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ORIGINAL ARTICLE

Obesity in children with different risk factors for obstructive sleep apnea: a community-based study

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Abstract This study investigated the association between obesity and obstructive sleep apnea (OSA) in preschool and school-age children. Parents of obese and randomly chosen normal weight children completed a questionnaire on sleep-related symptoms, demography, family, and medical history. All subjects were invited to undergo polysomnography (PSG). OSA cases were defined as obstructive apnea hypopnea index (OAHI) ≥1. A total of 5930 children were studied with 9.5 % obese (11.9 % boys/6.1 % girls), 205/2680 preschool and 360/3250 school children. There were 1030 children (535 obese/495 normal weight) who underwent PSG. OSA was higher in obese children and obese school children had higher OAHI, arousal index, and shorter total sleep time. However, there was no positive correlation between OSA and body mass index (BMI). The main risk factors for OSA in preschool children were adenotonsillar hypertrophy and recurrent respiratory tract infection. The main cause for OSA in school children was a history of parental snoring and obesity. Mallampati scores and sleep-related symptoms were found to be associated with OSA in both preschool and school children.

Conclusion: We demonstrated differential risk factors for OSA in obese children, which suggest that a different mechanism may be involved in OSA development in preschool and school-age children.

What is Known:

Various risk factors have been reported in obese children with OSA owing to the different age and different study design.

- *Obese children have a higher prevalence and severity of obstructive sleep apnea (OSA).*
- *OSA risk factors in obese children are affected by different ages and study designs.*

What is New:

- *A differential prevalence and risk factors for obese preschool and school-age children with OSA has been demonstrated.*

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Keywords Obesity · Obstructive sleep apnea · Prevalence · Risk factors

Abbreviations

BMI	Body mass index
OAHI	Obstructive apnea hypopnea index
OSA	Obstructive sleep apnea
PSG	Polysomnography
REM	Rapid eye movement

Introduction

Obesity is a common global health problem and can cause hypertension, diabetes, hyperlipidemia, and cardiovascular disease in children and adults [12, 13, 25]. In the past decades, the prevalence of obesity in children has doubled in the USA and China [26, 27, 32, 42]. Using the same definition, based on the International Obesity Task Force (IOTF) definition, the prevalence of obesity in children varies from 2.2 to 14.2 % [2, 10, 16, 30; Table 7]. However, according to the World Health Organization (WHO) standards, the varying prevalence rate of obesity in children is from 4.9 to 29.9 % [15, 29; Table 8], owing in part to age, ethnic group, and area.

Previous studies have reported a higher prevalence and severity of obstructive sleep apnea (OSA) in children with obesity [7, 8, 17, 36, 41]. Potential contributions of adiposity to the airway collapsibility during sleep in obese children may cause the occurrence and severity of OSA. Frequent fragmentary sleep in children with OSA may lead to short sleep duration, which may increase appetite and body mass index (BMI) [34, 38], leading to a vicious cycle. It has been hypothesized that OSA contributes to obesity and vice versa. However, recent studies have failed to support a positive relationship between the degree of obesity and the severity of OSA. The risk of OSA has not been shown to significantly increase with increased BMI in younger children [19]. Indeed, the validity of an association between obesity and OSA has recently been questioned [20]. Such discrepant findings partly reside in the limitations imposed by the definition of obesity in terms of BMI. It is important to note that some previous studies were limited by their selected patient recruitment and lack of matched control subjects for comparison [8, 17, 36, 41]. In this study, we aimed to identify the prevalence, sleep characteristics, and risk factors associated with obesity and to compare the different risks factors for OSA in preschool and school children with obesity.

Methods

Study population

First phase—sleep questionnaire

This study is part of an epidemiological study to examine the prevalence of obesity in Wenzhou Chinese children. The protocol was approved by the Institutional Ethics Review Committee with support from the public health and disease control center. Parents of all the obese children and an age, sex, and ethnicity-matched normal weight children aged 3–11 years in 13 randomly selected primary schools and kindergartens were selected and invited to participate and attend an education forum where details of the study were explained. A validated OSA screening sleep questionnaire for children completed by parents [22] and a personally addressed letter asking for consent were then distributed with the help of school teachers. Those who failed to return the questionnaire within a week were given another copy with a self-addressed envelope enclosed for ease of return.

The sleep questionnaire sought information from participants regarding sleep habits and problems in the previous 12 months. The information obtained included demography data (age, sex, living environment, parental snoring history, parental education, and physical measurements), medical history (adenotonsillar hypertrophy, recurrent respiratory tract infection and allergic rhinitis, sinusitis, asthma), respiratory symptoms (snoring, witnessed apnea, labored breathing, and mouth breathing), nocturnal symptoms (restless sleep, prone position, nocturnal enuresis, night sweats, night terrors, nightmares, somnambulism, sleep talking, bruxism), and daytime symptoms (morning headache, daytime fatigue, difficulty getting up in the morning, hyperactivity). Parents were asked to provide answers using a five-point frequency scale (0=never, 1="rarely" for 0–1 night per month, 2="sometimes" for 1–2 nights per month, 3="often" for 1–2 nights per week, 4="frequently" for 3 nights or more per week.) and "do not know" category. All the obese and matched normal weight children that had completed the sleep questionnaire were invited to undergo an overnight polysomnographic study.

Second phase—polysomnography

All the respondents were invited to undergo an overnight (at least 7 h) polysomnography (PSG) (Alice 5, Respironics Inc., Pittsburgh, PA, USA). The montage included eight channels (F3, F4, C3, C4, T3, T4, O1, and O2) of electroencephalogram; electrooculography; submental electromyography; vibration detector; thoracic and abdominal excursions by inductive plethysmography; air flow, which was measured by a nasal pressure catheter and supplemented by an oral thermistor; finger pulse oximetry; electrocardiography; and leg

electromyography [5]. All computerized sleep data were manually edited by experienced PSG technologists and clinicians according to standardized criteria [1]. Hypopnea was defined as a reduction of 50 % or more in the amplitude of the airflow signal. It was only quantified if longer than two baseline breaths and was associated with oxygen desaturation of at least 4 % and/or arousals. Obstructive apnea hypopnea index (OAHI) was defined as the number of apnea and hypopneas per hour of total sleep time (TST). The obstructive apnea index (OAI) was defined as the average number of apnea episodes. The arousal index was the total number of arousals per hour of sleep.

Definition of obesity and OSA

Body mass index (BMI) was calculated as weight (kg) divided by the height squared (m^2). Children were classified as being of normal weight, overweight, or obese according to the criteria defined by the international cutoff points for BMI for overweight and obesity by sex between 2 and 18 years [9]. Children were classified as obese or overweight if their BMI was above the 95th or between the 85th and 95th percentile on the sex- and age-specific growth chart, respectively. Those who were classified as being of normal weight were grouped as controls. For children who underwent PSG, they were separated into OSA cases and non-OSA controls using OAHI ≥1 as the cutoff. Adenotonsillar hypertrophy was defined as tonsil swelling over the pharynx arches and adenoid hypertrophy defined as swelling over more than half of the pharyngeal cavity as assessed by X-ray or laryngoscope.

Statistical analysis

Descriptive data were presented as percentages for discrete variables and as means (standard deviation) or medians (inter-quarter range) for continuous variables. Chi-squared test and Mann–Whitney *U* test were used to compare the variables between children with and without obesity. When performing comparisons between obese and non-obese participants in different age groups, ANOVAs with the factors of obesity and age were performed first. Age differences were only present if the factor age or the interaction was significant. This statistical approach was also used to compare the prevalence of obesity between school and preschool children. Exploratory factor analysis was used to identify the underlying pattern between the various sleep-related symptoms. Correlation coefficients were analyzed by principal component analysis and subsequent rotation according to the standard varimax criterion. With this analysis, the correlation between parameters was attributed to their common dependence on independent entities called "factors." The coefficients that link parameters to factors were called "factor loadings"; the number of factors was chosen to be as small as possible but large enough to account for most of the variation within the data. It was decided a priori that the number of factors in the varimax rotation would be based on the number of eigenvalues above 1.0 in the principal component analysis. Logistic regression analysis was used to determine the association between obesity and risk factors which were found to be significant in univariate analysis. All analyses were performed using SPSS version 15.0 (SPSS, Inc. Chicago, USA). All p values reported are two tailed with statistical significance set at <0.05.

Results

A total of 5930 children were analyzed. The data included 2405 girls and 3525 boys aged 3–11 years. The overall prevalence of obesity was 565/5930 (9.5 %), with boys 419/3525 (11.9 %) two times more likely than girls 146/2405 (6.1 %) to be affected. Among them, the prevalence of obesity in school children tended to be higher than that in preschool children (360/3250 [11.1 %] vs. 205/2680 [7.6 %], $p<0.01$) (Table 1).

The flow chart for selection of study subjects is shown in Fig. 1. Of all the 1130 subjects, 1030 respondents (91.1 %) completed the questionnaires. There were 535 obese children (94.7 %; 203 preschool children and 332 school children) and 495 normal weight children (87.6 %; 178 preschool children and 317 school children) who agreed to take part in the second phase of the overnight PSG study. The characteristics of the children were shown in Tables 2 and 3.

Based on the PSG study, there was higher prevalence of OSA in obese school children than in normal weight children (normal weight [11.0 %] vs. obesity [41.0 %], $p=0.006$), but this was not seen in obese preschool children (normal weight [20.2 %] vs. obesity [28.6 %], $p=0.08$) (Table 4). The school children with obesity were found to have higher OAHI (5.8 [2.1–14.8] vs. 1.4 [0.2–3.3], $p<0.01$), higher arousal index (9.8 [6.8–15.9] vs. 2.4 [3.2–7.3], $p<0.01$), and shorter sleep duration (412 [288–468] vs. 525 [288–468], $p<0.01$) than controls. Further analysis showed that wake time after sleep onset (WASO) was prolonged in obese in contrast with normal weight children (22 [12–36] vs. 36 [21–48], $p=0.035$). However, children with and without obesity had similar sleep latency and sleep efficiency in all the sleep stages (S1, S2, S3+4). In preschool children, the sleep parameter changes in obesity were not seen (Table 4).

Thirteen items in the questionnaire were categorized into three factors by factor analysis, morning or daytime symptoms, nocturnal symptoms, and breathing symptoms (Table 5). The total variance explained by this three-factor model was 52.5 %, and the factor scores were calculated using the regression method. In the first logistic regression model with 203 preschool children with obesity, several risk factors were found to be significantly associated with OSA, including adenotonsillar hypertrophy (odds ratio [OR] 3.52, $p<0.01$),

Table 1 Prevalence of obesity in children by sex in all study groups (additional)

Age (years)	Prevalence of obesity (%)		
	Boys	Girls	Total
3	20/440 (4.5)	18/452 (4.0)	38/892 (4.3)
4	45/448 (10.0)	37/442 (8.4)	82/890 (9.2)
5	53/547 (9.7)	32/351 (9.1)	85/898 (9.5)
3–5	118/1435 (8.2)	87/1245 (7.0)	205/2680 (7.6)*
6	38/325 (11.7)	16/213 (7.5)	54/538 (10.0)
7	41/322 (12.7)	21/218 (9.6)	62/540 (11.5)
8	45/335 (13.4)	18/207 (8.7)	63/542 (11.6)
9	39/336 (11.6)	16/208 (7.7)	55/544 (10.1)
10	44/332 (13.3)	18/216 (8.3)	62/548 (11.3)
11	45/328 (13.7)	19/210 (9.0)	64/538 (11.9)
6–11	252/1978 (12.7)	108/1272 (8.5)	360/3250 (11.1)*
3–11	419/3525 (11.9)$^{\triangle}$	146/2405 (6.1)$^{\triangle}$	565/5930 (9.5)

*$p<0.01$, statistical significance between preschool and school children; $^{\triangle}p<0.01$, comparison between boys and girls

recurrent respiratory tract infection (OR 2.57, $p<0.01$), nocturnal (including restless sleep, prone position, bruxism, and night sweating) (OR 1.38, $p<0.01$), morning and daytime (including morning headache, daytime fatigue, and difficulty getting up in the morning) (OR 2.63, $p<0.01$), breathing (including labored breathing witnessed apnea, mouth breathing, and habitual snoring) (OR 1.46, $p<0.01$), and Mallampati scores (OR 2.18, $p<0.01$) (Table 6).

Table 2 General characteristics of children with and without (control) obesity (3–11 years old)

General characteristics	Control (n=495)		Obesity (n=535)	
	Mean	SD	Mean	SD
Age (year)	6.2	3.7	6.7	4.3
Height (cm)	123.4	34.8	121.5	32.3
Weight (kg)*	21.6	10.2	36.8	16.4
BMI (kg/m^2)*	17.8	3.5	36.4	5.4
BMI z-score*	0.28	0.85	1.72	0.95

*$p<0.01$ compared with control group

In the second logistic model that included 351 school children with obesity who had undergone PSG, the risk factors that were found to be significantly associated with OSA were parental snoring history (OR 2.45, $p<0.01$), parental obesity (OR 2.12, $p<0.01$), nocturnal symptoms (including restless sleep, prone position, bruxism, night sweating, and enuresis) (OR 1.75, $p<0.01$), morning or daytime (including morning headache, daytime fatigue, difficulty getting up in the morning, and hyperactivity) (OR 1.53, $p<0.01$), breathing (OR 1.33, $p<0.01$), BMI z-score (OR 1.15, $p=0.04$), and Mallampati scores (OR 2.45, $p<0.01$) (Table 6).

Discussion

This study is a large community-based study on the association of obesity with OSA and other sleep-related symptoms.

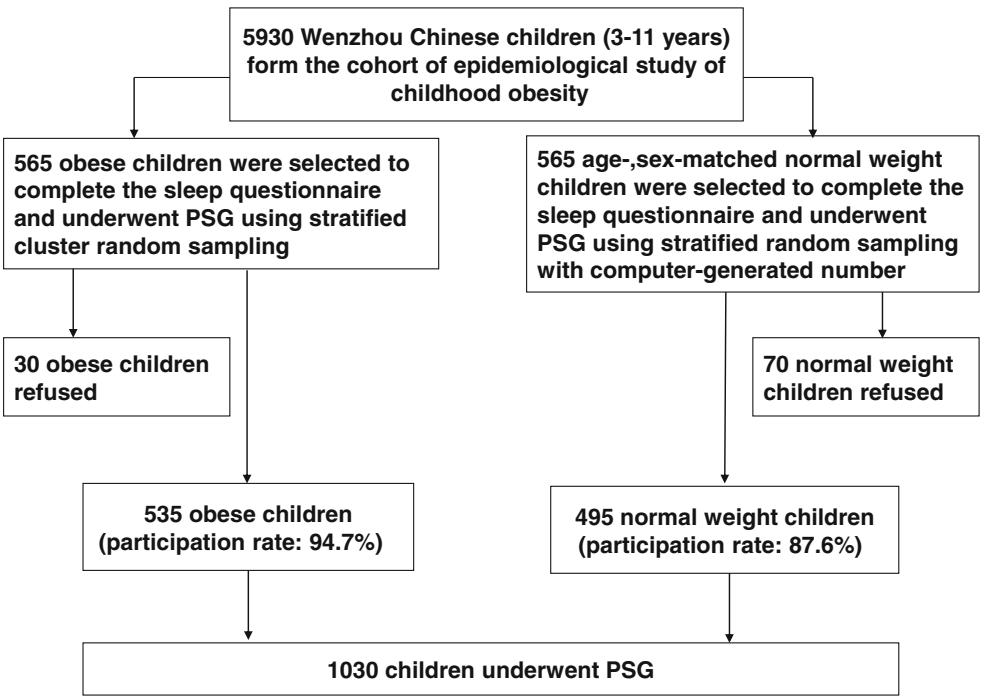

Fig. 1 Flow chart for recruited subjects in sleep study (additional)

Table 3 Clinical characteristics of study subjects with and without (control) obesity (3–11 years old)

Clinical characteristics	Control (n=495) n (%)	Obesity (n=535) n (%)
Boys	338 (68.3)	390 (72.9)
Mallampati scores (>II degree)*	13 (2.6)	125 (23.4)
Adenotonsillar hypertrophy	84 (17.0)	109 (20.4)
Respiratory diseases		
Allergic rhinitis	75 (15.2)	92 (17.2)
Sinusitis	18 (3.6)	29 (5.4)
Asthma	6 (1.2)	9 (1.7)
Recurrent respiratory tract infection*	95 (19.2)	158 (29.5)
Family information		
Parental snoring history*	84 (17.0)	145 (27.1)
Education (college level or above)	224 (45.3)	236 (44.1)
Parental obesity*	165 (33.3)	242 (45.2)

BMI body mass index

*p<0.01 compared with control group

The prevalence of obesity in our cohort of 3- to 11-year-old children was 9.5 %, and the prevalence of OSA was greater in obese than non-obese children with higher OAHI and sleep arousal. A novel finding was the differential age prevalence and risk factors for OSA in children with obesity. The main risk factors for OSA in preschool children were adenotonsillar hypertrophy and recurrent respiratory tract infection, but in school children the parental snoring history and obesity were the main causes for OSA.

The prevalence of obesity in children has dramatically increased worldwide in the last few decades. In the USA, the prevalence of childhood obesity doubled among children aged 6–11 years [26, 27]. According to the multi-center survey for Chinese children aged 6–18 years, the average prevalence of obesity in China had increased from 0.2 % in 1985 to 8.1 % in 2010 [32, 42]. For 3–5-year-old children, the prevalence of obesity is seldom reported. The present study found a prevalence of 9.5 %, which is comparable to studies that used the same definition and involved the same ethnic Chinese children of a similar age and geographical region. However, the prevalence of obesity was considerably lower than that in developed countries [26, 27]. A study involving Chinese children reported a rate of 14.2 % in Shanghai city, which is higher than in our study [16]. The differences in the results may be due to subject age differences or to different cutoffs for BMI, which in previous studies may also give rise to a wide range in the reported prevalence rate. A consistent finding in most studies is that boys have a higher prevalence of obesity,

Table 4 Sleep parameters of preschool and school aged children with and without obesity

Sleep parameters	Preschool children		p	School children		p
	Control (n=178)	Obesity (n=203)		Control (n=317)	Obesity (n=332)	
OAHI (/h)	1.5 (0.5–2.2)	2.3 (0.2–5.6)	0.086	1.4 (0.2–3.3)	5.8 (2.1–14.8)	0.002
OSA (%)	36/178 (20.2)	58/203 (28.6)	0.080	35/317 (11.0)	171/649 (26.3)	0.006
Arousal index (/h)	6.5 (4.0–8.5)	7.1 (5.2–10.8)	0.254	4.4 (3.2–7.3)	9.8 (6.8–15.9)	0.004
Sleep efficiency (%)	84.5 (78.5–91.5)	82.4 (73.5–90.0)	0.765	83.0 (80.5–92.2)	78.5 (76.1–88.2)	0.185
Sleep stage REM (min)	105 (85–116)	97 (72–125)	0.546	106 (83–122)	92 (83–105)	0.201
Sleep stage 1 (min)	35 (24–36)	38 (26–42)	0.155	32 (26–45)	29 (24–42)	0.158
Sleep stage 2 (min)	238 (205–258)	225 (165–254)	0.242	232 (198–258)	225 (189–235)	0.658
Slow wave sleep (min)	115 (95–135)	105 (82–126)	0.520	118 (96–126)	112 (98–141)	0.159
Total sleep time (min)	482 (448–525)	474 (392–532)	0.484	525 (450–586)	412 (388–468)	0.005
Sleep latency time (min)	19 (11–28)	22 (16–35)	0.185	18 (12–25)	25 (21–38)	0.112
WASO (min)	17 (12–25)	19 (13–28)	0.350	22 (12–36)	36 (21–48)	0.035

OAHI obstructive apnea hypopnea index, *WASO* wake time after sleep onset, p control vs. obesity

Table 5 Summary of items and factor loadings for three identified factors

Items	Factor loading	
	Preschool children	School children
Factor 1: nocturnal symptoms		
Restless sleep	0.775	0.725
Prone position	0.740	0.710
Bruxism	0.635	0.685
Night sweating	0.620	0.642
Enuresis*	–	0.586
Factor 2: morning/daytime symptoms		
Morning headache	0.687	0.680
Daytime fatigue	0.625	0.645
Difficulty getting up in the morning	0.610	0.629
Hyperactivity*	–	0.612
Factor 5: breathing symptoms		
Labored breathing	0.652	0.680
Witnessed apnea	0.635	0.620
Mouth breathing	0.585	0.612
Habitual snoring	0.564	0.590

*p <0.01 comparison between preschool and school aged children

and the prevalence increases with increasing age [2, 10, 15, 16, 29, 30; Tables 7 and 8].

Obese children may be at increased risk of OSA. The proportion of sleep-disordered breathing (SDB) has been found to be markedly increased among obese children [8, 36]. It has also been reported that a BMI of 1 kg/m^2 beyond the mean BMI for age and gender can increase the risk of OSA by 12 %. Similar trends demonstrating an increased risk of OSA among obese and overweight children have been reported from all over the world [7, 17, 41]. Our study showed a higher proportion of OSA in school children with obesity, which is consistent with most published studies. A 4-year follow-up study from Hong Kong reported that obesity is the most important risk factor for the development of OSA in children with primary snoring [24].

Because not all obese children will suffer from OSA, the question is which obese children with snoring will have OSA. The results from studies have shown that the risk factors for

Table 6 Association between independent factors and OSA in obese preschool and school aged children

Models	Preschool children (n=203)				School children (n=351)			
	OR	95 % CI		p	OR	95 % CI		p
		Lower	Upper			Lower	Upper	
Weight (kg)	0.58	0.41	0.78	0.18	0.69	0.51	0.91	0.12
BMI (kg/m^2)	0.86	0.62	1.25	0.17	0.92	0.53	1.68	0.08
BMI z-score	0.93	0.82	1.12	0.13	1.15	0.85	1.36	0.04
Boys	0.54	0.35	0.85	1.12	0.49	0.34	0.94	0.26
Mallampati scores>II degree	2.18	1.35	2.48	<0.01	2.45	2.19	2.85	<0.01
Adenotonsillar hypertrophy	3.52	2.58	3.96	<0.01	0.61	0.42	0.81	0.13
Recurrent respiratory tract infection	2.57	2.23	3.21	<0.01	0.87	0.54	1.05	0.21
Parental snoring history	0.87	0.65	1.15	0.12	2.45	2.15	3.42	<0.01
Parental obesity	0.85	0.56	1.24	0.24	2.12	1.58	2.92	<0.01
Nocturnal symptoms	1.38	1.14	1.62	<0.01	1.75	1.26	2.24	<0.01
Morning/daytime symptoms	2.63	1.85	3.18	<0.01	1.53	1.25	1.81	<0.01
Breathing symptoms	1.46	1.32	1.98	<0.01/	1.33	0.85	1.78	<0.01

BMI body mass index, *CI* confidence interval, *OR* odds ratio

OSA are variable due to differences in age and study design. It is accepted overall that the primary pathophysiologic mechanism involved in childhood OSA consists of adenotonsillar hypertrophy in the upper airway [14, 35], but several studies [4, 11, 21, 23] have failed to demonstrate the anticipated result. In our study, we demonstrated differential age risk factors for obese children in a large sample population including preschool and school children. School children with obesity were found to have higher OAHI than those without obesity ($p<0.01$), but there was no significant differences in preschool children. However, we did not find a significant linear correlation between BMI and OAHI in school children. Logistic regression models confirmed that the main risk factors for OSA in preschool children were adenotonsillar hypertrophy and recurrent respiratory infection, a result in agreement with most studies. It might be that the rapidly developed size of tonsils and adenoids in preschool-age children predisposed them to having complications with respiratory infections because of an immature immune function. In school children, we found the main risk factors were a history of parental snoring and obesity. This finding suggests that genetic factors might play a more important role in school children than in preschool children. This result is consistent with that from a previous study in which adenotonsillar hypertrophy played a smaller role for OAHI in obese children compared with non-obese children. Furthermore, a significant association between BMI z-score and Mallampati scores emerged for the whole cohort ($p<0.01$). The results suggest that Mallampati scores can reflect the true volume of pharyngeal cavity of the interactions between the multiple factors contributing to the upper airway collapsibility during sleep, such as neuromuscular response and other important anatomic and genetic factors [18, 28].

Several studies have reported the sleep characteristic in obese children with OSA, but because of different study designs and subjects, it is difficult to make comparisons. Our study randomly selected obese children aged 3–11 years and matched them with normal weight children by age, sex, and ethnicity. All the participants agreed to undergo the whole night PSG study. This sleep study demonstrated a higher prevalence of OSA in obese school children, and also found that they have higher OAHI and arousal as well as shorter total sleep time. This finding is compatible with published studies that suggest obese children with OSA tend to have worse nighttime sleep and excess daytime sleep. However, some studies did not show that children with OSA presented higher rates of obesity, or that obesity influenced its presentation clinically [19, 20, 22]. These studies with different results had probably been influenced by the characteristics of the studied population [3]. While most obese children appeared to have frequent arousal and short sleep duration, it has been reported that these are markers of emotional stress rather than a reflection of true sleep loss [39]. In our study, those obese children with greater sleep apnea and poor sleep may be the result of psychological distress and the interaction of the hypothalamic-pituitary-adrenal (HPA) axis and the pro-inflammatory cytokine response [40].

The relationship between obesity and sleep-related symptoms has also been reported in previous studies. Studies reporting both positive and negative associations have been published [31, 33]. This discrepancy may be explained by the differences in study design and sample size. In our study, a large sample of children underwent PSG, the current gold standard for diagnosing OSA, allowing us to accurately classify the subjects into OSA and non-OSA groups. We used factor analysis on various sleep-related symptoms, which were divided into three common factors. This study showed that the obese children with OSA were associated with morning or daytime symptoms, night symptoms, and respiratory symptoms. Further analysis found that the BMI z-score and the Mallampati scores were associated with OSA in school children, but not in preschool children. Nocturnal enuresis and hyperactivity during the daytime were also found to be important risk factors of OSA. These findings are consistent with our previous studies, as well as results reported in the literature [37].

There are certain limitations in our study. First, only the selected obese children and matched normal weight children completed our questionnaire survey. This could have introduced a degree of selection bias because parents whose children had sleep symptoms or problems with sleep were more likely to return the questionnaire, than parents whose children did not exhibit these symptoms or problems. Another potential selection bias is that a greater number of obese subjects with high risk factors for OSA underwent PSG in the second phase, despite our strict inclusion criteria. By comparing the demographic and socio-economic data of respondents and non-respondents, we could not find a significant difference between the two groups. Nonetheless, we believe that our study cohort still showed a good overall representation of the sample population. Second, the survey questionnaire was completed by parents, and very often, they did not sleep in the same room as their children, and therefore, would not know their actual sleep behavior. This is a known intrinsic problem associated with the use of questionnaire surveys. Despite this, a recent publication on preschool children showed a significant and independent association between parentally reported and objectively measured sleep symptoms [6]. Furthermore, the reliability of our screening questionnaire has been previously demonstrated [5].

The strength of this study is that it included a large number of subjects and used a validated, reliable, and locally applicable questionnaire. Furthermore, all subjects who agreed to take part in the second phase of this study underwent PSG to ascertain their OSA status, thus allowing an accurate comparison of the proportion of OSA between obese and normal weight children.

In summary, we found an obesity prevalence rate similar to that reported in studies carried out in ethnic Chinese children. We found a higher proportion of OSA in obese children, and a differential risk factor for OSA in preschool and school-aged children. The results suggest that different mechanisms may be involved in the development of OSA in preschool and school-age children with obesity.

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Contributors M-SS was the guarantor of integrity and conceived the study, participated in its design, carried out sleep studies, and prepared the manuscript; H-LZ participated in the literature research, performed the statistical analysis, and helped draft the manuscript; X-HC helped coordinate and design the study; YL participated in data acquisition and data analysis; P-NL selected the epidemiology survey and clinic data; Y-BZ and W-ZH carried out sleep questionnaire; C-CL and Y-FX were mentors for M-SS and helped the design, data analysis, and final revision of the manuscript. All authors read and approved the final manuscript.

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Appendix

Table 7 Published studies on prevalence of obesity in children using IOTF definition

Author, country, published year	Overweight (%)	Obesity (%)	Age (years old)
Paul Farajian, et al., Greece, 2011	29.6	11.8	10–12
Jiang XX, et al., Shanghai, China, 2011	25.7	14.2	8–15
Kumaravel V, et al., India, 2014	8.1	2.6	5–18
Rooster E, et al., Northern Europe, 2014		21	10–12
Abril V, et al., Developing countries, 2013	26	10.6	6–9
Emandi AC, et al., Romania, 2011	18.2	7.2	7–18
Lemelin L, et al., Quebec, 2010	16.6	3.1	5
Gulías-González R, et al., Castilla-La Mancha, 2012	26.7	11	6–11
Misra A, et al., India, 2011	14.4	2.8	8–18
Bingham DD, et al., Portugal, 2010	19.7	8.2	3–10
Salehi-Abargouei A, et al., Iran, 2013	10.8	2.2	11–15
Decelis A, et al., Maltese, 2013	20.4	14.2	10–11
Pwint MK, et al., Singapore, 2013	7.6	3.9	6–72 month
Raiah M, et al., Oran, 2011	10	3.1	6–11
Decelis A, et al., Albania, 2013		3.3	7–9.9

IOTF International Obesity Task Force

Table 8 Published studies on prevalence of obesity in children using WHO definition

Author, country (published year)	Overweight (%)	Obesity (%)	Age (years old)
Rajput N, et al., New Zealand, 2012	18.3	16.3	4
Hyska J, et al., Albania, 2013		7.7	7–9.9
Hernández-Herrera RJ, et al., Monterrey, 2014	15.5	29.9	0–14
Lasarte-Velillas, Zaragoza, 2014	12.75	11.2	2–14
Blake-Scarlettl BE, Jamaica 2009	10.6	7.1	6–10
Roberts KC, Canada, 2011		11.6	5–17
Jitnarin N, et al., Thailand, 2012	19	4.9	>3
Rito A, et al., Portugal, 2008	37.9	15.3	6–8
Lemelin L, et al., Quebec, 2010	26.3	6.2	5
Misra A, et al., India, 2011	18.5	5.3	8–18
Salehi-Abargouei A, et al., Iran, 2013	8.8	7.5	11–15
Decelis A, et al., Maltese, 2013	23.1	20.9	10–11

WHO World Health Organization

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