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SCIENTIFIC INVESTIGATIONS

Obesity and Risk of Sleep Related Upper Airway Obstruction in Caucasian Children

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Background: Obesity is thought to be a significant risk factor for upper airway obstruction during sleep in children. However, the moderating influences of age and ethnicity have not been well explored and the relative contribution of obesity per se to upper airway obstruction has yet to be quantified. Given the markedly increasing prevalence of childhood obesity, an objective understanding of the impact of obesity on upper airway obstruction is important. The purpose of the present study was to examine the interaction between obesity, age and upper airway obstruction in Australian Caucasian children referred for evaluation of snoring.

Methods: This was a retrospective case study involving 190 children (4-12 y) who were referred for evaluation of upper airway obstruction and underwent one night of polysomnography at the Adelaide Women's and Children's Hospital Sleep Disorders Unit. Children were classified as Infrequent Snorers (n = 80), Habitual Snorers (n = 68) or Obstructive Sleep Apnea Syndrome (OSAS) (n = 42) (i.e., obstructive apnea hypopnea index (OAHI) ≥ 1).

Results: Thirty-five percent (66/190) of children were overweight or obese. Body mass index but not age was a significant but weak predictor of OAHI (< 5% of the variance).

Conclusion: In Australian Caucasian children aged 4-12 years who snore, obesity but not age was a significant, albeit weak, predictor of upper airway obstruction during sleep.

Keywords: Obstructive sleep apnea syndrome, obesity, children, Caucasian.

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Childhood obesity is now recognized as a major pediatric health issue.[1] Over the past decade the prevalence of obesity in children has increased two- to threefold in the UK,[2] fourfold in USA and four- to sixfold in Australia.[3] It is associated with a range of known significant health and social problems (e.g., diabetes, hypertension, peer rejection)[4] and there is emerging evidence that childhood obesity may also be an important risk factor for obstructive sleep apnea syndrome (OSAS).[5] Moreover, the inflammatory effects of both obesity and upper airway obstruction may be additive.[6] However, the relationship between childhood obesity and OSAS may be more complex than previously thought.

In children the physiological effects of obesity on the respiratory system are believed to be similar to that of adults with an increased mass effect on the upper airway, deposition of fat in the pharyngeal muscles, decreased chest wall compliance, cephalad displacement of the diaphragm and blunting of central respiratory drive all increasing the potential severity of OSAS.[7] The rise in childhood obesity is therefore likely to predispose increasing numbers of children to OSAS.

Obesity in children may also be a risk factor for other sleep breathing disorders. Verhulst et al.[8] recently reported the intriguing finding that abdominal obesity was associated with central apnea (but not OSAS) in 91 overweight and obese children referred for evaluation of snoring. This work awaits further exploration.

Ethnicity is a recognized modulator of OSAS in children. For example, African American compared to Caucasian children are reported to be at greater risk of OSAS,[5,9,10] and while not directly compared with other groups, Asian children are thought to be at lower risk.[11] However, the detailed interaction, between ethnicity, obesity and OSAS has not been well explored. A summary of the literature exploring ethnicity and OSAS frequency in children is given in Appendix 1. In general, the studies have not specifically evaluated the relative contribution of ethnicity with most recruiting mixed ethnic samples. Inspection of the findings from studies which have largely sampled a single ethnic group would suggest that there is a modest association between obesity and OSAS in samples containing predominantly African American children,[12,13] mixed findings in Asian children with one group reporting no association (Chinese)[14] and two groups (both Chinese) a weak association[15,16] and little association in Caucasian children.[17,18] Given this background, the relative contribution of

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ethnicity remains to be more fully examined especially in Caucasian children where there is a paucity of data.

Age may also be an important factor modulating the impact of obesity on OSAS in children with an emerging consensus that the association may be stronger in older children.[19] Our review of the literature suggests that an association between obesity and OSAS is more often reported in older than younger children (Appendix 1). In the only study to date that has specifically examined the relationship between OSAS and obesity in different age groups, Stepanski et al[20] report that obesity was more frequent in older (8-12 years) compared to younger (< 8 years) children with OSAS.

Considering the findings of age and ethnicity in the genesis of OSAS in children an important question is once these factors have been controlled, what is the relative contribution of obesity to OSAS? The aim of the present study was to examine the interaction between obesity, age and upper airway obstruction in Australian Caucasian children referred for evaluation of snoring.

METHODS

Participants

This was retrospective case study. Consecutive healthy children aged 4 to 12 years who were referred for evaluation of snoring and possible OSAS to the Sleep Disorders Unit, Women's and Children Hospital, Adelaide, South Australia over the period October 1999 to December 2003 were included as participants in the present study. We excluded non-Caucasian children, children with specific syndromes (e.g., midface hypoplasia, Prader-Willi Syndrome), children prescribed psychotropic medication and children with a significant medical condition that could potentially result in hypoxemia and/or sleep fragmentation (e.g., uncontrolled asthma, cystic fibrous). A final total of 190 Caucasian children were selected, of which 55% were referred from respiratory medicine, 22% otorhinolaryngology, and 23% general pediatric clinics. The children were classified by polysomnography (PSG) and parental responses to the question "Does your child snore"? into three groups reflecting increasing severity: Intermittent Snorers (IS; normal PSG [OAHI < 1] and snoring < 3 nights per week, n = 80), Habitual Snorers (HS; normal PSG [OAHI < 1] and snoring ≥ 3 or more nights per week, n = 68) and Obstructive Sleep apnea Syndrome (OSAS; obstructive apnea and hypopnea index [OAHI ≥ 1], n = 42).

Established growth charts corrected for age and gender were used to determine body mass index (BMI) z-scores and classify overweight and obesity (BMI ≥ 85th and 95th percentile respectively.[21]

Because of previous evidence of an association between childhood OSAS and parental snoring,[22] smoking in the home[22] and socioeconomic status (SES)[23] these variables were also collected and covaried for in the analyses. Frequent snoring by either parent and smoking of cigarettes by any member of a household was determined by parental report. A measure of socioeconomic status (SES) was derived from the Australian Bureau of Statistics' Index of Relative Socio-economic Advantage/Disadvantage 2001 national census data (Australian Bureau of Statistics, Canberra, 2001). A higher score on this index indicates increased income and occupational skills and/or training within the geographical area of residence.

This study was approved by the Human Ethics Committee Women's and Children's Hospital Adelaide.

Polysomnography

Overnight PSG was conducted without sedation or sleep deprivation and began at each child's usual bedtime. A parent accompanied each child throughout the procedure. Polysomnography was performed using a computerized sleep data acquisition system (Compumedics S-Series Sleepwatch System, Melbourne, Australia). The following standard parameters were measured and recorded continuously utilizing the appropriate signal sampling and filtering protocols: electroencephalogram (EEG; C3-A2 or C4-A1), left and right electrooculogram (EOG), submental and intercostal electromyogram (EMG) with skin surface electrodes, leg movements by piezoelectric motion detection, heart rate by electrocardiogram (ECG), oronasal airflow by thermistor and nasal pressure, respiratory movements of the chest and abdominal wall using uncalibrated respiratory inductive plethysmography, arterial oxygen saturation (SpO_2) by pulse oximetry (Nellcor N2000) and transcutaneous CO_2 ($TcCO_2$) using a heated (41^{o}C) transcutaneous electrode (TINA, Radiometer Pacific). All data were digitized and stored on computer disk for subsequent analysis. Each child was continuously monitored and observed via infrared camera by a pediatric sleep technician who also documented observations of sleep behavior including the presence or absence of snoring.

Data Analysis

All polysomnograms were analyzed and scored manually by a sleep technician experienced and trained in analyzing pediatric sleep studies. The interscorer concordance (kappa) between two sleep technicians who randomly sampled a set of PSG used in this study was > 0.9. Sleep stages were scored in 30-sec epochs according to the standardized EEG, EOG, and EMG criteria.[24] Movement time was scored as a separate category and was not included in either sleep or awake time. Epochs were scored as movement if the EEG and EOG signals were obscured for > 50% of the epoch by muscle tension or artifact associated with movement of the subject.[24] Awake time refers to time spent awake during the recording period after initial sleep onset.

Respiratory variables were scored according to standard guidelines recommended for pediatric sleep studies.[25] Obstructive apneas were defined as the absence of airflow associated with continued chest and abdominal wall movement for duration of two or more respiratory cycles. Obstructive hypopneas were defined as a 50% to 80% reduction in the amplitude of the RIP and/or airflow signal associated with paradoxical chest/abdominal wall movement for duration of two or more respiratory cycles and associated with ≥ 3% oxygen desaturation. The presence of any other supportive data such as increased intercostal or submental EMG activity was also used to distinguish between obstructive and central hypopneas. The obstructive apnea and hypopnea index (OAHI) was calculated as the total number of obstructive apneas and obstructive hypopneas,

Table 1—Frequency (%) or Mean (95% CI) for Demographic and Sleep Variables According to Severity of Upper Airway Obstruction (Intermittent Snorer, Habitual Snorer and OSAS) with Chi-Square and F-Test Results

Demographic/Sleep variable	Severity of Upper Airway Obstruction						
	Intermittent Snorer (n=80)		Habitual Snorer (n = 68)		OSAS (n= 42)		
	n	%	n	%	n	%	Chi-square
Gender (male)	43	(54%)	40	(59%)	23	(55%)	0.47
Parental snoring	52	(65%)	44	(65%)	26	(62%)	0.07
Smoking in home	23	(29%)	22	(32%)	12	(29%)	0.28
	mean	95% CI	mean	95% CI	mean	95% CI	*F*-test
BMI z-score	0.63	0.40-0.86	0.67	0.42-0.92	1.26	0.87-1.61	5.07**
Age	7.20	6.66-7.74	6.54	5.88-7.20	6.93	6.11-7.74	1.20
Socioeconomic status	991	971-1011	972	955-989	958	932-983	2.52
Total sleep time (min)	439	430-448	426	414-437	422	405-437	2.67
Stage 1%	3.26	2.80-3.72	3.83	3.22-4.44	4.17	3.22-5.14	2.13
Stage 2%	45.2	43.8-46.6	45.4	43.8-47.1	43.0	41.3-45.2	1.62
SWS (3+4)%	30.6	29.1-31.8	30.6	29.0-32.3	31.6	29.6-33.3	0.33
REM%	20.9	19.9-22.0	20.1	18.9-21.3	21.1	19.8-22.5	0.85
REM latency (min)	103	92-115	112	98-127	122	106-138	1.70
OAHI	0.14	0.09-0.19	0.18	0.13-0.23	5.35	3.75-7.00	75.4****
OAHI-transformed[a]	0.91	0.88-0.94	0.88	0.84-0.90	0.25	0.20-0.29	353****
CAI	0.53	0.38-0.67	1.14	0.46-1.81	1.96	0.86-3.06	4.94**
CAI-transformed[a]	0.74	0.69-0.78	0.66	0.60-0.72	0.54	0.45-0.61	10.6****
Spontaneous Arousal Index	6.71	5.92-7.50	7.81	6.77-8.85	8.25	6.57-9.93	2.1
Respiratory Arousal Index	0.89	0.62-1.16	1.36	0.96-1.77	6.44	4.14-8.75	34.5****
SpO_2 nadir	93.0	92.3-93.8	92.3	91.5-93.1	88.0	86.1-89.6	24.6****

NB *denotes $p < 0.05$, **$p < 0.01$, ***$p < 0.005$ and ****$p < 0.001$. [a]Results for inverse functions indicate the opposite direction of actual severity, i.e., a smaller value indicates greater raw value and severity. OSAS = obstructive sleep apnea syndrome. Stage 1,2, SWS (stage 3+4 slow wave sleep) and REM sleep are presented as a percentage of total sleep time.

divided by the total sleep time and expressed as the number of events per hour of sleep. An OAHI > 1 was considered indicative of OSAS.

Spontaneous and respiratory arousals were scored according to the criteria of the American Sleep Disorders Task Force and are expressed as the total number of arousals per hour of sleep (spontaneous arousal index and respiratory arousal index).[25]

Central apneas were scored when there was an absence of respiratory effort to < 20% of baseline for ≥ 2 respiratory cycles in association with an absence of airflow and, as well, an absence of thoracoabdominal effort and reliable diaphragmatic EMG activity in the presence of ≥ 3% oxygen desaturation. The central apnea index (CAI) was calculated as the total number of central apneas divided by the total sleep time and expressed as the number of events per hour of sleep.

Statistical Analysis

Statistical analyses were conducted using SPSS version 14.0 for Windows (Chicago, IL). All p values reported are 2-tailed, with statistical significance determined at $\alpha = 0.05$. Data are presented as mean (95% CI) unless otherwise stated.

The severity of upper airway obstruction was entered as an independent variable (i.e., IS, HS, and OSAS) into a series of Chi-square analyses to explore group differences in categorical variables (gender, parental snoring, and smoking in the home). One-way analysis of variance (ANOVA) was used to test for group differences in the continuous variables, i.e., age, socioeconomic status, BMI z-score, and sleep variables. Games-Howell tests were used in post hoc analyses.

Apart from two exceptions all variables were normally distributed. The two exceptions were OAHI and CAI, which displayed significant positive skew and were, therefore, inversely transformed before entering into analyses [i.e. 1/(x+c), where x = data value and c = 1].[26] The transformed values are indicated as *OAHI-transformed* and *CAI-transformed*. Please note that lower transformed scores indicate more severe disease.

The relationship between Age and BMI z-score with demographic and sleep variables were explored using Pearson-r or in the case of ordinal data Spearman-rho correlational analyses and significance was tested using Fishers r-z transformations. Hierarchical regressions were used to explore the variance explained by age (step 1) and BMI z-score (step 2) in OAHI and, likewise, age (step 1) and BMI z-score (step 2) in CAI.

RESULTS

Heavier children were more likely to have OSAS. The percentage of overweight/obese was higher in OSAS (52% [22/42]) than either IS (30% [24/80]) or HS (29% [20/68]) children. Conversely, OSAS was more frequent in overweight/obese (22/66 [33%]) than normal weight (20/124 [16%]) children.

ANOVA revealed that OSAS children had a significantly higher BMI z-score than either IS or HS children (post hoc analysis: OSAS > IS = HS, $p < 0.05$) (Table 1). As expected, Respiratory Arousal Index scores were significantly higher (OSAS > IS = HS, $p < 0.05$) while OAHI-transformed (i.e. inverse OAHI) and SpO_2 nadir scores were significantly lower in OSAS than IS and HS children (both OSAS < IS = HS, $p < 0.05$). CAI-transformed (i.e. inverse CAI) scores were also

Table 2—Association between BMI Z-Score, Age, and Demographic and Sleep Variables (n=190)

Demographic/Sleep variable	Age	BMI z-score
Age (years)	-0.09	
BMI z-score	0.09	-
Socioeconomic status	-0.04	0.01
Parental snoring[a]	0.01	-0.02
Smoking in home[a]	0.10	0.04
Total sleep time (min)	-0.04***	-0.09
Stage 1%	0.11	0.09
Stage 2%	0.30****	-0.01
SWS%	-0.15*	-0.11
REM%	-0.30****	-0.11
REM latency (min)	0.16*	-0.02
(OAHI	-0.09	0.24***)
OAHI-transformed	0.04	-0.21**
(CAI	-0.04	0.16*)
CAI-transformed	0.19**	-0.18*
Spontaneous Arousals Index	-0.10	0.03
Respiratory Arousals Index	-0.03	0.19*
SpO_2 nadir	0.14	-0.17*

NB *denotes $p < 0.05$, **$p < 0.01$, ***$p < 0.005$ and ****$p < 0.001$. Stage 1, stage 2, SWS (slow wave sleep), and REM sleep are presented as a percentage of total sleep time. Spontaneous and Respiratory Arousals are number of events per hour of total sleep time. [a]Spearman correlation for ordinal data, remaining relationships Pearson-r correlations.

lower in OSAS than either IS and HS children (OSAS < IS = HS, $p < 0.05$). No significant group differences were observed in the remaining demographic or sleep variables.

Correlational analysis indicated that children with a higher BMI z-score demonstrated a significantly higher number of obstructive and central respiratory events during sleep, increased frequency of respiratory arousals and lower SpO_2 nadir (Table 2). As well, older age was associated with fewer central apneas.

To investigate the possible interaction between age with obesity and SDB, we divided the sample according to the criteria used by Stepanski et al (1999) into young (< 8 y) and old (≥ 8 y) children. Restricting our examination to children with obesity (n = 51), OSAS was more frequent in younger children (47% [14/30]) than older children (24% [5/21]). However, despite this trend the distribution of SDB subtypes was not significantly different between young and old children (chi-square = 2.9, $p > 0.05$).

As can be seen in Table 1, the Parental Snoring, Smoking in the Home and SES scores were comparable across the three SDB groups. Examination of the relationship between these variables and OAHI and, likewise, CAI revealed no significant correlations (all $p > 0.05$). A history of parental snoring, exposure to smoking in the home or lower SES were not associated with increased OAHI and CAI.

The regression results are summarized in Table 3. BMI z-score was predictive of upper airway obstruction. However, while significant, BMI z-score contributed only 4.5% of explained variance in OAHI-transformed. Age and BMI z-score were significant predictors of central apnea, but again only contributed a small percentage of the variance in CAI-transformed, 3.7% and 3.8% respectively.

Table 3—Hierarchical Regression Results with OAHI-Transformed and CAI-Transformed as Dependent Variables and Age (Step 1) and BMI Z-Score (Step 2) as Independent Variables

Variable	Regression Results				
	Step 1 β	Step 2 β	R^2	Adj R^2	R^2 change
OAHI-transformed					
Age	0.04	0.06	0.00	0.00	0.00
BMI z-score		-0.21***	0.05	0.04	0.04
CAI-transformed					
Age	0.19	0.21**	0.04	0.03	0.04
BMI z-score		-0.20**	0.08	0.07	0.04

NB *denotes $p < 0.05$, **$p < 0.01$ and ***$p < 0.005$.

DISCUSSION

In a large clinical sample of Australian Caucasian children undergoing overnight PSG for the assessment of snoring, those with confirmed OSAS had a greater body mass index than children with either intermittent or habitual snoring. Regression analyses revealed that increased body mass index was predictive of upper airway obstruction. However, the association was weak, with body mass index explaining less than 5% of the variance in obstructive apnea hypopnea index. This is comparable to findings reported by de la Eva et al[17] and Goodwin et al[18] in samples consisting mainly of Caucasian children. In the present study, increased body mass index together with younger age were also predictive of central apnea. Again however, the association was weak with age and body mass index together explaining less than 8% of the variance in central apnea index. Overall, the present findings suggest that obesity is a weak predictor of upper airway obstruction in Australian Caucasian children.

Recent reports suggest that older obese children are at higher risk for OSAS.[19] This is supported by inspection of the studies summarized in Appendix 1 where the relationship between obesity and OSAS is more frequently reported in studies performed in older compared to younger children. In contradistinction to these findings, age in the present study was not predictive of upper airway obstruction. The current literature examining the relationship between age, childhood obesity and upper airway obstruction is limited by several methodological shortcomings. These include small subject numbers a failure to normalize obesity for age and gender, lack of quantification of upper airway severity by full overnight polysomnography, lack of a comparison or control group and, in particular, a lack of control for ethnicity.[5,14,20,27-29]

In the present study we restricted our sample to Australian Caucasian children because they form the majority of the population seen in our sleep laboratory. Studies reporting an association between obesity and OSAS have included a relatively high percentage of African American and/or Asian children. Independent of obesity, African American children appear to be at higher risk of OSAS.[5,9,10] The relationship between obesity and OSAS is less well documented in Asian children, but Lam et al.[15] report a weak association in a sample of 480 Chinese children (r = 0.16). Unfortunately, to date, the interaction between obesity, ethnicity and OSAS has not been well explored and

while the percentage of obese children with OSAS is sometimes reported the percentage further divided by ethnicity is not (Appendix 1). Taken together, we suggest that ethnicity may be a more important modulator of increased risk of upper airway obstruction in obese children than has been previously assumed.

Like Verhulst and colleagues,[8] we found that body mass index was predictive of central apnea. In addition, we found that younger age was predictive of increased CAI severity. Nonetheless some caution is needed in the interpretation of these findings. Chest and abdominal movement recordings during PSG are particularly prone to artifact amongst obese patients. When increased abdominal adiposity is present and muscle function and respiratory drive are impaired, such as during REM, the ability to move the chest wall in response to an obstructed upper airway may be impaired.[30] Consequently, obstructive respiratory events may be erroneously scored as central events thereby underscoring the degree of obstruction.

A limitation of the present study is that we studied children referred to a sleep unit at a children's hospital. It is conceivable that obesity might be a differential factor for referral and this tendency would have increased the likelihood of overweight/obese children with suspected OSAS in our sample. We therefore reasoned that this would result in an increased likelihood of finding a relationship between obesity and OSAS. Thirty-five percent of our sample was overweight or obese. This is comparable to values reported elsewhere in a U.S. clinic sample.[6] In our "biased" sleep laboratory sample, we would have expected a higher prevalence of obese children with OSAS than was observed. Given this positive bias, the finding of only a weak relationship between childhood obesity and upper airway obstruction in our present study is therefore instructive. Nonetheless, because children referred to a sleep clinic may be dissimilar to the general population of children the present findings may not be applicable to the general pediatric community. A further limitation of the present study is that the contribution of regional adiposity was not evaluated. In adults with OSAS excess parapharyngeal fat is associated with OSAS severity [31] and whether the same applies to children is unclear. Finally, tonsillar size was not evaluated in the present study. Brooks et al[13] report a significant correlation between adenoidal-nasopharyngeal ratio and apnea duration while Lam et al[15] report a significant relationship between tonsillar size and OSAS severity. However, the relationship of tonsillar size to upper airway caliber during sleep may be the critical predictor of OSAS severity rather than tonsillar size per se.[32]

Socioeconomic status, parental snoring and smoking at home are reported to be associated with OSAS.[22,23] In the present study the mean and/or frequency of these factors were comparable across the three upper airway obstruction groups. In addition, lower socioeconomic status, a history parental snoring and smoking at home were not associated with increased disease severity. A similar pattern of results were observed for central apnea.

Recent studies have established that children who demonstrate UAO have increased upper airway collapsibility,[33] decreased upper airway volume,[32] but successful neuromuscular compensatory mechanisms which maintain airway patency during wakefulness and NREM sleep.[34] However, during obstructive events and REM sleep, upper airway tone is temporarily and dramatically reduced.[35] Further, developmental changes in upper airway tone may also play a significant role in upper airway obstruction as upper airway tone is reported to decrease with age.[36,37] No independent relationship between age and OSAS was demonstrated in the present study. The majority of studies reporting obesity to increase the risk of OSAS have recruited study participants with a mean age greater than 10 years.[12,16,38,40,41] Stepanski and colleagues[20] have shown that children displaying upper airway obstruction had significantly greater BMI than children with no symptoms only when older than 8 years. Rosen[10] in a sample of children with a mean age = 5.8 years, while finding an increased proportion of obese children amongst referrals to a sleep clinic, did not report any association between obesity and OSAS. If an age point exists at which OSAS becomes a risk, as opposed to not, this has yet to be established. However, it is not unreasonable to suspect developmental factors play an important intermediary role in the development of upper airway obstruction.

In conclusion, we found in a homogenous group of Australian Caucasian children referred to a sleep disorders clinic for suspected upper airway obstruction that obesity was a significant but weak predictor of OSAS. Given the dramatic recent increase in the prevalence of childhood overweight and obesity and the emerging literature suggesting that adenotonsillectomy may not result in the same degree of resolution of obstruction as in normal weight children this area of research will continue to require detailed study.

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Appendix 1. Comparison of Studies Investigating Obesity in Children and its Association with Upper Airway Obstruction

Author	Referral Source N (mean age ± SD)	Ethnicity	Definition of Obesity	Inclusion Criteria	% (n) Obese	% (n) Obese with OAHI ≥ 5	Relationship between obesity and apnea severity
Brooks et al.[13]	Hospital Clinic 17 (4.4 ± 0.8 y) RDI < 5 16 (5.1 ± 1.0 y) RDI ≥ 5	64% (21/33) AA 36% (12/33) Caucasian	Not reported	SDB	30% (5/16)	Not reported	Obesity correlated with RDI (r = 0.49)
Beebe et al.[38]	Obesity Clinic 22 (12.6 ± 1.7 y) Controls 60 (13.1 ± 1.8 y) Obese	55% (45/82) AA 41% (34/82) Caucasian 4% (3/82) Other	BMI > 95th %ile	Obesity	73% (60/82)	13% (8/60)	OR Obesity to OAHI > 1 = 6.6
Carroll et al.[39]	Hospital Clinic 48 (5.6 ± 3.4 y) OAHI < 1 35 (4.3 ± 2.4 y) OAHI ≥ 1	68% (56/82) AA 31% (25/82) Caucasian 1% (1/82)	BMI > 95th %ile	SDB	23% (19/83)	Not reported	Frequency of obesity same for OAHI < 1 & OAHI ≥ 1
Chay et al.[14]	Obesity Clinic 146 (6-18 y)	58% (85/146) Chinese 32% (47/146) Malay 8% (12/146) Indian 2% (2/146) Other	≥ 180% of IBW	Obesity or SDB	41% (60/146)	13% (8/60)	No significant association
de la Eva et al.[17]	Hospital Clinic 62 (10.9 ± 3.1 y)	80% (50) Caucasian 10% (6) Middle Eastern 7% (4) Asian 3% (2) Others	BMI > 95th %ile	Obesity and snoring	100% (62/62)	Not reported	No significant association
Dubern et al.[40]	Nutrition Clinic 54 (12.0 ± 2.6 y)	67% (36/54) Caucasian 33% (18/54) Black	BMI z-score>3 SD over mean (severe obesity)	Obesity	100% (54/54)	15% (8/54) RDI>10	Severe obesity correlated with RDI (r = 0.30)
Goodwin et al.[18]	Community 239 (6-11 y)	49% (117/239) Caucasian 51% (122/329) Hispanic	BMI > 95th %ile	Nil	12% (28/239)	Not reported	No significant association
Lam et al.[15]	Hospital Clinic 482 (median age = 6y; interquartile range 4-9 y)	100% (482/482) Chinese	BMI Z score > 1.96	SDB	30% (111/371)	49.5% (55/111)	Obesity correlated with (ln)AHI (r = 0.16). OR obesity to OAHI > 1.5 = 2.3
Mallory et al.[27]	Obesity Clinic 41 (10.3 ± 4.4 y)	Not reported	≥ 150% of IBW	Obesity and SDB	100% (41/41)	24% (10/41)	No significant association
Marcus et al.[12]	Hospital Clinic 22 (10.0 ± 5.0 y)	91% (20/22) AA 9% (2/22) Caucasian	> 120% of IBW or subscapular skinfold thickness > 85th %ile	Obesity	100% (22/22)	27% (6/22) obstructive apneas > 1/hr	Obesity correlated with obstructive apneas/h (r = 0.47)

Appendix 1 continues on next page.

Author	Referral Source N (mean age ± SD)	Ethnicity	Definition of Obesity	Inclusion Criteria	% (n) Obese	% (n) Obese with OAHI ≥ 5	Relationship between obesity and apnea severity
Reade et al.[41]	Hospital Clinic 90 (10.7: 4-18 y)	64.4% (58/90) AA 34.4% (31/90) Caucasian 1% (1/90) Hispanic	BMI > 95th %ile	SDB	62.2% (56/90)	53% (30/56) apnea Index > 1	Obesity correlated with OAHI (r = 0.29)
Rosen[10]	Hospital Clinic 326 (5.8 ± 3.0 y)	38% (137/326) AA 30% (98/326) Caucasian 31% (101/326) Hispanic	≥ 120% of IBW or BMI > 95th %ile	SDB	28% (91/326)	26% (24/91)	No significant association
Stepanski et al.[20]	Hospital Clinic 196 (5.9 ± 3.7y)	68% (133/196) AA 12% (24/196) Caucasian 19% (37/196) Hispanic 1% (2/196) Arabic	BMI > 95th ile	SDB	23% (47/198)	NR	Non-SDB BMI < SDB BMI (only for children > 8y)
Verhulst et al. [8]	Obesity Clinic 91 (11.2 ± 2.6 y)	Not reported	BMI > 95th ile	Obesity	70% (64/91)	8% (5/64)	No significant association
Wing et al. [16]	Obesity Clinic and Community 46 Obese (10.8 ± 2.3y) 44 Control (11.7 ± 2.1y)	100% Chinese	≥ 120% of IBW	Obesity	51.1% (46/90)	15% (7/46)	OR Obesity to RDI ≥ 5 = 1.2

To produce the table the terms "Obesity," "Children," and "Polysomnography" were entered as search terms in the PubMed database. From that search we identified 16 studies that report sufficient details to determine OAHI, ethnicity and frequency of obesity. Studies that used nonstandard polysomnographic protocols were excluded. Abbreviations: ADT = adenotonsillectomy, AA = African-American, BMI = body mass index, IBW = ideal body weight, SDB = sleep disordered breathing, RDI = respiratory disturbance index (total respiratory events per hour of sleep) and OR = Odds Ratio.