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Multilevel Risk Factors for Sleep-Disordered Breathing-Related Symptom Burden in an Urban Pediatric Community-Based Sample

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Abstract

BACKGROUND: Pediatric sleep-disordered breathing (SDB) disproportionately affects children with low socioeconomic status (SES). The multilevel risk factors that drive these associations are not well understood.

RESEARCH QUESTION: What are the associations between SDB risk factors, including individual health conditions (obesity, asthma, and allergies), household SES (maternal education), indoor exposures (environmental tobacco smoke [ETS] and pests), and neighborhood characteristics (neighborhood disadvantage), and pediatric SDB symptoms?

STUDY DESIGN AND METHODS: Cross-sectional analyses were performed on 303 children (aged 6-12 years) enrolled in the Environmental Assessment of Sleep Youth study from 2018 to 2022. Exposures were determined by caregiver reports, assays of measured settled dust from the child's bedroom, and neighborhood-level Census data (deriving the Childhood Opportunity Index to characterize neighborhood disadvantage). The primary outcome was the SDB-related symptom burden assessed by the OSA-18 questionnaire total score. Using linear regression models, we calculated associations between exposures and SDB-related symptom burden, adjusting for sociodemographic factors, then health conditions, indoor environment, and neighborhood factors.

RESULTS: The sample included 303 children (39% Hispanic, Latino, Latina, or Spanish origin; 30% Black or African American; 22% White; and 11% other). Increasing OSA-18 total scores were associated with low household SES after adjustment for demographic factors, and with asthma, allergies, ETS, pests (mouse, cockroach, and rodents), and an indoor environmental index (sum of the presence of pests and ETS; 0-2) after adjusting for sociodemographic factors. Even after further adjusting for asthma, allergies, and neighborhood disadvantage, ETS and pest exposure were associated with OSA-18 (ETS: $\beta = 12.80$; 95% CI, 7.07-18.53, also adjusted for pest; pest exposure: $\beta = 3.69$; 95% CI, 0.44-6.94, also adjusted for ETS).

INTERPRETATION: In addition to associations with ETS, a novel association was observed for indoor pest exposure and SDB symptom burden. Strategies to reduce household exposure to ETS and indoor allergens should be tested as approaches for reducing sleep health disparities.

Keywords

children; disparities; OSA; OSA-18

Pediatric sleep-disordered breathing (SDB), including habitual snoring and OSA, is prevalent in 7% to 15% and 1% to 5% of children, respectively, and disproportionally affects children from minoritized backgrounds, with low socioeconomic status (SES) households, or living in disadvantaged neighborhoods.[1-7] SDB is a risk factor for numerous health, neurobehavioral, and functional outcomes,[1] and therefore may contribute to health disparities. In generally healthy school-aged children, the most commonly recognized risk factors for SDB are adenotonsillar hypertrophy and obesity.[5] Black children have a fourfold increased risk for OSA compared with White children unexplained by obesity,[4,8] and have

decreased response to treatment of OSA with adenotonsillectomy compared with White children, suggesting that risk factors other than adenotonsillar hypertrophy and obesity contribute to OSA disparities.[9] Several studies suggest that neighborhood disadvantage associates with SDB prevalence or severity[6,10,11]; however, the underlying mechanisms are not understood.

A plausible explanation for disparities in SDB is that environmental exposures to allergens and irritants directly contribute to the pathogenesis or severity of SDB via effects on upper airway inflammation or indirectly influence SDB through effects on lower airway inflammation that increase risk of asthma. Asthma and allergic rhinitis frequently coaggregate with SDB, suggesting potential causal associations and shared risk factors.[12-15] Although allergen and irritant exposures, including rodents, cockroaches, and environmental smoke exposure (ETS), have been associated with increased asthma symptoms or increased asthma severity in children who are atopic,[16-25] little is known about how these exposures affect SDB.

Understanding multilevel risk factors as risk factors for SDB is critical to mitigating childhood sleep health disparities. We hypothesize that multilevel risk factors—particularly the indoor environment—are associated with increased risk for SDB symptom burden in children. We focused on SDB symptom burden given that measures of symptom burden are increasingly valued as important health outcomes, the apnea-hypopnea index (AHI) does not fully reflect the clinical manifestations and chronic symptom burden experienced by children with SDB,[26] and SDB symptoms better predict behavioral outcomes and response to intervention than the AHI.[27]

Study Design and Methods

The Environmental Assessment of Sleep Youth (EASY) study is a community-based study of children living in predominantly low-income neighborhoods of Boston, Massachusetts. A detailed description of the design for the EASY study and recruitment flow are included in e-Appendix 1 and Figure 1, respectively. Briefly, children were recruited from January 2018 to June 2022 primarily from two sources: (1) 41 urban public elementary schools that participated in school-wide population surveys administered to the targeted school population and completed by their caregivers as part of the School Inner-City Asthma Study (SICAS),[28] and (2) discharge lists from the Boston Children's Hospital ED. Children aged 6 to 12 years who lived in their current residence ≥ 50% of the time for at least 1 month before study enrollment with caregivers who spoke English or Spanish were eligible. Children with severe medical, neurobehavioral, neurodevelopmental, or psychiatric disorders that would limit their ability to participate and require special assistance were excluded. The study was approved by the Boston Children's Hospital institutional review board (No. IRB-P00025433). Written informed consent was obtained from participants' guardians, and assent was obtained from participants before enrollment.

Exposures

Individual-Level Factors: Medical history of asthma and allergic rhinitis were identified using the International Study of Asthma and Allergies in Childhood asthma and rhinitis

core questions. Asthma was defined as a score ≥ 5 on the International Study of Asthma and Allergies in Childhood eight-item global wheezing questionnaire. This definition has a sensitivity of 95% and a specificity of 100% for identifying asthma by physician diagnosis.[29] Allergic rhinitis was defined as having both sneezing and itchy and watery eyes symptoms in the past 12 months; this definition has a positive predictive value of 63%.[30] BMI was calculated with participants' measured height and weight. Obesity was defined as a BMI ≥ 95th percentile.

Household-Level Factors:

Household SES: This was characterized using caregiver-reported maternal education (including eight response options ranging from eighth grade or less to professional degree) and dichotomized as more than a high school degree and high school degree or less as the primary exposure for low household SES. This variable is additionally used as a covariate in some analyses.

Indoor Home Environment: From standardized household questionnaires modified from Adamkiewicz et al,[31] we created binary variables for ETS and pest exposure. Participants were considered exposed to ETS if the caregiver reported anyone living in the residence who smoked in the residence and exposed to pests if reported seeing mice, rats, or cockroaches in the past 12 months more than a few times a year (scale range: never, few times per year, few times per month, few times per week, and every day) in individual questions. A summed index, the Indoor Environmental Index, reflected the arithmetical sum of exposure to ETS and the presence of pests (range, 0-2), summarized as 0, 1, and 2.

Bedroom Dust Allergen Exposure: Settled dust samples were collected in each participant's bedroom by vacuuming a 2-m^2 area in the child's bedroom floor plus bedding with a handheld vacuum using a standardized protocol.[32] Concentrations of the mouse (Mus m 1) and German cockroach allergen (Bla g 2) were assessed using the Multiplex Array for Indoor Allergens (MARIA; Indoor Biotechnologies).[33] The lowest limit of detection was 0.002 ng/mg for Mus m 1 and 0.044 ng/mg for Bla g 2.

Neighborhood-Level Factors: Each participant's residential address was assigned to a US Census Tract database using the Geographic Information System ArcGIS Pro 2.8.7 software (Environmental Systems Research Institute) with 80% spelling sensitivity based on georeferenced 2014 US Census Bureau Master Address File/Topologically Integrated Geographic Encoding. A manual review was then performed to geocode unmatched addresses. The Child Opportunity Index (COI) 2.0 total scores were used to model neighborhood disadvantage. COI 2.0, a tool for studying children's neighborhoods, is composed of 29 variables in three domains (education, health/environment, and social/economic opportunity) and is calculated using data from several sources (diversitydatakids.org).[34,35] Nationally adjusted COI scores vary from 0 to 100 and were categorized as very low (0-20), low (20-40), moderate (40-60), high (60-80), and very high (80-100). Neighborhood disadvantage was defined as a COI score < 40 (very low or low).

Covariates: Children's characteristics included caregiver-reported age (years), sex (male/female), race (American Indian or Alaskan Native, Asian, Black or African American, White, more than one race, and other race), and ethnicity (Hispanic, Latino, Latina, or Spanish origin). Because of the paucity of individuals who identified as American Indian, Asian, and those with more than one race, these participants were recategorized to other. A dichotomized minority variable was then created to include Hispanic, Latino, Latina, or Spanish origin; Black or African American; and other compared with White children (reference group). Objective measures of SDB were collected with a single-night home assessment. Children were studied at home using the WatchPAT 200U (Itamar Medical) on their nondominant hand after the fit was checked by research staff (n = 169). Events were manually edited so that only events with ≥ 3% desaturation were used to calculate an AHI. Children in whom the WatchPAT 200U did not fit well were studied with a finger pulse oximeter (Nonin WristOx), which generated an oxygen desaturation index (n = 94). An AHI/oxygen desaturation index ≥ 5 was considered evidence for SDB.

Outcome

The study's outcomes were derived from the OSA-18 questionnaire. The OSA-18 questionnaire uses a point scale ranging from 1 (none of the time) to 7 (all the time) for the caregiver to rate the severity of the child's problems in five domains: sleep disturbances (eg, snoring, breathing pauses, fragmented sleep), physical symptoms (eg, mouth breathing), emotional distress (eg, hyperactive behavior), daytime problems (eg, sleepiness, attention span), and caregiver concerns (eg, not getting enough air) . Scores on each item are summed to produce a total score ranging from 18 to 126; higher scores correspond to a greater symptom burden.[36-38] Although the OSA-18 score was designed to evaluate changes in symptoms in children referred for evaluation and treatment of SDB, it provides a comprehensive assessment of SDB across relevant symptom dimensions and was found to have high internal validity (Cronbach $\alpha = 0.85$) in a general pediatric sample, supporting its more general use as a measure of SDB symptom burden.[39] The primary study outcome was defined as a continuous total OSA-18 score. Secondary analyses were conducted using continuous measures from each OSA-18 subdomain.

Statistical Analysis

Cross-sectional analyses were performed. We first examined the univariate association between each risk factor (individual, household, and neighborhood characteristics) and OSA-18 total score in separate linear regression models. We then modeled associations for individual health, household, and neighborhood exposures after adjusting for sociodemographics, including age, sex, racial or ethnic minority group, and maternal education (except for models that evaluated maternal education as the main predictor) (Table 2). Next, we focused on the roles of the indoor environment and OSA-18 by testing (1) the association between ETS and OSA-18 after adjusting for pest exposure (model 2), plus asthma and allergic rhinitis (model 3) and plus neighborhood disadvantage (model 4), in addition to sociodemographics (model 1); and (2) the association between presence of pests and OSA-18 after adjusting for ETS exposure (model 2), plus asthma and allergic rhinitis (model 3) and plus neighborhood disadvantage (model 4), in addition to covariates in model 1 (Table 3). We also investigated the associations between measured mouse (Mus m 1) and

cockroach (Bla g 2) allergen concentration (log-transformed) with OSA-18 in participants with bedroom dust allergen samples (n = 252) in similar models previously described (Table 4).

Secondary analysis included investigating the linear association between the risk factors for SDB and the five OSA-18 subdomains as continuously measured dependent variables (Table 5). Effect modification for the association between asthma and allergic rhinitis with OSA-18 score by caregiver-reported pest exposure was tested using the cross-product terms in multivariable models (e-Table 1).

Results

Selected characteristics of study participants are presented in Table 1. The sample included 303 children with a mean age of 9.5 years, and 44% were female. Of the sample, 39%, 31%, 22%, and 9% were of Hispanic, Latino, Latina, or Spanish origin; Black or African American; White; and other race or ethnicity, respectively. Twenty-seven percent of respondents reported maternal education attainment of a high school diploma or less, and 65% of the sample lived in disadvantaged neighborhoods. Of the children, 28% met the criteria for objective SDB (AHI/oxygen desaturation index ≥ 5/h). Compared with the larger school cohort (SICAS enrollment pool; N = 4,983) from which we recruited, the reported sample was similar regarding the prevalence of asthma (23% vs 19%) and allergic rhinitis (14% vs 19%) for the SICAS and EASY samples, respectively. Missingness for variables included in the primary and secondary analyses ranged from four (1%) to 20 (6%) (e-Table 2). Participants from racial or ethnic minority groups, lower maternal education, neighborhood disadvantage, and asthma were more likely to have missing data (e-Table 1).

OSA-18 and Multilevel Risk Factors

The mean OSA-18 total score was 34.5 ± 15.8. Table 2 shows the estimated β coefficients and 95% CIs from models of the association between risk factors for SDB and total OSA-18 score. In models adjusted for age, sex, racial minority groups, and ethnicity, low maternal education was associated with 7.55 (95% CI, 3.44-11.66; $P < .01$) increased OSA-18 score. In models adjusted for sociodemographics including maternal education, history of asthma and allergic rhinitis were associated with a 13.63 (95% CI, 9.44-17.82; $P < .01$) and 6.95 (95% CI, 2.62-11.29; $P < .02$) increased OSA-18 score, respectively. In adjusted analyses, ETS was associated with an 11.59 (95% CI, 5.84-17.35; $P < .01$) increased score. Any pest exposure was associated with a 3.80 (95% CI, 0.26-7.34; $P = .04$) higher adjusted score, whereas caregiver-reported cockroach exposure was associated with a 6.07 (95% CI, 1.15-10.99; $P = .02$) higher score. Notably, an Indoor Environmental Index score of 2 was associated with an 18.75 (95% CI, 10.96-26.53; $P < .01$) point increase. COI was associated with elevated OSA-18 only in the unadjusted model and not in the adjusted model (Table 2).

Associations With the Indoor Environment

Table 3 shows associations between measures of the indoor environment and OSA-18 score. The association between ETS and OSA-18 did not appreciably attenuate after additionally adjusting for pest exposures, asthma, allergic rhinitis, and neighborhood disadvantage. For

the association of pest exposure, the magnitude of associations with OSA-18 was about one-third of that for ETS but was significant after additionally adjusting for ETS, asthma, and allergic rhinitis. However, this association was no longer significant after adjusting for neighborhood disadvantage.

Associations With Bedroom Dust Allergens

There were 82% (n = 206) and 9% (n = 23) of homes with detectable levels of mouse (Mus m 1) and cockroach (Bla g 2) in the participants' bedrooms, respectively. The overall concentration (geometric mean) of mouse and cockroach allergens was 0.06 and 0.12 ng/mg, respectively. A 10-unit increase of log (Mus m 1) was associated with a 1.7 increase in the OSA-18 score (β = 1.69; 95% CI, 0.15-3.23; $P = .03$) after adjusting for sociodemographics. For log (Bla g 2), a 10-unit increase was associated with a 6.91 (95% CI, 0.28-13.54; $P = .04$) increase in OSA-18 score in models adjusted for sociodemographics. Further adjustment for ETS, asthma, and allergic rhinitis showed similar trends with mouse allergen, whereas the association with roach allergen attenuated. Note that caregiver-reported mouse exposure was moderately correlated with measured bedroom mouse allergen ($r = 0.57$; $P \geq .01$), whereas it was only weakly correlated with cockroach allergen ($r = 0.09$; $P \geq .01$).

Secondary Outcomes

Table 5 shows adjusted linear associations between the indoor environmental exposures and the five OSA-18 subdomains. ETS was positively associated with all subdomains except for physical suffering when adjusted for sociodemographic factors, pest exposure, asthma, and allergic rhinitis. In analyses adjusted for sociodemographics, any pests or mice were most consistently associated with the physical suffering subscale; this relationship did not appreciably change after adjusting for ETS, asthma, and allergic rhinitis. Cockroach exposure was associated with sleep disturbance, physical suffering, and caregiver concerns in most models; however, associations were attenuated for sleep disturbance and physical suffering after adjusting for asthma and allergic rhinitis.

There was no interaction found between asthma or allergic rhinitis with caregiver-reported pest exposures (e-Table 2).

Sensitivity Analysis

In the subgroup of children without asthma and allergic rhinitis (n = 213), the univariate and multivariate associations also were similar to the whole sample of children (N = 303) (e-Table 3).

Discussion

To our knowledge, this is the first study to evaluate both the indoor and neighborhood environment in relation to SDB symptom burden in children. Our findings reveal that multilevel risk factors are associated with SDB-related symptom burden (ie, individual health conditions [asthma and allergic rhinitis], family/household SES [maternal education], neighborhood disadvantage [COI score]). Within households, exposures to irritants and allergens (eg, ETS, pests) were associated with an approximately 4-point to 12-point

increase in total OSA-18 scores, and those with both exposures had an approximately 20-point increase in OSA-18 scores (approximately 1.3 SD increase) compared with children with no adverse indoor exposures.

These results are consistent with and extend those from prior studies that showed children living in low SES households have a high prevalence of SDB. In a Canadian cohort, children with mothers reporting less than high school education had nearly three times the odds of having OSA compared with university-educated participants.[7] The underlying drivers of this association, however, are poorly understood. Poor indoor air quality because of tobacco smoke and allergen exposures from rodents, mold, and cockroaches have been shown to contribute to asthma symptoms.[16,17,21-23] Although the pathogenesis for OSA and asthma differ, our results suggest that these conditions have overlapping risk factors. Irritants and allergens may exacerbate SDB by stimulating immune responses manifested as adenotonsillar hypertrophy and by amplifying nasopharyngeal inflammation, adversely affecting upper airway patency. ETS, although not common in this sample, was strongly associated with SDB symptoms, a finding that is consistent with previous reports.[40-44] We additionally showed an association between pest exposure with SDB symptoms. Moreover, we studied the associations of bedroom dust allergen exposures in a large proportion of the cohort and demonstrated similar associations for mouse and cockroach bedroom dust levels with caregiver-report information, supporting the importance of household and bedroom environmental conditions and sleep health.

In addition to adverse home environmental conditions, prior studies identified a relationship between OSA prevalence or severity and neighborhood-level risk factors.[6,11,35,44-46] In Cleveland, Ohio, residence in neighborhoods with low SES was associated with OSA prevalence after adjusting for prematurity, obesity, and race/ethnicity.[11] In the six-city analysis of the Childhood Adenotonsillectomy Study, neighborhood factors (high poverty rate, high population densities, and close distance to major roads) were associated with increased OSA severity.[6] Communities of largely Black and Hispanic families are disproportionately burdened by negative effects of zoning, highway placement, and investment because of previous discriminatory policies (eg, redlining beginning in the 1930s), potentially leading to lower rates of home ownership and adverse exposures to a variety of social and environmental stressors, including poor air quality.[47] In this sample, there were significant differences in measures of neighborhood disadvantaged (COI indices) in unadjusted analyses; however, this relationship was modestly attenuated and no longer significant after adjustment for demographics and maternal education. This may reflect the challenges in dissecting associations between race and ethnicity from those directly related to neighborhood because of within-neighborhood segregation. Future studies will need to examine wider geographic areas and address adverse outdoor environments.

Children in households with lower SES have been shown to experience increased chronic inflammatory diseases (eg, asthma, allergic rhinitis),[48] which commonly coaggregate with OSA.[4,12,13,49] We found approximately a 7-point to 14-point increase in elevated OSA-18 scores with allergic rhinitis and asthma, respectively. Our analyses underscore the potential to leverage approaches from asthma prevention interventions to additionally address SDB

disparities. We explored whether asthma or allergic rhinitis might amplify the effects of pest exposure and did not find evidence for statistical interaction.

Study strengths include the study of a pediatric population living in predominantly low-income and minoritized neighborhoods and the consideration of multilevel risk factors. A validated caregiver-reported measurement was our primary outcome, consistent with the recognition of the importance of perceived symptoms as important health-related targets.[50] Additionally, we showed an association between pest exposure and OSA-18 score using both caregiver report and bedroom dust allergen levels.

A study limitation includes a focus on a single geographic region. We recruited children from two sources—a school-based sample and a sample presenting for care at an ED. The relatively low participation rate of children in the broad target sample may have resulted in selection biases; however, this is unlikely to have weakened the internal validity of our findings. Although limiting inferences on prevalence rates, the prevalence of common health conditions (asthma and allergic rhinitis) was similar in the sample to the larger school-based sample, suggesting that children with health problems from low-income communities were not overrepresented. A large proportion of children had objective evidence for SDB, consistent with data indicating high rates of SDB in low socioeconomic communities and children from underrepresented minority groups.[1,3,7] Although the OSA-18 is most commonly used as a measure of SDB burden in children with known OSA, it has also been used with good psychometrics in general community samples.[39] Other factors other than SDB may contribute to symptoms assessed by OSA-18, such as subclinical behavioral health symptoms (eg, anxiety, depression, attention-deficit/hyperactivity disorder), which could be secondary to SDB and were not included in our analysis. It is plausible that some of the items may reflect symptoms of asthma or allergic rhinitis rather than SDB. However, our key results persisted in analyses that adjusted for both asthma and allergic rhinitis, or analyses restricted to children without these conditions. Moreover, domain-specific analyses showed that ETS was consistently associated with elevated scores across each symptom domain, including a specific sleep disturbance domain. Cockroach exposure was associated with sleep disturbance and physical suffering and caregiver concerns. As for total OSA-18 scores, most associations were not appreciably influenced by asthma and allergic rhinitis adjustment. Misclassification bias because of caregiver-reported data is possible; however, this would likely have biased the results toward the null. We used validated instruments when possible and corroborated OSA-18 associations based on caregiver reports for pests with objectively measured dust allergen levels. The weak correlation between caregiver and dust cockroach levels likely reflects the within-home differences in cockroaches (ie, higher in the kitchen than the bedroom, where sampling was done).[16] The cross-sectional design limits causal inference; however, it is unlikely that the child's SDB symptoms cause ETS or pest exposures. Moreover, the study results will provide the framework for future work on the understanding of the mechanistic pathways of environmental contributors to SDB pathophysiology in children.

Interpretation

A broad assessment of multilevel risk factors in children living in predominantly low-income neighborhoods found that SDB symptom burden was associated with exposures to ETS and pests and asthma and allergic rhinitis. These data highlight potential targets for improving sleep disparities, particularly indoor air quality, an underappreciated SDB-related risk factor. This study also identifies the need to research the mechanisms underlying these associations and consider the effectiveness of standard interventions for pediatric OSA (eg, adenotonsillectomy) in settings of chronic irritant and allergen exposures.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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ABBREVIATIONS:

AHI	apnea-hypopnea index
Bla g 2	Blattella germanica 2
COI	Child Opportunity Index
EASY	Environmental Assessment of Sleep Youth
ETS	environmental tobacco smoke
Mus m 1	Mus musculus 1
SDB	sleep-disordered breathing
SES	socioeconomic status

SICAS	School Inner-City Asthma Study

References

1. Marcus CL, Brooks LJ, Draper KA, et al. Diagnosis and management of childhood obstructive sleep apnea syndrome. Pediatrics. 2012;130(3):e714–e755. [PubMed: 22926176]
2. Katz ES, D'Ambrosio CM. Pathophysiology of pediatric obstructive sleep apnea. Proc Am Thorac Soc. 2008;5(2):253–262. [PubMed: 18250219]
3. Bixler EO, Vgontzas AN, Lin H-M, et al. Sleep disordered breathing in children in a general population sample: prevalence and risk factors. Sleep. 2009;32(6):731–736. [PubMed: 19544748]
4. 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. Am J Respir Crit Care Med. 1999;159(5 Pt 1):1527–1532. [PubMed: 10228121]
5. Rosen CL, Larkin EK, Kirchner HL, et al. Prevalence and risk factors for sleep-disordered breathing in 8- to 11-year-old children: association with race and prematurity. J Pediatr. 2003;142(4):383–389. [PubMed: 12712055]
6. Wang R, Dong Y, Weng J, et al. Associations among neighborhood, race, and sleep apnea severity in children. A six-city analysis. Ann Am Thorac Soc. 2017;14(1):76–84. [PubMed: 27768852]
7. Park JW, Hamoda MM, Almeida FR, et al. Socioeconomic inequalities in pediatric obstructive sleep apnea. J Clin Sleep Med. 2022;18(2):637–645. [PubMed: 34170224]
8. Redline S, Tishler PV, Hans MG, Tosteson TD, Strohl KP, Spry K. Racial differences in sleep-disordered breathing in African-Americans and Caucasians. Am J Respir Crit Care Med. 1997;155(1):186–192. [PubMed: 9001310]
9. Weinstock TG, Rosen CL, Marcus CL, et al. Predictors of obstructive sleep apnea severity in adenotonsillectomy candidates. Sleep. 2014;37(2):261–269. [PubMed: 24497655]
10. Mayne SL, Mitchell JA, Virudachalam S, Fiks AG, Williamson AA. Neighborhood environments and sleep among children and adolescents: a systematic review. Sleep Med Rev. 2021;57:101465. [PubMed: 33827031]
11. Spilsbury JC, Storfer-Isser A, Kirchner HL, et al. Neighborhood disadvantage as a risk factor for pediatric obstructive sleep apnea. J Pediatr. 2006;149(3):342–347. [PubMed: 16939744]
12. Gunnlaugsson S, Abul MH, Wright L, et al. Associations of snoring and asthma morbidity in the School Inner-City Asthma Study. J Allergy Clin Immunol Pract. 2021;9(10):3679–3685.e1. [PubMed: 34102347]
13. Ross KR, Storfer-Isser A, Hart MA, et al. Sleep-disordered breathing is associated with asthma severity in children. J Pediatr. 2012;160(5):736–742. [PubMed: 22133422]
14. Garza N, Witmans M, Salud M, Lagera PGD, Co VA, Tablizo MA. The association between asthma and OSA in children. Children (Basel). 2022;9(10).
15. Cao Y, Wu S, Zhang L, Yang Y, Cao S, Li Q. Association of allergic rhinitis with obstructive sleep apnea: a meta-analysis. Medicine (Baltimore). 2018;97(51):e13783. [PubMed: 30572534]
16. Salo PM, Arbes SJ, Crockett PW, Thorne PS, Cohn RD, Zeldin DC. Exposure to multiple indoor allergens in US homes and its relationship to asthma. J Allergy Clin Immunol. 2008;121(3):678–684.e2. [PubMed: 18255132]
17. Sheehan WJ, Permaul P, Petty CR, et al. Association between allergen exposure in inner-city schools and asthma morbidity among students. JAMA Pediatr. 2017;171(1):31–38. [PubMed: 27893060]
18. Baxi SN, Sheehan WJ, Sordillo JE, et al. Association between fungal spore exposure in inner-city schools and asthma morbidity. Ann Allergy Asthma Immunol. 2019;122(6):610–615.e1. [PubMed: 30904580]
19. Howard EJ, Vesper SJ, Guthrie BJ, et al. Asthma prevalence and mold levels in US northeastern schools. J Allergy Clin Immunol Pract. 2021;9(3):1312–1318. [PubMed: 33091637]
20. Esty B, Permaul P, DeLoreto K, Baxi SN, Phipatanakul W. Asthma and allergies in the school environment. Clin Rev Allergy Immunol. 2019;57(3):415–426. [PubMed: 31044354]

21. Maciag MC, Phipatanakul W. Update on indoor allergens and their impact on pediatric asthma. Ann Allergy Asthma Immunol. 2022;128(6):652–658. [PubMed: 35227902]
22. Permaul P, Hoffman E, Fu C, et al. Allergens in urban schools and homes of children with asthma. Pediatr Allergy Immunol. 2012;23(6):543–549. [PubMed: 22672325]
23. Wilson J, Dixon SL, Breysse P, et al. Housing and allergens: a pooled analysis of nine US studies. Environ Res. 2010;110(2):189–198. [PubMed: 19939359]
24. Akinbami LJ, Kit BK, Simon AE. Impact of environmental tobacco smoke on children with asthma, United States, 2003-2010. Acad Pediatr. 2013;13(6):508–516. [PubMed: 24021528]
25. Bakoula CG, Kafritsa YJ, Kavadias GD, et al. Objective passive-smoking indicators and respiratory morbidity in young children. Lancet. 1995;346(8970):280–281. [PubMed: 7630249]
26. Borsini E, Nogueira F, Nigro C. Apnea-hypopnea index in sleep studies and the risk of over-simplification. Sleep Sci. 2018;11(1):45–48. [PubMed: 29796201]
27. Isaiah A, Spanier AJ, Grattan LM, Wang Y, Pereira KD. Predictors of behavioral changes after adenotonsillectomy in pediatric obstructive sleep apnea: a secondary analysis of a randomized clinical trial. JAMA Otolaryngol Head Neck Surg. 2020;146(10):900–908. [PubMed: 32880655]
28. Phipatanakul W, Koutrakis P, Coull BA, et al. Effect of school integrated pest management or classroom air filter purifiers on asthma symptoms in students with active asthma: a randomized clinical trial. JAMA. 2021;326(9):839–850. [PubMed: 34547084]
29. Solé D, Vanna AT, Yamada E, Rizzo MC, Naspitz CK. International Study of Asthma and Allergies in Childhood (ISAAC) written questionnaire: validation of the asthma component among Brazilian children. J Investig Allergol Clin Immunol. 1998;8(6):376–382.
30. Braun-Fahrländer C, Wüthrich B, Gassner M, et al. Validation of a rhinitis symptom questionnaire (ISAAC core questions) in a population of Swiss school children visiting the school health services. SCARPOL-team. Swiss Study on Childhood Allergy and Respiratory Symptom with respect to Air Pollution and Climate. International Study of Asthma and Allergies in Childhood. Pediatr Allergy Immunol. 1997;8(2):75–82. [PubMed: 9617776]
31. Adamkiewicz G, Spengler JD, Harley AE, et al. Environmental conditions in low-income urban housing: clustering and associations with self-reported health. Am J Public Health. 2014;104(9):1650–1656. [PubMed: 24028244]
32. Celedón JC, Milton DK, Ramsey CD, et al. Exposure to dust mite allergen and endotoxin in early life and asthma and atopy in childhood. J Allergy Clin Immunol. 2007;120(1):144–149. [PubMed: 17507083]
33. King EM, Filep S, Smith B, et al. A multi-center ring trial of allergen analysis using fluorescent multiplex array technology. J Immunol Methods. 2013;387(1-2):89–95. [PubMed: 23085532]
34. Acevedo-Garcia D, McArdle N, Hardy EF, et al. The child opportunity index: improving collaboration between community development and public health. Health Aff (Millwood). 2014;33(11):1948–1957. [PubMed: 25367989]
35. Acevedo-Garcia D, Noelke C, McArdle N, et al. Racial and ethnic inequities in children's neighborhoods: evidence from the new child opportunity index 2.0. Health Aff (Millwood). 2020;39(10):1693–1701. [PubMed: 33017244]
36. Baldassari CM, Mitchell RB, Schubert C, Rudnick EF. Pediatric obstructive sleep apnea and quality of life: a meta-analysis. Otolaryngol Head Neck Surg. 2008;138(3):265–273. [PubMed: 18312869]
37. Kobayashi R, Miyazaki S, Karaki M, et al. Evaluation of adenotonsillectomy and tonsillectomy for pediatric obstructive sleep apnea by rhinomanometry and the OSA-18 questionnaire. Acta Otolaryngol. 2014;134(8):818–823. [PubMed: 24847948]
38. Mitchell RB, Kelly J, Call E, Yao N. Quality of life after adenotonsillectomy for obstructive sleep apnea in children. Arch Otolaryngol Head Neck Surg. 2004;130(2):190–194. [PubMed: 14967749]
39. Bannink N, Maliepaard M, Raat H, Joosten KFM, Mathijssen IMJ. Reliability and validity of the obstructive sleep apnea-18 survey in healthy children and children with syndromic craniosynostosis. J Dev Behav Pediatr. 2011;32(1):27–33. [PubMed: 21160438]
40. Sánchez T, Rojas C, Casals M, et al. Prevalence and risk factors for sleep-disordered breathing in Chilean schoolchildren [Article in Spanish]. Rev Chil Pediatr. 2018;89(6):718–725. [PubMed: 30725060]

41. Gokdemir Y, Civelek E, Cakir B, et al. Prevalence of sleep-disordered breathing and associated risk factors in primary school children in urban and rural environments. Sleep Breath. 2021;25(2):915–922. [PubMed: 33030645]

42. Groner JA, Nicholson L, Huang H, Bauer JA. Secondhand smoke exposure and sleep-related breathing problems in toddlers. Acad Pediatr. 2019;19(7):835–841. [PubMed: 30959225]

43. Yolton K, Xu Y, Khoury J, et al. Associations between secondhand smoke exposure and sleep patterns in children. Pediatrics. 2010;125(2):e261–e268. [PubMed: 20083521]

44. Liu J, Ghastine L, Um P, Rovit E, Wu T. Environmental exposures and sleep outcomes: a review of evidence, potential mechanisms, and implications. Environ Res. 2021;196:110406. [PubMed: 33130170]

45. Johnson DA, Drake C, Joseph CLM, Krajenta R, Hudgel DW, Cassidy-Bushrow AE. Influence of neighbourhood-level crowding on sleep-disordered breathing severity: mediation by body size. J Sleep Res. 2015;24(5):559–565. [PubMed: 25950087]

46. Gueye-Ndiaye S, Williamson AA, Redline S. Sleep disordered breathing. In: Harris D, Brigham E, Celedon JC, eds. Aiming to Improve Equity in Pulmonary Health, An Issue of Clinics in Chest Medicine. 1st ed., Volume 44, Issue 3. Elsevier; September 2023: 585–603.

47. Bailey ZD, Krieger N, Agénor M, Graves J, Linos N, Bassett MT. Structural racism and health inequities in the USA: evidence and interventions. Lancet. 2017;389(10077):1453–1463. [PubMed: 28402827]

48. Ganti P, Suman A, Chaudhary S, Sangha B, David L, Sekhsaria S. The effect of the socioeconomic status on the measurement of asthma control. Allergy Asthma Proc. 2022;43(1):e11–e16. [PubMed: 34983718]

49. Liu J, Wu Y, Wu P, Xu Z, Ni X. Analysis of the impact of allergic rhinitis on the children with sleep disordered breathing. Int J Pediatr Otorhinolaryngol. 2020;138:110380. [PubMed: 33152971]

50. Weldring T, Smith SMS. Patient-reported outcomes (PROs) and patient-reported outcome measures (PROMs). Health Serv Insights. 2013;6:61–68. [PubMed: 25114561]

Take-home Points

Study Question:

What are the associations between multilevel risk factors and sleep-disordered breathing symptom burden in children living in predominantly low-income neighborhoods?

Results:

Within households, exposures to irritants and allergens (eg, pests, environmental tobacco smoke) were associated with an approximately 4-point to 12-point increase in OSA-18 total score, and those with both exposures (Indoor Environmental Index score 2) had a nearly 20-point increase in OSA-18 total scores compared with children with no adverse indoor exposure in models adjusting for sociodemographics. Similar trends were observed in models additionally adjusted for asthma and allergies.

Interpretation:

A broad assessment of multilevel risk factors in children living in predominantly low-income neighborhoods found that sleep-disordered breathing symptom burden was associated with exposures to environmental tobacco smoke and pests and asthma and allergies.

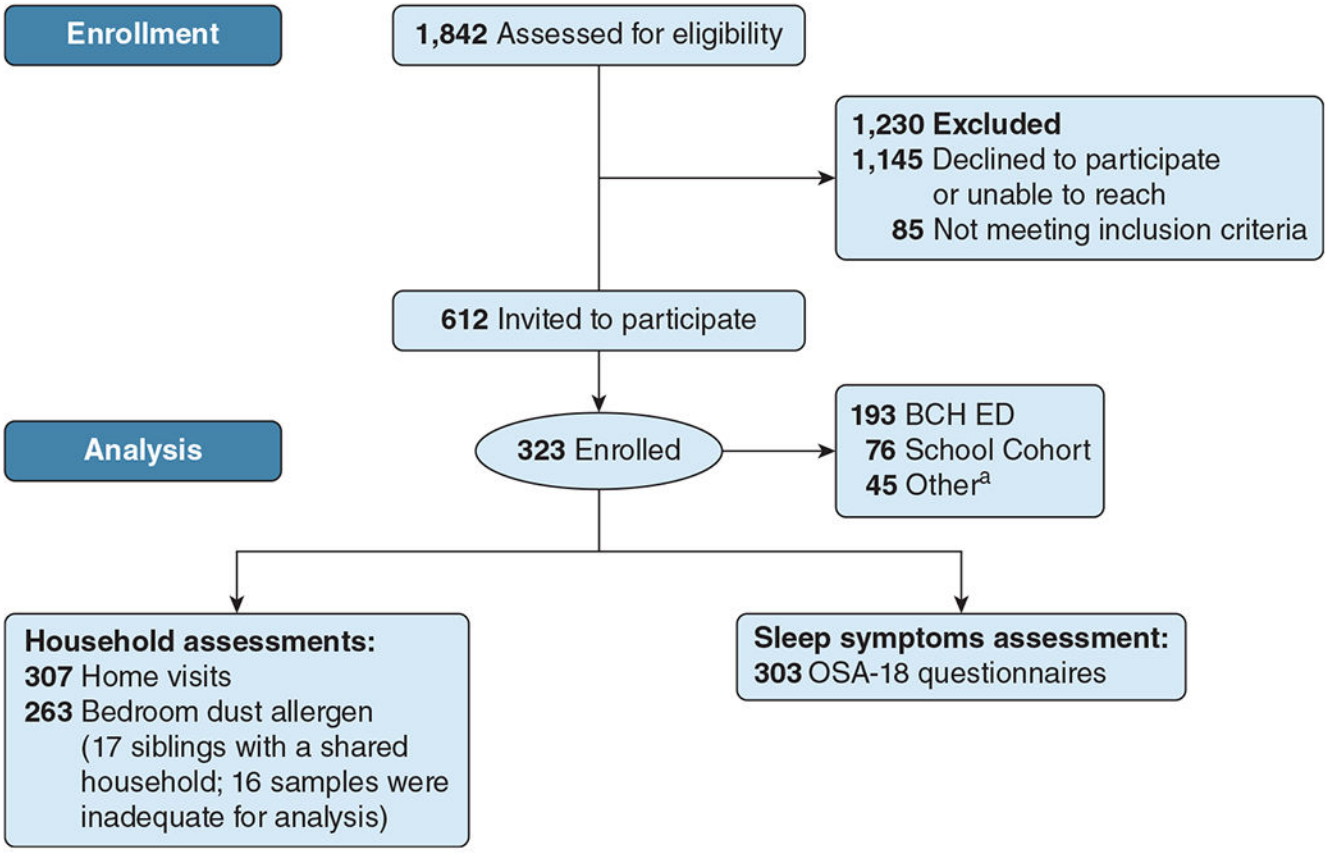

Figure 1 –.

Flow of participants in the Environmental Assessment of Sleep Youth (EASY) study. BCH = Boston Children's Hospital. [a]Other sources included a database with contacts for participants of other ongoing or previous studies at our hospital, siblings, and referrals made by participants and physicians.

TABLE 1]

Sample Characteristics Among Participants of the Environmental Assessment of Sleep in Youth Study (N = 303)

Characteristic	Overall (N = 303)
Sociodemographic	
Age, mean ± SD, y[a]	9.5 ± 1.9
Sex (female)[a]	133 (44)
Race and ethnicity[a]	
Black or African American	91 (31)
Hispanic, Latino, Latina, or Spanish origin	115 (39)
White	66 (22)
Other	26 (9)
Racial or ethnic minority group[a,b]	232 (78)
Household socioeconomic status	
Maternal education (≤ HS degree)	77 (27)
Health conditions	
BMI percentile[a]	
Mean ± SD	70.8 ± 29.6
≥ 95th percentile: obesity	93 (31)
Asthma	56 (19)
Allergic rhinitis	56 (19)
SDB	
AHI/ODI 3%	
Mean ± SD	4.2 ± 3.7
≥ 5 events/h (SBD)	76 (28)
Household environment	
Environmental tobacco smoke	28 (9)
Pests[c]	158 (55)
Mice	138 (49)
Cockroaches	49 (17)
Rats	21 (7)
Indoor Environment Index[d]	
0	114 (40)
1	156 (55)
2	15 (5)
Neighborhood environment	
Child Opportunity Index	
Mean ± SD	33.5 ± 29.6

Characteristic	Overall (N = 303)
< 40, (very low and low)	184 (65)

Values are No. (%) or as otherwise indicated. AHI = apnea-hypopnea index; HS = high school; ODI = oxygen desaturation index; SDB = sleep-disordered breathing.

[a]Missing data: n = 1; missingness for the other variables ranges from n = 4 to n = 20 (refer to e-Table 1).

[b]Racial or ethnic minority group defined as Black or African American; Hispanic, Latino, Latina, or Spanish origin; and other. Reference group = White.

[c]Pests are defined as reported exposure, mice, cockroaches, or rats in the home.

[d]Indoor Environment Index is defined as the arithmetical sum of two household exposures: environmental tobacco smoke and the presence of pests (mice, cockroaches, or rats).

TABLE 2]

Association Between Potential Sleep-Disordered Breathing Risk Factors and OSA-18 Total Score in Unadjusted and Adjusted Models

	Unadjusted Models	Adjusted Models[a]
Household socioeconomic status		
Maternal education (≤ HS degree)	**7.84 (3.97 to 11.72, < .01)**	**7.55 (3.44 to 11.66, < .01)**
Health conditions		
Obesity[b]	0.59 (−3.30 to 4.48, .77)	−1.26 (−5.18 to 2.66, .53)
Asthma	**14.67 (10.52 to 18.82, < .01)**	**13.63 (9.44 to 17.82, < .01)**
Allergic rhinitis	**7.73 (3.33 to 12.14, < .01)**	**6.95 (2.62 to 11.29, < .01)**
Household environment		
Environmental tobacco smoke	**12.82 (6.89 to 18.75, < .01)**	**11.59 (5.84 to 17.35, < .01)**
Pests[c]	**4.12 (0.61 to 7.63, .02)**	**3.80 (0.26 to 7.34, .04)**
Mice	2.55 (−0.77 to 5.88, .13)	2.51 (−0.78 to 5.81, .13)
Cockroaches	**6.76 (2.10 to 11.42, < .01)**	**6.07 (1.15 to 10.99, .02)**
Rats	3.79 (−3.08 to 10.65, .28)	1.76 (−5.14 to 8.66, .62)
Indoor Environment Index[d]		
0	Reference	Reference
1	**4.10 (0.59 to 7.61, .02)**	**3.94 (0.37 to 7.52, .03)**
2	**20.18 (12.35 to 28.00, < .01)**	**18.75 (10.96 to 26.53, < .01)**
Neighborhood environment		
Child Opportunity Index (< 40, low)	**5.36 (1.55 to 9.17, .01)**	3.46 (−0.82 to 7.74, .11)

Values are β (95% CI, *P* value). Bold font = significant associations; HS = high school.

[a]Each model is adjusted for age, sex, racial or ethnic minority group, and maternal education, except for the models predicting maternal education (adjusted for age, sex, and minority race and ethnicity). Note that each row represents a different exposure analyzed in separate models.

[b]Obesity is defined as BMI ≥ 95th percentile.

[c]Pest exposure is defined as caregiver-reported exposure to mice, rats, or cockroaches in the home.

[d]Indoor environment index is defined as the summary of two household exposures: environmental tobacco smoke and the presence of pests (mice, rats, or cockroaches).

TABLE 3]

Association Between Indoor Environment and OSA-18 Total Score in Subsequent Models

Variable	OSA-18 Total Score
Environmental tobacco smoke exposure	
Model 1 (sociodemographics)	**11.59 (5.84 to 17.35, < .01)**
Model 2 (model 1 + pest exposure)	**12.41 (6.79 to 18.03, < .01)**
Model 3 (model 2 + asthma and allergic rhinitis)	**11.64 (6.35 to 16.93, < .01)**
Model 4 (model 3 + neighborhood COI)	**11.32 (5.95 to 16.69, < .01)**
Pest exposure[a]	
Model 1 (sociodemographics)	**3.80 (0.26 to 7.34, .04)**
Model 2 (model 1 + environmental tobacco smoke)	**4.22 (0.78 to 7.66, .02)**
Model 3 (model 2 + asthma and allergic rhinitis)	**3.69 (0.44 to 6.94, .03)**
Model 4 (model 3 + neighborhood COI)	2.95 (−0.35 to 6.24, .08)

Values are β (95% CI, *P* value). Model 1 includes age, sex, racial or ethnic minority group, and maternal education. Bold font = significant associations; COI = Child Opportunity Index.

[a]Pest exposure is defined as caregiver-reported exposure to mice, rats, or cockroaches in the home.

TABLE 4]

Associations Between Measured Bedroom Dust Allergen Exposure and OSA-18 Total Score (n = 252)

	OSA-18 Total Score
Mouse (log Mus m 1)[a]	
Unadjusted model	**2.21 (0.77 to 3.66, < .01)**
Model 1	**1.69 (0.15 to 3.23, .03)**
Model 2	**1.67 (0.14 to 3.20, .03)**
Model 3	**2.00 (0.60 to 3.39, .01)**
Model 4	1.34 (−0.12 to 2.80, .07)
Cockroach (log Bla g 2)[a]	
Unadjusted model	**7.40 (0.73 to 14.08, .03)**
Model 1	**6.91 (0.28 to 13.54, .04)**
Model 2	6.31 (−0.83 to 13.45, .08)
Model 3	2.84 (−3.84 to 9.52, .40)
Model 4	3.28 (−3.37 to 9.93, .33)

Values are β (95% CI, *P* value). Model 1 is adjusted for age, sex, racial or ethnic minority groups, and maternal education. Model 2 is model 1 + environmental tobacco smoke. Model 3 is model 2 + asthma and allergies. Model 4 is model 3 + neighborhood disadvantage (by Childhood Opportunity Index). Bold font = significant associations.

[a]Exposure variables (Mus m 1 and Bla g 2 in ng/mg) were log-transformed (10-unit increase).

TABLE 5]

Associations of ETS and Pest Exposure With OSA-18 Total Score Subdomains

	OSA-18 Subdomains				
	Sleep Disturbance	**Physical Suffering**	**Emotional Distress**	**Daytime Problems**	**Caregiver Concerns**
ETS					
Model 1	**2.44 (0.84 to 4.05, < .01)**	1.13 (−0.29 to 2.54, .12)	**2.40 (0.76 to 4.04, < .01)**	**1.83 (0.47 to 3.19, .01)**	**3.79 (1.87 to 5.72, < .01)**
Model 2[a]	**2.65 (1.04 to 4.26, < .01)**	1.21 (−0.19 to 2.62, .09)	**2.57 (0.94 to 4.21, < .01)**	**1.93 (0.57 to 3.28, .01)**	**4.04 (2.17 to 5.92, < .01)**
Model 3	**2.55 (1.01 to 4.09, < .01)**	1.00 (−0.31 to 2.30, .14)	**2.52 (0.90 to 4.15, < .01)**	**1.79 (0.45 to 3.13, .01)**	**3.78 (2.01 to 5.55, < .01)**
Pest exposure[b]					
Model 1	**0.68 (−0.32 to 1.67, .18)**	**1.03 (0.17 to 1.89, .02)**	0.63 (−0.39 to 1.64, .23)	0.36 (−0.48 to 1.20, .40)	1.10 (−0.08 to 2.28, .07)
Model 2	0.77 (−0.21 to 1.76, .12)	**1.09 (0.23 to 1.95, .01)**	0.69 (−0.31 to 1.69, .18)	0.44 (−0.39 to 1.27, .30)	**1.23 (0.08 to 2.38, .04)**
Model 3	0.63 (−0.31 to 1.58, .19)	**0.93 (0.12 to 1.73, .02)**	0.76 (−0.24 to 1.76, .14)	0.36 (−0.46 to 1.18, .39)	1.01 (−0.08 to 2.10, .07)
Mice					
Model 1	0.63 (−0.31 to 1.58, .19)	**0.89 (0.08 to 1.70, .03)**	0.40 (−0.58 to 1.39, .42)	0.22 (−0.59 to 1.03, .59)	0.36 (−0.75 to 1.47, .52
Model 2	0.70 (−0.24 to 1.64, .14)	**0.92 (0.10 to 1.73, .03)**	0.44 (−0.54 to 1.42, .37)	0.28 (−0.53 to 1.08, .50)	0.42 (−0.69 to 1.53, .46)
Model 3	0.84 (−0.06 to 1.74, .07)	**0.97 (0.21 to 1.72, .01)**	0.48 (−0.50 to 1.45, .34)	0.23 (−0.57 to 1.03, .57)	0.37 (−0.68 to 1.42, .49)
Cockroaches					
Model 1	**1.55 (0.18 to 2.91,.03)**	**1.19 (0.02 to 2.36, .05)**	0.80 (−0.61 to 2.21, .26)	0.05 (−1.12 to 1.21, .94)	**2.48 (0.86 to 4.10, < .01)**
Model 2	**1.45 (0.08 to 2.82, .04)**	**1.20 (0.01 to 2.38, .05)**	0.57 (−0.85 to 1.99, .43)	−0.06 (−1.22 to 1.11, .93)	**2.22 (0.61 to 3.84, .01)**
Model 3	1.16 (−0.16 to 2.47, .09)	0.99 (−0.14 to 2.11, .09)	0.67 (−0.75 to 2.09, .35)	−0.05 (−1.22 to 1.11, .93)	**2.12 (0.60 to 3.64, .01)**

Values are β (95% CI, *P* value). Model 1 is adjusted for age, sex, racial or ethnic minority groups, and maternal education. Model 2 is model 1 + ETS. Model 3 is model 2 + asthma and allergies. OSA-18 subdomain content items include the following: sleep disturbance (loud snoring, breath holding or pauses in breathing, choking/gasping, and fragmented sleep), physical suffering (mouth breathing, frequent colds or upper respiratory infections, rhinorrhea, and dysphagia), emotional distress (mood swings or temper tantrums, aggressive or hyperactive behavior, and discipline problems), daytime problems (excessive drowsiness or sleepiness, poor attention span, and difficulty awakening), and caregiver concerns (caregiver worried over child health, concerned not getting enough air, missed activities, and frustration). Bold font = significant associations; ETS = environmental tobacco smoke.

[a]Model 2 is model 1 + pest exposure.

[b]Pest exposure is defined as caregiver-reported exposure to mice, rats, or cockroaches in the home.