

Risk Factors for Sleep-disordered Breathing in Children

Associations with Obesity, Race, and Respiratory Problems

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This study examined risk factors for sleep-disordered breathing (SDB) in children and adolescents; specifically, quantifying risk associated with obesity, race, and upper and lower respiratory problems. Subjects were participants in a genetic-epidemiologic study of SDB and included 399 children and adolescents 2 to 18 yr of age, recruited as members of families with a member (a proband) with known sleep apnea (31 index families) or as members of neighborhood control families (30 families). SDB was assessed with home overnight multichannel monitoring and SDB was defined based on an apnea-hypopnea index ≥ 10 (moderately affected) or < 5 (unaffected). SDB of moderate level was significantly associated with obesity (odds ratio, 4.59; 95% confidence interval [CI], 1.58 to 13.33) and African-American race (odds ratio, 3.49; 95% CI, 1.56 to 8.32) but not with sex or age. After adjusting for obesity, proband sampling, race and familial clustering, sinus problems and persistent wheeze each independently (of the other) predicted SDB. These data suggest the importance of upper and lower respiratory problems and obesity as risk factors for SDB in children and adolescents. Increased risk in African Americans appears to be independent of the effects of obesity or respiratory problems. **Redline S, Tishler PV, Schluchter M, Aylor J, Clark K, Graham G. Risk factors for sleep-disordered breathing in children: associations with obesity, race, and respiratory problems.**

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Sleep-disordered breathing (SDB) refers to the occurrence of repetitive episodes of complete or partial obstruction of the upper airway during sleep, usually in association with loud snoring and daytime sleepiness. Episodes of upper airway obstruction often are associated with arousals, sleep fragmentation, intermittent hypoxemia and hypercapnia, and nocturnal hypertension. The long-term consequences of SDB may include sustained daytime hypertension and increased cardiovascular and cerebrovascular morbidity and mortality. Relatively recent epidemiologic data have shown the disorder to be prevalent in adults, affecting $> 9\%$ of those 30 to 60 yr of age (1). Much less data are available for children. Despite an increased clinical experience with diagnosing and treating children with snoring and upper airway obstruction, there has not been an appreciable increase in knowledge regarding the overall prevalence of clinically significant SDB in children, risk factors for the disorder, and consequences of SDB of various degrees of severity. Characterization of risk factors for SDB could help identify susceptible children, which may pro-

vide insight into disease pathogenesis and help formulate screening strategies.

In this study, we assessed putative risk factors for SDB in a large sample of children, specifically quantifying the independent risks associated with obesity, sex, race, and upper and lower respiratory problems.

METHODS

Sample

The study population consisted of children 2 to 18 yr of age who participated in the Cleveland Family Study, a community-based genetic-epidemiologic study of sleep apnea. The larger cohort consists of index and control families, recruited using methods previously detailed (2, 3). Briefly, "index" families were identified because one member (the proband) had laboratory-confirmed sleep apnea. Neighborhood control families were chosen randomly from a list of names provided by the index proband of neighbors/friends with living relatives who resided in the area. *In toto*, 399 children 2 to 18 yr of age were identified who had a technically acceptable overnight sleep study. Of these children, 273 were members of 31 index families and 126 were members of 30 control families.

Protocol

All subjects were studied in their homes, where they were visited by a trained technician who distributed and administered questionnaires, made physical measurements, performed spirometry, and provided instruction on the use of an overnight sleep monitor. For subjects ≥ 13 yr of age, medical history, medication use, family history, and symptoms were assessed with the Sleep and Health Modified SCOR Questionnaire (4). This instrument included questions pertaining to respiratory symptoms and illnesses that are identical or similar to those

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used in the American Thoracic Association-DLD Standardized Respiratory Symptom Questionnaire for children (5). A modified version of this questionnaire, completed by the child's primary caretaker, was used for younger children. Height, neck circumference, and weight were measured directly. Spirometry was performed in a sitting position and with the use of noseclips, using a portable spirometer (Multi-Spiro, Irvine, CA). Three acceptable forced vital capacity maneuvers were obtained (6). Overnight sleep monitoring was accomplished with an Edentrace I or II monitor (Edentec, Inc., Eden Prairie, MN), with measurement of airflow (nasal/oral thermistry), chest wall impedance, finger pulse oximetry, and heart rate, as described previously (7).

Data Extraction

Pulmonary symptoms, illnesses, and exposures were identified by the following criteria.

Asthma. The child was reported as having had asthma and the diagnosis was reported to have been confirmed by a doctor.

Occasional wheeze. The child's chest was reported to sound "wheezy or whistling" during the last year, and this occurred at times other than "only with colds."

Persistent wheezing. Wheezing or whistling was reported as occurring "most days or nights."

Cough. Report of cough occurring "usually" during the previous year.

Sinus problems, hay fever. Reported to have occurred any time during the child's life.

Bronchitis. The child was reported to have "chronic bronchitis."

Maternal passive smoking. The child's mother reported smoking at least one cigarette per day during the previous month.

Body mass index (BMI) was computed by dividing weight (in kilograms) by height-squared (in meters). Obesity was considered present if the BMI was $> 28 \text{ kg/m}^2$ (8).

The FEV₁ and FVC were derived from the FVC maneuver with greatest summed value of FVC and FEV₁ that also met ATS standards of acceptability (6). The percent predicted values were based on published regression equations, with adjustments for age, height, and sex (9).

From the sleep studies, respiratory events were defined as cessations (apneas) or discrete reductions (hypopneas) in airflow or chest impedance, lasting ≥ 10 s and associated with $\geq 2.5\%$ fall in oxygen saturation. Sleep time was estimated from visual inspection of the sleep record, correlated with a sleep diary completed by the subject or the subject's parent. The apnea-hypopnea index (AHI) was determined by dividing the number of respiratory events by the estimated sleep time. The AHI measured with this approach has been shown previously to correlate excellently with in-laboratory polysomnography, which includes EEG, EOG, and EMG measurement (7). In adults, and more so in children, there is no consensus on what threshold level of AHI best identifies "affection," with levels of 5, 10, 15, 20, or 30 used in different studies and clinical settings (reviewed in [10]). It is clear that such thresholds need to be modified according to the method of monitoring and definitions of hypopnea (10). For this report, we chose an AHI threshold of 10 to identify a group with clearly increased apneic activity (SDB cases). This threshold is higher than what is often considered abnormal in clinical settings (> 5) and, thus, probably identifies a group with at least a moderate degree of SDB. Support for this threshold is derived from our previous work, which has shown that children with an AHI at least this high have significantly more symptoms of SDB (habitual snoring, nocturnal gasping/snorting, and sleepiness) than do children with lower AHIs (11). An AHI level < 5 was chosen to identify a control group with "minimal" or no SDB. To minimize misclassification, subjects with AHI levels between 5 and 10 were not included in analyses in which SDB was the outcome variable ($n = 35$).

Statistical Analyses

Comparisons of the distribution of demographic and risk factors according to group membership (either index/control family or SDB present/absent) were made with unpaired *t* tests (continuous variables), with *p* values adjusted for unequal variances when appropriate, or chi square analyses (dichotomous outcomes). The tables present continuous variables as means \pm standard deviations (SDs) or as me-

dians and the interquartile values (for non-normally distributed values). The *p* values for AHI and BMI were based on analyses of their natural logarithms, the distributions of which approximated normality. To account for the use of data on family members, in which outcomes may be correlated, a generalized linear model for correlated data was implemented using Generalized Estimating Equations (Proc Genmod, SAS PC 6.11; SAS Institute, Cary, NC) This method models the effects of covariates on marginal response probabilities using an appropriate link function (the logistic for binary data) (12). In these models, the family unit was considered the unit of clustering; additionally, an indicator variable was included to account for differences in recruitment sources (index family or control).

RESULTS

The distributions of physical and demographic characteristics and of risk factors in the study population are shown in Table 1 for children recruited as members of index families and neighborhood control subjects. Approximately one half of the sample was female. Twenty-seven percent of the sample was African-American, reflecting the racial makeup of the greater Cleveland area. The majority of the remaining children were white (the others consisted of three Hispanics, one Asian, and 14 "others"). Almost 13% of children had had a history of doctor-confirmed asthma and 5% reported persistent wheeze, percentages consistent with prevalence estimates for children of similar age and racial makeup (13). Subjects from the neighborhood control group and index families did not differ with respect to age, sex, or asthma prevalence. The frequency of obesity and sinus problems was greater in subjects from index families. SDB, based on an AHI of ≥ 10 , was observed in 1.6% of children from neighborhood control families, and in 8.4% of children from index families. The former is consistent with estimates of SDB based on two-sampling procedures from general population samples (14, 15). The latter is consistent with the 3- to 4-fold excess risk of SDB in members of families identified by a proband with SDB, as we have reported previously (16). Of the children with SDB, sleep apnea had been diagnosed in only two prior to study participation (i.e., the majority of children were found to have SDB as a result of their family's study participation.) No child had obvi-

TABLE 1
POPULATION CHARACTERISTICS OF CHILDREN < 18 yr
OF AGE, RELATIVES OF CONTROL SUBJECTS,
AND AFFECTED PROBANDS*

	Relatives of Control Subjects (<i>n</i> = 126)	Relatives of Affected Probands (<i>n</i> = 273)	<i>p</i> Value
Female, %	53.2	50.9	NS [‡]
African American, %	19.1	30.4	0.017
Age, yr	10.7 \pm 3.9	11.1 \pm 4.3	NS [‡]
BMI, kg/m ²	18.5 (16.9–21.8)	19.9 (16.4–24.0)	0.047 [†]
Obese (BMI > 28), %	4.8	11.7	0.031
History of asthma, %	13.5	11.4	NS [‡]
Occasional wheeze, %	19.1	19.8	NS [‡]
Persistent wheeze, %	5.6	4.8	NS [‡]
Sinus problems, %	13.9	23.4	0.030
Chronic Bronchitis, %	8.1	9.5	NS [‡]
AHI, events/h	1.37 (0.7–2.8)	1.77 (0.7–3.6)	NS ^{††}
AHI > 5 , %	10.3	17.2	0.073
AHI > 10 , %	1.6	8.4	0.009

Definition of abbreviations: AHI = apnea/hypopnea index; BMI = body mass index.

* Values of continuous variables are expressed as means \pm SD, or as medians (interquartile ranges) for non-normally distributed variables. See text for definition of disease/symptoms.

[†] Based on log-transformed values.

[‡] Not significant; *p* > 0.10 .

TABLE 2
DEMOGRAPHIC AND ANTHROPOMETRIC FEATURES
ACCORDING TO SLEEP-DISORDERED BREATHING*

	AHI < 5 (n = 339)	AHI > 10 (n = 25)	p Value
Female, %	57.5	48.0	NS [†]
African American, %	23.6	56.0	0.001
Age, yr	10.8 ± 4.2	10.5 ± 3.6	NS
BMI, kg/m ²	18.8 (16.4–22.6)	22.6 (17.7–28.8)	0.011 [†]
Obese (BMI > 28), %	6.7	28.0	0.001
Neck circumference, cm	29.8 ± 4.0	33.3 ± 4.6	0.006
Related to an Index Proband, %	66.7	92.0	0.009

For definition of abbreviations, see Table 1.

* Values of continuous variables are expressed as means ± SD, or as medians (interquartile ranges) for non-normally distributed variables.

[†] Based on log-transformed values.

[‡] Not significant; p > 0.10.

ous facial or congenital anomalies, other than one child with Down's syndrome.

A comparison of demographic and anthropometric factors in groups without and with moderate SDB (AHI < 5 and ≥ 10) is shown in Table 2. Groups did not differ in their sexual makeup. A larger proportion of children with moderate SDB than without SDB were African-American (56 versus 24%, p = 0.001). Children with SDB also had greater BMIs and had larger neck circumferences than did children without SDB. A majority of children with SDB were from families with another affected member. Age did not differ between affected and unaffected groups.

The distribution of upper and lower respiratory conditions in children with and without SDB is shown in Table 3. Children with SDB significantly more frequently reported or were reported to have had: usual cough, occasional wheeze, persistent wheeze, sinus problems, and doctor-diagnosed asthma. FEV₁ and FVC levels, which were technically acceptable for only 163 children (mean age, 11.6 yr), tended to be lower in the group with SDB, but these differences were not statistically significant.

Although obesity and sinus and respiratory problems were related to SDB, obesity was not significantly associated with either sinus problems (20% of nonobese children were reported to have sinus problems as compared with 25% of obese

children) or to asthma/wheeze (e.g., 5% of nonobese children had persistent wheeze compared with 3% of obese children).

The results of the analyses that adjusted for the familial correlations within the data and also for recruitment sources (neighborhood control or index family) are presented in Table 4. Significant associations exist between SDB and obesity, African American race, sinus problems, wheeze (occasional or persistent), cough, and doctor-diagnosed asthma. Obese children (defined by a BMI > 28) were four to five times more likely to have SDB than were nonobese children, and African Americans were approximately three and a half times more likely to have SDB than were other children. After additionally adjusting for obesity and African American race, significant relationships between SDB and respiratory symptoms and illnesses persisted. The strongest relationship was between SDB and usual cough; however, this symptom could be assessed only in children < 13 yr of age (the version of the questionnaire for older subjects did not contain this question). Persistent wheeze more strongly predicted SDB than occasional wheeze (adjusted odds ratio, 7.45 versus 3.29 for persistent and occasional wheeze, respectively). Children with doctor-diagnosed asthma were estimated to be at an almost 4-fold increased risk for SDB than were nonasthmatic children. A history of sinus problems also increased risk of SDB approximately five fold. Age did not significantly predict SDB.

Although sinus problems and asthma may occur in the same individual (17), a significant association between these two conditions was not seen in our data (sinus problems were reported by 27% of children with asthma and by 19% of the nonasthmatics; p > 0.25). To evaluate the extent to which upper (sinus) as compared with lower (asthma/wheeze) respiratory problems contributed to an excess risk of SDB, we assessed the extent to which each condition independently predicted SDB using a model that included obesity, race, recruitment source, sinus problems, and persistent wheeze as covariates, and SDB as the outcome. In this model, the adjusted odds ratio for sinus problems was 5.21 (95% confidence interval [CI], 1.66 to 16.12), and for persistent wheeze it was 8.76 (95% CI, 1.28 to 60.27), suggesting that each condition independently predicted SDB.

The relationship of race to SDB, independent of obesity is provided in Table 4 (second line, third and fourth columns). The relationship of race to SDB persisted when additionally considering the effects of asthma (odds ratio, adjusted for obesity and asthma, 3.49), sinus problems (obesity- and sinus-

TABLE 3
DISTRIBUTION OF UPPER AND LOWER RESPIRATORY PROBLEMS
AND PULMONARY FUNCTION ACCORDING TO
SLEEP-DISORDERED BREATHING

	AHI < 5 (n = 339)	AHI > 10 (n = 25)	p Value
Chronic bronchitis, %	8.5	17.4	NS [†]
Cough, % [‡]	23.1	77.8	0.001
Occasional wheeze, %	17.4	44.0	0.001
Persistent wheeze, %	4.4	16.0	0.012
Sinus problems, %	19.1	50.0	0.001
Hay fever, %	15.2	27.3	NS
Asthma, %	11.8	28.0	0.019
Bronchitis, %	7.9	17.4	NS
FEV ₁ , % pred [§]	95.2 ± 17.0	89.3 ± 15.3	NS
FVC, % pred [§]	98.2 ± 15.4	93.8 ± 12.6	NS

For definition of abbreviations, see Table 1.

* Continuous variables expressed as means ± SD.

[†] Not significant (p > 0.10).

[‡] Data only available for children ≤ 13 yr.

[§] Based on 163 subjects with acceptable spirometry.

TABLE 4
ODDS RATIOS, RELATING DEMOGRAPHIC FACTORS AND UPPER
AND LOWER RESPIRATORY PROBLEMS TO
SLEEP-DISORDERED BREATHING*

	Unadjusted		Adjusted for Race and Obesity	
	Odds Ratio	95% CI	Odds Ratio	95% CI
Obesity (BMI > 28)	4.59	1.58–13.33	4.69 [†]	1.59–14.15
African American	3.49	1.56–8.32	3.59 [†]	1.50–8.58
Sinus problems	3.41	1.32–9.68	5.10	1.78–15.18
Occasional wheeze	3.64	1.51–8.79	3.29	1.24–8.94
Persistent wheeze	4.71	1.30–16.76	7.45	2.03–27.39
Cough	10.52	3.28–33.68	8.83	2.29–34.05
History of asthma	3.22	1.39–7.54	3.83	1.39–10.55

Definition of abbreviations: CI = confidence interval. For other definitions, see Table 1.

* All values adjusted for recruitment source (control or index family). Analyses also account for clustered family data (see STATISTICAL METHODS).

[†] Values adjusted for obesity or African American race.

adjusted odds ratio, 6.23), and persistent wheeze (obesity- and wheeze-adjusted odds ratio, 3.81).

We also assessed whether a relationship between SDB and respiratory symptoms could be explained by current maternal (passive) smoke exposure. Forty-three percent of the mothers of children with SDB reported smoking at least one cigarette a day during the previous month as compared with 26% of the mothers of children without SDB. After adjusting for recruitment source, race, and obesity, and accounting for the clustered family data, no relationship between SDB and passive smoking was suggested (odds ratio, 1.02; 95% CI, 0.89 to 2.80).

Because there is uncertainty regarding the optimal cutoff values of BMI that identify obesity in children, analyses also were repeated for BMI as a continuous variable. This demonstrated that for each increase of 1 kg/m² of BMI above the mean, risk of SDB increased by 12%. Use of BMI, rather than obesity defined as a binary variable, did not alter the results of the multiple adjusted logistic regression analyses presented in Table 4.

Because the identification of hypopneas from the sleep records was dependent on observing a reduction in oxygen saturation with changes in breathing amplitude (from the thermistor or impedance channels), we assessed whether the higher AHIs in the asthmatic as compared with the nonasthmatic children could have resulted from differences in their levels of oxygen saturation. No differences were noted between the asthmatic and the nonasthmatic subjects in baseline oxygen saturation (97.7 ± 1.3 versus 97.5 ± 1.7 , $p = 0.51$ for asthmatics and nonasthmatics, respectively) or for average saturation with hypopneas/apneas (94.6 ± 1.9 versus 94.8 ± 2.1 , $p = 0.58$).

DISCUSSION

Although recent epidemiologic studies have helped define the prevalence and risk factors for SDB in adults, relatively little is known regarding the distribution of SDB and risk factors for SDB in pediatric populations. The majority of reports of SDB in children are of patients referred for clinical evaluation, often because of profound sleepiness, behavioral problems, or cor pulmonale (18–22). Case series of symptomatic children emphasize the importance of adenotonsillar hypertrophy, nasal obstruction, facial anomalies, micrognathia, neuromuscular disease, or certain inherited syndromes as risk factors for SDB (20). These data, however, are likely biased by the selection processes that influence referral of symptomatic children for evaluation. In the current study, we were able to exploit the relatively high prevalence of SDB in family members of affected probands to study risk factors for SDB in a large sample of children with a wide range of apneic activity, the majority of whom were not referred for clinical evaluation. The findings from this sample derived from an urban community in the United States show that children and adolescents < 18 yr of age at increased risk for SDB include children who are obese, African Americans, and children with both upper and lower respiratory conditions.

Susceptibility to SDB relates to the propensity for repetitive upper airway collapse. In any given person, this is determined by anatomic and neuromuscular factors that influence upper airway size and/or function. In adults, the strongest risk factors for SDB are male sex and obesity (23, 24). This study has demonstrated that, in children, sex does not significantly influence SDB risk. This is consistent with previous epidemiologic data that suggest no differences in snoring between boys and girls (15). Lack of a gender effect suggests that the sex differences seen in adults may be mediated by sex hormones (and their in-

fluence on respiratory control and/or body fat distribution), which likely play a smaller role in prepubertal children.

This study does demonstrate a significant association between obesity, as measured by the BMI, and increased neck circumference, with SDB. However, the magnitude of the effect is approximately one-half that which has been previously reported for adults (odds ratios approximately 5 compared with 10 to 12 in adults) (24, 25). A lesser effect of obesity in children may relate to relatively lower levels of obesity observed generally in children than in adults, or because obese children may be relatively “protected” because of other factors such as ventilatory control mechanisms.

We previously reported increased SDB in African Americans as compared with whites, an effect that was most pronounced in the age group < 25 yr. In the current report, focused on children < 18 yr, we estimate that African Americans are approximately 3.5 times more likely to have SDB than a racial group consisting predominantly of white individuals. This effect is independent of both obesity and the respiratory risk factors described below. It is possible that some of the excess risk might be related to adenotonsillar hypertrophy, which is difficult to objectively quantify. Although we have attempted to systematically evaluate adenotonsillar hypertrophy from physical examination, we have been unable to validate its quantification, and, thus, chose not to report these data here. It is also possible that racial differences result from sociocultural differences in parental behaviors and the home environment that influence the sleep patterns of children, such as cosleeping (26).

A focus of this report was to examine the relationship between upper and lower respiratory problems and SDB. We hypothesized that exposures or pathophysiologic processes that promote nasopharyngeal or airway inflammation influence SDB susceptibility. Data from an epidemiologic study from the United Kingdom indicated that the frequency of snoring was increased in asthmatics as compared with nonasthmatic subjects; this relationship was significant among adults < 40 yr of age (27). Larsson and colleagues (28) in Sweden found an increased prevalence of symptoms of SDB (snoring and apneas) in adults with chronic bronchitis when compared with a reference population. The Swedish study also reported an increased prevalence of snoring and apnea in subjects who reported wheezing and dyspnea. In children, questionnaire surveys have pointed out significant relationships of snoring with asthma and bronchitis (29, 30), and with allergic rhinitis and passive smoking (14, 29). Odds ratios, relating asthma and cough in snorers versus nonsnorers ranged from 2 to 3 (29). With the use of objective monitoring of breathing during sleep, we confirmed a significant association between upper and lower respiratory symptoms and asthma with SDB and demonstrated that these associations were independent of sex, obesity, family history, and race. Furthermore, with multivariate modeling, we were able to quantify the extent to which risk was attributable to upper versus lower respiratory problems. Our data suggest that symptoms of lower respiratory problems (wheeze or doctor-confirmed asthma) and a history of sinus problems both independently increase risk of SDB. Furthermore, risk of SDB in association with lower respiratory problems appears “dose-dependent,” in that children with persistent wheeze appear to be at greater risk than children with “occasional” wheeze. Additionally, these associations do not appear to be due to confounding by active maternal smoking.

There are a number of potential pathophysiologic processes that could account for the relationship of upper and lower respiratory conditions with SDB. The association between sinus problems and SDB may relate to increased upper

airway resistance often found in children with inflamed or abnormal upper airways (31, 32). An increased upper airway resistance may lead to the generation of negative inspiratory pressure and intraluminal collapse, promoting hypopneas or apneas (32).

The association of SDB with lower respiratory problems is probably more complex, and the direction of the predominant causal pathway is unclear. It is possible that SDB may cause or exacerbate airway hyperresponsiveness. With SDB, increased negative intrathoracic pressure is generated, which may promote gastroesophageal reflux, and, in turn, cause vagal stimulation and bronchoconstriction (33). Intrathoracic pressure swings and/or hypoxia also may increase cholinergic tone (potentiating bronchoconstriction) independent of esophageal reflux (34, 35). SDB may produce abnormalities in upper airway muscles and mucosa that may alter oropharyngeal reflexes, which potentiates bronchoconstriction (34, 35). A causative association between SDB and asthma is suggested by two studies that demonstrated improved asthma control in asthmatics with SDB or loud snoring that were treated with nasal continuous positive airway pressure (34, 36). The reverse causal pathway (asthma causing or exacerbating SDB) also is possible. Asthmatics frequently experience sleep disruption because of nocturnal bronchoconstriction (37, 38). Experimental sleep restriction has been reported to increase the frequency of apneas in snorers (39), possibly because of adverse effects of sleep deprivation on respiratory control. Finally, asthma and SDB may be linked because of common risk factors that promote airway inflammation and/or disturb neuromuscular control of breathing. The patency of the upper airway and overall bronchomotor tone may be influenced by common sleep-state-specific or circadian-rhythm-specific influences. Alterations in both bronchomotor tone and respiratory muscle function occur in REM sleep (37, 40), which also is the time when apneas are often the most pronounced.

It is possible that children with SDB with an affected family member may differ from children in whom the disorder occurs in a "nonfamilial" form. The relative impact of the risk factors discussed in this report are likely different for children with congenital anomalies, severe underlying comorbidities, and neuromuscular diseases. Because SDB occurs more frequently as a familial than as a sporadic disorder (2), and because family history is a significant risk factor for SDB in children (odds ratio, 4 to 5) (11), the data from this study should be broadly generalizable to the majority of children in the community with elevated AHI levels.

The measurement of the AHI in this study was based on a simple approach for collecting six channels of data in an unattended setting. This approach has been used extensively in epidemiologic studies to provide reliable estimates of the AHI that, in adults, correlates excellently with the AHI determined by conventional polysomnography (7). The optimal recording approach for sleep studies of children is an area of controversy. It is possible that the sometimes subtle changes in breathing pattern, in particular, periods of hypoventilation, that occur in children with SDB be identified best with inductance plethysmography and measurement of carbon dioxide (41). Recognizing that children may not desaturate as markedly as adults, we used a 2.5% level of desaturation rather than the 4% used in many adult studies (42) to help identify hypopneas. We also identified hypopneas on the basis of changes in the amplitude of either the impedance or the thermistor signal, an approach that may increase the sensitivity compared with use of a single channel to identify breathing changes. We previously demonstrated that this approach for monitoring and scoring successfully distinguishes children with and with-

out SDB symptoms (11). Additionally, in our analyses, we did not use clinical criteria for identifying abnormality (which are variable across centers and specific to the recording apparatus). Rather, we maximized the contrast between children with and without "exposure" to SDB by creating two groups of children with clearly different AHI levels. A level of 10 likely identifies children with at least moderate levels of SDB. A level of < 5, however, may include some children, who, if symptomatic, might be considered to have some degree of SDB. Redefining affection status differently, using a cutoff level of < 1 to define a normal population, and a level of > 5 as an abnormal level, resulted in generally similar but somewhat less significant associations between SDB and the risk factors under consideration (e.g., odds ratios of 2.2 for wheeze, 2.3 for race, and 6.1 for obesity). We also used a 10-s duration criterion to identify obstructed breathing events. This may differ from what is done in some pediatric sleep laboratories. It is possible that different recording approaches and SDB definitions may have identified a slightly different population of "affected" children. However, it seems unlikely that alternative definitions would result in qualitative differences in the findings of this study.

Both the prevalence and the clinical significance of SDB to general health and development in children are poorly understood. We observed a frequency of SDB in our control sample of children of 1.6%. The results of the studies of Gislason and Benediktsdottir (15) and Ali and colleagues (43), which focused on small samples of children with multiple symptoms drawn from their larger population-based samples, also indicate that the minimum prevalence of SDB is in the range of 1 to 3%. Clinical case series and limited epidemiologic data suggest that children with untreated SDB experience substantial morbidity. Even mild forms of SDB may be important in children, who may be particularly susceptible to adverse effects of sleep disruption and gas exchange abnormalities that may negatively impact growth and development. The findings from this study identify several risk factors for SDB in children, highlighting risk associated with obesity, African American race, and respiratory problems. Further understanding how these risk factors negatively impact upper airway patency may aid in elucidating the pathogenesis of SDB and optimizing treatment strategies.

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