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Current child, but not maternal, snoring is bi-directionally related to adiposity and cardiometabolic risk markers: A cross-sectional and a prospective cohort analysis

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Abstract

Purpose—Obstructive sleep apnea (OSA), typically manifested as snoring, is closely associated with obesity. However, the directionality of associations of OSA with cardiometabolic risk markers is unclear, as obesity increases risk for OSA, and OSA results in excess weight gain and its metabolic consequences. Less is known about how obesity and OSA may relate in children and adolescents and whether maternal OSA may influence the development of obesity and cardiometabolic dysfunction in offspring.

Basic Procedures—Among 1078 children from the Project Viva cohort, we examined cross-sectionally and prospectively associations of parent-reported child or maternal snoring with cardiometabolic outcomes, including adiposity, adipokines, and insulin resistance.

Main Findings—Cross-sectionally, child snoring was related to adiposity and metabolic risk, particularly body mass index (BMI; β 0.61 kg/m^2, 95% CI 0.33, 0.89; p < 0.001), trunk fat mass index (β 0.23 kg/m^2, CI 0.12, 0.34; p < 0.001), high-density lipoprotein cholesterol (β −1.47 mg/dL, CI −2.69, −0.25; p = 0.02), and metabolic risk z-score (β 0.08, CI 0.02, 0.14; p = 0.01) after correction for covariates. Prospectively, adiposity (BMI, trunk fat, fat mass, and waist

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Author Contributions

CSM designed the study data analysis and SRS analyzed the data. OMF wrote the manuscript. SRS, EO, ET, and CSM reviewed and revised the manuscript. SRS and OMF take responsibility for the data presented in the manuscript. All authors have reviewed and approved the final manuscript.

Disclosures

Authors have no conflicts to disclose.

circumference) and cardiometabolic (leptin, HOMA-IR, CRP, and global metabolic risk) measures at mid-childhood (~7 y) were associated with child snoring at the early teen visit (~12 y) after correction for covariates. Child snoring at ~9 y was related to changes in adiposity between mid-childhood and early teen visits.

Conclusions—Child but not maternal snoring, was related to child adiposity and cardiometabolic outcomes. Adiposity and child snoring are associated with each other cross-sectionally and are each predictive of the other among children/adolescents prospectively. These results suggest similar mechanisms in pediatric/adolescent populations as in adults for the development of sleep-disordered breathing and sleep apnea that will need to be confirmed in randomized clinical trials. Importantly, this research points to the need to target both sleep and obesity in order to break this vicious cycle.

Keywords

Snoring; Obstructive sleep apnea; Obesity; Child health; Cardiometabolic risk

1. Introduction

Both obesity and obstructive sleep apnea (OSA) are increasing in prevalence in the United States (US) and globally [1,2]. Approximately 35% of US adults are obese and 20% of adults have at least mild OSA [3]. Childhood and adolescent obesity (2–19 years) in the USA has remained stable from 2011 to 2014 at 17%, with gradually increasing rates across ages from 2 to 5 year-olds at 8.9% to 12–19 year-olds at about 20.5% [4]. The prevalence rate for OSA in children is lower, around 4% among children aged 8–11 years in the Cleveland Children's Sleep and Health Study [5] and 10.6% (apnea-hypopnea index, AHI ≥ 5) among children aged 9.2 ± 0.8 years in the Penn State Child Cohort [6]. It is critical to understand how OSA may be implicated in children/adolescents as this may set the stage for more serious consequences in adulthood.

Obesity can lead to OSA through mechanical effects on airways. In addition, evidence from adults suggests that OSA appears to exacerbate obesity and its comorbidities, such as diabetes and cardiovascular disease [7–10]. OSA results in disrupted sleep and hypoxia, both of which have been linked to weight gain and insulin resistance. More severe OSA is related to worsening diabetes [7,8,11–14]. OSA in adults has also been linked with poorer metabolic outcomes, including changes in insulin, leptin, and lipid profile [15–19], and the associations were only partially explained by obesity.

Limited studies have examined these associations in children and adolescents, but they are mostly cross-sectional in nature, underpowered and/or show conflicting results. A few prospective studies have looked at only inflammation and overweight/obesity in association with snoring and OSA, but not in-depth the complete spectrum of cardiometabolic biomarkers [20,21]. For instance, OSA in obese children was associated with greater visceral fat area derived using MRI [22]. The same investigators also demonstrated that obese children with OSA (versus obese children without OSA) had higher insulin resistance, triglycerides, and leptin [22], whereas another study showed no changes in leptin among children with versus without OSA [23].

In animal studies, maternal OSA (particularly during pregnancy) has been associated with poorer metabolic outcomes in the offspring. Body weight, food intake, adiposity index, fasting insulin, triglycerides, and cholesterol levels were higher in male rodent offspring exposed to intermittent hypoxia of maternal sleep during late gestation [24]. Body weight, visceral adipose tissue, and HOMA-IR were higher in male rodents exposed to late gestational maternal sleep fragmentation [25]. We are not aware of any human studies that have examined relationships of maternal sleep/OSA with child cardiometabolic outcomes. We begin with a cross-sectional analysis in the current study, assuming that current maternal sleep reflects pre-pregnancy sleep and/or genetic susceptibility to OSA.

To investigate associations of maternal and adolescent sleep with child obesity and metabolic risk, we used data from Project Viva, a longitudinal cohort study of mothers and children. We hypothesized that cross-sectional associations of mother-reported child sleep-disordered breathing and daytime sleepiness would be associated with child adiposity and metabolic biomarkers. As mother's sleep may indicate underlying genetics/predisposition to child OSA [26,27], maternal snoring may also impair child sleep directly, and current maternal sleep may indicate pregnancy sleep [28], we also hypothesized that the mother's own sleep-disordered breathing and daytime sleepiness would be associated with the same outcomes (adiposity and cardiometabolic bio-markers) either directly or indirectly, through genetic or other factors. We further hypothesized that the metabolic biomarkers and adiposity in mid-childhood would predict the development of sleep-disordered breathing and daytime sleepiness in early adolescence using prospective data.

2. Methods

Project Viva is a prospective cohort, which began in early pregnancy and has followed offspring into adolescence. Recruitment and procedures for Project Viva have been published previously [29,30]. Briefly, pregnant women were recruited between 1999 and 2002 from Atrius Harvard Vanguard Medical Associates and were eligible for the study if they spoke English, were <22 weeks with a singleton gestation, and had no current plans to move. Women provided written informed consent at each visit and children began providing verbal consent at mid-childhood. All procedures were approved by participating site institutional review boards. Mothers and children attended in-person research visits at mid-childhood (mean 7.9 years) and early adolescence (mean 13.2 years) and provided information via annual mailed/online questionnaires, including the 9-year questionnaire included in this analysis. Data from the 10-year questionnaire was substituted if data from the 9-year questionnaire was missing. Of the initial 2128 live births, 1177 completed any part of the early teen visit, and 1078 contributed results to any of the aims. Compared with the 1078 participants in this analysis, the 1050 non-participants were somewhat less likely to have college-educated mothers (57% v. 72%) and mean maternal age was slightly lower (31.3 v. 32.3 y). Maternal pre-pregnancy BMI (mean 25.1 v. 24.7 kg/m^2), pregnancy weight gain (mean 15.5 kg v. 15.5) and child sex (48% v. 49% girls), however, were similar.

We sought to examine the relationships between child and parent snoring with child adiposity and fat mass distribution measured by body mass index (BMI), waist circumference, skinfold thickness, and dual-energy X-ray absorptiometry (DXA) as well as

metabolic biomarkers, including indicators of cardiometabolic outcomes (total cholesterol, triglycerides, HDL-cholesterol, and hsCRP levels); insulin resistance (fasting glucose, HOMA-IR, and C-peptide); adipocyte function (plasma leptin and adiponectin); and metabolic risk scores. Data collected at the early teen visit (~12 y) were used for the cross-sectional analysis. To study the directionality of changes, anthropometry and cardiometabolic exposures from the mid-childhood (~7 y) visit were used for the prospective analysis with 12 y sleep measures as outcomes; reports from a mailed questionnaire at ~9 y, or ~10 y if ~9 y was missing, were used for sleep exposures in the prospective analysis with changes in anthropometry and cardiometabolic measures between the mid-childhood and early teen visit as outcomes, which allowed us to control for effects at the earlier age [31].

At each visit (or via mail/secure electronic communication), mothers and children completed questionnaires, which included sleep and lifestyle information (including hours watched of TV, fast food and sugar-sweetened beverage consumption, etc.). Trained research staff collected blood samples and anthropometric measurements using a stadiometer and scale for BMI with standard formulas and z-scores and tape measures for waist circumference. A DXA scan (Hologic, Bedford, MA) provided information on body composition, giving total body mass, total body fat mass (FM), trunk fat mass, and fat-free mass (FFM) percent and index. Trained staff measured skin fold thickness in the subscapular (SS) and triceps (TR) regions with Holtain calipers (Holtain Ltd., Crosswell, United Kingdom), which were summed (SS + TR). A phlebotomist collected blood samples, and research staff analyzed them as previously reported using validated assays [32,33]. Glucose, cholesterol, HDL, LDL, and triglycerides were measured enzymatically. C-peptide, and hsCRP were measured using chemiluminescence (Roche Diagnostics, Indianapolis, IN). Leptin and adiponectin were measured using a radioactive immunoassay (Linco Research, St. Charles, MO). Homeostatic Model of Insulin Resistance (HOMA-IR) was calculated as glucose × insulin/22.5. As previously reported [34], researchers calculated an overall metabolic risk score by averaging sex-specific internal z-scores for systolic blood pressure, log-transformed triglycerides, waist circumference, inversely-scaled HDL and log-transformed HOMA-IR.

2.1. Sleep Measures

Beginning at age ~9 years, mothers responded to a mailed/online questionnaire about the child's sleep and breathing as well as their own. Questions about the child's sleep included in this analysis were: "In the past month, how often has your child experienced the following during sleep?" which included the subquestions of "Breathing through mouth," "Snoring," "Loud snoring," and "Stopping breathing or gasping or snorting for breath," and a separate question of "In the past month, how often does your child have a problem with sleepiness during the day?" Parents responded on a 4-point scale (never, rarely, sometimes, or often) or that they "didn't know." Answers of "didn't know" were treated as missing variables. Mothers also answered questions about their own sleeping, including "In the past month, how often did you snore?" on a 5-point scale (almost never, occasionally, a few nights a week, most nights, and every night) and "In the past month, on average, how many hours per day did you sleep in a usual 24-hour period? (Answer separately for weekdays and for weekend days)."

2.2. Covariates

Upon entering the study, mothers reported their age, education, race/ethnicity, and smoking history. We defined gestational glucose tolerance in categories according to the results of routine prenatal screening. Atrius clinicians screened all pregnant women at 26–28 weeks of gestation with a non-fasting oral glucose challenge test, in which venous blood was sampled 1 h after a 50 g oral glucose load. If the blood glucose exceeded 140 mg/dL, the clinician referred the woman for a fasting 3-h 100 g oral glucose tolerance test (OGTT). Abnormal OGTT results were a blood glucose >95 mg/dL fasting, >180 mg/dL at 1 h, >155 mg/dL at 2 h, or >140 mg/dL at 3 h. Women with two or more abnormal values on the OGTT were diagnosed with GDM [35]. Researchers calculated pre-pregnancy BMI from mother's self-reported height and pre-pregnancy weight. Maternal weight gain was calculated by last weight measure before delivery minus pre-pregnancy weight. Parents reported child sugar-sweetened beverage (SSB) intake, fast food intake, and TV hours/day watched.

2.3. Statistical Analysis

We performed all analyses using SAS version 9.3 (SAS Institute, Cary NC)·Variables were examined for distribution, and variables not normally distributed were log transformed and denoted as such. Linear regressions (for continuous or ordinal outcomes) were used to assess the relationships between the exposures and outcomes. Successive multivariable models were used to include confounders and potential mediators (listed in the models below), which may interact with the outcomes studied herein. The first model was adjusted for child age and sex, the second included the first model plus maternal age at enrollment, education, and child race/ethnicity, maternal pre-pregnancy BMI, maternal pregnancy weight gain, gestational diabetes, and smoking during pregnancy, and the third model included early teen child intake of sugar-sweetened beverages and fast-food and hours/day of TV watched. These are broken into five models in the Supplementary Tables. Synergistic/interaction models were used to determine whether exposures were additive or independent. We used a 4-category exposure, where yes is any snoring and no is no snoring (yes child – yes mom; yes child – no mom; no child – yes mom; no child – no mom).

3. Results

Overall demographic and metabolic information for children included in the analysis are found in Table 1. The mid-childhood visit occurred at mean (SD) 7.9 (0.8) years, while the early teen visit occurred at 13.3 (1.0) years.

3.1. Cross-sectional Analysis of Sleep and Cardiometabolic Outcomes

Parent-reported child snoring in early adolescence showed strong associations with adiposity measures (BMI, fat mass index measured with DXA, waist circumference, and skinfold tests) that persisted even after adjustment for potential confounders (Table 2). Child snoring was also related to leptin, high-density lipoprotein (HDL) cholesterol levels, HOMA-IR, c-peptide, and metabolic risk score. However, only HDL and metabolic risk scores remained associated with child snoring in multivariable models.

Loud snoring was even more potently correlated with child anthropometry cross-sectionally: e.g. after adjustment for covariates (Model 3), BMI (β 1.10 kg/m^2, 95% CI 0.71, 1.50; p < 0.001), waist circumference (β 3.06 cm, 95% CI 2.02, 4.09; p < 0.001), DXA total fat mass index (β 0.75 kg/m^2, 95% CI 0.42, 1.07; p < 0.001), and DXA trunk fat mass index (0.38 kg/m^2, 95% CI 0.22, 0.53; p < 0.001) were all associated with child loud snoring. Leptin (β 2.71 ng/mL, 95% CI 1.11, 4.30; p < 0.001), HDL (β −1.61 mg/dL, 95% CI −3.20, −0.03; p = 0.05), HOMA-IR (β 0.44, 95% CI 0.13, 0.75; p < 0.01), C-peptide (β 0.14 ng/mL, 95% CI 0.01, 0.26; p = 0.03), and metabolic risk score were correlated with loud snoring, but only metabolic risk score (β 0.10, 95% CI 0.02, 0.18; p = 0.02) remained significantly related to loud snoring after correction for covariates.

After adjustment for covariates, parent-reported child stopping breathing and/or gasping for breath was related to BMI (β, 1.09 kg/m^2, 95% CI 0.16, 2.02; p = 0.02), waist circumference (β, 2.56 cm, 95% CI 0.11, 5.02; p = 0.04), DXA fat mass index (β 0.77 kg/m^2, 95% CI 0.00, 1.54; p = 0.05), and HOMA-IR (β 0.86, 95% CI 0.21, 1.51; p = 0.01). Child daytime sleepiness and breathing through mouth did not significantly relate to child anthropometry or cardiometabolic outcomes cross-sectionally after adjustment for covariates (data not shown).

Cross-sectionally, maternal snoring was related to measures of child adiposity, but these associations substantially attenuated after adjustment for maternal pre-pregnancy BMI (Table 3). Similar results were found with maternal hours of sleep relating to child adiposity measured by BMI, waist circumference, and skinfold but not DXA, and these remained significant when corrected for potential confounders (data not shown). Maternal snoring levels were related to child leptin, insulin, and HOMA-IR levels, but these associations attenuated to null with adjustment for maternal BMI and other characteristics. There were no synergistic or additive effects of child and maternal snoring on cardiometabolic outcomes (data not shown).

3.2. Prospective Analysis of Sleep and Cardiometabolic Outcomes

Prospectively, mid-childhood adiposity exposures, leptin, HOMA-IR, and CRP were related to the outcome of snoring at the early teen visit, even when adjusted for confounders (Table 4). Mid-childhood global metabolic risk score was also related to early teen levels of snoring, but this association attenuated to null when maternal pre-pregnancy BMI was included in the model. However, child adiposity, leptin, and CRP predicted child loud snoring at the early teen visit, and these associations remained when adjusted for covariates (Table 4). On the other hand, child snoring as an exposure at ~9 y predicted changes in some of the adiposity outcomes, even when adjusted for covariates (Table 5). Child's loud snoring as an exposure at the ~9 y timepoint predicted the same adiposity-related outcomes as child snoring, though these associations attenuated to null when adjusted for maternal pre-pregnancy BMI (Table 5). Loud snoring at ~ 9 y predicted changes in HOMA-IR which remained significant when corrected for covariates. Maternal snoring at ~ 9 y predicted adiposity-related changes in the child, though these disappeared when adjusted for maternal pre-pregnancy BMI (data not shown).

4. Discussion

We explored the associations of parent-reported child snoring and mother snoring with cardiometabolic outcomes cross-sectionally and prospectively. We observed that child snoring was related to child adiposity and metabolic risk, while maternal snoring was not related to child cardiometabolic biomarkers cross-sectionally. Prospectively, child adiposity biomarkers in mid-childhood were related to child snoring in early adolescence, while child snoring was also prospectively related to greater increases in adiposity but not cardiometabolic biomarkers during the relatively short time period of this study.

4.1. Cross-sectional Associations Between Sleep and Cardiometabolic Outcomes

In the cross-sectional study, we observe that parent-reported levels of child snoring, and particularly loud snoring, were associated with child adiposity, as we expected. Several studies have found relationships of body fat mass and/or BMI with sleep apnea or sleep-disordered breathing [7–9,23,36–38]. Additionally, we observed metabolic risk scores and HDL were associated with snoring, which support these previous findings of altered lipids and metabolic outcomes in individuals with sleep apnea [15–19]. Snoring did not seem to be significantly associated with other adipokines or cardiometabolic markers cross-sectionally. This indicates that adiposity, including central adiposity, is linked with snoring in children. It remains possible that other metabolic consequences of snoring would develop later in life, and this points to the critical importance of early and targeted therapy of OSA to combat metabolic consequences.

Furthermore, we did not observe cross-sectional associations of maternal snoring with cardiometabolic biomarkers. It appears that unlike previous studies in rodents [24,25], in humans, there is no strong association between maternal sleep outcomes and child metabolic health. These data will need to be confirmed with future prospective studies examining relationships between maternal sleep quality during gestation and offspring metabolic outcomes.

4.2. Prospective Associations Between Sleep and Cardiometabolic Outcomes

Prospectively, we found that mid-childhood adiposity and insulin resistance were related to early adolescent snoring. Snoring was related to changes in adiposity, but not insulin resistance, from mid-childhood to early teen timepoints. It remains possible that over time, OSA, and/or more severe OSA, may eventually lead to worsening insulin resistance. Our findings confirm the existence of a physiologic loop between worsening obesity with worsening sleep apnea, which in turn leads to worsened obesity [39]. However, we found stronger associations of cardiometabolic biomarkers and adiposity with the later development of sleep apnea, suggesting that, in children, early metabolic interventions, such as weight loss, may have more benefit on sleep outcomes than vice versa. These results confirm prior findings in adults of the Wisconsin Sleep Cohort Study, which followed patients prospectively and showed that a 10% increase in weight resulted in a 32% worsening of sleep apnea [40]. Similar results were found in the Cleveland Family Study, which found that BMI, cholesterol, age, and sex were all significantly associated with later sleep-disordered breathing in adults [41]. Further, weight loss improves the symptoms of

sleep apnea in adults, suggesting that the improvement of obesity leads to the improvement of sleep apnea [42,43]. While other studies have found links between OSA and the development of insulin resistance and diabetes in adults [12,13], we do not see a similar change in children and adolescents. This may be due to the relatively young age and/or examination of snoring at around 9 years of age and not at around the mid-childhood visit, which makes it closer in time to the early teen visit.

Regardless, we observe prospective associations that confirm a cycle between worsening obesity/adiposity and worsening OSA [39]. The risk of developing OSA increases with age and BMI [44], likely due to mechanical stress on airways. OSA may influence obesity and cardiometabolic health through other mechanisms, including central (brain) and peripheral (adipocytes, hormonal signaling) tissues [39]. For instance, adipocytes respond to insulin differently after acute sleep deprivation [18], which presents as impaired insulin sensitivity after sleep restriction and/or fragmentation, key features of OSA. Other studies have shown neurodegeneration with hypoxia and sleep fragmentation that result from OSA [45] and manifest in impaired performance on executive/cognitive function, such as inhibitory control, among patients with OSA [46]. Although OSA has not yet been studied in terms of how the brain responds to appetitive cues, sleep deprivation or restriction has been shown to increase the reward-related brain response to high fat or high calorie food cues in patients of normal body weight [47,48], which leads to increased caloric intake and weight gain. These changes may be more subtle in children and adolescents, although our results point to prospective connections between OSA and adiposity as early as adolescence which may be exacerbated in later life and point to the critical need for early intervention.

Limited studies have examined prospective relationships between OSA and cardiometabolic outcomes in pediatric/adolescent populations. In one study, children with the highest levels of sleep-disordered breathing versus those without any OSA showed higher risk of later becoming overweight [20]. In another study, OSA was related to higher diastolic and systolic blood pressure values but not changes in blood pressure after four years [49]. Similarly, children with resolved OSA showed increased baroreflex sensitivity and children with resolved or unresolved OSA demonstrated decreased blood pressure variability after four years [50]. Increased hsCRP, denoting increased inflammation, in childhood was related to later worsened OSA in adolescence in another study [21]. A full prospective evaluation of potential cardiometabolic biomarkers and adiposity with snoring is completed for the first time herein.

4.3. Limitations and Conclusions

This study has some limitations. The data is based on parent-reported measures of sleep quality collected at different timepoints. Other studies have used similar parent-reported measures for child sleep [51,52]. Additionally, there are several analyses being performed the results of which will be used to run power calculations for future, larger studies, to explore these interactions more in-depth. The strength of this cohort is that the data were collected at multiple timepoints and uses primarily investigator-collected data at clinic visits which should not be subject to bias. Furthermore, the sample size is relatively large and allows for the inclusion of several covariates in the analysis to eliminate other potential

confounders. Although we did not collect information on maternal snoring in pregnancy, we are able to account for potential genetic correlations by using the maternal current snoring as a surrogate marker in our analysis. The cross-sectional data is correlative in nature and cannot be used to support causality, but the strength of this paper is that it also includes a prospective analysis which could begin to infer causality. Future larger and longer studies will need to replicate these results, and importantly, future clinical trials are needed to study potential therapies and their benefits on sleep and metabolic outcomes in children and adolescents. There is some evidence that treatment with continuous positive air pressure (CPAP) in adults may improve insulin resistance and cardiometabolic outcomes [53–56], but this needs to be tested in larger samples and with adolescents. This study presents associations between child/adolescent snoring and cardiometabolic health and thus points to the need for early treatment and prevention of sleep apnea and/or obesity in children and adolescents.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Abbreviations

OSA	obstructive sleep apnea
MRI	magnetic resonance imaging
HOMA-IR	homeostatic assessment of insulin resistance
TNF-α	tumor necrosis factor alpha
TNFR2	tumor necrosis factor receptor 2
BMI	body mass index
DXA	dual energy X-ray absorptiometry
FM	fat mass
FFM	fat-free mass
hsCRP	high sensitivity c-reactive protein

HDL	high density lipoprotein
LDL	low density lipoprotein
AHI	apnea-hypopnea index
CPAP	continuous positive air pressure
CI	confidence interval

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Appendix A. Supplementary Data

Supplementary data to this article can be found online at http://dx.doi.org/10.1016/j.metabol.2017.06.008.

Table 1

Participant characteristics (n = 1078). Data shown are means (standard deviation) unless otherwise indicated.

Child characteristics	
Sex, n(%)	
Male	545 (50.6)
Female	533 (49.4)
Race/ethnicity, n(%)	
Black	173 (16.1)
Hispanic	48 (4.5)
Asian	34 (3.2)
White	696 (64.6)
Other	126 (11.7)
Mid-childhood visit	
Age, years	7.9 (0.8)
BMI, kg/m^2	17.2 (2.9)
BMI z-score	0.40 (0.99)
Waist circumference, cm	59.8 (8.1)
SS + TR, mm	19.7 (9.6)
DXA total fat, %	24.5 (6.3)
DXA FMI, kg/m^2	4.4 (1.9)
DXA FFMI, kg/m^2	13.0 (1.4)
DXA trunk FMI, kg/m^2	1.5 (0.8)
Leptin, ng/mL	6.0 (7.3)
Adiponectin, μg/mL	15.2 (8.0)
Cholesterol, mg/dL	160.0 (27.1)
HDL cholesterol, mg/dL	57.1 (13.6)
Triglycerides, mg/dL	57.7 (23.9)
Glucose, mg/dL	95.5 (15.3)
Insulin, uU/ml	7.9 (6.5)
HOMA-IR	1.9 (1.8)
hsCRP, mg/L	1.0 (3.0)
Metabolic risk, z-score	−0.00 (0.60)
Early teen visit	
Age, years	13.2 (0.9)
BMI, kg/m^2	20.9 (4.6)
BMI z-score	0.38 (1.06)
Waist circumference, cm	73.2 (11.6)
SS + TR, mm	28.2 (13.7)
DXA total fat, %	28.6 (7.6)
DXA FMI, kg/m^2	6.3 (3.1)
DXA FFMI, kg/m^2	14.9 (2.1)
DXA trunk FMI, kg/m^2	2.4 (1.5)

Leptin, ng/mL	12.0 (14.2)
Adiponectin, μg/mL	6.4 (2.8)
Cholesterol, mg/dL	156.1 (29.1)
HDL cholesterol, mg/dL	55.0 (13.5)
Triglycerides, mg/dL	70.9 (34.1)
Glucose, mg/dL	93.1 (22.9)
HOMA-IR	3.3 (2.4)
hsCRP, mg/L	0.9 (2.1)
Metabolic risk, z-score	0.00 (0.63)
C-peptide, ng/mL	2.2 (1.1)
Sugar sweetened beverage (SSB) intake, serv/d	0.8 (0.9)
Fast food intake, serv/d	0.7 (1.1)
TV, h/d	2.0 (1.4)
Child snoring (~9 y)	
Snoring, n(%)	
Never	418 (50.2)
Rarely	242 (29.1)
Sometimes	139 (16.7)
Often	33 (4.0)
Loud snoring, n(%)	
Never	694 (83.0)
Rarely	98 (11.7)
Sometimes	35 (4.2)
Often	9 (1.1)
Stop breathing or gasping for breath, n(%)	
Never	792 (97.9)
Rarely	14 (1.7)
Sometimes	2 (0.2)
Often	1 (0.1)
Maternal covariates	
Maternal age, years	32.3 (5.0)
Pre-pregnancy BMI, kg/m^2	24.7 (5.1)
Pregnancy weight gain, kg	15.5 (5.3)
GDM, n(%)	
No	1015 (95.3)
Yes	50 (4.7)
College graduate, n(%)	
No	303 (28.2)
Yes	772 (71.8)
Pregnancy smoking status, n(%)	
Never	765 (71.2)
Former	215 (20.0)
During pregnancy	95 (8.8)

Maternal snoring	
Maternal snoring, n(%)	
Almost never	393 (50.3)
Occasionally	236 (30.2)
A few nights a week	55 (7.0)
Most nights	62 (7.9)
Every night	36 (4.6)
Mom snoring early teen visit, n(%)	
Almost never	401 (47.1)
Occasionally	186 (21.9)
A few nights a week	78 (9.2)
Most nights	95 (11.2)
Every night	91 (10.7)
Early teen snoring	
Snoring, n(%)	
Never	449 (47.8)
Rarely	256 (27.2)
Sometimes	189 (20.1)
Often	46 (4.9)
Loud snoring, n(%)	
Never	782 (80.4)
Rarely	126 (12.9)
Sometimes	47 (4.8)
Often	18 (1.8)
Stop breathing or gasping for breath, n(%)	
Never	890 (96.7)
Rarely	21 (2.3)
Sometimes	7 (0.8)
Often	2 (0.2)
Daytime sleepiness, n(%)	
Never	449 (42.1)
Rarely	421 (39.5)
Sometimes	172 (16.1)
Often	25 (2.3)
Child’s sleep duration, h/d	8.7 (1.0)

hsCRP, high sensitivity c-reactive protein; DXA, dual energy X-ray absorptiometry; FMI, fat mass index; FFMI, fat free mass index; HOMA-IR, homeostatic model assessment of insulin resistance; HDL, high density lipoprotein; SS + TR, subscapular and triceps skinfold.

Table 2

Cross-sectional associations of snoring with cardiometabolic risk markers at the early teen visit.

Outcome	Model 1		Model 2		Model 3	
	β (95% CI)	p-Value	β (95% CI)	p-Value	β (95% CI)	p-Value
Exposure: parent-reported child snoring [a]						
BMI, kg/m^2	1.04 (0.73, 1.34)	<0.0001	0.61 (0.33, 0.88)	<0.0001	0.61 (0.33, 0.89)	<0.0001
BMI z-score	0.21 (0.14, 0.28)	<0.0001	0.13 (0.06, 0.20)	0.0002	0.13 (0.06, 0.20)	0.0002
Waist circumference, cm	2.58 (1.80, 3.35)	<0.0001	1.74 (1.01, 2.47)	<0.0001	1.71 (0.98, 2.44)	<0.0001
SS + TR, mm	2.28 (1.35, 3.21)	<0.0001	1.25 (0.36, 2.13)	0.01	1.17 (0.29, 2.05)	0.01
DXA total fat, %	1.05 (0.44, 1.65)	0.001	0.66 (0.07, 1.25)	0.03	0.71 (0.12, 1.30)	0.02
DXA FMI, kg/m^2	0.71 (0.46, 0.96)	<0.0001	0.46 (0.23, 0.69)	0.0001	0.48 (0.25, 0.71)	<0.0001
DXA FFMI, kg/m^2	0.59 (0.43, 0.76)	<0.0001	0.40 (0.26, 0.55)	<0.0001	0.41 (0.27, 0.56)	<0.0001
DXA trunk FMI, kg/m^2	0.35 (0.23, 0.47)	<0.0001	0.23 (0.12, 0.34)	<0.0001	0.23 (0.12, 0.34)	<0.0001
Leptin, ng/mL	1.62 (0.44, 2.79)	0.01	0.48 (−0.68, 1.63)	0.42	0.59 (−0.59, 1.77)	0.33
Adiponectin, μg/mL	−0.16 (−0.40, 0.08)	0.18	−0.03 (−0.28, 0.21)	0.79	−0.03 (−0.28, 0.22)	0.80
Cholesterol, mg/dL	−2.05 (−4.44, 0.34)	0.09	−2.48 (−4.96, −0.01)	0.05	−2.69 (−5.21, −0.18)	0.04
HDL cholesterol, mg/dL	−1.51 (−2.65, −0.36)	0.01	−1.32 (−2.52, −0.13)	0.03	−1.47 (−2.69, −0.25)	0.02
Triglycerides, mg/dL	1.78 (−0.88, 4.44)	0.19	1.33 (−1.29, 3.94)	0.32	1.80 (−0.85, 4.45)	0.18
Glucose, mg/dL	0.80 (−1.35, 2.95)	0.47	−0.77 (−2.27, 0.72)	0.31	−0.78 (−2.31, 0.75)	0.32
HOMA-IR	0.40 (0.17, 0.62)	0.001	0.18 (−0.04, 0.39)	0.11	0.20 (−0.02, 0.42)	0.07
hsCRP, mg/L (log transformed)	0.15 (0.04, 0.25)	0.01	0.09 (−0.01, 0.20)	0.08	0.09 (−0.01, 0.20)	0.09
Metabolic risk, z-score	0.11 (0.05, 0.16)	0.0002	0.07 (0.02, 0.13)	0.01	0.08 (0.02, 0.14)	0.01

Model 1. Adjusted for child age and sex.

Model 2. Model 1 + maternal age, education, pre-pregnancy body mass index (BMI), pregnancy weight gain, gestational diabetes and smoking during pregnancy and child race/ethnicity.

Model 3. Model 2 + child sugar-sweetened beverage (SSB) and fast food intake and TV h/d.

hsCRP, high sensitivity c-reactive protein; DXA, dual energy X-ray absorptiometry; FMI, fat mass index; FFMI, fat free mass index; HOMA-IR, homeostatic model assessment of insulin resistance; HDL, high density lipoprotein; SS + TR, subscapular and triceps skinfold.

[a]Snoring was treated as a continuous (ordinal) exposure with values ranging from 1 = never to 4 = often.

Table 3

Cross-sectional associations of mother snoring [a] and offspring cardiometabolic risk markers at the early teen visit.

Offspring outcomes	Model 1		Model 2		Model 3	
	β (95% CI)	p-Value	β (95% CI)	p-Value	β (95% CI)	p-Value
BMI, kg/m^2	0.43 (0.22, 0.64)	<0.0001	−0.12 (−0.31, 0.08)	0.25	−0.12 (−0.32, 0.08)	0.26
BMI z-score	0.08 (0.03, 0.13)	0.002	−0.04 (−0.09, 0.01)	0.13	−0.04 (−0.09, 0.01)	0.15
Waist circumference, cm	0.91 (0.37, 1.45)	0.001	−0.38 (−0.90, 0.15)	0.16	−0.42 (−0.95, 0.11)	0.12
SS + TR, mm	1.18 (0.53, 1.83)	0.0004	−0.31 (−0.95, 0.33)	0.34	−0.26 (−0.90, 0.39)	0.44
DXA total fat, %	0.40 (−0.03, 0.83)	0.07	−0.27 (−0.71, 0.16)	0.22	−0.21 (−0.65, 0.24)	0.36
DXA FMI, kg/m^2	0.29 (0.11, 0.46)	0.001	−0.05 (−0.22, 0.11)	0.52	−0.04 (−0.21, 0.13)	0.64
DXA FFMI, kg/m^2	0.27 (0.15, 0.38)	<0.0001	0.03 (−0.08, 0.15)	0.54	0.03 (−0.09, 0.14)	0.65
DXA trunk FMI, kg/m^2	0.15 (0.07, 0.23)	0.0003	−0.01 (−0.09, 0.07)	0.79	−0.01 (−0.09, 0.07)	0.89
Leptin, ng/mL	0.94 (0.17, 1.71)	0.02	−0.17 (−0.97, 0.62)	0.66	−0.16 (−0.97, 0.64)	0.69
Adiponectin, µg/mL	−0.02 (−0.19, 0.14)	0.79	0.15 (−0.03, 0.33)	0.10	0.15 (−0.03, 0.33)	0.10
Cholesterol, mg/dL	−0.64 (−2.39, 1.11)	0.47	−0.69 (−2.60, 1.22)	0.48	−1.21 (−3.17, 0.74)	0.22
HDL cholesterol, mg/dL	−0.45 (−1.22, 0.32)	0.25	−0.06 (−0.90, 0.77)	0.88	0.00 (−0.85, 0.86)	1.00
Triglycerides, mg/dL	0.37 (−1.59, 2.33)	0.71	0.23 (−1.89, 2.35)	0.83	0.05 (−2.09, 2.19)	0.96
Glucose, mg/dL	1.45 (−0.07, 2.96)	0.06	0.50 (−0.58, 1.58)	0.36	0.60 (−0.50, 1.70)	0.29
HOMA-IR	0.27 (0.13, 0.42)	0.0002	0.10 (−0.04, 0.23)	0.18	0.11 (−0.03, 0.25)	0.14
hsCRP, mg/L (log transformed)	0.02 (−0.05, 0.09)	0.55	−0.05 (−0.13, 0.02)	0.17	−0.06 (−0.13, 0.02)	0.17
Metabolic risk, z-score	0.04 (0.00, 0.08)	0.06	−0.01 (−0.05, 0.03)	0.72	−0.01 (−0.05, 0.03)	0.58

Model 1. Adjusted for child age and sex.

Model 2. Model 1 + maternal age, education, pre-pregnancy body mass index (BMI), pregnancy weight gain, gestational diabetes and smoking during pregnancy and child race/ethnicity.

Model 3. Model 2 + child sugar-sweetened beverage (SSB) and fast food intake and TV h/d.

hsCRP, high sensitivity c-reactive protein; DXA, dual energy X-ray absorptiometry; FMI, fat mass index; FFMI, fat free mass index; HOMA-IR, homeostatic model assessment of insulin resistance; HDL, high density lipoprotein; SS + TR, subscapular and triceps skinfold.

[a] Snoring was treated as a continuous (ordinal) exposure with values ranging from 1 = almost never to 5 = every night.

Table 4

Associations of mid-childhood cardiometabolic "exposures" (per IQR) with child snoring at early teen ("continuous" [ordinal 1–4 values]).

	Model 1		Model 2		Model 3	
	β (95% CI)	p-Value	β (95% CI)	p-Value	β (95% CI)	p-Value
Exposures (per IQR)						
Outcome = child snoring [a]						
BMI, kg/m^2	0.18 (0.11, 0.25)	<0.0001	0.12 (0.04, 0.19)	0.002	0.12 (0.05, 0.20)	0.002
BMI z-score	0.18 (0.09, 0.26)	<0.0001	0.10 (0.01, 0.20)	0.03	0.11 (0.01, 0.20)	0.03
Waist circumference, cm	0.17 (0.10, 0.24)	<0.0001	0.12 (0.04, 0.20)	0.002	0.12 (0.04, 0.20)	0.003
SS + TR, mm	0.15 (0.09, 0.21)	<0.0001	0.10 (0.03, 0.17)	0.004	0.10 (0.03, 0.17)	0.01
DXA total fat, %	0.16 (0.06, 0.26)	0.001	0.11 (0.01, 0.21)	0.03	0.12 (0.01, 0.22)	0.03
DXA FMI, kg/m^2	0.17 (0.10, 0.25)	<0.0001	0.12 (0.04, 0.20)	0.003	0.13 (0.05, 0.21)	0.003
DXA FFMI, kg/m^2	0.24 (0.14, 0.33)	<0.0001	0.18 (0.07, 0.28)	0.001	0.20 (0.09, 0.30)	0.0003
DXA trunk FMI, kg/m^2	0.16 (0.09, 0.22)	<0.0001	0.11 (0.04, 0.19)	0.002	0.12 (0.04, 0.19)	0.002
Leptin, ng/mL	0.07 (0.02, 0.11)	0.01	0.05 (0.01, 0.10)	0.03	0.06 (0.01, 0.11)	0.02
Adiponectin, μg/mL	−0.05 (−0.15, 0.06)	0.36	−0.02 (−0.13, 0.09)	0.72	−0.01 (−0.12, 0.09)	0.80
Cholesterol, mg/dL	0.01 (−0.10, 0.12)	0.90	0.01 (−0.10, 0.12)	0.86	0.01 (−0.10, 0.13)	0.83
HDL Cholesterol, mg/dL	−0.04 (−0.15, 0.07)	0.44	−0.04 (−0.15, 0.07)	0.46	−0.04 (−0.15, 0.08)	0.53
Triglycerides, mg/dL	0.00 (−0.09, 0.10)	0.92	0.01 (−0.09, 0.11)	0.82	0.01 (−0.10, 0.11)	0.90
Glucose, mg/dL	0.07 (−0.04, 0.19)	0.20	0.05 (−0.07, 0.17)	0.39	0.04 (−0.08, 0.16)	0.51
HOMA-IR	0.12 (0.05, 0.20)	0.001	0.07 (−0.01, 0.15)	0.07	0.08 (0.00, 0.17)	0.04
hsCRP, mg/L (log transformed)	0.14 (0.03, 0.25)	0.02	0.11 (0.00, 0.22)	0.06	0.12 (0.00, 0.23)	0.04
Metabolic risk, z-score	0.01 (−0.08, 0.09)	0.89	−0.01 (−0.10, 0.08)	0.87	0.04 (−0.05, 0.13)	0.40
Outcome = child loud snoring [a]						
BMI, kg/m^2	0.16 (0.12, 0.21)	<0.0001	0.11 (0.06, 0.16)	<0.0001	0.11 (0.05, 0.16)	<0.0001
BMI z-score	0.16 (0.10, 0.21)	<0.0001	0.09 (0.02, 0.15)	0.01	0.08 (0.02, 0.15)	0.01
Waist circumference, cm	0.17 (0.12, 0.22)	<0.0001	0.12 (0.06, 0.17)	<0.0001	0.11 (0.06, 0.17)	<0.0001
SS + TR, mm	0.14 (0.10, 0.18)	<0.0001	0.09 (0.05, 0.14)	<0.0001	0.09 (0.05, 0.14)	<0.0001
DXA total fat, %	0.17 (0.10, 0.24)	<0.0001	0.13 (0.05, 0.20)	0.001	0.13 (0.05, 0.20)	0.001
DXA FMI, kg/m^2	0.17 (0.11, 0.22)	<0.0001	0.12 (0.07, 0.18)	<0.0001	0.12 (0.06, 0.18)	<0.0001

	Model 1		Model 2		Model 3	
	β (95% CI)	p-Value	β (95% CI)	p-Value	β (95% CI)	p-Value
DXA FFMI, kg/m^2	0.18 (0.12, 0.25)	<0.0001	0.12 (0.04, 0.19)	0.002	0.12 (0.04, 0.19)	0.003
DXA trunk FMI, kg/m^2	0.15 (0.11, 0.20)	<0.0001	0.12 (0.06, 0.17)	<0.0001	0.12 (0.06, 0.17)	<0.0001
Leptin, ng/mL	0.06 (0.02, 0.09)	0.001	0.04 (0.00, 0.07)	0.03	0.04 (0.00, 0.07)	0.03
Adiponectin, μg/mL	−0.06 (−0.14, 0.01)	0.10	−0.03 (−0.10, 0.04)	0.43	−0.03 (−0.11, 0.05)	0.44
Cholesterol, mg/dL	0.01 (−0.07, 0.09)	0.80	0.02 (−0.06, 0.10)	0.58	0.03 (−0.06, 0.11)	0.54
HDL cholesterol, mg/dL	−0.03 (−0.11, 0.05)	0.42	−0.03 (−0.11, 0.04)	0.39	−0.03 (−0.11, 0.05)	0.40
Triglycerides, mg/dL	0.02 (−0.05, 0.09)	0.56	0.03 (−0.04, 0.11)	0.35	0.04 (−0.04, 0.11)	0.34
Glucose, mg/dL	0.05 (−0.03, 0.13)	0.22	0.02 (−0.07, 0.10)	0.68	0.02 (−0.07, 0.10)	0.69
HOMA-IR	0.09 (0.04, 0.15)	0.001	0.04 (−0.01, 0.10)	0.15	0.05 (−0.01, 0.11)	0.11
hsCRP, mg/L (log transformed)	0.12 (0.04, 0.20)	0.002	0.08 (0.00, 0.16)	0.05	0.09 (0.00, 0.17)	0.04
Metabolic risk, z-score	0.01 (−0.10, 0.12)	0.88	−0.02 (−0.15, 0.11)	0.77	0.02 (−0.11, 0.14)	0.81

Model 1. Adjusted for child age and sex.

Model 2. Model 1 + maternal age, education, pre-pregnancy body mass index (BMI), pregnancy weight gain, gestational diabetes and smoking during pregnancy and child race/ethnicity.

Model 3. Model 2 + child sugar-sweetened beverage (SSB) and fast food intake and TV h/d.

hsCRP, high sensitivity c-reactive protein; DXA, dual energy X-ray absorptiometry; FMI, fat mass index; FFMI, fat free mass index; HOMA-IR, homeostatic model assessment of insulin resistance; HDL, high density lipoprotein; SS + TR, subscapular and triceps skinfold.

[a]Snoring was treated as a continuous (ordinal) exposure with values ranging from 1 = never to 4 = often.

Table 5

Associations of parent-reported child snoring and loud snoring (~9 y) with change in outcomes from mid-childhood to early teen.

Outcome = change mid-childhood to early teen	Model 1		Model 2		Model 3	
	β (95% CI)	p-Value	β (95% CI)	p-Value	β (95% CI)	p-Value
Exposure: parent-reported child snoring ~9 y [a]						
BMI, kg/m^2	0.43 (0.22, 0.63)	<0.0001	0.26 (0.06, 0.45)	0.01	0.26 (0.06, 0.46)	0.01
BMI z-score	0.02 (−0.03, 0.07)	0.36	0.01 (−0.04, 0.06)	0.80	0.01 (−0.04, 0.06)	0.77
Waist circumference, cm	1.03 (0.43, 1.64)	0.001	0.77 (0.17, 1.37)	0.01	0.76 (0.16, 1.37)	0.01
SS + TR, mm	0.51 (−0.25, 1.28)	0.19	0.12 (−0.64, 0.88)	0.76	0.07 (−0.68, 0.83)	0.85
DXA total fat, %	0.11 (−0.35, 0.58)	0.63	0.00 (−0.48, 0.48)	1.00	0.05 (−0.43, 0.53)	0.84
DXA FMI, kg/m2	0.30 (0.12, 0.48)	0.001	0.20 (0.03, 0.38)	0.02	0.20 (0.03, 0.38)	0.02
DXA FFMI, kg/m2	0.29 (0.18, 0.41)	<0.0001	0.22 (0.10, 0.33)	0.00	0.20 (0.09, 0.32)	0.00
DXA trunk FMI, kg/m2	0.15 (0.06, 0.24)	0.001	0.10 (0.02, 0.19)	0.02	0.10 (0.01, 0.19)	0.02
Leptin, ng/mL	1.35 (−0.07, 2.77)	0.06	0.94 (−0.52, 2.41)	0.20	1.19 (−0.32, 2.69)	0.12
Adiponectin, μg/mL	0.14 (−0.69, 0.96)	0.75	0.18 (−0.69, 1.04)	0.69	0.09 (−0.81, 1.00)	0.84
Chol, mg/dL	−2.76 (−5.95, 0.43)	0.09	−2.97 (−6.25, 0.31)	0.08	−3.07 (−6.40, 0.27)	0.07
HDLC, mg/dL	−0.64 (−2.08, 0.81)	0.39	−0.40 (−1.90, 1.10)	0.60	−0.81 (−2.30, 0.68)	0.29
Trig, mg/dL	−1.58 (−5.26, 2.09)	0.40	−1.68 (−5.55, 2.18)	0.39	−0.44 (−4.38, 3.50)	0.83
Glucose, mg/dL	−0.61 (−3.29, 2.06)	0.65	−1.02 (−3.84, 1.81)	0.48	−1.51 (−4.35, 1.34)	0.30
HOMA-IR	0.05 (−0.33, 0.43)	0.79	−0.05 (−0.44, 0.34)	0.80	0.08 (−0.32, 0.48)	0.70
hsCRP, mg/L (log transformed)	−0.12 (−0.32, 0.08)	0.25	−0.10 (−0.30, 0.11)	0.36	−0.09 (−0.30, 0.12)	0.42
Metabolic risk, z-score	−0.03 (−0.11, 0.05)	0.50	−0.04 (−0.12, 0.05)	0.41	0.01 (−0.07, 0.10)	0.80
Exposure: parent-reported child loud snoring ~9 y [a]						
BMI, kg/m^2	0.48 (0.18, 0.78)	0.002	0.15 (−0.14, 0.44)	0.31	0.10 (−0.20, 0.39)	0.52
BMI z-score	−0.02 (−0.09, 0.06)	0.68	−0.04 (−0.12, 0.03)	0.28	−0.06 (−0.13, 0.02)	0.16
Waist circumference, cm	0.87 (−0.02, 1.76)	0.06	0.35 (−0.55, 1.24)	0.45	0.15 (−0.77, 1.06)	0.75
SS + TR, mm	0.64 (−0.48, 1.76)	0.26	−0.09 (−1.23, 1.04)	0.87	−0.44 (−1.57, 0.69)	0.45
DXA total fat, %	0.38 (−0.30, 1.06)	0.28	0.16 (−0.56, 0.87)	0.67	0.01 (−0.71, 0.74)	0.97
DXA FMI, kg/m2	0.49 (0.23, 0.76)	0.0003	0.27 (0.01, 0.53)	0.04	0.21 (−0.06, 0.47)	0.12
DXA FFMI, kg/m2	0.32 (0.15, 0.49)	0.0003	0.13 (−0.04, 0.30)	0.12	0.09 (−0.08, 0.27)	0.29

Outcome = change mid-childhood to early teen	Model 1		Model 2		Model 3	
	β (95% CI)	p-Value	β (95% CI)	p-Value	β (95% CI)	p-Value
DXA trunk FMI, kg/m2	0.24 (0.11, 0.37)	0.0003	0.14 (0.01, 0.27)	0.04	0.10 (−0.03, 0.23)	0.13
Leptin, ng/mL	0.31 (−1.76, 2.38)	0.77	−0.68 (−2.86, 1.51)	0.54	−0.70 (−2.95, 1.55)	0.54
Adiponectin, µg/mL	0.79 (−0.40, 1.98)	0.19	0.80 (−0.49, 2.10)	0.22	0.78 (−0.56, 2.12)	0.25
Chol, mg/dL	−0.92 (−5.47, 3.62)	0.69	−1.74 (−6.51, 3.02)	0.47	−1.58 (−6.46, 3.31)	0.53
HDLC, mg/dL	0.15 (−1.90, 2.20)	0.89	0.79 (−1.38, 2.97)	0.47	0.83 (−1.35, 3.00)	0.46
Trig, mg/dL	−2.15 (−7.37, 3.07)	0.42	−2.51 (−8.11, 3.09)	0.38	−1.78 (−7.51, 3.96)	0.54
Glucose, mg/dL	−1.15 (−4.94, 2.64)	0.55	−0.77 (−4.90, 3.37)	0.72	−1.00 (−5.14, 3.13)	0.63
HOMA-IR	0.60 (0.08, 1.12)	0.02	0.54 (−0.02, 1.09)	0.06	0.69 (0.13, 1.26)	0.02
hsCRP, mg/L (log transformed)	−0.18 (−0.47, 0.11)	0.22	−0.11 (−0.41, 0.20)	0.48	−0.16 (−0.47, 0.15)	0.31
Metabolic risk, z-score	−0.01 (−0.12, 0.11)	0.93	−0.02 (−0.14, 0.10)	0.75	0.03 (−0.10, 0.15)	0.68

Model 1. Adjusted for child age and sex.

Model 2. Model 1 + maternal age, education, pre-pregnancy body mass index (BMI), pregnancy weight gain, gestational diabetes and smoking during pregnancy and child race/ethnicity.

Model 3. Model 2 + child sugar-sweetened beverage (SSB) and fast food intake and TV h/d.

hsCRP, high sensitivity c-reactive protein; DXA, dual energy X-ray absorptiometry; FMI, fat mass index; FFMI, fat free mass index; HOMA-IR, homeostatic model assessment of insulin resistance; HDL, high density lipoprotein; SS + TR, subscapular and triceps skinfold.

[a]Snoring was treated as a continuous (ordinal) exposure with values ranging from 1 = never to 4 = often.