Chronic Sleep Curtailment and Adiposity

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WHAT'S KNOWN ON THIS SUBJECT: Curtailed sleep in children has been found to be associated with increased cardiovascular disease risk factors, including obesity. Few existing studies have examined measures of adiposity beyond BMI or have examined the effects of being chronically sleep curtailed.

WHAT THIS STUDY ADDS: In this cohort of children who had research-level measures of sleep, BMI, total fat mass, and fat mass distribution, we found that chronic sleep curtailment from infancy to age 7 years was associated with higher overall and central adiposity in mid-childhood.

abstract

OBJECTIVES: To examine the extent to which chronic sleep curtailment from infancy to mid-childhood is associated with total and central adiposity.

METHODS: We studied 1046 children participating in a prospective cohort study. At age 6 months and yearly from age 1 to 7 years, mothers reported their children's sleep duration in a usual 24-hour period. The main exposure was a sleep curtailment score from age 6 months to 7 years. The range of the total score was 0 to 13, where 0 indicated the maximal sleep curtailment and 13 indicated never having curtailed sleep. Outcomes in mid-childhood were BMI z score, dual X-ray absorptiometry total and trunk fat mass index (kg/m^2), and waist and hip circumferences (cm).

RESULTS: The mean (SD) sleep score was 10.2 (2.7); 4.4% scored a 0 to 4, indicating multiple exposures to sleep curtailment between age 6 months to 7 years, 12.3% scored 5 to 7, 14.1% scored 8 to 9, 28.8% scored 10 to 11, and 40.3% scored 12 to 13. In multivariable models, children who had a sleep score of 0 to 4 had a BMI z score that was 0.48 U (95% confidence interval, 0.13 to 0.83) higher than those who had a sleep score of 12 to 13. We observed similar associations of higher total and trunk fat mass index and waist and hip circumferences, and higher odds of obesity (odds ratio, 2.62; 95% confidence interval, 0.99 to 6.97) among children who had a score of 0 to 4 vs 12 to 13.

CONCLUSIONS: Chronic sleep curtailment from infancy to school age was associated with higher overall and central adiposity in mid-childhood. *Pediatrics* 2014;133:1013–1022

Across infancy, childhood, and adolescence, evidence from multiple US and international studies suggests a consistent rapid decline in sleep duration ranging from 30 to 60 minutes over the last 20 years.[1] Some[2] but not all studies[3,4] suggest that the most pronounced decreases in sleep have occurred among children younger than 5 years of age, owing largely to later bedtimes. Recent studies also suggest that the quality of sleep among young children is often compromised,[5] and that pediatric sleep disturbances often become chronic.[6,7]

Curtailed sleep in children, defined as shortened sleep relative to average sleep duration, has been found to be associated with increased cardiovascular disease risk factors such as obesity, hypertension, and elevated hemoglobin A1c levels. Cross-sectional studies of older children and adults[8–19] and increasingly some longitudinal studies, including those of infants and children younger than age 5 years, suggest an inverse association between sleep duration and obesity and cardiovascular disease risk factors.[20–26] Few studies, however, have examined the effects of chronic or repeated sleep curtailment on adiposity outcomes in children and few pediatric studies have examined measures of adiposity beyond BMI. Furthermore, questions remain on whether there is a "critical period" in early childhood in which curtailed sleep is more highly associated with adverse anthropometric outcomes as has been suggested in the published literature.[21]

The purpose of this study was to examine (1) the extent to which chronic sleep curtailment throughout infancy to mid-childhood is associated with obesity, adiposity, and central fast mass distribution in a cohort of US-born children and (2) independent associations of curtailed sleep in each of 3 distinct age periods, infancy, early childhood, and mid-childhood, to determine whether there are critical periods for the development of associations between sleep and obesity. We hypothesized that being chronically sleep curtailed throughout infancy to mid-childhood would be associated with more adverse anthropometric indices. We further hypothesized that sleep curtailment in infancy would be associated with adverse adiposity and obesity outcomes at age 7 years and that the magnitude of the associations would decrease with age. A better understanding of the extent to which chronic curtailed sleep in early childhood affects overall and central adiposity could help support interventions to promote healthful sleep as part of childhood obesity interventions.

METHODS

Subjects/Study Design

Study subjects were participants in Project Viva, a prospective, pre-birth cohort study that recruited women during early pregnancy from Harvard Vanguard Medical Associates, a multi-specialty group practice in eastern Massachusetts.[27] Details of recruitment and retention procedures are available elsewhere.[27] Of the 2128 women who delivered a live infant, 1683 children were eligible for 7-year (mid-childhood) follow-up, of whom 1116 attended a mid-childhood in-person visit. Because our main exposure was chronic sleep curtailment from age 6 months to 7 years, we excluded 70 participants who did not have sleep data for these time points. Thus, our sample size for analysis was 1046 children. Compared with the 1046 participants in this analysis, non-participants were less likely to have college-educated mothers (59% vs 71%) and to have annual household incomes exceeding $70 000 (52% vs 63%). Mean maternal age (31.3 years vs 32.3 years), however, was fairly similar.

After obtaining informed consent, we performed in-person study visits with the mother at the end of the first and second trimesters of pregnancy, and with mother and child in the first few days after delivery and in infancy (median, age 6.2 months), early childhood (median, age 3.3 years), and mid-childhood (median, age 7.7 years). Mothers completed mailed questionnaires at 1, 2, 4, 5, and 6 years after birth. Institutional Review Boards of participating institutions approved the study protocols.

Measurements

Main Exposures

At age 6 months and yearly from age 1 to 7 years, mothers reported their children's sleep duration in a usual 24-hour period. At 6 months, we asked mothers: (1) "In the past month, on average, for how long does your baby nap during the morning?" (2) "In the past month, on average, for how long does your baby nap during the afternoon?" and (3) "In the past month, on average, how many hours does your baby sleep during the night?" Response options were in hours and minutes. At age 1 year, we asked, "In the past month, on average, for how long does your child sleep in a usual 24-hour period? Please include morning naps, afternoon naps, and nighttime sleep." Response options were in hours and minutes. Between age 2 and 7 years, we asked parents to report the number of hours their child slept in a usual 24-hour period on an average weekday and weekend day in the past month. Response categories included "<9 hours, 9 hours a day, 10 hours a day, 11 hours a day, 12 hours a day, 13 hours a day, and 14 or more hours a day"; at age 7 years the response option was in hours and minutes.

The main exposure was a sleep curtailment score from age 6 months to 7 years. We based the sleep score on

mean sleep duration at each of the 8 measurement times: age 6 months and yearly from age 1 to 7 years. Using established thresholds from the published literature[28] of sleep durations that are associated with an increased risk for elevated BMI or obesity (BMI ≥95th percentile) in childhood, specific sleep duration cutoffs associated in crude analyses with increased obesity risk at age 7 years in the Project Viva cohort, and the National Sleep Foundation age-specific sleep recommendations,[29] we coded sleep duration at each time period as follows: from age 6 months to 2 years, score was 0 for <12 hours/day and 1 for ≥12 hours/day; from age 3 to 4 years, <10 hours/day = 0, 10 to <11 hours/day = 1, and ≥11 hours/day = 2; at age 5 to 7 years, <9 hours/day = 0, 9 to <10 hours/day = 1, and ≥10 hours/day = 2. The range of the total score was 0 to 13, where 0 indicated the maximal sleep curtailment and 13 indicated never having curtailed sleep.

As a secondary exposure we also examined sleep curtailment at each age separately but collapsed into 3 age periods, age 6 months to 2 years, 3 to 4 years, and 5 to 7 years, based on comparable results and ease of interpretation. We defined curtailed sleep as <12 hours/day from age 6 months to 2 years, <10 hours/day from age 3 to 4 years, and <9 hours/day from age 5 to 7 years.

Outcome Measures

At age 7 years, we measured height and weight of children using a calibrated stadiometer (Shorr Productions, Olney, MD) and scale (Seca model 881, Seca Corp, Hanover, MD). We calculated age- and gender-specific BMI z scores by using US national reference data.[30] We defined obesity as a BMI for age and gender ≥95th percentile.[30] We also measured total and trunk fat using dual-energy radiograph absorptiom-

etry and calculated fat mass index (FMI, kg/m^2), waist circumference (cm) and hip circumference (cm) using a Lefkin woven tape, and subscapular and triceps skinfold thicknesses (SS+TR) using Holtain calipers (Holtain LTD, Crosswell, United Kingdom), and calculated the sum (SS+TR) of the 2 thicknesses. Research assistants performing all measurements followed standardized techniques,[31] and participated in in-service training to ensure measurement validity.[32]

Other Measures

At enrollment, we collected information about maternal age, education, parity, household income, and child's race and ethnicity. In mid-childhood, we asked parents to report the number of hours their children watched television/ videos on an average weekday and weekend day in the past month. Response categories included, "none, <1 hour a day, 1–3 hours a day, 4–6 hours a day, 7–9 hours a day, and 10 or more hours a day." Mothers also reported their pre-pregnancy weight and height, from which we calculated their BMIs.

Statistical Analysis

We first examined bivariate relationships of child's sleep score and curtailed sleep with each covariate and with our anthropometric outcomes. We then used multivariable linear or logistic regression models to examine the associations of child's sleep score and curtailed sleep with our anthropometric outcomes with and without the inclusion of potential confounders. Our first model, Model 1, was adjusted for child age and gender only. We then additionally adjusted the multivariable models for potential confounders including sociodemographic variables and maternal BMI (Model 2) and child television viewing at mid-childhood (Model 3).

To examine the independent effects of curtailed sleep in each age period on the anthropometric outcomes, we ran separate multivariable models with each age period (Table 3). We then also adjusted curtailed sleep in the later age periods for curtailed sleep in earlier age periods by adding curtailment variables at earlier ages as additional covariates (Fig 2). The models of curtailed sleep from age 3 to 4 years were adjusted for curtailed sleep from age 6 months to 2 years; models of curtailed sleep from age 5 to 7 years were adjusted for curtailed sleep from age 6 months to 2 years and 3 to 4 years.

The confounding variables in our analyses were not available for all subjects. We therefore used multiple imputation to generate several plausible values for each missing value.[33,34] We used a chained equations approach with predictive mean matching based on linear regressions for approximately continuous variables and logistic or generalized logistic regression for dichotomous or more generally categorical variables. The "completed" data set comprises the observed data and 1 imputed value for each missing value. We replicated this analysis across completed data sets and then combined them in a structured fashion that accurately reflects the true amount of information in the observed data (ie, without erroneously presuming that the imputed values are known true values) but recovering the information in partially observed subjects. We generated 50 complete data sets[35] and combined multivariable modeling results (Proc MI ANALYZE) in SAS version 9.3 (SAS Institute, Cary, NC).

From these multiple imputation results, we report adjusted effect estimates or odds ratios (OR) from regressions and 95% confidence intervals (CI) for each sleep category with the lowest risk sleep category as the reference group.

RESULTS

Characteristics of study participants overall, and by sleep score, are shown in Table 1. Children who lived in homes with lower household incomes and lower maternal educational attainment were more likely to have lower sleep scores indicating more sleep curtailment from age 6 months to 7 years (Table 1). In addition, black, Hispanic, and other race/ethnicity children were more likely than white children to have curtailed sleep (Table 1). In mid-childhood, curtailed sleep was also associated with greater hours of television viewing (Table 1). In bivariate analyses (Table 1), children who had the lowest sleep score (most curtailed sleep) had higher indices of all of our anthropometric outcomes.

The mean (SD; range) total sleep score from age 6 months to 7 years was 10.2 (2.7; 0–13). The score frequencies are shown in Table 1. We collapsed scores of 0 to 4, 5 to 7, and 8 to 9 because of small frequencies and also of 10 to 11 and 12 to 13 because of comparable results. In multivariable models, adjusted for child's age, gender, race/ethnicity, and television viewing; maternal age, education, pre-pregnancy BMI, and parity; and household income, children who had lower sleep scores had higher anthropometric indices (Table 2, Model 3 and Fig 1). For example, children who had a score of 0 to 4 compared with a score of 12 to 13 had higher BMI z score (0.48 U; 95% CI, 0.13 to 0.83), sum of subscapular and triceps skinfold thickness (4.22 mm; 95% CI, 0.72 to 7.72), total FMI (0.72 kg/m^2; 95% CI, 0.02 to 1.42), trunk FMI (0.36 kg/m^2; 95% CI, 0.05 to 0.67), waist circumference (3.61 cm; 95% CI, 0.74 to 6.48), hip circumference (2.78 mm; 95% CI, 0.23 to 5.32), and a higher odds of obesity (OR, 2.62; 95% CI, 0.99 to 6.97).

Furthermore, BMI z score was 0.05 U (95% CI, -0.07 to -0.02) less for each additional increment in the sleep score.

In multivariable analyses, we found that across all 3 age periods, children who had the most curtailed sleep had higher anthropometric indices. For example, children who had curtailed sleep from age 6 months to 2 years had a BMI z score that was 0.15 U (95% CI, 0.02 to 0.28) higher than the referent group (Table 3, Model 3). Similarly, from age 3 to 4 years and from age 5 to 7 years, children who had curtailed sleep had a higher BMI z score than the referent group (Table 3). We observed a similar trend of curtailed sleep across all age periods with overall higher anthropometric outcomes, although CIs in some cases spanned 0.

In multivariable models in which we additionally adjusted sleep curtailment in later age periods for sleep

TABLE 1 Characteristics of 1046 Children From Project Viva, Overall, and by Sleep Curtailment Score

Characteristics		Sleep Curtailment Score: 0 = Maximum Curtailed Sleep to 13 = Never Curtailed Sleep				
	Overall	0 to 4	5 to 7	8 to 9	10 to 11	12 to 13
	N = 1046	46 (4.4%)	129 (12.3%)	148 (14.1%)	302 (28.8%)	421 (40.3%)
Maternal and household				Mean (SD) or %		
Maternal age (y)	32.3 (0.16)	30.5 (6.8)	31.3 (7.1)	32.3 (6.9)	32.8 (5.5)	32.5 (4.5)
Maternal BMI (kg/m^2)	24.6 (0.16)	27.5 (8.0)	24.9 (6.3)	25.4 (6.1)	24.6 (5.5)	23.9 (4.7)
Parity, nulliparous, %	47.9	28.0	41.9	50.7	45.0	53.0
Education, ≥college graduate, %	70.8	33.8	49.0	63.0	75.9	80.7
Household income >$70 000, %	63.4	25.6	43.8	52.9	66.8	74.9
Child						
Girl, %	50.3	54.8	40.2	48.3	49.4	54.2
Race/ethnicity, %						
White	66.3	21.0	36.9	53.7	70.3	81.8
Black	14.8	38.9	33.8	26.4	12.4	4.2
Hispanic	3.4	7.5	8.8	3.4	2.0	2.2
Other	15.5	32.6	20.5	16.5	15.4	11.8
Television viewing at age 7 y (h/d)	1.5 (1.0)	2.4 (1.4)	2 (1.3)	1.7 (1.2)	1.5 (1.0)	1.3 (0.8)
Age 7 years anthropometric outcomes						
BMI z score (units)	0.37 (0.03)	1.01 (1.18)	0.58 (1.21)	0.48 (1.12)	0.35 (1.08)	0.21 (0.96)
Sum of subscapular and triceps skinfolds (mm)	19.5 (0.29)	26.3 (15.6)	21.3 (12.6)	20.6 (11.7)	19.4 (9.9)	18 (7.7)
DXA FMI (kg/m^2)	4.4 (0.06)	5.6 (3.0)	4.6 (2.5)	4.5 (2.2)	4.4 (2.0)	4.1 (1.6)
DXA trunk FMI (kg/m^2)	1.5 (0.03)	2 (1.4)	1.6 (1.1)	1.5 (1.0)	1.5 (0.9)	1.3 (0.7)
Waist circumference (cm)	59.7 (0.25)	64.8 (12.3)	61.5 (11.1)	60.7 (9.9)	59.7 (8.4)	58.2 (6.7)
Hip circumference (cm)	68.0 (0.23)	72.9 (10.8)	69.9 (10.0)	68.8 (8.8)	68.1 (8.2)	66.5 (6.7)
BMI category						
<85th percentile	75.0	55.3	66.4	70.0	74.9	81.7
85th to 95th percentile	13.8	10.7	15.3	15.7	16.0	11.5
≥95th percentile	11.1	34.0	18.3	14.2	9.1	6.8

DXA, dual-energy radiograph absorptiometry.

TABLE 2 Multivariable Associations of the Sleep Curtailment Score With Anthropometric Outcomes at Mid-Childhood

Anthropometric Outcomes and Multivariable Models[a]	Sleep Curtailment Score: 0 = Maximum Curtailed Sleep to 13 = Never Curtailed Sleep[b]					
	0 to 4	5 to 7	8 to 9	10 to 11	12 to 13	Continuous score, 0 to 13
BMI z score	Effect Estimate (95% Confidence Interval)					
Model 1. Child age and gender	0.80 (0.45 to 1.14)	0.36 (0.14 to 0.58)	0.27 (0.06 to 0.47)	0.13 (−0.02 to 0.29)	0.0 (ref)	−0.07 (−0.10 to −0.05)
Model 2. Model 1 + SES	0.53 (0.19 to 0.88)	0.25 (0.03 to 0.47)	0.14 (−0.06 to 0.35)	0.09 (−0.06 to 0.24)	0.0 (ref)	−0.05 (−0.08 to −0.02)
Model 3. Model 2 + TV	0.48 (0.13 to 0.83)	0.22 (0.00 to 0.44)	0.13 (−0.08 to 0.33)	0.08 (−0.07 to 0.23)	0.0 (ref)	−0.05 (−0.07 to −0.02)
SS+TR skinfold thickness, mm						
Model 1. Child age and gender	7.57 (4.12 to 11.02)	3.15 (1.12 to 5.19)	2.21 (0.34 to 4.07)	1.17 (−0.24 to 2.58)	0.0 (ref)	−0.66 (−0.88 to −0.44)
Model 2. Model 1 + SES	4.90 (1.42 to 8.37)	2.00 (−0.05 to 4.06)	1.09 (−0.77 to 2.94)	0.72 (−0.63 to 2.07)	0.0 (ref)	−0.43 (−0.67 to −0.19)
Model 3. Model 2 + TV	4.22 (0.72 to 7.72)	1.64 (−0.41 to 3.69)	0.88 (−0.96 to 2.73)	0.57 (−0.79 to 1.92)	0.0 (ref)	−0.37 (−0.60 to −0.13)
DXA total FMI, kg/m²						
Model 1. Child age and gender	1.36 (0.66 to 2.05)	0.47 (0.08 to 0.87)	0.37 (0.00 to 0.74)	0.22 (−0.06 to 0.51)	0.0 (ref)	−0.11 (−0.15 to −0.07)
Model 2. Model 1 + SES	0.88 (0.18 to 1.57)	0.28 (−0.12 to 0.69)	0.18 (−0.19 to 0.54)	0.15 (−0.13 to 0.42)	0.0 (ref)	−0.07 (−0.12 to −0.02)
Model 3. Model 2 + TV	0.72 (0.02 to 1.42)	0.20 (−0.20 to0.60)	0.13 (−0.23 to 0.49)	0.11 (−0.17 to 0.39)	0.0 (ref)	−0.06 (−0.10 to −0.01)
DXA trunk FMI, kg/m²						
Model 1. Child age and gender	0.63 (0.32 to 0.95)	0.24 (0.06 to 0.42)	0.17 (0.00 to 0.34)	0.12 (−0.01 to 0.24)	0.0 (ref)	−0.05 (−0.07 to −0.03)
Model 2. Model 1 + SES	0.44 (0.13 to 0.75)	0.17 (−0.01 to, 0.35)	0.09 (−0.07 to 0.26)	0.09 (−0.04 to 0.21)	0.0 (ref)	−0.04 (−0.06 to −0.02)
Model 3. Model 2 + TV	0.36 (0.05 to 0.67)	0.13 (−0.05 to 0.31)	0.07 (−0.09 to 0.23)	0.07 (−0.05 to 0.19)	0.0 (ref)	−0.03 (−0.05 to −0.01)
Waist circumference, cm						
Model 1. Child age and gender	5.64 (2.80 to 8.49)	2.39 (0.66 to 4.12)	1.79 (0.21 to 3.36)	0.93 (−0.25 to 2.11)	0.0 (ref)	−0.49 (−0.67 to −0.31)
Model 2. Model 1 + SES	4.19 (1.33 to 7.05)	1.97 (0.23 to 3.71)	1.11 (−0.45 to 2.66)	0.68 (−0.45 to 1.81)	0.0 (ref)	−0.37 (−0.57 to −0.18)
Model 3. Model 2 + TV	3.61 (0.74 to 6.48)	1.65 (−0.08 to 3.38)	0.93 (−0.62 to 2.48)	0.55 (−0.58 to 1.68)	0.0 (ref)	−0.32 (−0.52 to −0.12)
Hip circumference, cm						
Model 1. Child age and gender	5.32 (2.81 to 7.83)	2.45 (0.94 to 3.97)	1.39 (0.00 to 2.78)	1.00 (−0.10 to 2.10)	0.0 (ref)	−0.47 (−0.63 to −0.31)
Model 2. Model 1 + SES	3.21 (0.68 to 5.73)	1.44 (−0.08 to 2.95)	0.39 (−0.99 to 1.77)	0.61 (−0.46 to 1.68)	0.0 (ref)	−0.28 (−0.45 to −0.10)
Model 3. Model 2 + TV	2.78 (0.23 to 5.32)	1.21 (−0.31 to 2.72)	0.26 (−1.12 to 1.64)	0.51 (−0.56 to 1.58)	0.0 (ref)	−0.24 (−0.41 to −0.06)
Obesity (BMI ≥95th vs 5th to 85th percentile)	OR (95% CI)					
Model 1. Child age and gender	6.85 (3.01 to 15.57)	3.15 (1.65 to 6.04)	2.30 (1.17 to 4.54)	1.43 (0.77 to 2.67)	1.0 (ref)	0.82 (0.77 to 0.88)
Model 2. Model 1 + SES	3.16 (1.21 to 8.24)	2.04 (0.97 to 4.27)	1.51 (0.72 to 3.16)	1.16 (0.61 to 2.23)	1.0 (ref)	0.88 (0.81 to 0.96)
Model 3. Model 2 + TV	2.62 (0.99 to 6.97)	1.79 (0.84 to .83)	1.41 (0.68 to 2.95)	1.11 (0.57 to 2.14)	1.0 (ref)	0.90 (0.83 to 0.98)

Data are from 1046 children in Project Viva. DXA, dual-energy radiograph absorptiometry; SES, socioeconomic status; TV, television.
[a] Model 1 is adjusted only for child age and gender. Model 2 is additionally adjusted for maternal age, education, BMI, and parity; household income; and child race/ethnicity. Model 3 is further adjusted for child television viewing at mid-childhood.
[b] The range of the total sleep score is 0 to13, where 0 indicates the maximal sleep curtailment and 13 indicates never having curtailed sleep.

curtailment in the younger age periods (Fig 2), we observed fairly similar effect estimates for BMI z score among those who had curtailed sleep across all age periods.

DISCUSSION

In this prospective cohort, chronic sleep curtailment throughout infancy to mid-childhood was associated with higher overall and central adiposity at age 7 years. The adverse effects of sleep curtailment on adiposity were consistent across infancy, early-, and mid-childhood, and we did not find evidence for a particular critical period. The associations between curtailed sleep and overall and central adiposity persisted even after adjustment for

many potential confounders of the relationship between sleep and adiposity. We first examined the association between *chronic* sleep curtailment and our BMI and adiposity outcomes. A 0 to 4 value on our chronic curtailment score indicated multiple times from age 6 months to 7 years during which sleep duration was suboptimal based on published associations with obesity or age-specific recommendations. Previous studies, such as that by Landhuis et al,[26] have used repeated sleep measures to relate lower average sleep duration during ages 5 to 11 years with higher odds of obesity at age 32 years. Another study[36] used repeated measures of time in bed from ages 10 to 13 years to create 3 time-in-bed trajectories: short sleepers, 10.5-

hour sleepers, and 11-hour sleepers. Seegers et al[36] found that the short-sleeper trajectory was associated with a higher OR for being overweight (OR, 1.99; 95% CI, 1.67 to 2.37) and obese (OR, 2.23; 95% CI, 2.18 to 2.27) at 13 years of age compared with the 11-hour sleeper trajectory. Using our sleep curtailment score, we found that children who had more sleep curtailment from age 6 months to 7 years with observed thresholds between 0 to 4 had higher anthropometric indices. Given similar directional associations of all of the anthropometric outcomes with sleep curtailment, recent studies that show high correlation between several anthropometric indices,[37] and comparable clinical significance for a range of metabolic risk factors,[38,39]

our study might suggest that in epidemiologic studies of school-aged children in which dual-energy radiograph absorptiometry is not available or is cost-prohibitive, more feasible anthropometric measures such as BMI and skinfold thicknesses are reasonable surrogate measures.

Previous studies have suggested infancy and early childhood as critical periods for development of associations between sleep and obesity.[40] In a study of children birth to 13 years of age,[40] shorter duration of nighttime sleep from birth to age 4 years, but not from age 5 to 13 years, was associated with subsequent risk for overweight and obesity (OR, 1.80; 95% CI, 1.16 to 2.80). The authors concluded that there is a critical window before age 5 years when nighttime sleep may be important for subsequent obesity status. In contrast to this study, we did not find evidence of a critical period of sleep curtailment. Instead we found that

across infancy, early-, and mid-childhood, curtailed sleep was consistently associated with adverse anthropometric outcomes. Our findings suggest that sleep curtailment across childhood appears to be a developmentally important risk factor for increased adiposity.

Among adults and more recently among children and adolescents, several mechanisms have been proposed relating curtailed sleep and obesity, including indirect influence on diet through neurohormonal controls of hunger and satiation[41] as well as direct influences on sympathetic activity, elevation of cortisol and ghrelin levels, decreases in leptin levels, and/or impairing glucose tolerance.[42–46] Other studies suggest that circadian misalignment can contribute to metabolic dysfunction and adiposity.[47] Molecular circadian clocks exist in almost all tissues and contribute to the coordination of gene transcription involved in a range of metabolic processes.[48]

Studies show that central and peripheral circadian molecular clocks interact to achieve appropriate internal alignment of metabolic signaling as well as external alignment of cellular processes with the environment. It is also possible that common genetic pathways may underlie associations of sleep, obesity, and metabolic dysfunction. Emerging evidence from genetic studies in animals shows links between circadian "clock" genes, sleep, and metabolic systems.[49] For example, a loss of function mutation in the Circadian Locomotor Output Cycles Kaput (*CLOCK*) gene, the first mammalian circadian gene identified,[50–52] has been shown to alter circadian rhythmicity,[50,53] sleep architecture, and homeostasis,[54] and to result in hyperphagia, obesity, and metabolic dysfunction in mice.[55]

In early childhood, insufficient sleep might be a consequence of parental behaviors at bedtime and during the night[56] or household routines that

FIGURE 1
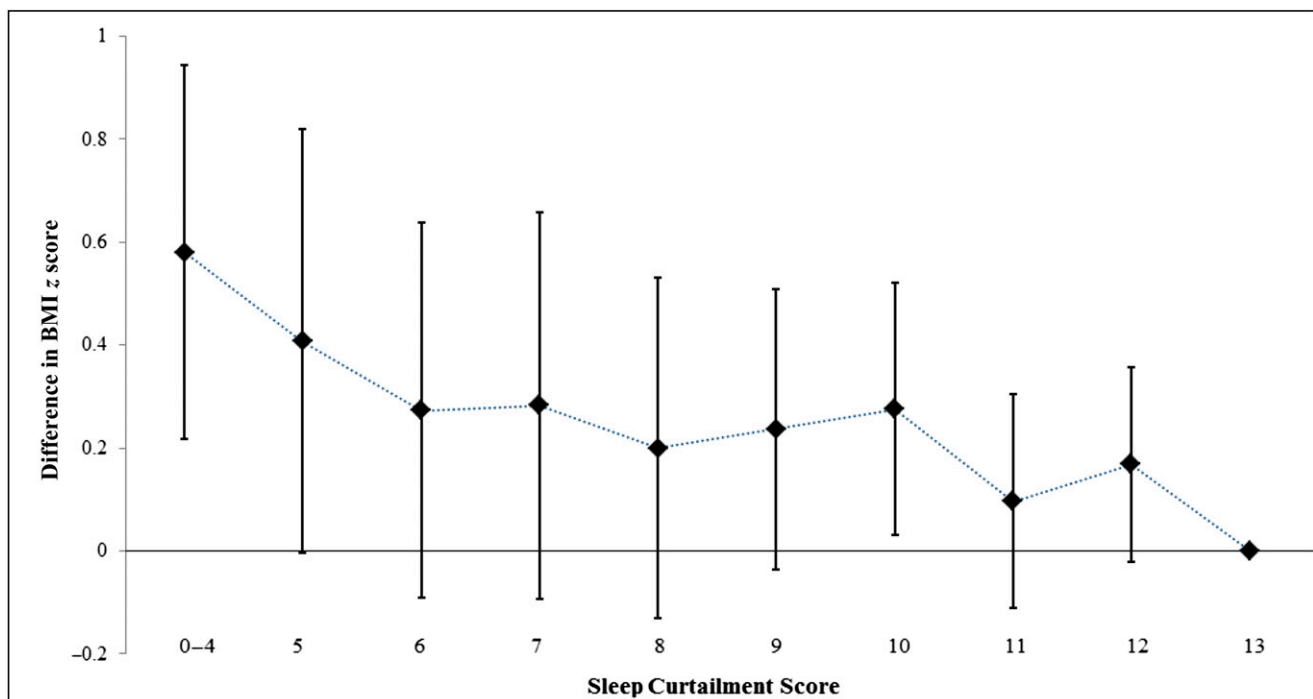Multivariable adjusted associations of the sleep curtailment score with BMI *z* score at mid-childhood. The range of the total sleep score is 0 to 13, where 0 indicates the maximal sleep curtailment and 13 indicates never having curtailed sleep. Models are adjusted for maternal age, education, BMI, and parity; household income; child race/ethnicity; and child television-viewing at mid-childhood.

TABLE 3 Multivariable Associations of Sleep Curtailment at Each Age Period With Anthropometric Outcomes at Mid-Childhood

Anthropometric Outcomes and Multivariable Models[a]

	Curtailed Sleep in Each Age Period							
	6 mo to 2 y		3 to 4 y			5 to 7 y		
	<12	≥12	<10	10 to <11	≥11	<9	9 to <10	≥10
	Effect Estimate (95% Confidence Interval)							
BMI z score								
Model 1. Child age and gender	0.25 (0.12 to 0.39)	0.0 (ref)	0.41 (0.21 to 0.61)	0.12 (−0.03 to 0.26)	0.0 (ref)	0.53 (0.21 to 0.85)	0.12 (−0.04 to 0.29)	0.0 (ref)
Model 2. Model 1 + SES	0.17 (0.04 to 0.30)	0.0 (ref)	0.23 (0.02 to 0.43)	0.08 (−0.06 to 0.22)	0.0 (ref)	0.29 (−0.04 to 0.61)	0.03 (−0.13 to 0.20)	0.0 (ref)
Model 3. Model 2 + TV	0.15 (0.02 to 0.28)	0.0 (ref)	0.20 (−0.01 to 0.40)	0.07 (−0.07 to 0.20)	0.0 (ref)	0.24 (−0.08 to 0.56)	0.01 (−0.15 to 0.18)	0.0 (ref)
SS+TR skinfold thickness, mm								
Model 1. Child age and gender	2.10 (0.92 to 3.29)	0.0 (ref)	3.08 (1.14 to 5.03)	0.97 (−0.31 to 2.25)	0.0 (ref)	5.77 (2.53 to 9.01)	1.56 (−0.01 to 3.13)	0.0 (ref)
Model 2. Model 1 + SES	1.24 (0.06 to 2.43)	0.0 (ref)	1.23 (−0.75 to 3.20)	0.54 (−0.71 to 1.78)	0.0 (ref)	3.64 (0.27 to 7.02)	0.76 (−0.78 to 2.31)	0.0 (ref)
Model 3. Model 2 + TV	1.00 (−0.19 to 2.19)	0.0 (ref)	0.86 (−1.12 to 2.83)	0.37 (−0.86 to 1.60)	0.0 (ref)	3.09 (−0.27 to 6.45)	0.54 (−0.99 to 2.07)	0.0 (ref)
DXA total FMI, kg/m^2								
Model 1. Child age and gender	0.39 (0.15 to 0.62)	0.0 (ref)	0.55 (0.17 to 0.93)	0.19 (−0.07 to 0.44)	0.0 (ref)	0.84 (0.20 to1.47)	0.24 (−0.07 to 0.55)	0.0 (ref)
Model 2. Model 1 + SES	0.24 (0.01 to 0.48)	0.0 (ref)	0.23 (−0.16 to 0.62)	0.11 (−0.13 to 0.36)	0.0 (ref)	0.46 (−0.20 to 1.12)	0.10 (−0.21 to 0.41)	0.0 (ref)
Model 3. Model 2 + TV	0.19 (−0.05 to 0.43)	0.0 (ref)	0.15 (−0.25 to 0.54)	0.07 (−0.17 to 0.32)	0.0 (ref)	0.33 (−0.32 to 0.98)	0.05 (−0.26 to 0.36)	0.0 (ref)
DXA trunk FMI, kg/m^2								
Model 1. Child age and gender	0.19 (0.09 to 0.30)	0.0 (ref)	0.27 (0.09 to 0.44)	0.10 (−0.01 to 0.21)	0.0 (ref)	0.37 (0.08 to 0.66)	0.12 (−0.02 to 0.26)	0.0 (ref)
Model 2. Model 1 + SES	0.13 (0.03 to 0.24)	0.0 (ref)	0.13 (−0.05 to 0.31)	0.06 (−0.04 to 0.17)	0.0 (ref)	0.21 (−0.09 to 0.51)	0.06 (−0.08 to 0.20)	0.0 (ref)
Model 3. Model 2 + TV	0.11 (0.00 to 0.21)	0.0 (ref)	0.09 (−0.09 to 0.27)	0.05 (−0.06 to 0.15)	0.0 (ref)	0.15 (−0.15 to 0.44)	0.04 (−0.10 to 0.18)	0.0 (ref)
Waist circumference, cm								
Model 1. Child age and gender	1.70 (0.72 to 2.69)	0.0 (ref)	2.37 (0.77 to 3.98)	0.85 (−0.22 to 1.92)	0.0 (ref)	3.65 (1.01 to 6.28)	0.99 (−0.33 to 2.31)	0.0 (ref)
Model 2. Model 1 + SES	1.23 (0.24 to 2.21)	0.0 (ref)	1.31 (−0.32 to 2.94)	0.60 (−0.43 to 1.64)	0.0 (ref)	2.41 (−0.27 to 5.08)	0.56 (−0.76 to 1.88)	0.0 (ref)
Model 3. Model 2 + TV	1.02 (0.03 to 2.01)	0.0 (ref)	0.99 (−0.64 to 2.62)	0.46 (−0.57 to 1.48)	0.0 (ref)	1.92 (−0.72 to 4.56)	0.36 (−0.94 to 1.67)	0.0 (ref)
Hip circumference, cm								
Model 1. Child age and gender	1.45 (0.55 to 2.35)	0.0 (ref)	2.53 (1.08 to 3.97)	0.93 (−0.05 to 1.91)	0.0 (ref)	4.16 (1.96 to 6.37)	0.79 (−0.38 to 1.96)	0.0 (ref)
Model 2. Model 1 + SES	0.76 (−0.13 to 1.64)	0.0 (ref)	1.16 (−0.31 to 2.62)	0.62 (−0.32 to 1.56)	0.0 (ref)	2.35 (0.09 to 4.60)	0.11 (−1.05 to 1.27)	0.0 (ref)
Model 3. Model 2 + TV	0.60 (−0.29 to 1.50)	0.0 (ref)	0.92 (−0.53 to 2.38)	0.51 (−0.42 to 1.45)	0.0 (ref)	1.99 (−0.27 to 4.24)	−0.03 (−1.19 to 1.12)	0.0 (ref)
Obesity (BMI ≥95th vs 5th to 85th percentile)	OR (95% CI)							
Model 1. Child age and gender	2.03 (1.33 to 3.08)	1.0 (ref)	2.64 (1.49 to 4.67)	1.17 (0.72 to, 1.90)	1.0 (ref)	4.63 (2.23 to 9.62)	1.60 (0.93 to 2.74)	1.0 (ref)
Model 2. Model 1 + SES	1.50 (0.93 to 2.40)	1.0 (ref)	1.41 (0.72 to 2.76)	1.04 (0.62 to 1.76)	1.0 (ref)	2.47 (1.05 to 5.77)	1.17 (0.65 to 2.13)	1.0 (ref)
Model 3. Model 2 + TV	1.36 (0.84 to 2.21)	1.0 (ref)	1.27 (0.64 to 2.52)	0.98 (0.58 to 1.66)	1.0 (ref)	2.12 (0.90 to 4.98)	1.09 (0.60 to 2.00)	1.0 (ref)

Data from 1046 children in Project Viva. SES, ; TV, television.
[a] Model 1 is adjusted only for child age and gender. Model 2 is additionally adjusted for maternal age, education, BMI, and parity; household income; and child race/ethnicity. Model 3 is further adjusted for child television viewing at mid-childhood.
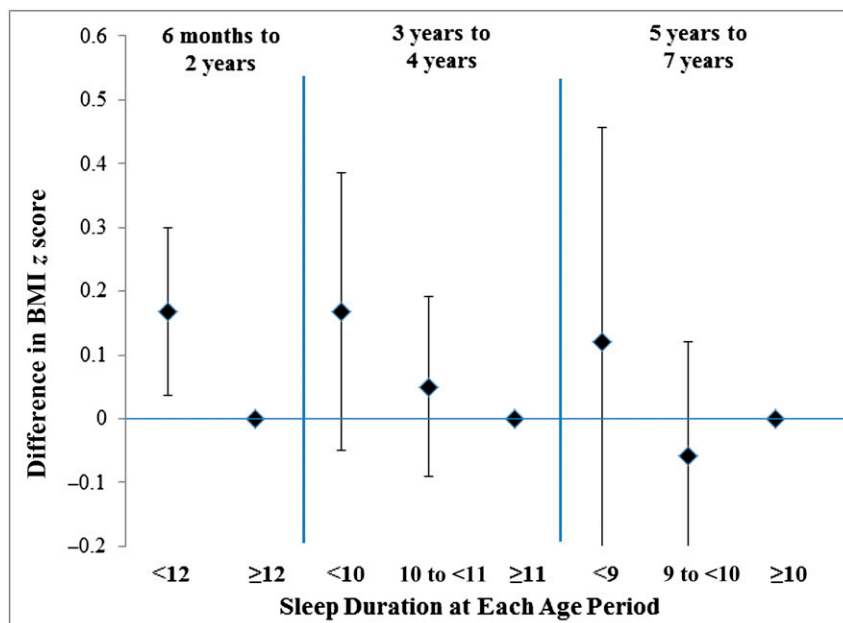
FIGURE 2
Multivariable adjusted associations of sleep curtailment at each age period with BMI z score at mid-childhood. Models of curtailed sleep in the later age periods are adjusted for curtailed sleep in earlier age periods. The models of curtailed sleep from age 3 to 4 years were adjusted for sleep duration from age 6 months to 2 years; models of curtailed sleep from age 5 to 7 years were adjusted for curtailed sleep from age 6 months to 2 years and 3 to 4 years, as well as for maternal age, education, BMI, and parity; household income; and child race/ethnicity.

might also increase risk for childhood obesity. Anderson et al[57] have found that among US preschool-aged children, exposure to 3 household routines (eg, regularly eating the evening meal as a family, obtaining adequate nighttime sleep, and having limited screen-viewing time) was associated with an approximately 40% lower prevalence of obesity than those exposed to none of these routines. Another potential mechanism that could explain our observed associations is the relationship between sleep duration, television viewing, and other screen-based technologies. Television viewing is a known risk factor for childhood obesity and is associated with short sleep duration. For these reasons, in the current study we adjusted for television viewing, which confounded the relationship between sleep and the adiposity, but sleep was still independently associated with our anthropometric outcomes. Other screen-based technologies such as computers, mobile telephones, and video gaming could also be associated with curtailed sleep and might be upstream or in the intermediate pathway in the relationship between sleep and obesity.[58]

Our study had several strengths. First, we collected longitudinal data on sleep duration beginning in early infancy through 7 years of age and used repeated measures of sleep to examine chronic sleep curtailment. Second, we analyzed several measures of adiposity in children, including skinfolds, FMIs, and circumferences as well as BMI from heights and weights. Most previous studies used BMI as the only outcome. Our study also had limitations. In this cohort, 38.9% of infants age 6 months to 2 years were defined as having sleep curtailment based on a cutoff of 12 hours of sleep. Although previous studies have also demonstrated a high prevalence of short sleep in infancy,[59] the cutoff of 12 hours may overestimate the number of children who are categorized as insufficient. We used the best available empirical evidence to define thresholds of sleep curtailment but because of biological variation and lack of mechanistic data, these cutoffs may not be appropriate across large populations. Maternal education and household income levels were relatively high, and we had underrepresentation of Hispanics in this cohort culled from eastern Massachusetts. Our results may not be generalizable to more socioeconomically disadvantaged populations, Hispanics, or to populations outside of Massachusetts. Second, in any observational study it is possible that unmeasured characteristics (eg, co-sleeping, bedroom sharing, and sleep routines) might explain the observed associations between exposure and outcome. Finally, we measured sleep duration by mother's report on the questionnaires as opposed to using an objective measure of sleep such as accelerometers or diaries. However, the potential misclassification of sleep duration is likely non-differential with respect to obesity and any resulting bias should be toward the null.

CONCLUSIONS

Sleep curtailment from infancy to school age was associated with higher overall and central adiposity in midchildhood. Improving sleep could be an achievable intervention for attenuating obesity risk.

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REFERENCES

1. Matricciani L, Olds T, Petkov J. In search of lost sleep: secular trends in the sleep time of school-aged children and adolescents. *Sleep Med Rev.* 2012;16(3):203–211

2. Matricciani LA, Olds TS, Blunden S, Rigney G, Williams MT. Never enough sleep: a brief history of sleep recommendations for children. *Pediatrics.* 2012;129(3):548–556

3. Williams JA, Zimmerman FJ, Bell JF. Norms and trends of sleep time among US children and adolescents. *JAMA Pediatr.* 2013; 167(1):55–60

4. Matricciani L, Olds T, Williams M. A review of evidence for the claim that children are sleeping less than in the past. *Sleep.* 2011; 34(5):651–659

5. National Sleep Foundation. *Sleep in America.* Princeton, NJ. 2008. Available at: http://sleepfoundation.org/. Accessed April 9, 2014

6. Kataria S, Swanson MS, Trevathan GE. Persistence of sleep disturbances in preschool children. *J Pediatr.* 1987;110(4):642–646

7. Pollock JI. Night-waking at five years of age: predictors and prognosis. *J Child Psychol Psychiatry.* 1994;35(4):699–708

8. 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(10):721–729

9. von Kries R, Toschke AM, Wurmser H, Sauerwald T, Koletzko B. Reduced risk for overweight and obesity in 5- and 6-y-old children by duration of sleep—a cross-sectional study. *Int J Obes Relat Metab Disord.* 2002;26(5):710–716

10. Brunet M, Chaput JP, Tremblay A, 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(2):163–170

11. Brunet M, Chaput JP, Tremblay A. The association between low physical fitness and high body mass index or waist circumference is increasing with age in children: the 'Québec en Forme' Project. *Int J Obes (Lond).* 2007 Apr;31(4):637–643

12. Gupta NK, Mueller WH, Chan W, Meininger JC. Is obesity associated with poor sleep quality in adolescents? *Am J Hum Biol.* 2002;14(6):762–768

13. Kagamimori S, Yamagami T, Sokejima S, et al. The relationship between lifestyle, social characteristics and obesity in 3-year-old Japanese children. *Child Care Health Dev.* 1999;25(3):235–247

14. Vioque J, Torres A, Quiles J. Time spent watching television, sleep duration and obesity in adults living in Valencia, Spain. *Int J Obes Relat Metab Disord.* 2000;24(12): 1683–1688

15. Padez C, Mourão I, Moreira P, Rosado V. Prevalence and risk factors for overweight and obesity in Portuguese children. *Acta Paediatr.* 2005;94(11):1550–1557

16. Knutson KL. Sex differences in the association between sleep and body mass index in adolescents. *J Pediatr.* 2005;147(6):830–834

17. Chen MY, Wang EK, Jeng YJ. Adequate sleep among adolescents is positively associated with health status and health-related behaviors. *BMC Public Health.* 2006;6:59

18. Seicean A, Redline S, Seicean S, Kirchner HL, Gao Y, Sekine M, Zhu X, Storfer-Isser A. Association between short sleeping hours and overweight in adolescents: results from a US Suburban High School survey. *Sleep Breath.* 2007;11(4):285–293

19. Eisenmann JC, Ekkekakis P, Holmes M. Sleep duration and overweight among Australian children and adolescents. *Acta Paediatr.* 2006;95(8):956–963

20. 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(4):305–311

21. Bell JF, Zimmerman FJ. Shortened nighttime sleep duration in early life and subsequent childhood obesity. *Arch Pediatr Adolesc Med.* 2010;164(9):840–845

22. Lumeng JC, Somashekar D, Appugliese D, Kaciroti N, Corwyn RF, Bradley RH. Shorter sleep duration is associated with increased risk for being overweight at ages 9 to 12 years. *Pediatrics.* 2007;120(5):1020–1029

23. Agras WS, Hammer LD, McNicholas F, Kraemer HC. Risk factors for childhood overweight: a prospective study from birth to 9.5 years. *J Pediatr.* 2004;145(1):20–25

24. Reilly JJ, Armstrong J, Dorosty AR, Emmett PM, Ness A, Rogers I, Steer C, Sherriff A; Avon Longitudinal Study of Parents and Children Study Team. Early life risk factors for obesity in childhood: cohort study. *BMJ.* 2005 Jun 11;330(7504):1357

25. Snell EK, Adam EK, Duncan GJ. Sleep and the body mass index and overweight status of children and adolescents. *Child Dev.* 2007;78(1):309–323

26. 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(5):955–960

27. Gillman MW, Rich-Edwards JW, Rifas-Shiman SL, Lieberman ES, Kleinman KP, Lipshultz SE. Maternal age and other predictors of newborn blood pressure. *J Pediatr.* 2004;144(2): 240–245

28. Hart CN, Cairns A, Jelalian E. Sleep and obesity in children and adolescents. *Pediatr Clin North Am.* 2011;58(3):715–733

29. Benjamin Neelon SE, Reyes-Morales H, Haines J, Gillman MW, Taveras EM. Nutritional quality of foods and beverages on child-care centre menus in Mexico. *Public Health Nutr.* 2013;16(11):2014–22

30. National Center for Health Statistics. CDC Growth Charts, United States. Available at: www.cdc.gov/growthcharts/. 2000

31. Shorr IJ. *How to Weigh and Measure Children.* New York, NY: United Nations; 1986

32. Mueller WH, Martorell R. Reliability and accuracy of measurement. In: Lohman TG, Roche AF, Martorell R, eds. *Anthropometric Standardization Reference Manual.* Champaign, IL: Human Kinetics Books; 1988

33. Rubin DB. *Multiple Imputation for Nonresponse in Surveys.* New York, NY: J. Wiley and Sons; 1987

34. Horton NJ, Kleinman KP. Much ado about nothing: a comparison of missing data methods and software to fit incomplete data regression models. *Am Stat.* 2007;61 (1):79–90

35. White IR, Royston P, Wood AM. Multiple imputation using chained equations: issues and guidance for practice. *Stat Med.* 2011; 30(4):377–399

36. Seegers V, Petit D, Falissard B, et al. Short sleep duration and body mass index: a prospective longitudinal study in preadolescence. *Am J Epidemiol.* 2011;173(6): 621–629

37. Boeke CE, Oken E, Kleinman KP, Rifas-Shiman SL, Taveras EM, Gillman MW. Correlations among adiposity measures in school-aged children. *BMC Pediatr.* 2013;Jun 24:13–99

38. Dowd JB, Zajacova A, Aiello AE. Predictors of inflammation in U.S. children aged 3-16 years. *Am J Prev Med.* 2010;39(4):314–320

39. Falaschetti E, Hingorani AD, Jones A, et al. Adiposity and cardiovascular risk factors in a large contemporary population of prepubertal children. *Eur Heart J.* 2010;31(24): 3063–3072

40. Bell JF, Zimmerman FJ. Shortened nighttime sleep duration in early life and subsequent childhood obesity. *Arch Pediatr Adolesc Med.* 2010;164(9):840–845

41. Mozaffarian D, Hao T, Rimm EB, Willett WC, Hu FB. Changes in diet and lifestyle and long-term weight gain in women and men. *N Engl J Med.* 2011;364(25):2392–2404

42. Buxton OM, Cain SW, O'Connor SP, et al. Adverse metabolic consequences in humans of

prolonged sleep restriction combined with circadian disruption. *Sci Transl Med*. 2012;4 (129):129ra143

43. Fletcher EC. Invited review: physiological consequences of intermittent hypoxia: systemic blood pressure. *J Appl Physiol (1985)*. 2001;90(4):1600–1605

44. Leproult R, Copinschi G, Buxton O, Van Cauter E. Sleep loss results in an elevation of cortisol levels the next evening. *Sleep*. 1997;20(10):865–870

45. Mehra R, Redline S. Sleep apnea: a proinflammatory disorder that coaggregates with obesity. *J Allergy Clin Immunol*. 2008; 121(5):1096–1102

46. Mullington JM, Haack M, Toth M, Serrador JM, Meier-Ewert HK. Cardiovascular, inflammatory, and metabolic consequences of sleep deprivation. *Prog Cardiovasc Dis*. 2009;51(4):294–302

47. Scheer FA, Hilton MF, Mantzoros CS, Shea SA. Adverse metabolic and cardiovascular consequences of circadian misalignment. *Proc Natl Acad Sci USA*. 2009;106(11):4453–4458

48. Ko CH, Takahashi JS. Molecular components of the mammalian circadian clock. *Hum Mol Genet*. 2006;15(Spec No 2):R271–R277

49. Marcheva B, Ramsey KM, Affinati A, Bass J. Clock genes and metabolic disease. *J Appl Physiol (1985)*. 2009;107(5):1638–1646

50. Antoch MP, Song EJ, Chang AM, et al. Functional identification of the mouse circadian Clock gene by transgenic BAC rescue. *Cell*. 1997;89(4):655–667

51. King DP, Zhao Y, Sangoram AM, et al. Positional cloning of the mouse circadian clock gene. *Cell*. 1997;89(4):641–653

52. Vitaterna MH, King DP, Chang AM, et al. Mutagenesis and mapping of a mouse gene, Clock, essential for circadian behavior. *Science*. 1994;264(5159):719–725

53. Vitaterna MH, Ko CH, Chang AM, et al. The mouse Clock mutation reduces circadian pacemaker amplitude and enhances efficacy of resetting stimuli and phase-response curve amplitude. *Proc Natl Acad Sci USA*. 2006;103(24):9327–9332

54. Naylor E, Bergmann BM, Krauski K, et al. The circadian clock mutation alters sleep homeostasis in the mouse. *J Neurosci*. 2000;20(21):8138–8143

55. Turek FW, Joshu C, Kohsaka A, et al. Obesity and metabolic syndrome in circadian Clock mutant mice. *Science*. 2005;308(5724):1043–1045

56. Mindell JA, Sadeh A, Kohyama J, How TH. Parental behaviors and sleep outcomes in infants and toddlers: a cross-cultural comparison. *Sleep Med*. 2010;11(4):393–399

57. Anderson SE, Whitaker RC. Household routines and obesity in US preschool-aged children. *Pediatrics*. 2010;125(3):420–428

58. Arora T, Hussain S, Hubert Lam KB, Lily Yao G, Neil Thomas G, Taheri S. Exploring the complex pathways among specific types of technology, self-reported sleep duration and body mass index in UK adolescents. *Int J Obes (Lond)*. 2013;37(9):1254–60

59. Demirci JR, Braxter BJ, Chasens ER. Breastfeeding and short sleep duration in mothers and 6-11-month-old infants. *Infant Behav Dev*. 2012;35(4):884–886

VENOM VS. VENOM: *Each year seems to bring a story about new, invasive species displacing the indigenous fauna of the United States. This year is a bit different, as the invasive species is displacing a previously highly successful invader. As reported in* The New York Times *(Science: February 13, 2014), 'crazy ants,' first seen in Houston and parts of Florida a decade ago, have expanded their habitat throughout the South. Remarkably, they have done so at the expense of the fire ant. Crazy ants,* Nylanderia fulva, *are so named because of their helter-skelter movements. They feed on the same types of food and like the same types of ground nests as fire ants. However, unlike other ants, they have been quite successful competing with fire ants for food. Importantly, they are also less likely to be injured by the venom of fire ants.*

Scientists have shown that crazy ants have developed a unique fire ant venom detoxification system. In experiments, researchers recorded crazy ants demonstrating prolonged grooming behaviors following exposure to fire ant venom. They were able to show that the crazy ant was using its own venom, formic acid, to detoxify the fire ant venom. Almost all crazy ants that were able to excrete and groom with their own venom survived exposure to fire ant venom. However, when the orifice that releases formic acid from their abdomen was blocked, only half of the crazy ants survived exposure to fire ant venom. How the crazy ant venom blocks the activity of the fire ant venom is not known, but it does not bode well for fire ants. The crazy ants are out-competing them on the ground and are impervious to their venom. However, it should be noted that while the strategy of smothering oneself with acid after a fire ant sting may work for crazy ants, it does not work well for humans and should not be tried at home.

Noted by WVR, MD