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Sleep, energy balance, and meal timing in school-aged children

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Abstract

Objective: To determine associations among objectively-measured nocturnal sleep time, bedtime and obesogenic behaviors, including dietary intake, timing of intake, and physical activity, in a diverse sample of school-aged children who presented for behavioral treatment to enhance sleep duration.

Methods: Eighty-seven children (8-11y, 66.7% female, zBMI: 0.86±1.0) who self-reported sleeping <9.5h/night were studied for one week using wrist actigraphy to estimate sleep; hip-worn accelerometers to measure physical activity; and 24h dietary recalls to capture dietary intake and

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meal timing. Pearson and Spearman's rho correlations and linear regressions controlling for age, gender and race were used for statistical analyses.

Results and Conclusion: Mean bedtime was 10:31 PM (\pm 58.2 minutes) and mean nocturnal sleep time was 7.7 hours (\pm 37.5 minutes). Although later bedtime was associated with shorter sleep time (r=-0.61, *p*<0.001), the two variables were differentially related to obesity risk factors. Later bedtime, but not sleep time, correlated with greater daily fat intake, later first meal of the day, and greater after-dinner intake (all *p*<0.05). Nocturnal sleep time, but not bedtime, correlated with zBMI (*p*=0.04). Both sleep time and later bedtime were associated with a later last meal of the day (all *p*<0.05). Findings remained consistent after controlling for demographic factors. In short-sleeping school-aged children, bedtime may be more predictive of dietary obesity risk factors whereas sleep duration may be more predictive of zBMI. Results suggest that health providers should consider both bedtime and sleep duration for reducing obesity risk in children.

Keywords

Pediatric; Obesity; Risk Factors; Sleep Timing; Sleep Duration

Parental reports show that 31% of 6-11 year olds sleep less than the recommended 9-11h/ night ^{1,2}. The fact that one-third of school-aged children in the United States may be habitually sleep deprived is problematic given the association between insufficient sleep and various adverse outcomes related to cognitive performance, mood and health^{3,4}. Increased risk for obesity has been consistently associated with habitual short sleep in children^{5–9}; a recent meta-analysis examining prospective associations between sleep duration and weight status in children and adolescents found that those who sleep for a shorter duration had twice the risk for being overweight/obese compared with those who sleep longer¹⁰. Given that children who are short sleepers are at increased risk for weight gain, it is important to examine relationships between sleep and obesogenic behaviors in this group as it provides important information on children who may present for intervention.

Recent observational studies have also focused on how later bedtime, independent from sleep duration, relates to weight gain and increased risk for obesity¹¹. Short sleep duration and later bedtime have been associated with dietary (e.g., increased daily caloric intake, lower fruit/vegetable consumption, greater soda and added sugar intake) and physical activity (e.g., more sedentary/light activity) risk factors for obesity in children^{12–20}. A common limitation of these studies, however, is the reliance on self/parent-report to assess sleep, which may not accurately reflect sleep duration and/or bedtime.

Growing evidence indicates that, in addition to the amount of calories consumed, the timing of caloric intake plays an important role in weight regulation²¹. Although a limited number of studies have examined meal timing and weight regulation in children, some evidence shows decreased morning caloric intake (e.g., breakfast skipping) and increased evening caloric intake are associated with overweight/obesity^{22–28}. Other evidence suggests that sleep may influence meal timing. For example, Thivel and colleagues found that children who consumed breakfast everyday had an earlier bedtime than those who did not, suggesting a relationship between bedtime and the timing of the first meal of the day¹⁷. In addition, a recent counterbalanced crossover experimental study found that compared to a week of sleep

extension (sleep was prolonged by 1.5h/night), children reported increased caloric intake during a week of sleep restriction (sleep was decreased by 1.5h/night) which was primarily due to extra calories consumed during the 3 additional evening hours that children were awake in the restriction condition²⁹. Finally, an observational study among adults showed that later sleep timing was associated with later meal timing (time of the last meal/snack of the day) and greater caloric intake after 8:00 PM³⁰. Therefore, sleep may impact obesity risk by affecting the timing of food intake. A study examining the relationship between objectively-measured sleep, focusing on both duration and timing, and obesity risk factors, including meal timing, in school-aged children is therefore needed.

To build upon previous studies examining sleep and obesity risk in children, the current study examined relationships among objectively-measured sleep duration and bed time and obesity risk factors, including meal timing, in a racially diverse sample of school-aged children (8-11 years) who presented for behavioral treatment to enhance sleep. This enabled focusing on a developmental period when children are at risk for both short sleep and obesity, yet is prior to adolescence (when circadian shifts may influence sleep patterns). To our knowledge, few of the previous studies have focused on examining the relationship between sleep behaviors and obesity risk factors in short-sleeping children. Given evidence that short-sleeping children are at increased risk for overweight/obesity, it is particularly important to understand how sleep affects eating and activity behaviors in this group. Furthermore, gaining a better understanding of associations between sleep, eating and physical activity behaviors in short-sleeping children can inform refinement of current intervention approaches aimed at improving sleep and health in this age group. We hypothesized that shorter sleep duration and later bedtime would be associated with higher zBMI (body mass index for age z-score), increased daily caloric intake, less time spent in moderate-to-vigorous physical activity, later meal timing, and greater evening caloric intake.

Materials and Methods

Participants

Children 8 to 11 years old who reported sleeping 9.5 hours per night most days of the week were eligible to participate in a randomized controlled trial to determine the relative efficacy of a brief behavioral intervention to enhance sleep³¹. Data presented in these analyses are from the baseline assessment which occurred prior to randomization. Parents had to be at least 18 years old, the child's primary caretaker, and able to understand and complete the study questionnaires in English. Children were ineligible if parents reported diagnosis of a medical or psychiatric condition (including a sleep disorder) or current medication that could affect sleep, eating behaviors, or weight status. Eighty-seven children (9.6±1.0 y, n=58 [66.7%] female) provided complete sleep data at the baseline assessment. The sample was diverse (Race: n=40 Black [46.0%], n=32 White [36.8%], n=14 Other [16.3%], n=1 Not reported; Ethnicity: n=75 Not Hispanic [86.2%], n=12 Hispanic [13.8%]) and included children with normal weight (n=47 [54.0%]) and overweight/obesity (n=39 [44.8%], n=1 Not measured).

Procedure

Families were recruited through direct mailings and posted advertisements. Trained staff screened families by phone and children who appeared eligible were scheduled for an orientation visit that included additional eligibility assessments. All procedures were approved by the Institutional Review Boards at the Miriam Hospital and Temple University. During the orientation visit, study aims and procedures were described, written consent and assent were obtained, and initial eligibility was determined. If eligible, families were provided with an Actiwatch 2 (Mini Mitter, A Respironics Company, Bend, OR) and an ActiGraph GT3X (ActiGraph, Pensacola, FL) for the child to wear and were instructed when, where, and how to wear the devices. Families were also provided with a 7-day sleep diary and instructed how to complete the diary as well as call-in to the study voicemail box to report sleep and wake times twice daily for one week. Families were contacted on 3 random days (two weekdays, 1 weekend day) during this week by phone to complete a 24-h dietary recall. All families were instructed to continue with their child's typical sleep schedule for this week. At the end of the week, families returned to the Center and research staff measured the child's height and weight. Actiwatch 2, sleep diary and call-in data were reviewed with the family to confirm sleep-wake periods³² and ActiGraph GT3X data were reviewed for wear time.

Measures

Sleep.—Actigraphy is a widely used, reliable and valid device for measuring sleep, which monitors motor activity to obtain continuous recordings of sleep-wake states^{32,33}. Actiwatch 2, a watch-size monitor worn on the wrist, was used with standard procedures (children wore the device on their non-dominant wrist across the 24-hour period, medium sensitivity threshold)^{32,34,35}. Actiwatch 2s were configured to store data in 1-minute epochs and 7 days of data were gathered during each assessment. Epochs were scored as sleep or wake by Actiware 5.0 software using an algorithm that interprets activity counts during a single epoch by activity produced in the surrounding 2-minute time period³⁶. In comparison to polysomnography, the sleep-wake algorithm is excellent at detecting sleep (sensitivity = .93)³⁴. Sleep diaries were completed by the child and parent; variables included bedtime, night wakings, wake up time, daytime naps, caffeine/medication use, and illness (e.g., colds). Diary information was used to verify and score Actiwatch 2 data³⁷. The primary variables of interest for the current study were weekly average bedtime (actigraph-estimated sleep onset) and weekly average total sleep time (minutes of Actiwatch 2 -scored sleep during the Actiwatch 2 -estimated sleep period).

Physical Activity.—Physical activity was measured using an ActiGraph GT3x, a valid objective assessment of physical activity in children^{38–40}. Families were instructed that the device should be worn on the child's right hip during all waking hours (except during activities where the device would become wet). The ActiGraph GT3x has a dynamic measurement range of +/-6 g's and the acceleration is digitized using a 12-bit analog to digital converter. Data are sampled at a user selected rate between 30 and 100 Hz (30 Hz was selected for the current study) that are low-pass filtered at a proprietary cutoff and stored and downloaded as raw acceleration. Time spent in moderate to vigorous physical activity (MVPA) was calculated using validated child-specific equations^{38,41}. For inclusion

in analyses, children had to have worn the accelerometer a minimum of 4 days for a minimum of 8 waking hours/day (n=86 (99%) children met this criteria). The variable of interest for the current study was weekly average time spent in MVPA (minutes); weekly average wear time (minutes) was entered as a covariate to account for individual differences in wear time.

Dietary Intake.—Three 24h dietary recalls (2 weekdays, 1 weekend day) during the 1week baseline assessment period using multiple pass methodology were used to measure dietary intake. Staff at the Cincinnati Center for Nutritional Research and Analysis (CCNRA) completed the recalls over the phone with the child and parent together (to enable capture of food intake when the child was not in the presence of his/her parent) using a laptop computer with the Nutrition Data System Software (NDS) developed by the Nutrition Coordinating Center at the University of Minnesota^{42–44}. Variables of interest included 3day average daily caloric intake and 3-day average daily percentages of calories from protein, carbohydrate and fat. N=84 (97%) children completed all three dietary recalls, the remaining three participants only completed 2 dietary recalls so data for those children reflects a 2-day average.

Meal information for each eating episode, including the time and label (provided by participants, i.e., breakfast, lunch, snack, dinner, other), was also collected during each dietary recall to provide caloric and macronutrient content for each meal/snack. Meal timing was operationalized as the 3-day average time of the first eating episode of the day and the 3-day average time of the last eating episode of the day, based on previous findings linking both morning^{23,25–27} and evening^{22,24,28} intake with health outcomes in children. An eating episode was defined as any amount of calories consumed via food or beverage. We also examined 3-day average caloric and macronutrient (kcal from protein, carbohydrate, fat) intake after the "dinner" eating episode, as previous studies in adults have shown that sleep patterns may be particularly important for caloric intake and fat intake during evening/late-night hours^{45–48}. Macronutrient intake after dinner was measured in terms of calories consumed (rather than as the percentage of total after-dinner intake) because a number of participants (n=24) did not consume any calories after dinner, thus preventing the percent calculation.

Anthropometrics.—Weight and height were measured in duplicate by trained research staff. Child weight was obtained on a digital scale and height was measured using a wall-mounted stadiometer. All measurements were obtained with children dressed in light clothes and without shoes. zBMI (BMI-for-age z-score) was calculated using CDC age and gender-specific population norms⁴⁹.

Statistical analysis.—Both sleep variables and zBMI were normally distributed whereas physical activity, daily and after-dinner intake, and meal timing variables were not. Therefore, Pearson and Spearman's rho (for non-normally distributed variables) correlations were used to assess associations between sleep, energy balance, and meal timing. Preliminary analyses revealed differences in sleep, energy balance, and meal timing variables between demographic groups (age, gender, and race). Therefore, general linear regression models, controlling for age, gender and race, were also run to control for potential

confounders. Statistical analyses were performed with IBM SPSS Statistics for Windows (version 20.0; IBM).

Results

Later bedtime was associated with shorter total sleep time (r=-0.61, p<0.001, see Table 1 for baseline sleep information). However, the two variables were differentially associated with energy balance measures. Later bedtime, but not total sleep time, correlated with less daily carbohydrate (% kcal; r_s=-0.25, p=0.022) and greater daily fat (% kcal; r_s=0.30, p=0.005) intake. Findings remained consistent after controlling for age, gender and race (bedtime and carbohydrate intake: B=-0.02, p=0.05; bedtime and fat intake: B=0.02, p=0.019). Shorter total sleep time, but not bedtime, correlated with higher zBMI (r=-0.22, p=0.04). Again, this finding remained after controlling for demographic variables (B=-0.008, p=0.017). Neither sleep variable was associated with time spent in moderate-to-vigorous physical activity, daily caloric intake or protein intake.

When examining relationships between sleep and meal timing, we observed that shorter total sleep time ($r_s=-0.25$, p=0.02) and later bedtime ($r_s=0.48$, p<0.001), correlated with a later last meal of the day; findings remained consistent after controlling for age, gender and race (total sleep time and last meal: B=-0.46, p=0.023; bedtime and last meal: B=0.57, p<0.001). Later bedtime, but not total sleep time, correlated with a later first meal of the day ($r_s=0.61$, p<0.001). Later bedtime was also marginally associated with more calories consumed after the dinner meal ($r_s=0.20$, p=0.063; however, this did not reach statistical significance) and specifically with more after-dinner calories from protein ($r_s=0.21$, p=0.048) and fat ($r_s=0.22$, p=0.041). Again, these findings remained after controlling for age, gender and race (bedtime and first meal: B=0.76, p<0.001; bedtime and caloric intake after dinner: B=0.81, p=0.014; bedtime and protein intake after dinner: B=0.08, p=0.041; bedtime and fat intake after dinner: B=0.37, p=0.015). Neither sleep variable was associated with carbohydrate intake after the dinner meal.

Discussion

Our findings demonstrate that, although moderately correlated with each other, bedtime and total sleep time are differentially associated with obesity risk factors in school-aged children who presented for behavioral treatment to enhance sleep duration. Specifically, later bedtime was associated with greater fat intake, later meal timing, and increased caloric intake after the dinner meal whereas shorter total sleep time was associated with higher zBMI and a later last meal of the day. These findings build upon previous research by using objective measures of sleep, physical activity and zBMI, focusing on a diverse sample of children at increased risk for weight gain, and by demonstrating that beyond sleep duration, bedtime may be an important correlate of eating behaviors, including meal timing.

Observed associations between bedtime and both daily fat intake (% daily kcal) and evening fat intake (kcal from fat consumed after the dinner meal) are consistent with previous experimental studies in adults^{45–47,50}. Healthy adults undergoing sleep restriction via a delayed bedtime in a laboratory setting exhibited an increase in daily fat intake^{46,47} and

consumed a greater percentage of calories from fat during late-night hours compared to morning/afternoon and evening hours⁴⁵. Fat intake during evening hours may be particularly problematic for weight management; Baron and colleagues found that the percentage of fat consumed after 22:00 was associated with greater total caloric intake and a higher BMI in adults⁵¹. Future studies are needed to examine how meal timing and macronutrient intake interact to influence obesity risk and what mechanisms underlie the relationship between sleep timing and fat intake in school-aged children. Alterations in brain areas associated with responses to food stimuli, such as those within the salience or reward networks, may represent one mechanism as these areas have been shown to be disrupted in sleep-restricted adults^{52–54}.

Bedtime was also associated with delayed meal timing (operationalized as the time of the first and last meal of the day) and was associated with greater evening intake (kcal consumed after the dinner meal). Breakfast skipping^{23,25,27}, which implies a delay in the timing of the first meal of the day, and evening intake^{22,24,28} have been associated with weight gain, increased adiposity, a larger waist circumference and higher BMI in pediatric populations. For example, children who reported a habit of skipping breakfast exhibited a greater increase in BMI during a 2-year longitudinal study, and those who also skipped lunch (suggesting an even greater delay in the time of the first meal of the day) experienced a larger increase in BMI during that time²⁷. In a large sample of 7-11 year olds, the proportion of daily intake consumed at dinner or as a night snack was significantly associated with increased adiposity (% fat mass)²⁸.

There is growing evidence from experimental studies in adults that sleep patterns influence meal timing²⁸. Healthy adults undergoing sleep restriction via a delayed bedtime in a laboratory setting exhibit an increase in evening/late-night intake and a subsequent decrease in morning intake^{45,48} suggesting that later sleep timing leads to a shift in the timing of caloric intake. Furthermore, in one sleep restriction protocol, bedtime was delayed until 04:00 AM for five consecutive nights (time-in-bed: 04:00 AM - 08:00 AM) and then participants were provided with two consecutive nights of recovery sleep (time-in-bed: 10:00 PM – 10:00 AM)⁴⁵. Interestingly, daily caloric intake was only increased during days when bedtime was delayed - it was not increased during the fifth day of sleep restriction, when participants transitioned to the recovery sleep schedule and bedtime was 10:00 PM⁴⁵. Furthermore, school-aged children exhibited increased daily energy intake (134 kcal/day) during one week of sleep restriction (sleep was decreased by 1.5h/night) compared to one week of sleep extension (sleep was prolonged by 1.5h/night) and the difference in intake seemed to be primarily due to the consumption of additional calories (103 kcal/day) during the 3 additional evening hours that children were awake in the restriction condition²⁹. This pattern suggests that bedtime, or total hours of wakefulness, may be better predictors of daily caloric intake compared to the amount of sleep obtained the previous night. In the current study, bedtime, but not total sleep time, was associated with caloric intake after the dinner meal in school-aged children. Collectively findings suggest that interventions promoting a consistent early bedtime may reduce obesity risk by promoting earlier meal timing and reduced evening intake in children.

Numerous cross-sectional and prospective association studies have demonstrated a relationship between self-reported short sleep duration and higher BMI in pediatric populations 5-10. Furthermore, a recent counterbalanced crossover experimental study found that school-aged children exhibited increased daily energy intake and greater weight gain during a week of sleep restriction (sleep was decreased by 1.5h/night) compared to a week of sleep extension (sleep was prolonged by 1.5h/night)²⁹. Consistent with these findings, in the current study, we observed a negative correlation between total sleep time, assessed using an Actiwatch, and zBMI. In our sample, sleeping 30 additional minutes per night was associated with a 0.24 lower zBMI. These results suggest that modest differences in sleep duration are associated with meaningful differences in zBMI⁵⁵. Research is needed to examine potential mechanisms that underlie the relationship between sleep duration and weight status and the efficacy of sleep interventions for promoting weight management in school-aged children. It is possible that delayed sleep onset and total sleep time impact obesity risk through different mechanisms; although we did not observe a relationship between our measures of caloric intake or moderate-to-vigorous physical activity with total sleep time; other factors, such as the regulation of lipolysis, gastric or intestinal satiation signals (e.g., gastric distention, cholecystokinin, glucagonlike peptide-1), or long-term adiposity signals (e.g., leptin and insulin) may be affected by sleep duration and not bedtime⁵⁶. There is limited evidence linking sleep duration and the regulation of metabolic hormones in adults⁵⁷; however, whether this is also true for school-aged children remains under-studied.

Findings should be considered within the context of strengths and limitations. Strengths of the study include the diversity of the sample in terms of race/ethnicity and weight status as well as its focus on children with short sleep who presented for behavioral intervention. Thus, findings generalize to a diverse, treatment-seeking population and can inform potential intervention targets for improving sleep and promoting weight management. Despite these strengths, it is important to acknowledge that these results may not generalize to children who obtain sufficient sleep. In addition, it is important to note that caloric intake was based upon self-report via 24-hour dietary recalls, which may be subject to reporter bias. Finally, we examined cross-sectional associations between baseline sleep patterns and obesity risk factors. Therefore, we cannot determine if differences in sleep duration or timing lead to changes in eating behaviors and weight regulation or if differences in eating behaviors and weight regulation lead to changes in sleep patterns. Future experimental studies are needed to deduce cause and effect.

Conclusions

In a sample of school-aged children (8 to 11 years) who reported sleeping 9.5 hours per night, we found that bedtime and total sleep time were differentially associated with obesity risk factors. Later bedtime was associated with greater fat intake, later meal timing, and increased caloric intake after the dinner meal whereas shorter total sleep time was associated with higher zBMI. These findings, using an objective measure of sleep duration and timing, provide additional evidence for the importance of sleep hygiene for weight regulation in children. In addition to focusing on sufficient sleep duration, health providers should also consider bedtime when working with children to reduce obesity risk.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Highlights

- bedtime and sleep duration differentially relate to obesity risk factors in children
- later bedtime was associated with greater daily fat intake and later meal timing
- shorter total sleep time was associated with higher zBMI

Table 1.

Sleep, Energy Balance and Meal Timing in School-aged Children (n=87)

	Mean ± SD	Range
Sleep Period (min)	520.8 ± 39.9	402.6 - 590.4
Total Sleep Time (min)	463.8 ± 37.5	364.7 - 532.7
Bedtime	10:31 PM \pm 58.2 min	8:29 PM - 12:58 AM
Wake Time	7:12 AM \pm 41.5 min	5:44 AM - 9:45 AM
Wake After Sleep Onset (min)	57.0 ± 18.9	25.2 - 133.8
Sleep Efficiency (Total Sleep Time/Sleep Period)	$89.1\pm3.4\%$	76.5 - 94.8%
zBMI	0.86 ± 1.0	-1.55 - 2.44
Daily caloric intake (kcal)	1807.9 ± 400.6	1057.8 - 2914.2
Daily protein intake (% kcal)	14.5 ± 3.0	6.9 - 24.7
Daily carbohydrate intake (% kcal)	54.4 ± 5.1	42.6 - 67.2
Daily fat intake (% kcal)	31.1 ± 4.5	20.4 - 44.0
% wake time in MVPA	11.2 ± 5.2	4.1 - 26.9
Time of first meal	$8:29~AM\pm67.0~min$	6:25 AM - 12:45 PM
Time of last meal	$7{:}56~PM \pm 64.5~min$	6:05 PM - 10:27 PM
Intake after 'Dinner' (kcal)	143.4 ± 166.9	0.0 - 700.6
Protein intake after 'Dinner' (kcal)	12.3 ± 20.6	0.0 - 99.9
Carbohydrate intake after 'Dinner' (kcal)	78.0 ± 83.1	0.0 - 332.4
Fat intake after 'Dinner' (kcal)	56.5 ± 77.7	0.0 - 345.1