DOI: 10.5665/SLEEP.1238

# Longitudinal Association between Short Sleep, Body Weight, and Emotional and Learning Problems in Hispanic and Caucasian Children

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**Study Objective:** To determine the impact of lower amounts of childhood sleep assessed by polysomnogram on development of obesity, being anxious or depressed, or having learning problems 5 years later.

**Design:** Prospective cohort.

**Participants:** Subjects were 304 community participants from the Tucson Children's Assessment of Sleep Apnea study, aged 6-12 years old at baseline.

**Measurements and Results:** Children were classified according to baseline sleep as those who slept ≥ 9 h/night, those who slept > 7.5 to < 9 h/night, and those who slept ≤ 7.5 h/night. Odds of overweight/obese (≥ 85$^{th}$ BMI percentile), obese (≥ 95$^{th}$ BMI percentile), anxious or depressed, and learning problems at follow-up were assessed according to baseline sleep categories. Children who slept ≤ 7.5 h/night had higher odds of being obese (OR = 3.3, P < 0.05) at follow-up than children who slept ≥ 9 h/night. Borderline significance for overweight/obese (OR = 2.2, P < 0.1), anxious or depressed (OR = 3.3, P < 0.1), and having learning problems (OR = 11.1, P < 0.1) were seen for children who slept ≤ 7.5 h/night as compared to those who slept ≥ 9 h/night. A mean increase in BMI of 1.7 kg/m$^2$ (P = 0.01) over the 5 years of follow-up was seen for children who slept ≤ 7.5 h/night compared to those who slept ≥ 9 h/night. These relationships did not differ between Hispanic and Caucasian children.

**Conclusions:** Children with reduced amounts of sleep (≤ 7.5 h/night) had an increased risk for higher body weight in early adolescence. Similarly, children who slept ≤ 7.5 h/night had higher risk of being anxious or depressed or having learning problems in early adolescence.

**Keywords:** Sleep time, obesity, childhood, body mass index

**Citation:** Silva GE; Goodwin JL; Parthasarathy S; Sherrill DL; Vana KD; Drescher AA; Quan SF. Longitudinal association between short sleep, body weight, and emotional and learning problems in Hispanic and Caucasian children. *SLEEP* 2011;34(9):1197-1205.

## INTRODUCTION

The prevalence of being overweight and obese among adolescents and young adults has increased radically during the last 30 years.[1] Among children and adolescents 2-19 years, 31.7% are at or above the 85$^{th}$ percentile of BMI for age.[2] Along with the increasing obesity epidemic, the number of hours that children and adolescents spend sleeping at night has been shown to be decreasing.[3] Moreover, recent epidemiological studies have pointed to a strong relationship between short sleep duration and higher body mass index in children.[4-8] Most of these studies have utilized subjective measures of sleep to determine sleep time. For example, in a cohort of 915 infants and toddlers, parents were asked to rate their children's sleep; those who slept < 12 h/day were 2.04 times more likely to become overweight by age 3 than children who slept longer.[9] Reported high levels of television viewing elevated the risk; children who slept less and who viewed ≥ 2 h of television per day had a 17% higher chance of becoming overweight compared with those who viewed television fewer hours. However, few of these studies have assessed sleep time using objective measures,[10] and fewer have evaluated the association longitudinally from childhood to adolescence.[11,12]

Obesity in childhood and adolescence is of special concern, because obesity itself is a risk factor for cardiovascular disease, hypertension, insulin resistance, and dyslipidemia. Attempted interventions to improve adolescent diet and physical activity patterns have had limited success.[13] Although sleep alone may not be the sole source driving weight gain, most studies' interventions have not addressed the amount of time spent sleeping as a possible contributing factor.

In addition to these potential physiological consequences of short sleep in adolescence, reduction in sleep duration may contribute to increased risks of cognitive and/or behavioral problems. The rise in adolescents' use of electronic media may contribute to a delayed sleep time and subsequent shorter sleep times.[14] Chronic sleep deprivation is prevalent in 21% to 75% of the adolescent population,[15] resulting in adolescents and young adults who are often excessively sleepy and at high risks for cognitive impairment, fatigue, behavioral or emotional problems, injuries, and motor vehicle accidents.[16,17]

The purpose of this study was to determine the impact of lower amounts of childhood sleep on the subsequent development of obesity independent of sleep disordered breathing (SDB) in a prospective cohort of children and young adolescents, and to determine if differences in this association exist between Caucasian and Hispanic children. The longitudinal ef-

A commentary on this article appears in this issue on page 1153.

**Submitted for publication September, 2010**
**Submitted in final revised form May, 2011**
**Accepted for publication June, 2011**

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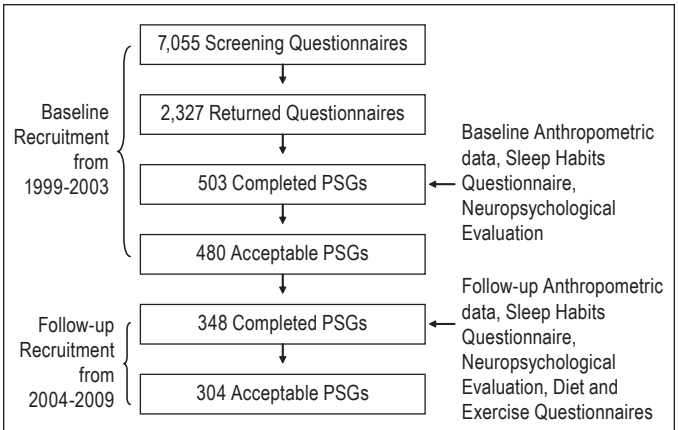


**Figure 1**—Flow diagram of sample selection.

fects of reduced sleep duration on anxious or depressed symptoms, learning problems, and excessive daytime sleepiness (EDS) in adolescents were also assessed.

## METHODS

The Tucson Children's Assessment of Sleep Apnea Study (TuCASA) is a prospective cohort study designed to investigate the natural history of SDB and its impact on school and neurocognitive performances. Hispanic and Caucasian children ages 6 to 12 years were recruited to undergo unattended home polysomnograms (PSGs) and perform neurocognitive assessments. Subjects were recruited through the Tucson Unified School District (TUSD), a very large district with a substantial elementary school population. Parents were asked to complete short screening questionnaires and to provide their contact information if they were willing to allow study personnel to contact them to determine if their child was eligible for the study. A total of 7,055 screening questionnaires were sent home with the children. Of these, 2,327 (33%) were returned. Recruitment information was supplied on 52% of the returned questionnaires from which we selected children, based on pre-established inclusion and exclusion criteria, to undergo PSG. Children were included if they were 6-12 years of age and were Caucasian or Hispanic. Excluded were children whose parents reported them to have a history of tonsillectomy, attention deficit hyperactivity disorder, mental retardation, or other mental or physiological conditions that would affect neurocognitive or sleep testing. An unattended home PSG was scheduled as soon as possible after recruitment. From 1999-2003, a total of 503 children completed home PSGs (Baseline); 480 were satisfactory. Approximately 5 years later (mean 4.7 years), 348 children agreed to participate in the second phase of the study, and 304 children (10 to 18 years old) had home visits with completion of acceptable in-home PSGs (Follow-up) from 2004 to 2009. We were unable to contact the rest of the participants, and they were lost to-follow-up. The socioeconomic status of the families of Hispanic participants was lower and their places of residence were less stable. This resulted in a greater lost-to-follow-up rate among Hispanics. On both occasions, all families completed sleep screening, sleep habits, and morning sleep questionnaires (see Figure 1). Comparisons were made between the 304 children who had baseline and follow-up PSGs to the 176 children with baseline but no follow-up PSGs. A lower proportion of children were Hispanic in the group with follow-up PSGs (36.8%) than in the group with no follow-up PSG (51.7%, $P = 0.001$). Prevalence of anxiety or depression was lower in the PSG follow-up group (15.3%) than in the group with no follow-up PSG (27.9%, $P = 0.003$). No other significant differences were seen between the groups for age, gender, BMI, or being overweight or obese.

The TuCASA study was approved by the University of Arizona Institutional Review Board (IRB), as well as the Tucson Unified School District (TUSD) Research Committee. Complete details of the TuCASA study design have been published previously.[18,19]

The data were collected using a 2-person, mixed-gender team who arrived at the home approximately 1 h prior to the child's normal bedtime. There was emphasis on making the night of the polysomnographic assessment as representative as possible of a usual night of sleep. Children's weekday or weekend bedtime routines were encouraged to be consistent with the day of the week and the calendar month the visits were made. PSG total sleep time was used in this analysis without discerning the weekday or month the study was performed. However, 83% of PSGs were done during a weekday, and studies were performed equally throughout the year.

Prior to performing any study procedures, parents gave informed consent, and the child gave assent to the study using language appropriate IRB-approved forms. Each child's height, weight, and blood pressure were measured. Body mass index (BMI, $kg/m^2$) was calculated, and the standardized z-score for BMI computed using the 2000 Centers for Disease and Control and Prevention (CDC) growth charts; CDC Statistical Analysis System (SAS) program files were used to adjust for age, gender, and height- and weight-for age.[20-22] Overweight/obese was defined as $\geq 85^{th}$ BMI percentile and obesity as $\geq 95^{th}$ BMI percentile.[2]

Parents were asked to complete comprehensive Sleep Habits Questionnaires (SHQs) that inquired about their children's sleep history and sleep characteristics. Single, unattended overnight PSGs were obtained using the Compumedics PS-2 system (Abbotsford, Victoria, Australia). The following signals were acquired as part of the TuCASA montage: C3/A2 and C4/A1 electroencephalogram, right and left electrooculogram (EEG), a bipolar submental electromyogram, thoracic and abdominal displacement (inductive plethysmography band), airflow (nasal/oral thermistor), nasal pressure cannula, finger pulse oximetry, ECG (single bipolar lead), snoring microphone, body position, and ambient light (sensor attached to the vest to record on/off).

Scoring of sleep was performed by a single registered polysomnographic technologist using Rechtschaffen and Kales criteria.[23] Arousals were identified using criteria published by the American Academy of Sleep Medicine.[24] Apneas were scored if the amplitude (peak to trough) of the airflow signal using the thermistor decreased below $\geq 25\%$ of the amplitude of baseline breathing (identified during a period of regular breathing with stable oxygen levels) and if this change lasted > 6 sec or 2 breath cycles. Hypopneas were designated if the amplitude of any respiratory signal decreased below (approximately) $\geq 70\%$ of the amplitude of baseline and if the thermistor signal did not meet the criterion for apnea. The respiratory disturbance index (RDI) was defined as the number of respiratory events (apneas

and hypopneas) per hour of the total sleep time. For this analysis, a 3% oxygen desaturation was required for an event to be counted in the total RDI (RDI3%). We considered a child to have SDB if their RDI3% was ≥ 1 event/h of total sleep time. Previous evidence supports that a RDI of 1, based on events with a 3% oxygen desaturation, is clinically significant.[19,25,26]

Neuropsychological evaluations were conducted approximately one month after the baseline and follow-up PSGs, and evaluators were blinded to both PSG findings. During the baseline and follow-up visits, parents completed a behavioral assessment including the Child Behavior Checklist (CBCL).[27] The CBCL consists of 118 items on a 3-point scale ranging from *not true*, *somewhat true*, to *often true*, which include social, attention, thought problems, anxiety, depression, and withdrawal scales. Raw scores were converted to age-standardized *T*-scores (Mean = 50 and SD = 10). *T*-scores ≥ 60 were considered within the borderline of clinical referral range, and thus, scores were dichotomized at this value. Dichotomized values of anxious or depressed were used to indicate subjects in the borderline clinical range. Syndrome scales for anxious or depressed were compared at baseline and follow-up in this study.

Baseline PSG total sleep hours were categorized into groups at the 25$^{th}$ and 75$^{th}$ percentile of distribution with cut points at 7.5 and 9 hours. Children were classified at baseline as those who slept ≥ 9 h/night, slept > 7.5 to < 9 h/night, and slept ≤ 7.5 h/night. This categorical variable allowed comparison between subjects who demonstrated high, medium, and low amounts of sleep time. Excessive daytime sleepiness (EDS) was defined if affirmative answers were given to any of the following questions: *is your child sleepy during the daytime, falls asleep at school, or falls asleep while watching television?* Children were classified as having learning problems if the parent answered *frequently* or *almost always* to the question, *does your child have learning problems?* Reports of *never*, *rarely*, or *occasionally* were considered negative.

During the follow-up survey of the TuCASA study, 2 questionnaires were added to evaluate physical activity and dietary intake. The *Block Kids Physical Activity Screener* was used and asks about physical activities—both frequency and duration—in the past 7 days. The tool includes 9 items querying leisure and school activities, chores, and part-time jobs, as well as time spent watching television, playing video games, and using the Internet. The main output variables estimate the total calories "expended" and minutes per day spent at moderate and vigorous activity levels. We included in this analysis the total number of calories expended. Food frequency was evaluated using the *Harvard Medical School K-95-1 Youth/Adolescent* (YAQ) Questionnaire. Assessments in this questionnaire include unit amounts for individual nutrients, as well as total calories, protein, various types of fat, carbohydrates, fiber, sucrose, vitamins, and minerals. This instrument has been validated qualitatively and quantitatively.[28-30] Since a previous report from this study showed association between caffeine consumption and sleep time,[31] the total number of calories as well as total amounts of caffeine consumed were analyzed in this study.

### Analyses

The $\chi^2$ test was used to compare differences in proportions between each of the categorical variables from baseline to follow-up and by ethnicity. Student's *t*-test was used to compare differences in mean values for continuous variables. Differences in proportions for categorical variables between baseline and follow-up were assessed using the *Z*-test for equality of proportions. Separate multivariate logistic regressions were fitted to evaluate odds of overweight/obese, obese, anxious or depressed, learning problems, and EDS at follow-up by baseline sleep categories. Models were adjusted for potential confounders such as BMI (kg/m$^2$), ethnicity, SDB, age, and caffeine use, as well as baseline values where appropriate. Models also were adjusted for caloric expenditure, caloric intake, parental education, sex, and hours of television or video use. However, these additional variables were not significant in any of the models and were excluded from the final logistic models. Variables included in the models were selected in accordance with possible biologic associations, significance, or in accordance with previously published studies. Separate logistic regression models were also fitted including minutes and percent time spent in REM stage, stage 3/4, and sleep efficiency at baseline. None of these predictive variables, however, yielded significant associations with being obese, overweight, having learning problems, or being anxious or depressed at follow-up and thus are not discussed further. In addition, we ran our logistic regression models excluding subjects with the highest RDI, those with RDI ≥ 5 (n = 30) and those with RDI 3% ≥ 5 (n = 3), again with no appreciable differences in results, and thus, we retained all the subjects in the analyses and adjusted for SDB in the models. In order to evaluate a possible interaction effect between baseline sleep and ethnicity, we created an indicator variable containing baseline sleep categories by ethnicity groups. We then included this variable in each of the logistic regression models. Linear contrasts were performed between the regression coefficients for Caucasians and Hispanics at each category of sleep time. No significant differences were found.

Multivariate mixed-effects linear regression models were fitted to evaluate mean differences in longitudinal increase in BMI associated with the baseline sleep categories using ≥ 9 h sleep/night as the reference category. Subjects were fitted as random effects to account for intra-subject serial correlation, which results from longitudinal BMI assessments. Ethnicity and SDB were included as fixed effects; age and total hours of sleep were included as time-dependent variables.

## RESULTS

### Difference by Ethnicity at Baseline and Follow-Up

The mean age at baseline was 8.9 years (range 6-12), and 13.7 years (range 10-18) at follow-up. Approximately 51% were boys and 49% were girls; 63.2% were Caucasian and 36.8% were Hispanic (Table 1). The mean values for BMI, BMI z-score, and BMI percentile increased significantly from baseline to follow-up, with Hispanic children having higher mean scores than Caucasian children at both surveys. A higher percent of Hispanic children than Caucasian children were overweight/obese at baseline and follow-up. The proportion of children classified as obese increased significantly from baseline to follow-up (14.1% vs 19.1%, P < 0.0001), with significant ethnic differences. A higher percent of Hispanic children than Caucasian children were obese at baseline (20.5% vs 10.4%,

**Table 1**—Baseline and follow-up characteristics by ethnicity (n = 304)*

| | Baseline | | | Follow-up | | |
|---|---|---|---|---|---|---|
| | Caucasian (N = 192) | Hispanic (N = 112) | All (N = 304) | Caucasian (N = 192) | Hispanic (N = 112) | All (N = 304) |
| Age, mean years (range) | 8.9 (6-12) | 9.1 (6-12) | 8.9 (6-12) | 13.5 (10-18) | 13.9 (10-18)[∞] | 13.7 (10-18)[†] |
| % Gender | | | | | | |
| Male | 48.5 | 54.5 | 50.7 | | | |
| Female | 51.5 | 45.5 | 49.3 | | | |
| % Ethnicity | 63.2 | 36.8 | | | | |
| % Parental education | | | | | | |
| 1-8 years | 0 | 7.4 | 2.5 | | | |
| 9-12 years | 20.2 | 44.2 | 28.4 | | | |
| > 13 years | 79.8 | 48.4[γ] | 69.1 | | | |
| BMI, kg/m$^2$ | 17.7 ± 4.6 | 19.1 ± 4.1[§] | 18.2 ± 4.5 | 20.6 ± 4.6 | 23.5 ± 5.6[£] | 21.6 ± 5.1[†] |
| BMI z-score | 0.15 ± 1.3 | 0.74 ± 1.1[£] | 0.36 ± 1.3 | 0.25 ± 1.2 | 0.87 ± 1.0[£] | 0.48 ± 1.2[‡] |
| BMI percentile | 54.7 ± 31.7 | 69.5 ± 28.1[£] | 60.2 ± 31.2 | 57.5 ± 31.7 | 72.9 ± 26.9[£] | 63.2 ± 30.9[‡] |
| % Overweight/obese | 26.0 | 42.9[α] | 32.2 | 30.2 | 45.5[α] | 35.8 |
| % Obese | 10.4 | 20.5[α] | 14.1 | 12.5 | 30.4[γ] | 19.1[β] |
| Total sleep, hours | 8.1 ± 1.5 | 7.9 ± 1.5 | 8.05 ± 1.5 | 7.9 ± 1.1 | 7.8 ± 1.0 | 7.85 ± 1.05[‡] |
| % Sleep category | | | | | | |
| ≥ 9 h/night | 28.1 | 20.5 | 25.3 | 11.9 | 8.0 | 10.5[θ] |
| > 7.5 to < 9 h/night | 47.9 | 54.5 | 50.3 | 54.2 | 54.5 | 54.3 |
| < 7.5 h/night | 24.0 | 25.0 | 24.4 | 33.9 | 37.5 | 35.2[θ] |
| Non-REM sleep | | | | | | |
| Stage 1 % sleep | 4.5 ± 3.3 | 4.4 ± 3.1 | 4.6 ± 3.2 | 3.8 ± 2.3 | 4.1 ± 2.3 | 3.9 ± 2.3[‡] |
| Stage 2 % sleep | 52.1 ± 5.7 | 53.2 ± 5.9 | 52.5 ± 5.8 | 54.6 ± 6.6 | 57.0 ± 6.9[§] | 55.5 ± 6.8[†] |
| Stage 3/4 % sleep | 22.0 ± 5.9 | 21.2 ± 6.2 | 22.1 ± 6.9 | 19.5 ± 6.6 | 16.6 ± 6.7[§] | 18.4 ± 6.8[£] |
| Stage 1 min | 22.7 ± 17.2 | 21.1 ± 13.8 | 22.1 ± 16.0 | 17.7 ± 9.8 | 18.8 ± 10.0 | 18.1 ± 9.9[†] |
| Stage 2 min | 257.1 ± 56.1 | 253.3 ± 60.4 | 255.7 ± 57.6 | 257.9 ± 46.7 | 267 ± 42.7[∞] | 261.5 ± 45.4 |
| Stage 3/4 min | 106.2 ± 23.4 | 99.6 ± 22.3[∞] | 103.8 ± 23.2 | 91.2 ± 29.9 | 78.4 ± 32.1[§] | 86.5 ± 31.4[†] |
| REM % | 21.3 ± 4.6 | 20.6 ± 5.2 | 21.0 ± 4.9 | 22.4 ± 4.7 | 23.0 ± 4.6 | 22.6 ± 4.7[†] |
| REM minutes | 107.2 ± 33.2 | 101.0 ± 35.1 | 104.9 ± 33.9 | 107 ± 31.0 | 108.9 ± 28.7 | 107.7 ± 30.2 |
| Sleep efficiency % | 90.2 ± 5.4 | 90.2 ± 4.9 | 90.2 ± 89.6 | 87.6 ± 6.8 | 86.3 ± 7.8 | 87.1 ± 7.2[†] |
| RDI 3% | 1.1 ± 2.7 | 0.9 ± 1.4 | 1.04 ± 2.3 | 0.47 ± 0.7 | 0.51 ± 1.0 | 0.5 ± 0.8[†] |
| % SDB (RDI 3% ≥ 1) | 27.1 | 27.7 | 27.3 | 14.6 | 16.1 | 15.1[Ω] |
| % EDS | 8.3 | 11.6 | 9.4 | 32.8 | 29.5 | 31.6[θ] |
| % Anxious/depressed | 15.0 | 15.9 | 15.3 | 13.9 | 12.1 | 13.4 |
| % Learning problems | 4.7 | 3.6 | 4.3 | 4.3 | 5.7 | 4.8 |
| Kcalories (expended) | | | | 531 ± 600 | 752 ± 632[§] | 613 ± 620 |
| Kcalories intake | | | | 1627 ± 642 | 1770 ± 685 | 1678 ± 660 |
| Hours of TV/video | | | | 4.0 ± 1.3 | 4.0 ± 1.3 | 4.0 ± 1.3 |
| Caffeine use, mg | | | | 26.5 ± 29.3 | 34.5 ± 3.3[∞] | 29.4 ± 31.2 |

*Mean ± SD unless otherwise noted. [‡]P < 0.05, [†]P < 0.0001 for *t*-test between baseline and follow-up. [∞]P < 0.05, [§]P < 0.001, [£]P < 0.0001 for *t*-test by ethnicity. [β]P < 0.05, [Ω]P < 0.001, [θ]P < 0.0001 for Z test for difference in proportions between baseline and follow-up. [α]P < 0.01, [γ]P < 0.0001 for $\chi^2$ test by ethnicity.

P < 0.01). The percent of obese children increased to 30.4% for Hispanics and 12.5% for Caucasians at follow-up (P < 0.0001).

The total number of sleep hours, however, decreased significantly from baseline (8.05 h) to follow-up (7.85 h, P < 0.05). The percent of children classified as sleeping ≥ 9 h/night decreased from baseline (25.3%) to follow-up (10.5%, P < 0.0001), while the percent of children who slept < 7.5 h/night increased from baseline (24.4%) to follow-up (35.2%, P < 0.0001). Percent in stage 1 sleep decreased from baseline (4.6%) to follow-up (3.9%, P < 0.05), while percent in stage 2 sleep increased from baseline (52.5%) to follow-up (55.5%, P < 0.0001). Similarly, percent in REM sleep increased (21.0% to 22.6%, P < 0.0001), while sleep efficiency decreased (90.2% to 87.1%, P < 0.0001).

The overall mean for RDI 3% decreased from baseline (1.04) to follow-up (0.5, P < 0.0001). The prevalence of SDB (RDI 3% ≥ 1) decreased from baseline to follow-up (27.3% vs 15.1%, P < 0.001), while the percent of children reporting EDS increased from 9.4% to 31.6% (P < 0.0001). In addition,

**Table 2**—Baseline and follow-up characteristics by baseline sleep categories (n = 304)*

| | Baseline | | | Follow-up | | |
|---|---|---|---|---|---|---|
| Baseline sleep category, h | ≥ 9 (N = 77) | > 7.5 to < 9 (N = 153) | < 7.5 (N = 74) | ≥ 9 (N = 77) | > 7.5 to < 9 (N = 153) | < 7.5 (N = 74) |
| Age, years | 8.3 ± 2 | 9.3 ± 2 | 9.0 ± 2† | 13.1 ± 2 | 13.9 ± 2 | 13.7 ± 2§ |
| Gender | | | | | | |
| % Male | 23.4 | 51.9 | 24.7 | | | |
| % Female | 27.3 | 48.7 | 24.0 | | | |
| Ethnicity | | | | | | |
| % Caucasian | 28.1 | 47.9 | 24.0 | | | |
| % Hispanic | 20.5 | 54.5 | 25.0 | | | |
| BMI, kg/m$^2$ | 17.4 ± 5 | 18.4 ± 4 | 18.7 ± 5 | 20.2 ± 4 | 21.9 ± 5 | 22.5 ± 6€ |
| BMI z-score | 0.15 ± 1 | 0.43 ± 1 | 0.45 ± 2 | 0.31 ± 1 | 0.52 ± 1 | 0.56 ± 1 |
| BMI percentile | 54.5 ± 30 | 61.3 ± 32 | 63.8 ± 30 | 59.8 ± 30 | 63.6 ± 30 | 65.8 ± 33 |
| % Overweight/obese | 23.4 | 35.3 | 35.2 | 27.3 | 34.0 | 44.6 |
| % Obese | 7.8 | 15.7 | 17.6 | 11.7 | 18.9 | 27.0‡ |
| Total sleep (h) | 9.5 ± 0.5 | 8.3 ± 0.5 | 5.9 ± 1† | 8.1 ± 1.0 | 7.8 ± 0.92 | 7.7 ± 1.3€ |
| NREM sleep | | | | | | |
| Stage 1 % sleep | 4.5 ± 2.9 | 4.6 ± 2.8 | 4.1 ± 4 | 3.9 ± 2.8 | 3.9 ± 2.1 | 3.9 ± 2.1 |
| Stage 2 % sleep | 52.6 ± 5.1 | 53.1 ± 5.8 | 51.2 ± 6.5 | 55.7 ± 6.9 | 55.7 ± 6.9 | 55.0 ± 6.4 |
| Stage 3/4 % sleep | 19.7 ± 4.5 | 20.5 ± 4.8 | 26.5 ± 7.2† | 18.6 ± 6.4 | 18.0 ± 6.9 | 19.1 ± 7.1 |
| Stage 1 min | 25.7 ± 17.2 | 23.5 ± 14.5 | 15.5 ± 16.2§ | 18.8 ± 11.9 | 18.2 ± 9.5 | 17.3 ± 8.2 |
| Stage 2 min | 300 ± 32.9 | 256.8 ± 32.5 | 188.7 ± 60.2§ | 271.2 ± 42.0 | 259.5 ± 41.6 | 255.2 ± 54.5π |
| Stage 3/4 min | 112.1 ± 24.9 | 102.3 ± 23.1 | 98.3 ± 19.1§ | 90.8 ± 34.2 | 83.7 ± 30.6 | 87.6 ± 29.5 |
| REM % | 23.3 ± 3.2 | 21.7 ± 3.7 | 17.2 ± 6.2† | 22.3 ± 4.2 | 22.9 ± 4.6 | 22.2 ± 5.2 |
| REM minutes | 133 ± 20.9 | 109 ± 20.4 | 66.8 ± 33.3† | 110 ± 29.5 | 108 ± 27.8 | 104.9 ± 35 |
| Sleep efficiency % | 92.2 ± 3.5 | 89.8 ± 5.2 | 88.9 ± 6.2† | 88.7 ± 6.1 | 87.2 ± 7.1 | 85.3 ± 8.0π |
| RDI 3% | 0.73 ± 0.6 | 1.1 ± 3 | 1.1 ± 1.8 | 0.6 ± 0.9 | 0.41 ± 0.7 | 0.59 ± 1 |
| % SDB (RDI 3% ≥ 1) | 28.6 | 25.5 | 29.7 | 22.1 | 10.5 | 17.6‡ |
| KCalories (expend) | | | | 480 ± 475 | 672 ± 651 | 629 ± 676 |
| KCalories intake | | | | 1715 ± 691 | 1703 ± 674 | 1588 ± 597 |
| Hours of TV/video | | | | 3.9 ± 1 | 4.0 ± 1 | 4.1 ± 1 |
| Caffeine use (mg) | | | | 27.1 ± 29 | 30.5 ± 32 | 29.5 ± 32 |
| % EDS | 6.5 | 9.8 | 12.2 | 29.9 | 32.7 | 31.1 |
| % Anxious/depressed | 13.4 | 14.0 | 19.7 | 8.5 | 11.6 | 20.7 |
| % Learning problems | 5.2 | 3.9 | 4.0 | 2.6 | 5.2 | 6.8 |

*Mean ± SD unless otherwise noted. BMI, body mass index; SDB, sleep disordered breathing. πP < 0.05, €P < 0.01, §P < 0.001, †P < 0.0001 for ANOVA test by sleep category. ‡P < 0.05 for $\chi^2$ test by sleep category.

Hispanic children expended more calories (752 Kcal) and consumed more caffeine (34.5 mg) than Caucasian children (531 Kcal and 26.5 mg) (P < 0.05 and P < 0.001, respectively).

### Differences by Baseline Sleep Categories

Descriptive characteristics by baseline sleep categories are presented in Table 2. Children who slept < 7.5 h/night at baseline had higher body weight indices at baseline and at follow-up than children who slept ≥ 9 h/night or > 7.5 to < 9 h/night. Significantly higher mean BMI values were seen at follow-up for children who slept < 7.5 h/night (22.5 kg/m$^2$) compared to those who slept ≥ 9 h/night (20.2 kg/m$^2$) and > 7.5 to < 9 h/night (21.9 kg/m$^2$, P < 0.01) at baseline. At follow-up, 27.0% of children who slept < 7.5 h/night were obese, while only 11.7% of those who slept ≥ 9 h/night and 18.9% of those who slept > 7.5 to < 9 h/night at baseline were obese (P < 0.05).

The percent of time in stage 3/4 sleep was higher for children who slept < 7.5 h/night at baseline (26.5) than those who slept ≥ 9 h/night (19.7) or those who slept > 7.5 to < 9 h/night (20.5) (P < 0.0001) at baseline. Conversely, REM %, minutes in REM, and % sleep efficiency were significantly lower for children who slept < 7.5 h/night than for those who slept > 7.5 to < 9 or ≥ 9 h/night. Lower values were seen for sleep efficiency at follow-up in children who slept < 7.5 h/night than those who slept > 7.5 to < 9 or ≥ 9 h/night at baseline. Although differences in SDB were seen at follow-up by baseline sleep categories, no particular trend was noted.

### Differences by Gender

Gender differences were noted for RDI at follow-up, with boys having higher mean values (0.59 ± 1.0 standard deviation [SD]) than girls (0.38 ± 0.06 SD, P = 0.03). Similarly, for

**Table 3**—Multiple logistic regression models predicting obesity/overweight, obesity, anxious/depressed, and having learning problems at follow-up by baseline sleep categories and other independent variables*

| Variables | Overweight/Obese | | Obese | | Anxious/Depressed | | Learning Problems | |
|---|---|---|---|---|---|---|---|---|
| | OR | 95% CI | OR | 95% CI | OR | 95% CI | OR | 95% CI |
| > 7.5 to < 9 h sleep/night | 1.5 | 0.69 – 3.15 | 2.0 | 0.73 – 5.64 | 2.4 | 0.61 – 9.44 | 11.3 | 0.91 – 141§ |
| < 7.5 h sleep/night | 2.2 | 0.95 – 5.09§ | 3.3 | 1.09 – 9.66† | 3.3 | 0.83 – 13.5§ | 11.1 | 0.86 – 140§ |
| Baseline BMI (kg/m$^2$) | 1.4 | 1.24 – 1.51∫ | 1.2 | 1.17 – 1.40∫ | 0.9 | 0.83 – 1.03 | 0.9 | 0.83 – 1.13 |
| Ethnicity (Hispanic) | 1.3 | 0.72 – 2.4 | 2.4 | 1.20 – 4.89† | 0.6 | 0.21 – 1.85 | 2.1 | 0.59 – 7.56 |
| SDB at follow-up | 4.2 | 1.83 – 9.61∞ | 2.5 | 0.99 – 6.24† | 2.7 | 0.88 – 8.34 | 4.9 | 1.18 – 20.3† |
| Age at follow-up | 0.9 | 0.76 – 1.08 | 0.8 | 0.98 – 1.04 | 0.9 | 0.64 – 1.20 | 0.7 | 0.49 – 1.0 |
| Caffeine use at follow-up | 1.0 | 0.99 – 1.01 | 1.1 | 0.99 – 1.01 | 1.0 | 0.99 – 1.01 | 1.0 | 0.98 – 1.02 |
| Baseline Anxious/Depressed | — | — | — | — | 8.6 | 3.2 – 23.1∫ | — | — |
| Baseline Learning Problems | — | — | — | — | — | — | 30.1 | 6.5 – 140∫ |

*OR, odds ratio. The reference category for sleep category is ≥ 9 h/night. Caucasian is the reference category for ethnicity. No SDB is the reference category for SDB. BMI, body mass index; SDB, sleep disordered breathing; 95%CI, 95% confidence interval. §P < 0.1, †P < 0.05, ∞P < 0.01, ∫P < 0.0001.

**Table 4**—Random effects linear regression model of BMI by baseline sleep categories and other predictive variables*

| | Coefficient | P-value | 95% Conf. Interval |
|---|---|---|---|
| > 7.5 to < 9 h sleep/night | 1.06 | 0.05 | -0.2 – 2.1 |
| < 7.5 h sleep/night | 1.7 | 0.01 | 0.4 – 3.1 |
| SDB 3% at baseline | 1.3 | 0.008 | 0.4 – 2.3 |
| SDB 3% at follow-up | 2.7 | < 0.0001 | 1.5 – 3.9 |
| Age (years) | 0.74 | < 0.0001 | 0.6 – 0.8 |
| Total sleep time (h) | 0.21 | 0.09 | -0.03 – 0.5 |
| Ethnicity (Hispanic) | 1.8 | < 0.0001 | 0.9 – 2.7 |
| Constant | 7.3 | < 0.0001 | 4.6 – 10.03 |

*≥ 9 h of sleep/night is the reference category for sleep category. Caucasian is the reference category for ethnicity. No SDB is the reference category for SDB. BMI, body mass index; SDB, sleep disordered breathing.

SDB prevalence, boys had a higher prevalence (21.4%) than girls (8.7%, P = 0.002). Boys also had higher energy expenditure (716 kcal ± 739 SD) than girls (507 kcal ± 449 SD, P = 0.003). There were no other significant gender differences (data not shown).

### Multivariate Modeling

Logistic regression models showed that after adjusting for covariates, the odds of being obese at follow-up were 3.3 times higher for children who slept < 7.5 h/night at baseline compared to those with who slept ≥ 9 h/night (P < 0.05; Table 3). Although borderline significant, children who slept < 7.5 h/night also had higher odds of being overweight/obese (OR = 2.2, P < 0.1) at follow-up, being anxious or depressed (OR = 3.3, P < 0.1), and having learning problems (OR = 11.1, P < 0.1) compared to children who slept ≥ 9 h/night at baseline. No significant associations were found with EDS and the baseline sleep categories in logistic regressions (data not shown). Baseline BMI was a significant predictor for being overweight/obese (OR = 1.4, P < 0.0001) and obese (OR = 1.2, P < 0.0001) at follow-up. SDB at follow-up also was significantly associated with being overweight/obese (OR = 4.2, P < 0.01), obese (OR = 2.5, P < 0.05), and having learning problems (OR = 4.9, P < 0.05).

Random effect models showed that after adjusting for potential confounders, children who slept < 7.5 h/night at baseline had an average increase in BMI of 1.7 kg/m$^2$ (P = 0.01) from baseline to follow-up compared to children who slept ≥ 9 h/night (see Table 4 and Figure 2). Models also showed that Hispanic children had a mean increase in BMI of 1.8 kg/m$^2$ (P = 0.0001) compared to Caucasian children, and that the average BMI increased 0.74 kg/m$^2$ (P = 0.0001) with each year of age. Having SDB at either baseline or follow-up were significant predictors of higher BMI at follow-up. Figure 3 shows median differences in follow-up BMI by baseline sleep categories for older Hispanic and Caucasian children.

## DISCUSSION

Results from this study showed that prevalence of obesity increased from childhood to young adolescence while objectively measured total sleep time decreased from baseline to follow-up. Furthermore, we demonstrated that children who slept < 7.5 hours/night at baseline had a higher average BMI and higher odds of developing obesity during young adolescence than children who slept ≥ 9 hours/night even after adjusting for SDB. Although we did not find an interaction effect between baseline sleep categories and ethnicity, results from this study also showed that the risk of obesity at follow-up was higher for Hispanic children than for Caucasian children. In addition, we found that children who slept < 7.5 hours/night during baseline had borderline significant odds ratio for being overweight/obese, anxious or depressed, and having learning problems at follow-up compared to those who slept ≥ 9 hours/night.

Other studies have found consistent relationships between short sleep duration and higher body weight in children.[4,6-8] Sekine et al. found a dose-response relationship between short sleep hours and childhood obesity in a cohort study of 8,274 Japanese children aged 6-7 years old.[32] This study showed that

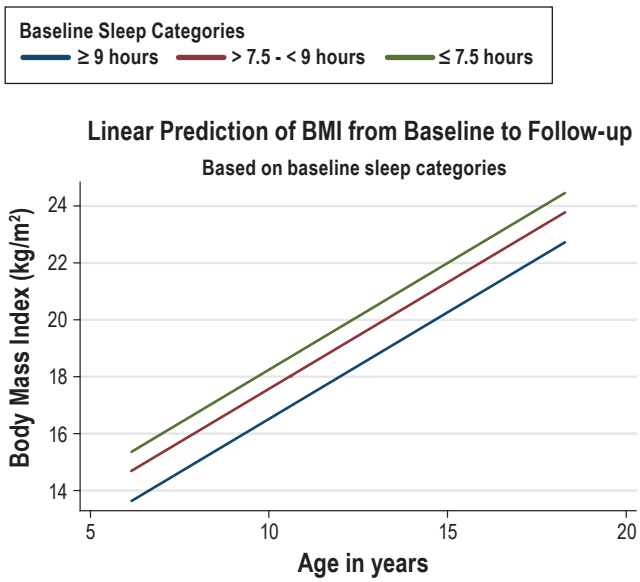


**Figure 2**—Linear prediction of BMI (kg/m$^2$) at follow-up by baseline sleep categories ≥ 9 h/night, > 7.5 to < 9 h/night, and ≤7.5 h/night (adjusted for baseline and follow-up SDB, age, sleep time, and ethnicity).

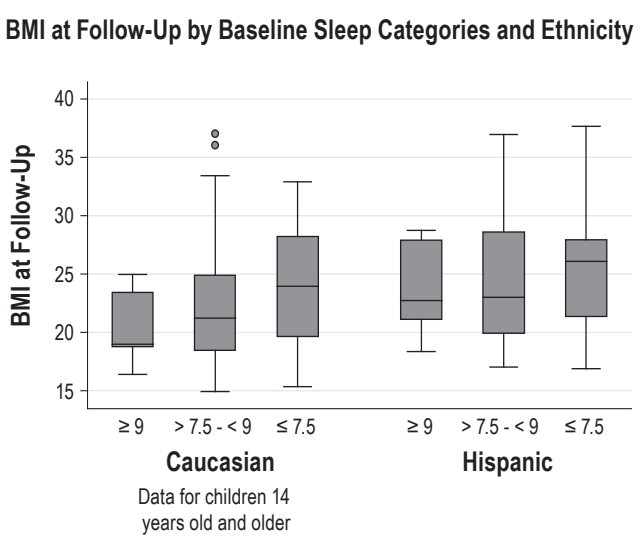


**Figure 3**—The lines in the middle of each box represent the median values for BMI (kg/m$^2$) at follow-up. Higher medians are seen for Hispanic than Caucasian children for ≥ 9 h/night (22.7 vs. 19.0), > 7.5 to < 9 h/night (23.0 vs. 21.2), and ≤7.5 h/night(26.1 vs. 23.9) sleep, respectively.

compared with children who slept ≥ 10 hours, the odds for obesity were 1.49 for those with 9-10 hours of sleep, 1.89 for those with 8-9 hours of sleep, and 2.87 for those with < 8 hours of sleep. The relationship was observed even after adjusting for age, gender, parental obesity, and children's television viewing. Similarly, Reilly et al. found that according to parental reports, sleeping fewer than 10.5 hours at age 3 increased the risk of obesity at age 7.[33] In a meta-analysis of cohort and cross-sectional studies in the general pediatric population, Chen et al. found that the pooled odds ratio for being overweight/obese was 1.58 (95% CI 1.26; 1.98) for children with short sleep duration compared to those with long sleep duration.[34] Similar to other studies, Chen et al.[35] also found that the effect of sleep on obesity differed for boys and girls.

Our results are consistent with previous findings relating short sleep to higher BMIs. Furthermore, our results extend these observations by documenting their occurrence in adolescents followed prospectively from childhood. In a cross-sectional study, Liu et al.[36] found that BMI z-scored was inversely associated with REM density. Although we did not assess REM density, our longitudinal evaluations using %REM and minutes in REM did not show similar associations. We also did not find gender differences between sleep time and BMI in the present study. However, unlike prior studies that utilized parent-reported sleep times, we used objective measurements of sleep derived from PSGs. The methodology for measuring total sleep time is important, because we know that parent-reported sleep times tend to overestimate sleep times derived from objective measurements of sleep—namely PSG.[37-39] Thus, we feel that PSG-derived sleep time provides a more reliable measure of actual time spent asleep.

Studies have shown that obesity rates and sleep patterns vary by ethnicity.[15] Hispanic adolescents have one of the highest rates of obesity and sleep disturbances in the United States.[40] Two studies found that among college students, Hispanics had higher rates of insomnia and greater dissatisfaction with sleep quality than Caucasian students.[41,42] Taveras et al. found that infants and toddlers who slept less than 12 hours a day were 2.04 times more likely to become overweight by age 3.[9] The authors also found that Hispanic children or those from other ethnicities were more likely to sleep less than Caucasian children, and that this association was sustained even after adjusting for maternal education, income, and marital status. Our findings demonstrate that the greater impact of reduced sleep in Hispanic children is consistent from childhood through adolescence.

Many reasons exist for the reduction of opportunity to sleep in children and adolescents. Adolescents spend more time on the internet and watching television.[43] Moreover, adolescents also may engage in extracurricular activities and employment, which further delay bedtime. Delayed sleep time, coupled with early school start times, imposes a significant constraint in adolescents' sleep schedules and predisposes them to reductions in total sleep time.[44,45] The present study found borderline significance for total sleep duration to be associated with learning problems. Such evidence supports the known relationship between sleep and memory retention.[46] Studies suggest that information acquired during the waking hours is "reactivated" or "consolidated" during subsequent REM sleep.[46,47] Therefore, disorders that affect sleep, especially REM sleep, can be linked to the development of learning problems. The association between sleep and behavioral or emotional problems in adolescents has been described both as a cause and consequence of short sleep.[48] We found that compared to those who slept ≥ 9 h/night, adolescents who slept < 7.5 h/night had a borderline higher odds of being anxious or depressed and a nearly significant higher odds for learning problems. These findings provide further evidence that obtaining sufficient sleep during childhood is potentially an important intervention to remediate subsequent learning and behavior problems.

Studies have previously reported on the longitudinal association between sleep problems and cognitive and behavioral

deficits.[49,50] The present study, however, is novel in that it utilizes objective sleep measures as opposed to parental sleep reports, which may reduce possible biases. Potential explanations for our findings may be that children with short sleep during their early years experienced behavioral and learning disruptions that further set the stage for a continuation of behavioral and learning challenges into adolescence. These mechanisms however, should be further evaluated in different cohort populations, which include other possible factors affecting cognitive and behavioral outcomes in adolescents such as family dynamics[51] that were not collected in our study.

We acknowledge some limitations with our study in that learning problems were based on one question and that answers to this question could be subject to parental bias or interpretation. Learning problems among children may be due to a wide range of factors including personal, school, or home environment. However, in the present study we did not assess these variables. In addition, our assessment of sleep time is based on a single-night, unattended, ambulatory sleep study. PSG measurements may be affected by subjects' discomfort from sensors and equipment and may not adequately represent habitual sleep patterns. Other studies however, have reported that unattended studies are reliable for measuring sleep time and have little first night effect.[52,53] Similarly, results from TuCASA analyses on night-to-night variability in key polysomnographic parameters showed a high degree of reproducibility on two different nights of study using identical protocols in the same child (n = 10).[18] Furthermore, the sleep architecture data presented in Table 1 shows that sleep in TuCASA children is comparable to normal sleep in this age. In a previous analysis, we compared parental-reported sleep time to PSG total sleep time.[37] We found that, consistent with other studies,[39,54] parents tended to overestimate their children's actual total sleep time, further supporting our use of PSG sleep time. In addition, we did not adjust for multiple comparisons which may have resulted in inflated type I error. However, given the consistency of our results with multiple endpoints, we think this is unlikely. We also did not discern whether PSGs were performed on the night of a weekday or weekend. However, 83% or the studies were performed on a weekday night, and thus, we feel this had little effect on our results. Furthermore, any effect regarding the use of weekend studies would result in dilution of our findings, given the propensity for adolescents to sleep longer on weekends, and a regression towards the null hypothesis.

Causal pathways linking chronic sleep deprivation with obesity point to neurohormonal effects that increase caloric intake. Studies have shown that sleep restriction results in reduced levels of the *satiety* hormone leptin and increased levels of the *hunger* hormone ghrelin.[55,56] Conceivably, the deleterious changes in the leptin-ghrelin axis due to sleep disruption may have long-term impacts that spill into adolescence. Understanding the complex relationship between sleep restriction and increase in obesity is crucial in order to design appropriate behavioral interventions for children and adolescents, which may include counseling interventions on appropriate sleep requirements. Such interventions, if effective, may potentially influence the growing threat of obesity.

In conclusion, results from this study strongly suggest that there is an association between reduced amounts of sleep during childhood and increase in body weight in early adolescence, and that this association is independent of SDB. Hispanics appear to be at higher risks for restricted sleep and increased BMI in adolescence than Caucasians. Whether this trend continues into late adolescence and early adulthood remains to be determined. Behavioral or other interventions aimed at improving sleep duration are needed to determine if a causal relationship exists between sleep deprivation and obesity, and in order to promote health and well-being of children and adolescents.


## ACKNOWLEDGMENTS

This study was performed at the University of Arizona, Tucson, AZ. The TuCASA study was supported by NHLBI grant HL 62373. Dr. Silva was supported by NHLBI grant HL 62373-05A2S1. The authors thank the participants of the TuCASA study.


## DISCLOSURE STATEMENT

This was not an industry supported study. The authors have indicated no financial conflicts of interest.

## REFERENCES


1. Kiess W, Gausche R, Keller A, Burmeister J, Willgerodt H, Keller E. Computer-guided, population-based screening system for growth disorders (CrescNet) and on-line generation of normative data for growth and development. Horm Res 2001;56 Suppl 1:59-66.
2. Ogden CL, Flegal KM. Changes in terminology for childhood overweight and obesity. National health statistics reports; no 25. Hyattsville, MD: National Center for Health Statistics, 2010.
3. Dollman J, Ridley K, Olds T, Lowe E. Trends in the duration of school-day sleep among 10- to 15-year-old South Australians between 1985 and 2004. Acta Paediatr 2007;96:1011-4.
4. Chaput JP, Brunet M, Tremblay A. Relationship between short sleeping hours and childhood overweight/obesity: results from the 'Quebec en Forme' Project. Int J Obes (Lond) 2006;30:1080-5.
5. Chaput JP, Despres JP, Bouchard C, Tremblay A. Short sleep duration is associated with reduced leptin levels and increased adiposity: Results from the Quebec family study. Obesity 2007;15:253-61.
6. Locard E, Mamelle N, Billette A, Miginiac M, Munoz F, Rey S. Risk factors of obesity in a five year old population. Parental versus environmental factors. Int J Obes Relat Metab Disord 1992;16:721-9.
7. Padez C, Mourao I, Moreira P, Rosado V. Prevalence and risk factors for overweight and obesity in Portuguese children. Acta Paediatr 2005;94:1550-7.
8. Patel SR, Hu FB. Short sleep duration and weight gain: a systematic review. Obesity 2008;16:643-53.
9. Taveras EM, Rifas-Shiman SL, Oken E, Gunderson EP, Gillman MW. Short sleep duration in infancy and risk of childhood overweight. Arch Pediatr Adolesc Med 2008;162:305-11.
10. Lauderdale DS, Knutson KL, Rathouz PJ, Yan LL, Hulley SB, Liu K. Cross-sectional and longitudinal associations between objectively measured sleep duration and body mass index: the CARDIA Sleep Study. Am J Epidemiol 2009;170:805-13.
11. Snell EK, Adam EK, Duncan GJ. Sleep and the body mass index and overweight status of children and adolescents. Child Dev2007;78:309-23.
12. Landhuis CE, Poulton R, Welch D, Hancox RJ. Childhood sleep time and long-term risk for obesity: a 32-year prospective birth cohort study. Pediatrics 2008;122:955-60.
13. Johnson-Taylor WL, Everhart JE. Modifiable environmental and behavioral determinants of overweight among children and adolescents: report of a workshop. Obesity 2006;14:929-66.
14. Carskadon MA. Factors influencing sleep patterns of adolescents. In: Carskadon MA, ed. Adolescent sleep patterns: Biological, social, and psychological influences. Cambridge, UK: Cambridge University Press, 2002:4-26.
15. 2010 Sleep in America Poll. National Sleep Foundation. Washington, DC. [cited 2010 May 12]; Available from: www.sleepfoundation.org

16. Carskadon MA, Harvey K, Duke P, Anders TF, Litt IF, Dement WC. Pubertal changes in daytime sleepiness. Sleep 1980;2:453-60.
17. Pizza F, Contardi S, Antognini AB, et al. Sleep quality and motor vehicle crashes in adolescents. J Clin Sleep Med 2010;6:41-5.
18. Goodwin JL, Enright PL, Kaemingk KL, et al. Feasibility of using unattended polysomnography in children for research--report of the Tucson Children's Assessment of Sleep Apnea study (TuCASA). Sleep 2001;24:937-44.
19. Goodwin JL, Kaemingk KL, Fregosi RF, et al. Clinical outcomes associated with sleep-disordered breathing in Caucasian and Hispanic children--the Tucson Children's Assessment of Sleep Apnea study (TuCASA). Sleep 2003;26:587-91.
20. Centers for disease control and prevention. A SAS program for the CDC growth charts. http://www.cdc.gov/nccdphp/dnpao/growthcharts/resources/sas.htm. Accessed July 21, 2010.
21. Dibley MJ, Goldsby JB, Staehling NW, Trowbridge FL. Development of normalized curves for the international growth reference: historical and technical considerations. Am J Clin Nutr 1987;46:736-48.
22. Dibley MJ, Staehling N, Nieburg P, Trowbridge FL. Interpretation of Z-score anthropometric indicators derived from the international growth reference. Am J Clin Nutr 1987;46:749-62.
23. Rechtschaffen A, Kales A. A manual of standardized terminology: techniques and scoring systems for sleep stages of human subjects. Los Angeles: UCLA Brain Information Service/Brain Research Institute, 1968.
24. Sleep-related breathing disorders in adults: recommendations for syndrome definition and measurement techniques in clinical research. The Report of an American Academy of Sleep Medicine Task Force. Sleep 1999;22:667-89.
25. Huang YS, Chen NH, Li HY, Wu YY, Chao CC, Guilleminault C. Sleep disorders in Taiwanese children with attention deficit/hyperactivity disorder. J Sleep Res 2004;13:269-77.
26. Rosen CL, Larkin EK, Kirchner HL, et al. Prevalence and risk factors for sleep-disordered breathing in 8- to 11-year-old children: association with race and prematurity. J Pediatr 2003;142:383-9.
27. Achenbach TM, Rescorla LA. Manual for ASEBA school-age forms & profiles. Burlington, VT: University of Vermont, Research Center for Children, Youth, & Families, 2001.
28. Rockett HR, Breitenbach M, Frazier AL, et al. Validation of a youth/adolescent food frequency questionnaire. Prev Med 1997;26:808-16.
29. Rockett HR, Colditz GA. Assessing diets of children and adolescents. Am J Clin Nutr 1997;65:1116S-22S.
30. Rockett HR, Wolf AM, Colditz GA. Development and reproducibility of a food frequency questionnaire to assess diets of older children and adolescents. J Am Diet Assoc 1995;95:336-40.
31. Drescher AA, Goodwin JL, Silva GE, Quan SF. Association between sleep, and dietary, exercise and electronic screen habits of adolescents in the Tucson Children's Assessment of Sleep Apnea (TuCASA) study. Sleep 2009;32:A106.
32. Sekine M, Yamagami T, Handa K, et al. A dose-response relationship between short sleeping hours and childhood obesity: results of the Toyama Birth Cohort Study. Child Care Health Dev 2002;28:163-70.
33. Reilly JJ, Armstrong J, Dorosty AR, et al. Early life risk factors for obesity in childhood: cohort study. BMJ 2005;330:1357.
34. Chen X, Beydoun MA, Wang Y. Is sleep duration associated with childhood obesity? A systematic review and meta-analysis. Obesity 2008;16:265-74.
35. St-Onge M, Perumean-Chaney S, Desmond R, et al. Gender differences in the association between sleep duration and body composition: The Cardia study. Int J of Endocrinol 2010;doi:10.1155/2010/726071.
36. Liu X, Forbes EE, Ryan ND, Rofey D, Hannon TS, Dahl RE. Rapid eye movement sleep in relation to overweight in children and adolescents. Arch Gen Psychiatry 2008;65:924-32.
37. Goodwin JL, Silva GE, Kaemingk KL, Sherrill DL, Morgan WJ, Quan SF. Comparison between reported and recorded total sleep time and sleep latency in 6- to 11-year-old children: the Tucson Children's Assessment of Sleep Apnea Study (TuCASA). Sleep Breath 2007;11:85-92.
38. Owens JA, Spirito A, McGuinn M, Nobile C. Sleep habits and sleep disturbance in elementary school-aged children. J Dev Behav Pediatr 2000;21:27-36.
39. Sekine M, Chen X, Hamanishi S, Wang H, Yamagami T, Kagamimori S. The validity of sleeping hours of healthy young children as reported by their parents. J Epidemiol 2002;12:237-42.
40. Anonymous. Obesity: preventing and managing the global epidemic. Report of a WHO consultation. World Health Organization Technical Report Series 2000;894:i-xii.
41. Hicks RA, Lucero-Gorman K, Bautista J, Hicks GJ. Ethnicity, sleep duration, and sleep satisfaction. Percept Mot Skills 1999;88:234-5.
42. Roberts RE, Roberts CR, Chen IG. Ethnocultural differences in sleep complaints among adolescents. J Nerv Ment Dis 2000;188:222-9.
43. Anderson SE, Economos CD, Must A. Active play and screen time in US children aged 4 to 11 years in relation to sociodemographic and weight status characteristics: a nationally representative cross-sectional analysis. BMC Public Health 2008;8:366.
44. Marshall SJ, Gorely T, Biddle SJ. A descriptive epidemiology of screen-based media use in youth: a review and critique. J Adolesc 2006;29:333-49.
45. Wolfson AR, Carskadon MA. Sleep schedules and daytime functioning in adolescents. Child Dev 1998;69:875-87.
46. Maquet P, Laureys S, Peigneux P, et al. Experience-dependent changes in cerebral activation during human REM sleep. Nat Neurosci 2000;3:831-6.
47. Wetzel W, Wagner T, Balschun D. REM sleep enhancement induced by different procedures improves memory retention in rats. Eur J Neurosci 2003;18:2611-7.
48. Morrison DN, McGee R, Stanton WR. Sleep problems in adolescence. J Am Acad Child Adolesc Psychiatry 1992;31:94-9.
49. Gregory AM, Caspi A, Moffitt TE, Poulton R. Sleep problems in childhood predict neuropsychological functioning in adolescence. Pediatrics 2009;123:1171-6.
50. Gregory AM, O'Connor TG. Sleep problems in childhood: a longitudinal study of developmental change and association with behavioral problems. J Am Acad Child Adolesc Psychiatry 2002;41:964-71.
51. Gregory AM, Eley TC, O'Connor TG, Rijsdijk FV, Plomin R. Family influences on the association between sleep problems and anxiety in a large sample of pre-school aged twins. Pers Individ Dif 2005;39:1337-48.
52. Gouveris H, Selivanova O, Bausmer U, Goepel B, Mann W. First-night-effect on polysomnographic respiratory sleep parameters in patients with sleep-disordered breathing and upper airway pathology. Eur Arch Otorhinolaryngol 2010;267:1449-53.
53. Verhulst SL, Schrauwen N, De Backer WA, Desager KN. First night effect for polysomnographic data in children and adolescents with suspected sleep disordered breathing. Arch Dis Child 2006;91:233-7.
54. Tremaine RB, Dorrian J, Blunden S. Subjective and objective sleep in children and adolescents: Measurement, age, and gender differences. Sleep Biol Rhythms 2010;8:229-38.
55. Spiegel K, Tasali E, Penev P, Van Cauter E. Sleep curtailment in healthy young men is associated with decreased leptin levels, elevated ghrelin levels, and increased hunger and appetite. Ann Intern Med 2004;141:846-51.
56. Taheri S, Lin L, Austin D, Young T, Mignot E. Short sleep duration is associated with reduced leptin, elevated ghrelin, and increased body mass index. PLoS Med 2004;1:e62.