that BMI is known to inconsistently capture differences in adiposity (193), fat mass (kg) was also assessed directly by dual-energy x-ray absorptiometry (GE Healthcare, Madison WI).

Depressive symptoms

Depressive symptoms over the previous two weeks were assessed by the Children's Depressive Inventory- Second Edition (234) a self-report measure that demonstrates excellent reliability and validity among children and adolescents (235).

Statistical Analyses

Analyses were conducted in SPSS version 25 (236). EMA practice days were not included in analyses. EMA reports and measures of sleep were matched on the date the individual woke up. Such that if an individual went to bed on Sunday and woke up on Monday, characteristics of that night's sleep were matched with EMA surveys completed on Monday. For descriptive analyses, average food craving and LOC-eating scores were computed by aggregating ratings within persons. To account for variation in the number of EMA surveys reported each day, daily averages in sleep duration/timing were computed. These daily values were then aggregated within-person to obtain a single rating of average sleep duration/timing across the two-weeks. Sleep onset time demonstrated a significant linear increase across the study, such that participants fell asleep later over the course of the study. Therefore, sleep onset was detrended by regressing each participant's sleep onset on day in the study [e.g., day 3 of participation; (237)]. Then, weekly sleep onset was centered on the individual's intercept (sleep onset on the first day in the study) and daily sleep onset was centered on the individual's regression slope (predicted sleep onset). No other measures of sleep changed over the course of the study. Thus, weekly sleep duration, midpoint and waketime variables were mean-centered by subtracting the entire sample's average sleep duration/timing (grand means) from each individual's own average sleep duration/timing (individual means). Nightly shifts in sleep were mean centered by subtracting each participant's average sleep duration/timing (individual means) from their previous night's sleep duration/timing [a specific night's sleep duration/timing; (238)]. Thus, all weekly sleep variables represent the direction and degree to which a person's average sleep duration/timing deviated from the sample's average sleep duration/timing, and nightly sleep variables represent the direction and degree to which participants' last night's sleep duration/timing deviated from their average night's sleep duration/timing.

Two generalized linear mixed models (GLMMs) tested the contribution of each facet of sleep while adjusting for all other sleep variables. Outcome variables for the two models were food cravings and LOC-eating severity. In both models, participant ID number was entered as a random effect, as sleep and eating measurements were nested within persons. A standard variance components covariance structure was used for random effects. Distributions of food cravings and LOC-eating severity were positively skewed; therefore, models assumed a Gamma distribution and logit function.

Models were re-run adjusting for covariates that were selected a priori and included weekend vs. weekday sleep (1 = weekday, 0 = weekend day), fat mass (kg), height (cm), snoring [1 = presence, 0 = absence; (239)], sex (1 = Female, 0 = Male), socioeconomic status (240), age, race/ethnicity (1 = non-Hispanic white, 0 = other), and depressive symptoms (202, 203). All facets of sleep were included in analyses as continuous predictors, but to facilitate visualization of the results, facets of sleep were dichotomized as one standard deviation above and below the sample's mean.

RESULTS

Sample Characteristics

The opportunity to participate in the EMA protocol was first offered to youth in September 2016, approximately 18 months after protocol initiation. Of the 252 participants in the parent study to date, 169 participants provided usable actigraphy data. Of the 120 youth who elected to complete the optional EMA protocol, 68 concurrently wore an actigraphy watch. Twenty participants were excluded from analyses because they reported one or fewer eating episodes

during the EMA protocol, had less than 30% compliance with EMA surveys, or had sleep data from fewer than 3 weekdays and 2 weekends. Participants included in analyses had a higher percentage of females ($\chi^2 = 4.68, p = .03$), but did not differ from the parent study sample on percentage of non-Hispanic White individuals ($\chi^2 = 0.30, p = .58$), age ($t = -0.84, p = .41$), or BMIz ($t = -0.19, p = .85$). Data from 48 boys and girls (68.8% female; 12.8 ± 2.7 y; 14.6% Non-Hispanic Black or African American; 33.3% with overweight/obesity) were analyzed. See Table 1 for final sample characteristics.

On average, EMA compliance was adequate ($M = 73.97\%$, $SD = 17.78$). Craving analyses comprised 548 nights of sleep and 2,380 craving ratings. LOC-eating analyses included 518 nights of sleep and 1,073 eating episodes. Generally, food cravings and LOC-eating severity were low, but varied (food cravings: $M = 1.51, SD = 0.72$; LOC-eating: $M = 1.25, SD = 4.17$) and weekly/daily shifts in sleep were more variable (range 0.18 - 4.25 hours). Average food cravings, LOC-eating severity, and weekly and nightly shifts in all facets of sleep are reported in Table 1.

Associations of Sleep with Food Cravings

Weekly and nightly measures of sleep were not significantly related to food cravings when accounting for all facets of sleep, (est. β s = -0.13 - 0.15, $ps = .12 - .69$) or when adjusting for covariates (est. β s = -0.15 - 0.10, $ps = .08 - .93$).

Associations of Sleep with LOC-Eating Severity

When accounting for all facets of sleep, weekly sleep duration (est. $\beta = -0.31, p = .004$), sleep midpoint (est. $\beta = -0.47, p = .01$), and wake time (est. $\beta = 0.49, p = .01$) were associated with greater LOC-eating severity (Figure 1). Specifically, youth who had shorter average sleep duration, an earlier average sleep midpoint, and, on average, woke up later reported greater

LOC-eating severity during the EMA period (Figure 1). Shifts in daily facets of sleep were not significantly related to subsequent LOC-eating severity (est. β s = -0.01 - 0.02, p s = .11 - .92). Results remained significant after adjusting for all covariates. Results from unadjusted and fully adjusted models are displayed in Table 2.

DISCUSSION

The current study utilized an objective measurement of sleep and concurrent EMA to examine the associations among weekly and daily shifts in sleep duration/timing and food cravings and LOC-eating severity among a diverse sample of girls and boys of a broad age and weight range. When adjusting for the effects of various facets of sleep, sleep was not associated with food cravings. However, in both unadjusted and adjusted models, youth with shorter average sleep duration, earlier average sleep midpoint, and later average waketime during the two-weeks reported greater LOC-severity. Overall, these results demonstrate that when accounting for the entire sleep pattern, between-person differences in sleep duration and timing may be associated with eating-disordered behaviors.

When using objective measurement of sleep and accounting for the entire sleep pattern and relevant covariates, shifts in weekly and nightly sleep were not associated with food cravings. These findings are inconsistent with previous experimental studies among youth suggesting links between weekly sleep duration and bedtime and food cravings (158, 225). The differences in the findings between our study and previous studies could be due to a relatively small sample and analyses that adjusted for all facets of sleep. Our ability to detect an association may have been obscured due to our inclusion of youth of various ages and BMIs. Variability of sleep needs across development (128) and weight status could be explored in future studies as potential

moderators of the associations among sleep patterns and food cravings. Other characteristics of sleep that were not studied (e.g., sleep efficiency, sleep chronotype) may also be linked to food cravings. Additionally, acute changes in sleep duration and timing may influence other factors (e.g., hormones, physical activity, affect, executive function) that promote LOC-eating in youth. The mechanisms underlying the relationships between sleep patterns and LOC-eating are likely complex, therefore future studies among larger samples are needed to identify the moderators and mechanisms linking interdependent facets of sleep to LOC-eating.

We found that greater variation in sleep duration and timing during a two-week period is positively associated with LOC-eating, suggesting that greater shifts, or more frequent shifts, in sleep duration and timing across two weeks appear to be linked to LOC-eating severity. These findings are inconsistent with preliminary cross-sectional studies in youth at risk for type 2 diabetes and with overweight and obesity which have failed to detect links between sleep and LOC-eating (54, 183). However, extant studies only examined the independent effects of a single facet of sleep on drives to eat and LOC-eating (54, 183), and have not disentangled the impact of weekly and nightly shifts in sleep. Thus, differences are likely due to disparate statistical and methodological approaches. Future experimental studies are needed to elucidate the possible additive and interactive effects of nightly and weekly shifts in sleep duration/timing and their impact on factors that promote LOC-eating.

Study strengths include the use of actigraphy to examine several facets of sleep collected over two weeks. We also utilized EMA, which allowed for the examination of the associations between facets of sleep and both food cravings and LOC-eating severity in the natural environment. Additionally, EMA and sleep data were measured concurrently, which allowed for the examination of weekly and nightly shifts in sleep and food cravings and LOC-eating severity.

Examination of youth in generally good health enabled us to study factors associated with LOC-eating severity prior to the development of significant eating issues, thereby elucidating potential risk factors for the onset of obesity and related health comorbidities.

Study limitations include small average shifts in sleep and low average severity of food cravings and LOC-eating. Further, the craving measure used in the present study has been validated in samples of adults, but not yet in children or adolescents. Therefore, although the measure demonstrated good internal consistency, the low severity reports of food cravings may suggest the need for measures of food cravings that are adapted for and validated in youth. Moreover, it is unknown if continuous severity, rather than a specific cut-off score, is most appropriate for detecting LOC-eating in the natural environment among youth. It is possible that the present study identified “false positives,” such that some reports of LOC-eating may not have met a clinical threshold. There are no empirically determined cutoffs of LOC-eating severity in youth; therefore, we examined LOC-eating severity as a continuous measure to maintain statistical power and consistency with previous EMA studies in children and adolescents (72, 228). Future studies are needed to determine the clinical significance of LOC-eating severity and replicate findings from the current study in samples of youth enriched for LOC-eating. Nevertheless, detection of associations among sleep and severity of LOC-eating, even in a sample comprising healthy youths with low scores on this measure, supports sleep as a potentially modifiable risk factor for pediatric obesity and disordered eating behaviors.

CONCLUSION

Interactions among sleep duration and sleep timing may be important for the promotion of LOC-eating. We did not observe statistically significant associations among facets of sleep and

food cravings. Thus, LOC-eating may be a candidate mechanism linking sleep to excess weight gain. Longitudinal data are needed to provide clarification on how shifts in sleep duration and timing prospectively impact appetite, eating behaviors, and onset of obesity. Future studies that aim to determine the biological and psychological mechanisms that promote risk for developing obesity may further elucidate how sleep can be harnessed to maximize the efficiency of pediatric obesity prevention programs.

AUTHOR CONTRIBUTIONS: JAY and MTK designed the parent study in collaboration with NRK and SZY. RDC and SGE provided consultation on the design of the EMA protocol. MNP, SLR, ER, LMS, MEB, TNS, KGD, EAK, LMF, AZ, and SMB collected data. MNP managed the dataset and conducted statistical analyses. SLR, ER, and NRK cleaned and analyzed the sleep dataset. RDC supervised data analysis. MNP and SLR co-wrote the first draft of the manuscript. MTK, NAS, and JAY supervised the project and provided substantial editing of the manuscript. MTK, NAS, and JAY had primary responsibility for final content. All authors edited the manuscript and approved the final draft submitted.

For confirmation of publication submission, see Appendix B.

Table 1.

Participant Characteristics	<i>n</i>	%		
Sex				
Girls	33	68.8		
Boys	15	31.2		
Race				
White	24	50.0		
Black	7	14.6		
Asian	11	22.9		
Multiracial	4	8.3		
Unknown	2	4.2		
Ethnicity				
Non-Hispanic	41	85.4		
Hispanic	6	12.5		
Unknown	1	2.1		
Presence of Snoring	13	27.1		
Overweight or Obesity	16	33.3		
	<i>M</i>	<i>SD</i>	Minimum	Maximum
Age (years)	12.88	2.69	8.00	17.00
Socioeconomic Status ^a	2.23	1.06	1.00	5.00
Depressive Symptoms	7.29	5.66	0.00	24.00
EMA Ratings				
Food Cravings	1.51	0.72	1.00	4.86
LOC-eating Severity	1.25	4.17	1.00	3.49
Shifts in Facets of Sleep (hours)				
Sleep Duration Daily	-0.03	0.24	-1.09	0.65
Sleep Duration Weekly	0.09	0.90	-2.01	1.83
Sleep Onset Daily	-0.01	0.15	-0.28	0.43
Sleep Onset Weekly	0.23	0.81	-2.86	3.61
Midpoint Daily	-0.02	0.14	-0.33	0.32

Midpoint Weekly	0.07	1.08	-1.54	3.10
Waketime Daily	1.01	0.71	0.18	4.25
Waketime Weekly	0.12	1.02	-1.32	3.00

Table 1. Participant characteristics and descriptive statistics

BMIz, standardized body mass index; LOC-eating, loss-of-control eating; M, mean; SD, standard deviation. ^aThree participants did not have reports of socioeconomic status. Average facets of sleep were obtained from the 548 nights of sleep analyzed in the craving analyses. Daily averages and ranges in facets of sleep were obtained by aggregating daily shifts in sleep within persons. Weekly averages and ranges in facets of sleep were obtained by aggregating weekly shifts within days and persons. Negative sleep duration values represent shorter sleep duration (e.g., 6 hours vs. 8 hours). Negative sleep onset, midpoint, and waketime represent later timing (e.g., 3am vs. 12am).

Table 2.

Variable	Food Cravings					LOC-eating Severity				
	est. β	SE	t	p	95% CI	est. β	SE	t	p	95% CI
Unadjusted models										
Intercept	0.34	0.34	1.01	.32	-0.32 - 1.00	0.19	0.21	0.90	.37	-0.22 - 0.59
Sleep Duration Weekly	0.05	0.10	0.52	.61	-0.15 - 0.25	-0.31	0.11	-2.86	.004	-0.52 - -0.10
Sleep Duration Daily	0.01	0.01	0.77	.44	-0.01 - 0.02	-0.003	0.01	-0.42	.67	-0.02 - 0.01
Sleep Onset Weekly	-0.12	0.08	-1.54	.12	-0.27 - 0.03	0.03	0.04	0.60	.55	-0.06 - 0.11
Sleep Onset Daily	0.01	0.01	1.25	.21	-0.01 - 0.03	-0.01	0.01	-1.09	.27	-0.03 - 0.01
Midpoint Weekly	0.15	0.17	0.85	.40	-0.19 - 0.48	-0.47	0.18	-2.68	.01	-0.82 - -0.13
Midpoint Daily	0.00	0.01	-0.41	.69	-0.02 - 0.01	0.02	0.01	1.59	.11	-0.004 - 0.04
Waketime Weekly	-0.13	0.18	-0.71	.48	-0.49 - 0.23	0.49	0.18	2.75	.01	0.14 - 0.84
Waketime Daily	0.01	0.01	1.04	.30	-0.01 - 0.04	0.001	0.01	0.10	.92	-0.02 - 0.02
Fully adjusted models										
Intercept	0.34	0.90	0.38	.71	-1.43 - 2.11	0.38	0.49	0.77	.44	-0.58 - 1.33
Weekend/weekday Sleep	0.06	0.02	2.48	.01	0.01 - 0.10	0.002	0.02	0.11	.91	-0.03 - 0.03
Fat mass	-0.01	0.01	-1.22	.22	-0.02 - 0.01	-0.002	0.004	-0.53	.59	-0.01 - 0.01
Height	-0.002	0.01	-0.24	.81	-0.02 - 0.01	-0.01	0.004	-1.28	.20	-0.01 - 0.003
Snoring	0.15	0.19	0.79	.43	-0.22 - 0.52	-0.09	0.10	-0.89	.37	-0.29 - 0.11
Sex	0.01	0.15	0.10	.92	-0.27 - 0.30	0.09	0.09	1.04	.30	-0.08 - 0.26
SES	0.05	0.08	0.70	.49	-0.09 - 0.20	0.01	0.04	0.30	.77	-0.07 - 0.10

Age	0.01	0.04	0.12	.91	-0.07 - 0.08	0.04	0.02	1.78	.08	-0.004 - 0.08
Race/Ethnicity	0.08	0.15	0.57	.57	-0.20 - 0.37	0.15	0.08	1.76	.08	-0.02 - 0.31
Depressive Symptoms	0.02	0.01	1.48	.14	-0.01 - 0.04	-0.001	0.01	-0.07	.94	-0.02 - 0.01
Sleep Duration Weekly	0.07	0.13	0.59	.56	-0.17 - 0.32	-0.26	0.11	-2.40	.02	-0.47 - -0.05
Sleep Duration Daily	0.01	0.01	0.99	.32	-0.01 - 0.03	-0.003	0.01	-0.40	.69	-0.02 - 0.01
Sleep Onset Weekly	-0.15	0.08	-1.76	.08	-0.31 - 0.02	0.05	0.05	0.98	.33	-0.05 - 0.13
Sleep Onset Daily	0.02	0.01	1.76	.08	-0.002 - 0.04	-0.01	0.01	-0.99	.32	-0.03 - 0.01
Midpoint Weekly	0.10	0.21	0.46	.65	-0.31 - 0.50	-0.49	0.18	-2.78	.01	-0.84 - -0.14
Midpoint Daily	0.00	0.01	-0.09	.93	-0.02 - 0.02	0.02	0.01	1.73	.08	-0.002 - 0.04
Waketime Weekly	-0.10	0.22	-0.46	.65	-0.52 - 0.32	0.47	0.18	2.69	.01	0.13 - 0.82
Waketime Daily	0.02	0.02	1.25	.21	-0.01 - 0.05	-0.001	0.01	-0.07	.95	-0.02 - 0.02

Table 2. Unadjusted and adjusted GLMMs testing the associations among sleep, food cravings, and LOC-eating

est. β , coefficient; SE, standard error; CI, confidence interval; LOC-eating, loss-of-control eating, SES; social economic status.

Significant p-values are in bold type font

Figure 1.

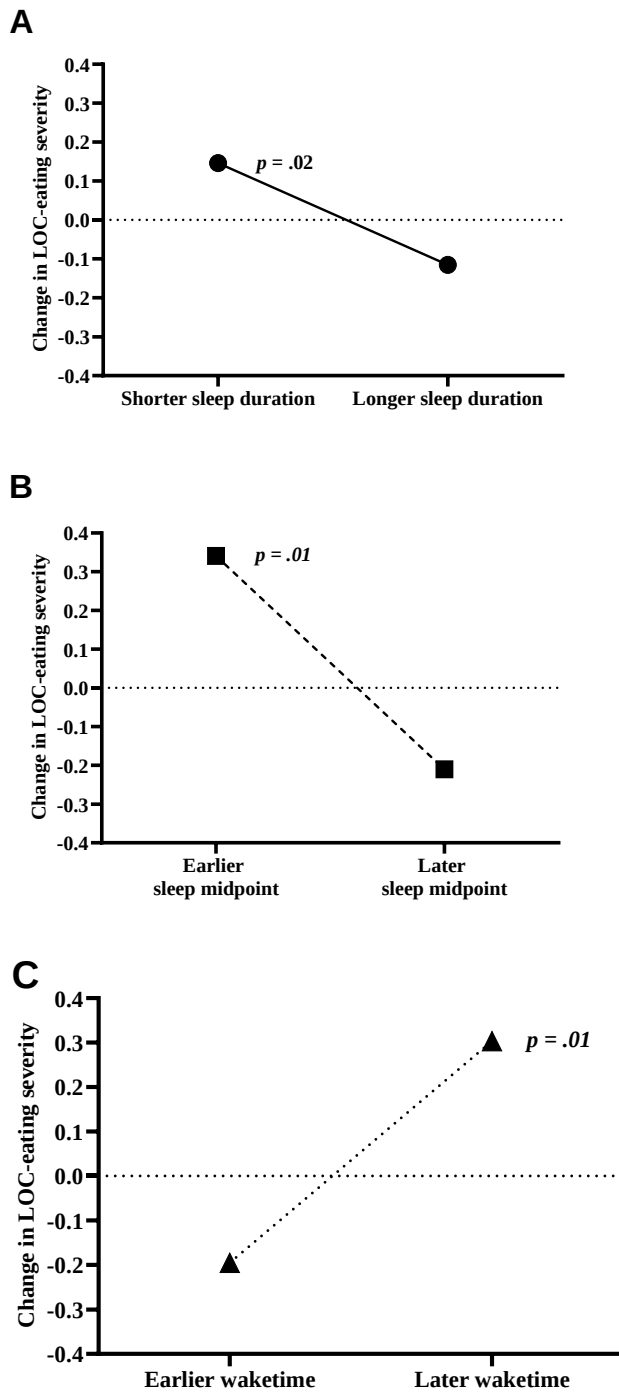


Figure 1. Facets of weekly sleep and LOC-eating severity **A:** Associations among LOC-eating severity and weekly sleep duration. **B:** Associations among LOC-eating severity and weekly sleep midpoint. **C:** Associations among LOC-eating severity and weekly waketime.

In all graphs, LOC-eating, loss-of-control eating. To facilitate visualization, facets of sleep were dichotomized as 1 SD below/above the average for each facet of sleep. Estimated Betas were used to predict LOC-eating severity at 1 SD below the mean, 1 SD above the mean, and at the mean for each facet of sleep. Predicted values were adjusted for weekend/weekday sleep, fat mass, height, snoring, socioeconomic status, age, race/ethnicity, and depressive symptoms. The Y-axis is centered on predicted LOC-eating severity at the average shift in each facet of sleep. Therefore, the difference between LOC-eating severity predicted for the average sleep duration, sleep midpoint, and waketime facets of sleep and 1 SD below/above means of each facet of sleep are presented.

CHAPTER 4: Study 3 Longitudinal Associations Between Facets of Sleep and Adiposity in Youth

ABSTRACT

Background: Beyond sleep duration, other facets of sleep, such as variability and timing, may be associated with obesity risk in youth. Yet, data are limited. **Objective:** Using a longitudinal design, we tested whether multiple facets of sleep were associated with fat mass gain over 1 year. **Methods:** A convenience sample of non-treatment seeking youth (8-17y) wore actigraphy monitors for 14 days. Average weekly sleep duration, within-subject sleep duration variability, weekend catch-up sleep, bed and waketime shift, social jet lag, bedtime, waketime, and sleep midpoint were calculated. The association of each facet of baseline sleep with 1-year fat mass (1yFM), adjusting for baseline fat mass (BLFM) and height, was examined. **Results:** One-hundred-thirty-seven youths (54.0% female; 12.5 ± 2.6 y; 28.4% Non-Hispanic Black or African American; BLFM: 15.3 ± 8.9 kg; 1yFM: 17.0 ± 10.0 kg; 28.5% with baseline overweight/obesity) were studied. Waketime ($p=.03$) and sleep midpoint ($p=.02$) were inversely associated with 1yFM, such that earlier waketime and midpoint were associated with higher 1yFM. No other facet of sleep was significantly associated with 1yFM ($ps>.09$). **Conclusion:** Using objective measures, youth with earlier waketimes and sleep midpoints had greater gains in fat mass. Additional research is needed to determine if sleep timing may be a modifiable target to prevent pediatric obesity.

Keywords: youth, sleep, adiposity, obesity, actigraphy, energy intake

INTRODUCTION

Research has demonstrated a consistent association between short nightly sleep duration and increased risk for higher body mass index (BMI), overweight, and obesity in youth (39, 146). A recent systematic review examining the associations between multiple dimensions of sleep and obesity in children found the majority of studies reported an inverse association between sleep duration (hours per night) and weight (40). This relationship has been observed both cross-sectionally, as well as prospectively (39, 40). For example, among children 6-12 years old across the weight spectrum, those who slept 7.5 hours or fewer per night had a three times greater risk of having obesity five years later than those who slept 9 hours or more each night (146). These data suggest that age-appropriate sufficient sleep may be particularly important for promoting healthy weight in youth.

However, the mechanisms linking sleep and obesity risk among youth are poorly understood. It is possible that the association between shorter sleep duration and high weight is at least partially due to increased energy intake and/or poor diet quality (41). Indeed, experimental studies have found that restricting nightly age-specific sleep recommendations is associated with increased energy intake in youth (158). Observational studies, using both objective (160) and self-report (55) measures of sleep, have also found an association between insufficient sleep duration and poorer diet quality, such as greater intake of added sugar, fats, and carbohydrates (55, 160). For example, one study found that among children and adolescents, those who slept less than 7 hours nightly consumed a higher percentage of their intake from carbohydrates at a buffet test meal than those who slept 7 hours or more (160), potentially increasing risk for excess weight gain. Yet, the data are not entirely consistent regarding the associations of sleep duration and food intake in youth (241, 242). For example, one study found that adolescents who reported

sleeping more than 8 hours per night were more likely to report eating highly palatable calorically dense foods [e.g. French fries and cake (242)]. Mixed results suggest that it may be important to use objective measures to examine other facets of sleep, in addition to sleep duration, in order to fully understand the associations between sleep, food intake, and weight.

Sleep variability may be particularly salient for obesity risk and food intake in youth (55, 166, 167, 171). There are several facets of sleep variability (Table 1), including variability of sleep duration [i.e., measures based on hours per night of sleep, such as weekend catch-up sleep, calculated as the difference between weekend sleep duration and weekday sleep duration (168)] and behavioral estimations of circadian rhythm shifts [i.e., metrics based on sleep timing such as social jet lag, calculated as the difference between weekend sleep midpoint and weekday sleep midpoint (143)]. Data generally support the role of sleep variability in energy intake, BMI, and adiposity beyond what is attributable to sleep duration alone (40, 41). For example, objectively measured within-subject sleep duration variability has been associated with greater energy intake, specifically from fats and carbohydrates and abdominal obesity (171). These studies did not, however, observe any association between total sleep duration and energy intake or abdominal obesity when adjusting for relevant covariates. Additionally, preliminary data in youth suggest that objectively measured (143) as well as parent-reported (176) social jet lag is associated with greater energy intake, higher BMIz, waist circumference, and fat mass. However, there is a relative paucity of data examining objectively measured sleep variability in children and adolescents.

Sleep timing is another facet of sleep that may play a role in obesity risk in youth. For instance, bedtime (i.e. the time one falls asleep) has been associated with weight in youth (40), with data generally supporting a relationship between later self- and parent-reported bedtime and

higher weight (178, 179). While waketime may also be important, few studies have examined the relationship between waketime and obesity risk in youth, with only one study finding a cross-sectional relationship between early waketime and greater odds of having obesity (243). It is also possible that the combination of bed and waketimes, or overall sleep timing pattern, may be particularly relevant for obesity risk in youth. A combination of self-reported later bed and wake times, as well as later sleep midpoint, have been associated with greater intake of sugar-sweetened beverages and nutrient-poor, energy-dense foods independent of sleep duration (181, 182). Although the mechanisms remain unclear, preliminary data indicate that, similar to sleep variability, sleep timing may be associated with circadian rhythm misalignment (i.e., sleep patterns that are incongruent with physiologically-driven circadian preference), which is known to be associated with obesogenic behaviors and high weight in children and adolescents (218). However, studies utilizing objectively measured sleep timing are limited in youth (40, 41).

The majority of studies examining sleep characteristics are cross-sectional, thereby precluding an understanding of the temporal relationships between facets of sleep, food intake, and obesity risk. Additionally, it is unknown which sleep facets may be most relevant to the development of high weight. Further, adiposity is known to be a better predictor of cardiometabolic health than BMI (244), and more data are needed examining the association between facets of sleep and body composition. Thus, longitudinal data examining different sleep facets are needed to fully understand the association between sleep, obesogenic behaviors, and adiposity gain in youth over time.

Therefore, we examined relationships between multiple facets of actigraphy-measured sleep (including average weekly sleep duration, within-subject sleep duration variability, weekend catch-up sleep, bed and waketime shift, social jet lag, bedtime, waketime, and sleep midpoint),

food intake at a laboratory test meal, and change in fat mass at 1-year follow-up among youth (8-17 years old). We hypothesized that shorter sleep duration, greater sleep variability, and later sleep timing would be associated with a greater increase in fat mass at 1-year follow-up. Further, we explored whether the association between these facets of sleep and change in fat mass would be mediated by total energy and macronutrient intake, as assessed by a laboratory test meal.

METHODS

Participants

Boys and girls (8-17 years old) in good general health across the weight spectrum were recruited to participate in the Children's Growth and Behavior Study [Clinical Trials Identifier: NCT02390765 (52, 160)], an ongoing prospective study designed to understand the relationships between eating behaviors and body weight in children. Youth were recruited through mailings to parents in the greater Washington, D.C. area and flyers posted at local public facilities (e.g., libraries) in communities near Bethesda, M.D. Additionally, flyers were distributed through local primary and secondary schools and parent listservs.

Youth were eligible if they were 8-17 years old, cognitively capable of completing study procedures, and had a BMI $\geq$ 5th percentile for age and sex according to the Centers for Disease Control and Prevention 2000 U.S. standards (151). Individuals were excluded if they had a history of brain injury, major medical or psychiatric illness, current pregnancy or history of pregnancy, current and regular use of illicit drugs, greater than 5% body weight reduction during the three months prior to screening, or regular use of medications known to impact eating

behavior, weight, autonomic functioning, or endocrine functioning. The study procedure was approved by the National Institutes of Health (NIH) Institutional Review Board.

Procedures

Following a telephone screen, potentially eligible families were seen at the NIH Hatfield Clinical Research Center for all visits. The baseline screening comprised two visits (Visits #1 and #2), and the 1-year follow-up was a single visit (Visit #3). The study's purposes, testing procedures, and possible study risks were reviewed in detail with families. Interested parents and youth provided written consent and assent, respectively. All data were collected prior to the COVID-19 pandemic. Participants underwent the following procedures and measures at each visit.

Visits #1 and #2

Anthropometric Measures. Fasting body weight was measured to the nearest 0.1 kg using a calibrated scale. Height (to the nearest 0.1 cm) was measured in triplicate by stadiometer and the average was used to calculate BMI_z, adjusting for age and sex according to CDC growth standards (151).

Body Composition. Kilograms of lean mass and total fat mass were measured via dual-energy x-ray absorptiometry (iDEXA system, GE Healthcare, Madison, WI). Percent fat mass was calculated from the values obtained.

Physical Examination. Medical history (including parent-report of child snoring), vital signs, and history of past or current medical and psychiatric illness were assessed by a pediatric endocrinologist or a nurse practitioner.

Children's Depression Inventory. Depressive symptoms were assessed using the Children's Depression Inventory - Second Edition (198). Respondents indicate to what degree they

experienced depressive symptoms during the prior weeks. Each item response ranges from 0-2 and total scores range between 0 and 52, with higher total scores indicating more depressive symptoms. This measure is a widely used and demonstrated adequate internal consistency for the present sample (Cronbach's $\alpha = .79$).

Socioeconomic Status. Socioeconomic status was determined by parents' reported educational and occupational status via the Hollingshead Two Factor Index of Socioeconomic Status. Possible scores range from 8-66 with higher scores indicating higher family socioeconomic status (233).

Standardized Breakfast Shake. At approximately 10:00 AM, participants consumed 21% of their estimated energy needs as a breakfast shake (17% protein, 16% fat, and 67% carbohydrate), prepared by the NIH metabolic kitchen staff.

Multi-Item Buffet Laboratory Test Meal. At approximately 12:30 PM, after completing rating scales on hunger and fullness, participants were introduced to the multi-item test meal (approximately 10,000 kcal; 12% protein, 32% fat, and 56% carbohydrate) with tape-recorded instructions to "Let yourself go and eat as much as you want" (43). Rating scales for hunger and fullness were repeated post-meal. The NIH metabolic kitchen staff weighed each item before and after the test meal from which energy and macronutrient consumption was determined. Energy and macronutrient content for each food item was determined according to U.S.D.A. Nutrient Database for Standard Reference and information supplied by food manufacturers.

Sleep. Wrist-worn actigraphy was used to assess sleep behavior in youth for approximately 14 days following their first screening visit. Participants were given an ActiGraph GT3X+ activity monitor (ActiGraph, Pensacola, FL), which is worn as a wristwatch and delivers 24-hour physical activity and sleep/wake measurements. In young adults, the GT3X+ is a valid and

reliable device for detecting sleep/wake diurnal patterns and has good concordance with polysomnography (195). The GT3X+ wrist actigraphy has also been used in youth ages 8-17 years (143, 245). To be included in analyses, participants were required to have valid data from ≥ 3 weekday nights of sleep and ≥ 1 weekend night of sleep. Sleep data were downloaded from devices using Actilife software version 6.13.3 (ActiGraph, Pensacola, FL). The Sedeh sleep detection algorithm, which has been previously validated for youth [e.g., (194)], was applied to data in order to detect periods of sleep. Data were visually inspected, and sleep periods were confirmed by cross-referencing self-reported sleep logs.

Average weekly sleep duration (hour/night) of all nights (Sunday-Saturday) was calculated. Additionally, weekend catch-up sleep was calculated by subtracting average hours per night of weekday (Sunday through Thursday nights) from average hours per night of weekend (Friday and Saturday nights) sleep (170). Further, within-subject sleep duration variability was calculated as the intra-subject standard deviation of average sleep duration across the data collection period (167, 171). Bedtime shift was calculated by taking the absolute value of average weekday sleep onset time minus average weekend sleep onset time. Similarly, waketime shift was calculated as the absolute value of average weekend waketime minus average weekday waketime (55). Based on sleep onset and waketimes, sleep midpoint was calculated as the time halfway between the start and end of the primary nightly sleep period (143). Social jet lag was then calculated as the absolute difference between mean sleep midpoint on weekend days minus weekdays (143).

Visit #3

Approximately one year after completing the screening visits, participants returned to the NIH and repeated the following procedures: anthropometric measures, (fasting weight and height), body composition (via dual-energy x-ray absorptiometry), and physical examination.

Data Analytic Plan

All analyses were performed with IBM SPSS Statistics version 27 (SPSS, INC., Chicago, IL). Data were screened for normality and arcsine square root transformations were conducted for percentages of macronutrient intake and percent fat mass to enhance normality (160). Within-subject sleep duration variability, baseline fat mass, fat mass at one year follow-up, and depressive symptoms were not normally distributed; therefore, scores were log base 10 transformed to achieve normality. Additionally, weekend catch-up sleep and social jet lag were not normally distributed, and log base 10 transformation did not achieve normality. Therefore, outliers were recoded to within three standard deviations from the mean which resulted in normal distribution (246).

Nine multiple linear regressions were conducted to examine the association of facets of sleep with fat mass at one year follow-up (1-year fat mass). Several covariates were considered for these models. Given the differences in academic and social demands throughout the year, time of year when sleep data were obtained (coded as four categorical seasons) was considered as a covariate for all models (143). Additionally, due to established links of depressive symptoms with sleep and weight status in children and adolescents (247), the total score on the Children's Depression Inventory- Second Edition was considered as a covariate for all models. Studies have also demonstrated links between socioeconomic status and poor sleep and weight status in youth (248); therefore, socioeconomic status was considered as a covariate for all models. Given

evidence suggesting that age and puberty status differentially affect sleep in youth (249), both variables were considered as covariates. However, age and puberty status were highly correlated ($r = .80$); therefore, age rather than puberty was considered in order to prioritize the continuous variable. Adiposity also develops differentially across childhood and adolescence by sex (250); therefore, sex (0= male, 1= female) was also considered. Additionally, given the links between high weight and obstructive sleep apnea (239), snoring (0 = no snoring, 1 = snoring present) was considered as a covariate in all models. Due to the potential interdependence between facets of sleep and total sleep duration, average weekly sleep duration was also considered in all models. The final covariate considered was race (0 = Other; 1 = Non-Hispanic Black or African American). In an effort to conserve power, models were all run twice: once with only those covariates significantly associated with 1-year fat mass at $p < .05$ (partially adjusted) and once with all aforementioned covariates (fully adjusted; data available upon request).

For facets of sleep that were significantly related to 1-year fat mass, exploratory mediation models were conducted using Hayes Process macro version 3.5 for SPSS (251). Models examined whether the association between facets of sleep and change in percent fat mass were mediated by energy and macronutrient content intake, specifically total energy intake (kcal), sugar intake (g), and percent intake from fat, carbohydrate, and protein (Figure 1). Significant mediation is demonstrated whenever the confidence interval of the indirect effect (ab) does not include 0. Exploratory analyses of energy intake data included baseline height (cm), lean mass (kg), percent fat mass (252), and days between visits as covariates. All tests were two-tailed, and differences were considered significant when $p < .05$.

RESULTS

Participants

One-hundred-thirty-seven youths (54.0% female; 12.5 ± 2.6 y; 28.4% Non-Hispanic Black or African American; baseline fat mass: 15.3 ± 8.9 kg; 1-year fat mass: 17.0 ± 10.0 kg; 28.5% with overweight/obesity at baseline) were studied (Table 2). On average, data were available for 13.7 (± 2.9) days of weekly sleep, 9.8 (± 2.2) days of weekday sleep, and 3.9 (± 1.0) days of weekend sleep. Characteristics of facets of sleep are described in Table 2.

Facets of Sleep and 1-Year Fat Mass

Results of partially adjusted analyses [adjusting for baseline fat mass (kg) and baseline height (cm) only] are presented in Table 3. No considered covariate, other than baseline fat mass, was statistically significant in any model ($ps > .08$). Adjusting for baseline fat mass and height, average weekly sleep duration was not significantly associated with 1-year fat mass [$F(2,137) = 420.53$, $\beta = -0.01$, $p = .44$, $R^2 = .91$]. Additionally, no facet of sleep variability [weekend catch-up sleep $F(2,137) = 419.41$, $\beta = -0.003$, $p = .60$, $R^2 = .91$; within-subject sleep duration variability $F(2,137) = 418.95$, $\beta = 0.02$, $p = .69$, $R^2 = .91$; bedtime shift $F(2,137) = 421.05$, $\beta = -0.01$, $p = .38$, $R^2 = .91$; waketime shift $F(2,137) = 418.65$, $\beta = -0.002$, $p = .79$, $R^2 = .91$; social jet lag $F(2,137) = 424.48$, $\beta = -0.01$, $p = .19$, $R^2 = .91$] nor bedtime [$F(2,137) = 428.71$, $\beta = -0.23$, $p = .09$, $R^2 = .91$] was significantly associated with 1-year fat mass. However, waketime [$F(2,137) = 435.12$, $\beta = -0.32$, $p = .03$, $R^2 = .91$] was negatively associated with 1-year fat mass such that waking up earlier was associated with greater fat mass at 1-year follow-up (Figure 2a). Likewise, sleep midpoint was inversely associated with 1-year fat mass [$F(2,137) = 438.64$, $\beta = -0.35$, $p = .02$, $R^2 = .91$], such that having an earlier sleep midpoint was associated with greater 1-year fat mass (Figure 2b).

Exploratory Mediation Analyses

Adjusting for baseline height (cm), lean mass (kg), percent fat mass, and days between visits, waketime and sleep midpoint were not associated with intake at the laboratory test meal [specifically from total energy intake (kcal), sugar intake (g), and percent intake from fat, carbohydrates, or protein; i.e., a path; $p_s > .18$]. Energy and macronutrient intake were also not associated with change in percent fat mass (i.e., b path; $p_s > .13$). Finally, energy and macronutrient intake did not significantly mediate the relationship between waketime or sleep midpoint and change in percent fat mass (Table 4).

DISCUSSION

The current study utilized objective measures of sleep, fat mass, and energy intake to examine the associations between facets of sleep and fat mass at 1-year follow-up among youth of a broad age and weight range. Average weekly sleep duration, sleep variability (weekend catch-up sleep, within-subject sleep duration variability, bed and waketime shift, and social jet lag), and bedtime were not significantly associated with fat mass at 1-year follow-up. However, earlier waketime and earlier sleep midpoint were associated with greater fat mass at 1-year follow-up, after adjusting for baseline fat mass and height. These results suggest that wake timing may be associated with greater gains in fat mass over time.

Null results between average weekly sleep duration, sleep variability and bedtime and 1-year fat mass are somewhat inconsistent with prior literature among youth (146, 166, 171, 178). This could be due to methodological differences, such as a somewhat smaller sample size of a wide age range. Alternatively, few studies among youth have utilized actigraphy to examine several nuanced facets of sleep over a 2-week period in relation to objectively measured adiposity (39, 40). It is possible that bias of self-report measures may link nonsignificant relationships, such

that using objective measures provided a more accurate representation of nuanced facets of sleep. Indeed, the majority of prior studies rely on child- or parent-report of sleep, with many using single item measures, such as “on an average night, how many hours of sleep do you get on weekdays and weekend days” (41). It is possible that asking participants to estimate a weekly average does not adequately capture nightly sleep differences.

Notably, both early waketime and sleep midpoint were associated with greater gains in fat mass. While contrary to our original hypotheses, these findings are consistent with one study in children that found earlier wake times to be associated with greater odds of having obesity (243). Additionally, Garipy et al. (2018) found that delayed school start time, and ostensibly later waketime, was associated with lower BMIz among youth 10-18 years old (253). Our results complement these previous data by highlighting that earlier waketime and sleep midpoint may be understudied components of broader sleep-timing, which may play a role in the promotion of high weight among youth. Emerging data suggest that sleep patterns that are incongruent with physiologically-driven circadian preference, which often shifts later beginning in middle childhood (254), are associated with higher BMIz, waist-to-height ratio, waist circumference, and fat mass (143). While preliminary data suggest that social jet lag may represent circadian rhythm misalignment (143), we did not observe a significant association between social jet lag and fat mass at 1-year follow-up. Given that in our sample, mean social jet lag was relatively low (1.1 ± 1.0 hr), it is possible that participants were not able to sleep according to their body’s circadian preference on the weekends (which would increase social jet lag) due to social demands (e.g., waking early to attend sporting events). Therefore, we may not have accurately captured circadian rhythm misalignment through this construct and it may instead be better represented by early average waketime and sleep midpoint time. Supporting this notion, a recent

study found the positive association between reported social jet lag and age-adjusted BMI percentiles to be highest among individuals with two or more hours of social jet lag (255). Data specifically examining waketime and sleep midpoint among youth are scarce. The current findings suggest these facets may serve as additional indicators of circadian rhythm misalignment and may therefore be important to examine in future research. Additional longitudinal data considering multiple facets of sleep timing as well as a measure of circadian preference are needed to better elucidate these associations.

Study strengths include use of actigraphy to examine several facets of sleep over two weeks, which provided an objective measure that more accurately quantifies sleep as compared to self-report assessments (256). Additionally, the current study included fat mass as measured by dual-energy x-ray absorptiometry, thereby minimizing the known limitations of using BMI (244). Further, we objectively measured food intake through a validated laboratory buffet test meal (43, 257). Finally, examination of youth in generally good health enabled us to study factors that may promote high weight before the development of overweight or obesity, thereby elucidating potential risk factors for the onset of pediatric obesity.

Study limitations include a relatively small sample size of a broad age strata which precluded us from further assessing varying sleep needs across the developmental trajectory (128). Similarly, our sample did not include enough youth with overweight or obesity to assess the influence of weight status on outcomes. Given that emerging data have found links between sleep timing and weight status (175), future studies should recruit samples enriched for children with overweight and obesity in order to better elucidate potential differences among groups. It is also possible that the 1-year timeframe was not sufficient to see meaningful changes in fat mass in our sample, especially given that adiposity develops differentially by sex across puberty (250),

suggesting future studies should include a longer follow-up period. Additionally, one test meal may not be an accurate reflection of real-world eating patterns (e.g., the foods provided may not reflect typical dietary intake) and therefore may have reduced ecological validity. However, this test meal has demonstrated validity in measuring differences in eating behaviors and macronutrient consumption in youth (43, 257). Future studies should consider ecologically valid measures of food intake such as ecological momentary assessments when assessing the association of facets of sleep and energy consumption. Finally, given that assessing the association between several facets of sleep and 1-year fat mass was not a primary aim of the original study, no *a priori* power estimates were made for the current study aims. This study also utilized nine regression analyses which could have inflated the chance of a type 1 error; thus, this study should be considered hypothesis-generating.

CONCLUSION

Sleep timing, specifically waketime and sleep midpoint, may be relevant for the development of excess fat gain in youth. No significant associations were detected between other facets of sleep, including average weekly sleep duration or sleep variability, and adiposity. Thus, sleep timing specifically may be a potentially modifiable factor in the development of excess weight gain in youth. More data are needed to provide clarification on how circadian preference impacts the association between sleep timing and onset of obesity.

AUTHOR CONTRIBUTIONS: JAY and MTK designed the parent study in collaboration with NRK and SZY. SLR, MNP, ER, LMS, MEB, TS, KGD, EAK, LMF, AZ, JY, and SMB collected data. SLR managed the dataset and conducted statistical analyses. SLR, ER, and NRK

cleaned and analyzed the sleep dataset. SLR wrote the first draft of the manuscript. MTK, NAS, and JAY supervised the project and provided substantial editing of the manuscript. MTK, NAS, and JAY had primary responsibility for final content. All authors edited the manuscript and approved the final draft submitted.

For confirmation of publication submission, see Appendix C.

Table 1. Sleep constructs

Construct	Definition/ Calculation
Average Weekly Sleep Duration	Average hours per night of sleep over one week.
Bedtime	The time one falls asleep; when using actigraphy data, defined as the first minute of a five-minute period of no actigraphy-detected period of activity (6).
Bedtime Shift	The absolute difference in weekend and weekday bedtime, calculated as average weekend bedtime minus average weekday bedtime (7).
Social Jet Lag	Calculated as the absolute difference between the midpoint of nightly sleep on the weekends and midpoint of sleep on weekdays (14).
Waketime	The time one awakens; when using actigraphy data, defined as the first minute of a five-minute period of actigraphy-detected after a period of inactivity (6).
Waketime Shift	The absolute difference in weekend and weekday waketime calculated as average weekend waketime minus average weekday waketime (7).
Weekday Sleep	Average hours per night of sleep over Sunday night through Thursday night sleep (13).
Weekend Catch-Up Sleep	The difference between average hours per night of weekend sleep and weekday sleep; calculated as average hours per night of weekend (Friday and Saturday nights) sleep minus average hours per night of weekday (Sunday through Thursday nights) sleep (13).
Weekend Sleep	Average hours per night of sleep over Friday and Saturday nights' sleep (6).
Within-Subject Sleep Duration Variability	Night-to-night variation in hours of sleep from the average; calculated in hours as the intra-subject standard deviation of average nightly sleep duration across a multi-day period (10).

Note. Table 1 had to be pasted as a picture to preserve formatting, therefore reference numbers do not accurately match those listed in the reference section. For accurate reference numbers, please see Table 1 in the Introduction of this dissertation (p. 16)

Table 2. Sample characteristics

Characteristic ^a	Total Sample (N = 137)	Total Sample Range
Age (years)	12.5 ± 2.6	8.0 – 17.0
Sex (% female)	54.0	
Race/Ethnicity (%)		
Hispanic or Latino	6.0	
Asian	12.7	
Black or African American	28.4	
Multiple Races	7.5	
White	47.0	
Unknown	0.7	
Baseline Fat Mass (kg)	15.3 ± 8.9	3.8 – 47.0
1-Year Fat Mass (kg)	17.0 ± 10.0	4.5 – 49.2
Overweight/Obesity (≥ 85 th percentile, %)	28.5	
BMIz Score	0.5 ± 1.0	-1.6 – 2.8
Average Weekly Sleep Duration (hr/night)	7.2 ± 0.9	4.6 – 8.8
Weekend Catch-Up Sleep (hr/night)	0.3 ± 1.0	-1.5 – 3.7
Within-Subject Sleep Duration Variability	1.2 ± 0.5	0.4 – 3.7
Bedtime Shift (hr)	0.9 ± 0.7	0.0 – 3.2
Waketime Shift (hr)	1.3 ± 1.1	0.0 – 5.3
Social Jet Lag (hr)	1.1 ± 1.0	0.0 – 7.3
Bedtime (hh:mm ±hr)	11:31PM ± 1.3	8:52PM- 3:21AM
Waketime (hh:mm ±hr)	7:53AM ± 1.1	5:02AM – 11:31AM
Sleep Midpoint (hh:mm ±hr)	3:46AM ± 1.1	1:40AM – 7:26AM

^aValues presented are mean ± standard deviation, unless otherwise noted as percentage.

Table 3. Associations of facets of sleep and fat mass at 1-year follow-up

	<i>F</i>	β	<i>p</i>	R ²
Partially Adjusted Models				
Average Weekly Sleep Duration	420.53	-0.01	.44	.91
Weekend Catch-Up Sleep	419.41	-0.003	.60	.91
Within-Subject Sleep Duration Variability	418.95	0.02	.69	.91
Bedtime Shift	421.05	-0.01	.38	.91
Waketime Shift	418.65	-0.002	.79	.91
Social Jet Lag	424.48	-0.01	.19	.91
Bedtime	428.71	-0.23	.09	.91
Waketime	435.12	-0.32	.03	.91
Sleep Midpoint	438.64	-0.35	.02	.91

Note. All models adjusted for baseline fat mass (kg) and baseline height (cm).

Table 4. Mediation analyses of the associations between facets of sleep and change in percent fat mass through energy and macronutrient intake

	<i>a</i> path			<i>b</i> path			Direct Effect (<i>c</i> ' path)			Indirect Effect (<i>ab</i> path)		
	β	<i>SE</i>	<i>p</i>	β	<i>SE</i>	<i>p</i>	β	<i>SE</i>	<i>p</i>	<i>ab</i>	<i>SE</i>	95% CI
Total Caloric Intake (kcal)												
Waketime	8.21	700.60	0.99	0.00	0.00	0.14	-0.22	0.11	0.05	0.00	0.02	-0.03 - 0.04
Sleep Midpoint	-55.74	720.25	0.94	0.00	0.00	0.13	-0.26	0.11	0.02	0.00	0.02	-0.04 - 0.05
Sugar Intake (g)												
Waketime	-36.43	49.45	0.46	0.00	0.00	0.28	-0.22	0.11	0.05	0.01	0.02	-0.01 - 0.05
Sleep Midpoint	-6.99	50.94	0.89	0.00	0.00	0.32	-0.26	0.11	0.03	0.00	0.02	-0.03 - 0.04
% Intake from Fat												
Waketime	0.26	0.15	0.08	0.07	0.06	0.32	-0.23	0.11	0.04	0.02	0.02	-0.02 - 0.07
Sleep Midpoint	0.18	0.16	0.26	0.06	0.06	0.36	-0.27	0.11	0.02	0.01	0.02	-0.03 - 0.05
% Intake from Carbohydrates												
Waketime	-0.21	0.16	0.18	-0.08	0.06	0.18	-0.23	0.12	0.04	0.02	0.02	-0.02 - 0.06
Sleep Midpoint	-0.08	0.16	0.62	-0.08	0.06	0.23	-0.26	0.11	0.02	0.01	0.02	-0.03 - 0.04
% Intake from Protein												
Waketime	-0.06	0.10	0.57	0.11	0.10	0.27	-0.21	0.11	0.06	-0.01	0.01	-0.04 - 0.02
Sleep Midpoint	-0.13	0.10	0.22	0.10	0.10	0.33	-0.24	0.11	0.03	-0.01	0.01	-0.05 - 0.01

Note. SE, standard error; CI, confidence interval. All models adjusted for baseline percent fat mass, baseline height (cm), baseline lean mass (kg), and days between visits. Three participants did not provide test meal data; therefore, this sample includes 134 participants.

Figure 1:

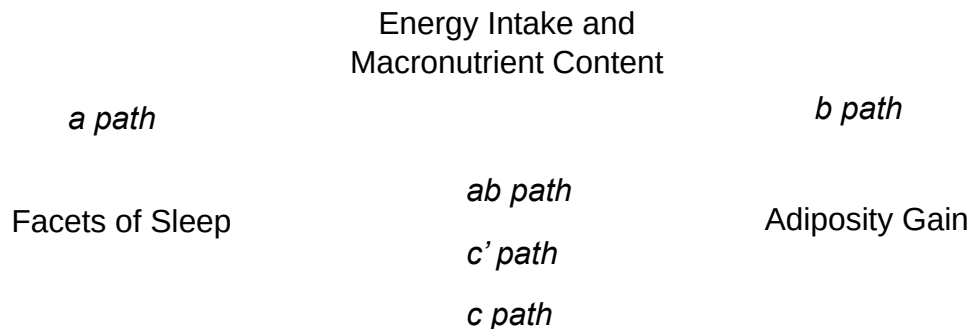
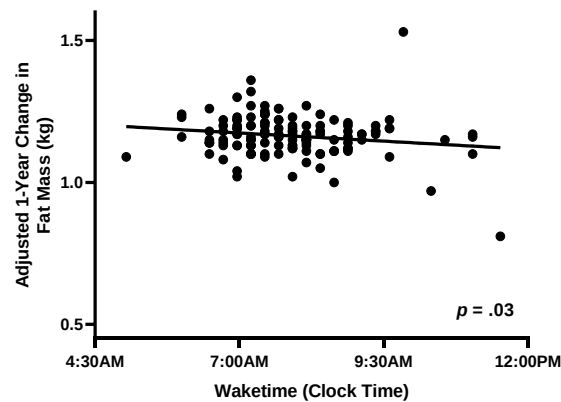


Figure 1. Mediation model of the association between facets of sleep, energy and macronutrient content intake, and adiposity gain. The *a* pathway represents the relationship between facets of sleep and energy and macronutrient content intake, the *b* pathway represents the relationship between energy and macronutrient content intake and adiposity gain, and the *c* pathway represents the total relationship between facets of sleep and adiposity gain. The *c* pathway represents the direct relationship between facets of sleep and adiposity gain, after adjusting for energy and macronutrient content intake. The *ab* pathway represents the unstandardized indirect relationship between facets of sleep and adiposity gain, through the energy and macronutrient content intake pathway. Models adjusted for baseline percent fat mass, baseline height (cm), baseline lean mass (kg), and days between visits.

Figure 2:
(a)



(b)

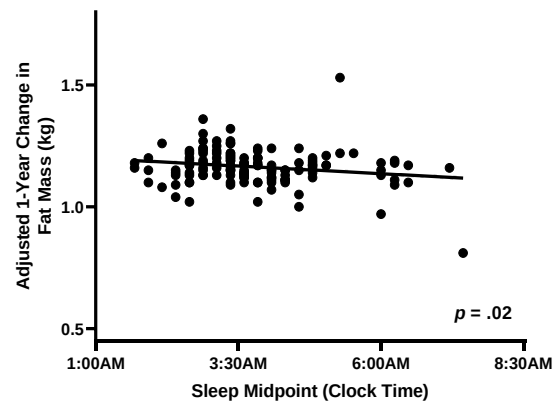


Figure 2. The association of waketime and sleep midpoint and 1-year change in fat mass.

Waketime (a; $p = .03$) and sleep midpoint (b; $p = .02$) were significantly inversely associated with fat mass at 1-year follow-up. Analyses adjusted for baseline fat mass (kg) and height (cm). For ease of visualization, raw wake and sleep midpoint times were used rather than adjusted wake and sleep midpoint times.

CHAPTER 5: General Discussion

This series of studies provide a better understanding how nuanced facets of sleep are associated with disinhibited eating, energy intake, and adiposity in youth by: 1) demonstrating a positive association between weekend catch-up sleep and EAH, 2) utilizing EMA data to elucidate temporal associations between shorter weekly sleep duration, earlier weekly sleep midpoint, and later weekly waketime and LOC-eating severity, and 3) finding longitudinal links between earlier wake and sleep midpoint time and adiposity gain (see table 1 for full list of results)

These studies have several strengths, including the use of 14 days of actigraphy data, with high compliance, to measure sleep, EMA to assess the acute and cumulative effects of sleep on disinhibited eating, the use of a laboratory test meal to assess energy intake, and objectively measured adiposity at baseline and a one-year follow-up. Further, these studies utilize a racially and ethnically diverse sample of healthy youth. Therefore, results may highlight early modifiable risk factors for high weight.

Given the novelty of these data and somewhat discrepant findings, experimental studies are needed to better understand how specific facets of sleep are related to aberrant eating behaviors and energy intake in youth. For an experimental trial could manipulate specific facets of sleep (e.g. weekend catch-up sleep), while holding others constant (e.g. sleep midpoint) and examine how changes in facets of sleep impact food cravings, LOC-eating, and energy intake in a controlled environment. Results of additional experimental and longitudinal data could better inform clinical intervention.

Clinical Implications

These studies suggest that targeting specific facets of sleep could be an effective and efficient means of reducing obesity risk in susceptible youth, as well as improving sleep patterns. For instance, among adults, sleep has been shown to be highly modifiable through short-term cognitive behavioral interventions (258-260). A recent meta-analysis suggested that as few as four face-to face meetings is sufficient to change sleep behavior (260). Additionally, there are preliminary data in youth to suggest that internet-delivered cognitive behavioral interventions for sleep are comparable to face-to-face interventions (261). For example, a recent randomized-controlled trial among adolescents evaluated the efficacy of cognitive-behavioral therapy for insomnia (CBT-I) in-person versus over the internet (261). Participants assigned to in-person treatment received six weekly sessions of CBT-I which comprised psychoeducation, sleep hygiene, stimulus control, sleep restriction, relaxation techniques, and cognitive restructuring. The group assigned to the internet-based therapy engaged with a guided self-help website and received written feedback from a sleep therapist. Results demonstrated that participants in both groups improved significantly on several facets of sleep compared with participants on the waiting list.

CBT has also been shown to be effective in reducing disinhibited eating among youth [CBT-E; (94)]. Interestingly, many of the skills for improving sleep are similar to those used for improving eating behavior. For example, one of the first focuses of CBT-I is to regularize sleeping schedule, and one of the first focuses of CBT-E is to regularize eating patterns (94, 262). Both require planful scheduling of behaviors and activities and could be addressed concurrently. Restructuring maladaptive cognitions is also a core skill in both CBT-I and CBT-E, and it may be possible for clinicians to address both eating and sleep- related thoughts in the

same session. For example, clinicians could encourage clients to challenge all or nothing thinking regarding both eating and sleep (e.g., “I already blew my diet, I may as well eat whatever I want” and “I cannot sleep tonight, I may as well get up and play video games). It may also be effective to incorporate mindfulness into any combined CBT-E/I protocol given that mindfulness has been shown effective at reducing EAH, LOC, and sleep problems (263-265). Mindfulness is described as focusing one’s attention, non-judgmentally, on the present moment which allows for recognition of internal states as well as external stimuli without the pressure to change them (266). Developing mindfulness skills may be particularly helpful in promoting the emotion- and self-regulation for both healthful eating and sleep patterns (265, 267-269). Thus, given that results of these studies support that weekend catch-up sleep, short weekly sleep duration, earlier weekly sleep midpoint, and later weekly waketime are associated with EAH and LOC-eating, future experimental studies could consider utilizing a brief course of CBT-I/E+ mindfulness that focuses on skills for improving both sleep and eating behavior as a cost-effective, efficient treatment strategy.

Indeed, a next step in this programmatic line of research could be a pilot study to test the feasibility of a CBT-I/E + mindfulness approach for the concurrent treatment of sleep and eating concerns in late adolescence/early adulthood. For instance, given the high rates of obesity (270), sleep problems (271, 272), and aberrant eating behaviors (273, 274) in college students, a study in the college counseling center might integrate CBT-I/E + mindfulness skills into a treatment group. After initial assessment, participants presenting with sleep and/or eating concerns could self-select to engage with a brief 4-session skills-based behavioral health group that would focus on 1) psychoeducation on healthy sleep, eating, and physical activity, 2) behavioral recommendations for regularizing sleep, eating, and physical activity patterns, 3) building

cognitive restructuring skills, and 4) learning mindfulness exercises. If data indicate this protocol to be acceptable and feasible, the next goal would be to conduct a randomized control trial comparing the efficacy of brief CBT-I/E + mindfulness group treatment to standard 8-12 session individual CBT-I or CBT-E treatments for the improvement of sleep, disinhibited eating behaviors, and weight.

It will be important for any future clinical studies to consider the potential moderating role of chronotype. Indeed, data among child, adolescent, and adult samples support that timing of energy intake and exercise are affected by chronotype and may impact treatment outcomes (275-278). For example, in adults the timing of physical activity is associated with regulation of an individual's overall circadian system (276, 279) such that physical activity has been shown to re-set circadian cycles and ameliorate the negative health effects of sleep disruption (280). Interestingly, however, preliminary data suggest that chronotype moderates these effects. For instance, a recent study in college undergraduate students (mean age of 19.6 years), found that the positive association between evening exercise and later bedtimes was more robust for individuals with morning chronotypes than for those with evening chronotypes (276). Studies also indicate that energy intake plays a role in regulating biological clocks, and preliminary data suggest that timing of food intake, or chrono-nutrition, is impacted by chronotype and related to energy consumption (278). For example, in a study among children and adolescents (grouped by age in 2-year intervals from 3-18), evening preference for energy intake increased (i.e., shifted to later in the day) progressively from ages 3/4-17/18 (281). Further, starting in adolescence (11/12 years), later preference for eating was associated with greater total daily energy intake such that individuals in the latest group had 11% higher energy intake than those in the earliest (281). Therefore, given the circadian nature of physical activity and energy intake and the moderating

role of chronotype, experimental data are needed to more fully understand how chronotype may be utilized in prevention and treatment of high weight in youth. For example, providers utilizing a CBT-E protocol for reduction of disinhibited eating and excess weight gain could assess and tailor behavioral recommendations based on chronotype [i.e., given that later exercise and eating is associated with later bedtime, especially for those with later chronotypes, providers could recommend individuals with evening chronotype shift their eating and exercise to the morning (276, 278, 281)].

Additionally, a better understanding of the links between nuanced facets of sleep and obesity risk in youth may inform clinical assessments and subsequent care. For instance, it may be important for clinicians to assess multiple facets of sleep rather than duration alone to best treat obesity-promoting sleep problems. While data support that structured interviews are necessary to capture nuanced facets of sleep in order to accurately diagnose sleep disorders (282, 283), it remains unclear how these assessments impact intervention. Therefore, future research should directly assess the impact of self-/parent-report versus more detailed clinical interview on treatment recommendations and sleep and weight outcomes. Further, additional experimental and prospective studies are needed to elucidate the specific facets of sleep most responsive to intervention and those most robustly linked with risk for aberrant eating behaviors and excess weight to help inform future clinical application. Finally, a better understanding of these links may have important public health implications (284). In addition to providing individualized behavioral recommendations, programs focusing on improving multiple facets of sleep could be initiated in schools and health clinics as a potentially effective obesity prevention strategy. Given the amount of time children typically spend in schools (either in person or virtually), teachers may be uniquely situated to provide informal interventions such as instructing about basic sleep

hygiene. Similarly, given that most children visit their pediatrician annually, healthcare workers may have an opportunity to assess multiple facets of sleep and offer brief recommendations for sleep improvement. Recommendations could include emphasizing the importance of consistent sleep schedules across weekday and weekend nights as well as sufficient total nightly sleep duration. Importantly, given the known differences in sleep behaviors based on race, ethnicity, and culture (139, 141), it will be important for future public health efforts to consider these factors when implementing sleep improvement interventions. Indeed, sleep improvement recommendations would likely be most effective if offered in cultural context. For example, sleep improvement recommendations offered at a well-child visit may consider whether co-sleeping is problematic or a normative cultural practice before advising parents to ask their children to sleep in independent beds and rooms.

Future Directions

These studies also have limitations that should be addressed in future research. The first two studies utilize cross-sectional data, and therefore no causal or mechanistic conclusions can be drawn. Additionally, given that the second study did not include dietary recall, analyses were limited to self-reported LOC- eating severity and food cravings, which could contribute to discrepant findings in studies 2 and 3. Study 2 found that later weekly waketime and earlier weekly sleep midpoint were associated with greater LOC-eating severity, yet contrary to expectations (42, 43), study 3 did not find an association between any facet of sleep and test meal intake. It is possible that a single test meal may not be an accurate reflection of real-world eating patterns and therefore may have reduced ecological validity. Indeed, it is likely rare that a participant encounters a nearly 10,000 calorie meal and is allowed to eat however much they want while alone. It is also possible that the foods presented to participants did not reflect typical

dietary and could include items not typically consumed. For example, it may not be common for a participant to have access to several chicken nuggets as well as Oreos, chips and salsa, and M & Ms. Therefore, it is possible that participants may have been more inclined to consume differently than they would in the natural environment. However, laboratory test meals avoid many of the confounding variables found in self-report measures, such as under-reporting (285) and this test meal has demonstrated validity in measuring differences in eating behaviors and macronutrient consumption in youth (43, 257). Further, while EMA data are collected in real-time, they are still based on subjective experience. Therefore, additional data are needed to more fully understand how test meal data map onto eating in the natural environment in youth. Additionally, future EMA studies should include dietary recall in order to gain a more robust understanding of how nightly sleep impacts next day eating behaviors. Finally, use of a continuous measure of LOC-eating severity in study 2 may contribute to discrepant findings. To date, it is unknown if continuous severity, rather than a specific cut-off score, is most appropriate for detecting LOC-eating in the natural environment among youth. It is possible that study 2 identified “false positives,” such that some reports of LOC-eating may not have met a clinical threshold and were rather a “prodromal” feeling out of control while eating. Because there are no empirically determined cut-offs of LOC-eating severity in youth, we chose to examine LOC-eating severity as a continuous measure to maintain statistical power and consistency with previous EMA studies in children and adolescents (72, 228). Future studies should aim to validate clinical cut-off scores for EMA surveys assessing LOC-eating.

There were also limitations in statistical approaches utilized throughout the three studies. First, the sample sizes were not large enough to assess differences based on age group (i.e., child versus adolescent), or weight status (i.e., healthy weight versus high weight). Due to the current

restrictions on data collection during the COVID-19 pandemic, the current samples are smaller than originally anticipated and additional data are needed to more fully understand differences in results by these groups. Another limitation of these studies is the use of multiple comparisons resulting in the potential of inflated type-1 error rates. However, many statisticians support this is an acceptable risk when conducting hypothesis-generating studies (286, 287). For example, Althouse (2015) argues that in confirmatory studies which may impact clinical care, it is important to more conservatively reduce the risk false positives by adjusting for multiple comparisons, however in exploratory studies this is less of a concern (287). Rothman (1990) additionally asserts that adjusting for multiple comparisons increases the risk of type-2 errors (or false negatives) and that it is preferable to not make adjustments for multiple comparisons in order to minimize errors in interpretation, especially when data are observations in nature (286). Therefore, given the novelty of these data and their exploratory nature, we chose to not adjust for multiple comparisons, but rather to clearly report the number of tests conducted as well as the unadjusted p-values for each test. As such, each study should be considered hypothesis-generating and results should be interpreted cautiously.

A final limitation of this series of studies is potentially missing data. Due to the nature of actigraphy collection (i.e., continuous data collection over 14 days in the natural environment) and the differences in analyses across the studies, we considered both between- and within-person missing data. To address between-person missing data, we first utilized previously recommended exclusion criteria for analysis of actigraphy data [i.e., requiring participants to provide approximately one-third of total possible data (288, 289)]. Additionally, we considered use of mean imputation for the reduction of missing data (290). Mean imputation utilizes a technique in which missing data are replaced by group means [e.g., individuals who provide

outcome variables but are missing sleep data could have the sleep data replaced with the group mean for each facet of sleep (290)]. However, mean imputation can add bias to the data (290), and some statisticians note that missing data are ignorable under a specific set of conditions, one of which is when data are missing at random (291). In each study, we found no demographic differences between participants missing data and those without which allowed for the assumption that the data were indeed missing at random (292) and therefore ignorable not requiring imputation (291). In order to avoid any potential bias of mean imputation and to ensure the parameters of the models had the same set of data and statistics (293), we chose to handle missing data through list-wise deletion (294).

Within-person missing is also a common problem in actigraphy data collection [e.g., participants taking their watches off during the study period (295)], which can be handled through imputation. However, given that these studies utilized weekly averages for facets of sleep, mean imputation would not have changed or enhanced the data and was therefore not used. Regarding the analyses examining the association between nightly shifts in sleep and eating behaviors in study 2, the combination of EMA and actigraphy is relatively novel and there are few recommendations for handling missing data. However, we utilized previously recommended approaches by requiring participants provide approximately one third of total possible data (292), using list-wise deletion for missing cases (294), and reporting compliance (183). Future studies could consider the use of mean imputation of sleep data to increase the number of EMA surveys available for analysis. There are additional environmental and parental factors that could impact sleep duration and timing as well as eating behaviors and obesity risk in youth that should be considered in future studies. For example, ecological variables such as community of origin or household make up may reduce sleep duration and/or alter sleep timing

as well as increase obesity risk. Indeed, data from objective and self-report measures support that children and adolescents from urban settings have shorter nightly later bed- and waketimes, and more evening chronotypes than individuals from rural settings due to increased environmental stimuli (e.g., neighborhood noise) and light pollution (296-300). Similarly, data generally support an association between living in an economically disadvantaged neighborhood and increased risk for obesity in youth due to decreased access to nutritional foods and physical activity resources [e.g. well-stocked grocery stores and playgrounds; (301)]. For example, one study found that among a nationally representative sample of 10-17 year old children and adolescents, odds of having overweight or obesity was significantly higher for individuals living in neighborhoods with poor housing, and no parks, sidewalks, or recreation centers (302). Parental factors may also play an important role in child and adolescent sleep behaviors and obesity risk. A recent large meta-analysis found that parental rule setting for bedtimes was associated with longer sleep duration among adolescents (303). The same meta-analysis found parent and adolescent sleep duration and timing to be similar, and that healthy parental sleep behavior was predictive of longer sleep duration among youth. Further, another large nationally representative study of 2,454 youth ages 5.5-19.1, found parental warmth to be associated with increased average reported sleep duration among children, and stricter household rules to be protective among adolescents (304). Parental style has also been associated with obesity risk. For example, a large longitudinal study that followed participants from 5-21 years, found less parental warmth and acceptance were associated with accelerated BMI trajectories (305). Further research is warranted to more fully understand how these sociocultural and parental variables may impact child and adolescent sleep behaviors longitudinally.

Additionally, there are endocrinological factors associated with sleep, eating behaviors, and weight status that could be considered in future studies. For example, disruptions in sleep timing may affect appetite hormone secretion as they are circadian in nature. One hormone that may play a key role is leptin. Leptin, secreted primarily from adipose tissue, helps regulate hunger and energy intake (306). Experimentally increasing leptin concentrations leads to decreased appetite and weight loss (306). Although several studies have found short objectively measured and reported sleep duration is linked to reductions in leptin, the data are somewhat inconsistent (156, 307-310). Leptin is diurnally patterned with peak concentrations at night and a nadir observed during the daytime (311). Ghrelin, an appetitive hormone secreted by peptides in the stomach and responsible for signaling fullness, is another appetitive hormone that is diurnal in nature (312) and may contribute to the link between sleep and obesity risk. While the data are somewhat mixed (313), studies support an inverse association between reported sleep duration and ghrelin levels (314, 315). Conceivably, given the diurnal pattern of leptin and ghrelin, circadian rhythm shift may be associated with overall decreased leptin and increased ghrelin, or mismatched receptor sensitivity, leading to greater energy intake (311, 316). It is possible that changes in circadian rhythm and appetitive hormone secretion could lead to preference in meal timing which could then impact energy consumption (317). However, there are no longitudinal data examining objectively measured facets of sleep and leptin and ghrelin in youth, and to date, no studies have explored meal chronotype (i.e., biological preference of meal timing). Other hormones may also be linked with sleep and obesity risk in youth. For instance, data from a recent meta-analysis support that short sleep duration is associated with the development of insulin resistance, a precursor for type-two diabetes, in youth (318). Considering energy or macronutrient intake at a single lab test meal did not significantly mediate the relationship

between facets of sleep and adiposity development in study 3, future analyses could consider the mediating role of leptin and ghrelin secretion and insulin resistance as other potential mechanisms. Further, this series of studies did not directly assess the self-control depletion model proposed as the theoretical paradigm explaining the links between insufficient and/or inconsistent sleep, disinhibited eating, food craving, and energy intake as it was outside the scope of the data. Future research should consider directly testing this model by collecting measures of self-control [e.g. the Self-Scoring Self-Control Scale (319, 320)] via EMA while participants concurrently report eating behaviors and wear actigraphy monitors. A better understanding of how sleep impacts self-control and eating behaviors could inform prevention and intervention efforts. Finally, given the known associations between sleep duration, disinhibited eating, and weight status (39, 58, 60, 130, 146, 148, 149), future research should recruit individuals at-risk for overweight and obesity in order to better elucidate potential differences among groups with and without high weight.

Conclusion

In conclusion, these studies aid in better understanding the complex associations between multiple facets of sleep and risk for overweight and obesity in youth. By conducting these three studies, we have addressed important gaps in the literature, specifically: 1) elucidating that weekend catch-up sleep is associated with greater EAH, 2) utilizing EMA data to observe temporal associations between shorter weekly sleep duration, earlier weekly sleep midpoint, and later weekly waketime and LOC-eating, 3) finding longitudinal associations between earlier waketime and sleep midpoint and adiposity gain. A better understanding of the links between multiple facets of sleep and risk factors for obesity and disinhibited eating in youth may aid in the development of prevention and intervention strategies for reducing obesity in youth.

Table 1. Findings across the three studies between facets of sleep, eating behaviors, and adiposity

Facet of sleep	Study 1	Study 2	Study 3
Average weekly sleep duration	• No association with EAH • No association with fat mass	• No association with food cravings • nightly shifts not associated with LOC-eating severity • shorter weekly sleep duration associated with greater LOC-eating severity	• No association with 1-year fat mass
Within-subject sleep duration variability	• Not assessed	• No association with food cravings • No association with LOC-eating severity	• No association with 1-year fat mass
Weekend catch-up sleep	• Associated with greater EAH • No association with fat mass	• Not assessed	• No association with 1-year fat mass
Bedtime shift	• Not assessed	• Not assessed	• No association with 1-year fat mass
Waketime shift	• Not assessed	• Not assessed	• No association with 1-year fat mass
Social jet lag	• Not assessed	• Not assessed	• No association with 1-year fat mass
Bedtime	• Not assessed	• No association with food cravings • No association with LOC-eating severity	• No association with 1-year fat mass
Waketime	• Not assessed	• No association with food cravings • Daily shifts not associated with LOC-eating severity • Later weekly waketime associated with greater LOC-eating severity	• Earlier waketime associated with greater gains in fat mass over 1 year
Sleep Midpoint	• Not assessed	• No association with food cravings • Daily shifts not associated with LOC-eating severity • Earlier sleep midpoint associated with greater LOC-eating severity	• Earlier sleep midpoint associated with greater gains in fat mass over 1 year

MEASURES

Children's Depression Inventory (CDI)

Remember, pick out the sentences that describes your feelings and ideas in the past two weeks.

1. _____ I am sad once in awhile
_____ I am sad many times
_____ I am sad all the time
2. _____ Nothing will ever work out for me
_____ I am not sure if things will work out for me
_____ Things will work out for me ok
3. _____ I do most things ok
_____ I do many things ok
_____ I do everything wrong
4. _____ I have fun in many things
_____ I have fun in some things
_____ Nothing is fun at all
5. _____ I am bad all the time
_____ I am bad many times
_____ I am bad once in awhile
6. _____ I think about bad things happening to me once in awhile
_____ I worry that bad things will happen to me
_____ I am sure that terrible things will happen to me
7. _____ I hate myself
_____ I do not like myself
_____ I like myself
8. _____ All bad things are my fault
_____ Many bad things are my fault
_____ Bad things are usually not my fault
9. _____ I do not think about killing myself
_____ I think about killing myself, but would not do it
_____ I want to kill myself
10. _____ I feel like crying everyday
_____ I feel like crying many days
_____ I feel like crying once in awhile

11. _____ Things bother me all the time
_____ Things bother me many of the times
_____ Things bother me once in awhile
12. _____ I like being with people
_____ I like being with people many times
_____ I do not want to be with people at all
13. _____ I can not make up my mind about things
_____ It is hard to make up my mind about things
_____ I make up my mind about things easily
14. _____ I look ok
_____ There are some bad things about my looks
_____ I look ugly
15. _____ I have to push myself all the time to do my schoolwork
_____ I have to push myself many times to do my schoolwork
_____ Doing schoolwork is not a big problem
16. _____ I have trouble sleeping every night
_____ I have trouble sleeping many nights
_____ I sleep pretty well
17. _____ I am tired once in awhile
_____ I am tired many times
_____ I am tired all the time
18. _____ Most days I do not feel like eating
_____ Many days I do not feel like eating
_____ I eat pretty well
19. _____ I do not worry about aches and pains
_____ I worry about aches and pains many times
_____ I worry about aches and pains all the time
20. _____ I do not feel alone
_____ I feel alone many times
_____ I feel alone all the time
21. _____ I never have fun at school
_____ I have fun at school only once in awhile
_____ I have fun in school many times

Eating in the Absence of Hunger (EAH) Questionnaire

Please let us know about your eating by putting an “X” and a number in the appropriate boxes.

EXAMPLE

How often do you...	Never	Rarely	Some-times	Often	Always	On average, how many days a week? (0-7)
...eat dinner at a restaurant?			X			3

Imagine that you are eating a meal or snack at home, work, or in a restaurant. Imagine that you eat enough of your meal so that you are no longer hungry.

	In this situation, how often do you keep eating because	Never	Rarely	Some-times	Often	Always	On average, how many days a week? (0-7)
1.	... the food looks, tastes or smells so good?						
2.	... others are still eating?						
3.	... you are feeling sad or depressed?						
4.	... you are feeling bored?						
5.	... you are feeling angry or frustrated?						
6.	... you are feeling tired?						
7.	... you are feeling anxious or nervous?						

Now imagine that you finished eating a meal or snack some time ago and you are not yet hungry.

	In this situation, how often do you start eating because...	Never	Rarely	Some-times	Often	Always	On average, how many days a week? (0-7)
8.	...you are near food that looks, tastes or smells so good?						
9.	... you are with other people who are eating?						
10.	... you are feeling sad or depressed?						
11.	... you are feeling bored?						
12.	... you are feeling angry or frustrated?						
13.	... you are feeling tired?						
14.	... you are feeling anxious or nervous						

EMA Items

Measure	Items
Food cravings	How strong is your desire to eat one or more specific foods? How much do you crave one or more specific foods? How strongly do you want to eat one or more specific foods? How much does your desire or craving to eat have power over you?
Loc-eating	How much did you lose control during this eating episode? Did you feel that you could not keep yourself from eating? Did you feel that you could not stop eating once you started? During the eating episode you just finished how much did you feel a sense of loss of control? How upset or distressed are you about how much you just ate? How much did you feel driven to eat?

Hollingshead Index

Parent's Name: _____

Your relationship to the child: _____

Child's Name: _____

What has been the mother's highest paying occupation ever? (Please list occupation, not actual salary amount)

What has been the father's highest paying occupation ever? (Please list occupation, not actual salary amount)

Please select the mother's highest level of education:

1. Graduate Professional Training
2. Standard College or University
3. Partial College Training
4. High School Graduate
5. Partial High School
6. Junior High School
7. Less than 7 years of education

Please select the father's highest level of education:

1. Graduate Professional Training
2. Standard College or University
3. Partial College Training
4. High School Graduate
5. Partial High School
6. Junior High School
7. Less than 7 years of education

Sleep Log



Sleep Diary: to be completed every day for 14 days

Start Date: ____/____/____ CGB#: _____

WEEK 1	Day 1	Day 2	Day 3	Day 4	Day 5	Day 6	Day 7
Day of Week: _____	_____	_____	_____	_____	_____	_____	_____
I went to bed last night at: _____ AM/PM	_____ AM/PM	_____ AM/PM	_____ AM/PM	_____ AM/PM	_____ AM/PM	_____ AM/PM	_____ AM/PM
I got out of bed this morning at: _____ AM/PM	_____ AM/PM	_____ AM/PM	_____ AM/PM	_____ AM/PM	_____ AM/PM	_____ AM/PM	_____ AM/PM
Notes: Write down anything that might have affected your sleep (like a storm, getting sick, someone waking you up).							
WEEK 2	Day 1	Day 2	Day 3	Day 4	Day 5	Day 6	Day 7
Day of Week: _____	_____	_____	_____	_____	_____	_____	_____
I went to bed last night at: _____ AM/PM	_____ AM/PM	_____ AM/PM	_____ AM/PM	_____ AM/PM	_____ AM/PM	_____ AM/PM	_____ AM/PM
I got out of bed this morning at: _____ AM/PM	_____ AM/PM	_____ AM/PM	_____ AM/PM	_____ AM/PM	_____ AM/PM	_____ AM/PM	_____ AM/PM
Notes: Write down anything that might have affected your sleep (like a storm, getting sick, someone waking you up).							



Article

Associations of Weekday and Weekend Sleep with Children's Reported Eating in the Absence of Hunger

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Abstract: Insufficient average sleep duration has been inconsistently associated with poor diet and obesity risks in youth. Inconsistencies in findings across studies may be due to a general failure to examine associations in weekday versus weekend sleep. We hypothesized that greater variations in weekday and weekend sleep duration would be associated with more disinhibited eating behaviors, which, in turn, might be involved in the relationship between sleep and weight. We, therefore, examined, among healthy, non-treatment seeking youth, the associations of average weekly, weekend, and weekday sleep duration with eating in the absence of hunger (EAH), a disinhibited eating behavior associated with disordered eating and obesity. Sleep was assessed via actigraphy for 14 days. Participants completed a self-report measure of EAH. Adiposity was measured by dual-energy X-ray absorptiometry. Linear regressions were used to test the associations of sleep duration with EAH and the associations of sleep duration and EAH, with fat mass. Among 123 participants (8–17 years, 52.0% female, and 30.9% with overweight), there was no significant association between average weekly sleep and EAH. Further, there was no significant association among average weekly sleep duration or EAH and fat mass. However, average weekday sleep was negatively associated, and average weekend sleep was positively associated, with EAH ($ps < 0.02$). Weekend “catch-up” sleep (the difference between weekend and weekday sleep) was positively associated with EAH ($p < 0.01$). Findings indicate that shorter weekday sleep and greater weekend “catch-up” sleep are associated with EAH, which may place youth at risk for the development of excess weight gain over time.

Keywords: eating in the absence of hunger; sleep; child and adolescent; fat mass; adiposity

1. Introduction

In the United States, over one third of children and adolescents currently have overweight and nearly 20% have obesity [1,2]. Given the negative long-term health impacts of overweight and obesity [3–5], the elucidation of early, modifiable risk factors for excess weight gain is necessary to enhance the effectiveness of obesity prevention and treatment in children and adolescents. Two such

APPENDIX B- Confirmation of Submission of Study 2 to *Pediatric Obesity*



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Re: Pediatric Obesity - Manuscript ID IJPO-2021-0204

8 messages

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To: Sarah Lemay-Russell <sarah.russell@usuhs.edu>

Mon, May 10, 2021 at 4:30 PM

Submitted! Please send the final files (manuscript and figure) to co-authors and submit for clearance. Thanks!!

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Thank you for submitting your manuscript to *Pediatric Obesity*.

Sincerely,
Pediatric Obesity Editorial Office

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APPENDIX C- Confirmation of Submission of Study 3 to *Obesity*



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Obesity - Manuscript ID 21-0414-OS

1 message

Obesity <onbehalf@manuscriptcentral.com>

Thu, May 13, 2021 at 5:17 PM

Reply-To: editorial@obesity.org

To: marian.tanofsky-kraff@usuhs.edu

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13-May-2021

Dear Dr. Tanofsky-Kraff:

Your manuscript titled "Longitudinal Associations Between Facets of Sleep and Adiposity in Youth" has been received by the journal *Obesity* via its online submission system.

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