Obstructive Sleep Apnea in Obese Adolescents and Cardiometabolic Risk Markers

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Abstract

Background—Pediatric studies examining the association between obstructive sleep apnea (OSA) and insulin sensitivity/cardiometabolic risk are limited and conflicting.

Objective—To determine if cardiometabolic risk markers are increased among obese youth with obstructive sleep apnea as compared with their equally obese peers without OSA.

Methods—We performed a retrospective analysis of 96 patients (age 14.2 ± 1.4 years) who underwent polysomnography for suspected OSA. Fasting lipids, glucose, insulin, and hemoglobin A_1c (HbA_1c) were performed as part of routine clinical evaluation. Patients were categorized into two groups by degree of OSA as measured by the apnea hypopnea index (AHI): none or mild OSA (AHI < 5) and moderate or severe OSA (AHI ≥ 5).

Results—Despite similar degrees of obesity, patients with moderate or severe OSA had higher fasting insulin ($p = 0.037$) and homeostasis model assessment-insulin resistance [HOMA-IR ($p = 0.0497$)], as compared with those with mild or no OSA. After controlling for body mass index, there was a positive association between the AHI and log HOMA-IR ($p = 0.005$). There was a positive relationship between arousals plus awakenings during the polysomnography and fasting triglycerides.

Conclusions—OSA is linked with greater cardiometabolic risk markers in obese youth.

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Introduction

Obstructive sleep apnea (OSA) is associated with obesity in children and adolescents, and the degree of OSA is positively related to levels of visceral fat (1–4). In adults, OSA is linked not only to obesity and insulin resistance, but also to a high prevalence of cardiovascular disease (5, 6) and type 2 diabetes (7–9). However, pediatric studies examining the association between OSA and measures of cardiometabolic risk are very limited and conflicting. Some studies have shown a negative relationship between the degree of OSA and fasting markers of insulin sensitivity [higher fasting insulin levels and homeostasis model assessment-insulin resistance (HOMA-IR)] (10, 11), while others have shown no association between OSA and these measures or with hemoglobin A_1c (HbA_1c) among normal weight and obese children (12–14). Nevertheless, increasing awareness of the association between OSA and cardiometabolic disease and type 2 diabetes risk in adults has led to more obese youth being referred for sleep evaluation.

To evaluate the hypothesis that OSA is associated with decreased fasting insulin sensitivity and increased cardiometabolic risk markers in obese youth, we performed a retrospective analysis of clinical data from patients referred for evaluation of suspected obesity-related OSA. Specific aims included determining the associations between OSA measured during overnight polysomnography (PSG) and 1) fasting markers of insulin sensitivity (fasting insulin and HOMA-IR), and 2) cardiovascular risk factors (fasting lipid profile, blood pressure) performed during clinical evaluation.

Methods

The study was approved by the Indiana University Institutional Review Board. Clinical data were obtained from medical records of patients who presented for evaluation and treatment of obesity at the Pediatric Overweight Education and Research (POWER) Program at Riley Hospital for Children at Indiana University Health between January 2009 and November 2011 and were subsequently referred for PSG. The POWER Program is a referral clinic that treats pediatric patients who are obese [body mass index (BMI) 95% for age and sex; BMI 85% with complications associated with obesity]. Routine assessment at the initial visit consisted of history and physical examination, anthropometric measures, as well as laboratory evaluation including fasting lipids, liver enzymes, glucose, insulin, and HbA_1c. Criteria warranting referral for PSG included parental concern of OSA or complaints of snoring accompanied by excessive daytime sleepiness, morning headache, and/or behavioral problems suspected to be related to sleep disruption. Tonsillar or adenoid hypertrophy, neuromuscular disease, and craniofacial abnormalities were not noted. One patient was referred to otolaryngology after an abnormal sleep study. Because pre-pubertal children have different metabolic profiles compared with adolescent children, we limited the analysis to patients aged 12 to 16 years to minimize the number of pre-pubertal adolescents included

in the analyses, as Tanner staging was not uniformly available. The final analysis was based on data from 96 patients, all of whom were obese and had complete sleep data. Fasting glucose was missing for 3 patients.

Patients had overnight PSG performed under the direction of the Riley Hospital for Children at IU Health Sleep Disorders Center using the American Academy of Sleep Medicine (AASM) guidelines for sleep assessment in children and adolescents. The PSGs were performed as part of a clinical evaluation; they were interpreted by one of three sleep medicine physicians. PSG data were recorded using the Sandman Elite 9.1 sleep diagnostic software, and applying the following EEG montage: F3M2, F4M1, C3M2, C4M1, O2M1, O1M2, L-EOG, R-EOG, chin EMG, limb EMG, and the following cardiorespiratory parameters: SpO2 and pulse (Masimo), ETCO2 (Microstream NPB 70 and Capnograph Sleep by BCI), nasal pressure, airflow (nasal or oral thermistor), thoracic and abdominal excursion (uncalibrated respirator inductance plethysmography), pulse and ECG. The apnea-hypopnea index (AHI), which is the total number of obstructive apnea and hypopnea events per one hour of sleep, was calculated following the AASM manual for scoring guidelines (15).

Laboratory measures were performed in the Indiana University Health pathology lab. Plasma glucose was measured by the Beckman Coulter DXC 800 using the glucose hexokinase method (CV 2%). HbA_1c was quantified by the Tosoh G7 ion exchange column using the high-performance liquid chromatography method (CV 0.3%). Plasma insulin was determined by the Beckman Coulter DXI 800 using a chemiluminescent sandwich assay (CV 6%). Fasting lipid profile was measured using the Beckman Coulter DXC 800. Homeostasis model assessment of insulin resistance (HOMA-IR) was calculated to express basal insulin resistance (16).

Patient characteristics were summarized using sample mean and standard deviation for continuous variables, and frequency and proportion for categorical variables. We dichotomized the patients into AHI classes: none or mild OSA (AHI < 5) and moderate or severe OSA (AHI 5) based on clinically accepted cut-offs for significant OSA. Two sample t-tests were used to compare normally distributed continuous variables between AHI groups and Mann-Whitney U tests were used for non-normally distributed continuous variables. For comparison of categorical variables, Pearson's Chi-squared test or Fisher's exact test was used. Spearman correlation coefficients were calculated between sleep parameters and cardiometabolic measures. Linear regression models were used to assess the associations between AHI and other measures of sleep disruption (arousals, awakenings, S_aO_2, ETCO2) and glucose, insulin, and lipid measures while adjusting for the effects of gender, race, age, and BMI. Natural logarithmic transformation was performed on HOMA-IR, fasting insulin, and triglycerides to accommodate the right skewness of the observed data. Linear regression models were used to assess the interactions between the AHI and gender, race, age, and BMI. R software was used to perform all statistical analysis. P values less than 0.05 were considered statistically significant.

Results

Characteristics of the study population are shown in Table 1. Of the 96 patients aged 12–16 years who reported symptoms associated with OSA, 80 (83.3%) had either mild or no OSA on overnight PSG and 16 patients (16.7%) had moderate or severe OSA (AHI 5). The study included 3 patients with a diagnosis of diabetes, 2 of whom were being treated (1 each with insulin and metformin). There were 27 patients found to have impaired fasting glucose by American Diabetes Association criteria. While no patients were on medications for hypertension or hyperlipidemia, 1 patient had a prior diagnosis of hypertension and 3 had a diagnosis of hyperlipidemia. In the group with the lowest AHIs, 60 (62.5%) had no OSA indicated by an AHI <1.5 and 20 (20.8%) had mild OSA indicated by an AHI between 1.5 and 4.9. Nine patients (9.4%) had moderate OSA (AHI 5 – 9.9) and 7 (7.3%) had more severe OSA (AHI > 9.9). Significantly more males had either moderate or severe OSA as compared with females ($p = 0.02$), despite similar degrees of obesity. There were no significant racial differences between the groups. Blood pressure, BMI, and HbA$_1$c did not differ between the 2 groups. There were significant between-group differences in fasting insulin ($p = 0.016$, Figure 1), HOMA-IR ($p = 0.038$, Figure 1), and triglycerides ($p = 0.016$) with higher values seen in those with moderate or severe OSA. No such association was seen with cholesterol, hemoglobin, or liver transaminases (AST/ALT).

Spearman correlation analysis showed a significant relationship between AHI and insulin / HOMA-IR ($r = 0.28$, $p = 0.006$/$r = 0.26$, $p = 0.01$) and between triglyceride levels and arousals plus awakenings ($r = 0.26$, $p = 0.01$). However, after correcting for BMI, race, and age the only significant association was between triglyceride levels and arousals plus awakenings (regression coefficient = 0.23, $p = 0.03$). After controlling for the effects of BMI, a significant positive association existed between the AHI and log HOMA-IR ($p = 0.004$, R^2 with AHI = 0.19, R^2 without AHI = 0.12, Table 2). Similarly, higher AHI was associated with higher fasting insulin levels ($p = 0.001$, R^2 with AHI = 0.2, R^2 without AHI = 0.1, Table 3). The association of AHI with log HOMA-IR scores was greatest among Hispanic patients ($p = 0.043$).

Discussion

In this retrospective study of patients undergoing evaluation in a clinical pediatric obesity program, we found that obese youth (aged 12 –16 years) with greater degrees of OSA characterized by higher AHI, have lower fasting insulin sensitivity (higher fasting insulin and HOMA-IR values) compared with equally obese peers with mild or no OSA. After controlling for confounding factors, including BMI, the degree of OSA was significantly positively associated with both fasting insulin and HOMA-IR levels. We also found an association between triglyceride levels and arousals plus awakenings while controlling for BMI, age, and race. This finding is consistent with the elevated triglyceride levels that can be seen with lower insulin sensitivity (17).

As has been previously reported, males were more likely than females to have significant OSA (18); however the degree of obesity was not significantly related to the degree of OSA in males. Associations between OSA and markers of cardiometabolic risk appear strongest

among Hispanic male youth; however, the number of Hispanic youth in the study was very limited and so further study will be needed to confirm this finding. Overall, these findings support our hypothesis that OSA is associated with increased insulin resistance in obese non-diabetic youth.

This is one of few studies addressing race/ethnicity differences in obesity-related OSA, and is the only pediatric study we are aware of to compare race-related differences in OSA and obesity associated cardiometabolic risk markers. Our study population included white, black, and Hispanic youth who were referred for sleep evaluation due to symptoms of OSA (parental concern of OSA or complaints of snoring together with excessive daytime sleepiness, morning headache, and/or behavioral problems suspected to be related to sleep disruption). Overall, the percentage of these youth found to have OSA on overnight PSG was small. This is consistent with prior findings that symptoms, such as daytime somnolence and snoring, do not reliably identify patients with OSA in this population (19). The majority of the study population was black or white and there were no differences in the prevalence of OSA between these two races, nor was there a race effect on the association between HOMA-IR or fasting insulin and AHI in blacks or whites. However 29% of all males (11 of 38) and 40% of Hispanic males (2 of 5) who reported symptoms of impaired sleep and completed overnight PSG had either moderate or severe OSA. This suggests that obese male youth may be at increased risk for OSA as compared with obese female youth. We did not evaluate for PCOS, which has been shown to be associated with OSA in females (20). Our data is in agreement with the Multi-Ethnic Study of Atherosclerosis performed in adults, which collected self-reported sleep measures and found that Hispanics had a higher rate of OSA than whites (either prior diagnosis of OSA or symptoms 3 or more nights a week) (21). A prior family study to evaluate sleep disordered breathing in blacks versus whites did include children as young as 2 years of age and found that black participants <25 years old had more apneic events as compared with whites, though the clinical significance of this finding is not known (22). Our findings indicate that further work is needed to address race/ethnicity determinants of OSA and associated cardiometabolic risk in youth, especially among males.

While other studies in children with varying BMIs undergoing sleep evaluation have shown obesity to be the primary determinant of insulin sensitivity rather than the degree of OSA (11, 13), studies in adults have shown OSA to be associated with insulin resistance, independent of BMI (7). A strength of our study is the inclusion of only obese youth. While there is clearly an association between BMI and OSA in the pediatric population as a whole, there were no such associations in this study population because of the severity of obesity in the population. Because there were no between group differences in BMI according to level of OSA (mild and moderate or severe), this allowed for the examination of associations between the degree of OSA and cardiometabolic risk factors without the confounding associations between obesity and OSA. As expected, BMI was associated with fasting insulin and HOMA-IR, thus we controlled for BMI when evaluating associations between the AHI and measures of fasting insulin sensitivity and cardiovascular risk. Previous studies performed in youth have shown an association between the AHI and HOMA-IR (10); while this relationship has not been significant in other pediatric studies (13, 14). This is likely because of smaller numbers of patients and the inclusion of non-obese patients, as the degree

of obesity is a significant confounder. Our findings add to the evidence that there is a significant association between OSA, fasting insulin sensitivity and cardiometabolic risk markers not only among obese adults but in the obese adolescent population as well. While prior studies in adult populations have also shown a strong association between OSA and type 2 diabetes (9, 23), there are no published pediatric studies reporting whether or not type 2 diabetes and OSA are linked in youth. Our findings indicate that this is an area deserving of further research to determine whether or not similar associations exist in youth.

There are limitations associated with this study that must be noted. Because of the retrospective, clinical nature of the current study, it is not possible to draw conclusions with regard to mechanistic pathways for the relationship between the degree of OSA and cardiometabolic risk factors. Moreover, there were no uniform measures of glucose tolerance other than fasting laboratory values utilized for clinical purposes available for analysis. Further prospective studies including more sensitive measures of glucose tolerance, insulin sensitivity, markers of inflammation and measurements of visceral adiposity will allow for mechanistic questions to be addressed. While no patients were noted to have tonsillar or adenoid hypertrophy, neuromuscular disease, or other craniofacial abnormalities associated with obstructive sleep apnea, prospective work would allow exclusion of patients with these potential confounding conditions. Because of the lack of uniform documentation of pubertal staging from the clinical records, we used age as a surrogate marker of pubertal status and limited the analysis to patients who were between 12 and 16 years of age. Due to the differences in glucose metabolism in pre-pubertal and adolescent children, future prospective work should take pubertal status into account. Finally, because of referral bias associated with the study of specialty clinic patients, the results cannot be generalized to the broader pediatric population without further study.

In conclusion, obese adolescents with OSA characterized by higher arousals, awakenings, and AHI during PSG show evidence of worse fasting cardiometabolic profiles, including higher triglyceride, fasting insulin and HOMA-IR levels, compared with their equally obese peers who do not have clinically significant OSA. A preliminary finding that deserves more study is that fasting insulin and HOMA-IR may be especially linked with OSA among obese male Hispanic adolescents, suggesting that indications for further evaluation for symptoms of OSA not only vary by gender but also possibly by race/ethnicity. From a clinical perspective, the relationship between OSA and cardiometabolic abnormalities in obese adolescents should be considered when evaluating patients found to have OSA. From a research perspective, these relationships should be investigated further to determine physiologic mechanisms and if youth with impaired glucose tolerance or type 2 diabetes are at greater risk for OSA.

Acknowledgments

Abbreviations

OSA	obstructive sleep apnea
HOMA-IR	homeostasis model assessment-insulin resistance
HbA$_1$c	hemoglobin A$_1$c
PSG	polysomnography
BMI	body mass index
AHI	apnea hypopnea index

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What is already known about this subject

- In pediatric patients, obstructive sleep apnea is associated with adiposity, especially visceral adiposity.

- In adults, obstructive sleep apnea is also associated with a higher prevalence of cardiovascular disease and type 2 diabetes.

- There are limited and conflicting pediatric studies examining the association between obstructive sleep apnea and biomarkers of risk for cardiovascular disease and type 2 diabetes in youth.

What this study adds

- Obstructive sleep apnea is linked with greater cardiometabolic risk markers in obese adolescents.

- Fasting insulin and homeostasis model assessment-insulin resistance may be especially linked with obstructive sleep apnea among obese male Hispanic adolescents.

- The relationship between obstructive sleep apnea and cardiometabolic abnormalities in obese adolescents should be considered when evaluating patients found to have obstructive sleep apnea.
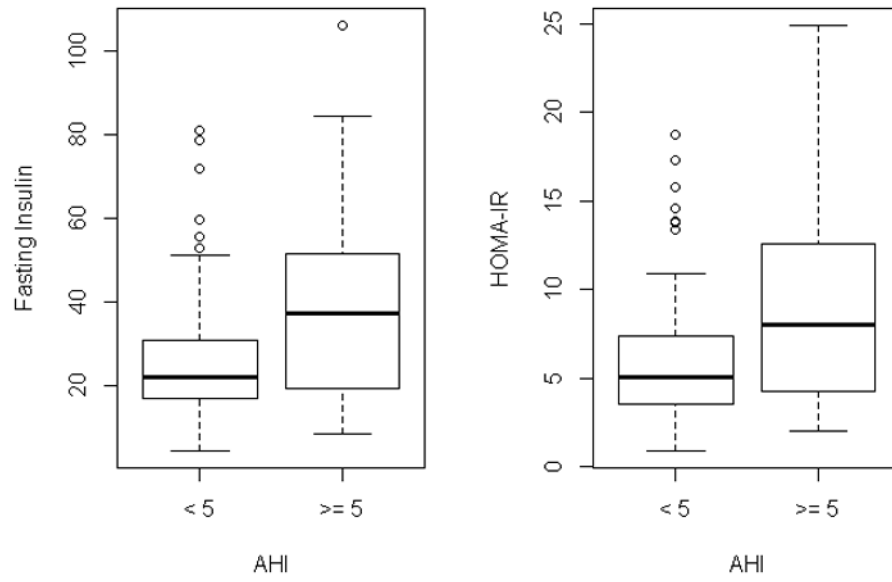
Figure 1.
Mean levels of homeostasis model assessment-insulin resistance (HOMA-IR) score and fasting insulin with 95% confidence intervals for the two apnea-hypopnea index (AHI) groups.

Table 1

Demographic and clinical characteristics of study subjects by AHI classification

	AHI <5 (n = 80)	AHI 5 (n = 16)	p-value
Gender			0.020*
Male	27 (34%)	11 (7%)	
Female	53 (66%)	5 (3%)	
Race			0.44
White	33 (41%)	6 (38%)	
Black	40 (50%)	7 (44%)	
Hispanic	6 (8%)	2 (12%)	
Other	1 (1%)	1 (6%)	
Age (years)	14.20 (1.45)	14.23 (1.34)	0.93
Age distribution			
Min	12.00	12.25	
Q1	13.04	13.08	
Median	13.99	14.17	
Q3	15.25	15.02	
Max	16.98	16.67	
SBP (mmHg)	115.9 (12.4)	115.7 (17.0)	0.96
DBP (mmHg)	70.8 (9.6)	71.9 (10.9)	0.70
BMI (kg/m^2)	40.1 (8.2)	40 (7.7)	0.96
BMI distribution			
Min	28.2	29.6	
Q1	34.6	34.5	
Median	38.3	38.7	
Q3	44	43.4	
Max	66.5	55.2	
BMI SDS	2.6 (0.3)	2.6 (0.3)	0.52
BMI percentile	99.3 (0.7)	99.4 (0.5)	0.49
Fasting glucose (mg/dl)	93.7 (9.6)	93.1 (16.7)	0.89
HbA1C (%)	5.62 (0.38)	5.75 (0.37)	0.23
Fasting insulin	25.86 (15.35)	41.65 (26.98)	0.016*
Fasting Insulin distribution			
Min	4.32	8.28	
Q1	16.67	19.32	
Median	21.97	37.14	
Q3	30.71	50.72	
Max	81.00	106.20	
HOMA-IR	6.01 (3.70)	9.68 (6.73)	0.038*
HOMA-IR distribution			

	AHI <5 (n = 80)	AHI ≥ 5 (n = 16)	p-value
Min	0.94	2.02	
Q1	3.55	4.39	
Median	5.09	8.05	
Q3	7.16	12.41	
Max	18.78	24.88	
Fasting HDL (mg/dl)	38.3 (8.5)	35.6 (10.2)	0.33
Fasting LDL (mg/dl)	97.4 (26.0)	101.7 (22.9)	0.52
Cholesterol (mg/dl)	158.2 (31.5)	170.7 (32.4)	0.19
Triglycerides (mg/dl)	114.1 (85.5)	155.9 (87.7)	0.016*
Triglycerides distribution			
Min	35.0	65.0	
Q1	59.0	105.2	
Median	88.5	120.5	
Q3	134.8	185.0	
Max	627.0	397.0	
Non-HDL Cholesterol (mg/dl)	119.7 (31.6)	134.7 (30.2)	0.10
AST (Units/L)	24.2 (8.8)	23.8 (5.8)	0.80
ALT (Units/L)	24.7 (14.3)	26.2 (14.5)	0.71
Hg (gm/dL)	13.29 (1.23)	13.28 (1.16)	0.97
Arousals	41.1 (27.0)	70.9 (64.0)	0.086
Awakenings	9.4 (6.9)	13.6 (8.5)	0.085
Obstructive Apneas	2.7 (2.8)	15.9 (17.7)	<0.001*
Obstructive Apneas distribution			
Min	0.0	1.0	
Q1	1.0	3.5	
Median	2.0	12.0	
Q3	4.0	18.5	
Max	18.0	71.0	
Hypopneas	2.1 (6.0)	19.5 (25.5)	0.028*
Hypopneas distribution			
Min	0.0	0.0	
Q1	0.0	0.0	
Median	0.0	0.0	
Q3	1.0	38.5	
Max	31.0	67.0	
ETCO2 mean	42.9 (5.0)	42.8 (2.5)	0.96
ETCO2 max	51.3 (4.6)	53.2 (3.0)	0.0496*

AHI = Apnea-hypopnea index, SBP = systolic blood pressure, DBP = diastolic blood pressure, BMI = body mass index, HbA$_1$c = hemoglobin A$_1$c, HOMA-IR = homeostasis model assessment of insulin resistance, HDL = high-density lipoprotein, LDL = low-density lipoprotein, AST = aspartate aminotransferase, ALT = alanine aminotransferase, ETCO2 = end-tidal carbon dioxide concentration

Table 2

Estimated AHI effect on log HOMA-IR adjusted for sex, race, age, and BMI-Z score

	Coefficient Estimate	95% CI	p-value
AHI	0.033	(0.011, 0.055)	0.004*
Female sex	0.207	(−0.045, 0.460)	0.107
Black race	0.013	(−0.234, 0.261)	0.915
Hispanic Race	0.485	(0.043, 0.928)	0.032*
Other Race	0.435	(−0.377, 1.248)	0.290
Age	−0.011	(−0.097, 0.074)	0.795
BMI-Z	0.600	(0.197, 1.002)	0.004*

AHI = Apnea Hypopnea Index, BMI = Body Mass Index

Table 3

Estimated AHI effect on log fasting insulin level adjusted for sex, race, age, and BMI-Z score

	Coefficient Estimate	95% CI	p-value
AHI	0.035	(0.014, 0.056)	0.001*
Female sex	0.242	(0.0004, 0.484)	0.0496*
Black race	−0.011	(−0.248, 0.227)	0.930
Hispanic Race	0.436	(0.013, 0.860)	0.044*
Other Race	0.363	(−0.415, 1.141)	0.356
Age	−0.007	(−0.089, 0.075)	0.873
BMI-Z	0.539	(0.154, 0.923)	0.007*

AHI = Apnea-Hypopnea Index, BMI = Body Mass Index