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Allergic sensitization and objective measures of sleep in urban school-age children with asthma

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Introduction

Allergic Sensitization, Asthma, and Sleep

Associations between allergic sensitization, decreased pulmonary function, and increased asthma morbidity in children have been demonstrated consistently across studies.^{1–3} Specific allergic sensitivity to dog, cat, alternaria, but especially to cockroach and rodents have been associated with increased bronchial responsiveness,⁴ increased risk of ED visits for asthma,⁵ and higher asthma morbidity.^{3,6–10}

Sleep is an important outcome to evaluate when considering allergen sensitization in asthma, because it can be a vulnerable time for children with asthma for various physiologic reasons.^{11,12} To date, most studies of allergic asthma and sleep in children are based on self-report assessments using diary data,^{13,14} which are subject to poor recall and bias. More objective assessments of sleep quality in asthma are needed.¹¹

Urban minority children appear to be at greater risk. Children from African American and Latino, predominantly Puerto Rican, backgrounds have poor asthma outcomes.^{15,16} In

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particular, African Americans are two times more likely to report nocturnal asthma compared to non-Latino Whites (NLWs) in genetic studies.¹⁷ African Americans, as early as age 2 years, are also disproportionately affected by higher allergic sensitization compared to NLWs of the same age.¹⁸ Our previous work has shown that both urban African Americans and Latinos with asthma have higher total sleep problem scores compared to NLWs,¹⁹ and that well-controlled asthma and better lung function were more likely in children with “good” sleep health.²⁰ We have yet to study the contribution of allergic sensitization to objective measures of sleep quality in urban children with asthma. Addressing this gap in the literature has important implications for environmental and behavioral interventions to promote good sleep health in urban minority groups.

Current Study

This study first investigated the association between increased allergic sensitization (number of positive allergy tests measured by skin prick test or specific IgE) and children’s sleep outcomes (measured objectively using actigraphy; sleep quality or efficiency, variability in sleep efficiency, mean number of awakenings and sleep duration) in a carefully evaluated urban sample of children with persistent asthma. We expected that higher allergic sensitization would be associated with poorer sleep quality and duration. Given higher levels of morbidity in minority groups,²¹ we expected this association would be more pronounced in children from African American and Latino backgrounds compared to NLW children. Second, we determined whether sleep outcomes differed across sensitization to specific indoor perennial allergens (dust mite, cat, dog, cockroach, mouse), potentially relevant to the bedroom environment. Based on prior literature in urban environments, we expected that children with cockroach and mouse sensitization would have poorer sleep outcomes. Finally, we sought to determine whether asthma status indicators (FEV₁, FEV₁ variability, and asthma control) moderated associations between allergic sensitization and sleep outcomes, and expected these associations to be more robust in the context of lower FEV₁, greater FEV₁ variability, and poor asthma control.

Methods

Data for this study were collected from a larger study, Project NAPS (Nocturnal Asthma and Performance in School, *citation blinded for review*), that examines the co-occurrence of asthma and allergic rhinitis symptoms, sleep quality, and academic function in urban children with persistent asthma (7–9 years of age) across one academic year. Data presented in this sub-study include the 196 participants who completed allergy testing at the clinic study visit that occurred during the fall/early winter monitoring period. The Institutional Review Board at Rhode Island Hospital approved the study protocol.

Participants were recruited from the four largest school districts in an urban Northeastern US city, from hospital and ambulatory pediatric clinics, and from a hospital-based asthma educational program by convenience sampling. Inclusion criteria specified child age 7–9 years, child’s legal guardian was willing to participate, caregiver race/ethnicity was self-identified as black/African American, Latino (Dominican or Puerto Rican), or NLW, and child attended public school in one of the targeted school districts in [*blinded for review*].

Children needed to meet criteria for persistent asthma either by prescription for asthma controller medication or recurrent asthma symptoms, e.g., daytime symptoms greater than 2 days a week, nocturnal symptoms greater than 2 times a month, short acting beta agonist use greater than 2 days a week, at least some limitation in normal activity, and/or 2 or more oral steroid bursts per year.²² Exclusion criteria were moderate to severe cognitive impairment as determined by school placement, use of stimulant medication for attention deficit/hyperactivity disorder, additional pulmonary disease or chronic health condition, or a diagnosed sleep disorder (e.g., restless leg syndrome) that would confound the primary hypotheses of the larger study. Children with sleep disordered breathing (SDB) were not excluded as this is highly comorbid among this population.

Data for the current study were collected during the fall and early winter period of each study year (August 15– December 31 from 2010 to 2014). At the initial study visit, demographic information and medication use were collected, followed by a second session at the hospital-based clinic at least 2 weeks later, when asthma severity and allergy status were evaluated. The clinic visit was followed by a one month home-monitoring period in which children wore an actigraph to assess sleep quality and used a handheld device to assess FEV₁. Midway through the monitoring period, study staff conducted a home visit to download and review electronic sleep and pulmonary function data. At the end of the monitoring period, asthma control was assessed.

Standardized training procedures for device use were provided throughout to encourage protocol adherence and ensure appropriate use of the devices.²⁰ Assessments were administered in English or Spanish according to participants' preference, and standardized procedures were used for the translation of measures.²³

Measures

Demographic and Descriptive Information—Primary caregivers provided demographic information (see Table 1). Poverty status was determined by dividing the family's annual income by the Federal per capita poverty threshold for a family of that size.²⁴

Asthma Severity—The clinic study visit consisted of a medical history, physical examination, allergy testing, pulmonary function testing, and review of medications to confirm the diagnosis of asthma and classify asthma severity according to NHLBI EPR-3 guidelines.²²

Allergy Testing—Allergy skin prick testing was performed (Multi Test II device, Lincoln Diagnostics; Decatur, IL) with allergen extracts in addition to positive (histamine 6mg/ml) and negative (50% glycerin) control after adequate medication washout. The following extracts were used from Greer laboratories: *D. pteronyssinus* (10,000 au/ml), *D. farinae* (10,000 au/ml), dog ep. (1:20 w/v), cat hair (10,000 BAU/ml), cockroach mix (German and American) (1: 20 w/v), mold mix 1 (1:20 w/v), *Cladosporium herbarum* (1:40 w/v), mouse (1:20 w/v), ragweed mix (1:20 w/v), Eastern tree mix (1:20 w/v), grass mix 7 (100,000 BAU/ml), Timothy (100,000 BAU/ml), plantain (1:20 w/v), oak (red) (1:20 w/v), birch (red) (1:20 w/v), and maple (sugar) pollen (1:20 w/v). The results of a test were considered to be

positive if the maximum diameter of the wheal was at least 3mm after subtraction of the negative control.

Participants who could not washout of antihistamine (n=10), underwent specific IgE testing (sIgE) (Quest Diagnostics, Chantilly, VA) to Northeast Environmental Panel 76 which contains the following allergens: Dermatophagoides pteronyssinus (d1), Dermatophagoides farinae (d2), cat dander (e1), dog dander (e5), Timothy grass (g6), cockroach (i6), Penicillium notatum (m1), Cladosporium herbarum (m2), Aspergillus fumigatus (m3), Alternaria alternata (m6), maple (Box Elder) (t1), birch (t5), oak (t7), elm (t8), common ragweed (short) (w1), giant ragweed (tall) (w3), English plantain (w9), and Lamb's quarters (goose foot) (w10), with the addition of mouse (e88) and mouse epithelia (e76). A result was considered positive if it was greater than or equal to 0.35kU/L.

Evaluated allergens were subsequently grouped into a categorical variable (Positive/Negative) from either the skin test or sIgE for the following 10 allergen categories: mold, dust mite, dog, cat, cockroach, grass, ragweed, spring trees, plantain weed, and mouse. We used two variables to measure atopy: (1) *Number of positive allergy tests* was a continuous variable ranging from 0 to 10 positive allergy tests. This continuous variable included participants with zero positive results. (2) After reviewing the distribution of positive allergy tests, we created a second categorical variable, *degree of atopy*, for the participants with at least one positive test, to reflect increasing categories of sensitization: 1–2, 3–5, and 6–10 positive allergy tests.

Sleep Disordered Breathing—Primary caregivers completed the Sleep Related Breathing Disorders subscale of the Pediatric Sleep Questionnaire, a well-validated and reliable measure of SDB.²⁵

Environmental Control Subscale—Caregivers reported child exposure to environmental triggers and family efforts to control these exposures during a well-validated semi-structured interview, the Family Asthma Management System Scale (FAMSS).²⁶ Trained interviewers rated participants on an environmental control domain, using a subscale from 1 to 9 to assess child exposure to secondhand tobacco smoke, carpeting in the child's room, family pets, problems with pests, as well as family knowledge and implementation of typical asthma trigger controls. Lower environmental control scores indicated higher levels of exposures to triggers, as well as fewer family efforts to control trigger exposure.²⁶ Interviews were audio-recorded and coded according to a standardized coding system.²⁶

Lung Function—During the monitoring period, children's lung function was measured twice daily by a handheld computerized spirometer (Jaeger AM2; VIASYS Healthcare; Yorba Linda, CA). Participants were instructed to complete 3 “blows” prior to any asthma or allergy medications in the morning and evening. The highest of 3 FEV₁ % predicted values per trial was retained. A series of data cleaning and reduction steps were used, which are available upon request.²⁷ FEV₁ variability, a measure of poor asthma control, was measured by coefficient of variation.²⁸

Sleep Measures—Sleep methods have been described in detail previously.²⁰ Participants wore an actigraph on their non-dominant wrist for the monitoring period (MiniMitter Company, Bend, OR, USA). Sleep outcome variables were computed as the aggregate across the monitoring period. Three sleep quality variables were included: (1) *sleep efficiency* was defined as the % time asleep divided by the total time in bed; (2) *variability in sleep efficiency* was the standard deviation across all monitored days, and (3) *mean # of awakening* per night of at least 3 minutes in duration. Our final sleep outcome variable included *sleep duration*, defined as the total time between evening sleep onset and morning waking.²⁰

Asthma control—At the end of the monitoring period, parents and children completed the Childhood Asthma Control Test (cACT),²⁹ a well-validated questionnaire of asthma control for this age group. Item scores are summed for a total score (0–27) with lower scores indicating poorer asthma control. A score 19 and below denotes poorly controlled asthma.

Statistical Analysis Plan

Analyses were conducted using IBM SPSS Statistics for Windows, V23.0 (IBM Corp., Armonk, NY, USA). For preliminary analyses, a chi-square was used to evaluate differences in race/ethnic group by poverty threshold. An analysis of variance (ANOVA) was used to determine differences in environmental control scores and number of positive allergy tests across race/ethnic group; post-hoc Least Significant Difference (LSD) analyses were used to pinpoint differences between groups. Child age, sex, poverty threshold, environmental control score, antihistamine use, and risk for SDB were evaluated as potential covariates in associations between number of positive allergy tests and sleep outcome variables using Pearson r correlations and t-test analyses. We controlled for covariates that were significantly associated with both predictor and outcome variables in subsequent analyses.

To determine associations between number of positive allergy tests (0–10 continuous variable) and sleep outcomes in addressing our first hypothesis, we used hierarchical regressions and controlled for significant covariates. An ANOVA with post-hoc LSD analyses determined differences in sleep outcomes across degree of atopy (1–2, 3–5, and 6–10 positive allergy tests) and race/ethnic groups. We also looked within each race/ethnic group and used (1) Pearson's r correlations to determine associations between number of positive allergy tests and each sleep outcome, and (2) ANOVAs to determine differences in sleep outcomes across degree of atopy.

To address our second hypothesis, t-test analyses were conducted to determine differences in sleep outcome variables by sensitization to specific indoor allergens (yes/no sensitized to mouse, cockroach, dust mite, cat, or dog).

Finally, we tested FEV₁, FEV₁ variability, and asthma control as moderators in associations between number of positive allergy tests and sleep outcome variables for the entire group to address our third hypothesis. In hierarchical regressions predicting sleep variables, covariates were entered as the first step, number of positive allergy tests second, the moderator third, and the interaction term between number of positive allergy tests and the moderator as the final step. Prior to moderation analyses, predictor and moderator variables

were centered and a product term was created from the centered predictor and moderator. In analyses with a significant interaction term, the moderator was dichotomized by median split and follow-up regression analyses were conducted examining the association between number of positive allergy tests and the sleep outcome at high and low levels of the moderating variable. Simple slopes were also plotted to aid in interpretation of moderation results.

Results

Descriptive Data

Characteristics of the 196 child participants who underwent allergy testing in Project NAPS can be found in Table 1. Child participants studied were 53% male, 85% minority and 70% living at or below poverty threshold, 44% moderate/severe persistent in asthma severity, and 70% on inhaled corticosteroid (ICS) therapy by self-report. Allergic sensitization and environmental control data are presented in Table 2. For seasonal pollen, 4.6% of participants were ragweed-sensitized and in-season. Other types of pollen were not applicable to the time-frame of this study.

Preliminary Analyses

Differences in variables by race/ethnic group and child sex are presented in Table 3. Child age was significantly correlated with sleep duration ($r = -.28, p = .00$), with younger children experiencing longer sleep duration. Children who were at or below poverty threshold had more variability in their sleep efficiency than children above poverty threshold ($t(169) = -2.91, p = .004$). Environmental control scores from the FAMSS, risk for SDB, and first generation antihistamine use were not associated with number of positive allergy tests, degree of atopy, or any sleep outcomes. Second generation antihistamine use was only associated with sleep duration ($t(175) = -2.164, p = .032$). Given that second generation antihistamine use was not associated with our predictor variables, it was not included as a covariate in analyses with sleep duration.

Associations between allergic sensitization and objective sleep outcomes

Controlling for child sex, number of positive allergy tests (0–10) predicted mean *sleep efficiency* ($F(1,176) = 5.60, \beta = -.18, p = .02$) and mean number of *night awakenings* ($F(1,179) = 4.04, \beta = .15, p = .046$). Children with a higher number of positive allergy tests experienced less efficient sleep and more night awakenings. Number of positive allergy tests was also significantly associated with *variability in sleep efficiency* ($\beta = .22, p = .003$), such that children with more positive allergy tests had more variability in their sleep efficiency. No significant association emerged between number of positive allergy tests and sleep duration.

For those with at least one positive test, *variability in sleep efficiency* also differed across degree of atopy ($F(2, 147) = 6.36, p = .002$). Children with 3–5 or 6–10 positive allergy tests experienced more variability in sleep efficiency ($M = 4.36$ vs. 4.92 , respectively) than children with 1–2 positive allergy tests ($M = 3.51$). Mean sleep efficiency, night awakenings and sleep duration did not differ across degree of atopy.

Race/ethnic differences in sleep outcomes

African American children experienced less efficient sleep than both Latino and NLW children ($F(2, 176) = 3.67, p = .027, \eta^2 = .04$). Within ethnic groups, no significant associations emerged between number of positive allergy tests/degree of atopy and sleep outcomes in African American and NLW children. However, within Latino children, number of positive allergy tests was significantly associated with variability in sleep efficiency ($r = .30, p = .004$). Latino children with more positive allergy tests experienced more variability in their sleep efficiency than Latinos with less positive allergy tests. Latino children with 6–10 positive allergy tests also experienced more variability in sleep efficiency than Latinos with 1–2 or 3–5 positive allergy tests ($F(2, 70) = 6.43, p = .003$). Similarly, mean sleep duration was lowest for Latinos in the 6–10 positive allergy test category.

Sensitization to specific indoor perennial allergens

We found associations in several mean sleep outcomes with *cockroach* and *cat* sensitization. Cockroach sensitization was associated with shorter sleep duration. Cat sensitization was associated with less efficient sleep and more variability in sleep efficiency (Table 4). Sleep outcomes did not differ across sensitization to other indoor allergens (e.g., mouse, dust mite, dog).

Moderation analyses in associations between number of positive allergy tests and sleep outcomes

We considered FEV₁, FEV₁ variability, and asthma control as moderators in associations between number of positive allergy tests and sleep efficiency, variability in sleep efficiency, and mean number of night awakenings. We controlled for child sex in the models including sleep efficiency and number of night awakenings.

FEV₁ variability was a significant moderator in the association between number of positive allergy tests and variability in sleep efficiency ($\beta = .16, p = .04, R^2 \text{ adjusted} = .07$; see Table 5). FEV₁ variability for each participant was computed by dividing the standard deviation of FEV₁ across the monitoring period by the mean FEV₁ (across the monitoring period) and multiplying by 100. The median FEV₁ variability score for the sample was calculated and dichotomized by median split. Scores above the median were labeled as “1” (high level) and scores below the median were labeled as “0” (low level). Follow-up regression analyses were conducted examining the association between number of positive allergy tests and variability in sleep efficiency (See Figure 1). For children above the median FEV₁ value of variability, more allergy tests were associated with more variability in sleep efficiency ($\beta = .38, p = .00, R^2 \text{ adjusted} = .13$). On the other hand, for children below the median (less variability in FEV₁), there was no significant association between number of positive allergy tests and variability in sleep efficiency ($\beta = .04, p = .62, R^2 \text{ adjusted} = -.01$). We did not find that FEV₁ or asthma control were significant moderators in associations between number of positive allergy tests and sleep outcomes.

Discussion

To our knowledge, this is the first study to measure allergic sensitization in asthma and objective measures of sleep using actigraphy in children. In this sample of urban school-children with persistent asthma, the majority of whom reported using ICS therapy, we found that children who were sensitized to more aeroallergens had lower overall sleep efficiency, more variability in their sleep efficiency, and more night awakenings. These are important findings given that to date, most measures of sleep in pivotal studies of nocturnal asthma^{14,17} have used subjective diary data. Not only did we demonstrate a link between allergic sensitization and specific sleep outcomes assessed through objective methods, but we found the association to be moderated by FEV₁ variability. For children with increased FEV₁ variability (an objective measure of poor asthma control), an increased number of positive allergy tests was associated with more changes in children's sleep efficiency (an objective measure of poor sleep). This variability in sleep efficiency can be an important marker of uncontrolled asthma or an indicator of frequent disruptions related to wakings due to asthma or other urban stressors.

Our study also found objective measures of sleep outcomes to be associated with cockroach sensitization, but not mouse sensitization. In the literature, there has been an ongoing debate regarding which of these two allergens is more significantly linked with morbidity in urban environments, sometimes revealing paradoxical results.^{30–34} We also found cat sensitization to be associated with sleep outcomes. Neither dust mite nor dog sensitization were associated with sleep outcomes in this urban group. Recent studies from the Inner City Asthma Consortium (ICAC) have found that multiple allergen sensitization (not just individual allergens) has effects on asthma severity and is related to difficult-to-control asthma.^{35,36} Likewise, seasonal data show evidence for the role of multiple allergic sensitization in increased asthma exacerbations during the fall season as inner-city children return to school.³⁷ Our findings suggest an association between increased number of positive allergy tests and poor sleep outcomes during a similar time frame in urban children with asthma.

This sample was primarily minority in race/ethnicity. Our findings within the Latino subgroup suggest that Latino children with more positive allergy tests experienced more variability in sleep efficiency, and that the sub-group with 6–10 positive allergy tests were at higher risk for more variable sleep efficiency and less sleep duration than Latinos with 1–5 positive tests. This is interesting because African American children in our sample had a higher mean number of positive allergy tests and higher proportion in the 6–10 positive category over other groups, but did not follow the same patterns in increased sensitization and sleep as Latinos. To date, genetic studies do not indicate a clear and overwhelming role for genetic ancestry in the observed disparity in allergic sensitization for African American and Latinos, which may suggest a greater influence of local exposures or location of residence on atopy.^{38,39} We found no differences in environmental control scores, however, between our African American and Latino sub-groups. Further study is needed with more proportionate sampling of ethnic sub-groups.

Avenues for Intervention

Studies of nocturnal asthma cite potential therapies for nocturnal asthma which include chronopharmacologic dosing strategies during exacerbations (e.g., dose prednisone at 3pm), use of long acting beta-agonists (LABAs) in conjunction with ICS, and use of sustained release theophylline to achieve peak concentration when airflow limitation is greatest.⁴⁰ Notably, although 70% of our sample reported ICS therapy, only 5% of our sample reported using LABAs. Use of combination therapy with LABAs presents a clear target for future intervention in similar samples.

The overall goal of intervention is to develop an integrated and multifaceted environmental control strategy⁴¹ that focuses on allergic triggers in the bedroom. Additional goals include increasing access to allergy testing to identify allergic sensitization, improving medication adherence particularly if nocturnal asthma is present prior to bed, and developing interventions focusing on sleep behaviors as demonstrated by our prior work.¹⁹

Limitations

This study is limited by its small sample size, convenience sampling, and disproportionate number of families per ethnic group. The disproportionate number of Latino participants skews the results of the pooled data for the association between degree of atopy and sleep outcomes and limits its general applicability. We also note that our moderational analyses were conducted on 156 participants due to missing PFT and sleep outcome data, which warrants replication in larger samples. We acknowledge that our environmental exposure data are limited. Indoor environmental exposures were not measured objectively, as collecting allergen samples was beyond the scope of our study design. Self-report of exposure to pests did not specify whether it was mouse or cockroach. Pollen counts were obtained from a regional source; however, the bulk of the monitoring period occurred outside the ragweed pollen season and were not applicable for the following reasons: recruitment began with the start of the schoolyear (end August/September); continued on a rolling basis; and skin testing followed by the month-long monitoring period did not overlap often with elevated ragweed pollen counts, which typically ended by September 30 (pollen data available upon request). For indoor allergens, associations between individual allergens and sleep outcomes need further study with objective sampling and larger numbers. Allergic sensitization on testing does not always translate into clinically significant symptoms caused by individual allergens⁴². Race also was measured by self-report as genetic sampling was beyond the scope of this study. Finally, neither allergic rhinitis nor weight status were a focus of this report. Associations between allergic sensitization, allergic rhinitis control, weight status, degree of sleep disordered breathing, and objective markers of sleep are future areas of study. Likewise, future studies should assess medication adherence, specifically access to LABAs, availability of rescue therapy at night, and the role of the environment in Latino sub-groups.

Conclusion

Results from this important study of urban school-children with persistent asthma suggest that increased allergic sensitization was associated with poor sleep outcomes objectively measured during a one-month monitoring period in the fall/early winter. The association

between allergic sensitization and sleep outcomes depended on variability in lung function, so that children with more variable FEV₁ were more at risk for increased variability in sleep efficiency. These results have important implications on intervening through several avenues (e.g. behavioral, environmental, access to testing) to enhance the sleep quality of this group, as sleep can affect multiple areas of children's functioning. The value of this study is its real-time monitoring of sleep and asthma outcomes in a sample of difficult-to-reach, atopic school-age children.

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Abbreviations

ANOVA	Analysis of Variance
cACT	Childhood Asthma Control Test
CAMP	Childhood Asthma Management Program
EPR-3	Third Expert Panel Report
FAMSS	Family Asthma Management System Scale
FEV1	Forced Expiratory Volume in 1 second
ICAC	Inner City Asthma Consortium
ICS	Inhaled Corticosteroid
LABA	Long-acting Beta Agonist
LSD	Least Significant Difference
NAPS	Nocturnal Asthma and School Performance
NHANES	National Health and Nutrition Examination Survey
NHLBI	National Heart Lung and Blood Institute
NLW	Non-Latino White
RI	Rhode Island
SDB	Sleep Disordered Breathing
sIgE	Specific IgE

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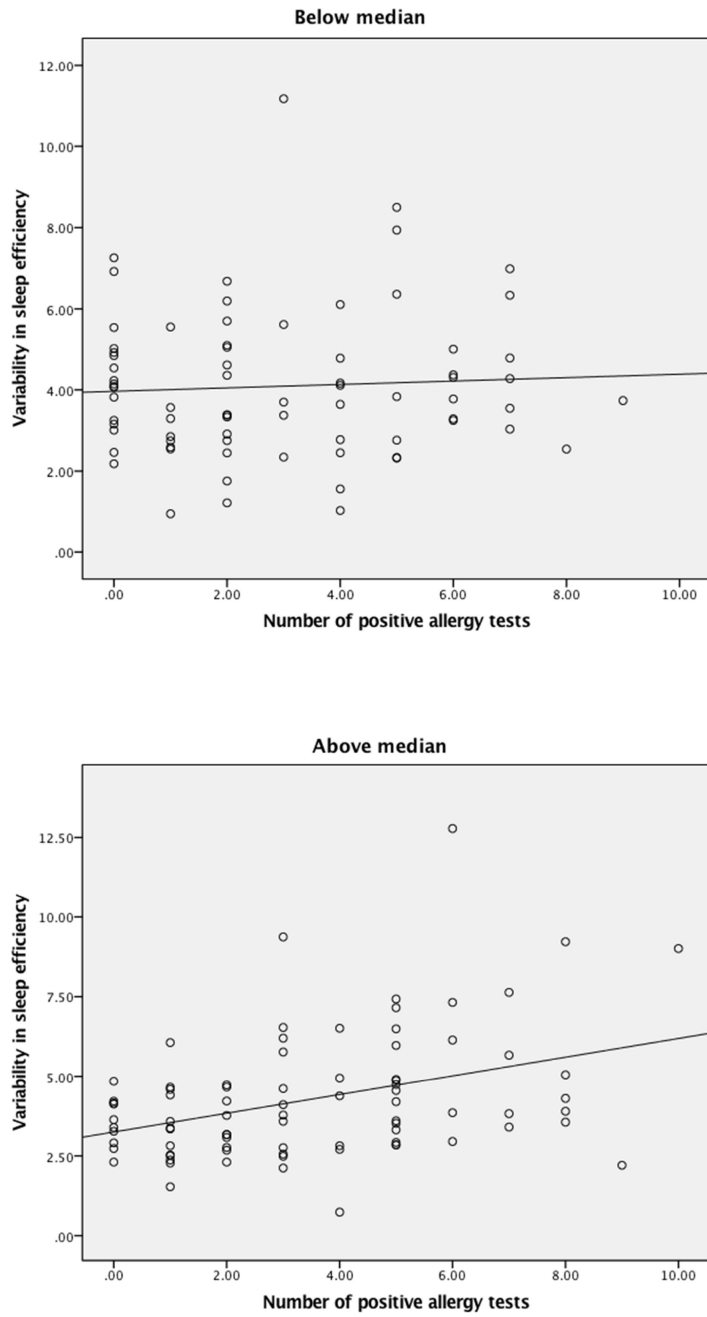


Figure 1. Number of positive allergy tests and variability in sleep efficiency by variability in FEV₁ (median split)

Table 1

Sample characteristics (n= 196)

Demographic variable	
Child age in years, <i>M (SD)</i>	8.31 (.87)
Male (%)	53
Caregiver race/ethnicity (%)	
Black	34
Latino	51
Non-Latino White	15
At/below poverty threshold (%)	70
Number of people in household, <i>M (SD)</i>	4.70 (1.76)
Asthma Severity (%)	
Mild persistent	56
Moderate persistent	33
Severe persistent	11
Asthma poorly controlled (%)	44
Asthma medications (%)	
Inhaled corticosteroid	70
Long-acting beta agonists	5
AR medications (%)	
Intranasal steroid	11
Antihistamine use (%)	
1 st generation antihistamine	18
2 nd generation antihistamine	26
FEV ₁ % predicted, <i>M (SD)</i>	83.9 (12.80)
Variability in FEV ₁ % predicted, <i>M (SD)</i>	14.97 (5.96)
Risk for sleep disordered breathing (%)	49
BMI percentile for age, <i>M (SD)</i>	74.32 (25.22)
Sleep health indicators	
Sleep efficiency (%), <i>M (SD)</i>	86.54 (3.48)
Variability in sleep efficiency, <i>M (SD)</i>	4.17 (1.87)
Sleep duration (minutes), <i>M (SD)</i>	554.49 (35.04)
Mean number of awakenings per night, <i>M (SD)</i>	5.35 (2.42)

Table 2

Allergic Sensitization Data (n= 196)

Variable	
Number of positive allergy tests (0–10), <i>M (SD)</i>	3.18 (2.47)
Degree of atopy (%) [*]	
1–2 positive allergy tests	29
3–5 positive allergy tests	36
6–10 positive allergy tests	18
Proportion sensitized to allergens (%)	
Mouse	37
Cockroach	28
Dust mite	61
Cat	32
Dog	25
Ragweed	24
Spring Trees	47
Grass	28
Plantain pollen	10
Mold	28
Mean environmental control score (1–9), <i>M (SD)</i>	3.74 (2.56)
Pest exposure (%)	40
Carpet in sleeping room	43
Dog ownership	24
Cat ownership	11
Smoke exposure	41

^{*} 17% of the sample did not have a positive allergy test

Table 3

Preliminary analyses

Differences by race/ethnicity						
	Black ^a (n=66)	Latino ^b (n=100)	NLW ^c (n=30)	Test statistic	<i>p</i>	Post hoc comparisons
At or below poverty threshold	66%	79%	46%	$\chi^2(2) = 11.75$.003***	--
Mean environmental control score (1-9)	3.80 (2.59)	4.04 (2.61)	2.60 (2.04)	$F(2, 192) = 3.78$.025*	a>c; b>c; a=b
# of positive allergy tests (0-10)	3.92 (1.79)	2.92 (2.48)	2.43 (1.79)	$F(2, 193) = 5.11$.007***	a>b; a>c; b=c
Degree of atopy, [†]				$\chi^2(4) = 11.02$.026***	--
1-2	21%	30%	43%			
3-5	39%	33%	37%			
6-10	29%	16%	3%			
Differences by child sex						
	Male (n=104)	Female (n=92)		Test statistic	<i>p</i>	
# of positive allergy tests (0-10)	3.56 (2.53)	2.76 (2.36)		$t(194) = 2.27$.024*	
Sleep efficiency	85.93 (3.84)	87.26 (2.85)		$t(177) = -2.61$.01*	
Variability in sleep efficiency	4.23 (1.71)	4.11 (2.04)		$t(177) = .44$.66	
# of night awakenings	5.72 (2.81)	4.91 (1.81)		$t(180) = 2.27$.024***	
Sleep duration	553.63 (36.0)	555.51 (34.05)		$t(177) = -.36$.72	

Note.

* $p < .05$,

*** $p < .01$;

Test statistic with $\chi^2(df)$ refers to chi-square, $F(df)$ refers to ANOVA, and $t(df)$ refers to t-test.

[†] 11% of Black, 21% of Latino, and 17% of NLW children did not have a positive allergy test.

Table 4

Sleep outcomes by cockroach and cat sensitization

	No sensitization ^a	Sensitization ^b	<i>t</i>	<i>df</i>	<i>p</i>
Cockroach	(n=128)	(n=50)			
Sleep efficiency	86.69 (3.28)	86.34 (3.74)	.62	176	.54
Variability in sleep efficiency	4.08 (1.71)	4.43 (2.25)	-1.12	176	.26
Night awakenings	5.29 (2.32)	5.34 (2.48)	-.12	176	.91
Sleep duration	558.45 (34.46)	554.78 (35.14)	2.37	176	.02*
Cat	(n=122)	(n = 57)			
Sleep efficiency	86.92 (3.33)	85.73 (3.67)	2.62	177	.03*
Variability in sleep efficiency	3.97 (1.65)	4.61(2.22)	4.87	177	.03*
Night awakenings	5.18 (2.42)	5.70 (2.40)	-1.37	180	.17
Sleep duration	554.08 (34.82)	555.36 (35.79)	-.23	177	.82

*Note.**** $p < .01$;* $p < .05$

Table 5

Results testing moderators in associations between number of positive allergy tests and sleep outcomes (n = 156)

	Variability in Sleep Efficiency			
	β	B	95% CI for B	R^2 adjusted
Number of positive allergy tests	.23**	.17	.06 – .29	
FEV ₁ variability	.001	.00	-.05 – .05	
Allergy tests × FEV ₁ variability	.16*	.02	.001 – .04	.07

Note.

* $p < .05$,

** $p < .01$;

Results for models with nonsignificant interaction terms are not shown.