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

Xiao-Xue Qu1, Issy C Esangbedo2, Xiu-Juan Zhang1, Shu-Jun Liu1, Lian-Xia Li3, Shan Gao1, Ming Li3

1Department of Endocrinology, Chaoyang Hospital, Capital Medical University, Beijing 100043, China
2Health Weight Program Clinic, Children’s Hospital of Philadelphia, Perelman School of Medicine at University of Pennsylvania, USA
3Department of Endocrinology, Key Laboratory of Endocrinology, Ministry of Health, Peking Union Medical College Hospital, Chinese Academy of Medical Sciences and Peking Union Medical College, Beijing 100730, China

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 Redline8 analyses on 270 adolescents showed that OSA was associated with elevated systolic...
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.\[10,11\]

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,\[12,13\] 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 $\geq 5.2$ mmol/L, elevated triglycerides (TGs) $\geq 1.7$ mmol/L, or elevated fasting glucose $\geq 5.6$ 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 $\geq 24$ kg/m², or obese if BMI $\geq 28$ kg/m². Hypertension was classified as SBP and DBP $\geq 95$th percentile for age, sex, and height (<18 years old) or $\geq 140/90$ mmHg. WC was converted to age, gender percentile values based on BCAMS study in 2004.\[12\] Subjects were classified as abdominal obesity if WC $\geq 90$th percentile for child and adolescent or if WC $\geq 90$ cm for male or $\geq 80$ cm for female ($\geq 18$ 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. The diagnostic criteria include abdominal obesity characterized by the presence of WC ≥90 cm 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 males, <1.29 mmol/L in females, 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 circumference, 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 (%) |                |                 |                   |       |
| 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|
| WC (cm)*               | 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|
| WC (cm)*               | 4.35 (0.92)    | 4.29 (0.86)     | 4.41 (0.99)       | 0.131 |
| HDL-C (mmol/L)*        | 2.53 (0.79)    | 2.56 (0.72)     | 2.50 (0.86)       | 0.371 |
| AST (U/L)              | 2.53 (0.79)    | 2.56 (0.72)     | 2.50 (0.86)       | <0.001|
| ALT (U/L)              | 1.44 (0.32)    | 1.34 (0.28)     | 1.54 (0.34)       | <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; 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/\(t^2\) | 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; BMI: Body mass index; 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/χ²     | P     |
|----------------|-------------------|------------------|----------|-------|
| **Lipids**     |                   |                  |          |       |
| HDL (mmol/L)   | 1.27 (0.29)       | 1.46 (0.32)      | -4.84    | <.001 |
| LDL (mmol/L)   | 2.94 (0.80)       | 2.47 (0.77)      | 4.73     | <.001 |
| TG (mmol/L)    | 1.73 (1.42)       | 1.05 (0.66)      | 6.86     | <.001 |
| TC (mmol/L)    | 4.69 (0.98)       | 4.30 (0.90)      | 3.40     | <.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     | <.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     | <.001 |
| DBP (mmHg)     | 80.81 (10.49)     | 71.99 (10.05)    | 6.91     | <.001 |
| **Cardiac structures** |            |                  |          |       |
| IVST (cm)      | 0.94 (0.13)       | 0.88 (0.12)      | 4.03     | <.001 |
| LVED (cm)      | 4.63 (0.41)       | 4.38 (0.49)      | 3.90     | <.001 |
| LVPWT (cm)     | 0.95 (0.12)       | 0.88 (0.11)      | 4.84     | <.001 |
| **Liver function** |                 |                  |          |       |
| AST (U/L)      | 26.97 (24.49)     | 19.54 (12.79)    | 3.97     | <.001 |
| ALT (U/L)      | 38.82 (39.77)     | 20.83 (13.38)    | 7.53     | <.001 |
| **NAFLD (%)**  | 49 (71.01)        | 109 (24.22)      | 84.22    | <.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 | Low risk for OSA | ORs (95% CIs) | P     |
|----------------|-------------------|------------------|---------------|-------|
| **Abdominal obesity** |               |                  |               |       |
| NC             | 65 (90.3)         | 230 (47.2)       | 11.16 (4.96–25.10) | <.001 |
| **High BP**    | 30 (41.7)         | 76 (15.6)        | 2.97 (1.70–5.17) | <.001 |
| **High TG**    | 29 (40.3)         | 51 (10.5)        | 4.77 (2.70–8.41) | <.001 |
| **Low HDL-C**  | 27 (37.5)         | 66 (13.6)        | 5.67 (2.88–11.13) | <.001 |
| **High glucose** | 10 (13.8)        | 32 (6.7)         | 2.49 (1.14–5.44) | 0.023 |
| **NAFLD (%)**  | 28 (38.9)         | 34 (7.0)         | 9.28 (4.91–17.53) | <.001 |
| Data are n (%). *ORs and 95% CIs for MS and its components after adjusting for age and sex. ORs: Odds ratios; CIs: 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 | ORs (95% CI) | P     |
|----------------|--------|--------|------|--------------|-------|
| **Neck circumference (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) | <.001 |
| **TG (mmol/L)** | 0.294  | 0.146  | 4.041| 1.34 (1.01–1.79) | 0.044 |
| SE: Standard error; ORs: Odds ratios; CIs: 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\(^{[9]} \) 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.

Acknowledgment
Nil.

Financial support and sponsorship
This work was supported by grants from Beijing Municipal Science and Technology Commission (No. D111100000611002, No. D111100000611001).

Conflicts of interest
There are no conflicts of interest.

References
1. Ogden CL, Yanovski SZ, Carroll MD, Flegal KM. The epidemiology of obesity. Gastroenterology 2007;132:2087-102.
2. Wang Y, Beydoun MA. The obesity epidemic in the United States – Gender, age, socioeconomic, racial/ethnic, and geographic characteristics: A systematic review and meta-regression analysis. Epidemiol Rev 2007;29:6-28.
3. Al Lawati NM, Patel SR, Ayas NT. Epidemiology, risk factors, and consequences of obstructive sleep apnea and short sleep duration. Prog Cardiovasc Dis 2009;51:285-93.
4. Punjabi NM, Caffo BS, Goodwin JL, Gottlieb DJ, Newman AB, O’Connor GT, et al. Sleep-disordered breathing and mortality: A prospective cohort study. PLoS Med 2009;6:e1000132.
5. Young T, Shahar E, Nieto FJ, Redline S, Newman AB, Gottlieb DJ, et al. Predictors of sleep-disordered breathing in community-dwelling
adults: The Sleep Heart Health Study. Arch Intern Med 2002;162:893-900.
6. Guilleminault C, Lee JH, Chan A. Pediatric obstructive sleep apnea syndrome. Arch Pediatr Adolesc Med 2005;159:775-85.
7. Chay OM, Goh A, Abisheganaden J, Tang J, Lim WH, Chan YH, et al. Obstructive sleep apnea syndrome in obese Singapore children. Pediatr Pulmonol 2000;29:284-90.
8. Redline S, Storfer-Isser A, Rosen CL, Johnson NL, Kirchner HL, Emancipator J, et al. Association between metabolic syndrome and sleep-disordered breathing in adolescents. Am J Respir Crit Care Med 2007;176:401-8.
9. Spicuzza L, Leonardi S, La Rosa M. Pediatric sleep apnea: Early onset of the ‘syndrome’? Sleep Med Rev 2009;13:485-91.
10. Netzer NC, Strohs RA, Netzer CM, Clark K, Strohl KP. Using the Berlin Questionnaire to identify patients at risk for the sleep apnea syndrome. Ann Intern Med 1999;131:485-91.
11. Amra B, Farajzadegan Z, Golshan M, Fietze I, Penzel T. Prevalence of sleep apnea-related symptoms in a Persian population. Sleep Breath 2011;15:425-9.
12. Li M, Fisette A, Zhao XY, Deng JY, Mi J, Cianflone K. Serum resistin correlates with central obesity but weakly with insulin resistance in Chinese children and adolescents. Int J Obes (Lond) 2009;33:424-39.
13. Wang Q, Yin J, Xu L, Cheng H, Zhao X, Xiang H, et al. Prevalence of metabolic syndrome in a cohort of Chinese schoolchildren: Comparison of two definitions and assessment of adipokines as components by factor analysis. BMC Public Health 2013;13:249.
14. Group of China Obesity Task Force. Body mass index reference norm for screening overweight and obesity in Chinese children and adolescents (in Chinese). Chin J Epidemiol 2004;25:97-102.
15. Kang K, Seo JG, Seo SH, Park KS, Lee HW. Prevalence and related factors for high-risk of obstructive sleep apnea in a large Korean population: Results of a questionnaire-based study. J Clin Neurol 2014;10:42-9.
16. Gozal D, Capdevila OS, Kheirandish-Gozal L. Metabolic alterations and systemic inflammation in obstructive sleep apnea among nonobese and obese prepubertal children. Am J Respir Crit Care Med 2008;177:142-9.
17. Korostovtseva LS, Sviryaev YV, Zvartau NE, Konadi AO, Kalinkin AL. Prognosis and cardiovascular morbidity and mortality in prospective study of hypertensive patients with obstructive sleep apnea syndrome in St Petersburg, Russia. Med Sci Monit 2011;17:CR146-53.
18. Coughlin SR, Mawdsley L, Mugarza JA, Calverley PM, Wilding JP. Obstructive sleep apnoea is independently associated with an increased prevalence of metabolic syndrome. Eur Heart J 2004;25:735-41.
19. Roche F, Sforza E, Pichot V, Maudoux D, Garenc A, Celle S, et al. Obstructive sleep apnoea/hypopnea influences high-density lipoprotein cholesterol in the elderly. Sleep Med 2009;10:882-6.
20. Drager LF, Li J, Reinecke C, Bevans-Fonti S, Jun JC, Piotou SK. Intermittent hypoxia exacerbates metabolic effects of diet-induced obesity. Obesity (Silver Spring) 2011;19:2167-74.
21. Panjabi NM, Beamer BA. Alterations in Glucose Disposal in Sleep-disordered Breathing. Am J Respir Crit Care Med 2009;179:235-40.
22. Steiropoulos P, Papanas N, Bouros D, Maltezos E. Obstructive sleep apnea aggravates glycemic control across the continuum of glucose homeostasis. Am J Respir Crit Care Med 2010;182:286.
23. Bradley TD, Floras JS. Obstructive sleep apnoea and its cardiovascular consequences. Lancet 2009;373:82-93.
24. Nieto FJ, Young TB, Lind BK, Shahar E, Samet JM, Redline S, et al. Association of sleep-disordered breathing, sleep apnea, and hypertension in a large community-based study. Sleep Heart Health Study. JAMA 2000;283:1829-36.
25. Lavie P, Herer P, Hoffer V. Obstructive sleep apnoea syndrome as a risk factor for hypertension: Population study. BMJ 2000;320:479-82.
26. Amin RS, Kimball TR, Bean JA, Jeffries JL, Willging JP, Cotton RT, et al. Left ventricular hypertrophy and abnormal ventricular geometry in children and adolescents with obstructive sleep apnea. Am J Respir Crit Care Med 2002;165:1395-9.
27. Amin RS, Kimball TR, Kalra M, Jeffries JL, Carroll JL, Bean JA, et al. Left ventricular function in children with sleep-disordered breathing. Am J Cardiol 2005;95:801-4.