Beyond Sleep Duration: Distinct Sleep Dimensions are Associated with Obesity in Children and Adolescent's

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Abstract

Objective—Short sleep duration is recognized as a significant risk factor in childhood obesity; however, the question as to how sleep contributes to the development of obesity remains largely unknown. The majority of pediatric studies have relied on sleep duration as the exclusive measure of sleep; this insular approach may be misleading given that sleep is a dynamic multidimensional construct beyond sleep duration, including sleep *disturbances* and *patterns*. While these sleep dimensions partly overlap, it is necessary to determine their independent relation with obesity, which in turn, may inform a more comprehensive understanding of putative pathophysiological mechanisms linking sleep and obesity. The aim of the present study was to investigate whether sleep dimensions including sleep duration, disturbances, and patterns were individually associated with obesity, independent of multiple covariates. The second objective was to examine whether sleep disturbances and patterns were independently associated with obesity, after adjusting for sleep duration.

Method—Participants included 240 healthy children and adolescents (M_{age}=12.60, SD=1.98; 45.8% females). Anthropometric measures included measured waist and hip circumference, body mass index Z-score and percent body fat. Subjective sleep measures included sleep duration, sleep disturbances, sleep quality, and sleep patterns from youth- and parental-report.

Results—Youth with larger adiposity and body composition measures reported poorer sleep quality (β_{avg}=−0.14, p<.01), more sleep disturbances (β_{avg}=0.13, p<.05), and showed a delayed sleep phase pattern (β_{avg}=0.15, p<.05), independent of age, sex, pubertal status, physical activity,

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screen time, socioeconomic status, and sleep duration. Shorter sleep duration was significantly associated with obesity; however, this link was attenuated after adjustment of covariates.

Conclusions—Results suggest sleep measures beyond duration may more precisely capture influences that drive the negative association between sleep and obesity, and thus, yield more robust associations. As such, future studies are needed to better understand how distinct sleep dimensions confer risk for childhood obesity.

Keywords

Over the past decade, sleep curtailment has been identified as a significant risk factor in the etiology and maintenance of childhood obesity. A solid and consistent association between short sleep duration and obesity, cross-sectionally as well as prospectively, has been reported even after controlling for a number of relevant covariates, such as age, sex, and other obesity-related behaviors (e.g., physical activity and snacking; refs. 1 & 2). Within pediatric populations, meta-analyses demonstrate a clear pattern suggesting short sleep duration is implicated in the etiology and maintenance of obesity (refs. 1 & 2). Despite these generally robust findings, some research suggests the relation between sleep duration and obesity in youth is attenuated after adjustment for covariates (e.g., refs. 3–5). Alas, the majority of pediatric studies have relied on "sleep duration" as the exclusive measure of sleep. This insular approach may be misleading given that sleep is a dynamic multidimensional construct beyond sleep duration, including sleep *disturbances* and *patterns*.

Sleep duration is derived based on the number of hours slept per night. Measures are used to capture average sleep duration ranging from one night to one month with actual or categorical estimates (e.g., >10hrs, 8–9hrs, 6–7hrs, <6 hrs). Sleep duration reflects factors such as biological and developmental sleep needs as well as contextual or lifestyle demands (e.g., school start times, extracurricular activities). However, length of time spent sleeping is directly influenced by other sleep dimensions. Indeed, sleep dimensions distinct from sleep duration are commonly used as diagnostic criteria for sleep and arousal disorders (ref. 6). Sleep disturbances refer to myriad dimensions including sleep fragmentation (e.g., arousals or awakenings), sleep disorders (e.g., sleep apnea, parasomnias), and poor sleep quality. Sleep *patterns* refer to circadian rhythm preference and sleep-wake schedules (e.g., sleep timing). These sleep dimensions may contribute to obesity through their impact on specific pathophysiological mechanisms.

Sleep physiology is composed of two major states (rapid eye movement, REM; non- rapid eye movement, NREM) and a cyclical alternating pattern or architecture. REM is characterized by an increase in heart rate, blood pressure, and respiration level compared to NREM sleep (refs. 7–8). NREM sleep is subdivided into four stages: stages 1 and 2 (light sleep) and stages 3 and 4 (deep or slow-wave-sleep). Slow wave sleep is characterized by increased parasympathetic and decreased sympathetic activation (i.e., reduced brain activity, heart rate, cardiac output, breathing, and blood pressure compared to wake and REM sleep) and coincides with the most prominent changes in the endocrine system (i.e., stimulating and inhibiting hormone secretion; refs. 7–9). Greater time spent in slow wave sleep is

considered to be more restorative than other sleep stages given its predominant parasympathetic drive (refs. 7–10). These sleep dimensions have been linked to adverse physiological processes (ref. 9).

Sleep disturbances are largely characterized by recurrent nocturnal awakenings defined by specific EEG events (i.e., micro-arousals) or behavioral markers (e.g., reported awakenings; ref. 11). Nocturnal awakenings cause abrupt physiological changes markedly increasing sympathetic and hypothalamic pituitary adrenal activity (e.g., increased respiration, heart rate, blood pressure, cortisol; refs. 11–13). In sleep apnea, respiration is repeatedly disrupted, resulting in frequent awakenings and micro-arousals that in turn affect sleep quality. Interestingly, these systems are postulated to contribute to an increased deposit of fat, particularly in the abdominal regions in both adults and youth (refs. 14–16).

Sleep patterns related to circadian rhythm preference are biologically governed by the suprachiasmatic nucleus and have a bi-directional relationship with metabolism (refs. 17–19). Circadian clock mutant mice absorb more carbohydrates and lipids than peptides (ref. 17), show increased levels of cholesterol, triglycerides, glucose, leptin, and have decreased insulin resistance (refs. 18–19). In turn, metabolic factors feedback onto the regulation of circadian timing, disturbing sleep architecture (time spent in sleep stages), wake schedules, as well as locomotor and feeding behaviors (refs. 18–19). Taken together, it is speculated that unique sleep dimensions may play critical, yet distinct roles beyond sleep duration (e.g., via pathophysiological mechanisms) in the development and maintenance of obesity.

Among adults, frequent sleep complaints related to initiating and maintaining continuous sleep are significantly associated with greater body mass index (BMI; refs. 20–21) and future weight gain, independent of sleep duration (ref. 22). Objectively measured sleep fragmentation (i.e., actigraphy) has been significantly associated with greater BMI (ref. 23), even in models adjusted for sleep apnea (ref. 24). Further, adults with erratic sleep patterns (e.g., shift work schedules) typically show greater indices of overweight, obesity, and metabolic syndrome compared to those with routine sleep patterns (ref. 25). Among pediatric populations, overweight youth evidence greater sleep disturbances (e.g., sleep-disordered breathing), more arousals (e.g., sleep fragmentation), sleep disorders (e.g., parasomnias), and longer time spent falling asleep (i.e., sleep latency), compared to healthy weight youth (ref. 26). Additionally, obese youth report more erratic sleep patterns and later bedtimes, independent of age, sex, and sleep duration (refs. 27–28).

The question as to *how* sleep contributes to the development of obesity remains largely unknown. The use of sleep duration as the predominant measure of sleep is problematic because it does not reflect the nuanced dimensions underlying sleep which themselves reflect unique physiological processes. In other words, it is unclear whether short sleep duration is directly linked to obesity or whether sleep disturbances (e.g., fragmentation, apnea, quality) and sleep patterns better explain the association. While these sleep dimensions partly overlap, it is necessary to determine their independent relation with obesity, which in turn, may inform a more comprehensive understanding of putative pathophysiological mechanisms linking sleep and obesity.

The aim of the present study was to investigate whether sleep dimensions including sleep duration, sleep disturbances, and sleep patterns were individually associated with obesity in a sample of healthy youth. The second objective was to examine whether sleep disturbances and sleep patterns were independently associated with obesity, after adjusting for sleep duration. First, it was hypothesized that sleep duration (i.e., school night, weekend night), sleep disturbances (e.g., poor sleep quality), and sleep patterns (e.g., weekend oversleep, weekend delay) would each be significantly associated with greater adiposity (i.e., waist and hip circumference) and body composition indices (i.e., BMI Z-score, percent body fat). Second, it was hypothesized that the associations between sleep disturbances and sleep patterns with obesity would remain significant, even after controlling for sleep duration.

Method

Participants

Youth (*N*=240) aged 8 to 17 years and their parents took part in the larger Healthy Heart Project, a longitudinal study that investigates childhood risk factors of cardiovascular disease. Youth were recruited using flyers posted around the community and bookmarks distributed by teachers in classrooms. Exclusionary criteria included serious psychopathology, medical conditions, or use of medications with known cardiovascular effects. The study was approved by the Concordia University Research Ethics Board (# UH2005-077-4). Informed consent and assent were obtained before the start of the study. Participants were financially compensated for their participation time.

Obesity Measures

While youth were dressed in light clothing, weight and height were taken by a pair of trained research assistants following standard procedures for anthropometric measurements (ref. 29). Height was measured using a standard stadiometer at maximal breath with shoes off. With a standard measuring tape, waist circumference was measured at the minimum circumference of the body between the lowest rib cage and the iliac crest; hip circumference was measured at the widest part of the body over the buttocks (i.e., greater trochanters). Height, waist, and hip circumference were measured in duplicate, to the nearest 0.2 cm; if they differed by more than 0.5 cm, a third measure was taken. The mean of the two closest measures was used in data analyses. Weight and percent body fat were measured with a bioelectrical impedance scale, which was routinely tested for accuracy and calibrated (Tanita Body Composition Analyzer BF-350). Bioelectrical impedance methods have demonstrated moderate agreement with the gold standard method for measuring body fat (Dual Energy X-ray Absorptiometry: *r*=0.40 to 0.69; ref. 30). Age-and sex-specific BMI Z-scores were determined using the growth charts published by the U.S. Centers for Disease Control and Prevention (ref. 31). All research assistants were trained on anthropometric measurement procedures prior to data collection; the principal investigator observed each assistant until reaching criterion consistently.

Sleep Measures

Sleep Duration—Sleep duration was obtained by youth self-report in response to: "During the past month, what time do you usually go to bed/wake up on school nights/weekends?"

Sleep duration for school nights and weekends was calculated as the difference between bed- and wake time. Youth self-report estimates of sleep duration have been previously shown to be correlated with objective measures of sleep duration (actigraphy: r=0.53; ref. 32).

Sleep Disturbances—The Children's Sleep Habits Questionnaire is a 43-item scale that screens for common sleep problems (e.g., parasomnias, sleep-disordered breathing) over a one week interval (ref. 33). On a 3-pt scale (rarely, sometimes, usually), parents reported the frequency of their child's sleep habits (e.g., "my child awakes more than once"). Items are summed to obtain a total score, with higher scores indicating considerable sleep disturbances. The scale has demonstrated test-retest reliability, validity, and internal consistency (ref. 33). Youth also rated their overall sleep quality (i.e., the subjective perception of how sleep is experienced) on a scale of 1 to 10 (1=very bad to 10=very good). This question is commonly used in studies assessing explicit perceptions about feeling rested and satisfied with sleep upon awakening (ref. 34).

Sleep Patterns—Sleep patterns were measured with self-reported bed- and wake time during school and weekends. These times were used to derive weekend oversleep (difference total sleep duration on weekends and school nights), weekend delay (difference between bedtime on weekend and school nights), and weekend awakening delay (difference between waketime on weekend and school days; refs. 35–36). Greater differences indicate more sleep, later bedtimes, and later wake times on weekends, respectively. Self-reported bed- and wake times are significantly correlated with objective measures of sleep (actigraphy: r=0.70) and wake-onset times (actigraphy: r=0.77; ref. 32).

Covariates

Pubertal Status—Using a validated self-report measure of puberty (Growing and Changing Questionnaire; ref. 37), youth indicated their adrenarche pubertal stage based on sex-specific illustrations corresponding to Tanner stages I-V of prepubertal to complete sexual maturity. Although physician assessment of pubertal development is considered the gold standard, self-report has demonstrated good reliability and validity among youth (r= 0.77 to 0.91; ref. 38).

Locomotor Activity—Using a modified version of the Self-Administered Physical Activity Checklist (SAPAC), youth reported the number of days in the past week during which they had engaged in nine moderate (e.g., jazz) and nine vigorous (e.g., swimming) physical activities during and outside of school hours for at least 15 min straight (ref. 39). The duration of moderate and vigorous activities in the week was calculated and used an index of physical activity. The SAPAC was previously validated with school-aged children, with a moderate criterion validity (r=0.57 to 0.75) and a 1-week test-retest reliability (r= 0.60; refs. 39–40). Youth also reported the number of hours spent each day watching TV, using the computer or internet, and playing videogames in the past week. The sum was used as an index of screen time. Self-report estimates of screen time demonstrate high test-retest reliability (ICC=0.98) and validity (ICC=0.50–0.80) among youth (ref. 41).

Socioeconomic Status—Socioeconomic status was based on parent-reported highest level of schooling achieved from nine categories that range from "no formal schooling" to "doctorate". The number of years in educational institutions were obtained for each schooling category and treated as ordinal. Parental education is considered a robust indicator of socioeconomic status.

Statistical Analysis

Data were analyzed with SPSS 20 software (SPSS, Inc., Chicago, IL), kept continuous to maximize statistical power, and were checked for normality and linearity. To examine the extent of overlap among the sleep dimensions, partial correlational analyses controlling for age were conducted. Next, to test the hypotheses, sequential regression analyses were modeled individually for each obesity measure (i.e., waist circumference, hip circumference, BMI Z-score, percent body fat). First, each sleep dimension (i.e., sleep duration, sleep disturbances, sleep quality, bed- and wake time, weekend oversleep, weekend delay, weekend awakening delay) was entered singularly to examine its unique effect on each obesity measure. Second, covariates were entered into these models (age, sex, pubertal status, physical activity, screen time, parental education). Third, sleep duration was added as an additional covariate to test the association between each sleep dimension with obesity, beyond sleep duration. Alpha levels were set to 0.05 (2-tailed), and Bonferroni corrections were applied as appropriate.

Results

Participant demographics are presented in Table 1. The majority of youth were male (54%), Caucasian (59%), and of normal weight status (70% BMI <85th percentile). Youth were 12.66 years (*SD*=2.03), in the intermediate stages of pubertal adrenarche (*M*=2.98, *SD*=1.59), were physically active for 144 min/week (*SD*=124.87), and had screen-time for 4.48 hours/day (*SD*=3.61; TV, computer/internet, videogames). Youth typically reported their sleep quality as "good", and slept about 9 hours on school nights, and 10 hours on weekend nights (see Table 2). The mean and standard deviations for sleep disturbances subscales were consistent with previous research (ref. 33). Finally, parents were largely university educated (*M*=15.7 yrs, *SD*=2.99).

After controlling for age, partial correlational analyses revealed small to moderate inter-correlations among the sleep dimensions (Table 3). Sleep duration on school and weekend nights was weakly correlated with sleep disturbances and weekend delay. Moderate to large correlations were observed for weekend oversleep and weekend awakening delay for weekend sleep duration only. However, these higher correlations are largely attributable to use of bed- and wake times to derive both of these measures. Sleep disturbances and sleep patterns also yielded low correlations. Collectively, these data suggest the sleep dimensions were largely unique.

In the first step of the sequential regression models, each sleep dimension was entered singularly (see Table 4: *univariate*). School night sleep duration was significantly associated with waist and hip circumference. The presence of sleep disturbances were significantly associated waist circumference and BMI, while sleep quality was significantly associated

with all obesity measures. Sleep patterns were associated with waist and hip circumference and BMI, but not percent body fat. These findings are largely consistent with previously reported findings.

Second, when covariates (age, sex, pubertal status, physical activity, screen time, parental education) were entered into the models (see Table 4: *multivariate*), sleep duration was no longer associated with any obesity measures. Sleep disturbances were significantly associated with all obesity measures. Sleep patterns reflected by overall bedtimes were most associated with obesity measures, while indicators of weekend sleep debt (e.g., oversleep, delay) were not related.

Third, when sleep duration was added as another covariate, analyses revealed nearly identical results (data not shown for parsimony). Namely, sleep disturbances remained significantly associated with adiposity and body composition indices. Similarly, sleep patterns of later school night bed- and wake times and later weekend bedtimes were still significantly associated with adiposity and body composition measures. Finally, to address the possible issue of non-linearity for age-related changes, polynomial curve estimations for all models were also tested; there were no significant improvements in model fit over the linear models (data not shown).

Discussion

The relation between sleep and obesity has been predominantly limited to the use of sleep duration. However, sleep duration is a broad measure that does not capture the unique aspects of other sleep dimensions. Importantly, different sleep dimensions may provide more precise information to better elucidate the relation between sleep and obesity due to their distinct underlying physiological mechanisms. The aim of the present study was to assess whether obesity measures are influenced by sleep disturbances and sleep patterns, beyond sleep duration.

Consistent with past research, school night sleep duration was significantly associated with central adiposity measures of obesity in youth (refs. 1 & 2), such that short sleep duration was significantly related with greater waist and hip circumference. However, this association was attenuated after the adjustment of multiple covariates; similar results have also been previously reported (refs. 3–5). Sleep disturbances, on the other hand, remained significantly associated with obesity in both unadjusted and adjusted models. Youth exhibiting frequent sleep disturbances had larger waist and hip circumferences as well as greater percent body fat. Beebe et al (ref. 26) found greater parent-reports of parasomnias, daytime sleepiness, and bedtime resistance among clinically obese youth, compared to healthy weight controls. In the present sample, pre-sleep anxiety and bedtime resistance were significantly related with obesity (data not shown). Similarly, frequent childhood sleep disturbances were associated with an almost two-fold increased risk of being overweight or obese at age 21 (ref. 42). Childhood sleep problems (e.g., pre-sleep anxiety), if untreated, may evolve into eventual sleep disorders (e.g., insomnia; ref. 43). Notably, there is physiological evidence showing markedly reduced parasympathetic and increased sympathetic activation during the

day and night among children diagnosed with sleep disorders, such as sleep-disordered breathing (ref. 44) and periodic leg movements (ref. 45).

Consistent with past studies, sleep quality was significantly associated with obesity (ref. 46). Youth reporting poor sleep quality had larger hip circumference, BMI Z-score, and percent body fat, after controlling for covariates. Poor sleep quality has been linked with an increased likelihood of having high blood pressure among adolescents (ref. 47) and greater waist circumference, BMI, percent body fat, insulin and glucose concentrations, and insulin resistance among adults (ref. 48). It has been postulated that estimates of sleep quality may be an indirect marker of restorative slow wave sleep (ref. 10). Compared to other sleep stages, slow wave sleep is particularly relevant for metabolic, hormonal, and neurophysiologic homeostasis (refs. 7, 9). During slow wave sleep, there is an overall dominance of parasympathetic activity and concomitant reductions in sympathetic activity, glucose use, and corticotropic release (i.e., cortisol and ACTH; refs. 7, 9), and is thus, posited to be an especially important sleep stage to obtain.

Experimental evidence indicates that selective deprivation of slow wave sleep may contribute to poor metabolism via the dysregulation of insulin, increase in cortisol secretion, and reduced secretion of growth hormone (ref. 49). Less time in slow wave sleep was associated with greater BMI, waist and hip circumference, percent body fat, and waist-to-hip ratio after controlling for physical activity, sleep efficiency, snoring, and sleep duration among older men (ref. 50) and middle-aged women (ref. 51).

Sleep patterns were also associated with obesity. Youth reporting later weekend bedtimes and exhibiting a delayed sleep phase on school days (i.e., later sleep and wake times) had greater adiposity and body composition measures of obesity, regardless of sleep duration. This may reflect the plausible accumulation of small differences in habitual sleep duration across long periods of time (rather than one single data point) that may contribute to obesity. Further, these results are consistent with evidence that metabolism may be more influenced by the *timing* of sleep (i.e., obtaining sleep at one's natural/ideal point in their circadian rhythm), rather than the actual *quantity* of sleep obtained (ref. 52). In fact, compared to youth who reported early bedtimes, youth with late bedtimes were 1.5 times more likely to have higher BMI values, 1.8 times more likely to be inactive, and almost 3 times more likely to have longer screen time (ref 28), suggesting that a delayed sleep phase may increase exposure to an obesogenic environment (i.e., missing breakfast, more sedentary behaviors, less physical activity). Although the present study found no significant association between irregular weekend sleep schedules and obesity in youth after adjusting for covariates, the observed direction is similar to those previously reported (ref. 28). Collectively, youth who exhibit more sleep disturbances, perceive their sleep quality as poor, and have a delayed sleep phase show significantly greater adiposity and body composition indices of obesity, irrespective of sleep duration.

Potential Underlying Physiological Mechanisms

The parasympathetic nervous system is dominant during sleep. Nocturnal awakenings or the transition to wakefulness are associated with concomitant increases in sympathetic (i.e., norepinephrine, sympathetic muscle nerve activity, blood pressure, heart rate) and

hypothalamic pituitary adrenal activity (i.e., cortisol; refs. 7, 9). Recurrent nocturnal awakenings considerably reduce sleep quality, increase daytime sleepiness, and alter sleep architecture, reducing restorative slow wave sleep and REM sleep (refs. 12–13). This reduction in slow wave sleep leads to decreases in growth hormone secretion, insulin sensitivity, and glucose effectiveness, as well as increases in morning cortisol and cholesterol levels; all irrespective of sleep duration (refs. 12–13). Additionally, sleep disruptions (sleep fragmentation) may modify the ability of appetite-regulating hormones (e.g., glucose, leptin, ghrelin) to accurately signal appropriate energy intake and expenditure, leading to increased food consumption, particularly for unhealthy foods (i.e., high-carbohydrate foods) and weight gain (ref. 7). It is postulated that the integrated activation of these systems contributes to increased metabolism of specific lipid-accumulating key enzymes, targeting lipolytically sensitive adipose tissue regions (refs. 14–15). Thus, the pathophysiological responses related to shallow fragmented sleep may promote a nocturnal stress response within the nervous and endocrine system, expediting the progression of obesity; examples of this include data from those diagnosed with sleep apnea (ref. 51).

Another plausible mechanism underlying the relation between sleep and obesity is the neurotransmitter hypocretin (Hcrt), also referred to as orexin. Hcrt is related to stress induced wakefulness, which leads to a hyperarousal state commonly characterized by increased fatigue, anxiety, and insomnia (ref. 52). It is also implicated in autonomic functions (i.e., increased arterial blood pressure, heart rate, and overall sympathetic activation) and has direct effects on the regulation of sleep-wake behaviors (ref. 52). Interestingly, Hcrt activation may also promote increased food intake, particularly palatable food, via activation of appetite regulating neurons (e.g., neuropeptide Y and agouti-related peptide) and inhibition of appetite suppressing neurons (e.g., proopiomelanocortin and cocaine-and amphetamine-related transcripts; refs. 53–55). Taken together, the integrated activation of the sympathetic and hypothalamic pituitary adrenal systems in combination with the Hcrt system are putative physiological mechanisms underlying the association between sleep disturbance and obesity (ref. 9).

Strengths and Limitations

One limitation of the current study was the cross-sectional design; the present findings cannot determine the causal or temporal nature of the relation between sleep and obesity. Other behavioral (e.g., dietary intake), psychological (e.g., stress), or environmental (e.g., neighborhood context) variables may be putative mediators underlying the association between sleep and obesity.

A second limitation involved the subjective measures of sleep indices, which precluded diagnosis of sleep disorders, such as sleep apnea or restless leg syndrome. Although, the subjective measures used in the present study are likely to be more related to habitual sleep duration than a single laboratory measure; and they have demonstrated reliability and validity in the literature when compared with objective sleep measures (ref. 56). Further, unlike previous studies, multiple indicators of obesity and sleep were measured. Sleep parameters were assessed by multiple informants, which provide a more comprehensive representation of sleep complaints, sleep patterns on both school and weekends, as well as

explicit ratings of sleep quality by youth. This was corroborated when sleep demographics (e.g., average sleep duration) within our sample were similar with those reported in past pediatric research (ref. 27). Similarly, the sample was representative of the general population, with prevalence rates of healthy-weight, overweight, and obese youth similar to population-based studies (ref. 57). This multi-method approach likely provides more robust measures of obesity and sleep in youth.

Future Studies and Conclusions

Given that obesity is a risk factor for multiple chronic diseases (e.g., cardiovascular disease), disability, and premature mortality (ref. 58), a better understanding of the role of sleep in the pathogenesis of obesity is of great importance. Ideally, it would be informative to conduct experimental sleep-restriction studies to establish causality; however, this has practical and ecologically-valid limitations. Longitudinal, prospective designs offer the possibility of examining the temporal nature of the complex relation between childhood obesity and sleep. Longitudinal designs with repeated obesity and sleep measures could elucidate how weight gain is influenced by distinct sleep dimensions (i.e., sleep disturbances and patterns). Specifically, investigation of the role of chronobiological (e.g., evening/morning preference), physiological (e.g., autonomic and metabolic activity profiles across specific sleep stages), contextual (e.g., school start-times, parental monitoring), behavioral (e.g., physical activity, screen time), and psychological (e.g., stress) factors across the life course is recommended to better delineate the nature and direction of the obesity-sleep relation (ref. 59). It may be advantageous to consider using ambulatory polysomnography, actigraphy, and daily sleep logs completed in the participant's usual environment, which would provide both objective and subjective information on behaviors (e.g., activity) sleep schedules (e.g., during school vs. summer vacations) and habits (e.g., watching TV in bed), as well as potentially identify additional risk and protective factors (e.g., sleep duration thresholds) previously omitted in studies (ref. 59). Given the dynamic nature of sleep and obesity during childhood, future research has the potential to identify important obesity prevention strategies such as lifestyle habits (e.g., sleep hygiene) that develop during the particularly vulnerable childhood life stage.

Overall, consistent with the literature, short sleep duration was associated with childhood obesity. The results suggest sleep measures beyond duration may more precisely capture influences that drive the negative association between sleep and obesity, and thus, yield more robust associations. Sleep disturbances and sleep delayed phase sleep pattern were independently associated with greater adiposity and body composition indices of obesity in youth, irrespective of obesity-related covariates and sleep duration.

Acknowledgments

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Table 1

Demographic Information of Youth

Variable	Mean (n)	SD (%)
Race		
Caucasian	(142)	(59.7)
Asian	(26)	(10.8)
Black	(20)	(8.3)
Latino	(10)	(4.2)
Other/Mixed	(42)	(17.5)
Anthropometric Measures		
Waist circumference (cm)	72.09	9.23
Hip circumference (cm)	90.59	10.47
Body Mass Index (%^{percentile})	63.21	26.78
Percent body fat (%)	21.92	9.26
Body weight status[1]		
Normal (5th – <85th percentile)	(169)	(70.4)
Overweight (85th – 95th percentile)	(43)	(17.9)
Obesity (95th percentile)	(25)	(10.4)
Underweight (<5th percentile)	(3)	(1.3)

Note. N=240.

[1]Distribution of age-and sex-specific body weight based on Centers for Disease Control values.

Table 2

Demographic Measures of Sleep Dimensions in Youth

Sleep Dimensions	Mean	SD
Sleep Duration		
School night (min)	545.65	62.94
Weekend night (min)	598.18	82.76
Sleep Disturbances[1]		
Sleep Behavior Problems[1] (range 0–132)	41.28	5.23
Bedtime Resistance (range 5–15)	6.94	1.46
Sleep Onset Latency (range 1–3)	1.30	0.58
Sleep Duration (range 3–9)	4.30	1.54
Sleep Anxiety (range 3–9)	4.46	0.88
Night Awakenings (range 3–6)	3.29	0.66
Parasomnias (range 6–17)	7.65	1.15
Sleep-Disordered Breathing (range 3–5)	3.14	0.42
Daytime Sleepiness (range 8–20)	12.42	3.11
Sleep Quality (1–10)	6.78	2.07
Sleep Patterns		
School Night		
Wake time (hr:min)	6:51	00:35
Bedtime (hr:min)	21:46	00:52
Weekend Night		
Wake time (hr:min)	9:05	1:34
Bedtime (hr:min)	23:07	1:22
Weekend Oversleep (min)	52	95
Weekend Delay (min)	80	66
Weekend Awakening (min)	133	100

Note. N=240. (hr : min = hour : minutes).

[1]Children's Sleep Habits Questionnaire.

Table 3

Partial Correlations among Sleep Dimensions, Controlling for Age

	Sleep Duration		Sleep Disturbances		Weekend Oversleep	Sleep Patterns	
	School Night	Weekend Night	Sleep Disturbances	Sleep Quality	Weekend Oversleep	Weekend Delay	Weekend Awakening Delay
School Night	-	0.23 ***	−0.15 **	0.18 **	−0.35 ***	−0.01	−0.10
Weekend Night		-	−0.17 **	−0.01	0.82 ***	−0.18 **	0.27 ***
Sleep Disturbances			-	−0.20 **	−0.07	0.05	−0.06
Sleep Quality				-	−0.12	−0.00	−0.08
Weekend Oversleep					-	−0.16 **	0.32 ***
Weekend Delay						-	0.38 ***
Weekend Awakening Delay							-

*
$p < .05$.

**
$p < .01$.

***
$p < .001$.

Table 4

Standardized Regression Coefficients of Sleep Dimensions Entered Simultaneously with Covariates

| | Waist Circumference | | Hip Circumference | | Body Mass Index[1] | | Percent Body Fat | |
| | | | | β | | | | |
Model	Univariate	Multivariate	Univariate	Multivariate	Univariate	Multivariate	Univariate	Multivariate
Sleep Duration								
School Night	**-0.22*****	-0.01	**-0.40*****	-0.08	0.03	-0.01	-0.05	0.02
Weekend Night	-0.05	-0.04	-0.02	-0.04	-0.08	-0.09	-0.02	-0.08
Sleep Disturbances								
Sleep Disturbances[2]	0.16**	0.15**	0.12†	0.11*	0.13*	0.12†	0.11	0.12*
Sleep Quality	-0.16**	-0.11	**-0.27*****	-0.15**	-0.13*	-0.15*	-0.16*	-0.12
Sleep Patterns								
School Bedtime	**0.24*****	0.09	**0.31*****	0.10†	0.10	0.14*	0.07	0.14*
School Wake time	0.06	0.13*	-0.06	0.06	0.15*	0.17**	0.10	0.16**
Weekend Bedtime	**0.25*****	0.14	**0.29*****	0.15**	0.18**	0.20**	0.08	0.13*
Weekend Wake time	0.14*	0.07	0.21**	0.10†	0.03	0.04	0.02	0.01
Weekend Oversleep	0.10	-0.04	**0.24*****	0.01	-0.09	-0.09	0.01	-0.10
Weekend Delay	0.11	0.10	0.12†	0.10†	0.15*	0.14*	0.04	0.05
Weekend Awakening Delay	0.11	0.03	0.22**	0.07	-0.02	-0.01	-0.01	-0.05

Note.

[1] Body mass index in Z-score.

[2] Children's Sleep Habits Questionnaire. β = standardized regression coefficient. Univariate model includes each sleep variable entered singularly. Multivariate model include each sleep variable entered simultaneously with covariates (age, sex, pubertal status, physical activity, screen time, socioeconomic status). Results remained consistent while adjusting for sleep duration (data not shown). Bolded values indicate statistical significance after Bonferonni corrections (p=0.0001).

* p < .05.

** p < .01.

*** p < .001.

† p < .09.