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Objective Sleep Characteristics and Cardiometabolic Health in Young Adolescents

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Abstract

BACKGROUND And Objective: 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, HDL-cholesterol scaled inversely, and log-transformed triglycerides and HOMA-IR), for which higher scores indicate greater metabolic risk. Secondary outcomes included score components and DXA fat mass. We measured socioeconomic status, race/ethnicity, pubertal status and obesity-related behaviors (television-viewing and fast food and sugar-sweetened beverage consumption) using questionnaires.

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CONTRIBUTORS' STATEMENT.

Drs. Cespedes Feliciano conceptualized and designed the study and wrote the initial draft of the manuscript and subsequent revisions.

Dr. Quante conceptualized and designed the study.

Drs. Taveras and Redline obtained funding for the study and conceptualized and designed the study.

Dr. Oken obtained funding for the study and oversaw data collection.

Ms. Rifas-Shiman conducted all analyses.

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

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Results: The sample was 51.5% female; mean (SD) age 13.2 (0.9) years, median (IQR) sleep duration was 441.1 (54.8) minutes/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, −0.02, per inter-quartile 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, HDL-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.

Table of Contents Summary:

Using objective measurements, this study found that longer sleep duration and greater sleep efficiency are associated with more favorable cardiometabolic profiles in early adolescence.

BACKGROUND

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: accumulating evidence, including our prior work, has observed that shorter childhood sleep duration leads to higher body mass index (BMI).[4–7] However, few studies examine whether child or adolescent sleep duration is associated with other aspects of cardiometabolic risk (e.g., blood pressure, blood lipids, and insulin resistance),[8] and fewer yet examine the contribution of sleep quality (e.g., efficiency) to cardiometabolic risk.[9,10] Further, most studies used subjectively-reported sleep characteristics. Those with objective measurements (e.g., polysomnography[11–17] or actigraphy[18–27]) often had small sample sizes or could not control for confounders such as socioeconomic status or potential mediators such as physical activity, television viewing, or diet quality.

The present study fills 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/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 to 16.6 years) and were eligible for the sleep exam. Of these, 829 provided valid actigraphy measurements. A smaller number completed blood draws and 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 mid-childhood. All procedures were approved by the relevant institutional review boards.

Actigraphy Protocol & Sleep Exposures

We measured nighttime sleep and daytime physical activity using wrist actigraphy (Actigraph™ GT3X+) analyzed using ActiLife-6® software (ActiGraph, Inc., Pensacola, FL). Actigraphs collected activity data in 60-second epochs. We asked adolescents to wear the device on their non-dominant wrist for 7–10 consecutive days and nights and complete daily sleep logs. We identified each day's main rest interval as the primary sleep period based on 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-Kripke's algorithm to classify sleep and wake periods.[31]

Our sleep exposures were from overnight sleep periods, averaged over all nights of valid recording: (1) duration (sleep time in minutes) and (2) maintenance efficiency (percentage of time between sleep onset and final awakening spent asleep). Secondary exposures included: (3) variability (standard deviation 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 in kilograms per meters squared (kg/m^2) and age- and sex-specific *z*-scores using national reference data.[32,33] We measured total body fat and trunk fat using dual-energy x-ray absorptiometry (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 (General Electric/Critikon, 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 using an electro-chemiluminescence immunoassay on the Roche Modular system and fasting glucose was measured enzymatically using Roche Diagnostics reagents.[35,36] We calculated the Homeostatic Model

Assessment of Insulin Resistance (HOMA-IR, insulin [μU/mL] × glucose [mg/dL]/405). Triglycerides and high-density lipoprotein (HDL) cholesterol were measured enzymatically.

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 indicate greater metabolic risk. While there is no consistent definition of the metabolic syndrome in children, prior research has utilized similar scores.[38–40]

Other Measures

We derived moderate-to-vigorous physical activity (MVPA) from actigraphy. From total activity (vertical axis counts/min) excluding sleep and non-wear time, we applied Chandler's cut-points to derive average MVPA minutes/day.[41] We defined season of measurement as "Spring" March 1st-May 31th; "Summer" June 1st-August 31st, "Autumn" September 1st-November 30th, or "Winter" December 1st-February 28th. Via questionnaire, mothers reported their educational attainment and household incomes and adolescents reported their pubertal status (Tanner stage derived from a validated 5-point rating scale on pubic hair growth, with higher values indicating greater pubertal development),[42] the number of hours/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, Cary, NC). We examined correlations among the sleep exposures, and the normality of biomarker measurements. For comparability, we reported associations per inter-quartile range (IQR) of sleep exposures. Our multivariable linear regression models adjusted for confounding variables selected *a priori* from prior literature; all models included demographics (adolescents' age, sex, and race/ethnicity), socioeconomic status (mothers' education and household income), pubertal status, and season.

To examine the associations of sleep exposures independent of other obesity-related behaviors, we additionally adjusted models for MVPA, television-viewing, and fast food and sugar-sweetened beverages, which prior studies suggest mediate and/or confound the sleep-cardiometabolic risk relationship.[9,10,43–46] Similarly, to examine associations independent 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 participants were female (Table 1). Median (IQR) sleep duration was 441.1 (54.8) minutes/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 (NSF)'s recommended sleep duration (>480 minutes [8 hours]/day for 14–17-year olds and >540 minutes [9 hours]/day for 11–13-year olds).[47] Very short sleep (<420 minutes/day) occurred in 31% of the sample (n=257). A majority were classified as having low sleep efficiency using the threshold ≤85% (n=484, 58.4%). Sleep characteristics are reported separately for weekdays and weekend recording in eTable 1.

Adiposity

Each increment of sleep duration (55 minutes/day) was inversely associated with adiposity, independent of adjustment for sociodemographics, puberty and season (Figure 1), including BMI z-score (−0.15 per IQR; 95% CI: −0.26, −0.05), sum of skinfold thicknesses (−2.05 millimeters; 95% CI: −3.43, −0.68), DXA fat mass index (−0.52 kg/m^2; 95% CI: −0.89, −0.15), and DXA trunk fat mass index (−0.26 kg/m^2; 95% CI: −0.43, −0.08).

Likewise, sleep efficiency (IQR: 6%) was inversely associated with adiposity measured by DXA (Figure 1), including total fat mass index (−0.37 kg/m^2; 95% CI: −0.71, −0.03), and trunk fat mass index (−0.21 kg/m^2; 95% CI: −0.37, −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 (Figure 2A): −0.11 points/IQR; 95% CI: −0.19, −0.02, and −0.08 points; 95% CI: −0.16, −0.01, respectively. Examining the score's individual components, this finding was driven by smaller waist circumference (Figure 1E: −2.81 cm; 95% CI: −3.96, −1.65 and −1.69 cm; 95% CI: −2.78, −0.61, respectively), lower systolic blood pressure (Figure 2B: −1.36 mmHg; 95% CI: −2.24, −0.47 and −1.44 mmHg; 95% CI: −2.26, −0.61, respectively), and higher HDL-cholesterol (Figure 2C: 1.99 mg/dL; 95% CI: 0.30, 3.67 and 1.49 mg/dL; 95% CI: −0.04, 3.02). Associations with the triglyceride and HOMA-IR components were in the expected direction but confidence intervals were wide (Figure 2D–E). 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 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: there was a robust association in girls that was not found in boys (p-interactions<0.05, eTable 2). 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 towards the null once adjusted for sleep duration (eTable 3). Associations of WASO and variability with most outcomes remained null regardless of adjustment for sleep duration.

DISCUSSION

This study is one of the largest and most comprehensive to date, assessing 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%. While few children met the NSF-recommended sleep duration, self or parent-reported sleep duration over-estimates sleep compared to actigraphy;[48] additional research is needed to establish normative data 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: 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,[49–66] we found that both shorter sleep duration and lower efficiency were associated with central adiposity (waist circumference and DXA trunk fat), which strongly predicts risk of diabetes and other metabolic derangements.[67] A limited number of actigraphy studies examined sleep duration and abdominal adiposity in children or adolescents;[22,23,27,68,69] even fewer examined sleep efficiency or DXA trunk fat.[12,19,68,70] 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. While few studies are directly comparable,[68] in adults, both short and extended duration of sleep have longitudinal associations with metabolic risks independent of BMI, including diabetes,[71] hypertension[72] and

hyperlipidemia.[73,74] Meanwhile, our prior longitudinal work in younger children found that the association between parent-reported sleep duration and mid-childhood metabolic risk was mediated through mid-childhood adiposity (mean age 7 years).[37] Here, we examine the same cohort as they enter adolescence (mean age 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.

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. While the literature on sleep and blood pressure is sparse among children and adolescents,[20,24,25,75] the few existing studies that objectively-measured sleep confirm our findings: an early study of this topic found that low efficiency and short duration were both associated with higher odds of pre-hypertension 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. Though some studies with objectively-measured sleep found inverse associations of sleep duration with triglycerides,[27,76] most have not observed associations with HDL-cholesterol.[68,76] 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 under-studied and inconsistent among children and adolescents, perhaps due to the challenge of obtaining fasting blood samples.[22,68] While we found no statistically-significant associations with HOMA-IR or insulin, higher sleep efficiency was associated with lower glucose. Other actigraphy studies reported an inverse association of sleep duration and HOMA-IR,[22,68] or a U-shaped relationship,[21] depending on participant age and study design: for example, a Danish study found no cross-sectional association of HOMA-IR, but longitudinally positive changes in sleep duration were associated with beneficial changes in HOMA-IR.[68]

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, the Cleveland Study found that short sleep's association with dietary fat intake was stronger in girls,[77] while its association with BMI and glucose homeostasis was stronger in boys.[22] Adult experimental studies also report that females have a greater inflammatory and immune response to sleep restriction than males.[78] Sex may modify the physiological response to sleep disturbance. The complex inter-relationship 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, underscoring that both quantity and quality/continuity of sleep are both important to cardiometabolic health in adolescence. Typically, sleep duration and efficiency are examined in separate models; very few studies reported independent associations.[20] For example, in Danish children, sleep efficiency was associated with waist circumference after adjustment for sleep duration,[23] and others have found that sleep efficiency[23] and variability, [19] but not duration, are associated with greater adiposity. This runs counter to our finding that while 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 explain the association between sleep duration and quality and cardiometabolic health[79–81] including physiologic and behavioral changes that impact energy intake and expenditure. In adults,[82–91] and, to a limited extent, in children[92] and adolescents,[93,94] experimental research suggests that inadequate sleep duration leads to increased energy intake, altered food choices, changes in the brain's response to reward stimuli,[80] lowered leptin and increased ghrelin.[84] Inadequate quantity and quality of sleep have also been associated with decreased physical activity, increased screen time,[95,96] decreased fat oxidation,[80] and abnormalities in the autonomic nervous system and hypothalamus-pituitary-adrenocortical axis.[80,81] These, in turn, influence the metabolic outcomes examined in this study including abdominal adiposity, insulin resistance, dyslipidemia, and elevated blood pressure. While this web of bi-directional 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

Though causality cannot be determined from cross-sectional data, pediatricians should be aware that poor sleep quality, e.g. frequent awakenings – not just insufficient duration of sleep – is associated with increased cardiometabolic risk. Optimizing children's health should include strategies for addressing child and adolescent sleep, including duration and efficiency, and screening for sleep problems and disorders. While greater clarity is needed on the independent effects of sleep quantity and quality, our findings suggest that both sleep duration and efficiency associate with cardiometabolic health in early adolescence. Intervention trials have attempted to extend sleep duration, but very few target sleep efficiency or other aspects of sleep quality. In adults, exercise increases sleep efficiency,[97,98] and in children television-viewing and other forms of screen time reduce sleep efficiency. Preventive interventions to improve sleep health should target behavioral and environmental factors that influence sleep continuity as well as duration, such as screen time,[99] stress, noise, caffeine and exercise.[98] Such multimodal interventions are likely to improve multiple aspects of sleep.[100]

Strengths and Limitations

Among its strengths, this study objectively-measured 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 followed since birth allowed us to address limitations of prior 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. Further, while actigraphy is a practical method to measure sleep over multiple days with minimal participant burden,[101] actigraphy differs from the gold-standard (polysomnography)[48] and there are important exposures (e.g., sleep stage distribution) not captured by actigraphy.[81]

CONCLUSION

Sleep quantity and quality are associated with a more favorable cardiometabolic profile in early adolescence. Though 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 do not fully account for the associations, suggesting 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 future studies should examine intervention strategies to improve sleep quality as well as quantity in adolescents.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Abbreviations:

BMI	Body mass index
SD	Standard deviation
IQR	Inter-Quartile Range
WASO	Wake after sleep onset
HOMA-IR	Homeostatic Model Assessment of Insulin Resistance

HDL-cholesterol	High-density lipoprotein cholesterol
DXA	dual-energy x-ray absorptiometry
MVPA	moderate-to-vigorous physical activity

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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 using actigraphy - are associated with a more favorable cardiometabolic profile, including lower abdominal adiposity, lower systolic blood pressure and higher HDL-cholesterol. Many associations were independent of obesity-related behaviors and BMI.

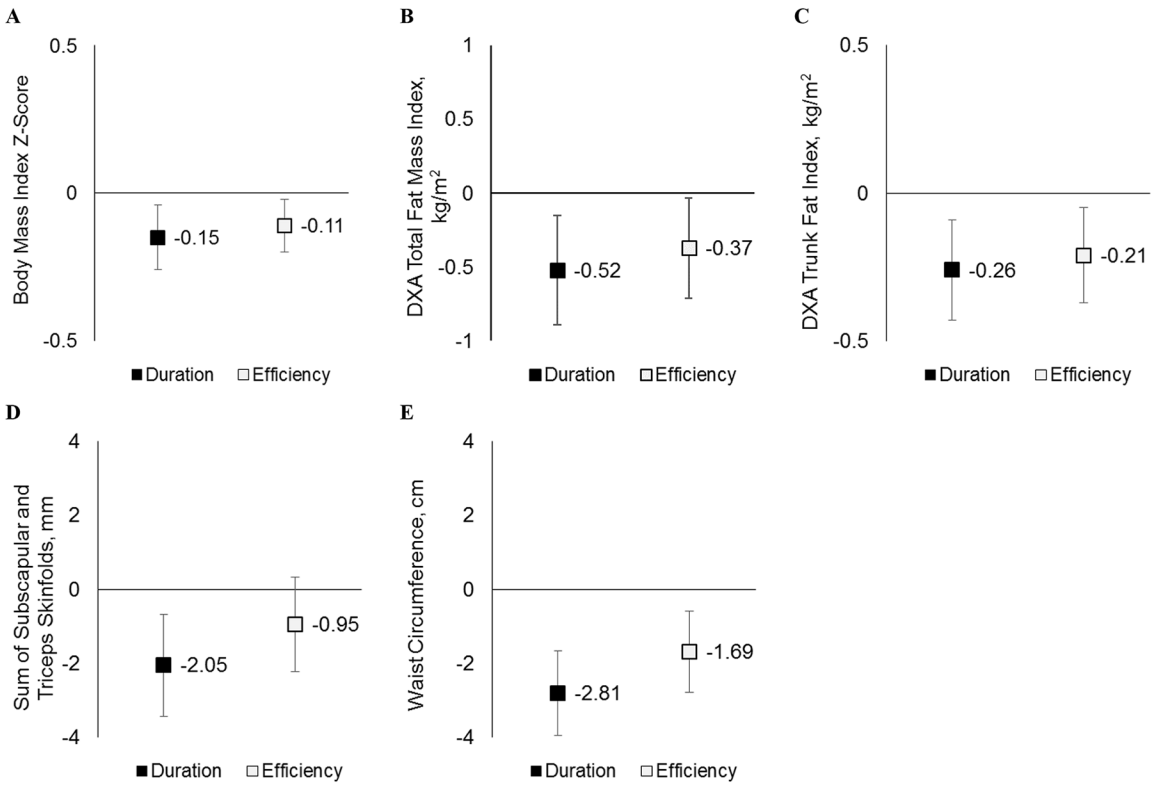

Figure 1. Multivariable-Adjusted associations of sleep duration and efficiency (per inter-quartile increase) with adiposity outcomes.

Results for adiposity outcomes (A. Body Mass Index Z-score, B. DXA Total Fat Mass Index, C. DXA Trunk Fat Index, D. Sum of Subscapular and Triceps Skinfolds, and E. Waist Circumference) are shown per inter-quartile range of sleep exposures: duration (55 minutes) and efficiency (6%). Models adjust for maternal education and household income, season of measurement, and adolescent age, sex, race/ethnicity, and puberty (Tanner stage).

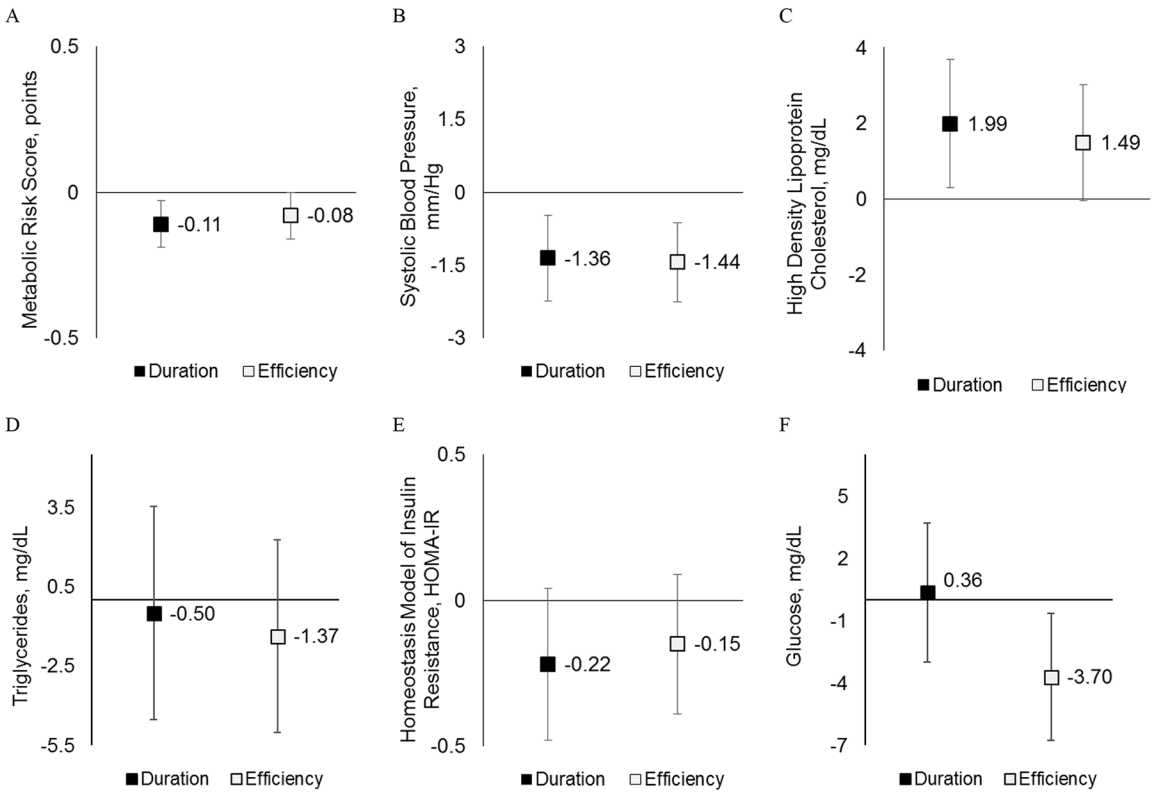

Figure 2. Multivariable-Adjusted associations of sleep duration and efficiency (per inter-quartile increase) with cardiometabolic biomarker outcomes.

Results for cardiometabolic outcomes (A. Metabolic Risk Score, B. Systolic Blood Pressure, C. High Density Lipoprotein [HDL]-Cholesterol, D. Triglycerides, E. Homeostatis Model Assessment of Insulin Resistance [HOMA-IR], and F. Glucose) are shown per inter-quartile range of sleep exposures: duration (55 minutes) and efficiency (6%). Models adjust for maternal education and household income, season of measurement, and adolescent age, sex, race/ethnicity, and puberty (Tanner stage).

Table 1.

Parent, household and child characteristics according to actigraphy-estimated sleep duration in early adolescence. Data from 829 participants from Project Viva.

	Overall n=829	<480 minutes/day n=692	≥480 minutes/day n=137
Mother/household characteristics		Mean (SD)	
Maternal age at enrollment, years	32.3 (5.0)	32.3 (5.1)	32.6 (4.7)
Mother graduated college		N (%)	
No	228 (27.5)	198 (28.7)	30 (21.9)
Yes	600 (72.5)	493 (71.3)	107 (78.1)
Household income >$70,000/year at early teen visit		N (%)	
No	177 (22.2)	154 (23.1)	23 (17.4)
Yes	622 (77.8)	513 (76.9)	109 (82.6)
Adolescents' characteristics			
Female		N (%)	
No	402 (48.5)	357 (51.6)	45 (32.8)
Yes	427 (51.5)	335 (48.4)	92 (67.2)
Race/ethnicity		N (%)	
Black	131 (15.8)	117 (16.9)	14 (10.2)
Hispanic	36 (4.3)	33 (4.8)	3 (2.2)
Asian	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		N (%)	
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)
		Mean (SD)	
Tanner stage[1]	3.6 (1.0)	3.6 (1.0)	3.5 (1.1)
Age, years	13.2 (0.9)	13.2 (0.9)	13.1 (0.8)
Television viewing, hours/day	2.0 (1.4)	2.1 (1.4)	1.7 (1.1)
Fast food intake, servings/week	0.7 (1.1)	0.7 (1.1)	0.6 (1.1)
Sugar sweetened beverage intake, servings/day	0.8 (0.9)	0.8 (0.9)	0.7 (0.7)
BMI z-score	0.36 (1.07)	0.41 (1.06)	0.11 (1.07)
Actigraphy measurements:		Median (IQR)	
Average daily moderate-to-vigorous physical activity, minutes	7.0 (17.0)	7.0 (16.0)	7.0 (16.5)
Average daily sleep duration, minutes	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 wake after sleep onset, minutes	74.8 (36.2)	75.9 (36.3)	69.2 (33.9)
Day-to-day variability in sleep duration, standard deviation[2]	56.0 (35.4)	55.7 (35.8)	58.5 (34.1)
Adiposity outcomes		Mean (SD)	

	Overall	**<480 minutes/day**	**≥480 minutes/day**
	n=829	**n=692**	**n=137**
Body mass index z-score, n=827	0.36 (1.07)	0.41 (1.06)	0.11 (1.07)
Waist circumference, cm, n=829	72.8 (11.7)	73.4 (11.9)	69.7 (10.5)
Sum of skinfolds, mm, n=826	28.2 (13.7)	28.6 (14.2)	26.2 (11.2)
DXA fat mass index, kg/m^2, n=619	6.3 (3.1)	6.4 (3.2)	5.9 (2.3)
DXA trunk fat mass index, kg/m^2, n=619	2.4 (1.5)	2.4 (1.5)	2.2 (1.1)
Cardiometabolic outcomes		Mean (SD)	
Metabolic risk, z-score[3], n=493	−0.02 (0.60)	0.00 (0.61)	−0.12 (0.56)
HOMA-IR, n=496	3.1 (1.9)	3.2 (2.0)	2.8 (1.4)
Insulin, uU/ml n=560	15.9 (14.9)	16.4 (15.9)	12.9 (6.1)
Glucose, mg/dL n=513	92.7 (23.5)	93.0 (25.1)	91.1 (11.5)
Systolic BP, mmHg, n=823	107.2 (8.9)	107.7 (8.9)	104.9 (9.0)
Triglycerides, mg/dL, n=559	69.8 (31.3)	69.1 (31.3)	73.4 (31.3)
HDL-cholesterol, mg/dL, n=560	55.3 (13.1)	55.1 (13.2)	56.9 (12.5)

[1] Tanner stage pubic hair (5-point scale)

[2] Standard deviation of daily time spent asleep (all days), minutes

[3] Mean of 5 sex- and cohort-specific z-scores for waist circumference, systolic blood pressure, high-density lipoprotein (HDL)-cholesterol scaled inversely, and log-transformed triglycerides and homeostatic model assessment of insulin resistance (HOMA-IR); higher scores indicate greater metabolic risk.

Table 2.

Multivariable-adjusted associations of sleep measures with adiposity and cardiometabolic outcomes

	Model 1	Model 2	Model 3
Sleep Duration			
(per 55 minutes)			
Body mass index 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, uU/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, mmHg	−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%)			
Body mass index 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, uU/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, mmHg	−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)
Wake After Sleep Onset			
(per 36 minutes)			
Body mass index 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, uU/ml	1.20 (−0.34, 2.73)	0.51 (−1.24, 2.26)	0.52 (−1.15, 2.18)

	Model 1	Model 2	Model 3
Glucose, mg/dL	1.51 (−1.08, 4.10)	2.72 (−0.52, 5.97)	2.70 (−0.52, 5.93)
Systolic blood pressure, mmHg	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 minutes)			
Body mass index 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, uU/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, mmHg	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. Adjusted for age and sex

Model 2. Model 1 + maternal education and household income, season of measurement, and adolescent race/ethnicity, puberty (Tanner stage), and obesity-behaviors (moderate-vigorous physical activity, television-viewing hours/day and fast food and sugar-sweetened beverage servings/day).

Model 3. Model 2 + BMI z-score (for cardiometabolic biomarkers only).