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## Longitudinal Associations of Sleep Curtailment with Metabolic Risk in Mid-Childhood

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### Abstract

**Objective**—To examine associations of chronic insufficient sleep with mid-childhood cardio-metabolic health.

**Design and methods**—At 6 months and yearly from 1–7 years, mothers participating in the Project Viva cohort reported children's 24-hour sleep duration. The main exposure was a sleep curtailment score, ranging from 0 (maximal curtailment) to 13 (never having curtailed sleep). The main outcome was a mid-childhood metabolic risk score, derived as the 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 indicate higher risk.

**Results**—The mean (standard deviation [SD]) sleep score was 10.0 (2.8); 5.1% scored 0–4, 13.9% scored 5–7, 14.1% scored 8–9, 28.7% scored 10–11, and 38.3% scored 12–13. Mean (SD, range) metabolic risk score was –0.03 (0.6, –1.8 to 2.6). In multivariable models, the metabolic risk score difference for children with most versus least curtailed sleep was 0.29 units (95% Confidence Interval [CI]: 0.02, 0.57). Further adjustment for mid-childhood BMI-z score attenuated this difference to 0.08 units (95% CI: –0.14, 0.30).

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**Competing interests:** The authors have no competing interests.

**Conclusions**—Chronic insufficient sleep from infancy to school-age was associated with higher mid-childhood metabolic risk. This association was explained by sleep duration's influence on mid-childhood adiposity.

### Keywords

sleep curtailment; cardio-metabolic health; early childhood

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### Introduction

Cross-sectional and longitudinal studies have established associations between shorter sleep duration and increased risk of obesity in children.<sup>1, 2</sup> Patterns of shorter sleep duration (<10 hours/day) during early childhood are preserved over long periods and increase risk of excess weight in later childhood.<sup>3</sup> Independent of obesity, inadequate sleep may contribute to metabolic dysfunction: in adults, experimental evidence supports a role for shorter sleep in decreased insulin sensitivity,<sup>4</sup> disrupted glucose homeostasis and appetite regulation,<sup>5</sup> as well as cardiac risk factors such as hypertension<sup>6</sup> and incident diabetes.<sup>7</sup> Among adults, suggested mechanisms include the impact of insufficient sleep on glucose regulation as well as adipokines, e.g. increasing interleukin-6 (IL-6) and decreasing adiponectin.

Limited research has examined sleep duration and metabolic dysfunction in children. Recently, Spruyt et al found longer actigraph-recorded sleep duration among school-age children was cross-sectionally associated with a lower probability of metabolic dysfunction, as measured through glucose, insulin, cholesterol, triglycerides and high sensitivity C-reactive protein (hsCRP), independent of body mass index (BMI).<sup>8</sup> Most, but not all,<sup>9</sup> cross-sectional studies have linked shorter sleep duration to adverse metabolic outcomes in children (e.g. higher Homeostatic Model Assessment for assessing Insulin Resistance (HOMA-IR), lower adiponectin,<sup>10</sup> increased non-high-density lipoprotein (HDL) cholesterol,<sup>11</sup> and higher blood pressure).<sup>12</sup> To our knowledge, no previous study has assessed prospective relationships of chronic insufficient sleep with an array of biomarkers of glucose and lipid metabolism, blood pressure, adiponectin, and inflammation in a cohort of young children without diagnosed sleep disorders.

The goal of this study was to examine whether chronic insufficient sleep from infancy to mid-childhood was associated with cardio-metabolic risk in mid-childhood, and the extent to which associations were explained by attained adiposity. This biomarker study builds on prior research showing associations of chronic sleep curtailment from infancy to mid-childhood with adiposity in early and mid-childhood.<sup>13</sup> We hypothesized that chronic insufficient sleep would be associated with an adverse cardio-metabolic state in mid-childhood, and that adjustment for mid-childhood BMI z-scores would attenuate these associations. A better understanding of the contributions of chronic insufficient sleep to later metabolic health may inform early life interventions to prevent obesity and cardiovascular disease.

## Methods

### Subjects/Study Design

Study subjects were participants in Project Viva, a prospective cohort study that recruited women during early pregnancy from Harvard Vanguard Medical Associates, a multi-specialty group practice in eastern Massachusetts. Details of recruitment and retention procedures are available elsewhere.<sup>14</sup> Of the 2128 women who delivered a live infant, 1116 families attended a 7 year (“mid-childhood”) in-person visit and 702 children provided blood samples. Since our main exposure was chronic sleep curtailment from 6 months to 7 years, we further excluded participants who did not have sleep data for these time points. Our sample for analysis was 652 children. Participants that were not included had similar characteristics to those who were: 56% not included versus 62% included had household incomes >\$70,000/year, 63% versus 67% had a college degree, and maternal age at enrollment was 32 years for both groups.

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

### Measurements

**Main Exposures**—At 6 months and yearly from 1 to 7 years, mothers reported children’s sleep duration in a usual 24-hour period. At 6 months, we asked mother to report separately in hours/minutes their baby’s average length of morning nap, afternoon nap and nighttime sleep in the past month. At 1-year, mothers reported in hours/minutes the child’s usual 24-hour sleep duration in the past month including morning naps, afternoon naps, and nighttime sleep. Between 2–7 years, mothers reported number of hours the child slept in a usual 24-hour period in the past month, separating weekends and weekdays. Response categories included, “< 9, 9, 10, 11, 12, 13, and 14 hours/day”; at age 7 the response option was in hours/minutes.

The main exposure was a sleep curtailment score from infancy to mid-childhood. The sleep score was derived from mean sleep duration at each of the 8 measurement times: 6 months and yearly from 1–7 years. Using established thresholds from the published literature and durations associated with an increased risk for elevated BMI (> 95<sup>th</sup> percentile) in childhood<sup>15</sup> and the National Sleep Foundation age-specific recommendations,<sup>16</sup> we scored sleep duration as follows: from 6 months to 2 years, the score was 0 for <12 hours/day and 1 for ≥ 12 hours/day; from 3 to 4 years, <10 hours/day = 0, 10–<11 hours/day = 1, and ≥ 11 hours/day = 2; at 5 to 7 years, <9 hours/day = 0, 9–<10 hours/day = 1, and ≥ 10 hours/day = 2. The range of the total score was 0–13, where 0 indicated maximal sleep curtailment and 13 indicated never having curtailed sleep. To examine potential non-linear associations we categorized this score and collapsed scores of 0–4, 5–7 and 8–9 due to small frequencies and

also of 10–11 and 12–13 due to comparable results shown in prior analyses for a total of five categories.<sup>13</sup>

**Outcome Measures**—At the early and mid-childhood visits, we measured height and weight using a calibrated stadiometer (Shorr Productions, Olney, MD) and scale (Seca model 881, Seca Corporation, Hanover, MD). We calculated age- and sex-specific BMI z-scores using US national reference data.<sup>17</sup> At the mid-childhood visit, we measured waist circumference (cm) using a Lefkin tape and systolic (SBP) and diastolic (DBP) blood pressures with a Dinamap (Critikon, Inc., Tampa, Florida) Pro 100 automated oscillometric recorder. We obtained 5 blood pressure measurements taken 1 minute apart and calculated SBP as the average of 5 measurements since the intra-class coefficient (ICC) was high (ICC = 0.74). Research assistants performing measurements followed standardized techniques<sup>18</sup> and participated in training to ensure validity.<sup>19</sup>

Phlebotomists collected blood from the antecubital vein in vacutainer tubes protected from sunlight and transported on ice to the laboratory for processing and storage within 24-hours. We centrifuged samples and stored plasma aliquots in liquid nitrogen at  $-80^{\circ}\text{C}$  until analysis. We measured plasma fasting insulin using an electrochemiluminescence immunoassay on the Roche E Modular system. Fasting glucose was measured enzymatically using Roche Diagnostics reagents (Roche Diagnostics, Indianapolis, IN). We calculated insulin resistance using the HOMA-IR (fasting insulin [ $\mu\text{U}/\text{mL}$ ] x fasting glucose [ $\text{mg}/\text{dL}$ ]/405). Triglycerides were measured enzymatically with correction for endogenous glycerol. We measured plasma concentrations of adiponectin with a radioimmunoassay (Linco Research Inc., St Charles, MO).<sup>20, 21</sup> We used an immunoturbidimetric high-sensitivity assay on a Hitachi 911 analyzer to determine hsCRP concentrations (Roche Diagnostics – Indianapolis, IN). Plasma IL-6 was measured by enzyme-linked immunosorbent assay (ELISA).

The main outcome was a mid-childhood metabolic risk score, derived as the mean of 5 sex- and cohort-specific z-scores for waist circumference, SBP, HDL cholesterol (scaled inversely), and log-transformed triglycerides and log-transformed HOMA-IR. While there is no consistent definition of the metabolic syndrome in young children, prior research has utilized similar metabolic risks scores.<sup>22–24</sup> Higher scores indicate higher metabolic risk. We also examined the individual components of the metabolic risk score as outcomes, as well as adiponectin, IL-6, hsCRP and Tumor Necrosis Factor Alpha (TNF- $\alpha$ ) because of previously observed associations of these biomarkers with sleep duration and with cardio-metabolic health.<sup>25, 26</sup>

**Other measures**—At enrollment, we collected information about maternal age, education, parity, and household income. At the early childhood visit parents reported the child's race and ethnicity. In mid-childhood, we asked mothers to report their children's average hours of TV/video time in the past month, separating weekdays and weekends. Similarly, mothers reported children's weekly active play and physical activity on an average week in the past month through separate questions about walking and light, moderate or vigorous activities such as sports. Mothers reported the child's consumption of different types of sugary drinks, including fruit drinks and soda, which we combined into a single sugary beverages variable

in servings/day. Mothers reported their pre-pregnancy weight and height, from which we calculated their BMIs.

### Statistical Analysis

We first examined bivariate relationships of the sleep curtailment score with each covariate and with anthropometric (waist circumference) and biomarker outcomes. We then used multivariable linear regression models to examine associations of the sleep score as a continuous and categorical variable. Since prior research has suggested sex differences in the metabolic response to chronic sleep curtailment,<sup>27, 28</sup> we repeated analyses separately in boys and girls.

Our first model, Model 1, was adjusted for child age and sex only. We additionally adjusted for potential confounding by household income, maternal education, age, and pre-pregnancy BMI and child race/ethnicity (Model 2). Finally, we assessed the extent to which associations were attenuated after adjustment for attained adiposity as represented by BMI z-score in mid-childhood (Model 3). We also considered adjustment for BMI z-score in early childhood, servings/day of sugary drinks and hours/day of TV/video-viewing and active play in mid-childhood, but these did not change estimated associations or conclusions and were not included in final models.

In sensitivity analyses, we considered alternative derivations of the sleep score in which sleep duration in infancy and toddlerhood was treated in three categories, allowing each period of childhood to contribute equally to the total score. We also adjusted for the time of day of blood draw to assess whether circadian rhythms in the biomarkers might influence the results.

We examined the residuals of each model, which appeared normal with the exception of IL-6 and hsCRP; these we log-transformed. Point estimates and confidence intervals were exponentiated for presentation in tables. Thus, model coefficients can be interpreted as the mean difference in the metabolic outcomes in their native scale with the exception of IL-6 and hsCRP, which should be interpreted as relative changes in these outcomes.

The confounding variables in our analyses were not available for all subjects. We used multiple imputation to generate several plausible values for each missing value.<sup>29, 30</sup> We used a chained equations approach with predictive mean matching based on linear regressions for approximately continuous variables and logistic or generalized logistic regression for dichotomous or more generally categorical variables. The “completed” data set comprises the observed data and one imputed value for each missing value. We replicated this analysis across completed data sets and combined them in a structured fashion that reflects the true amount of information in the observed data, i.e., without presuming that the imputed values are known true values, but recovering the information in partially observed subjects. We generated 50 complete data sets<sup>31</sup> and combined multivariable modeling results (Proc MI ANALYZE) in SAS version 9.3 (SAS Institute, Cary NC). From these multiple imputation results, we report adjusted effect estimates from regressions and 95% confidence intervals (CI) for each sleep category with the lowest risk sleep category as the reference.

## Results

Characteristics of study participants overall, and by sleep curtailment score, are shown in Table 1. Children from families with lower incomes and lower education had lower sleep scores, indicating greater exposure to insufficient sleep from infancy to mid-childhood (Table 1). In addition, black and Hispanic children, as well as children of “other” races/ethnicities, had more curtailed sleep than non-Hispanic white children (Table 1). In mid-childhood, curtailed sleep was associated with greater hours of television and greater servings of sugary drinks but not with physical activity (Table 1). In bivariate analyses (Table 1), children with the lowest sleep scores (most curtailed sleep) had higher indices of BMI, waist circumference and HOMA-IR.

The mean (SD, range) of the sleep curtailment score from infancy to mid-childhood was 10.0 (2.8, 0 – 13) units. Score frequencies are shown in Table 1. Metabolic risk scores for children with the most versus least curtailed sleep (sleep score 0–4 versus 12–13) were 0.29 units higher (95% CI: 0.02, 0.57; Table 2, Model 2 and Figure 1) in multivariable models adjusted for child’s age, sex, and race/ethnicity and maternal age, education, pre-pregnancy BMI, parity and household income. Of the metabolic risk score components, waist circumference contributed most to this difference; waist circumference for children with the most versus least curtailed sleep was 4.76 cm higher (95% CI: 1.18, 8.34). Further adjustment for mid-childhood BMI-z score attenuated the mean differences in metabolic risk score to 0.08 units (95% CI: –0.14, 0.30; Table 2, Model 3 and Figure 1) and in waist circumference to 0.86 cm (95% CI: –1.25, 2.98; Table 2, Model 3). Though results did not achieve statistical significance for other markers, there was a suggestion of a trend towards increased risk from high to low sleep score categories for insulin, systolic blood pressure, triglycerides, HDL cholesterol, IL-6, and hsCRP (Supplemental Table 1).

Results using the continuous sleep score mirrored those with the categorical score: before adjustment for mid-childhood BMI z-score, for each incremental increase in the sleep score, the metabolic risk score was 0.03 units (95% CI: –0.05, –0.01) lower and waist circumference was 0.46 cm (95% CI: –0.72, –0.20) lower (Table 2, Model 2). Associations of higher sleep scores with lower HOMA-IR (–0.06 units; 95% CI: –0.11, –0.002) and insulin (–0.21 mg/dL; 95% CI: –0.41, –0.003) were of borderline significance (Table 2, Model 2 and Supplemental Table 1, Model 2). The sleep score was not associated with other cardio-metabolic markers in mid-childhood, and all associations were attenuated after adjustment for BMI z-score in mid-childhood.

When we repeated analyses separately by sex, we found stronger associations among girls than boys; however, confidence intervals overlapped and tests for interactions of sex with the sleep score were non-significant. Sensitivity analyses considering alternative scoring methods for deriving the sleep curtailment score and adjusting for time of day of blood draw yielded nearly identical results (data not shown).

## Discussion

In this prospective cohort, chronic sleep curtailment from infancy to mid-childhood was associated with higher metabolic risk score in mid-childhood, as well as higher levels of certain metabolic components including higher waist circumference and insulin. All associations attenuated after adjustment for mid-childhood BMI z-score, suggesting that increased metabolic risk was related to mid-childhood adiposity.

Prior work in Project Viva has demonstrated cumulative effects of sleep curtailment on mid-childhood adiposity; in this study, we show that while chronic insufficient sleep is an obesity risk factor, young children may not yet experience metabolic derangements beyond the direct consequences of excess adiposity. Early childhood is a time when weight trajectories are being established that may carry forward into adolescence and adulthood, underscoring the importance of establishing healthy sleep routines at a young age before more severe metabolic consequences are apparent.<sup>32</sup> Proposed mechanisms linking insufficient sleep to metabolic risk include direct effects as well as indirect effects, such as shorter sleep's influence on weight gain and obesogenic behavior. For example, sleep deprivation appears to impact hormonal signaling, leading to elevated evening cortisol levels and disrupted growth hormone, which could lead eventually to disrupted glucose homeostasis. Experimentally, sleep deprivation has been shown to increase ghrelin and decrease leptin levels in adults, stimulating hunger. This may combine with decreased impulse control and extended exposure to an obesogenic environment to decrease diet quality and increase energy intake.<sup>7, 33</sup> In children, these behaviors are unfolding in a home environment under some degree of parental control, suggesting that changes to household routines focused on improving not only sleep habits but also screen time, dietary intake and other obesogenic behaviors might have mutually reinforcing beneficial effects.<sup>34</sup>

While chronic insufficient sleep among the young children in this study did not exert metabolic effects beyond those attributable to adiposity, this may not hold true in older children or for quality rather than quantity of sleep. In cross-sectional studies shorter and poorer quality sleep have adverse metabolic consequences independent of BMI. For example, shorter sleep measured by actigraphy predicts elevated blood pressure independent of obesity among adolescents, with stronger associations for sleep efficiency than duration, suggesting that sleep quality as well as duration influences blood pressure levels.<sup>35</sup>

Our longitudinal results indicate that curtailed sleep is associated with higher insulin and HOMA-IR, and that associations are explained by mid-childhood BMI. By contrast, cross-sectional evidence in adolescents links both shorter<sup>36</sup> and longer<sup>37</sup> sleep duration with higher HOMA-IR independent of BMI. Additionally, we observed a non-significant trend towards higher hsCRP and IL-6 with more curtailed sleep while cross-sectional studies in schoolchildren and adolescents linked more irregular<sup>8</sup> and shorter sleep<sup>38</sup> with higher hsCRP plasma concentrations independent of BMI. Finally, our study also did not find any significant associations with lipid levels though cross-sectional studies of healthy schoolchildren in Hong Kong<sup>39</sup> and Kentucky<sup>8</sup> have found inverse associations of sleep duration with total and LDL-cholesterol.

This study makes an innovative contribution to the literature through its repeated measures of sleep duration and the availability of cardio-metabolic biomarkers from a cohort of children followed from infancy to mid-childhood. Though prospective evidence that shorter sleep leads to adiposity in children is accumulating, most research including additional markers of cardio-metabolic health has been cross-sectional and has examined individual markers rather than a summary metabolic risk score as used in this study.

There are also important limitations to this study: first, sleep measures were from maternal report. In a recent validation study, though parental report of sleep duration in children 4–6 years old correlated well with actigraphy among healthy controls ( $\rho = 0.85$ ), there were significant discrepancies between reported sleep times and actigraphy.<sup>40</sup> Measurement error in sleep is most likely non-differential with respect to metabolic risk and would attenuate observed effects. Second, we did not have information about sleep quality, which may influence both sleep duration and metabolic health.<sup>7, 11</sup> Third, ideally blood samples would be collected on multiple occasions at identical times of day to minimize the influence of circadian rhythms and random fluctuations. Though collected bloods at one morning occasion, and not at identical times of day, adjustment for time of day did not alter point estimates or conclusions in multivariable models. Fourth, the majority of mothers in our study were non-Hispanic white and college-educated with household incomes  $\leq$  \$70,000/year; it is possible that results may not be generalizable to populations with different socioeconomic or racial/ethnic compositions where household routines and cultural contexts surrounding children's sleep might modify sleep's impact on metabolic health. However, analyses stratified on race/ethnicity showed similar results. Finally, as in all observational studies, unmeasured confounding by aspects of the home or neighborhood environment not controlled for through adjustment for the characteristics included in our models is possible.

## Conclusions

Our study suggests that chronic insufficient sleep in childhood may have lasting effects on adiposity, and, as a consequence, on metabolic health. Improving sleep duration and quality in childhood may be potential intervention targets to promote cardio-metabolic health.

## Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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EMC conducted all data analysis, drafted the manuscript, and completed all revisions. EMT conceptualized the analysis. All authors advised in presentation of analysis results, revised drafts critically for important intellectual content and approved a final version.



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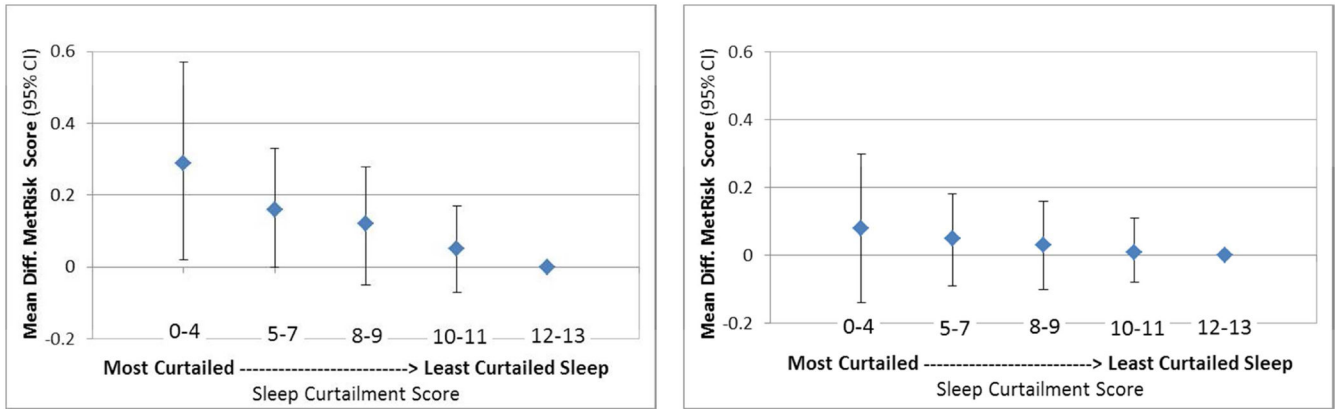
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**What is already known about this subject**

- Shorter sleep in children is associated cross-sectionally with many cardiovascular disease risk factors including obesity.
- Few longitudinal studies have examined chronic insufficient sleep or biomarkers of cardio-metabolic health in mid-childhood.

**What this study adds**

- In this cohort of children with cardio-metabolic markers including waist circumference, blood pressure, lipids, glucose, insulin, and inflammation, chronic insufficient sleep from infancy to mid-childhood is associated with greater metabolic risk.
- Associations are explained by mid-childhood adiposity.



**Figure 1.**

Figure 1a: Metabolic Risk Score by Sleep Curtailment without adjustment for BMI z-score in Mid-Childhood

Figure 1b: Metabolic Risk Score by Sleep Curtailment with adjustment for BMI z-score in Mid-Childhood

Adjusted for maternal age, education, pre-pregnancy BMI, parity; household income; child race/ethnicity & age at outcome assessment, then additionally for BMI z-score in mid-childhood

**Table 1**  
 Characteristics of 652 Children from Project Viva, Overall, and by Sleep Curtailment Score <sup>†</sup>

Characteristics	All N=652	Sleep Curtailment Score				
		0 to 4 33 (5.1%)	5 to 7 91 (13.9%)	8 to 9 92 (14.1%)	10 to 11 187 (28.7%)	12 to 13 250 (38.3%)
Maternal and Household		Mean (SD) or %				
Maternal age at enrollment, years	32.1 (5.4)	30.0 (6.7)	31.0 (6.9)	31.4 (7.4)	32.6 (5.7)	32.6 (4.9)
Maternal BMI, kg/m <sup>2</sup>	24.9 (5.3)	27.2 (7.8)	25.1 (6.6)	26.2 (7)	24.9 (5.8)	24.1 (4.6)
Nulliparous, %	43.1	23.0	41.0	47.6	40.9	46.5
Education, College grad, %	67.3	23.2	45.3	60.2	75.7	77.4
Household income >\$70k/yr, %	61.6	21.6	40.6	49.0	66.3	75.6
<b>Child</b>						
Age at blood draw, years	7.9 (0.8)	8.1 (1.2)	8.1 (1.1)	8.1 (1)	7.9 (0.8)	7.7 (0.7)
Girl, %	47.6	51.8	40.4	49.2	43.8	51.8
Race/ethnicity, %						
Black	19.4	42.5	39.8	34.3	15.9	5.9
Hispanic	4.1	10.8	7.7	4.3	2.3	3.3
Other	14.6	28.1	22.2	17.9	13.8	9.4
White	61.9	18.7	30.3	43.5	67.9	81.4
<i>Mid-childhood behaviors</i>						
Television viewing, hours/day	1.6 (1.0)	2.6 (1.5)	2.0 (1.3)	1.7 (1.2)	1.6 (0.9)	1.4 (0.8)
Physical activity, hours/day	1.8 (1.3)	1.8 (1.8)	1.8 (1.5)	1.7 (1.6)	1.8 (1.4)	1.9 (1.3)
Sugary drinks, servings/day	0.4 (0.8)	0.7 (1.4)	0.7 (1.2)	0.5 (1.2)	0.4 (0.7)	0.3 (0.5)
<i>Cardio-metabolic outcomes</i>						
Body mass index, z-score	0.40 (1.01)	1.00 (5.58)	0.60 (3.16)	0.58 (3.07)	0.35 (2.12)	0.21 (1.61)
Metabolic Risk Score *	-0.03 (0.6)	0.22 (0.9)	0.05 (0.7)	0.05 (0.7)	-0.04 (0.7)	-0.11 (0.6)
Waist circumference, cm	59.9 (8.5)	65.0 (13.1)	61.9 (11.2)	61.2 (9.8)	59.8 (8.9)	58.1 (7.1)
Diastolic blood pressure, mm/Hg	54.3 (5.7)	54.6 (6.9)	54.6 (6.8)	54.2 (5.9)	53.9 (5.7)	54.5 (6.2)
Systolic blood pressure, mm/Hg	94.4 (8.8)	95.6 (11.1)	95.1 (11.5)	95.5 (9.6)	94.4 (9.7)	93.7 (8.7)
Triglycerides, mg/dL	57.9 (26.3)	60.6 (34.6)	56.2 (24.9)	56.4 (26.3)	58.6 (29.1)	58.1 (26.2)
Total cholesterol, mg/dL	160.1 (29.0)	160.9 (34.3)	160.8 (32.6)	161.3 (33.1)	159.4 (30.1)	160.0 (29.5)
HDL cholesterol, mg/dL	57.4 (14.2)	55.3 (16.3)	58.1 (13.9)	57.1 (14.2)	58.0 (15.4)	57.0 (14.5)

Characteristics	Sleep Curtailment Score					
	All N=652	0 to 4 33 (5.1%)	5 to 7 91 (13.9%)	8 to 9 92 (14.1%)	10 to 11 187 (28.7%)	12 to 13 250 (38.3%)
HOMA-IR	1.8 (1.6)	2.4 (2.1)	2.0 (2.1)	2.0 (1.8)	1.8 (1.9)	1.5 (1.2)
Fasting glucose, mg/dL	94.6 (16.1)	95.6 (19.9)	95.2 (17.7)	95.8 (17.8)	95.4 (16.6)	93.2 (16.1)
Fasting insulin, uU/mL	7.5 (5.9)	10.2 (8.4)	8.4 (7.4)	8.2 (6.8)	7.4 (6.9)	6.7 (4.8)
Adiponectin, ug/mL	15.6 (8.9)	15.0 (11.9)	14.8 (8.9)	16.1 (11)	15.3 (9.7)	15.9 (9.4)
TNF- $\alpha$ , pg/mL	2555 (605)	2568 (771)	2577 (646)	2481 (604)	2539 (622)	2586 (647)
		<b>Median (IQR)</b>				
IL-6, pg/mL	0.6 (0.7)	0.9 (0.8)	0.7 (0.7)	0.6 (0.6)	0.6 (0.6)	0.6 (0.7)
hsCRP, mg/dL	0.2 (0.5)	0.3 (1.6)	0.2 (0.6)	0.2 (0.5)	0.2 (0.5)	0.1 (0.3)

<sup>†</sup>The range of the total sleep score is 0–13, where 0 indicates the maximal sleep curtailment and 13 indicates never having curtailed sleep.

\*The metabolic risk score is composed of the mean of 5 sex-specific internal z-scores for waist circumference, systolic blood pressure, inverted HDL cholesterol, and log-transformed triglycerides and HOMA-IR.

**Table 2**

Association of Sleep Curtailment Score with the Metabolic Risk Score and Components ( $n = 652$ )<sup>†</sup>

<u>Sleep Curtailment Score</u>	<b>0 to 4</b> 5.1%	<b>5 to 7</b> 13.9%	<b>8 to 9</b> 14.1%	<b>10 to 11</b> 28.7%	<b>12 to 13</b> 38.30%	<i>Continuous Score</i>
	<b>Mean Difference (95% Confidence Interval)</b>					
<b>Metabolic Risk Score</b>						
Model 1. Child age & sex	<b>0.28 (0.01, 0.54)</b>	0.12 (-0.04, 0.27)	0.11 (-0.05, 0.27)	0.05 (-0.07, 0.17)	0.0 (ref)	<b>-0.03 (-0.04, -0.01)</b>
Model 2. Model 1 + SES	<b>0.29 (0.02, 0.57)</b>	0.16 (0.00, 0.33)	0.12 (-0.05, 0.28)	0.05 (-0.07, 0.17)	0.0 (ref)	<b>-0.03 (-0.05, -0.01)</b>
Model 3. Model 2 + BMI	0.08 (-0.14, 0.30)	0.05 (-0.09, 0.18)	0.03 (-0.10, 0.16)	0.01 (-0.08, 0.11)	0.0 (ref)	-0.01 (-0.03, 0.01)
<i>Components</i>						
<b>Waist circumference, cm</b>						
Model 1. Child age & sex	<b>5.56 (2.04, 9.08)</b>	<b>2.74 (0.55, 4.93)</b>	1.98 (-0.26, 4.23)	1.34 (-0.31, 2.98)	0.0 (ref)	<b>-0.51 (-0.74, -0.27)</b>
Model 2. Model 1 + SES	<b>4.76 (1.18, 8.34)</b>	<b>2.7 (0.46, 4.94)</b>	1.34 (-0.84, 3.52)	1.07 (-0.51, 2.64)	0.0 (ref)	<b>-0.46 (-0.72, -0.20)</b>
Model 3. Model 2 + BMI	0.86 (-1.25, 2.98)	0.6 (-0.74, 1.94)	-0.17 (-1.47, 1.13)	0.42 (-0.52, 1.35)	0.0 (ref)	-0.06 (-0.22, 0.09)
<b>Systolic blood pressure, mm/Hg</b>						
Model 1. Child age & sex	1.07 (-2.81, 4.94)	0.74 (-1.65, 3.12)	1.15 (-1.17, 3.47)	-0.75 (-1.93, 0.43)	0.0 (ref)	-0.14 (-0.40, 0.12)
Model 2. Model 1 + SES	1.22 (-2.75, 5.19)	0.98 (-1.53, 3.48)	0.70 (-1.67, 3.08)	0.29 (-1.48, 2.06)	0.0 (ref)	-0.16 (-0.45, 0.12)
Model 3. Model 2 + BMI	-0.85 (-4.51, 2.81)	-0.14 (-2.51, 2.23)	-0.10 (-2.28, 2.08)	-0.06 (-1.68, 1.56)	0.0 (ref)	0.05 (-0.22, 0.32)
<b>Triglycerides, mg/dL</b>						
Model 1. Child age & sex	2.36 (-8.10, 12.82)	-1.91 (-8.83, 5.02)	-1.77 (-8.36, 4.82)	0.62 (-4.66, 5.89)	0.0 (ref)	-0.01 (-0.78, 0.75)
Model 2. Model 1 + SES	6.78 (-4.40, 17.95)	2.25 (-5.08, 9.59)	1.50 (-5.39, 8.39)	1.72 (-3.59, 7.02)	0.0 (ref)	-0.64 (-1.51, 0.23)
Model 3. Model 2 + BMI	3.54 (-7.42, 14.49)	0.50 (-6.64, 7.63)	0.24 (-6.58, 7.06)	1.18 (-4.07, 6.42)	0.0 (ref)	-0.31 (-1.17, 0.55)
<b>HDL cholesterol, mg/dL</b>						
Model 1. Child age & sex	-2.16 (-7.95, 3.64)	0.41 (-3.23, 4.06)	-0.32 (-3.97, 3.34)	0.58 (-2.18, 3.35)	0.0 (ref)	0.12 (-0.31, 0.55)
Model 2. Model 1 + SES	-3.38 (-9.64, 2.87)	-0.81 (-4.74, 3.11)	-1.21 (-5.02, 2.61)	0.16 (-2.65, 2.96)	0.0 (ref)	0.30 (-0.19, 0.78)
Model 3. Model 2 + BMI	-1.84 (-8.16, 4.48)	0.02 (-3.85, 3.88)	-0.61 (-4.37, 3.15)	0.41 (-2.35, 3.17)	0.0 (ref)	0.14 (-0.35, 0.63)
<b>HOMA-IR</b>						
Model 1. Child age & sex	<b>0.83 (0.17, 1.50)</b>	<b>0.49 (0.07, 0.91)</b>	0.40 (-0.02, 0.81)	0.26 (-0.06, 0.58)	0.0 (ref)	<b>-0.09 (-0.13, -0.04)</b>
Model 2. Model 1 + SES	0.47 (-0.23, 1.18)	0.31 (-0.15, 0.77)	0.22 (-0.20, 0.65)	0.19 (-0.12, 0.51)	0.0 (ref)	<b>-0.06 (-0.11, -0.002)</b>
Model 3. Model 2 + BMI	0.17 (-0.49, 0.83)	0.15 (-0.29, 0.59)	0.11 (-0.30, 0.51)	0.14 (-0.15, 0.44)	0.0 (ref)	-0.02 (-0.08, 0.03)

<sup>†</sup> All analyses use linear regression. Model 1 is adjusted only for child age and sex. Model 2 is additionally adjusted for maternal education, pre-pregnancy BMI, age at enrollment and nullparity; household income; and child race/ethnicity. Model 3 is further adjusted for child BMI z-score at mid-childhood.



The range of the total sleep score is 0–13, where 0 indicates the maximal sleep curtailment and 13 indicates never having curtailed sleep.

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