Changes in Children’s Sleep Duration on Food Intake, Weight, and Leptin

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KEY WORDS
sleep duration, food intake, weight, leptin, ghrelin, 24-hour dietary recall, food reinforcement

ABBREVIATIONS
AEBSF-4—(2-Aminoethyl) benzenesulfonyl fluoride hydrochloride
CDC—Centers for Disease Control and Prevention
TIB—time in bed
zBMI—body mass index z-score

Dr Hart conceptualized and designed the study, obtained funding, oversaw execution of the study, and drafted the initial manuscript; Dr Carskadon helped to design and execute the study and reviewed and revised the manuscript; Dr Considine helped to design the study, analyzed blood samples, and reviewed and revised the manuscript; Dr Fava carried out data analyses and reviewed and revised the manuscript; Ms Lawton coordinated data collection, scored actigraphy data, and reviewed and revised the manuscript; Dr Raynor helped conceptualize and design the study and reviewed and revised the manuscript; Dr Jelalian helped design the study and reviewed and revised the manuscript; Dr Owens helped design the study and reviewed and revised the manuscript; Dr Wing helped conceptualize and design the study and reviewed and revised the manuscript; and all authors approved the final manuscript as submitted.

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All families were reimbursed for their time and effort on the study, and staff at Cincinnati’s Center for Nutritional Research were paid for completion of dietary recalls.

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(Continued on last page)
Given the current pediatric obesity epidemic, there is heightened interest in identifying novel treatment approaches for obesity in children. An area receiving considerable attention is children’s sleep duration. Meta-analyses demonstrate that short sleep in childhood is associated with a 58% to 89% increased risk for obesity. Experimental studies with adolescents and adults suggest a causal pathway through the neuroendocrine control of appetite and food intake. Compared with a rested condition, sleep restriction is associated with decreases in leptin, and increases in ghrelin and reported hunger when caloric intake and energy expenditure are carefully controlled. These changes could affect food reinforcement or how motivated an individual is to obtain food, a pathway supporting the association of sleep restriction with increased caloric intake in several studies. Nevertheless, findings have been mixed for both measured hormones and food intake.

We are unaware of experimental sleep studies with school-age children to examine this pathway. This is striking, given evidence that children may be more susceptible than adults to the effects of sleep on obesity risk. The purpose of the current study was to determine whether experimental changes in school-age children’s sleep duration would result in changes in caloric intake, food reinforcement, appetite-regulating hormones, and measured weight. We hypothesized that compared with decreasing time in bed (TIB), increasing TIB would result in lower reported caloric intake and percent calories (kcal) consumed from fat, reduced reinforcing value of food, higher fasting morning leptin and lower ghrelin, and lower measured weight.

**METHODS**

**Participants**

Children 8 to 11 years old who reported ~9.5 hours/night TIB (ie, time between lights out/trying to fall asleep and waking) were eligible. Eligibility based on TIB was chosen to ensure that children could extend and restrict TIB without reaching a ceiling for what could be achieved (ie, 10–11 hours/night of sleep is recommended at this age) while limiting excessive sleep deprivation. Children needed to be >5th percentile BMI for their age and gender, but no more than 100% overweight (ie, twice the BMI value at the 50th percentile for their age and gender), and to like at least 1 of the foods used in the reinforcement paradigm. 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; per parent report, females could not have started menarche.

Figure 1 shows that 316 children were assessed for eligibility with 277 (88%) excluded. Thirty-nine children were randomized and 37 (95%) completed the study. Mean (SD) age of participants was 9.6 (1.0) years, and mean (SD) body mass index z-score (zBMI) was 0.21 (0.89); 27% were overweight/obese. Fifty-seven percent were male, and 81% were non-Hispanic white. Mean score on the pubertal development scale was 1.4 (0.4), suggesting limited pubertal development. Mean (SD) actigraph-defined sleep period time at baseline was 563 (20) minutes (ie, 9 hours 23 minutes).

**Procedures**

Families from southeastern New England were recruited between March 2009 and December 2011 through mailings, newspaper advertisements, and flyers posted in the community and on hospital/study websites. After an initial phone screen, eligible families attended individual orientations during which written consent was obtained from parents and assent from children. At this visit, children sampled and rated (on a 5-point Likert-type scale) energy-dense breakfast foods (~4 kcal/g) used to assess food reinforcement: blueberry muffins, cereal bars, coffee cake, or a bacon, egg, and cheese pastry. Each child’s highest rated food was used throughout. Eligible children completed a baseline assessment of their typical sleep, which served as both final eligibility determination (ie,
self-reported TIB was confirmed with actigraphy) and to determine the starting point for prescribed changes in TIB to increase and decrease sleep. Participants were randomized post-baseline using a variably sized, stratified (normal weight versus overweight/obese) permuted blocks randomization procedure to increase or decrease TIB by 1.5 hours/night for 1 week. The alternate schedule was completed the subsequent week. This procedure resulted in a targeted 3-hour TIB difference between conditions. Children otherwise completed identical procedures each week: wore actigraph on non-dominant wrist, completed sleep diaries, called the research center twice daily, and completed 3 24-hour dietary recalls (i.e., 6 total recalls across experimental weeks). Parent-child dyads completed self-report measures together. On day 8 of each study week, children came into the center ~1 hour after waking and after an overnight fast for a blood draw and measures of height, weight, and food reinforcement. Modifications of children’s sleep were based on mean TIB achieved (and confirmed by actigraphy) during the baseline week, and achieved by changing bedtimes; wake times remained constant. This procedure approximates changes in sleep in the “real world” (i.e., weekday wake times remain relatively constant), and controls for the influence of wake time on measured hormones. Naps were not allowed. Sleep schedule adherence was promoted by providing fixed bedtimes and wake times during the experimental weeks, and was monitored through twice-daily phone calls to the center. If children missed a call-in time by greater than ±5 minutes, they received a phone call from study staff to increase adherence. Children were compensated for keeping the sleep schedule and twice-daily calls ($5 per day), and for each in-laboratory assessment ($40). All study weeks were scheduled to avoid school vacations and changes in daylight savings time. If the child became ill or did not fast on the morning of center assessments, they were rescheduled. All procedures were approved by the Institutional Review Board at the Miriam Hospital.

Measures
Sleep
Children wore actigraphs (Actiwatch 2; Philips Respironics, Bend, OR) on their non-dominant wrist across the 24-hour period throughout the study. Actigraphs were configured to collect information in 1-minute epochs by using a medium sensitivity threshold, which has demonstrated high sensitivity for detecting sleep compared with polysomnography. Data were scored by using Actiware software, version 5.59.0015. Self-reported bedtimes and wake times from sleep diaries and call-ins were used to score actigraph data by using established procedures. Discrepancies between actigraphy and self-report that were not rectified after review with participating families were brought to consensus meetings with CNH, MAC, and JL. All children had at least 5 nights per condition of valid actigraphy-scored sleep. Variables of interest were mean scores across each week for: actigraph-defined sleep period (i.e., the period between scored sleep onset and scored wake), actigraph scored sleep (minutes of scored sleep within the actigraph-defined sleep period), wake after sleep onset (minutes of scored wake within the actigraph-defined sleep period), and sleep efficiency (actigraph scored sleep/actigraph-defined sleep period).

Anthropometrics
Height and weight were measured by trained staff while children wore street clothes without shoes. Weight was measured on the same calibrated digital scale (Tanita BWB-800; Arlington Heights, IL) to the nearest 0.01 pound, and was converted into kilograms (kg) for analysis. Height was measured to the nearest mm with wall-mounted stadiometers (Perspective Enterprises, Portage, MI). To characterize the sample, zBMI was calculated by using the 50th BMI percentile and corresponding SD of the age- and sex-appropriate sample from the Centers for Disease Control and Prevention (CDC) normative data. Given the short study timeframe, changes in measured weight (kg) were used to assess differences in children’s weight status.

Pubertal Status
A parent-report version of the Self-Administered Rating Scale for Pubertal Development was used. There is a male and female version of the measure assessing growth in height, body hair, and skin changes, as well as breast development and menstruation (girls only) or deepening of voice and facial hair growth (boys only). For each item, parents respond on a 4-point Likert-type scale from “not yet started” to “seems complete.” Scores are averaged to compute a Pubertal Development Scale score.

Food Intake
The USDA automated multiple pass method for 24-hour dietary recalls was used to assess child food intake on 2 weekdays and 1 weekend day during each study week. Families were provided with instructions and aids for portion estimation; parents as proxies (with child help as needed) completed recalls with blinded staff by phone. It is considered the most accurate approach in determining child energy intake when compared with the doubly labeled water method. Dietary data were entered and coded in the Nutrition Data System for Research (Nutrition Coordinating Center, University of Minnesota, Minneapolis, MN). Primary
variables of interest were mean kcal intake and mean percent kcal consumed from fat, carbohydrates, and protein. Given the additional 3 hours children were awake in the decrease condition, post-hoc analyses assessed kcal intake from snacks during this time. Specifically, kcal intake reported to have occurred after the prescribed bedtime for the increase condition was calculated; this was done for both the increase (ie, in case a child was off-schedule and consumed food) and decrease conditions.

**Food Reinforcement**

The Behavioral Choice Task is a validated computer activity that assesses children's motivation for a food reward (ie, how hard they work or the number of points earned for food). Studies demonstrated that food reinforcement is positively associated with increased hunger and higher weight status. The task is like a matching game with multiple trials. Children earn points when they click on a computer mouse and the shape and color of items on the screen match. The frequency of matches was predetermined using an ascending variable ratio reinforcement schedule that started with matches occurring at approximately every 4 clicks and doubling thereafter for each trial played. After each trial, children received ~25 g of their food reward (ie, 100 kcal) and continued playing until they no longer wanted to work. Primary variable of interest was number of points earned.

**Assays**

Blood samples were collected ~1 hour after waking on the last day of each 1-week sleep condition after an overnight fast. EDTA plasma was stored at −80°C until the end of the study. 4-(2-Aminothyl) benzenesulfonyl fluoride hydrochloride (AEBSF: Sigma Aldrich, St Louis, MO) was added to the blood collection tubes to protect acylated ghrelin from degradation. Leptin was measured by radioimmunoassay (HL-81K) and octanolyated ghrelin by ELISA (EZGRA-88K; Millipore-Linco Research, St Charles, MO). Using these assays, the minimum detectable level of leptin was 0.5 ng/mL and ghrelin was 8 pg/mL, with average intra-assay coefficients of variation (CV) of 3.7% and 1.3%, respectively. All samples were run in duplicate in the same assay.

**Sample Size and Statistical Analysis**

Analyses were conducted by using IBM SPSS Statistics for Windows, Release 20.0.0 (@IBM Corp., 2011, Armonk, NY; www.ibm.com). Three children were excluded from blood chemistry analyses owing to inability to draw blood during both conditions. One child was excluded from dietary recall analyses because of inability to obtain at least 1 weekend day during both weeks. Data on food reinforcement were available on N = 29 children owing to changes in the food reinforcement design after the first 8 children completed the study. To limit the influence of other factors on differences in weight we excluded 2 participants from weight analyses because their increase and decrease sleep conditions were not run consecutively.

Preliminary analyses assessed for normality of distribution using the Shapiro-Wilk statistic. At least 1 variable across condition (ie, increase or decrease) was skewed for wake after sleep onset and leptin. Both sets of variables were log transformed. Although condition order was counterbalanced, preliminary analyses used repeated measures analysis of variance with order as a between-subjects factor and condition as a within-subjects factor to confirm no order effect on study outcomes. None were found; final analyses used paired samples t tests to assess the effect of condition on study variables. Statistical significance was set at P < .05.

**RESULTS**

Figure 2 shows differences in the increase and decrease conditions for the actigraph-defined sleep period time for all participants, and demonstrates that all participants achieved changes in their sleep duration. Summary statistics across sleep parameters are presented in Table 1. Specifically, participants achieved a mean 141 minute/night (ie, 2 hour; 21 minutes) difference in the actigraph-defined sleep period time between the increase and decrease conditions (P < .001). As would be expected, wake after sleep onset and sleep efficiency differed between the 2 conditions such that on average during the increase condition, children were awake during the sleep period for ~34 minutes more than during the decrease condition and their sleep efficiency decreased by ~3% (P < 0.001).

Consistent with study hypotheses, children reported consuming 134 kcal/day less during the increase than the decrease condition (P = .04) (see Table 1). Most of the difference in kcal intake occurred during the additional 3 hours that children were awake during the decrease condition with children reporting 103 kcal/day more during this time (P < .001). There were no differences in reported macronutrient consumption (P > .05). Fasting morning leptin was significantly lower during the increase than the decrease condition (P < .05); there was no difference for fasting morning ghrelin (P = .21). Furthermore, children weighed less at the end of the increase than the decrease condition (P < .001) with a mean difference in weight of 0.22 kg, and associated effect size of d = 0.95. Twenty-four (69%) children weighed less at the end of the increase condition; 4 were weight stable. Differences
in weight were correlated with differences in leptin \( (r = 0.42, P = .03; \text{Fig 3}) \), but not with differences in reported caloric intake \( (P > .05) \). Post-hoc comparisons found no effect of child zBMI, age, or parent-reported pubertal status on any of the above-noted outcomes.

**DISCUSSION**

The current study is the first to use an experimental design in a naturalistic setting to determine whether changes in school-age children’s sleep duration are associated with changes in eating behaviors, appetite-regulating hormones, and measured weight. We found that an experimental increase in children’s average nightly sleep of 2 hours, 21 minutes resulted in a reported 134 kcal/day reduction, lower leptin levels, and lower body weight. These findings are consistent with observational studies, and extend findings by documenting that food intake, leptin, and weight decrease as a result of increases in sleep. Findings also build on previous experimental studies by focusing on a school-age sample.

Consistent with previous adult laboratory studies, we found that when children increased their sleep duration, they reported consuming ~134 kcal/day less than when they decreased sleep. On a population level a reduction in energy intake of this magnitude could result in prevention of excess fat accumulation, which could have significant implications for the obesity epidemic. Moreover, interventions designed to create an energy deficit comparable to that observed in the current study have demonstrated effectiveness in decreasing weight status in school-age children. Considered within this context, the observed decreases in reported caloric intake are compelling. Leptin levels were also lower during the increase sleep condition. Although these findings are contrary to many with adults, this may be attributable

**TABLE 1**

<table>
<thead>
<tr>
<th></th>
<th>Increase</th>
<th>Decrease</th>
<th>Difference(^{a}) (Increase – Decrease)</th>
<th>Paired Mean</th>
<th>Mean 95% CI</th>
<th>( P \text{-value} )</th>
<th>Cohen’s ( d )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Actigraph-defined sleep period (min/night)</td>
<td>627</td>
<td>618 to 636</td>
<td>486</td>
<td>479 to 493</td>
<td>141</td>
<td>135 to 148</td>
<td>39.00</td>
</tr>
<tr>
<td>Actigraph-scored sleep minutes (min/night)</td>
<td>545</td>
<td>535 to 556</td>
<td>439</td>
<td>431 to 446</td>
<td>106</td>
<td>98.4 to 114.4</td>
<td>26.92</td>
</tr>
<tr>
<td>Wake after sleep onset (min/night)</td>
<td>78.8</td>
<td>72.4 to 85.9</td>
<td>45.1</td>
<td>40.8 to 49.8</td>
<td>33.7</td>
<td>−4.1 to −2.5</td>
<td>8.57</td>
</tr>
<tr>
<td>Sleep efficiency (% wake/night during sleep period)</td>
<td>87.0</td>
<td>85.9 to 86.1</td>
<td>90.3</td>
<td>89.3 to 91.3</td>
<td>−3.3</td>
<td>−4.1 to −2.5</td>
<td>8.57</td>
</tr>
<tr>
<td>Mean caloric intake (kcal/d)</td>
<td>1751</td>
<td>1650 to 1872</td>
<td>1885</td>
<td>1726 to 2045</td>
<td>−134</td>
<td>−261 to −8</td>
<td>2.16</td>
</tr>
<tr>
<td>Mean caloric intake in the 3 h post-increase bedtime (kcal/d)(^{b})</td>
<td>2</td>
<td>0 to 5</td>
<td>105</td>
<td>66 to 143</td>
<td>102</td>
<td>64 to 141</td>
<td>5.38</td>
</tr>
<tr>
<td>Mean percent kcal from fat (%/d)</td>
<td>28.8</td>
<td>27.1 to 30.4</td>
<td>29.7</td>
<td>27.9 to 31.5</td>
<td>−1.0</td>
<td>−2.7 to 0.8</td>
<td>1.13</td>
</tr>
<tr>
<td>Mean percent kcal from carbohydrates (%/d)</td>
<td>55.4</td>
<td>53.8 to 57.3</td>
<td>54.8</td>
<td>53.0 to 56.6</td>
<td>0.7</td>
<td>−1.4 to 2.7</td>
<td>0.64</td>
</tr>
<tr>
<td>Mean percent kcal from protein (%/d)</td>
<td>15.8</td>
<td>14.8 to 16.7</td>
<td>15.5</td>
<td>14.5 to 16.5</td>
<td>0.3</td>
<td>−0.6 to 1.2</td>
<td>0.70</td>
</tr>
<tr>
<td>RRV (points earned)</td>
<td>21.9</td>
<td>18.9 to 24.9</td>
<td>20.3</td>
<td>17.1 to 23.6</td>
<td>1.6</td>
<td>−0.4 to 3.5</td>
<td>1.61</td>
</tr>
<tr>
<td>Leptin (ng/mL)</td>
<td>5.67</td>
<td>4.59 to 7.01</td>
<td>6.97</td>
<td>5.55 to 8.77</td>
<td>−18.6%</td>
<td>−1.0% to −33.1%</td>
<td>2.14</td>
</tr>
<tr>
<td>Ghrelin (pg/mL)</td>
<td>909.1</td>
<td>785.8 to 1032.4</td>
<td>866.8</td>
<td>745.3 to 988.3</td>
<td>42.3</td>
<td>−24.4 to 109.0</td>
<td>1.29</td>
</tr>
<tr>
<td>Wt (kg)</td>
<td>34.8</td>
<td>31.7 to 38.0</td>
<td>35.0</td>
<td>31.9 to 38.2</td>
<td>−0.22</td>
<td>−0.33 to −0.10</td>
<td>3.91</td>
</tr>
</tbody>
</table>

All values represent means and their 95% confidence interval [CI]. For wake after sleep onset and leptin, values represent geometric means, exponentiation (antilogs) of the natural log transformed data used in analyses; paired t-values and associated \( P \)-values for these variables are for transformed data.

\(^a\) Values presented for dietary data are for \( N = 36 \); for leptin and ghrelin, \( N = 34 \); for RRV \( N = 29 \). All other values are for the entire sample \( (N = 37) \).

\(^b\) For wake after sleep onset and leptin, values represent percent differences between the geometric means.

\(^{c}\) Given a child cannot consume a snack with negative kcal, we have truncated the lower bound of the 95% CI for this variable at 0. Owing to significant skew in the increase condition (ie, all but 2 children were adherent to their prescribed bedtime during the increase sleep condition and did not consume any food after that time), \(^{d}\) analyses were also run to examine post-hoc comparisons of kcal intake from snacks during the 3 additional hours children were awake during the decrease sleep condition; findings were consistent.
to important methodologic differences across studies. When caloric intake was carefully controlled and weight maintained, studies with adults demonstrated decreases in leptin on sleep restriction. Additional studies with adults that did not control food intake have not found such changes. Given positive associations between leptin and percentage body fat, and the correlation between leptin and weight in the current study, it is possible that decreased leptin levels during the increase sleep condition are more reflective of small changes in weight than of changes in satiety signaling (as is typically hypothesized in adult experimental studies). Given the 3-hour shift in bedtimes, a circadian influence on observed differences in leptin is also possible.

Differences observed in measured weight provide a potentially compelling link between differences in sleep, reported dietary intake, and fasting leptin. Similar to a laboratory study with adults, compared with the end of the decrease sleep condition, on average children weighed ~0.22 kg less at the end of the increase sleep condition. Although differences of this magnitude may seem small, the size of the effect is large, and the consistency in findings across participants is striking. If the observed effect on weight is maintained over the longer-term, it could have implications for the importance of sleep in weight regulation.

We did not find differences in either food reinforcement or ghrelin. Although contrary to hypotheses, our findings regarding ghrelin fit within the mixed findings from experimental studies with adults. As with leptin, reported differences in food intake and weight status may account for our null finding. Null findings for food reinforcement, which was measured ~1 hour after waking, may be at least partially explained by post-hoc analyses showing that differences in caloric intake occurred later in the day. Future studies should consider assessment of food reinforcement later in the day when children may be more susceptible to the effect of differences in sleep on eating behaviors.

Strengths of the study include its experimental design, focus on school-age children, and high level of participant adherence (ie, achieving an average 2 hour, 21 minute difference in the actigraph sleep period time within the context of a 3-hour TIB difference). Limitations include the small sample and assessment of acute (1-week) changes in sleep. It is also important to acknowledge that sleep duration was changed by changing bedtimes, which may result in shifts in circadian rhythm. It is thus not possible to tease apart the relative influence of changes in sleep duration versus possible shifts in circadian timing on study outcomes. Furthermore, differences in sleep between the 2 conditions were relatively large. Although the current study was designed more as a proof of concept than a clinical intervention (warranting the 3-hour difference in TIB) it is unclear whether smaller changes would yield similar results. Given the careful experimental controls, generalizability of findings may also be limited. Finally, there was no wash-out period between the 2 conditions, which may have dampened our ability to detect differences in study outcomes.

CONCLUSIONS

Increases in sleep duration in school-age children are associated with decreased reported energy intake, lower fasting leptin levels, and weighing less. Findings provide compelling evidence for further understanding how intervening on sleep duration could impact children’s weight status.
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