

Upper Airway Structure and Body Fat Composition in Obese Children with Obstructive Sleep Apnea Syndrome

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Rationale: Mechanisms leading to obstructive sleep apnea syndrome (OSAS) in obese children are not well understood.

Objectives: The aim of the study was to determine anatomical risk factors associated with OSAS in obese children as compared with obese control subjects without OSAS.

Methods: Magnetic resonance imaging was used to determine the size of upper airway structure, and body fat composition. Paired analysis was used to compare between groups. Mixed effects regression models and conditional multiple logistic regression models were used to determine whether body mass index (BMI) Z-score was an effect modifier of each anatomic characteristic as it relates to OSAS.

Measurements and Main Results: We studied 22 obese subjects with OSAS (12.5 ± 2.8 yr; BMI Z-score, 2.4 ± 0.4) and 22 obese control subjects (12.3 ± 2.9 yr; BMI Z-score, 2.3 ± 0.3). As compared with control subjects, subjects with OSAS had a smaller oropharynx ($P < 0.05$) and larger adenoid ($P < 0.01$), tonsils ($P < 0.05$), and retropharyngeal nodes ($P < 0.05$). The size of lymphoid tissues correlated with severity of OSAS whereas BMI Z-score did not have a modifier effect on these tissues. Subjects with OSAS demonstrated increased size of parapharyngeal fat pads ($P < 0.05$) and abdominal visceral fat ($P < 0.05$). The size of these tissues did not correlate with severity of OSAS and BMI Z-score did not have a modifier effect on these tissues. **Conclusions:** Upper airway lymphoid hypertrophy is significant in obese children with OSAS. The lack of correlation of lymphoid tissue size with obesity suggests that this hypertrophy is caused by other mechanisms. Although the parapharyngeal fat pads and abdominal visceral fat are larger in obese children with OSAS we could not find a direct association with severity of OSAS or with obesity.

Keywords: lymphoid hypertrophy; MRI; obese children

It is estimated that the prevalence of obstructive sleep apnea syndrome (OSAS) in obese children is much higher than the 1–4% reported in the general pediatric population (1). This is based on several observations. A large epidemiological study of children between 2 and 18 years of age noted that children with obesity had the highest risk to develop OSAS, with an odds ratio of 4.5 (2). Other studies support this finding: the occurrence of OSAS in unselected obese children undergoing polysomnography was 46% (3), and in morbidly obese children the reported rate was 55% (4). Last, when studying obese children referred for evaluation of OSAS, 59% had the disorder (5). It seems that children with metabolic syndrome, which is closely

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AT A GLANCE COMMENTARY

Current Scientific Knowledge on the Subject

The pathophysiology of obstructive sleep apnea syndrome (OSAS) in obese children is not well established. It is not known what anatomical changes occur and contribute to OSAS in this population.

What This Study Adds to the Field

This study enhances several new important aspects of the anatomical changes in the upper airway and body fat composition contributing to OSAS in obese children.

associated with obesity, are at particular risk for developing OSAS (6, 7). However, the anatomical and functional correlates leading to OSAS in obese children, and why some have the disorder whereas others do not, are not well understood and have not been studied systematically.

Several investigators emphasize the role of adenoid and tonsillar hypertrophy in the pathophysiology of the disorder in obese children (8–12). In addition, the American Academy of Pediatrics recommends considering adenotonsillectomy as the first treatment modality for obese children with OSAS (13). However, after adenotonsillectomy, residual OSAS continues in a significant number of children (8–12), suggesting that anatomical and/or functional factors may continue to restrict and/or allow restriction of the upper airway and lead to OSAS in these children.

In obese adults with OSAS, adenoid and tonsillar hypertrophy are not usually considered a risk factor. However, other soft tissues such as the soft palate, lateral pharyngeal walls, tongue, and parapharyngeal fat pads have been found to be larger and to restrict the upper airway (14–17). In addition, increased abdominal visceral fat has been shown to be associated with OSAS (18, 19), possibly by lowering lung volumes and promoting a smaller and more collapse-prone upper airway (20). Similar alterations in upper airway structure and body fat composition noted in obese adults should be considered in obese children with OSAS.

In this study, using magnetic resonance imaging (MRI), we performed a quantitative analysis of anatomical factors associated with OSAS in obese 8- to 17-year-old children. We hypothesized that the size of lymphoid tissues surrounding the upper airway is larger in the obese OSAS group as compared with obese control subjects, as found in young nonobese children with OSAS (21). Our secondary analysis examined other possible anatomical causes that have been previously noted in obese adults to determine whether these may play a role in the pathogenesis of the disorder in obese children, as well. Portions of this study have

been presented previously in abstract form at the 2010 American Thoracic Society International Conference, May 14–19, 2010, New Orleans, Louisiana (22).

METHODS

The study was approved by the Committee of Clinical Investigations at Albert Einstein College of Medicine (Bronx, NY). Informed consent was obtained from each subject and/or parent. Sequential obese subjects (body mass index [BMI] > 95th percentile for age and sex) (23) were recruited from the adolescent, endocrine, and general pediatric obesity clinics at the Children's Hospital at Montefiore (Bronx, NY). Subjects had normal development, intact adenoid and tonsils, and did not have a known metabolic or endocrine disorder other than prediabetes or type 2 diabetes. Determination of OSAS or control was made after overnight polysomnography in our hospital. Control subjects were matched to subjects with OSAS by age, sex, height, weight, BMI Z-score, Tanner stage, and ethnicity.

Overnight Polysomnography

Overnight polysomnography (XLTEK, Oakville, ON, Canada) was performed at the Sleep Disorders Center at the Children's Hospital at Montefiore. Sleep staging and scoring of arousals were performed according to standard criteria (24) by one blinded scorer. OSAS was determined if the obstructive apnea index was greater than 1/hour or if the apnea–hypopnea index (AHI) was greater than or equal to 5/hour (25) (for details on methodology *see* the online supplement).

Magnetic Resonance Imaging

MRI studies were performed in the Department of Radiology at the Children's Hospital at Montefiore within 4 weeks of recruitment and after polysomnography using a 16-channel Philips 3.0-tesla Achieva Quasar TX scanner (Philips Medical Systems, Best, The Netherlands). Subjects were awake during the period of imaging and were monitored continuously by pulse oximetry and direct observation. (For details of MRI methodology, sequences, and image processing *see* the online supplement).

Image Analysis

Image analysis was performed using Amira software (Version 4.1.1; Visage Imaging GmbH). Volumetric measurements were performed on each slice, using intensity threshold after normalization.

Upper airway structure. The volumes of the following structures were determined:

- A. Airway: The upper airway was subdivided to the following segments: *Nasopharynx*, defined as the region located superior to the level of the soft palate and continuous anteriorly, through the choanae, with the nasal cavities; *oropharynx*, defined as the region located between the level of the soft palate and the larynx, communicating anteriorly with the oral cavity, and having the posterior one-third of the tongue as its anterior border; and *hypopharynx*, defined as the region posterolateral to the larynx, and communicating with the cavity of the larynx through the auditus and including the pyriform recesses and the valleculae
- B. Lymphoid tissues: *Adenoid*, combined *palatine tonsils*, combined *retropharyngeal nodes* (defined as lymph nodes located between the internal carotid arteries from the base of the skull to the hyoid bone), and the combined *deep cervical lymph nodes* (defined as level II nodes, located along the internal jugular vein from base of the skull to the level of the hyoid bone)
- C. Tongue (including the genioglossus and geniohyoid muscles)
- D. Soft palate
- E. Mandible

Head, neck, and abdomen fat composition. Fat tissues of the head and neck included parapharyngeal and subcutaneous fat, and in the abdomen the visceral and subcutaneous fat compartments.

Data Analysis

Statistical analysis was conducted using SPSS version 18. Means and standard deviations were used to summarize continuous variables. For comparisons between the groups for demographics, polysomnography, and MRI data, we used two-tailed paired *t* tests, Wilcoxon signed rank tests, or McNemar's tests. Pearson correlations were derived between AHI and BMI Z-score and each anatomical variable. Mixed-effects regression models using AHI as a dependent variable, and conditional multiple logistic regression models with OSAS versus non-OSAS as the dependent variable, were derived to determine whether BMI Z-score was an effect modifier of each anatomic characteristic as it relates to OSAS. A *P* value less than 0.05 was considered significant.

RESULTS

Twenty-two obese children with OSAS (mean age, 12.5 ± 2.8 yr [range, 8.6–17.8 yr]; mean BMI Z-score, 2.4 ± 0.4) were compared with 22 obese control subjects without OSAS (mean age, 12.3 ± 2.9 yr [range, 8.5–17.8 yr]; mean BMI Z-score, 2.3 ± 0.3). There were no significant differences in demographic, anthropometric, and Tanner stage characteristics between groups (Table 1). One subject in the OSAS group had glucose intolerance and one had type 2 diabetes. None of the control subjects had prediabetes or type 2 diabetes.

Polysomnography

Subjects with OSAS exhibited mild–severe airway obstruction during sleep as compared with control subjects, having normal polysomnographic data (Table 2). There were significant differences between groups with respect to the following: AHI, 16.3 ± 12.8 versus 1.1 ± 1.1 events/hour (*P* < 0.001); SpO₂ (oxygen saturation as measured by pulse oximetry) nadir, 85.2 ± 6.1 versus 94.2 ± 2.9% (*P* < 0.001); and arousal index, 15.7 ± 12.1 versus 8.0 ± 4.2 events/hour (*P* < 0.05).

Magnetic Resonance Imaging

Upper airway structure. Volumetric analysis based on 3-mm slice thickness axial images of the upper airway structure is shown in Table 3. A three-dimensional depiction in four different views of the upper airway tissues of a subject with OSAS is presented graphically in Figure 1 (dynamic illustrations are shown in the online supplement as E Movie 1.1 and E Movie 1.2).

Airway: We noted a significantly smaller oropharyngeal airway in the OSAS group in comparison with the control group, 3.5 ± 1.7 versus 4.9 ± 2.0 cm³ (*P* < 0.05). No significant differences were noted with respect to the volume of either the nasopharynx or hypopharynx.

Lymphoid tissues: Except for the deep cervical nodes, all lymphoid tissues surrounding the upper airway were significantly

TABLE 1. DEMOGRAPHICS

	OSAS (n = 22)	Control Subjects (n = 22)	<i>P</i> Value
Age, yr	12.5 ± 2.8	12.3 ± 2.9	NS
Sex	M, 14; F, 8	M, 14; F, 8	NS
Height, cm	158.5 ± 17.7	158.8 ± 16.3	NS
Weight, kg	89.8 ± 35.2	84.6 ± 29.8	NS
BMI, kg/m ²	34.6 ± 8.3	32.4 ± 6.9	NS
BMI Z-score	2.4 ± 0.4	2.3 ± 0.3	NS
Tanner stage	3.4 ± 1.7	3.3 ± 1.6	NS
Ethnicity (AA/Hispanic/W/other)	12/8/1/1	12/7/3/0	NS

Definition of abbreviations: AA = African American; BMI = body mass index; F = female; M = male; NS = not significant; OSAS = obstructive sleep apnea syndrome; W = white.

Data shown represent means ± SD.

TABLE 2. POLYSOMNOGRAPHY

	OSAS (n = 22)	Control Subjects (n = 22)	P Value
Total sleep time, h	6.3 ± 1.5	6.2 ± 0.7	NS
Sleep efficiency, %	78.5 ± 16.5	84.3 ± 8.2	NS
Apnea index, events/h	3.1 ± 5.4	0.2 ± 0.4	NS
Apnea-hypopnea index	16.3 ± 12.8	1.1 ± 1.1	<0.001
Baseline SpO ₂ , %	98.8 ± 1.4	98.5 ± 1.2	NS
SpO ₂ nadir, %	85.2 ± 6.1	94.2 ± 2.9	<0.001
Baseline ETco ₂ , mm Hg	38.7 ± 7.7	41.2 ± 2.5	NS
Peak ETco ₂ , mm Hg	50.1 ± 4.4	47.6 ± 3.9	NS
Arousal index, events/h	15.7 ± 12.1	8.0 ± 4.2	<0.05

Definition of abbreviations: ETco₂ = end-tidal carbon dioxide; NS = not significant; OSAS = obstructive sleep apnea syndrome; SpO₂ = oxygen saturation as measured by pulse oximetry;

Data shown represent means ± SD.

larger in OSAS as compared with control subjects: the mean adenoid volume was 10.5 ± 4.4 versus 7.1 ± 3.6 cm³ ($P < 0.01$), the mean palatine tonsil volume was 10.1 ± 3.9 versus 7.8 ± 2.5 cm³ ($P < 0.05$), and the mean volume of the retropharyngeal lymph nodes was 4.7 ± 2.4 versus 3.1 ± 1.6 cm³ ($P < 0.05$). No significant differences were noted in the size of the tongue, the soft palate, or the mandible.

Regional fat distribution in neck and abdomen. Body fat composition is shown in Table 3. A three-dimensional depiction and axial, coronal, and sagittal views of the head and neck and abdomen are presented graphically in Figure 2 and Figure 3, respectively (respective dynamic illustrations are shown in the online supplement as E Movie 2 and E Movie 3). Accordingly, in the head and neck region, subjects with OSAS had significantly larger parapharyngeal fat pads, 10.6 ± 4.7 versus 8.4 ± 2.3 cm³ ($P < 0.05$), but no difference in head and neck subcutaneous fat. Similarly, subjects with OSAS had significantly more abdominal visceral fat, 1,434.9 ± 459.6 versus 1,101.4 ± 423.7 cm³ ($P < 0.05$), but no difference in abdominal subcutaneous fat.

Correlations between AHI and BMI Z-score and each of the anatomical variables are shown in Table 4. We noted a significant negative correlation between AHI and oropharyngeal size. A positive correlation was noted between AHI and the size of adenoid, tonsils, and retropharyngeal nodes with no

significant correlation with BMI Z-score. An illustration of the correlation between the AHI and the combined volume of the tonsils, adenoid, and retropharyngeal nodes is shown in Figure 4 ($r = 0.42$, $P < 0.01$). No significant correlations between the AHI and the size of the soft palate, tongue, mandible, and body fat composition were noted. BMI Z-score correlated significantly with the size of the tongue and mandible, and body fat tissues except abdominal visceral fat. Mixed-effects regression models and conditional multiple logistic regression models did not demonstrate that BMI Z-score was an effect modifier of anatomic characteristics as they relate to AHI or to OSAS versus subjects without OSAS.

DISCUSSION

Our study demonstrated several anatomical characteristics differentiating obese children with OSAS and obese children without OSAS. These include the increased size of upper airway lymphoid tissues and increased size of the parapharyngeal fat pads and abdominal visceral fat, noted in children with OSAS. However, our results did not demonstrate that variation in our subjects' BMI Z-score was an effect modifier on the above anatomical characteristics or severity of OSAS in these subjects.

To maximize the statistical power of comparisons between the two groups our study used matched pairs. We matched each obese subject with OSAS by age, height, weight, BMI Z-score, sex, and ethnicity with an obese control. Such matching serves to minimize the effect of confounding factors related to demographics and anthropometrics, which can be highly variable in children during growth and development.

When considering the various mechanisms leading to OSAS in obese children one should initially consider whether these may be related to an augmented effect of known causative factors resulting from their obesity and/or a distinct underlying pathophysiology of the disorder distinguishing it from OSAS in nonobese children. Because obesity is characterized by extensive somatic growth, we postulated, as previously described in younger nonobese children with OSAS (21), that older obese children with OSAS will have an excess of upper airway lymphoid tissues compared with obese subjects without OSAS.

In this study, in addition to comparing the size of the adenoid and tonsils, we have compared the difference in retropharyngeal

TABLE 3. UPPER AIRWAY STRUCTURE AND BODY COMPOSITION: VOLUMETRIC MEASUREMENTS

	OSAS (n = 22)	Control Subjects (n = 22)	P Value
Airway			
Nasopharynx	8.7 ± 3.5	9.8 ± 2.8	NS
Oropharynx	3.5 ± 1.7	4.9 ± 2.0	<0.05
Hypopharynx	3.2 ± 1.3	3.5 ± 1.5	NS
Lymphoid tissues			
Adenoid	10.5 ± 4.4	7.1 ± 3.6	<0.01
Tonsils	10.1 ± 3.9	7.8 ± 2.5	<0.05
Retropharyngeal nodes	4.7 ± 2.4	3.1 ± 1.6	<0.05
Deep cervical nodes	32.0 ± 11.9	26.8 ± 5.9	NS
Other tissues			
Soft palate	8.6 ± 2.9	8.6 ± 1.8	NS
Tongue	88.1 ± 17.0	89.7 ± 31.8	NS
Mandible	63.1 ± 12.7	64.9 ± 18.5	NS
Regional body fat composition			
Head and neck subcutaneous fat	483.2 ± 170.1	420.1 ± 111.0	NS
Neck parapharyngeal fat	10.6 ± 4.7	8.4 ± 2.3	<0.05
Abdominal subcutaneous fat	6,939.5 ± 3,115.0	5,916.9 ± 2,289.2	NS
Abdominal visceral fat	1,434.9 ± 459.6	1,101.4 ± 423.7	<0.05

Definition of abbreviations: NS = not significant; OSAS = obstructive sleep apnea syndrome.

Data shown represent mean cubic centimeters ± SD.

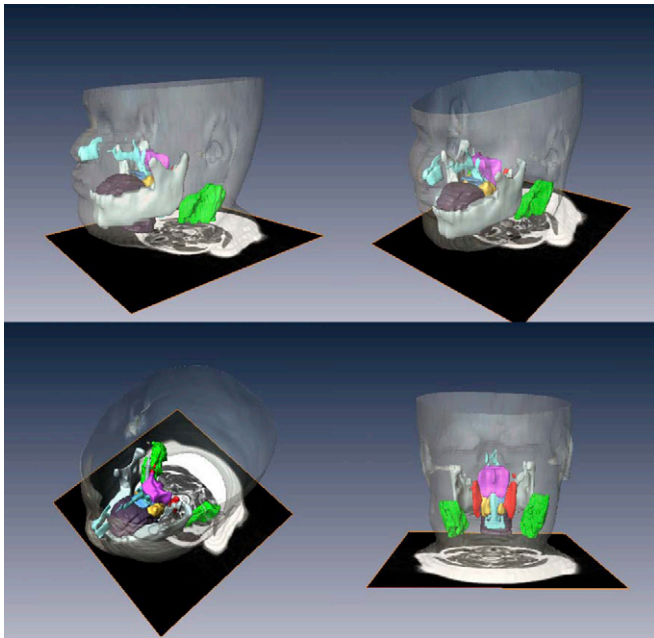


Figure 1. Upper airway, soft tissues, and mandibular reconstructions. Head and neck surface rendering with three-dimensional reconstructions of the upper airway, soft tissues, and mandible of a subject with obstructive sleep apnea syndrome in various views: lateral (*top left*), anterior oblique (*top right*), superior oblique (*bottom left*) and posterior (*bottom right*). Airway (*light blue*), tongue (*brown*), mandible (*white*), soft palate (*blue*), tonsils (*yellow*), adenoid (*magenta*), retropharyngeal nodes (*red*), and deep cervical nodes (*green*).

nodes, which may also contribute to reduced airway volume. The retropharyngeal nodes are defined as lymph nodes located between the internal carotid arteries from the base of the skull

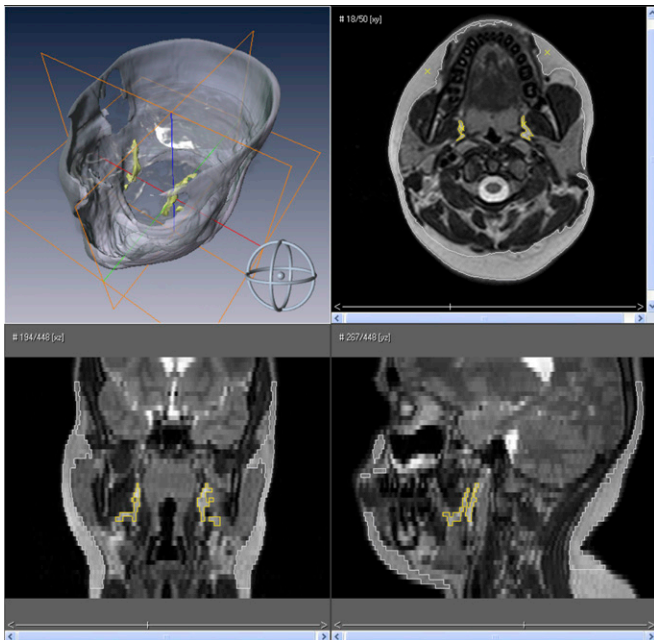


Figure 2. Head and neck fat composition. Surface rendering of the head and neck with three-dimensional reconstructions of the subcutaneous fat (*gray*) and parapharyngeal fat pads (*yellow*) of a subject with obstructive sleep apnea syndrome (*top left*), midtonsillar axial view (*top right*), coronal view (*bottom left*), and sagittal view (*bottom right*).

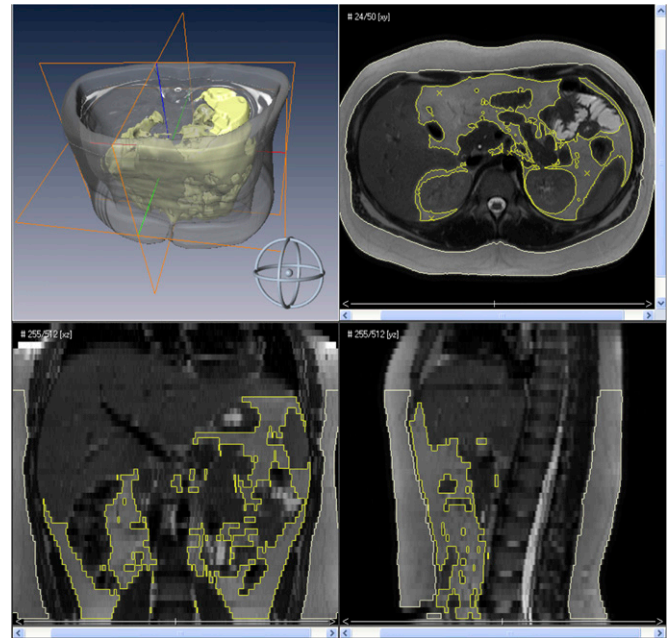


Figure 3. Abdominal fat composition. Surface rendering of the abdomen with three-dimensional reconstructions of the subcutaneous fat (*gray*) and visceral fat (*yellow*) of a subject with obstructive sleep apnea syndrome (*top left*), axial view (*top right*), coronal view (*bottom left*), and sagittal view (*bottom right*).

to the hyoid bone. The afferents of these nodes drain the pharyngeal nasal cavity, adenoid, paranasal sinuses, and the auditory tubes and their efferents pass to the deep cervical lymph nodes. Because of the proximity of the retropharyngeal nodes to the nasopharynx and oropharynx, and because no rigid anatomic barrier lies between these nodes and the upper airway, the retropharyngeal nodes may contribute directly to reduction of the volume of the upper airway when they are enlarged. This is the first study to examine how these nodes contribute to airway restriction. Although we have focused in this study on obese children, these nodes may play a similar role in nonobese children with OSAS as well.

Several important relationships that enhance our understanding about the pathophysiology of OSAS in obese children have been observed in our study: first, the larger size of the adenoid (48%), tonsils (29%), and retropharyngeal nodes (54%) in the obese OSAS group, which is not offset by other soft tissue volumes being reduced, could explain the 28% reduction in oropharyngeal size, as compared with control subjects. It should be noted that the oropharynx loses 1.4 cm³ whereas lymphatic tissue gains 7.3 cm³; however, not all the lymphatic tissue actually impinges into the oropharyngeal space. This difference, measured during wakefulness, may predispose to upper airway obstruction during sleep. The oropharyngeal airway (velopharynx) has been identified to be an area of vulnerability to airway collapse in both children (26, 27) and adults (28, 29). The velopharynx in children has also been described as the “overlap region” (27, 30), because it is where the lower portion of the adenoid and upper poles of the tonsils overlap with each other and the soft palate. Second, the size of each of the upper airway lymphoid tissues significantly correlates with severity of OSAS whereas BMI Z-score did not have an effect modifier on these tissues. This suggests that lymphoid proliferation as it relates to OSAS is independent of obesity and could possibly be linked to other conditions such as local or systemic inflammation described in nonobese children with

TABLE 4. CORRELATION COEFFICIENTS BETWEEN UPPER AIRWAY STRUCTURE AND BODY COMPOSITION VERSUS OBSTRUCTIVE SLEEP APNEA SEVERITY AND BODY MASS INDEX

	AHI (n = 44)		BMI Z-Score (n = 44)	
	r	P Value	r	P Value
Airway				
Nasopharynx	-0.261	NS	0.346	<0.05
Oropharynx	-0.297	<0.05	0.076	NS
Hypopharynx	-0.122	NS	0.075	NS
Lymphoid tissues				
Adenoid	0.359	<0.05	-0.165	NS
Tonsils	0.307	<0.05	-0.070	NS
Retropharyngeal nodes	0.371	<0.05	0.072	NS
Deep cervical nodes	0.02	NS	0.404	<0.01
Other tissues				
Soft palate	-0.181	NS	0.053	NS
Tongue	-0.153	NS	0.325	<0.05
Mandible	-0.114	NS	0.292	<0.05
Body fat				
Head and neck subcutaneous fat	0.255	NS	0.649	<0.001
Neck parapharyngeal fat	0.038	NS	0.526	<0.01
Abdominal subcutaneous fat	0.287	NS	0.682	<0.001
Abdominal visceral fat	0.153	NS	0.272	NS

Definition of abbreviations: AHI = apnea-hypopnea index; BMI = body mass index; NS = not significant.

OSAS (31–33) as well as in obese children with the disorder (34). Third, the soft palate, tongue, and mandible did not differ in size between groups or correlate with OSAS severity. However, the tongue and mandible did correlate with BMI Z-score. Thus, these tissues may not contribute to OSAS in obese children as has been reported in adults (16, 35). It is possible that these tissues develop as risk factors later in life and contribute to the adult form of OSAS.

Regarding body fat composition, we found significant differences between groups. On average, children with OSAS had a 28 and 30% increase in size of the parapharyngeal fat pads and abdominal visceral fat, respectively. However, we could not demonstrate a significant correlation between severity of OSAS and alterations in body fat composition, or establish BMI Z-score as an effect modifier on these tissues. These findings could reflect the nature of our design in that only obese subjects within a narrow range of BMI Z-score (median, 2.4; range, 1.7–

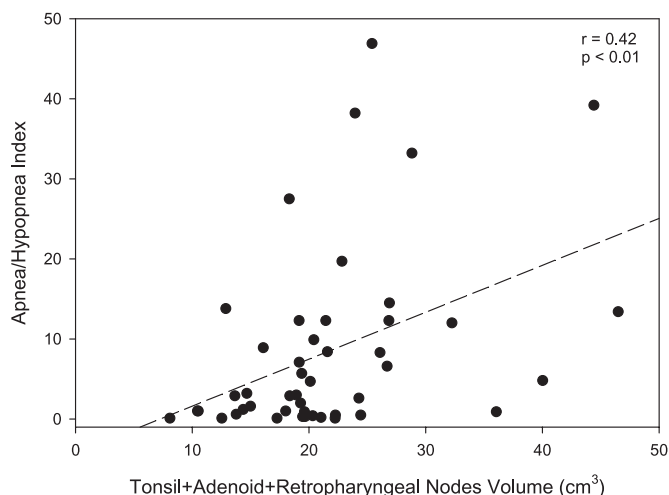


Figure 4. Correlation between apnea-hypopnea index and combined upper airway lymphoid size (cm³): $r = 0.42$, $P < 0.01$.

3.1) were included. Thus, it would be important to confirm these findings in a larger scale study.

Nevertheless, the finding of increased parapharyngeal fat pad size contributing to airway restriction in subjects with OSAS in this study is similar to that noted in obese adults with OSAS (14, 17), but not in nonobese adults (15) or children with the disorder (21). Similarly, increased abdominal visceral fat in our subjects with OSAS parallels the observation in obese children with metabolic syndrome and OSAS that used an indirect anthropometric measure of waist circumference (6). Thus, increased visceral fat in the neck and body has several important clinical implications in relation to OSAS. Visceral fat can directly affect chest wall and upper airway mechanics by reducing functional residual capacity, making such subjects more vulnerable to hypoxemia during sleep (20). Such a reduction of lung volume could also predispose to airway collapse by reducing tracheal traction and airway stability (36). In addition, visceral obesity is strongly associated with the proinflammatory and prothrombotic conditions that increase risk for insulin resistance, type 2 diabetes, atherosclerosis, and OSAS (37). On the other hand, studies have demonstrated that OSAS can independently induce metabolic syndrome by decreasing insulin sensitivity in both animal and humans (34, 38–40).

Although we noted that one subject in the OSAS group had glucose intolerance and one had type 2 diabetes, and none of the control subjects had evidence of prediabetes or type 2 diabetes, our study was not designed to correlate between OSAS, anatomical measures, and the biomarkers of metabolic syndrome, such as insulin resistance, dyslipidemia, inflammation, and so on. Therefore, we are limited in drawing conclusions regarding this possible association. Nevertheless, we suggest that the two groups emerging from the present study with similar BMI Z-score and that were *a priori* selected according to the existence or nonexistence of OSAS, present distinct phenotypes of childhood obesity: the first, with marked visceral adiposity, upper airway lymphoid hypertrophy, and OSAS, and a second with less profound visceral adiposity, smaller upper airway lymphoid tissues, and no evidence of OSAS. We speculate that the first phenotype is more prone to develop metabolic syndrome and its sequelae because OSAS was shown to independently lead to the disorder in both children and adults (6, 34, 38).

In summary, this study noted generalized overgrowth of upper airway lymphoid tissues in obese children with OSAS, restricting their upper airway. However, because surgical modalities, particularly adenotonsillectomy, in obese children often do not completely ameliorate OSAS (8, 11, 12), a prospective randomized control trial evaluating outcomes of this approach versus other treatment alternatives such as noninvasive ventilation and weight management would be helpful. Such a study should also include the metabolic outcomes linked to obesity and OSAS, such as insulin resistance and metabolic syndrome.

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