Objective: To test associations between daytime and nighttime sleep duration and subsequent obesity in children and adolescents.

Design: Prospective cohort.


Participants: Subjects aged 0 to 13 years (n=1930) at baseline (1997).

Main Exposures: Binary indicators of short daytime and nighttime sleep duration (<25th percentile of age-normalized sleep scores) at baseline.

Main Outcome Measures: Body mass index at follow-up (2002) was converted to age- and sex-specific z-scores and trichotomized (normal weight, overweight, obese) using established cut points. Ordered logistic regression was used to model body mass index classification as a function of short daytime and nighttime sleep at baseline and follow-up, and important covariates included socioeconomic status, parents' body mass index, and, for children older than 4 years, body mass index at baseline.

Results: For younger children (aged 0-4 years at baseline), short duration of nighttime sleep at baseline was strongly associated with increased risk of subsequent overweight or obesity (odds ratio = 1.80; 95% confidence interval, 1.16-2.80). For older children (aged 5-13 years), baseline sleep was not associated with subsequent weight status; however, contemporaneous sleep was inversely associated. Daytime sleep had little effect on subsequent obesity in either group.

Conclusions: Shortened sleep duration in early life is a modifiable risk factor with important implications for obesity prevention and treatment. Insufficient nighttime sleep among infants and preschool-aged children may be a lasting risk factor for subsequent obesity. Napping does not appear to be a substitute for nighttime sleep in terms of obesity prevention.

During the last 3 decades, the prevalence of overweight and obesity has increased dramatically among children and adolescents. Obesity—defined as having age- and sex-specific body mass index (BMI; calculated as weight in kilograms divided by height in meters squared) at or above the 95th percentile of national growth standards—has doubled among children aged 2 to 5 years and adolescents aged 12 to 19 years and has tripled among those aged 6 to 11 years. In 2003 to 2004, 17% of children and adolescents were obese and 34% were overweight (defined as having BMI >85th and <95th percentiles of the same standards). Short sleep duration may increase the risk of obesity in children and adolescents. Evidence is accumulating from cross-sectional population studies to support a robust contemporaneous relationship between shortened sleep duration and unhealthy weight status in children and adolescents. In several studies, a strong dose-response relationship is evident with increasing odds of overweight/obesity associated with fewer hours spent sleeping. The mechanisms through which sleep operates to influence the balance between energy intake and expenditure are unknown. Shortened sleep duration has been hypothesized to influence weight status through decreased physical activity due to tiredness and increased energy intake given greater opportunity to eat. An equally compelling pathway may be through influence on the hypothalamic mechanisms that regulate body weight and metabolism via key hormones such as leptin and ghrelin. Hunger and appetite increase with lower leptin levels and higher ghrelin levels, and both low leptin
and high ghrelin levels have been linked to short sleep duration in adults, however, less is known about these relationships in children.

To date, most studies of sleep duration and obesity in children and adolescents have been cross-sectional and it is unknown whether the relationship is truly causal. Very few studies have assessed prospective associations of sleep and obesity. A US sample followed from birth to age 3 years generated 2 studies showing that sleep duration of less than 12 hours in infancy was associated with increased odds of overweight and with higher BMI z-scores and skinfold thickness all measured at age 3 years. A third study of 150 children found that children’s hours of sleep at ages 3 and 4 years were associated with increased odds of overweight at age 9.5 years. Finally, a study of 900 children in the United Kingdom found that shortened sleep duration at age 3 years was associated with obesity at age 7 years. Population-based studies are required to test these associations over longer follow-up periods and in larger samples. Moreover, longitudinal studies are needed to examine the independent effects of daytime and nighttime sleep on obesity as none of the prior studies made this distinction. Although the precise physiological functions of sleep are not fully understood, daytime sleep and nighttime sleep may have independent functions and thus distinct effects on subsequent obesity.

The current study uses existing national, longitudinal, panel survey data collected for children and adolescents to test the following: (1) whether poor sleep in early life has lasting effects on obesity measured 5 years later; (2) whether daytime and nighttime sleep have independent effects on subsequent obesity; and (3) whether the relationship between sleep and obesity is different for adolescents and preadolescents.

**METHODS**

We conducted a longitudinal analysis of the Panel Survey of Income Dynamics (PSID) Child Development Supplement (CDS) collected for the same children and adolescents in 1997 and again in 2002. Data are in the public domain and the study was approved by the Institutional Review Board of Children’s Hospital and Regional Medical Center in Seattle, Washington.

**DATA SOURCE**

The PSID is a longitudinal survey directed by the National Science Foundation. Since 1968, 4800 families have been followed. In 1997 (ie. baseline) and 2002 (ie. follow-up), a CDS funded by the National Institute of Child Health and Human Development was administered to the primary caregivers of 3563 children aged 0 to 13 years. The CDS questionnaire included detailed demographic data as well as psychological and behavioral assessments of parents and children. Time-use diary data were collected from 1 randomly chosen weekday and 1 randomly chosen weekend day during a school year (September-May). The time diaries recorded both primary and secondary activities for each child during a 24-hour period. Such time diaries have been used extensively in research and have excellent validity when compared with direct observation of activities.

**SAMPLE**

Of the 2569 children in the PSID at follow-up, 990 were aged 0 to 59 months (younger cohort) and 1579 were aged 60 to 154 months (older cohort) at baseline. Children were excluded from the study if they were underweight (n=24) or had implausibly low BMI (<12) or missing BMI at follow-up or if they had missing time diary data that provide the estimates of sleep time. The total final sample size was 822 children in the younger cohort and 1108 children in the older cohort at baseline measurement. We selected age 5 years as the sample cut point for assignment to the younger vs older cohort to account for differences in sleep patterns between preschool-aged and school-aged children. By age 5 years, the vast majority of children do not take naps. Of the older children, 125 were missing BMI at baseline. Accordingly, the models included baseline BMI were estimated with a sample size of 983. The BMI at baseline was not available for the younger children because height and weight were not assessed in the 1997 survey for children younger than 5 years.

**DEPENDENT VARIABLE**

The dependent variable was BMI in 2002, converted to age- and sex-specific z scores using the 2000 growth charts published by the Centers for Disease Control and Prevention and tri-chotomized based on established cut points: normal weight (BMI ≤85th percentile), overweight (BMI >85th and <95th percentiles), or obese (BMI ≥95th percentile). Both height and weight were measured at follow-up, whereas at baseline the height of children aged 5 years and older was measured and their weight was recorded from parental report.

**INDEPENDENT VARIABLES**

Family time data were used to calculate the average duration of daytime and nighttime sleep in hours at baseline and at follow-up. Sleeping hours differ with normal growth and development, with younger age associated with longer sleeping hours. Accordingly, we developed age-normalized sleep scores for each child in the data set using external norms derived in a large sample of Swiss children. While Swiss children tend to sleep a little more on average than the children in this study, using an external data set has the advantage of avoiding the additional estimation error that would come from using within-sample norms, particularly given the relatively small sample size. No other data sources were found that could be used to compute sleep norms in US children across our study’s age range, and no articles describing such norms were found. The age-specific sleep scores were used to develop dichotomous indicators of low sleep for age—identifying those children below the 25th percentile in sleep for age—separately by category of daytime sleep and nighttime sleep. The 25th percentile was chosen to identify children and adolescents who get a low amount of sleep for their age while classifying enough of them as having low sleep to retain adequate statistical power.

More than 90% of children in the sample did not nap at follow-up when all were at least aged 5 years, and most of those who napped did so for a very short duration (<30 minutes). The results were completely robust to the inclusion of napping at follow-up; however, as this variable contained little information and was not significant, it was subsequently dropped from the analysis.

**COVARIATES**

All regression models control for child and family attributes that could affect sleep and obesity. Covariates include child age, sample mean, and baseline BMI. The BMI at baseline was not available for the younger children because height and weight were not assessed in the 1997 survey for children younger than 5 years.
at baseline (linear and quadratic terms in months), sex (female; male [reference]), race/ethnicity (white, non-Hispanic [reference]; black, non-Hispanic; Hispanic), birth order (only child [reference]; youngest; middle; oldest), family income in 1997 (in dollars, log-transformed), maternal education at baseline (in years, log-transformed), the child’s birth weight (in grams), and the mother’s and father’s BMI (each self-reported in 1999). The parents’ BMI is a proxy for both the diet and physical activity patterns in the household as well as genetic factors that might influence the child’s BMI. Indicator variables were included for low birth weight (<2500 g), macrosomia (>4500 g), and presence of the father in the household (each coded 1 for yes or 0 for no). Television viewing and other media may disrupt sleep patterns of children and adolescents; therefore, continuous variables representing the hours per day of entertainment television and noncommercial television (eg, educational programs or DVDs) watched at baseline and at follow-up were included as controls. For the older cohort only, time diary data were used to compute the hours per day in which the child engaged in physical activity at baseline and at follow-up.

For children older than 5 years, the child’s baseline BMI z score was controlled to account for the possibility that an association between sleep duration and subsequent obesity reflects an unmeasured tendency of obese children to sleep fewer hours. As height and weight were not measured for the younger cohort at baseline, it was not possible to include baseline BMI in regression models for this age group.

None of the covariates had more than 10% missing data. For each, missing data points were imputed with the sample mean or mode. An alternative approach of dropping observations from the sample if covariates were missing produced similar findings.

### Statistical Analysis

All statistical analysis was conducted with Stata version 10.1 statistical software (StataCorp LP, College Station, Texas) and developed separately for the younger and older cohorts. Two models were estimated in the older cohort, one including baseline BMI and one without this variable. Results from the 2 models were compared to assess the contribution of baseline BMI.
with the estimated association between sleep duration and subsequent BMI. This comparison allows us to gauge whether missing baseline BMI in the younger cohort is likely to affect the results. Because the dependent variable included 3 ordered categories, ordered logistic regression was used to model child weight status at follow-up as a function of sleep duration at baseline and at follow-up, controlling for all covariates and, for children older than 5 years, the child’s baseline BMI. The assumption of proportional odds required for ordered logistic regression was tested formally. In all models, sampling weights were used to account for the complex sampling design and to allow inferences valid for the population.

### RESULTS

The characteristics of the study sample are summarized in Table 1. At follow-up, 33% of the younger cohort and 36% of the older cohort were overweight or obese. On average, the younger cohort slept approximately 10 hours per night in 1997 and 2002 and napped for about an hour each day at baseline. Sleep duration in the older cohort averaged 9.7 hours per night at baseline, 9.2 hours per night at follow-up, and only 12 minutes of daytime sleep at baseline.

The sleep variables were not highly correlated (results not shown), with respective correlation coefficients in the younger and older cohorts of \( r = -0.23 \) and \(-0.14\) for baseline napping and baseline nighttime sleep, \(0.08\) and \(0.02\) for baseline napping and nighttime sleep at follow-up, and \(0.08\) and \(0.21\) for nighttime sleep at baseline and follow-up.

Figure 1 and Figure 2 describe the cutoff points used to identify low sleep (defined as <25th percentile) in the younger and older cohorts, respectively. A \(\chi^2\) test statistic failed to reject the null hypothesis of parallel odds, with \(P\) values of .45 for the younger cohort and .70 for the older cohort. Further investigation confirmed that the sleep variables did not individually violate the assumption of parallel odds. Therefore, results are reported for the ordered logit models in Table 2. Because the parallel odds assumption is met, the ordered logit results can be interpreted as the odds of a shift from normal weight to overweight or as the odds of a shift from overweight to obesity.

### YOUNGER COHORT

For the younger children (Table 2, model 1), low nighttime sleep at baseline was significantly associated with increased odds of overweight (vs normal weight) and increased odds of obesity (vs overweight) at follow-up (odds ratio = 1.80; 95% confidence interval, 1.16-2.81). The association of nighttime sleep at follow-up and obesity was attenuated with the inclusion of baseline BMI, while the estimate for nighttime sleep at baseline remained unchanged (Table 2, models 2 and 3).

### OLDER COHORT

In the older cohort (Table 2, model 2), low nighttime sleep at baseline was not associated with any shift in weight status at follow-up. Contemporaneous sleep, however, was associated with increased odds of a shift from normal weight to overweight or from overweight to obesity at follow-up (odds ratio = 1.80; 95% confidence interval, 1.16-2.81). The association of nighttime sleep at follow-up and obesity was attenuated with the inclusion of baseline BMI, while the estimate for nighttime sleep at baseline remained unchanged (Table 2, models 2 and 3).

### COMMENT

In a national sample, we found a robust longitudinal association between duration of nighttime sleep in early life and subsequent obesity measured at ages 5 to 9 years. These findings persisted when controlling for contemporaneous sleep and other important confounding variables including parents’ BMI, family socioeconomic status (parental education, income, single parent), and hours of television viewed. For the younger cohort, duration of sleep at baseline was more closely associated with obesity at follow-up than was duration of nighttime sleep at follow-up. In the older cohort, nighttime sleep at follow-up was associated with marginally increased odds of obesity at follow-up, while sleep duration 5 years prior had no significant effect. These findings suggest that there is a critical window prior to age 5 years when nighttime sleep may be important for subsequent obesity status.

Our findings also indicate that daytime sleep had little effect on subsequent obesity at any age. This result sug-
suggests that napping is not a substitute for nighttime sleep. There is some evidence that nighttime sleep and naps serve different physiological functions. Naps may reduce daytime psychosocial stress, increase attention span, and increase alertness for learning, while nighttime sleep involves complex biological, psychosocial, and restorative functions.²⁴ Problem napping and disruptive behaviors are associated with higher cortisol levels and shorter nap duration.²⁴

Interestingly, some variables associated with obesity in other work,² notably race/ethnicity, were not significant in our models. Significant racial/ethnic disparities have been reported in nighttime sleep duration.³⁵ Consistent with our results, sleep duration may be a plausible contributor to well-documented racial/ethnic disparities in obesity.

Study strengths include a large, nationally representative sample and data collected at 2 points 5 years apart, which permit assessment of causal ordering in the relationship between sleep and obesity. Interpretation of the study findings is, however, subject to limitations. The PSID CDS collects sleep data for only 2 days in a year—a random weekday and a random weekend day. With only 2 measurements, the reported sleep patterns may not reflect the children’s usual sleep patterns throughout the year. The resulting measurement error may introduce a conservative bias into the analyses. Second, BMI at baseline would be unlikely to alter the main study findings. Third, physical activity and diet are potential confounders of the associations of sleep with obesity. Few variables are available in the PSID to measure diet, and those available were of insufficient quality to be included in the regression models. Our results were robust to the inclusion of the number of hours per day the child engaged in physical activity. Furthermore, inclusion of parents’ BMI in the models serves as a proxy for the diet and physical activity patterns in the household. Fourth, weight for children at baseline was obtained by parent report, which could lead to misclassification, especially for the older children in the cohort for whom parents may minimize problems with overweight.³⁵,³⁶ Accordingly, the associations we report may be conservative.

Sleep duration is a modifiable risk factor with potentially important implications for obesity prevention and treatment. Insufficient nighttime sleep among infants and preschool-aged children appears to be a lasting risk factor for subsequent obesity, while contemporaneous sleep appears to be important to weight status in adolescents. Napping had no effects on the development of obesity.²⁴

Table 2. Ordered Logistic Regression Models of Weight Classification at Follow-up

<table>
<thead>
<tr>
<th>Variable</th>
<th>Model 1, Younger Cohort (n=822)</th>
<th>Model 2, Older Cohort, Baseline BMI Not Included (n=1108)</th>
<th>Model 3, Older Cohort, Baseline BMI Included (n=983)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sleep duration, h/d</td>
<td>Low nighttime sleep at baseline</td>
<td>1.80 (1.16-2.80)²⁶</td>
<td>1.21 (0.85-1.73)</td>
</tr>
<tr>
<td></td>
<td>Low daytime sleep at baseline</td>
<td>1.24 (0.75-2.04)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Low nighttime sleep at follow-up</td>
<td>1.36 (0.83-2.23)</td>
<td>1.80 (1.16-2.81)¹²</td>
</tr>
<tr>
<td>Child BMI z score at baseline</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Race/ethnicity</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>White, non-Hispanic</td>
<td>0.70 (0.38-1.31)</td>
<td>1.08 (0.67-1.74)</td>
<td>0.84 (0.59-1.43)</td>
</tr>
<tr>
<td>Black, non-Hispanic</td>
<td>1.60 (0.71-3.60)</td>
<td>1.18 (0.55-2.51)</td>
<td>0.72 (0.33-1.59)</td>
</tr>
<tr>
<td>Hispanic</td>
<td>1.05 (0.80-1.38)</td>
<td>1.50 (1.10-2.05)</td>
<td>1.54 (1.14-2.07)</td>
</tr>
<tr>
<td>Family income, log, $</td>
<td>0.83 (0.39-1.77)</td>
<td>0.35 (0.14-0.86)</td>
<td>0.21 (0.09-0.50)</td>
</tr>
<tr>
<td>Maternal education, log, y</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Parents’ BMI in 1999</td>
<td>Mother</td>
<td>1.04 (0.99-1.10)</td>
<td>1.07 (1.03-1.11)¹²</td>
</tr>
<tr>
<td></td>
<td>Father</td>
<td>1.06 (1.02-1.11)²⁶</td>
<td>1.08 (1.04-1.12)</td>
</tr>
<tr>
<td>Physical activity, h/d</td>
<td>At baseline</td>
<td>0.99 (0.83-1.19)</td>
<td>1.01 (0.84-1.22)</td>
</tr>
<tr>
<td></td>
<td>At follow-up</td>
<td>0.78 (0.64-0.95)</td>
<td>0.78 (0.64-0.95)</td>
</tr>
<tr>
<td>Pseudo R²</td>
<td></td>
<td>0.08</td>
<td>0.10</td>
</tr>
</tbody>
</table>

Abbreviations: BMI, body mass index (calculated as weight in kilograms divided by height in meters squared); CI, confidence interval; OR, odds ratio.

The younger cohort includes those aged 0 to 59 months at baseline; the older cohort includes those aged 60 to 154 months at baseline. Model results are interpreted as the odds of shifting from normal weight to overweight or from overweight to obesity. All models also controlled for age, sex, birth weight, father present, hours per day of television viewing, birth order, and urban residence.

Low sleep is defined as less than the 25th percentile of age-specific norms.

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The Archives of Facial Plastic Surgery will be publishing a theme issue on pediatric facial plastic and reconstructive surgery in May/June 2011. Manuscripts received by mid-September 2010 will have the best chance for consideration. Please visit the manuscript submission and review Web site at http://manuscripts.archfacial.com/cgi-bin/main.plex.
could occur for multiple reasons other than there being no depression (eg, the topic did not come up during the physician visit). Finally, the deprivation index was tied to the community in which families resided and may not accurately reflect the individual household.

Major strengths of this work are the knowledge generated on children’s exposure to depression in their parents. These findings reflect depression’s qualities of being recurrent as well as the commonality of a spouse/partner also being depressed. The study stands in sharp contrast to many studies of children of depressed parents, which relied on a single measure of depression, capturing depression symptom levels only at that time. The work also responds to the call for a more developmentally sensitive approach to describing children’s exposure to parents’ depression by detailing children’s first exposures. The authors also appreciated that adult depression’s onset may precede the birth of the child. Given concerns about prenatal depression, an important next step in this line of research will be to capture children’s true first exposure (during fetal development) rather than merging all depression prior to the birth of the child.

Of most importance in considering this work is the promise it holds for future findings in that the authors indicate that data will be available for children aged up to 12 years. For the article in this month’s Archives, only 5% of the sample had data to 12 years of age. Ultimately, the authors will be able to fulfill their promise of presenting “the first ever study assessing the incidence of depression in both parents recorded in general practice across the course of their offspring’s childhood.”

Given the preliminary nature of the data, the numbers may dramatically underrepresent children’s potential exposures and overly represent early (postpartum) exposures given that most of the sample had not yet had the opportunity to provide data for the child’s first 12 years of life. For example, the finding that 77% of mothers had only 1 episode of depression in children’s first 12 years of life must be considered in light of 83% of mothers having had data only through children’s first year of life. Thus, readers might greatly anticipate the findings from the completed study once the youngest children in the cohort attain 12 years of age. Also important will be to pursue the potential influence on the data of a noted cohort effect (the incidence was found to increase over the years of study). The authors are likely correct in suggesting that the change reflects an increasing trend toward prescribing antidepressants. Along with incidence data, the interesting findings from the multivariate analyses require the same caution in interpreting the findings beyond the first-year data.

In all, by innovatively linking data on depression in adults with data on children, Davé et al took important steps toward addressing the need for knowledge on the rates of depression in adults who are parents of young children and on factors that increase or decrease those rates. Readers will obtain valuable knowledge from these early findings and look forward to the findings from the completed study. Even these early findings have important implications for policy and practice. In particular, the findings underscore the need to determine the parenthood status of adults being screened or treated for depression and reducing obstacles toward coordinated care that takes a developmentally sensitive approach to assessing and addressing the needs of the children.

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