Obstructive Sleep Apnea Syndrome is Associated with Metabolic Syndrome among Adolescents and Youth in Beijing: Data from Beijing Child and Adolescent Metabolic Syndrome Study

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Abstract

Background: Obstructive sleep apnea (OSA) syndrome has a negative impact on the health of millions of adolescents and youth. The aim of this study was to evaluate the associations of OSA syndrome with obesity and cardiometabolic risk factors among adolescents and youth at risk for metabolic syndrome (MS).

Methods: A total of 558 subjects aged 14–28 years were recruited from the Beijing Child and Adolescent Metabolic Syndrome Study. Each underwent a 2-h oral glucose tolerance test (OGTT), echocardiography, and liver ultrasonography. Anthropometric measures, blood levels of glucose, lipids, and liver enzymes were assessed. Subjects with high or low risk for OSA were identified by Berlin Questionnaire (BQ).

Results: Among the subjects in obesity, 33.7% of whom were likely to have OSA by BQ. Subjects with high risk for OSA had higher neck and waist circumference and fat mass percentage compared to those with low risk for OSA ($P < 0.001$). Moreover, significant differences in levels of lipids, glucose after OGTT, and liver enzymes, as well as echocardiographic parameters were found between the two groups with high or low risk for OSA ($P < 0.05$). The rates of nonalcoholic fatty liver disease (71.0% vs. 24.2%), MS (38.9% vs. 7.0%), and its components in high-risk group were significantly higher than in low-risk group.

Conclusions: The prevalence of OSA by BQ was high in obese adolescents and youth. A high risk for OSA indicates a high cardiometabolic risk. Mechanisms mediating the observed associations require further investigation.

Key words: Adolescents; Cardiometabolic Risk Factors; Obesity; Obstructive Sleep Apnea Syndrome; Youth

INTRODUCTION

The prevalence of obesity in adolescents and youth has increased rapidly in recent years.[1,2] Obesity is a known risk factor for major chronic diseases such as diabetes, hypertension, metabolic syndrome (MS), and obstructive sleep apnea (OSA). Metabolically unhealthy obesity in adolescents and youth has become a public health issue worldwide.

OSA is characterized by the collapse of the upper airway during sleep, leading to airflow cessation or reduction.[3] It is associated with obesity and advanced age especially in men, if not treated in time and may lead to an increased incidence of cardiovascular diseases.[4,5] OSA is becoming increasingly common in childhood, especially with the increased prevalence of obesity in recent years. The prevalence of OSA among children is 2–3%,[6] and a survey in Singapore shows that the prevalence of OSA among obese children could be as high as 5.7%.[7] Redline[8] analyses on 270 adolescents showed that OSA was associated with elevated systolic

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blood pressure (SBP) and diastolic blood pressure (DBP), low-density lipoprotein cholesterol (LDL-C), and fasting insulin levels. OSA may also induce cardiovascular diseases and contribute to the development of MS in obese children.[9]

Polysomnography (PSG) is generally considered the gold standard for making the diagnosis of OSA. However, its utilization is not widespread because it is expensive and time-consuming. The Berlin Questionnaire (BQ) was first introduced in 1996 at the Conference on Sleep in Primary Care in Berlin, Germany. It consists of three categories which include questions about snoring, daytime fatigue and sleepiness, high blood pressure and body mass index (BMI). It is a known convenient and validated tool used in screening populations who are at high risk for OSA.[10,11]

OSA and obesity have been largely studied in adult populations, but few studies have been conducted in adolescents with risk for MS. Based on a large cohort study of MS,[12] we performed a comprehensive cardiometabolic evaluation of adolescents and youth with risk for OSA, including fasting lipid profile, blood glucose (BG), liver enzymes, blood pressure, liver ultrasonography, and echocardiography. The purpose of the study was to evaluate the correlation between obesity and OSA, and to identify the cardiometabolic risk factors in subjects with high risk for OSA.

Methods

Ethics statement
The study was approved by the Ethics Committee at the Capital Institute of Pediatrics in Beijing. Informed consent was obtained from patients or parents (caregivers) on behalf of minors.

Participants
The data were obtained from the Beijing Child and Adolescent Metabolic Syndrome (BCAMS) study.[12,13] The BCAMS study evaluated the prevalence of obesity and related metabolic abnormalities such as hypertension, hyperglycemia, and dyslipidemia in Beijing school-age children (n = 19,593, ages 6–18 years, 50% male) between April and October 2004. Within this cohort, 4500 were identified as high risk due to having one of the following: Overweight defined by BMI, elevated cholesterol ≥5.2 mmol/L, elevated triglycerides (TGs) ≥1.7 mmol/L, or elevated fasting glucose ≥5.6 mmol/L based on finger capillary blood tests. Current study was the follow-up study beginning in 2012. Subjects were recruited consecutively to undergo a medical examination at a center in Beijing Chaoyang Hospital. Total 558 subjects who completed medical examination were included in this analysis.

Berlin questionnaire (for sleep apnea)
BQ was used to identify subjects at risk for the OSA.[11] The BQ includes three categories with ten questions, five questions related to snoring behaviors/witnessed apneas (category 1), three about daytime

sleepiness (category 2), and two about the patient's history of hypertension and/or obesity (category 3). Subjects were considered to be at high risk for OSA, if they had at least two of the following conditions: (1) Two questions positive in category 1 that indicated the presence of persistent snoring (more than 3 times a week) with snoring sufficiently loud to be heard in the next room, and/or persistent apneas (more than 3 times a week or everyday); (2) daytime sleepiness defined as ever falling asleep while driving or as having at least two of the following symptoms at least 3 days a week: Feeling tired or fatigued after sleep, or feeling tired or fatigued during wake time (category 2); and (3) presence of hypertension or obesity. Subjects who denied chronic symptoms or had chronic symptoms or signs in only 1 category were classified as low risk for OSA.[10,11]

Clinical and laboratory measurements
Height, weight, neck and waist circumference (WC) and body fat percentage (FAT%) were measured by trained field workers. Participants removed bulky clothing and shoes prior to measurement. Height was measured to the nearest 0.1 cm using a portable stadiometer. Neck circumference (NC) was measured in the midway of the neck between mid-cervical spine and mid-anterior neck to 0.5 cm just below the laryngeal prominence. WC was measured midway between the lowest rib and the top of the iliac crest. Weight and FAT% was measured to the nearest 0.1 kg using a TANITA Body Composition Analyzer (Model TBF-300A (Tanita, Japan)). Measurements of right arm SBP and DBP were performed 3 times 10 min apart, and the mean values of the latter two measurements were recorded. BMI was calculated as weight divided by height squared. BMI was converted to age- and sex-standardized percentiles based on the Centers for Disease Control and Prevention 2000 growth charts, which are not race specific.[14] Subjects of children and teens were classified as overweight, if BMI was between the 85th and 95th percentile, or obese if BMI was above 95th percentile. Subjects older than 18 years old were classified overweight if BMI ≥24 kg/m², or obese if BMI ≥28 kg/m². Hypertension was classified as SBP and DBP ≥95th percentile for age, sex, and height (<18 years old) or ≥140/90 mmHg. WC was converted to age, gender percentile values based on BCAMS study in 2004.[12] Subjects were classified as abdominal obesity if WC ≥90th percentile for child and adolescent or if WC ≥90 cm for male or ≥80 cm for female (≥18 years old).

Venous blood samples were collected after an overnight (≥10 h) fast. An oral glucose tolerance test using 75 g glucose load was performed on each participant. The concentrations of plasma glucose, TGs, total cholesterol (TC), LDL-C and high-density lipoprotein cholesterol (HDL-C), aspartate aminotransferase (AST), and alanine aminotransferase (ALT) were assayed using the Hitachi 7060 C automatic biochemistry analysis system.

Echocardiography and liver ultrasonography
Noninvasive transthoracic echocardiogram was performed by a sonographer, and the following cardiac measurements were obtained: Interventricular septum thickness (IVST),

left ventricular end diastolic diameter (LVED), and left ventricular posterior wall thickness (LVPWT). Liver ultrasonography was also conducted for the diagnosis of nonalcoholic fatty liver disease (NAFLD).

Diagnoses of metabolic syndrome

MS in adolescents was diagnosed according to the 2007 consensus definition by the International Diabetes Federation.[13] The diagnostic criteria include abdominal obesity characterized by the presence of WC ≥90th in 10–16 years, WC ≥90 cm for boys and WC ≥80 cm for girls in over 16 years old plus the presence of 2 or more of the following criteria: (1) Fasting blood glucose (FBG) ≥5.6 mmol/L and/or 2 h glucose ≥7.8 mmol/L, or a diagnosis of diabetes; (2) SBP ≥130 mmHg or DBP ≥ 85 mmHg or presence of drug treatment for hypertension; (3) HDL-C <1.03 mmol/L in male, <1.29 mmol/L in female or presence of drug treatment; and (4) TG ≥1.70 mmol/L or drug treatment for elevated TG levels.

Data analysis

All statistical analyses were carried out using the Statistical Package for Social Sciences (SPSS 19.0 for Windows, SPSS Inc., USA). Results are expressed as mean and standard deviation for continuous variables, and counts and percentages for categorical variables. Comparisons between the groups were achieved using the t-test or analysis of variance for continuous variables, while categorical variables were explored using the Chi-square test. Logistic regression was used to calculate the odds ratio (OR) and confidence interval (CI) of variables, while adjusting for possible confounders. Level of significance was accepted as $P < 0.05$.

RESULTS

Characteristics of the study cohort

The clinical and biochemical features of the subjects stratified by gender are shown in Table 1. The average age was 20.2 years (14–28 years). Of the total 559 subjects, 72 (12.9%) were likely to have OSA by BQ, with high percentage of male. The prevalence rates of obesity, hypertension, NAFLD, and MS were 32%, 9.0%, 10.4%, and 28.2%, respectively, with much higher in males than in females. HDL-C were higher in females ($P < 0.001$), while FBG, TG, AST, ALT, SBP, and DBP were higher in males ($P < 0.001$).

Relationship between obesity and risk of obstructive sleep apnea

The associations between risk of OSA and multiple anthropometric indices related to general obesity and abdominal obesity are shown in Table 2. Subjects with high risk for OSA showed significantly higher BMI, neck and waist circumstance, FAT% and waist to height ratio than in low risk for OSA ($P < 0.001$). Those significant differences were not modified by adjustment of sex and age. Particularly, subjects with high risk for OSA had a higher neck size even after further adjusted for BMI ($P = 0.004$). The proportions of

Table 1: Characteristics of the study population

Items	Total (n=559)	Males (n=294)	Females (n=265)	P
High risk for OSA, n (%)	72 (12.9)	50 (17.0)	22 (8.3)	0.002
Age (years)*	20.1 (2.9)	20.0 (3.0)	20.4 (2.8)	0.110
BMI (kg/m²)*	25.7 (5.7)	27.0 (5.8)	24.3 (5.3)	<0.001
WC (cm)*	85.2 (14.6)	90.5 (14.6)	79.3 (12.1)	<0.001
NC (cm)*	34.7 (4.1)	37.2 (3.6)	32.0 (2.9)	<0.001
Fat%*	30.43 (10.26)	27.92 (9.38)	33.21 (10.49)	<0.001
FBG (mmol/L)	4.92 (0.69)	5.00 (0.86)	4.82 (0.41)	<0.001
2 h-BG (mmol/L)	6.06 (1.84)	6.17 (2.07)	5.95 (1.54)	0.179
SBP (mmHg)	115 (14)	121 (14)	108 (11)	<0.001
DBP (mmHg)	73 (11)	76 (10)	70 (10)	<0.001
TG (mmol/L)	1.13 (0.83)	1.25 (1.01)	1.01 (0.54)	<0.001
TC (mmol/L)	4.35 (0.92)	4.29 (0.86)	4.41 (0.99)	0.131
LDL-C (mmol/L)	2.53 (0.79)	2.56 (0.72)	2.50 (0.86)	0.371
HDL-C (mmol/L)	1.44 (0.32)	1.34 (0.28)	1.54 (0.34)	<0.001
AST (U/L)	20.5 (15)	22.7 (19.8)	18.0 (5.4)	<0.001
ALT (U/L)	23.1 (19.8)	28.2 (24.3)	17.1 (10.8)	<0.001
Overweight, n (%)	135 (24)	75 (26)	60 (23)	<0.001
Obesity, n (%)	181 (32)	121 (41)	60 (23)	<0.001
Hypertension, n (%)	53 (9)	45 (15)	8 (3)	<0.001
MS, n (%)	58 (10.4)	46 (15.6)	12 (4.5)	<0.001
NAFLD, n (%)	158 (28.2)	107 (36.4)	51 (20.2)	<0.001

*Data are means (SD). SD: Standard deviation; OSA: Obstructive sleep apnea; BMI: Body mass index; WC: Waist circumference; NC: Neck circumference; BG: Blood glucose; FBG: Fasting blood glucose; SBP: Systolic blood pressure; DBP: Diastolic blood pressure; TG: Triglycerides; TC: Total cholesterol; LDL-C: Low-density lipoprotein cholesterol; HDL-C: High-density lipoprotein cholesterol; AST: Aspartate aminotransferase; ALT: Alanine aminotransferase; MS: Metabolic syndrome; NAFLD: Nonalcoholic fatty liver disease; FAT%: Body fat percentage.

Table 2: Multiple anthropometric indices of obesity and risk of OSA

Items	High risk for OSA (n=72)	Low risk for OSA (n=487)	t/χ²	P
male/female	50/22	244/243	9.42	0.002
Age (years)*	20.9 (3.0)	20.0 (2.9)	2.38	0.018
BMI (kg/m²)*	32.5 (5.5)	24.7 (5.0)	12.14	<0.001
WC (cm)*	101.4 (13.1)	82.8 (13.2)	11.17	<0.001
NC (cm)*	39.0 (4.3)	34.1 (3.7)	10.34	<0.001
FAT%*	39.4 (11.2)	29.1 (9.4)	8.44	<0.001
Waist to height ratio*	0.59 (0.08)	0.49 (0.07)	11.93	<0.001
BMI categories				
Normal	7 (9.7)	236 (48.5)	103.40	<0.001
Overweight	4 (5.6)	131 (26.9)		
Obesity	61 (84.7)	120 (24.6)		
WC categories				
Non-abdominal obesity	7 (9.7)	257 (52.8)	46.60	<0.001
Abdominal obesity	65 (90.3)	230 (47.2)		

*Data are means (SD). SD: Standard deviation; WC: Waist circumference; NC: Neck circumference; OSA: Obstructive sleep apnea; BMI: Body mass index; FAT%: Body fat percentage.

normal, overweight, and obesity defined by BMI percentile were 9.7%, 5.6%, and 84.7% in the high-risk group of OSA, respectively. The detection rate of abdominal obesity was high up to 90.3% in the high-risk group compared to 47.2% in the low-risk group. On the other hand, 33.7% (61/181) of subjects with general obesity and 22.0% of subjects with abdominal obesity were likely to have OSA. Both general and central obesity were significance associated with high risk for OSA ($P < 0.001$).

Relationship between obstructive sleep apnea and cardiovascular risk factors

Cardiovascular risk factors in two groups with high or low risk of OSA are shown in Table 3. Mean levels of TC, LDL-C, and TG were significantly higher in high-risk group than those in low-risk group, while HDL-C was significantly lower in high-risk group. Compared with subjects with low risk for OSA, subjects with high risk had higher levels of 0.5 h- and 2 h-BG ($P < 0.001$ and $P < 0.05$), higher SBP ($P < 0.001$) and DBP ($P < 0.001$), and higher IVST, LVED, and LVPWT ($P < 0.001$). Furthermore, levels of liver enzyme (AST and ALT) as well as rates of NAFLD were significantly higher in high-risk group of OSA than in low-risk group ($P < 0.001$).

Relationship between obstructive sleep apnea and metabolic syndrome

Table 4 shows the detection rates of MS and its components, according to risk for OSA. Subjects with high risk for OSA had higher proportion of MS compared to low risk for OSA (38.9% vs. 7.0%), with an age- and sex-adjusted odd ratio of 9.28 (95% *CI*, 4.91–17.53). The detection rates of abdominal obesity, high BP, high TG, low HDL-C, and high glucose in high-risk group were 91.2%, 41.7%, 40.3%, 37.5%, and 13.8%, respectively, which were significantly higher compared with low risk group.

Independent associations between above adiposity-, and cardiometabolic-related variables [Tables 2 and 3] and OSA according to logistic analysis are shown in Table 5. After multiple factors adjustment, NC, BMI, and TG are the independent factors of OSA, with NC *OR* 1.12 (95% *CI*, 1.02–1.24), BMI *OR* 1.20 (95% *CI*, 1.12–1.29), and TG *OR* 1.34 (95% *CI*, 1.01–1.79). For additional 1 cm each in neck size, the occurrence risk of OSA increased 1.12-fold; for additional 1 kg/m² each in BMI, the occurrence risk of OSA increased 1.2-fold, and for additional 1 mmol/L each in TG, the occurrence risk of OSA increased 1.34-fold.

DISCUSSION

In the present study, the estimated prevalence of OSA by BQ in obese adolescents and youth was 33.7%, which was much higher compared to 12.4% in a Korea population-based study conducted by the Korea Center for Disease Control and Prevention using the same instrument of BQ.[15] On the other hand, the prevalence rates of general obesity and abdominal obesity in high-risk subjects were high up to 84.7%–90.3%, suggesting that both general and central obesity were strongly

Table 3: Comparisons of cardiovascular risk factors between the two groups of OSA

Items	High risk for OSA	Low risk for OSA	t/χ^2	P
Lipids				
HDL (mmol/L)	1.27 (0.29)	1.46 (0.32)	−4.84	<0.001
LDL (mmol/L)	2.94 (0.80)	2.47 (0.77)	4.73	<0.001
TG (mmol/L)	1.73 (1.42)	1.05 (0.66)	6.86	<0.001
TC (mmol/L)	4.69 (0.98)	4.30 (0.90)	3.40	<0.001
Glucose				
FBG (mmol/L)	5.06 (0.59)	4.90 (0.71)	1.85	0.064
0.5 h-BG (mmol/L)	8.60 (1.31)	7.84 (1.51)	3.90	<0.001
2 h-BG (mmol/L)	6.66 (1.48)	5.98 (1.87)	2.92	0.004
Blood pressure				
SBP (mmHg)	124.74 (14.60)	112.98 (13.73)	6.73	<0.001
DBP (mmol/L)	80.81 (10.49)	71.99 (10.05)	6.91	<0.001
Cardiac structures				
IVST (cm)	0.94 (0.13)	0.88 (0.12)	4.03	<0.001
LVED (cm)	4.63 (0.41)	4.38 (0.49)	3.90	<0.001
LVPWT (cm)	0.95 (0.12)	0.88 (0.11)	4.84	<0.001
Liver function				
AST (U/L)	26.97 (24.49)	19.54 (12.79)	3.97	<0.001
ALT (U/L)	38.82 (39.77)	20.83 (13.38)	7.53	<0.001
NAFLD (%)	49 (71.01)	109 (24.22)	84.22	<0.001

Data are means (SD) or *n* (%). SD: Standard deviation; BG: Blood glucose; FBG: Fasting blood glucose; SBP: Systolic blood pressure; DBP: Diastolic blood pressure; TG: Triglycerides; TC: Total cholesterol; LDL: Low-density lipoprotein; HDL: High-density lipoprotein; AST: Aspartate aminotransferase; ALT: Alanine aminotransferase; MS: Metabolic syndrome; NAFLD: Nonalcoholic fatty liver disease; IVST: Interventricular septum thickness; LVED: Left ventricular end diastolic diameter; LVPWT: Left ventricular posterior wall thickness.

Table 4: Frequencies of MS and its components in the two groups of OSA

Items	High risk for OSA (n=72)	Low risk for OSA (n=487)	*OR*s (95% *CI*s)*	P
Abdominal obesity	65 (90.3)	230 (47.2)	11.16 (4.96–25.10)	<0.001
High BP	30 (41.7)	76 (15.6)	2.97 (1.70–5.17)	<0.001
High TG	29 (40.3)	51 (10.5)	4.77 (2.70–8.41)	<0.001
Low HDL-C	27 (37.5)	66 (13.6)	5.67 (2.88–11.13)	<0.001
High glucose	10 (13.8)	32 (6.7)	2.49 (1.14–5.44)	0.023
MS	28 (38.9)	34 (7.0)	9.28 (4.91–17.53)	<0.001

Data are *n* (%). **OR*s and 95% *CI*s for MS and its components after adjusting for age and sex. *OR*s: Odds ratios; *CI*s: Confidence intervals; MS: Metabolic syndrome; BP: Blood pressure; TG: Triglycerides; HDL-C: High-density lipoprotein cholesterol; OSA: Obstructive sleep apnea.

Table 5: Logistic regression model for the independent factors associated with OSA

Items	B	SE	Wals	*OR*s (95% *CI*)	P
Neck circumstance (cm)	0.115	0.050	5.268	1.12 (1.02–1.24)	0.022
BMI (kg/m²)	0.186	0.036	27.367	1.20 (1.12–1.29)	<0.001
TG (mmol/L)	0.294	0.146	4.041	1.34 (1.01–1.79)	0.044

SE: Standard error; *OR*s: Odds ratios; *CI*s: Confidence intervals; TG: Triglycerides; BMI: Body mass index; OSA: Obstructive sleep apnea.

associated with OSA. Moreover, our study demonstrated that NC was a potential predictor of OSA independent of BMI or WC, thus neck size might be a powerful physical sign when considering OSA in obese youth.

In our study, subjects belonging to high risk for OSA groups showed significantly higher obesity-related indices such as BMI, WC, FAT%, and waist-to-height ratio (WHtR) than those in low risk for OSA ($P < 0.001$). These results were similar to those of previous studies. Gozal et al.[16] investigated 62 children with OSA aged 7.4 ± 2.6 years, found that high-risk OSA had higher BMI, WC, FAT% comparing with low-risk OSA. Moreover, our study demonstrated that both BMI and NC but not waist or WHtR independently predict OSA in adolescents and youth, implying that obesity mediates its effect through fat deposition in the neck.

OSA patients often have obesity and experience higher incidence of cardiovascular disease, arterial hypertension, and cerebrovascular disease.[17] OSA patients resulted in increased cardiovascular morbidity and mortality in the adult population, but few studies have been conducted so far on the influence between OSA and cardiometabolic risk factors in adolescents and youth. There is even less research exploring the relationship between OSA and lipid metabolism.

A large cohort study has shown an association between high-risk OSA and higher TG, lower HDL-C.[18,19] Our study shows that mean level of TC, LDL-C, TG in high risk for OSA was significantly higher than in low risk for OSA ($P < 0.001$), while HDL-C was significantly lower ($P < 0.001$). We also found that adolescents and youth in high-risk group of OSA had a high level of AST and ALT, and high rates of NAFLD than in low-risk group. For the mechanism, animal studies suggested chronic intermittent hypoxia plays an important role in the development of fatty liver in obesity.[20]

With respect to glucose metabolism, in some reports there was a positive association between high risk of OSA and hyperglycemia.[21,22] In our study, we found no differences in FBG between groups of high risk for OSA and low risk for OSA, but postload plasma glucose levels were significantly increased in high-risk group, implying that an impaired glucose tolerance was associated with OSA.

Hypertension has been widely studied in OSA patients.[23] There was a statistical significance between low risk for OSA and high risk for OSA in SBP ($P < 0.001$) and DBP ($P < 0.001$). Our findings were consistent with large studies showing a high prevalence of hypertension among OSA individuals.[24,25] Furthermore, our report shows there was a statistical significance between low risk for OSA and high risk for OSA in IVST, LVED, and LVPWT ($P < 0.001$). Some published studies confirm that OSA is associated with left ventricular hypertrophy, abnormal ventricular geometry, and a decreased diastolic function through measuring left ventricular mass index and geometry in children and adolescents.[26,27] Thus, as high risk for OSA can not only affect blood pressure, but may also affect the cardiac structures, suggesting that OSA may be an important element in changing the ventricular, structure of the heart increasing the risk for early cardiovascular disease.

In the present study, subjects in high risk of OSA had higher prevalence of MS (38.9%), with an OR of 9.3 compared to those in low risk for OSA ($P < 0.001$), suggesting that OSA may contribute to the development of MS in obese adolescents and youth. Redline[8] investigated 270 adolescents with mean age of 13.6 years, adolescents with OSA had a 6.9 increased odds of MS compared with adolescents without OSA. OSA increases the risk of both metabolic diseases and cardiovascular diseases in adolescents and youth, therefore, providing a cost-effective way to appropriately screen for OSA is necessary.

There were several limitations to the study. First, the questionnaire-based method used to estimate the prevalence of OSA and participants did not undergo PSG which is the gold standard for making a diagnosis. Second, age distribution of participants was widespread including both adolescents and youth with different criteria being used to diagnose obesity and MS based on age and these limitations also included assessing cardiometabolic risk factors due to age-specific parameters.

As the obesity epidemic is rising, the prevalence of OSA is likely to increase, thus may lead to an important public health problem. From the study, it is clear that both OSA and obesity, which often co-exist, may act concurrently to determine the cardiometabolic risk factors. On the other hand, the present study also provides evidence that OSA in adolescents and youth adversely affects the components associated with MS. A better understanding of the complications of OSA could reduce the occurrence of cardiovascular disease and to a reduction of the social impact of this condition.

Conflicts of interest
There are no conflicts of interest.

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