

Does Sleep Duration Predict Metabolic Risk in Obese Adolescents Attending Tertiary Services? A Cross-Sectional Study

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Study Objectives: To determine, in a clinical sample of obese adolescents, whether shorter sleep duration is associated with metabolic risk and obesity severity.

Design: Cross-sectional study.

Setting: Tertiary care weight-management clinic in Cincinnati, OH, USA.

Participants: 133 obese adolescents aged 10-16.9 years.

Interventions: N/A.

Measurements: Multifaceted sleep duration data were examined with fasting venipuncture and anthropometric data collected during clinical care. *Primary Outcome:* presence of metabolic syndrome. *Secondary Outcomes:* waist circumference, triglycerides, HDL-cholesterol, blood pressure, glucose, insulin resistance (HOMA-IR), and body mass index (BMI). *Predictors:* Sleep duration by (1) parent-report, (2) self-report, and (3) multi-night actigraphy. *Analysis:* Relationships between sleep duration and each outcome were examined via regression models, adjusted for potential confounders.

Results: Regardless of how measured, sleep duration showed no strong association with metabolic syndrome (OR 1.1 to 1.5, $P = 0.2$ to 0.8), BMI ($\beta -0.03$ to -0.01 , $P = 0.2$ to 0.8), or most other outcomes. Lower triglycerides were predicted by shorter sleep duration by self-report ($\beta 12.3$, $P = 0.01$) and actigraphy ($\beta 13.6$, $P = 0.03$), and shorter parent-reported sleep duration was associated with higher HDL-cholesterol ($\beta = -2.7$, $P = 0.002$).

Conclusions: Contrary to expectations, sleep duration was not associated with metabolic outcomes, and showed limited associations with lipid profiles. Although inadequate sleep may affect other areas of functioning, it appears premature to expect that lengthening sleep will improve BMI or metabolic outcomes in clinical samples of obese adolescents.

Keywords: Sleep, obesity, metabolic syndrome, adolescent, cross sectional studies

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INTRODUCTION

Childhood Obesity and the Metabolic Syndrome

The epidemic of childhood obesity presents a major public health problem, with up to 32% of US children and adolescents overweight or obese, and up to 16% obese (Centers for Disease Control [CDC] definitions).¹ Complications from obesity involve multiple organ systems, which further compound the health risk to these children.

One cluster of obesity-linked complications, known as the metabolic syndrome, strongly predicts diabetes and early cardiovascular morbidity.²⁻⁵ In 2007, the International Diabetes Foundation defined metabolic syndrome in children as the presence of central obesity together with any 2 of the following 4 age-dependent factors: hypertriglyceridemia, low high-density lipoprotein (HDL) cholesterol, high blood pressure, and high fasting blood glucose.⁶ Metabolic syndrome has been reported

in 25% to 50% of obese children and adolescents from clinical samples,^{7,8} and in 2% to 10% of obese and non-obese adolescents in population samples.^{9,10} Finding modifiable risk factors for metabolic syndrome could therefore prevent substantial cardiovascular morbidity, particularly in obese adolescents.

Sleep Duration, Childhood Obesity, and Metabolic Syndrome

A trend toward decreasing sleep duration has paralleled the obesity epidemic. A 2005 US national survey showed that 45% of adolescents have insufficient sleep (< 8 h/night) and a further 31% have borderline (8-9 h) sleep.¹¹ Further, many studies now support an inverse linear relationship between short sleep duration and body mass index (BMI) in preadolescent children.¹²⁻¹⁶ This raises the possibility of a causal relationship, and therefore the potential for sleep related interventions.

However, results for adolescents have been less consistent,¹³ with some¹⁷⁻²¹ but not all²²⁻²⁴ reporting similar findings in population-based samples. One case-control study found differences between obese and demographically matched lean adolescents across a number of sleep variables, including duration, but did not report on associations between sleep duration and adiposity within the obese subgroup.²⁵ A major weakness of research in this area is the exclusive reliance on subjective parent- or self-reported sleep measures, with one study of 7-year-old children and 3 studies of adolescents including actigraphy^{20,25-27} as objective measures of sleep.

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Adult studies have shown sleep duration to be associated with the metabolic syndrome and diabetes.²⁸ The very few metabolic studies exploring associations in children suggest relationships between sleep duration and insulin resistance and waist circumference,²⁹⁻³¹ but are inconsistent. No study has found associations between sleep duration and hypertension, glucose levels, or lipid abnormalities in the pediatric population. Further, although obstructive sleep apnea (OSA) is common among obese youth,²⁵ there is considerable disagreement as to whether its presence or severity does³²⁻³⁷ or does not³⁸⁻⁴⁰ contribute to adolescent metabolic syndrome, over and above the contribution of obesity itself. Thus OSA could obscure or confound the relationship between sleep duration and the metabolic syndrome, especially in obese adolescents.

Aims and Hypotheses

Despite this conflicting literature on associations among child and adolescent sleep duration, BMI, and metabolic risk, sleep is being explored as a possible treatment target to minimize the risk for cardiovascular morbidity. This seems premature until associations are confirmed and quantified and, if found to be important, causal directions established. To address these issues, we studied a large clinical sample of children and adolescents attending a tertiary hospital outpatient weight management program for whom measured sleep duration, anthropometry, and markers of metabolic syndrome were all available.

Specifically, we aimed to determine, in obese 10- to 16.9-year-olds, cross-sectional associations between sleep duration and the metabolic syndrome. We also aimed to determine if associations existed between sleep duration and individual components of the metabolic syndrome (i.e., lipid markers, blood pressure, fasting glucose), as well as insulin resistance, and adiposity. We hypothesized that sleep duration would be inversely associated with metabolic syndrome, its individual components, adiposity, and insulin resistance in a manner that is statistically independent of OSA.

METHODS

Design and Setting

A sample of obese adolescents was consecutively recruited between 2003 and 2008 at intake into the Cincinnati Children's Hospital Medical Center (CCHMC) multidisciplinary pediatric weight-management clinic, as part of a larger study investigating the relationship between sleep and neurobehavioral functioning in adolescents.⁴¹ Demographic and sleep data were prospectively collected, and anthropometric and metabolic data were collected as part of each family's comprehensive intake evaluation for the clinic. Both the original study and the merging of research and clinical databases for current analyses were approved and overseen by the Institutional Review Board (IRB) of CCHMC. All parents provided written informed consent, and all participants provided written assent for collection of research-specific data and for review of the participant's medical record for data collected during clinical care.

Sample

Adolescents attending the clinic were eligible if they were 10 to 16.9 years of age and had a body mass index (BMI, kg/m²)

≥ 95th percentile for age and sex, using CDC definitions.⁴² Exclusion criteria for current analyses included neurological history (e.g., seizures), craniofacial abnormalities, developmental disorders (e.g., Down syndrome), conditions involving daytime hypoxia (e.g., poorly controlled asthma), use of psychiatric or neurological medication, and treatment for OSA in the past 2 years.

Overview of Procedures

At the initial visit to the clinic, demographics and anthropometric data, including weight, height, waist circumference, and blood pressure were routinely collected. Metabolic markers, including triglycerides, HDL-cholesterol, glucose, and insulin, were routinely collected by a 12-h fasting blood test performed within a month of the initial medical visit. All serum analyses were conducted at the same clinically accredited lab at CCHMC, and the procedure for obtaining the fasting laboratory measures was the same in all cases. During the initial clinic visit, an investigator (DWB) briefly introduced himself and the study, then followed up with interested families via telephone to conduct a more detailed discussion and screening interview. Once eligibility and participation were confirmed, a booking was made for an overnight polysomnogram (PSG) and for actigraphy, usually in the week immediately before the PSG. The PSGs were conducted at a mean of 14 weeks after the initial clinic visit. Parent and participant questionnaires were independently completed in the context of an afternoon neurobehavioral assessment (detailed in our prior report),⁴¹ which in nearly all cases occurred the afternoon prior to the PSG, and in all cases occurred within 1 month of the PSG.

MEASURES

Sleep Duration Predictor Measures

(1) Subjective sleep parameters: Parent-reported adolescent sleep duration was recorded by the Child Sleep Habits Questionnaire,⁴³ and self-reported sleep duration by the Youth Sleep Habits Survey.^{44,45} Both reporters were asked how long the participant typically slept, as well as when he or she typically goes to bed and rises for the day, on school nights. Similar questions are widely used in sleep surveys, and have been found to correlate strongly with objective actigraphy data, especially on school nights.⁴⁴ For each reporter, we computed sleep duration as the mean of the direct report of sleep duration and the arithmetic difference between reported bedtime and rise time.

(2) Objective actigraphy: As previously described,²⁵ adolescent participants were asked to wear motion-sensitive actigraphs (Mini-Motionlogger, Ambulatory Monitoring, Inc., NY) the size of sports wristwatches on their non-dominant wrists during evening and overnight hours for a week. The units were provided in person by study staff or via overnight mail, accompanied by detailed written instructions that were also reviewed via telephone. Subjects were asked to wear the units at home during evening and overnight hours during their normal sleep routine. Raw data on participant movement patterns were cross-checked against a nightly sleep diary to help screen for artifacts (e.g., actigraph removal) prior to being submitted to an automated scoring algorithm that estimated

sleep onset and offset based upon movement patterns; the results of this algorithm have been shown to correlate highly with PSG-determined sleep.⁴⁶ Sleep duration was calculated as the offset minus onset times averaged over the nights the unit was worn.

Laboratory Measures

All serum analyses were conducted at the same clinically accredited lab at CCHMC, and the procedure for obtaining the fasting blood work and laboratory measures was the same in all cases. Plasma glucose was measured using a Hitachi model 704 glucose analyzer. Plasma insulin was measured by RIA using an anti-insulin serum raised in guinea pigs, ¹²⁵I labeled insulin (Linco, St. Louis, MO), and a double antibody method to separate bound from free tracer. Assays of fasting plasma lipid profiles were carried out in a laboratory that is National Heart, Lung, and Blood Institute/Centers for Disease Control and Prevention standardized, with the low-density lipoprotein (LDL) cholesterol concentration calculated using the Friedewald equation.

Anthropometric Measures

Weight was measured by a digital scale, and height measured using a calibrated wall-mounted stadiometer while subjects were in light clothing and without shoes. Waist circumference was measured using a standardized protocol where the circumference measurement is taken just above the uppermost lateral border of the right iliac crest.⁴⁷ Blood pressure was measured using an appropriate-sized cuff, and the mean blood pressure value was taken from 2 readings.

Definitions and Calculations

The presence of metabolic syndrome was defined according to the 2007 International Diabetes Foundation criteria^{6,48} as: waist circumference \geq 90th percentile for age/gender⁴⁹ and 2 of the following: (a) fasting triglycerides \geq 150 mg/dL, (b) fasting HDL-cholesterol $<$ 40 mg/dL, (c) systolic or diastolic BP \geq 90th percentile,⁵⁰ and (d) fasting glucose \geq 100 mg/dL. HOMA-IR was calculated by [fasting serum insulin (μ U/mL) \times fasting plasma glucose (mmol/L)] divided by 22.5.⁵¹⁻⁵³ BMI (kg/m^2) was transformed to BMI z-scores using published CDC tables that adjust for age and sex.⁴²

Potential Confounders/Covariate Measures

All potential confounders were identified a priori. Parent-reported demographic variables included age, gender, race (coded as white/non-white, with 96% of non-white participants being African American) and socioeconomic status, all of which are known to be associated with sleep duration, metabolic risk, or both. Socioeconomic status was represented as a SES composite index, computed as the mean z-score within the sample across 4 variables: maternal education, paternal education, annual family income and median local income based upon the 2000 US Census for the subject's postal code of residence, prorating for any missing fields (e.g., paternal education unreported).⁴¹ For the metabolic analyses, BMI z-score was also included because of its previously reported independent associations with both sleep duration and metabolic syndrome.

Finally, OSA was entered as a covariate because OSA severity may independently influence metabolic markers, is associated with obesity, may affect sleep duration,^{25,41} and may introduce variance into the quality of sleep of any duration. OSA, as indexed by the apnea+hypopnea index (AHI), was measured during overnight PSG, conducted during a single night in an inpatient pediatric sleep unit.^{25,41} The AHI was computed as the sum of obstructive apneas and hypopneas divided by hours slept. Obstructive apnea was defined as $>$ 80% decline in airflow over 2 breaths, despite continued chest/abdominal wall movement. Obstructive hypopneas were defined as a decrease of 50% to 80% in airflow over \geq 2 breaths associated with (a) paradoxical respiration and (b) oxyhemoglobin desaturation (\geq 4%) or a subsequent arousal.

Statistical Analysis

For our primary analysis, 3 logistic regression models examined the change in odds of participants displaying metabolic syndrome by sleep duration for each of the 3 sleep duration measures (parent-report, self-report, and actigraphy). Similarly, the secondary outcomes (all continuous variables) were examined using multiple linear regression models. All regression models included the covariates identified above, with AHI log-transformed to approximate normality, and BMI z-score included only in the metabolic outcome analyses only.

Analyses were conducted using SAS 9.2 software (SAS Institute, Cary, NC). Post hoc power analyses were conducted based upon the available sample size and our observed distributions of the predictors, outcomes, and covariates. Assuming a population base rate of metabolic syndrome of 25% in obese adolescents (the observed rate in this sample was 23%), $\alpha = 0.05$, and 120 subjects, our primary analyses had 80% power to detect an odds ratio of 1.95 for a 1-h increase in sleep duration. Power would be even higher if the base rate of metabolic syndrome in obese adolescents exceeded 25, as suggested by some authors.^{7,8,54,55}

RESULTS

Sample Description

Figure 1 shows the participant flow and reasons for non-participation. Of the 380 families attending clinic, 247 were approached and deemed to be eligible for the study, of whom 133 (54%) participated and provided anthropometric and metabolic data. Of these, 92% of parents and adolescents also reported sleep duration, and 91% had actigraphy data (worn on average 5.8 ± 1.3 nights).

Table 1 summarizes the demographic, sleep, anthropometric, and metabolic features of the sample. Adolescents who participated in the study did not significantly differ from non-participants in age or sex ($P = 0.4-0.5$). Because we did not have consent to access additional information on non-participants, it is not known whether participants and non-participants differed on other study variables. By design, the sample was obese, with a mean BMI of $37 \text{ kg}/\text{m}^2$; and participants' mean sleep duration was $<$ 9 h. More than half of the sample (56%) experienced some degree of sleep disordered breathing, and 23% met criteria for the diagnosis of metabolic syndrome. A minority had individually significant markers of metabolic risk, except for

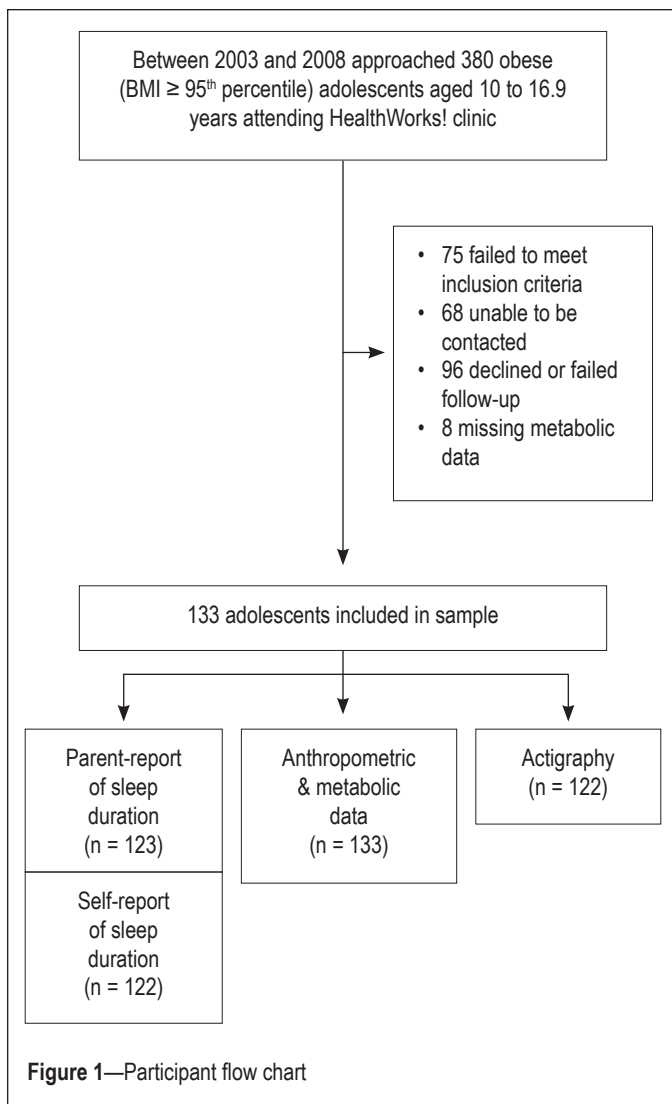


Figure 1—Participant flow chart

low HDL-cholesterol, which occurred in more than half of the sample, and high waist circumference, which was present in almost the entire sample.

Primary Outcome

There were no significant differences in sleep duration among adolescents with a diagnosis of metabolic syndrome and those without (Table 2), although small increases in the odds of having metabolic syndrome were observed for each h increase in sleep duration (parent-report OR = 1.2, 95% CI = 0.7-2.0; self-report OR = 1.1, 95% CI = 0.6-1.8; actigraphy OR = 1.5, 95% CI = 0.8-2.7, P = 0.2-0.8). Further, AHI was not a significant covariate in any of the logistic regression models.

Secondary Outcomes

There was a lack of relationship between sleep duration and all secondary outcomes, except for triglycerides and HDL-cholesterol that were in the opposite direction to that hypothesized (Table 3). Longer sleep duration by self-report and actigraphy predicted worse (i.e., higher) triglyceride levels ($\beta = 12.3$ [P = 0.01] and 13.6 [P = 0.03], respectively; Figs. 2A and 2B), while longer sleep duration by parent-report predicted worse (i.e., lower) HDL-cholesterol levels ($\beta = -2.7$ [P = 0.002]; Figure 2C). These unanticipated relationships between sleep

Table 1—Demographic, sleep and metabolic characteristics of the sample (n = 133)

Characteristics	% or Mean (SD) or Median (range)
Child	
Male sex (%)	34
Age (years), mean (SD)	13.2 (1.8)
Race: white (%)	32
BMI (kg/m ²), mean (SD)	37.0 (7.2)
BMI-z score, mean (SD)	2.5 (0.3)
Sleep duration (hours per night), median (range)	
Parent-report	8.5 (6.3 – 11.8)
Self-report	8.3 (5.5 – 13.2)
Actigraphy	7.8 (4.9 – 9.5)
Obstructive sleep apnea: AHI ≥ 1 (%)	56
Metabolic Syndrome present (%)	23
Markers of metabolic risk (%)	
Waist circumference ≥ 90 th percentile	98
Triglycerides ≥ 150 mg/dL	17
HDL cholesterol < 40 mg/dL	52
Systolic blood pressure ≥ 90 th percentile	13
Diastolic blood pressure ≥ 90 th percentile	12
Fasting glucose levels ≥ 100 mmol/L	11

Table 2—Sleep duration amongst adolescents with and without the diagnosis of metabolic syndrome

Sleep duration (hours per night), median (range)	With metabolic syndrome (n = 28)	Without metabolic syndrome (n = 105)
Parent-report	8.5 (6.5 – 11.5)	8.5 (6.3 – 11.8)
Self-report	8.5 (6.2 – 9.7)	8.1 (5.5 – 13.2)
Actigraphy	7.9 (5.6 – 9.5)	7.8 (4.9 – 9.3)

duration and triglycerides/cholesterol displayed heteroscedasticity—i.e., there was greater variability at one end of the sleep duration than at the other. Re-analysis of each secondary outcome using multiple logistic regression and dichotomizing according to cutpoints used to determine metabolic syndrome yielded nearly identical conclusions to the linear analyses.

After controlling for covariates, sleep duration did not predict BMI, BMI z-score, or waist circumference, nor did it predict insulin resistance as measured by HOMA-IR and fasting insulin levels (all P > 0.05). AHI was a significant covariate for modeling waist-circumference against self-reported sleep duration (β estimate for log-AHI = 5.68, P = 0.03) and baseline-BMI z-score against actigraphy sleep duration (β estimate for log-AHI = 0.14, P = 0.04). AHI was not a significant covariate in any of the other models for secondary outcomes.

DISCUSSION

Shorter sleep duration did not predict the presence of the metabolic syndrome, adiposity, or insulin resistance in this sample of obese children and adolescents. Moreover, short sleep duration was unexpectedly associated with *better* triglyceride and HDL-cholesterol levels, although the strength and statistical

Table 3—Regression of (Secondary) BMI and metabolic outcomes on sleep duration

Outcome Variable	Sleep duration (hours/night)								
	Parent-report			Self-report			Actigraphy		
	Beta Coeff	95% CI	P	Beta Coeff	95% CI	P	Beta Coeff	95% CI	P
BMI z-score ^a	-0.02	-0.07, 0.02	0.4	-0.01	-0.05, 0.04	0.8	-0.03	-0.09, 0.02	0.2
Metabolic markers ^b									
Waist circumference (cm)	-1.6	-3.6, 0.5	0.1	0.6	-1.2, 2.3	0.5	0.3	-1.8, 2.4	0.8
Triglycerides (mg/dL)	2.3	-9.6, 14.1	0.7	12.3*	2.5, 22.1	0.01	13.6*	1.1, 26.1	0.03
HDL-cholesterol (mg/dL)	-2.7*	-4.4, -1.0	0.002	-0.9	-2.6, 0.7	0.3	-0.8	-2.8, 1.2	0.4
Systolic blood pressure (mmHg)	0.05	-0.2, 0.3	0.6	-0.04	-0.2, 0.1	0.7	0.05	-0.2, 0.3	0.7
Diastolic blood pressure (mmHg)	0.01	-0.2, 0.2	1.0	-0.03	-0.2, 0.1	0.7	-0.2	-0.2, 0.02	0.1
Fasting glucose (mg/dL)	-0.5	-2.1, 1.1	0.5	-1.3	-2.6, 0.1	0.07	-0.8	-2.6, 0.9	0.4
HOMA-IR	0.2	-0.8, 1.2	0.7	-0.02	-0.8, 0.8	1.0	0.8	-0.2, 1.8	0.1

*Significant association in opposite direction to hypothesized relationship. ^aAdjusted for age, gender, race, socio-economic status, and obstructive apnea.
^bAdjusted for age, gender, race, socio-economic status, BMI z-score, and obstructive apnea.

significance of these effects varied across three different measures of sleep duration.

This study is the first to comprehensively describe the relationship between measured sleep duration and all aspects of the metabolic syndrome, while adjusting for potential confounders, in a group of high-risk obese children and adolescents. While conventional statistical methods are not designed to accept the null hypothesis, this study had a number of strengths that lend confidence that, at least within this subpopulation, any relationship between short sleep duration and the presence of metabolic syndrome or its elements is, at most, quite small. A post hoc power analysis suggested that the sample was of adequate size to detect even a relatively small effect at a liberal significance level of 0.05. For our primary outcome and several secondary outcomes, the nonsignificant trends ran *opposite* to predictions, suggesting that increasing the sample size would be extremely unlikely to find effects in the direction hypothesized. Further, the failure to find significant effects across multiple measures of sleep duration, including multi-night actigraphy as well as parent- and self-report, strongly argues against measure invalidity as a cause of nonsignificant results. The only other commonly used sleep measure in this age range, PSG, is the gold standard for measuring OSA, but is a poor measure of typical free-living sleep duration because it introduces artificial influences that can fundamentally alter sleep patterns (e.g., presence of monitoring leads and equipment).

Complementing our strong sleep measurement, this study adopted an internationally recognized classification of metabolic syndrome. Individual metabolic markers were also analyzed to ensure that potentially important findings did not emerge for specific elements of the metabolic syndrome, suggesting that alternate metabolic syndrome classifications would not change our conclusions. Further, aside from waist circumference, there was good variability in both the predictors and outcomes, suggesting that associations were not attenuated by a restriction in range. Finally, potential confounders were identified a priori and included in the analyses. In particular, OSA, which was prevalent in this sample, was objectively measured and included as a covariate.

There are some limitations to this study. As this was a cross-sectional study, it remains possible that short sleep increases the risk for the initial development of obesity and/or precursors of metabolic syndrome, which are then maintained and exacerbated by other factors. Such an effect would only be evident in a longitudinal model, perhaps complemented by within-subjects experimental sleep restriction studies that allow for better control of between-subjects variance. Measures for the current study were obtained at different time-points, and there was a general time-lag between the metabolic and sleep measures; insofar as sleep or metabolic functioning might have fluctuated over time this could have attenuated associations. While our recruitment from a weight management clinic allowed us to efficiently enroll significantly obese adolescents, these findings may not generalize to obese, non-treatment-seeking individuals. Detailed information on physical activity, diet, medication, smoking, and alcohol drinking were not systematically collected in the study, and therefore could not be considered as potential confounders. Although we observed considerable variability in sleep duration and the large majority of participants obtained less than the recommended 9 hours of sleep, very few had sleep duration < 6 hours (< 2% per parent-report or self-report, 6% per actigraphy). Thus it remains possible that markedly short sleep duration is associated with metabolic syndrome. Finally, this was not an intervention study that could more definitively speak to whether sleep duration modification alters metabolic risk factors within individuals; however, across obese adolescents, there appears to be minimal relationship between sleep duration and metabolic risk.

Some of our results are similar to, and some differ from, previous studies in the pediatric population. We did not find shorter sleep duration to be associated with insulin resistance (in contrast to Flint’s retrospective chart review of 40 obese 3.5- to 18.5-year-olds undergoing overnight PSG²⁹) or waist circumference (in contrast to Verhulst’s PSG study of 104 obese 6- to 17-year-olds³⁰ and Hitze’s population study of 414 6- to 20-year-olds³¹). However, in common with all three of these studies, sleep duration was not associated with blood pressure.²⁹⁻³¹

To our knowledge, this is the only study that has found an association between *longer* sleep duration and worse triglyc-

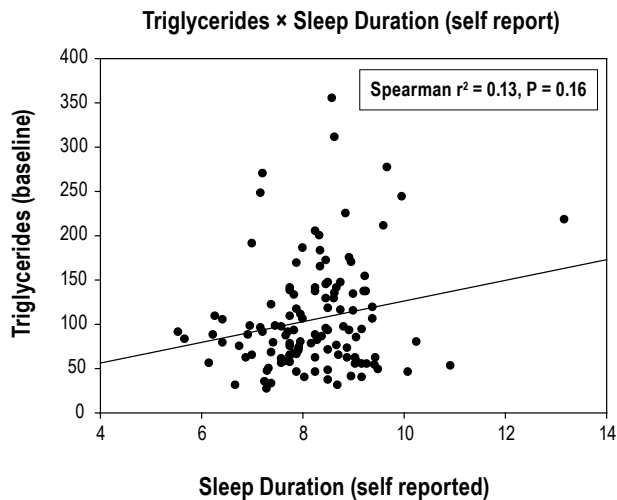


Figure 2A—Relationship between self-reported sleep duration (hours/night) and fasting triglyceride levels (mg/dL)

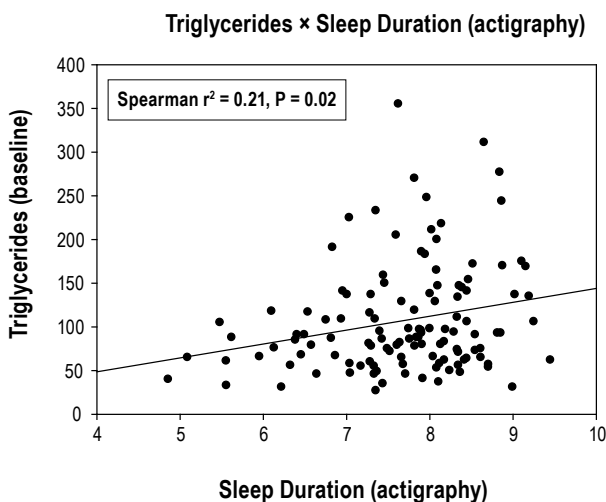


Figure 2B—Relationship between actigraphy-measured sleep duration (hours/night) and fasting triglyceride levels (mg/dL)

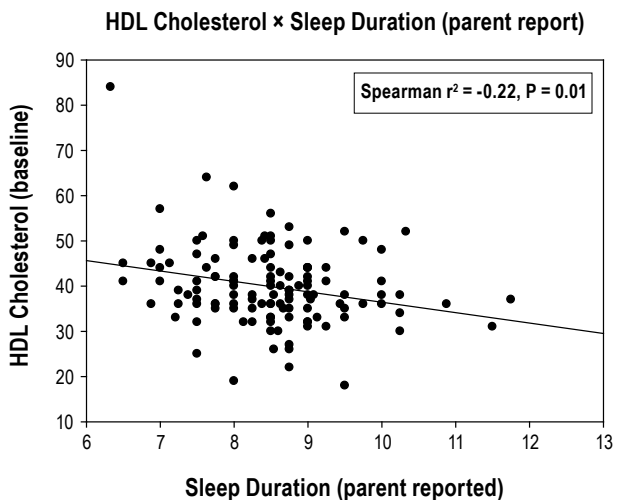


Figure 2C—Relationship between parent-reported sleep duration (hours/night) and fasting HDL cholesterol levels (mg/dL)

erides and HDL-cholesterol. While it is possible that this is a chance finding, it seems unlikely that we have missed a true association in the opposite direction, particularly given the objective measurement of free-living sleep duration. Several factors could explain the differences in results. No previous study that examined sleep and triglycerides or HDL-cholesterol included OSA or BMI z-score as covariates in the analyses. Flint's and Verhulst's studies reported sleep duration from a single night's PSG which, for reasons noted above, may not accurately reflect free-living sleep duration, while Hintze's study measured sleep duration only by child report in a non-overweight sample. Further research using at least equally reliable measures is needed to ascertain any true associations between sleep duration and lipids in obese children and adolescents. Such research should entertain the possibility of non-linear relationships, such as the U-shaped associations of sleep duration with obesity⁵⁶ and some metabolic markers^{57,58} seen in some adult studies.

Like Flint and Verhulst, we did not find an association between short sleep duration and high BMI within the subpopulation of obese adolescents. This contrasts with the widely reported inverse relationship between sleep duration and BMI in preadolescent and adolescent non-obese samples.¹²⁻²¹ While a range of adiposity was represented within our sample, that range was nevertheless restricted by definition, which could have statistically attenuated any sleep-weight relationship. Also, body mass index and related age- and sex-adjusted BMI scores may become less sensitive measures of adiposity in the most obese subpopulations.⁵⁹ Alternatively, the impact of sleep on body mass may be greatest within the large majority of the population which, despite secular trends towards overweight, continues to fall well below eligibility criteria for the current study.

Given that the relationship between obesity and metabolic risk is well established, the current science suggests that primary interventions to prevent or address obesity are more likely to reduce metabolic risk than are interventions to improve sleep in adolescents who are already obese. This is not to say that sleep is unimportant. There is evidence that OSA, which is common in this population, is associated with poor attention and classroom learning⁴¹ and that the effects of inadequate sleep on neurobehavioral functioning have been impressively larger than the relationships between short sleep and either obesity or metabolic syndrome.⁶⁰ Thus, interventions to improve the sleep of obese youth may yield important health benefits, even if not in the weight or metabolic arenas.

CONCLUSION

Despite evidence in adults that sleep duration is associated with the metabolic syndrome, hypertension, diabetes, cardiovascular morbidity, and obesity, these associations may not apply to clinically obese adolescents. Obese adolescents often have poor sleep quality, and improving this may have a number of health benefits (e.g., improved behavioral functioning and quality of life). However, prolonging sleep duration may not improve metabolic risk in this clinically at-risk group.

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DISCLOSURE STATEMENT

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