

Objective Sleep Characteristics and Cardiometabolic Health in Young Adolescents

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abstract

BACKGROUND AND OBJECTIVES: Shorter sleep duration is associated with childhood obesity. Few studies measure sleep quantity and quality objectively or examine cardiometabolic biomarkers other than obesity.

METHODS: This cross-sectional study of 829 adolescents derived sleep duration, efficiency and moderate-to-vigorous physical activity from >5 days of wrist actigraphy recording for >10 hours/day. The main outcome was a metabolic risk score (mean of 5 sex-specific z-scores for waist circumference, systolic blood pressure, high-density lipoprotein cholesterol scaled inversely, and log-transformed triglycerides and homeostatic model assessment of insulin resistance), for which higher scores indicate greater metabolic risk. Secondary outcomes included score components and dual-energy radiograph absorptiometry fat mass. We measured socioeconomic status, race and/or ethnicity, pubertal status, and obesity-related behaviors (television-viewing and fast food and sugar-sweetened beverage consumption) using questionnaires.

RESULTS: The sample was 51.5% girls; mean (SD) age 13.2 (0.9) years, median (interquartile range) sleep duration was 441.1 (54.8) minutes per day and sleep efficiency was 84.0% (6.3). Longer sleep duration was associated with lower metabolic risk scores (−0.11 points; 95% CI: −0.19 to −0.02, per interquartile range). Associations with sleep efficiency were similar and persisted after adjustment for BMI z score and physical activity, television-viewing, and diet quality. Longer sleep duration and greater sleep efficiency were also favorably associated with waist circumference, systolic blood pressure, high-density lipoprotein cholesterol, and fat mass.

CONCLUSIONS: Longer sleep duration and higher sleep efficiency were associated with a more favorable cardiometabolic profile in early adolescence, independent of other obesity-related behaviors. These results support the need to assess the role of sleep quantity and quality interventions as strategies for improving cardiovascular risk profiles of adolescents.



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WHAT'S KNOWN ON THIS SUBJECT: Current evidence, composed primarily of cross-sectional studies with self-reported sleep duration, suggests that short sleep increases childhood obesity risk. Limited evidence, mainly in adults, links poor sleep quality, including sleep fragmentation, and increased variability in sleep duration, to cardiometabolic risk.

WHAT THIS STUDY ADDS: Longer sleep duration and higher sleep efficiency (measured by using actigraphy) are associated with a more favorable cardiometabolic profile, including lower abdominal adiposity, lower systolic blood pressure, and higher high-density lipoprotein cholesterol. Many associations were independent of obesity-related behaviors and BMI.

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Obesity and cardiovascular risk factors in childhood promote cardiovascular disease later in life.¹⁻³ Sleep has emerged as a potential target for obesity prevention: in accumulating evidence, including in our previous work, authors have observed that shorter childhood sleep duration leads to higher BMI.⁴⁻⁷ However, authors of few studies examine whether child or adolescent sleep duration is associated with other aspects of cardiometabolic risk (eg, blood pressure, blood lipids, and insulin resistance),⁸ and fewer yet examine the contribution of sleep quality (eg, efficiency) to cardiometabolic risk.^{9,10} In addition, authors of most studies used subjectively reported sleep characteristics. Studies in which authors used objective measurements (eg, polysomnography¹¹⁻¹⁷ or actigraphy¹⁸⁻²⁷) often had small sample sizes or had no control for confounders, such as socioeconomic status, or potential mediators, such as physical activity, television viewing, or diet quality.

With the current study, we fill these gaps by examining actigraphy-estimated sleep characteristics with cardiometabolic health in a large sample of children entering adolescence, a period with dramatic changes in sleep architecture and duration, high prevalence of inadequate sleep,²⁸ and emerging cardiovascular risk profiles.^{1-3,29} In this cross-sectional study, we examine associations of sleep quantity and quality with a comprehensive range of measurements of adiposity and cardiometabolic risk factors including blood pressure, lipids, and insulin resistance.³⁰

METHODS

Study Participants

Project Viva recruited pregnant women from their initial prenatal

visit at Atrius Health in eastern Massachusetts (1999–2002) and has followed mother-child pairs since. After delivery, mothers reported their child's race and/or ethnicity. Of the 2128 children enrolled, 1038 participated in the adolescent in-person visit (mean [SD] age was 13.2 [0.9] years; range: 11.9–16.6 years) and were eligible for the sleep examination. Of these, 829 provided valid actigraphy measurements. A smaller number completed blood draws and dual-energy radiograph absorptiometry (DXA) scans; sample sizes for each outcome are in Table 1. Women provided written informed consent at each visit, and children began providing verbal consent at midchildhood. All procedures were approved by the relevant institutional review boards.

Actigraphy Protocol and Sleep Exposures

We measured nighttime sleep and daytime physical activity using wrist actigraphy (Actigraph GT3X+) analyzed by using ActiLife-6 software (ActiGraph, Inc, Pensacola, FL). Actigraphs were used to collect activity data in 60-second epochs. We asked adolescents to wear the device on their nondominant wrist for 7 to 10 consecutive days and nights and complete daily sleep logs. We identified each day's main rest interval as the primary sleep period on the basis of logs and observation of a sharp decrease in activity with subsequent increase. Participants with ≥ 5 days of recording with ≥ 10 hours of wear-time were included. If the device was removed for ≥ 1 hour within the in-bed interval, then the primary sleep period was considered invalid. We applied Cole et al's³¹ algorithm to classify sleep and wake periods.

Our sleep exposures were from overnight sleep periods, averaged over all nights of valid recording and are as follows: (1) duration (sleep time in minutes) and (2)

maintenance efficiency (percentage of time between sleep onset and final awakening spent asleep). Secondary exposures included the following: (3) variability (SD of sleep duration over all valid nights) and (4) wake after sleep onset (WASO) (time awake after sleep onset in minutes).

Adiposity Outcomes

We measured children's height using calibrated stadiometers (Shorr Productions, Olney, MD) and weight with calibrated Tanita scales (model TBF-300A; Tanita Corporation of America, Inc, Arlington Heights, IL). We calculated BMI and age- and sex-specific z scores using national reference data.^{32,33} We measured total body fat and trunk fat using DXA and calculated fat mass indices (kg/m^2). We measured waist circumference (cm) using a Lefkin woven tape and subscapular and triceps skinfold thicknesses (mm) using Holtain calipers (Holtain LTD, Crosswell, UK) and calculated their sum. Trained research assistants performed all measurements following standardized techniques.³⁴

Cardiometabolic Biomarkers

We measured systolic blood pressure with a Dinamap Pro-100 (Critikon of General Electric, Inc, Tampa, FL). We obtained 5 measurements 1 minute apart and calculated mean systolic blood pressure.

A phlebotomist collected blood samples, which were stored at -70°C until they were assayed. Plasma fasting insulin was measured by using an electro-chemiluminescence immunoassay on the Roche Modular system, and fasting glucose was measured enzymatically by using Roche Diagnostics reagents.^{35,36} We calculated the homeostatic model assessment of insulin resistance (HOMA-IR) ($\text{insulin } [\mu\text{U}/\text{mL}] \times \text{glucose } [\text{mg}/\text{dL}] / 405$). Triglycerides and high-density lipoprotein (HDL) cholesterol were measured enzymatically.

TABLE 1 Parent, Household, and Child Characteristics According to Actigraphy-Estimated Sleep Duration in Early Adolescence

Characteristics	Overall, <i>n</i> = 829	<480 min per d, <i>n</i> = 692	≥480 min per d, <i>n</i> = 137
Mother and/or household characteristics, mean (SD)			
Maternal age at enrollment, y	32.3 (5.0)	32.3 (5.1)	32.6 (4.7)
Mother graduated college, <i>n</i> (%)			
No	228 (27.5)	198 (28.7)	30 (21.9)
Yes	600 (72.5)	493 (71.3)	107 (78.1)
Household income >\$70 000 per y at early teenager visit, <i>n</i> (%)			
No	177 (22.2)	154 (23.1)	23 (17.4)
Yes	622 (77.8)	513 (76.9)	109 (82.6)
Adolescent's characteristics			
Girl, <i>n</i> (%)			
No	402 (48.5)	357 (51.6)	45 (32.8)
Yes	427 (51.5)	335 (48.4)	92 (67.2)
Race and/or ethnicity, <i>n</i> (%)			
African American	131 (15.8)	117 (16.9)	14 (10.2)
Hispanic	36 (4.3)	33 (4.8)	3 (2.2)
Asian American	22 (2.7)	21 (3.0)	1 (0.7)
White	532 (64.3)	428 (61.9)	104 (75.9)
Other	107 (12.9)	92 (13.3)	15 (10.9)
Season at early adolescent visit, <i>n</i> (%)			
Winter	170 (20.5)	140 (20.2)	30 (21.9)
Spring	222 (26.8)	187 (27.0)	35 (25.5)
Summer	275 (33.2)	229 (33.1)	46 (33.6)
Fall	162 (19.5)	136 (19.7)	26 (19.0)
Tanner stage, ^a mean (SD)	3.6 (1.0)	3.6 (1.0)	3.5 (1.1)
Age, y, mean (SD)	13.2 (0.9)	13.2 (0.9)	13.1 (0.8)
Television viewing, h per d, mean (SD)	2.0 (1.4)	2.1 (1.4)	1.7 (1.1)
Fast food intake, servings per wk, mean (SD)	0.7 (1.1)	0.7 (1.1)	0.6 (1.1)
Sugar sweetened beverage intake, servings per d, mean (SD)	0.8 (0.9)	0.8 (0.9)	0.7 (0.7)
BMI z score, mean (SD)	0.36 (1.07)	0.41 (1.06)	0.11 (1.07)
Actigraphy measurements, median (IQR)			
Average daily MVPA, min	7.0 (17.0)	7.0 (16.0)	7.0 (16.5)
Average daily sleep duration, min	441.1 (54.8)	432.7 (46.0)	495.8 (21.9)
Average daily sleep efficiency, %	84.0 (6.3)	83.5 (6.1)	86.3 (4.9)
Average daily WASO, min	74.8 (36.2)	75.9 (36.3)	69.2 (33.9)
Day-to-day variability in sleep duration, SD ^b	56.0 (35.4)	55.7 (35.8)	58.5 (34.1)
Adiposity outcomes, mean (SD)			
BMI z score, <i>n</i> = 827	0.36 (1.07)	0.41 (1.06)	0.11 (1.07)
Waist circumference, cm, <i>n</i> = 829	72.8 (11.7)	73.4 (11.9)	69.7 (10.5)
Sum of skinfolds, mm, <i>n</i> = 826	28.2 (13.7)	28.6 (14.2)	26.2 (11.2)
DXA fat mass index, kg/m ² , <i>n</i> = 619	6.3 (3.1)	6.4 (3.2)	5.9 (2.3)
DXA trunk fat mass index, kg/m ² , <i>n</i> = 619	2.4 (1.5)	2.4 (1.5)	2.2 (1.1)
Cardiometabolic outcomes, mean (SD)			
Metabolic risk, z score, ^c <i>n</i> = 493	−0.02 (0.60)	0.00 (0.61)	−0.12 (0.56)
HOMA-IR, <i>n</i> = 496	3.1 (1.9)	3.2 (2.0)	2.8 (1.4)
Insulin, μU/mL, <i>n</i> = 560	15.9 (14.9)	16.4 (15.9)	12.9 (6.1)
Glucose, mg/dL, <i>n</i> = 513	92.7 (23.5)	93.0 (25.1)	91.1 (11.5)
Systolic BP, mm Hg, <i>n</i> = 823	107.2 (8.9)	107.7 (8.9)	104.9 (9.0)
Triglycerides, mg/dL, <i>n</i> = 559	69.8 (31.3)	69.1 (31.3)	73.4 (31.3)
HDL cholesterol, mg/dL, <i>n</i> = 560	55.3 (13.1)	55.1 (13.2)	56.9 (12.5)

Data from 829 participants from Project Viva. BP, blood pressure.

^a Tanner stage pubic hair (5-point scale).

^b SD of daily time spent asleep (all days), minutes.

^c Mean of 5 sex- and cohort-specific z scores for waist circumference, systolic blood pressure, HDL cholesterol scaled inversely, and log-transformed triglycerides and HOMA-IR; higher scores are indicative of greater metabolic risk.

We derived a metabolic risk score, described previously,³⁷ as the mean of sex- and cohort-specific z scores for waist circumference, systolic blood pressure, HDL cholesterol (scaled inversely), log-transformed

HOMA-IR, and log-transformed triglycerides. Higher scores were indicative of greater metabolic risk. Although there is no consistent definition of the metabolic syndrome in children, researchers of previous

work have used similar scores.^{38–40}

Other Measures

We derived moderate-to-vigorous physical activity (MVPA) from

actigraphy. From total activity (vertical axis counts per minutes) excluding sleep and non-wear-time, we applied Chandler's cut points to derive average MVPA minutes per day.⁴¹ We defined the season of measurement as "Spring" being from March 1 to May 31, "Summer" from June 1 to August 31, "Autumn" from September 1 to November 30, or "Winter" from December 1 to February 28. Via questionnaire, mothers reported their educational attainment and household incomes, and adolescents reported their pubertal status (Tanner stage was derived from a validated 5-point rating scale on pubic hair growth, with higher values indicative of greater pubertal development),⁴² the number of hours per day on weekdays and weekends spent watching television, and the servings of fast food and sugar-sweetened beverages consumed per day.

Statistical Analysis

We performed all analyses using SAS version 9.4 (SAS Institute, Inc, Cary, NC). We examined correlations among the sleep exposures and the normality of biomarker measurements. For comparability, we reported associations per interquartile range (IQR) of sleep exposures. Our multivariable linear regression models adjusted for confounding variables selected a priori from previous literature; all models included demographics (adolescents' age, sex, and race and/or ethnicity), socioeconomic status (mothers' education and household income), pubertal status, and season.

To examine the associations of sleep exposures independently of other obesity-related behaviors, we additionally adjusted models for MVPA, television-viewing, and fast food and sugar-sweetened beverages, which authors of previous studies suggest mediate and/or confound the sleep-cardiometabolic risk relationship.^{9,10,43-46} Similarly, to

examine associations independently of overall adiposity, we adjusted blood biomarker outcomes for BMI z score.

To explore whether associations of sleep duration and efficiency were independent, we entered these exposures (Spearman correlation = 0.41) into the same model and reported mutually adjusted results.

We evaluated interactions of sex with sleep exposures through stratified analyses and product terms in multivariable models.

RESULTS

Mean (SD) age was 13.2 (0.9) years, and 51.5% of the sample were girls (Table 1). Median (IQR) sleep duration was 441.1 (54.8) minutes per day, sleep efficiency percentage was 84.0% (6.3), WASO was 74.8 (36.2) minutes, and variability was 56.0 (35.4) minutes. Using actigraphy-estimated sleep duration, a minority ($n = 18$; 2.2%) of adolescents met the lower bound of the National Sleep Foundation's recommended sleep duration (>480 minutes [8 hours] per day for participants 14-17 years old and >540 minutes [9 hours] per day for participants 11-13 years old).⁴⁷ Short sleep (<420 minutes per day) occurred in 31% of the sample ($n = 257$). A majority were classified as having low sleep efficiency by using the threshold $\leq 85\%$ ($n = 484$; 58.4%). Sleep characteristics are reported separately for weekdays and weekend recording in Supplemental Table 3.

Adiposity

Each increment of sleep duration (55 minutes per day) was inversely associated with adiposity, independent of the adjustment for sociodemographics, puberty, and season (Fig 1), including BMI z score (-0.15 per IQR; 95% confidence interval [CI]: -0.26 to -0.05), sum of skinfold thicknesses (-2.05 mm;

95% CI: -3.43 to -0.68), DXA fat mass index (-0.52 kg/m²; 95% CI: -0.89 to -0.15), and DXA trunk fat mass index (-0.26 kg/m²; 95% CI: -0.43 to -0.08).

Likewise, sleep efficiency (IQR: 6%) was inversely associated with adiposity measured by DXA (Fig 1), including total fat mass index (-0.37 kg/m²; 95% CI: -0.71 to -0.03) and trunk fat mass index (-0.21 kg/m²; 95% CI: -0.37 to -0.05).

Sleep variability (IQR: 35 minutes) and WASO (IQR: 36 minutes) had no association with any adiposity measures after multivariable adjustment (Table 2).

Most associations persisted after adjusting for MVPA, television-viewing, and sugar-sweetened beverage and fast food consumption (Table 2, Model 2).

Metabolic Risk Score, Blood Pressure, and Blood Lipids

Longer sleep duration and higher sleep efficiency were associated with lower metabolic risk scores (Fig 2A; -0.11 points per IQR; 95% CI: -0.19 to -0.02 and -0.08 points; 95% CI: -0.16 to -0.01, respectively). Examining the score's individual components, we found these results were driven by a smaller waist circumference (Fig 1E; -2.81 cm; 95% CI: -3.96 to -1.65 and -1.69 cm; 95% CI: -2.78 to -0.61, respectively), lower systolic blood pressure (Fig 2B; -1.36 mm Hg; 95% CI: -2.24 to -0.47 and -1.44 mm Hg; 95% CI: -2.26 to -0.61, respectively), and higher HDL cholesterol (Fig 2C; 1.99 mg/dL; 95% CI: 0.30 to 3.67 and 1.49 mg/dL; 95% CI: -0.04 to 3.02). Associations with the triglyceride and HOMA-IR components were in the expected direction, but CIs were wide (Figs 2D and 2E). Associations of both sleep duration and efficiency with systolic blood pressure and of efficiency with the metabolic risk score and HDL cholesterol persisted with additional adjustment for BMI z score and

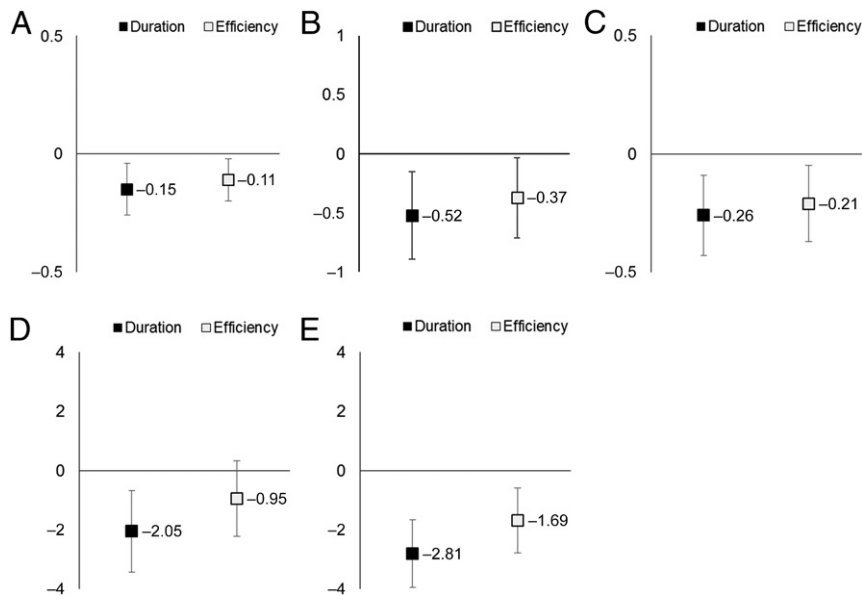


FIGURE 1 Multivariable-adjusted associations of sleep duration and efficiency (per interquartile increase) with adiposity outcomes. Results for adiposity outcomes are shown per IQR of the following sleep exposures: duration (55 minutes) and efficiency (6%). Models adjust for maternal education and household income, season of measurement, and adolescent age, sex, race and/or ethnicity, and puberty (Tanner stage). A, BMI z score. B, DXA total fat mass index (in kilograms per meter squared). C, DXA trunk fat index (in kilograms per meter squared). D, Sum of subscapular and triceps skinfolds (millimeters). E, Waist circumference (in centimeters).

MVPA, television-viewing, and diet quality (Table 2).

Sex Differences

The associations of sleep efficiency and WASO with triglycerides and variability with the adiposity measures varied by sex in that there was a robust association in girls that was not found in boys (*P*-interactions < .05; Supplemental Table 4).

Otherwise, associations did not vary by sex.

Sleep Quantity Versus Quality

When sleep duration and efficiency were in the same model, each was independently associated with systolic blood pressure. Associations of sleep duration with adiposity (BMI z score and waist circumference) and the metabolic risk score were robust to adjustment for sleep efficiency, whereas associations of sleep efficiency with adiposity and the metabolic risk score attenuated toward the null once they were adjusted for sleep duration

(Supplemental Table 5). Associations of WASO and variability with most outcomes remained null regardless of adjustment for sleep duration.

DISCUSSION

This study is 1 of the largest and most comprehensive to date, in which we assess multiple sleep characteristics and adiposity and cardiometabolic outcomes in adolescents. Inadequate sleep as estimated by actigraphy was highly prevalent (31% of adolescents slept <420 minutes, and 42% had sleep efficiencies <85%). Although few children met the National Sleep Foundation–recommended sleep duration, self- or parent-reported sleep duration overestimates sleep compared with actigraphy⁴⁸; additional research is needed to establish normative data by using objective sleep assessments. Adolescents with longer sleep duration and higher sleep efficiency had a more favorable cardiometabolic profile, including

components of the metabolic syndrome, namely central adiposity, systolic blood pressure, and HDL cholesterol. Greater sleep efficiency was associated with lower metabolic risk scores independent both of other obesity-related behaviors and of BMI z score. These findings are clinically important in that each of the outcomes examined influences not only current cardiometabolic health but also future cardiovascular risk.

Adiposity

Moving beyond the existing literature on sleep duration and BMI,^{21,23,49–64} we found that both shorter sleep duration and lower efficiency were associated with central adiposity (waist circumference and DXA trunk fat), which strongly predict risk of diabetes and other metabolic derangements.⁶⁵ A limited number of actigraphy studies are used to examine sleep duration and abdominal adiposity in children or adolescents^{22,23,27,61,66}; even fewer are used to examine sleep efficiency or DXA trunk fat.^{12,19,66,67} Adding new rigor to this evidence, we demonstrate that associations of inadequate sleep with abdominal adiposity are independent of confounders (socioeconomic status) and other obesity-related behaviors.

Metabolic Risk

Sleep duration and efficiency were associated with our aggregate measure of cardiometabolic risk, the metabolic risk score. Although few studies are directly comparable,⁶⁶ in adults, both short and extended duration of sleep have longitudinal associations with metabolic risks independent of BMI, including diabetes,⁶⁸ hypertension,⁶⁹ and hyperlipidemia.^{70,71} Meanwhile, with our previous longitudinal work in younger children, we found that the association between parent-reported sleep duration and midchildhood metabolic risk was mediated through midchildhood adiposity (mean age

TABLE 2 Multivariable-Adjusted Associations of Sleep Measures With Adiposity and Cardiometabolic Outcomes

Outcome	Model 1, β (95% CI)	Model 2, β (95% CI)	Model 3, β (95% CI)
Sleep duration (per 55 min)			
BMI z score	-0.23 (-0.33, -0.13)	-0.13 (-0.24, -0.03)	—
Waist circumference, cm	-3.06 (-4.12, -2.00)	-2.66 (-3.84, -1.49)	—
Sum of skinfolds, mm	-2.53 (-3.80, -1.26)	-1.85 (-3.22, -0.48)	—
DXA fat mass index, kg/m ²	-0.67 (-1.01, -0.34)	-0.49 (-0.86, -0.11)	—
DXA trunk fat index, kg/m ²	-0.33 (-0.49, -0.17)	-0.24 (-0.42, -0.05)	—
Metabolic risk score	-0.11 (-0.18, -0.03)	-0.08 (-0.17, 0.00)	-0.06 (-0.13, 0.01)
HOMA-IR	-0.34 (-0.58, -0.10)	-0.16 (-0.44, 0.11)	-0.12 (-0.38, 0.14)
Insulin, μ U/mL	-2.69 (-4.38, -1.01)	-0.81 (-2.76, 1.14)	-0.93 (-2.78, 0.93)
Glucose, mg/dL	-0.90 (-3.76, 1.97)	0.09 (-3.51, 3.70)	0.37 (-3.22, 3.97)
Systolic blood pressure, mm Hg	-1.73 (-2.53, -0.93)	-1.49 (-2.40, -0.57)	-1.42 (-2.34, -0.51)
Triglycerides, mg/dL	0.73 (-2.88, 4.34)	0.68 (-3.50, 4.85)	1.21 (-2.92, 5.34)
HDL cholesterol, mg/dL	1.66 (0.16, 3.15)	1.63 (-0.12, 3.37)	1.44 (-0.25, 3.14)
Sleep efficiency (per 6%)			
BMI z score	-0.05 (-0.15, 0.04)	-0.10 (-0.20, 0.00)	—
Waist circumference, cm	-1.16 (-2.18, -0.14)	-1.65 (-2.76, -0.55)	—
Sum of skinfolds, mm	-0.57 (-1.78, 0.63)	-0.72 (-1.99, 0.56)	—
DXA fat mass index, kg/m ²	-0.17 (-0.50, 0.15)	-0.30 (-0.65, 0.04)	—
DXA trunk fat index, kg/m ²	-0.11 (-0.26, 0.05)	-0.18 (-0.34, -0.01)	—
Metabolic risk score	-0.07 (-0.14, 0.00)	-0.09 (-0.17, -0.01)	-0.08 (-0.14, -0.02)
HOMA-IR	-0.08 (-0.31, 0.14)	-0.11 (-0.37, 0.14)	-0.10 (-0.34, 0.14)
Insulin, μ U/mL	-1.80 (-3.35, -0.24)	-0.65 (-2.42, 1.13)	-0.62 (-2.31, 1.07)
Glucose, mg/dL	-2.71 (-5.38, -0.05)	-3.91 (-7.24, -0.58)	-3.80 (-7.12, -0.49)
Systolic blood pressure, mm Hg	-1.17 (-1.93, -0.41)	-1.53 (-2.39, -0.68)	-1.46 (-2.32, -0.61)
Triglycerides, mg/dL	-2.68 (-5.99, 0.62)	-1.16 (-4.98, 2.65)	-0.84 (-4.60, 2.92)
HDL cholesterol, mg/dL	1.47 (0.10, 2.84)	1.65 (0.05, 3.24)	1.46 (-0.08, 3.00)
WASO (per 36 min)			
BMI z score	0.00 (-0.10, 0.09)	0.07 (-0.03, 0.16)	—
Waist circumference, cm	0.44 (-0.58, 1.46)	1.03 (-0.07, 2.13)	—
Sum of skinfolds, mm	-0.08 (-1.28, 1.12)	0.18 (-1.08, 1.45)	—
DXA fat mass index, kg/m ²	0.02 (-0.30, 0.33)	0.19 (-0.15, 0.53)	—
DXA trunk fat index, kg/m ²	0.03 (-0.12, 0.18)	0.12 (-0.04, 0.29)	—
Metabolic risk score	0.04 (-0.03, 0.11)	0.06 (-0.02, 0.13)	0.06 (-0.01, 0.12)
HOMA-IR	-0.02 (-0.23, 0.20)	0.04 (-0.20, 0.29)	0.04 (-0.19, 0.28)
Insulin, μ U/mL	1.20 (-0.34, 2.73)	0.51 (-1.24, 2.26)	0.52 (-1.15, 2.18)
Glucose, mg/dL	1.51 (-1.08, 4.10)	2.72 (-0.52, 5.97)	2.70 (-0.52, 5.93)
Systolic blood pressure, mm Hg	0.74 (-0.02, 1.50)	1.13 (0.27, 1.98)	1.08 (0.23, 1.93)
Triglycerides, mg/dL	2.87 (-0.39, 6.12)	1.15 (-2.60, 4.90)	0.97 (-2.73, 4.67)
HDL cholesterol, mg/dL	-1.13 (-2.48, 0.22)	-1.30 (-2.87, 0.27)	-1.16 (-2.68, 0.36)
Sleep duration variability (per 35 min)			
BMI z score	0.12 (0.03, 0.22)	-0.01 (-0.11, 0.09)	—
Waist circumference, cm	0.90 (-0.13, 1.93)	-0.18 (-1.31, 0.95)	—
Sum of skinfolds, mm	2.06 (0.84, 3.27)	0.79 (-0.51, 2.09)	—
DXA fat mass index, kg/m ²	0.33 (0.01, 0.65)	0.05 (-0.30, 0.41)	—
DXA trunk fat index, kg/m ²	0.15 (0.00, 0.30)	0.03 (-0.15, 0.20)	—
Metabolic risk score	0.03 (-0.04, 0.09)	-0.02 (-0.09, 0.06)	-0.01 (-0.08, 0.05)
HOMA-IR	0.22 (0.00, 0.43)	-0.02 (-0.26, 0.23)	0.00 (-0.23, 0.24)
Insulin, μ U/mL	0.89 (-0.67, 2.45)	-0.19 (-1.93, 1.55)	-0.23 (-1.88, 1.43)
Glucose, mg/dL	3.03 (0.45, 5.60)	2.60 (-0.62, 5.82)	2.65 (-0.55, 5.86)
Systolic blood pressure, mm Hg	0.08 (-0.70, 0.85)	0.07 (-0.82, 0.95)	0.08 (-0.80, 0.95)
Triglycerides, mg/dL	-1.18 (-4.50, 2.13)	-2.04 (-5.76, 1.69)	-2.24 (-5.92, 1.44)
HDL cholesterol, mg/dL	0.61 (-0.77, 1.99)	1.36 (-0.20, 2.92)	1.40 (-0.11, 2.91)

Model 1 was adjusted for age and sex. Model 2 comprised model 1 and maternal education and household income, season of measurement, adolescent race and/or ethnicity, puberty (Tanner stage), and obesity-related behaviors (MVPA, television-viewing hours per day, and fast food and sugar-sweetened beverage servings per day). Model 3 comprised model 2 and BMI z score (for cardiometabolic biomarkers only). —, not applicable.

was 7 years).³⁷ Here, we examine the same cohort as they enter adolescence (mean age was 13 years) and estimate sleep duration and efficiency using actigraphy. As before, the association

of sleep duration with the metabolic risk score is dependent on BMI z score; however, sleep efficiency was associated with metabolic risk independent of BMI z score,

suggesting that adolescent sleep quality, as measured by increased waking during the sleep period, contributes to cardiometabolic health through pathways other than BMI.

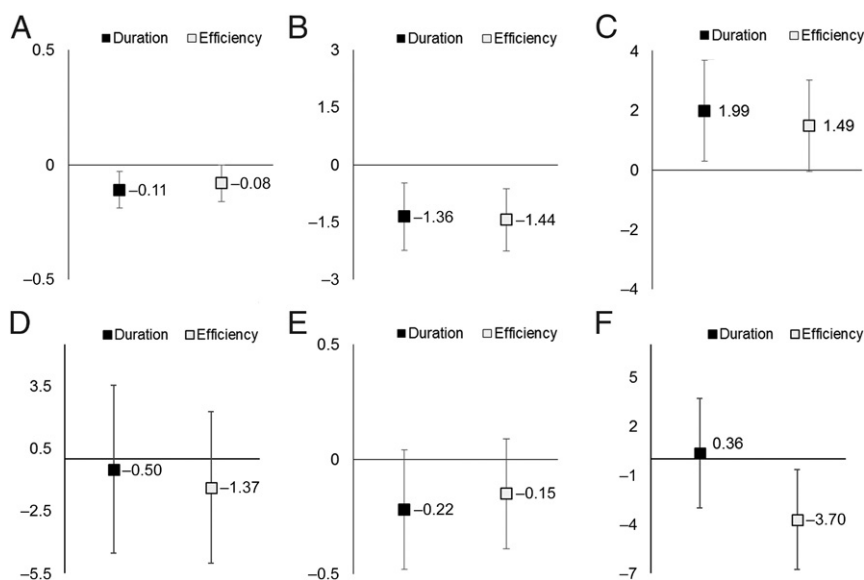


FIGURE 2 Multivariable-adjusted associations of sleep duration and efficiency (per interquartile increase) with cardiometabolic biomarker outcomes. Results for cardiometabolic outcomes are shown per IQR of the following sleep exposures: duration (55 minutes) and efficiency (6%). Models adjust for maternal education and household income, season of measurement, and adolescent age, sex, race and/or ethnicity, and puberty (Tanner stage). A, Metabolic risk score (points). B, Systolic blood pressure (in millimeters of mercury). C, HDL cholesterol (in milligrams per deciliter). D, Triglycerides (in milligrams per deciliter). E, HOMA-IR. F, Glucose (in milligrams per deciliter).

Blood Pressure and Blood Lipids

We observed associations of sleep duration and efficiency with higher systolic blood pressure independent of BMI z score and obesity-related behaviors. Although the literature on sleep and blood pressure is sparse among children and adolescents,^{20,24,25,72} authors of the few existing studies in which sleep is objectively measured confirm our findings: authors of an early study of this topic found that low efficiency and short duration were both associated with higher odds of prehypertension among adolescents.¹⁷ With respect to blood lipids, we found that longer sleep was associated with higher HDL cholesterol in all subjects and with lower triglycerides in girls only. Although authors of some studies in which objectively measured sleep was researched found inverse associations of sleep duration with triglycerides,^{27,73} most authors have not observed associations with HDL cholesterol.^{66,73} This

makes our finding that greater sleep efficiency is associated with higher HDL cholesterol independent of other obesity-related behaviors of particular interest.

Glucose Homeostasis

The associations of sleep duration with glucose homeostasis are understudied and inconsistent among children and adolescents, perhaps because of the challenge of obtaining fasting blood samples.^{22,66} Although we found no statistically significant associations with HOMA-IR or insulin, we found that higher sleep efficiency was associated with lower glucose. Authors of other actigraphy studies reported an inverse association between sleep duration and HOMA-IR,^{22,66} or a U-shaped relationship,²¹ depending on the participant age and study design. For example, authors of a Danish study found no cross-sectional association of HOMA-IR, but they found that longitudinally positive changes in sleep duration were associated with beneficial changes in HOMA-IR.⁶⁶

Sex Differences

We found associations of sleep efficiency and WASO with triglycerides and variability with adiposity only among girls; others have also found effect modification by sex when examining sleep characteristics. For example, authors of the Cleveland Study found that the association between short sleep and dietary fat intake was stronger in girls,⁷⁴ whereas its association with BMI and glucose homeostasis was stronger in boys.²² Authors of adult experimental studies also report that female subjects have a greater inflammatory and immune response to sleep restriction than male subjects.⁷⁵ Sex may modify the physiologic response to sleep disturbance. The complex interrelationship of sex hormones, sleep, and obesity-related behaviors in adolescence warrants further investigation.

Sleep Quantity Versus Sleep Quality

The associations of sleep duration and efficiency with systolic blood pressure were robust to mutual adjustment, were used to underscore that both quantity and quality or continuity of sleep are both important to cardiometabolic health in adolescence. Typically, sleep duration and efficiency are examined in separate models; authors of few studies reported independent associations.²⁰ For example, in Danish children, sleep efficiency was associated with waist circumference after adjustment for sleep duration,⁶⁶ and others have found that sleep efficiency²³ and variability,¹⁹ but not duration, are associated with greater adiposity. This runs counter to our finding that although duration was associated with BMI z score, and waist circumference was independent of efficiency, associations of efficiency with adiposity outcomes attenuated after adjusting for duration.

Proposed Mechanisms

Several proposed mechanisms can be used to explain the association between sleep duration and quality and cardiometabolic health,^{76–78} including physiologic and behavioral changes that impact energy intake and expenditure. In adults^{79–88} and, to a limited extent, in children⁸⁹ and adolescents,^{90,91} authors of experimental research suggest that inadequate sleep duration leads to increased energy intake, altered food choices, changes in the brain's response to reward stimuli,⁷⁷ lowered leptin, and increased ghrelin.⁸¹ Inadequate quantity and quality of sleep have also been associated with decreased physical activity, increased screen time,^{92,93} decreased fat oxidation,⁷⁷ and abnormalities in the autonomic nervous system and hypothalamus-pituitary-adrenocortical axis.^{77,78} These, in turn, influence the metabolic outcomes examined in this study including abdominal adiposity, insulin resistance, dyslipidemia, and elevated blood pressure. Although this web of bidirectional relationships cannot be disentangled in a cross-sectional study, it is striking that many of the associations were independent both of BMI z score and of obesity-related behaviors including MVPA, television-viewing, and fast food and sugar-sweetened beverage intake.

Clinical Implications

Although causality cannot be determined from cross-sectional data, pediatricians should be aware that poor sleep quality (eg, frequent awakenings) and not just insufficient duration of sleep is associated with increased cardiometabolic risk. Optimizing children's health should include strategies used to address

child and adolescent sleep, including duration and efficiency, and to screen for sleep problems and disorders. Although greater clarity is needed on the independent effects of sleep quantity and quality, with our findings, we suggest that both sleep duration and efficiency are associated with cardiometabolic health in early adolescence. Intervention trials have been used to attempt to extend sleep duration, but few have been used to target sleep efficiency or other aspects of sleep quality. In adults, exercise increases sleep efficiency,^{94,95} and in children, television-viewing and other forms of screen time reduce sleep efficiency. Preventive interventions to improve sleep health should be used to target behavioral and environmental factors that influence sleep continuity as well as duration, such as screen time,⁹⁶ stress, noise, caffeine, and exercise.⁹⁵ Such multimodal interventions are likely to improve multiple aspects of sleep.⁹⁷

Strengths and Limitations

Among its strengths, this study was used to objectively measure sleep quantity as well as sleep quality in relation to a composite metabolic risk score and its components. The use of data from a well-established cohort of adolescents managed since birth allowed us to address the limitations of previous studies through control for sociodemographic confounders and obesity-related behaviors. However, our study was cross-sectional, a key limitation that complicates its interpretation; longitudinal studies with repeated, objective measures are needed to establish the temporal order of inadequate sleep and metabolic risk. In addition, although actigraphy is a practical method used to measure sleep over multiple days with minimal participant burden,⁹⁸

actigraphy differs from the gold standard (polysomnography),⁴⁸ and there are important exposures (eg, sleep stage distribution) not captured by actigraphy.⁷⁸

CONCLUSIONS

Sleep quantity and quality are associated with a more favorable cardiometabolic profile in early adolescence. Although associations vary by sleep parameter or metabolic marker examined, they are strongest and most consistent for sleep duration and efficiency in relationship to central adiposity and blood pressure. BMI and obesity-related behaviors cannot be used to account fully for the associations, which suggests there may be additional physiologic pathways such as abnormalities in hypothalamic-pituitary-adrenal axis and the autonomic nervous system that account for sleep's influence on cardiometabolic health in early adolescence. Sleep quantity and quality are pillars of health alongside diet and physical activity, and authors of future studies should examine intervention strategies to improve sleep quality as well as quantity in adolescents.

ABBREVIATIONS

CI: confidence interval
DXA: dual-energy radiograph absorptiometry
HDL: high-density lipoprotein
HOMA-IR: homeostatic model assessment of insulin resistance
IQR: interquartile range
MVPA: moderate-to-vigorous physical activity
WASO: wake after sleep onset

all analyses; and all authors approved, revised, and critically reviewed the manuscript for important intellectual content and the final manuscript as submitted and agree to be accountable for all aspects of the work.

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Objective Sleep Characteristics and Cardiometabolic Health in Young Adolescents

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