

Sleep and the Body Mass Index and Overweight Status of Children and Adolescents

Emily K. Snell, Emma K. Adam, and Greg J. Duncan

Northwestern University

Associations between sleep and the body mass index (BMI) and overweight status of children and adolescents were estimated using longitudinal data from a nationally representative sample of 2,281 children aged 3–12 years at baseline. Controlling for baseline BMI, children who slept less, went to bed later, or got up earlier at the time of the first assessment had higher BMIs 5 years later and were more likely to be overweight. Child age moderated the relationship between bedtime and BMI. In addition, the study reports nationally representative data on the sleep habits of American children aged 3–18 years. This study underscores the likely importance of sleep on children's physical health and suggests that sleep is important for understanding childhood weight problems.

Sleep is vitally important for a child's day-to-day functioning. As the pace of modern life hastens, however, researchers, policymakers, and parents have become increasingly concerned about whether children get enough sleep and whether lack of sleep has lasting effects on children's cognitive, socioemotional, and physical development. In this paper, we describe the sleep behaviors of American children and adolescents and examine the influence of sleep patterns in early and middle childhood on one aspect of child and adolescent physical well-being: body mass index (BMI) and overweight status.

A growing body of work has found associations between sleep and children's cognitive and social functioning. Sleep problems, such as low hours of sleep, night terrors, sleep talking, and sleep apnea, have been associated with maladjustment in preschoolers (Bates, Viken, Alexander, Beyers, & Stockton, 2002), internalizing problems in middle childhood (Hofferth & Sandberg, 2001), and depression and school problems in adolescents (Fredriksen, Rhodes, Reddy, & Way, 2004; Wolfson & Carskadon 1998). Experimental studies have found that children's daytime cognitive and behavioral functioning are substantially impaired following even a slight truncation (by 1 hr) in the amount of sleep the previous night (Sadeh, Gruber, & Raviv, 2003). Lack of sleep among adolescents has also been shown to put them at increased risk of accidents while driving (Carskadon, Acebo, & Jenni, 2004).

As a result, scholars have called for more research aimed at understanding the short- and long-term effects of inadequate sleep as well as better guidelines for parents and physicians on the optimal level of sleep for children and adolescents (Dahl & Lewin, 2002; Wolfson & Carskadon, 2003).

Given the nation's pediatric obesity crisis, researchers have noted the need for investigating the relationship between children's sleep and weight (Bass & Turek, 2005). Several cross-sectional studies have found that overweight children sleep less than children of normal weight (Gupta, Mueller, Chan, & Meininger, 2002; Locard et al., 1992; Sekine et al., 2002). Obesity in particular is associated with high rates of sleep problems in both children (Marcus et al., 1996) and adults (Nieto et al., 2000; Vgontzas, Bixler, & Chrousos, 2005). Prospective studies of the sleep behavior of very young children have also found links between sleep and children's overweight status. Shorter daytime sleep (Agras, Hammer, McNicholas, & Kraemer, 2004), as well as shorter night-time sleep (Reilly et al., 2005) in early childhood have been linked to obesity during middle childhood.

Still, the causal relationship between sleep and weight remains unclear. Many previous studies control neither for prior weight nor for potentially confounding variables, and hence we cannot know whether child overweight status contributed to sleep problems or whether child or family characteristics (such as child health status or family schedules) differed in some other way that influenced both sleep behavior and weight.

Correspondence concerning this article should be addressed to Emily K. Snell, Institute for Policy Research, 2040 Sheridan Road, Evanston, IL 60208. Electronic mail may be sent to e-snell@northwestern.edu.

Finally, little is known about the potentially moderating influences of age on the relationships between sleep and developmental outcomes, although we know that there are age-related shifts in children's sleep needs and the circadian timing of sleep, particularly as children enter the adolescent years. The shifts include phase delay, or the tendency toward later sleeping and waking times, and irregular sleep patterns, with weekend sleep schedules being significantly different from sleep schedules during weekdays (Acebo & Carskadon, 2002).

In this paper, we first describe sleep behavior in a nationally representative sample of children and adolescents aged 3–17 years. We then examine whether sleep behaviors influence children's subsequent BMI and overweight status (which includes children classified as either overweight or obese). Our models relate BMI and overweight status outcomes to prior sleep patterns, controlling for prior BMI or overweight status, as well as child and family characteristics.

Rather than relying on global estimates of hours of sleep, our measurements of when children go to bed, when they wake up, and how long they sleep are derived from a time diary method, in which parents and/or children describe the allocation of their time across a randomly selected day on a minute-by-minute basis. Time diaries have been shown to estimate time allocation more accurately than questionnaire reporting of recalled hours of activity (Juster, Ono, & Stafford, 2003). We also examine whether the associations between sleep and BMI and overweight status vary by children's age and gender.

We hypothesize that children who sleep less will have higher BMIs at Time 2, controlling for their BMIs at Time 1. We anticipate that fewer sleep hours overall at Time 1 will be associated with greater BMIs for both younger and older children. Owing to the above-mentioned age and pubertal-stage-related shifts in the circadian timing of children's versus adolescents' sleep needs, we further hypothesize that later bedtimes will be more strongly related to increased BMIs for younger children, whereas earlier wakeup times will be more strongly associated with increased BMIs for adolescents.

A major analytic challenge for the estimation of the causal effect of sleep on children's weight is to disentangle whether sleep actually affects children's weight or whether it is simply that children who already are overweight, or have other characteristics that correlate with being overweight, tend to sleep poorly. Many studies examining the association between sleep and child outcomes have statistically

controlled for some of the measurable family and child influences on both children's sleep patterns and children's outcomes. We attempt to limit the potential influence of unmeasured confounding variables with the following longitudinal model:

$$\begin{aligned} \text{Child outcome}_{2i} = & \beta_0 + \beta_1 \text{Child outcome}_{1i} \\ & + \beta_2 \text{Sleep measure}_{1i} \\ & + \beta_3 \text{Child controls}_{1i} + \beta_4 \text{Family controls}_{1i} + \varepsilon_i. \end{aligned} \quad (1)$$

In this model, outcomes at Time 2 for child *i* are expressed as a function of sleep behavior at Time 1, controlling for a Time 1 measure of the child outcome (BMI or overweight status). Including the Time 1 measures of the child outcome helps control for time-invariant child and family influences on weight (both measurable and not) that were present at the first wave of data collection. To further limit the possibility that children with particular individual or family characteristics may choose to sleep more or less, we also include measures of parental income and education at Time 1, as well as child gender, race, and age at both Times 1 and 2.

Our approach assumes that unobserved, confounding variables have similar impacts on both early and later outcomes and that Time 1 sleep is measured without error. To the extent that the impact of child and family characteristics on sleep and child outcomes differs between the Times 1 and 2 assessments, estimates of the relation between sleep and children's development may be biased. While our longitudinal approach is stronger than many prior studies of associations between sleep and child outcomes, our results should be interpreted in light of this limitation.

Method

Sample

We use data from the first and second waves of the Child Development Supplement (CDS) of the Panel Survey of Income Dynamics (PSID). The CDS is one component of the PSID, a longitudinal study of a representative sample of U.S. individuals and the families in which they reside. In 1997, the PSID supplemented its main data collection with additional data on children aged 0–12 years old and their parents. The objective was to provide researchers with a comprehensive, nationally representative, and longitudinal database of children and their families with which to study the dynamic process of early human capital formation.

In the first wave of the CDS, which took place in 1997, 2,394 families participated, providing information on a total sample of 3,563 children. The second wave of data collection took place in 2002–2003 when 2,021 families were successfully reinterviewed, resulting in a total of 2,907 child interviews. The reduction in the sample is due to reclassification of the eligibility status of some sample participants (83 children) and also due to nonresponse among the remaining eligible CDS families (573 children) from the first wave. Accounting for both sources of attrition in the first wave, the second wave reinterview rate is 81.6%. Because of concerns that the nature of night-time sleep may be quite different for infants and toddlers than for older children, we exclude 626 children from our sample who were younger than 3 years at the time of the first wave. Thus, our sample for the descriptive analysis of sleep behavior includes 2,281 children. Our sample for the sleep and weight analysis is smaller (1,441 children) due to

missing height and weight data at one or both assessments.

Outcome Measures

As summarized in Table 1, our dependent variables include measures of children's standardized BMI and overweight status at Time 2. Children's BMI at Time 2 was computed using height and weight measured by the interviewer. Because raw BMI scores are not comparable across age and gender and are not distributed normally, the raw BMI scores were then standardized by child age and gender and adjusted for skew. The procedure for measuring height and weight was as follows: if the child was under 6 years, interviewers asked the primary care giver or a family member for assistance in taking off the child's shoes, helping the child onto the scale, and encouraging the child to stand still. Directions

Table 1
Description of Sample, Sleep Variables, and Outcome Variables

	Observed	Mean	Standard deviation
Body mass index (BMI) measures			
Outcome variables			
Child's BMI (standardized) at Time 2	1,677	0.55	1.14
Child is overweight or obese at Time 2	1,677	0.36	0.48
Time 1 weight controls			
Child's BMI (standardized) at Time 1	1,677	0.30	1.45
Child is overweight or obese at Time 1	1,677	0.29	0.45
Sleep measures			
Time diary measures as continuous			
Number of hours of sleep at Time 1	1,885	9.91	1.07
Time diary measures as dummies			
Slept less than 8 hr at Time 1	1,885	0.04	0.19
Slept 8–9 hr at Time 1	1,885	0.15	0.35
Slept 9–10 hr at Time 1	1,885	0.35	0.48
Slept 10–11 hr at Time 1	1,885	0.33	0.47
Slept 11+ hr at Time 1	1,885	0.13	0.34
Sleep measures			
Bedtime and waketime			
Bedtime at Time 1	1,885	9:25 p.m.	60 min
Wake-up time at Time 1	1,885	7:30 a.m.	50 min
Demographic measures			
Child's age at Time 1	2,281	8.12	2.91
Child's age at Time 2	2,281	13.65	2.93
Child is male	2,281	0.50	0.50
Family income in \$10,000s	2,281	7.32	9.03
Average parental education	2,281	12.89	2.84
Child is non-Hispanic black	2,281	16.1%	0.37
Child is non-Hispanic White	2,281	62.9%	0.48
Child is Hispanic	2,281	13.3%	0.34
Child is other race/ethnicity	2,281	7.6%	0.27

were given directly to both the child and family member assisting the interviewer. If the child was 6 years or older, the interviewer instructed the child directly and asked a family member for assistance only if necessary. To measure the child's height, the interviewer asked the child to take off his or her shoes and stand against a wall or door. The interviewer placed a Post-it[®] note on the wall right above the child's height, and then positioned a rafter's square against the wall directly over the child's head. A mark was placed on the Post-it[®] note at the child's height. When the child moved away from the wall, the interviewer positioned the end of the tape measure under a door jamb or under her foot and measured from the floor to the mark on the Post-it[®].

To weigh the child, the interviewer asked the child to empty his or her pockets and remove his or her shoes, activated the scale, and set it to zero. Once the scale showed "0.0" the child stepped onto and stood in the center of the scale for 5–10 s. The same model and brand of scale (the Strain-Gauge Lithium Bath Scales "THINNER MS-7") was used for all children. If the child or youth refused to be measured (<1% of the sample refused), the interviewer asked them or the primary caregiver (depending on the child's age) to report height and weight.

The Time 1 measure of BMI was generated from height as assessed by the interviewer (with the same protocol as at Time 2) and weight as reported by the primary caregiver. Unfortunately, we do not know when heights were reported by the parent rather than directly measured by the interviewer at Time 1. However, given that <1% refused to have their height measured at Time 2, it seems unlikely that a large proportion of subjects had their height reported rather than measured at Time 1. Intensive study-specific training was provided to interviewers for the anthropometric measures at both Times 1 and 2. Interviewers were given time to practice and were evaluated on their performance before entering the field.

We calculated standardized BMI at both waves using the STATA program "zanthro" (Vidmar, Carlin, Hesketh, & Cole, 2004), which calculates the Center for Disease Control's standardized BMI given the child's height, weight, gender, and age using methodology developed by Cole (1990). This procedure converts BMI into a standardized measure reflecting each child's BMI relative to children of the same age and sex, thus allowing for comparison of children of different ages and genders. The program also adjusts for the skew in the distribution of BMI, giving a normally distributed variable.

We also generated a binary indicator of children's overweight status at Time 1 and a binary indicator of children's overweight status at Time 2 following international guidelines (Cole, Bellizzi, Flegal, & Dietz, 2000). The binary indicator of normal weight versus overweight we use is analogous to the adult categories of normal weight (BMI <25) and overweight (BMI ≥25), although the actual BMI cutoffs differ from adults and vary by child age and gender.

Sleep Measures for Causal Analysis of Sleep on BMI/Overweight Status

We used information from the PSID–CDS time diaries from the first wave to measure the total amount of time each child was asleep. The time diaries provide detailed accounting of the type, number, duration, and location of activities for two 24-hr periods for each child: one on a weekend and the other on a weekday. The diaries were filled out by the parents of younger children and by the older children themselves with the assistance of parents as needed.

With child-based sampling weights, the time diaries provide a nationally representative estimate of how children spend their time. Both the weekday (Monday–Friday) and weekend day (Saturday or Sunday) time diary days were randomly selected when the interviewer completed the initial contact for the household. There was no substitution of diary days once they were assigned. In both waves, a paper CDS time diary was mailed several days before the scheduled interview, with instructions to complete the diary before the interview. During the in-house CDS interview or by telephone, the interviewer reviewed and edited the diary with the child and primary caregiver. If the diary was not completed in advance, the interviewer administered the diary as an interview about the day it was supposed to have been completed.

From the time diary reports, we created three types of sleep variables: a linear measure of total amount of sleep in hours, a nonlinear measure of total amount of sleep hours, and linear measures of bedtime and waketime. We created a measure of the total amount of night-time sleep at Time 1 by averaging weekday and weekend sleep (weighted 5:2 for weekday vs. weekend to account for the distribution of days in a week).

The PSID–CDS time diary data do not include a specific measure of daytime naps, although there is a measure of total time spent napping, resting, or laying down. We did not include this measure of

daytime nap or rest in measuring total sleep hours, although we did analyze it as a separate variable.

In order to test for nonlinear associations between sleep and BMI, we created dummy indicators for amount of sleep (dummy indicators for sleeping less than 8, 8–8.9, 10–10.9 hr, and more than 11 hr, with sleeping 9–9.9 hr as the reference category). The nonlinear measures allowed us to examine alternative functional forms of the relationship between sleep and weight, as it allows for changes in the strength of the effect of sleep on child outcomes at different hours of sleep. In order to determine how bedtimes and waketimes related to outcomes, we created linear measures of the average bedtime and average waketime at Time 1 (again weighted 5:2 for weekday vs. weekend).

Additional analyses regressed BMI/overweight status on measures of weekend sleep behavior alone and weekday sleep behavior alone, and regressed BMI/overweight status on a measure of sleep that weighted weekday sleep and weekend sleep equally. The results from these analyses are similar to those using the weighted average measure.

Sleep Measures for Descriptive Analysis of Sleep Behavior Across Childhood and Adolescence

In order to describe sleep behaviors of American children aged 3–18, we pooled time diary data from Time 1, when the children were aged 3–12, and Time 2, when the children were aged 8–19. We dropped the 19-year-olds from the descriptive analysis, however, because there were so few of them ($n = 3$). The sleep measures were created in the same way as described above, except that we examined weekday and weekend sleep separately.

Control Measures

Our control measures consisted of household, parent, and child characteristics. We included continuous measures of family income in 2000 in tens of thousands of dollars and parental education, which was obtained by averaging each parent's total number of years of education; one parent's education was missing for about one third of the families, in which case we used the only available parent's education level. We also included controls for child's race (dummy indicators, with non-Hispanic White being the reference group), child's age at the Time 1 interview (continuous measure), child's age at the Time 2 interview (continuous measure), and whether the child is male (dummy indicator). We included child's age at both waves because the length of time

that passed between the two assessments was not constant; it averaged five and a half years, but ranged from 4 to 7 years.

All analyses were weighted with PSID-supplied probability weights that are inversely proportional to the likelihood of being selected into and continuing to participate in the sample. Probability weights allow us to generalize to our population of inference, which includes all children living in the United States.

All of our regressions include dummy variables indicating who has missing data; mean values are assigned in these cases. In all regressions, standard errors have been adjusted for the lack of independence caused by family clustering of sample children using Huber–White methods.

After examining correlations between variables of interest, describing nationally representative patterns in children's sleep behavior, and determining the main effects of sleep on children's subsequent BMI and overweight status, we explore whether the effects of sleep on children's weight vary as a function of child gender and age. This was done by including interaction terms in the regression presented in Equation 1. The interactions involved child gender (1 = male) and child age at baseline (a dummy variable indicating that the child was aged 3–7.9 years vs. 8–12.9 years at the Time 1 assessment).

Results

Correlations among the various health and sleep measures assessed at Times 1 and 2 are shown in Table 2. Although we do not find an association between the linear measure of sleep time at Time 1 and BMI or overweight status at Time 1, we do find cross-sectional associations between measures of weight and both sleeping very little and having later bedtimes. For example, sleeping less than 8 hr a night is correlated with higher BMI as well as being overweight at Time 1, while sleeping between 10 and 11 hr a night is correlated with not being overweight at Time 1. In addition, later bedtimes are correlated with being overweight at Time 1. We also find measures of sleep at Time 1 to be correlated with BMI and overweight status at Time 2. The linear measure of sleep at Time 1 is negatively correlated with BMI and being overweight at Time 2. The nonlinear measure of sleep finds similar results to the cross-sectional results: Sleeping less than 8 hr a night is correlated with higher BMI and being overweight at Time 2, while sleeping between 10 and 11 hr a night or more than 11 hr is correlated with lower BMI and not being overweight at Time 2. Later

Table 2
Correlations Among BMI, Overweight, Sleep, and Demographic Variables

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18
1 BMI at Time 2	1.00																	
2 BMI at Time 1	0.40**	1.00																
3 Overweight at Time 2	0.78**	0.30**	1.00															
4 Overweight at Time 1	0.39**	0.72**	0.32**	1.00														
5 Sleep hours at Time 1	-0.10**	-0.01	-0.09**	-0.03	1.00													
6 Slept less than 8 hr a day at Time 1	0.08**	0.06*	0.04	0.06*	-0.55**	1.00												
7 Slept 8-9 hr a day at Time 1	0.04	-0.03	0.05	0.00	-0.46**	-0.09**	1.00											
8 Slept 9-10 hr a day at Time 1	0.02	0.01	0.02	0.01	-0.21**	-0.16**	-0.50**	1.00										
9 Slept 10-11 hr a day at Time 1	-0.05*	-0.03	-0.04	-0.06*	0.35**	-0.15**	-0.28**	-0.50**	1.00									
10 Slept 11+ hr a day at Time 1	-0.05*	0.01	-0.05*	0.02	0.60**	-0.09**	-0.17**	-0.30**	-0.28**	1.00								
11 Bedtime at Time 1	0.08**	0.04	0.06**	0.06**	-0.71**	0.51**	0.32**	0.08**	-0.32**	-0.31**	1.00							
12 Waketime at Time 1	-0.04	0.03	-0.05	0.03	0.46**	-0.08**	-0.24**	-0.19**	0.09**	0.44**	0.29**	1.00						
13 Family income in \$10,000s	-0.07*	0.00	-0.03	-0.04	0.01	-0.02	0.01	-0.02	0.02	0.00	0.01	0.01	1.00					
14 Parental education	-0.09**	-0.03	-0.09**	-0.08**	-0.03	0.03	-0.04	0.03	0.02	-0.05	0.01	-0.03	0.26**	1.00				
15 Child's age at Time 1	0.01	-0.03	0.02	0.01	-0.35**	0.09**	0.20**	0.17**	-0.14**	-0.30**	0.21**	-0.22**	0.07*	0.01	1.00			
16 African American	0.12**	0.02	0.08**	0.08**	-0.13**	0.12**	0.08**	0.01	-0.12**	-0.01	0.13**	-0.02	-0.23**	-0.16**	0.02	1.00		
17 Hispanic	0.06*	0.06*	0.07*	0.07*	0.04	-0.05	0.00	-0.02	0.02	0.03	-0.05	0.00	-0.09**	-0.25**	0.00	-0.21**	1.00	
18 Non-Hispanic White	-0.14**	-0.04	-0.12**	-0.11**	0.11**	-0.10**	-0.09**	0.02	0.11**	-0.01	-0.10**	0.03	0.27**	0.32**	-0.02	-0.80**	-0.27**	1.00

Note. BMI = body mass index.
* $p < .05$, ** $p < .01$.

bedtimes at Time 1 also predict higher BMI and being overweight at Time 2, although waketime does not. These patterns are similar to those found in the child sleep and overweight literature.

Parental demographic information is strongly correlated with BMI and obesity: Children in wealthier families and children with better educated parents have lower BMIs and are less likely to be overweight. However, parental income and education are not associated with children's sleep behaviors. Child age is strongly associated with sleep behavior: Older children sleep less, go to bed later, and wake up earlier than younger children. Child race and ethnic background are associated with BMI and sleep behaviors. African American children are heavier, sleep less, and go to bed later than other groups. Hispanic ethnicity is correlated with higher BMIs. Non-Hispanic White children tend to be lighter, sleep more, and go to bed earlier than other groups.

Developmental Trends in Sleep Behaviors

To describe developmental patterns in our nationally representative sample of children, we first examined children's average nightly sleep behavior. On average, total sleep time declined as children age; older children wake up later, but also went to bed later (Figures 1 and 2).

The story is somewhat more complex when weekend and weekday sleep behaviors are examined separately. As shown in Figure 1, children's sleep time declined moderately on weekends but declined quite rapidly on weekdays over childhood and adolescence. Weekend sleep declined by about 40 min between the ages of 3 and 17: from 10.9 to 10.2 hr. In contrast, sleep time on weekdays declined more than 2 hr between early childhood and later

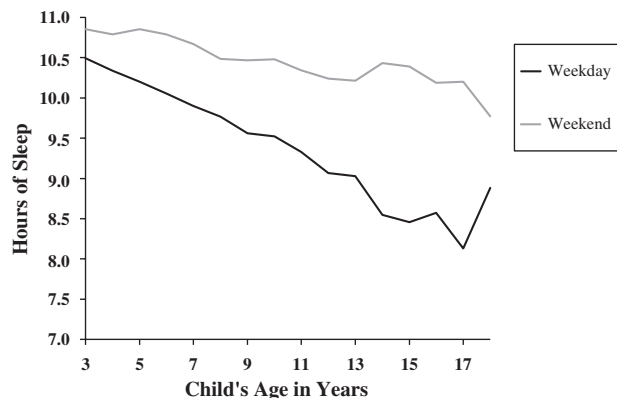


Figure 1. Children's weekday and weekend hours of sleep, by age.

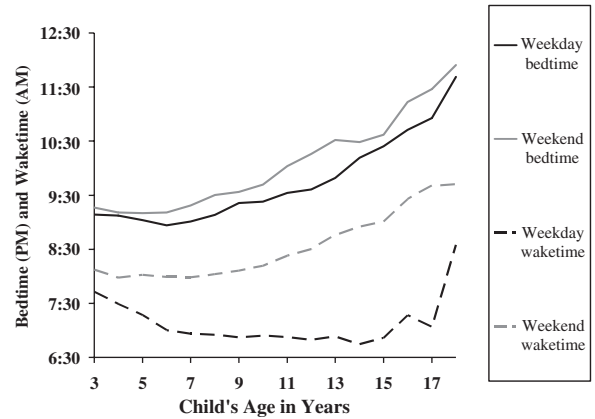


Figure 2. Children's weekday and weekend bedtimes and waketimes, by age.

adolescence. Children aged 3 slept nearly 10.5 hr a night on weekdays and 6-year-olds slept just over 10 hr, while 10-year-olds slept 9.5 hr, 14-year-olds slept 8.5 hr, and 17-year-olds slept barely 8 hr. The decline in sleep time was fairly linear except for a prominent fall-off in weekday sleep between ages 13 and 14, probably due to the transition from middle to high school. Weekday sleep time actually increased between ages 17 and 18, when many children transition out of high school and thus have greater choice over their own sleep-wake schedules.

Weekday waketimes were surprisingly constant across most of childhood and adolescence, averaging between 6:45 a.m. and 7:00 a.m. between ages 6 and 15 (Figure 2). A notable trend toward earlier waketimes on weekdays occurred between ages 3 and 5, when children first enter school. Weekday waketimes became later again at the end of adolescence, when children typically leave secondary school. In contrast, children's weekend waketimes changed considerably through late childhood and adolescence. Weekend waketime was fairly constant until age 9, when it begins to rise by about 15 min/year. Nine-year-olds woke up at 8:06 a.m. on average, while 17-year-olds woke up at 9:40 a.m. on average, perhaps attempting to compensate for deficits in their weekday sleep.

Unlike waketimes, which on weekdays stayed constant and on weekends became substantially later across adolescence, children's bedtimes on both weekends and weekdays became later as they aged. Weekday bedtime is fairly constant across ages 3-8; again, it is only at age 9 that it begins to rise substantially, from 9:00 p.m. for children aged 8 and under, to 9:30 p.m. for 11-year-olds, to 10:10 p.m. for 14-year-olds, to 11:00 p.m. for 17-year-olds. Weekend bedtimes also became later as children aged, from

Table 3
 OLS Regressions Examining the Influence of Sleep on Children's Subsequent BMI (Standardized)

Independent variable	All children (1)	Older children (2)	Younger children (3)
Model A: Linear measure of sleep time at Time 1 on subsequent BMI			
Hours of sleep	-0.115** (0.036)	-0.063 (0.046)	-0.153** (0.050)
R ²	0.238	0.356	0.179
Model B: Nonlinear measure of sleep time at Time 1 on subsequent BMI			
Sleeps less than 8 hr	0.108 (0.165)	-0.058 (0.163)	0.385 (0.308)
Sleeps 8–8.9 hr	0.033 (0.109)	0.025 (0.110)	0.107 (0.243)
Sleeps 9–9.9 hr	Omitted	Omitted	Omitted
Sleeps 10–10.9 hr	-0.164 ⁺ (0.086)	-0.162 ⁺ (0.098)	-0.191 (0.139)
Sleeps 11 hr or more	-0.257* (0.122)	0.009 (0.224)	-0.298* (0.151)
R ²	0.236	0.358	0.177
Model C: Linear measure of bedtime and waketime at Time 1 on subsequent BMI			
Bedtime (hr) ^a	0.116** (0.042)	0.029 (0.050)	0.189** (0.060)
Waketime (hr)	-0.120* (0.051)	-0.123 ⁺ (0.067)	-0.117 ⁺ (0.068)
R ²	0.238	0.359	0.181
Control variables from Model A ^b			
BMI at Time 1 ^a	0.341** (0.028)	0.410** (0.036)	0.290** (0.041)
Child is male	0.053 (0.069)	0.026 (0.086)	0.042 (0.104)
Child's age at Time 1	0.111 (0.162)	0.073 (0.201)	0.131 (0.227)
Child's age at Time 2	-0.109 (0.162)	-0.060 (0.205)	-0.093 (0.224)
Family income in \$10,000s	-0.004 (0.003)	-0.004 (0.004)	-0.005 (0.005)
Parental education	-0.056** (0.016)	-0.060** (0.019)	-0.047* (0.023)
Child is African American	0.103 (0.093)	0.101 (0.109)	0.092 (0.144)
Child is Hispanic	-0.018 (0.146)	0.062 (0.161)	-0.103 (0.227)
Child is other race	0.149 (0.169)	0.326 (0.228)	0.005 (0.229)
N	1,441	672	769

Note. BMI = body mass index.

Numbers in parentheses are standard errors.

Standard errors have been adjusted using Huber–White methods.

BMI at Times 1 and 2 is standardized using discussed protocols (see Method section).

^aYounger child coefficient is significantly different from older child coefficient at $p < .05$.

^bWe display the control variable coefficients from Model A only. Control variable coefficients for Models B and C are very similar.

⁺ $p < .10$, * $p < .05$, ** $p < .01$.

about 9:15 p.m. for children aged 7 and under, to 10:00 p.m. for 11-year-olds, to 10:30 p.m. for 14-year-olds, to 11:30 p.m. for 17-year-olds. As noted above, the trend for 18-year-olds is somewhat different, perhaps because so many of them are no longer in school; their average bedtimes were midnight for both weekends and weekdays.

In summary, weekday bedtimes become later throughout childhood and adolescence, while weekday waketimes remain fairly constant; thus children's weekday total sleep time declines by nearly two and a half hours between ages 3 and 17, from 10.5 to 8.1 hr. On weekends, both bedtimes and waketimes become later; the net decline in total weekend sleep is <1 hr between ages 3 and 18.

Sleep and BMI

We next turn to our primary question of the effect of sleep on children's standardized BMI (Table 3).

Model A presents the linear association between sleep and subsequent BMI; Model B allows for nonlinear associations; and Model C examines whether aspects of sleep timing, as reflected by bedtimes and waketimes, are associated with subsequent BMI. Although we included the same battery of control variables for Models A, B, and C, for the sake of brevity we only display the coefficients in Table 3 from Model A because they are very similar across the models.

In Model A, we find that for every additional hour of sleep at Time 1, children's BMI (standardized by age and gender) decreases by .12 *SD*. On average, this means an additional hour of sleep reduces BMI by about 0.75 kg/m², which, for the average child in the sample, means a BMI of 21.75 kg/m² rather than 22.5 kg/m² at Time 2.

Model B shows that children sleeping 11 or more hours a night have BMIs about .26 *SD* lower than children who slept between 9 and 10 hr at Time 1.

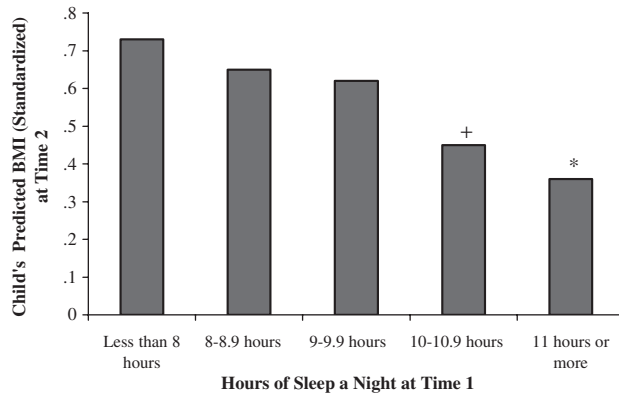


Figure 3. Hours of sleep a night at Time 1 and children's predicted standardized body mass index (BMI) at Time 2.

Children who sleep between 10 and 11 hr a night have BMIs that are .16 *SD* lower than those who slept between 9 and 10 hr at Time 1, although this result is significant only at $p < .10$. The coefficients from the dummy models suggest that sleep has a nonlinear association with children's future BMI; only for sleep above 10 hr a night was there a (marginally significant) influence on children's BMI, and only above 11 hr a night that we see an association that was statistically significant at conventional levels. This monotonic but somewhat nonlinear pattern is shown in Figure 3.

We also find strong associations between BMI and both bedtime and waketime (Model C). For every additional hour the child stays up at night before bedtime at Time 1, BMI at Time 2 increases by .12 *SD*. For every additional hour in the morning the child was asleep, BMI decreases by .12 *SD*.

We also examine whether these associations differed by age and find that additional hours of sleep for younger children are associated with a statistically significant .15 *SD* decrease in BMI, while for older children the coefficient suggests a statistically insignificant .06 *SD* decrease. These two coefficients, however, are not statistically different from one another at standard levels of significance, and hence we cannot conclude that our linear measure of sleep has a different effect on younger than older children.

In Model B, which models a nonlinear relationship between sleep and BMI, we find that younger children sleeping 11 hr or more nightly experience a .30 *SD* decrease in their BMI compared with the reference group, while older children's coefficient is .01 *SD*. Again, however, these coefficients are not different from one another at standard levels of significance.

In Model C, we examine interactions between age and bedtime and waketime behaviors and find

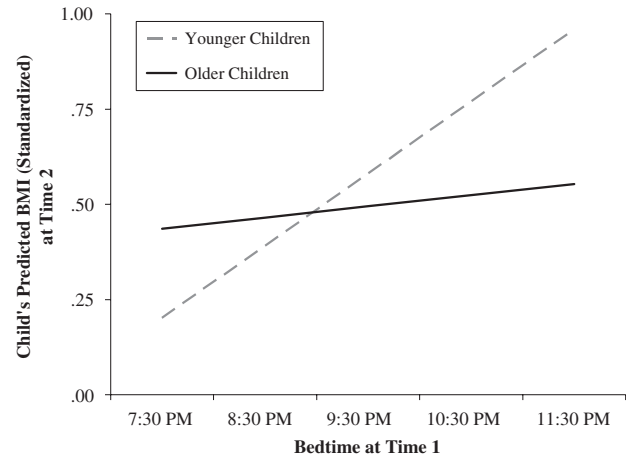


Figure 4. Bedtime at Time 1 and children's predicted standardized body mass index (BMI) at Time 2: interaction by age group.

significant age interactions for bedtime, but not for waketime. For every additional hour children are awake at night before bedtime, younger children experience a .19 *SD* increase in BMI, while older children experience a .03 *SD* increase; these two results are different from one another at a $p < .05$ level. The graphical representation of the interaction between bedtime and age is shown in Figure 4. For an average younger child, going to bed an hour later translates into a BMI of 21.8 kg/m² rather than 20.7 kg/m² at Time 2. We fail to find an interaction between age and waketime, as both younger and older groups experience a marginally significant decline in BMI as their average waketime becomes later. Both younger and older children experience a .12 *SD* decrease in BMI for every additional hour they stay asleep in the morning.

Overall, the results suggest that although total sleep time is related to both older (8–12.9 years) and younger children's (3–7.9 years) subsequent BMI, later bedtimes appear to have a greater effect on younger children's weight. Although greater amounts of sleep (particularly over 11 hr), earlier bedtime, and later waketime were all associated with a lower BMI for younger children, only sleeping 10–11 hr and having a later waketime were associated with a lower BMI for older children, and only at the $p < .10$ level.

Sleep and Overweight Status

We were also interested in assessing the associations between sleep time and a child's risk for subsequent overweight status, controlling for baseline overweight status. Table 4 presents marginal effects from a logistic regression of children's over-

Table 4
 Logistic Regressions Examining the Marginal Effects of Sleep on Children's Subsequent Overweight Status

Independent variable	All children (1)	Older children (2)	Younger children (3)
Model A: Linear measure of sleep time at Time 1 on subsequent overweight			
Hours of sleep	-0.053** (0.017)	-0.045 ⁺ (0.026)	-0.061** (0.023)
R ²	0.128	0.240	0.074
Model B: Nonlinear measure of sleep time at Time 1 on subsequent overweight			
Sleeps less than 8 hr	-0.009 (0.073)	-0.093 (0.089)	0.084 (0.126)
Sleeps 8–8.9 hr	0.014 (0.054)	0.036 (0.072)	0.041 (0.092)
Sleeps 9–9.9 hr	Omitted	Omitted	Omitted
Sleeps 10–10.9 hr	-0.070 ⁺ (0.044)	-0.084 (0.066)	-0.062 (0.060)
Sleeps 11 hr or more	-0.171** (0.063)	-0.108 (0.115)	-0.158** (0.072)
R ²	0.129	0.242	0.074
Model C: Linear measure of bedtime and waketime at Time 1 on subsequent overweight			
Bedtime (hr) ^a	0.056** (0.019)	0.026 (0.030)	0.083** (0.027)
Waketime (hr)	-0.058* (0.024)	-0.093* (0.040)	-0.039 (0.029)
R ²	0.129	0.245	0.078
Control variables from Model A ^b			
Overweight at Time 1 ^a	0.343** (0.036)	0.506** (0.057)	0.208** (0.049)
Child is male	0.095** (0.035)	0.116* (0.053)	0.055 (0.045)
Child's age at Time 1	0.121 (0.078)	0.188 (0.116)	0.089 (0.097)
Child's age at Time 2	-0.122 (0.077)	-0.176 (0.118)	-0.062 (0.095)
Family income in \$10,000s	0.001 (0.002)	0.003 (0.002)	-0.002 (0.003)
Parental education	-0.027** (0.008)	-0.033** (0.011)	-0.020 ⁺ (0.011)
Child is African American	0.019 (0.044)	-0.026 (0.066)	0.027 (0.056)
Child is Hispanic	-0.005 (0.069)	-0.028 (0.099)	0.015 (0.091)
Child is other race ^a	0.153 ⁺ (0.084)	0.313* (0.123)	0.041 (0.103)
N	1,441	672	769

Note. Numbers in parentheses are standard errors.

Standard errors have been adjusted using Huber–White methods.

Coefficients represent the marginal effect (percentage point change) on the mean probability of being overweight.

^aYounger child coefficient is significantly different from older child coefficient at $p < .05$.

^bWe display the control variable coefficients from Model A only. Control variable coefficients for Models B and C are very similar.

⁺ $p < .10$, * $p < .05$, ** $p < .01$.

weight status at Time 2 on Time 1 sleep measures, Time 1 overweight status, and control variables. The marginal effect represents the association between an additional unit of sleep measurement and the subsequent probability of being overweight. Using a linear measure of sleep, we find that an additional hour of sleep is associated with a 5.3 percentage point decline in the probability of being overweight (Model A). Results from the nonlinear or dummy measure of sleep (Model B) indicate that, compared with those sleeping 9–9.9 hr a night, those children who slept more than 11 hr a night experienced a 17.1 percentage point decline in the probability of being overweight. We find that the marginal effect of going to sleep one additional hour later (Model C) is a 5.6 percentage point increase in the probability of being overweight while waking up 1 hr later than average results in a 5.8 percentage point decrease in the probability of being overweight.

We also present the results of our regression model by age subgroups in Table 4. Interactions are statistically insignificant in both the linear and dummy-variable models, although in the latter only younger children experience a significant 16 percentage point decline in subsequent probability of being overweight when they sleep 11 hr or more.

We also investigated whether the regression-adjusted associations depended on the distinction between obesity and overweight. Coefficients were somewhat smaller in the obesity analysis.

Control Variables

Table 3 shows that a child's Time 1 BMI is a very strong predictor of their Time 2 BMI. In addition, parental education is also a significant predictor. For every additional year of average parental education,

children's BMI is .06 *SD* lower at Time 2. Family income, child race, and child gender, however, do not have significant associations with Time 2 BMI. The results are similar for overweight status (Table 4): Time 1 overweight status and parental education both are associated with Time 2 BMI. Males and older children of "other" racial status are also more likely to become overweight at Time 2.

Additional Analyses

Interactions between sleep and child's gender were conducted and found not to be significant. We were also interested in whether other behaviors at Time 1 might mediate the relationship between sleep and BMI. We ran a regression that included sleep measures as well as the amount of physical activity children were getting at Time 1, measured by the amount of time they spent playing active games or sports as recorded in the time diary and found no evidence that physical activity mediates the pathway between sleep and BMI. We were also interested in napping and resting behaviors. By age, the following percentage napped or rested and for those who napped or rested, their total amount of time napping or resting was: 3-year-olds, 67%, 83 min; 4-year-olds, 38%, 64 min; 5-year-olds, 28%, 44 min; 6-year-olds, 25%, 40 min; and 7-year-olds, 19%, 53 min. About 14% of children aged 8–12 napped or rested (averaging 54 min) while about 23% of children aged 13–17 napped or rested (averaging 58 min). When we included a measure of whether children were napping or resting in our models, the coefficient on the sleep measure declined only slightly.

We also investigated whether children who were sleeping more at Time 1 were less likely to be overweight at Time 2 because they were growing more in height but not weight. We did this with regressions examining the influence of sleep on standardized height and weight. We found no evidence that children who were sleeping more were also growing more. In fact, children who slept more were .05 *SD* shorter at Time 2, although this effect was statistically insignificant. However, these children also gained much less weight. For every additional hour of sleep, they weighed .09 *SD* less ($p < .01$). Consequently, their standardized BMI decreased compared with their peers.

Discussion

Using nationally representative, longitudinal data on American children, we describe developmental

trends in sleep behaviors across childhood and adolescence and estimate longitudinal models of the association between sleep and children's subsequent BMI and overweight status.

Age Trends in Sleep Behaviors

Ours is the first study that presents nationally representative estimates of sleep behaviors, recorded in time diaries, for children aged 3–18 on both weekends and weekdays. We document large declines in weekday sleep across middle childhood and adolescence, driven largely by later weekday bedtimes. We also find major differences in weekday versus weekend sleep behaviors, even among younger children, but particularly among older children.

Although our average sleep times for younger children were similar to what has been reported in other studies (Bates et al., 2002), average weekday sleep times for adolescents were at least 1 hr greater than what has been reported in another large study of adolescent sleep behaviors (Wolfson & Carskadon, 1998). This difference could be caused by differences in self-report versus time diary report of sleep length and/or by the very early start times at the four Rhode Island high schools from which Wolfson and Carskadon drew their sample.

For example, responses to separate questionnaires administered to adolescents in our sample show that, when asked how much they normally slept on weekdays, most reported lengths of time similar to those reported by Wolfson and Carskadon (1998). Although respondents were asked to differentiate between time in bed and time asleep in their time diaries, it is possible that respondents mistakenly included time in bed watching television or reading, but not actual sleeping. It is also possible that respondents tend to underestimate how much time they actually sleep when they use stylized or global reports, which seems reasonable given the current social pressure for both adolescents and adults to do more and sleep less.

The other possibility is that because the school day rise time was very early in the Rhode Island sample, nearly an hour earlier than in our sample, this led to lower total sleep times for the Wolfson and Carskadon report. Further evidence that it is in part the influence of school start time on waketime that drives the differences in total hours, rather than inaccuracies in reporting, comes from the fact that the CDS sample and the Rhode Island samples report going to bed on weekdays at about the same time.

Many studies have reported adolescents' shifts toward later bedtimes and waketimes, particularly on weekends (Wolfson & Carskadon, 1998). Other studies have suggested that older adolescents' natural time to fall asleep is 11:00 p.m. or later, and we also find that older adolescents report weekend and weekday bedtimes nearing 11:00 p.m. However, we also find evidence that this shift toward later weekday bedtimes might begin earlier than some researchers have suspected, starting around ages 8 or 9. Delay in weekend waketimes also followed a clear age trend; starting around ages 8 or 9, children woke up on average shortly later every year such that 17- and 18-year-olds woke up on average at 9:40 a.m. on the weekends, about 1 hr and 40 min later than 8-year-olds.

Thus, an additional finding of this study is that a shift in bedtimes and waketimes may begin in pre-adolescence, as early as ages 8 or 9. This finding may be of interest not only to sleep researchers but also to those concerned about the mismatch between children's sleep rhythms and early school start times. Although prior research has highlighted the mismatch for high school-aged students, our results suggest that early school start times might be problematic for middle school students, or even elementary school students as well. Indeed, our analysis of the effects of waketime and bedtime on subsequent weight problems finds that while younger children (aged 3–7.9) experience harmful effects on BMI and overweight status from staying up late, older children (aged 8–12.9) do not, but that both groups experience marginally significant negative effects on BMI from getting up early (older children also experience negative effects on overweight status).

Our descriptive results of sleep behaviors highlight the fact that weekday and weekend sleep behaviors differ quite drastically from one another in a naturalistic setting. This implies that those interested in studying children's and adolescents' sleep may benefit from assessing both weekday and weekend sleep behaviors. Although researchers studying adolescent sleep have long noted different patterns for weekday versus weekend sleep, we find that these differences apply for younger children as well. Even children aged 3–5 years had different sleep patterns on the weekday versus weekend, with weekday waketimes 25–45 min earlier than weekend waketimes (e.g., 7:29 a.m. on weekdays and 7:58 a.m. on weekends for 4-year-olds). It is likely that these differences for the youngest age group are driven at least in part by the sleep choices and preferences of the older members of their households; regardless of origin, these results suggest that the weekend–

weekday sleep differential is a pattern that begins early in life. This differential is of potential concern for health and well-being, given prior research showing that variability in sleep timing is associated with negative outcomes beyond the effects of total hours of sleep (Acebo & Carskadon, 2002).

Sleep experts have suggested that children and adolescents need, at a minimum, 9–10 hr of sleep a night (Carskadon et al., 1980; Mercer, Merritt, & Cowell, 1998). Our study finds that the majority of children and adolescents achieve 10 hr of sleep on weekend nights. Children's and adolescents' weekday sleep, however, is more troubling. By age 7, children are achieving less than 10 hr of sleep on weekday nights, and by age 14, children are achieving less than 9 hr of sleep on weekday nights. A substantial minority of children sleep even less on weekdays: about 13% of all children aged 3–7 years slept less than 9 hr, 11% of children aged 8–12 years slept less than 8 hr, and 16% of children aged 13–18 years slept less than 7 hr on weekday nights. The fact that a substantial portion of American children achieve such small amounts of sleep should be of concern in light of findings from prior studies suggesting associations between poor sleep hygiene and decreased cognitive and social functioning as well as the findings from our own study that suggest that such sleep deficits may contribute to the development of weight problems.

Sleep and Subsequent BMI and Overweight Status

Children who sleep more are estimated to have lower BMIs and lower rates of overweight status 5 years later, controlling for their baseline BMI or overweight status and demographic variables including parent income and education and child race/ethnicity. Our subgroup analyses indicate that sleep matters more for younger children's BMI and overweight status than older children's BMI and overweight status, but that child gender does not moderate the relationship between sleep and weight.

Our results suggest that even one additional hour of sleep may have a significant and meaningful effect on BMI and overweight status; for example, the chance of being overweight at Time 2 declines by 5.3 percentage points, from 35.6% to 30.3%, when average sleep length at Time 1 increases from the sample average of 9.9–10.9 hr.

Our measure of sleep has limitations because it comes from a single assessment of time allocation on one randomly chosen weekday and weekend day. These selected days are thus a sample of days in the year and are subject to sampling error, which can

impart downward bias to our estimates of the association between sleep and subsequent BMI. A virtue of our measure of sleep is that it is derived from a time diary report and is thus less likely to be subject to recall bias or socially desirable reporting; the days reported on were also randomly selected weekdays and weekend days, making them representative of all weekdays and weekends in the lives of the sample children. An additional benefit is that sleep is not measured concurrently with our outcome of interest, and prior weight is included in the model, which means that our results are not plagued by concerns of reverse causality.

Another potential concern is the role that puberty might play in the associations between sleep and weight. Because BMI increases dramatically during puberty, and because sleep amounts can influence the endocrine hormones that affect the timing and onset of puberty, puberty could act as a major mediator or moderator of causal linkages between sleep and weight. In addition, because the onset of puberty pertains more to our older group of children, this could have implications for our findings that sleep matters more for younger than older children. That is, perhaps we find that sleep matters more for younger children's BMI because our inability to account for pubertal status weakens the association between BMI and sleep for older children. Unfortunately, the CDS does not measure children's pubertal status and therefore we are unable to investigate these possibilities in the context of this study.

Causal Pathways Between Sleep and Weight

What might be the mediating pathway between inadequate sleep and subsequent weight gain? Recent findings from the biomedical literature suggest that inadequate sleep might disrupt hormones that regulate appetite and metabolism, with insufficient sleep hours causing reduced levels of leptin and increased levels of ghrelin, a hormonal profile associated with increased hunger and appetite for carbohydrate-rich foods (Spiegel, Tasali, Penev, & Van Cauter, 2004). Many recent papers suggest that these hormones might be implicated in the link between adult sleep and obesity (Patel et al., 2004; Saresranta & Polo, 2004; Taheri, Lin, Austin, Young, & Mignot, 2004). Our findings suggest that the association between sleep and obesity may be just as important for children, particularly younger children, although future research needs to identify whether similar biological mechanisms are at work.

The relationship between sleep and subsequent weight is much stronger for younger (aged 3–7.9

years) than older children (aged 8–12.9 years). Although the trends were generally in the same direction as younger children, only one coefficient for older children reached a standard level of statistical significance: an additional hour of sleep in the morning was associated with a 9.3 percentage point decrease in their likelihood of being overweight or obese (note, however, that it was not statistically different from the younger child coefficient).

Why might the associations between sleep behaviors and weight be stronger for younger children? Although we saw a shift to later bedtimes starting with younger children and continuing through adolescence, perhaps this shift to later bedtimes (resulting in reduced total sleep hours) is less developmentally appropriate for younger children, resulting in a more detrimental effect. There is clear evidence for the appropriateness of later bedtimes for adolescents, as these changes in bedtimes may be biologically driven (Carskadon et al., 2004). For younger children, however, the change to later bedtimes may be driven more by social factors rather than changes in biology, resulting in a mismatch between sleep schedules and biological needs regarding sleep timing. Given the evidence from the sleep literature that later bedtimes, as well as later waketimes, may be the natural pattern for pubertal adolescents (Carskadon et al., 2004), however, it makes sense that adolescents would attain greater benefits from increased sleep hours due to later waketimes than they do from earlier bedtimes.

Our analytic strategy attempts to reduce the likelihood of omitted variables explaining the relationship between sleep and weight both by including control variables and by using a longitudinal design. Our results suggest that parental education, but not parental income, plays a role in children's BMI and overweight trajectories. Children whose parents were more highly educated had lower BMIs and were less likely to be overweight at Time 2, controlling for all other factors. In addition, being male or an older child of "other race" was associated with a higher probability of being overweight at Time 2. These results suggest that parental education, or parental behaviors associated with higher levels of education, may play an important role in children's weight. Despite including these control variables, because we were still concerned that it was other differences between children and families that caused the association between sleep and weight, we also ran a set of additional regressions that examined whether differences in time spent napping or resting and time spent being physically active could explain the relationship between sleep and subsequent

weight. We failed to find any mediating effect of either physical activity or napping/resting. However, the measures we had of both physical activity and napping/resting were not ideal, and further research is needed to examine the links between physical activity, napping, sleep behaviors, and children's overweight status.

Implications for Policy and Practice

Our results suggest that sleep in childhood may be linked to subsequent weight and overweight status. Moreover, earlier bedtime may be more important for younger children's and later waketime more important for older children's subsequent BMI and overweight status. If so, parents should be encouraged to put their younger children to bed early enough so that they can sleep at least 10 or 11 hr a night. For older children, however, only later waketimes were associated with lower BMI and lower rates of overweight. This result supports findings from the growing sleep literature encouraging later school start times, particularly for adolescents. For example, a study by Wahlstrom and Freeman (1997) found that high-school students who attended schools that delayed their start times reported increased hours of sleep, less erratic sleep behaviors and less depressive feelings and behaviors, and better grades. Another recent study found that earlier high school start times contribute to sleep deprivation (Hansen, Janssen, Schiff, Zee, & Dubocovich, 2005). A combination of strategies targeting both earlier bedtimes and later waketimes, with the relative emphasis on each dependent on children's ages, might well improve multiple aspects of children's health, emotional well-being, and academic performance.

Our findings on the association between sleep and BMI and overweight status have particularly important implications for those concerned about the American obesity epidemic. Overweight children suffer from reduced socioemotional and physical well-being in childhood, and obese children have quality-of-life levels similar to children with cancer (Schwimmer, Burwinkle, & Varni, 2003). In addition, overweight and obese children are more likely to be overweight and obese as adults (Strauss, 1999), and thus suffer the wide range of social and physical health problems associated with adult obesity, including increased risk for heart disease, diabetes, some cancers, and decreased life expectancy (Halsam & James, 2005). Adult overweight and even borderline overweight have been associated with an increased risk for morbidity as well; in adults, hav-

ing a BMI of 23 or above—below the cutoff for overweight status—is associated with increased risk of diabetes (Halsam & James, 2005).

As a result, public health officials and the medical research community have called for a better understanding of the etiology of overweight and obesity and better treatments for childhood weight problems. The effect of an additional hour of sleep on changes in BMI and changes in overweight status rival the effects from successful childhood obesity interventions reviewed in Epstein, Myers, Raynor, and Saelens (1998). If our results represent a true causal relationship between sleep and weight, encouraging parents to put their younger children to bed earlier at night and allowing both younger and older children to sleep longer in the morning, as well as urging school districts to avoid very early school start times for later elementary and middle school-aged children, might represent an important and relatively low-cost strategy to reduce childhood weight problems.

References

- Acebo, C., & Carskadon, M. A. (2002). Influence of irregular sleep patterns on waking behavior. In M. A. Carskadon (Ed.), *Adolescent sleep patterns: Biological, social and psychological influences* (pp. 220–235). New York: Cambridge University Press.
- Agras, W. S., Hammer, L. D., McNicholas, F., & Kraemer, H. C. (2004). Risk factors for childhood overweight: A prospective study from birth to 9.5 years. *Journal of Pediatrics*, *145*, 20–25.
- Bass, J., & Turek, F. W. (2005). Sleepless in America: A pathway to obesity and the metabolic syndrome? *Archives of Internal Medicine*, *165*, 15–16.
- Bates, J. E., Viken, R. J., Alexander, D. B., Beyers, J., & Stockton, L. (2002). Sleep and adjustment in preschool children: Sleep diary reports by mothers relate to behavior reports by teachers. *Child Development*, *73*, 62–75.
- Carskadon, M. A., Acebo, C., & Jenni, O. G. (2004). Regulation of adolescent sleep: Implications for behavior. *Annals of the New York Academy of Sciences*, *1021*, 276–291.
- Carskadon, M. A., Harvey, K., Duke, P., Anders, T. F., Litt, I. F., & Dement, W. C. (1980). Pubertal changes in daytime sleepiness. *Sleep*, *2*, 453–460.
- Cole, T. J. (1990). The LMS method for constructing normalized growth standards. *European Journal of Clinical Nutrition*, *44*, 45–60.
- Cole, T. J., Bellizzi, M. C., Flegal, K. M., & Dietz, W. H. (2000). Establishing a standard definition for child overweight and obesity worldwide: International survey. *British Medical Journal*, *320*, 1240–1243.
- Dahl, R. E., & Lewin, D. S. (2002). Pathways to adolescent health: Sleep regulation and behavior. *Journal of Adolescent Health*, *31*, 175–184.

- Epstein, L. H., Myers, M., Raynor, H. A., & Saelens, B. E. (1998). Treatment of pediatric obesity. *Pediatrics*, *101*, 554–570.
- Fredriksen, K., Rhodes, J., Reddy, R., & Way, N. (2004). Sleepless in Chicago: Tracking the effects of adolescent sleep loss during the middle school years. *Child Development*, *75*, 84–95.
- Gupta, N. J., Mueller, W. H., Chan, W., & Meininger, J. C. (2002). Is obesity associated with poor sleep quality in adolescents? *American Journal of Human Biology*, *14*, 762–768.
- Halsam, D. W., & James, W. P. T. (2005). Obesity. *Lancet*, *366*, 1197–1209.
- Hansen, M., Janssen, I., Schiff, A., Zee, P. C., & Dubocovich, M. L. (2005). The impact of school daily schedule on adolescent sleep. *Pediatrics*, *115*, 1555–1562.
- Hofferth, S., & Sandberg, J. (2001). How American children spend their time. *Journal of Marriage and the Family*, *63*, 295–308.
- Juster, F. T., Ono, H., & Stafford, F. P. (2003). An assessment of alternative measures of time use. *Sociological Methodology*, *33*, 19–54.
- Locard, E., Mamelle, N., Billette, A., Miginiac, M., Munoz, F., & Rey, S. (1992). Risk factors of obesity in a five year old population. Parental versus environmental factors. *International Journal of Obesity and Related Metabolic Disorders*, *16*, 721–729.
- Marcus, C. L., Curtis, S., Koerner, C. B., Joffe, A., Serwint, J. R., & Loughlin, G. M. (1996). Evaluation of pulmonary function and polysomnography in obese children and adolescents. *Pediatric Pulmonology*, *21*, 176–183.
- Mercer, P. W., Merritt, S. L., & Cowell, J. M. (1998). Differences in reported sleep need among adolescents. *Journal of Adolescent Health*, *23*, 259–263.
- Nieto, F. J., Young, T. B., Lind, B. K., Shahar, E., Samet, J. M., Redline, S., et al. (2000). Association of sleep-disordered breathing, sleep apnea, and hypertension in a large community-based study. Sleep heart health study. *Journal of the American Medical Association*, *283*, 1829–1836.
- Patel, S. R., Palmer, L. J., Larkin, E. K., Jenny, N. S., White, D. P., & Redline, S. (2004). Relationship between obstructive sleep apnea and diurnal leptin rhythms. *Sleep*, *27*, 235–239.
- Reilly, J. J., Armstrong, J., Dorosty, A. R., Emmett, P. M., Ness, A., Rogers, I., et al. (2005). Early life risk factors for obesity in childhood: A cohort study. *British Medical Journal*, *330*, 1357–1362.
- Saareanta, T., & Polo, O. (2004). Does leptin link sleep loss and breathing disturbances with major public diseases? *Annals of Medicine*, *36*, 172–183.
- Sadeh, A., Gruber, R., & Raviv, A. (2003). The effects of sleep restriction and extension on school-age children: What a difference an hour makes. *Child Development*, *74*, 444–455.
- Schwimmer, J. B., Burwinkle, T. M., & Varni, J. W. (2003). Health related quality of life for severely obese children and adolescents. *Journal of the American Medical Association*, *289*, 1813–1819.
- Sekine, M., Yamagami, T., Hamanishi, S., Handa, K., Tomohiro, S., & Nanri, S. (2002). Parental obesity, lifestyle factors and obesity in preschool children: Results of the Toyama Birth Cohort Study. *Journal of Epidemiology*, *12*, 33–39.
- Spiegel, K., Tasali, E., Penev, P., & Van Cauter, E. (2004). Brief communication: Sleep curtailment in healthy young men is associated with decreased leptin levels, elevated ghrelin levels, and increased hunger and appetite. *Annals of Internal Medicine*, *141*, 846–850.
- Strauss, R. S. (1999). Childhood obesity. *Current Problems in Pediatrics*, *29*, 1–29.
- Taheri, S., Lin, L., Austin, D., Young, T., & Mignot, E. (2004). Short sleep duration is associated with reduced leptin, elevated ghrelin, and increased body mass index. *PLoS Medicine*, *1*, 210–217.
- Vgontzas, A. N., Bixler, E. O., & Chrousos, G. P. (2005). Sleep apnea is a manifestation of the metabolic syndrome. *Sleep Medicine Review*, *9*, 211–224.
- Vidmar, S., Carlin, J., Hesketh, K., & Cole, T. (2004). Standardizing anthropometric measures in children and adolescents with new functions for egen. *The Stata Journal*, *4*, 50–55.
- Wahlstrom, K. L., & Freeman, C. M. (1997). School Start Time Study: Final report summary. The Center for Applied Research and Educational Improvement, College of Education and Human Development, University of Minnesota.
- Wolfson, A. R., & Carskadon, M. A. (1998). Sleep schedules and daytime functioning in adolescents. *Child Development*, *69*, 875–887.
- Wolfson, A. R., & Carskadon, M. A. (2003). Understanding adolescents' sleep patterns and school performance: A critical appraisal. *Child Development*, *69*, 875–887.