

Sociodemographic correlates of indoor allergen sensitivity among United States children

Lori A. Stevenson, MPH,^{a,b} Peter J. Gergen, MD, MPH,^c
Donald R. Hoover, PhD, MPH,^{a,d} David Rosenstreich, MD,^e
David M. Mannino, MD,^f and Thomas D. Matte, MD^{a,f} *New York and
Bronx, NY, Rockville, Md, Piscataway, NJ, and Atlanta, Ga*

Background: Exposure to indoor allergens is associated with asthma morbidity. Nationally, asthma morbidity disproportionately affects socially disadvantaged populations, but it is unclear whether exposure to indoor allergens follows a similar pattern.

Objective: We sought to examine the national prevalences and demographic correlates of sensitivity to indoor allergens related to asthma.

Methods: Analysis of a cross-sectional survey of a representative sample of 4164 United States children aged 6 to 16 years who participated in allergen testing in the Third National Health and Nutrition Examination Survey from 1988 to 1994 was performed. The main outcome measures were sensitivity reactions to cockroach, dust mite, cat, and *Alternaria alternata*, as measured via skin prick testing.

Results: Multivariate models, including sex, age, race-ethnicity, education, poverty, family history, region of country, housing age, crowding, and urban residence, revealed significant racial-ethnic disparities in sensitivity. Compared with white children, African American children had higher odds ratios (ORs) of cockroach or dust mite sensitivity (cockroach OR, 2.5 [95% CI, 1.9-3.2]; dust mite OR, 1.3 [95% CI, 1.0-1.7]), as did Mexican American children (cockroach OR, 1.9 [95% CI, 1.3-2.8]; dust mite OR, 1.6 [95% CI, 1.2-2.2]). African American children also had significantly higher odds of sensitivity to *A. alternata* (OR, 2.1 [95% CI, 1.5-2.8]).

Conclusions: African American and Mexican American children are substantially more likely than white children to be sensitized to allergens important in asthma. Differences in indoor allergen sensitivity are consistent with racial differences in asthma morbidity. Along with other data, these findings

suggest that racial disparities in housing, community, or both environmental factors play a role in determining national patterns of asthma morbidity. (*J Allergy Clin Immunol* 2001;108:747-52.)

Key words: Race, indoor allergens, sensitivity, children, prevalence, NHANES

Asthma is a leading cause of childhood hospitalization, emergency department use, and school absences, representing a substantial portion of allergy-related morbidity.¹ Sensitivity to allergens is a hallmark of childhood asthma.² In particular, sensitivity to the common indoor allergens dust mite, cat, cockroach, and *Alternaria alternata* (an indoor and an outdoor fungus) has been associated with asthma morbidity or severity in numerous studies.³⁻⁹

Asthma morbidity and severity disproportionately affects socially disadvantaged populations, including African Americans and residents of low-income inner-city neighborhoods.¹⁰⁻¹³ Although racial differences in asthma prevalence are small nationally, African Americans have substantially higher rates of asthma mortality, hospitalization, and emergency department visits than whites.¹² To reduce these social disparities in asthma morbidity at the national level, the factors contributing to such disparities must be identified. Differences in exposure and sensitivity to indoor allergens have been explored as one set of factors accounting for the racial gradient in asthma morbidity.^{7,8,14}

Allergic sensitivity is a consequence of a genetic predisposition toward atopy and exposure to sufficient levels of allergen to provoke an immune response. Exposure to common indoor allergens is influenced by housing and social conditions,^{15,16} as well as regional factors, like climate, that may favor the proliferation of common allergens, such as cockroach, dust mite, or mold.¹⁷⁻²⁰ For example, in the arid climate of Los Alamos, New Mexico, dust mite exposure is rare,¹⁹ whereas in the humid climates of Atlanta and New Orleans, dust mite is a predominant allergen.^{7,20} However, geographic variation in allergen exposure limits the generalizability of local studies. Moreover, past studies documenting the prevalence and demographic distribution of allergen sensitivity in the general US population^{21,22} are now dated and did not assess cockroach allergy, a potentially important contributor to asthma morbidity. Therefore we assessed the prevalence and demographic correlates of sensitivity to 4 indoor allergens (German cockroach, dust mite, cat, and *A*

From ^aNew York Academy of Medicine, Center for Urban Epidemiologic Studies, New York; ^bNew York City Health and Hospitals Corporation, New York; ^cCenter for Primary Care and Research, Agency For Healthcare Research and Quality, Rockville; ^dthe Department of Statistics and Center for Health, Health Care Policy and Aging Research, Rutgers University, Piscataway; ^ethe Division of Allergy and Immunology Montefiore Medical Center, Albert Einstein College of Medicine, Bronx; and ^fthe Division of Environmental Hazards and Health Effects, National Center for Environmental Health, Centers for Disease Control and Prevention, Atlanta.

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Reprint requests: Lori A. Stevenson, MPH, Center for Urban Epidemiologic Studies, New York Academy of Medicine, 1216 Fifth Ave, New York, NY 10029.

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Abbreviations used

AHS: American Housing Survey
 NHANES III: Third National Health and Nutrition
 Examination Survey
 OR: Odds ratio
 SMSA: Standardized metropolitan standard area

alternata) in a nationally representative sample of children from the third National Health and Nutrition Examination Survey 1988-1994 (NHANES III).

METHODS

NHANES III (1988-1994) was a national survey of the health status of the US civilian, noninstitutionalized population. The survey used a complex, stratified probability design over 2 independent phases to examine a representative sample of the US civilian noninstitutionalized population. NHANES weighting methodology takes into account the probabilities of selection, including the intentional oversampling of certain racial-ethnic and age groups and nonresponse to provide an unbiased estimate of health statistics for the US population. A detailed description of the design and operation of NHANES III has been published elsewhere.²³

We restricted analyses to children aged 6 to 16 years of white non-Hispanic, black non-Hispanic, or Mexican American race-ethnicity (n = 5425). Children with missing-incomplete (n = 465) or invalid (n = 287) allergen test results or those missing information for specific covariates (n = 133) were excluded. Overall, 73% (4164) of interviewed children were included in these analyses.

Testing for immediate hypersensitivity to dust mite (*Dermatophagoides farinae*), German cockroach, cat, *A alternata*, and positive (histamine phosphate) and negative (50% glycerol saline) controls was performed via prick-puncture test with a 25-gauge needle by trained technicians according to a standardized protocol. All allergens were Food and Drug Administration–approved, commercially available mixtures.²³

Skin reactions were read 15 to 20 minutes after testing. The allergen test battery was considered valid if the mean wheal diameter of the positive control was at least 3 mm larger than that of the negative control. Invalid tests were excluded from analysis. Reactions for each allergen were considered positive if the mean wheal diameter of the reaction was at least 3 mm after subtracting the mean diameter of the negative control wheal.

SUDAAN, a statistical package designed for the complex survey design and weights of NHANES, was used for final weighted analyses.²⁴ Overall and covariate-specific prevalence estimates and standard errors for sensitivity to each allergen were calculated. Multivariate logistic regression was used to determine the relationship between allergen sensitivity and sociodemographic variables. We included sociodemographic covariates that had been associated with indoor allergen sensitivity or exposure by others or that were associated ($P < .05$) with allergen sensitivity in univariate analyses. Significance testing for each covariate was based on the Satterthwaite adjusted χ^2 . The following variables were included: sex (male, female); race-ethnicity (as noted above); age in years (6-11 or 12-16); education of the adult household respondent (<12th grade or ≥ 12 th grade); poverty/income ratio, which is the ratio of the reported annual family income from all sources to the annual poverty threshold adjusted for family size²⁵ (low, <1.3; mid, 1.3-2.99; high, ≥ 3.0 ,²⁶ or unknown [n = 316]); region of residence; and family history of atopy-allergy (a report of a biologic parent with a doctor's diagnosis of asthma or hay fever). Additionally, households were

classified as not crowded (<0.5 persons/room), crowded (0.51 persons/room to 1.50 persons/room), or severely crowded (≥ 1.51 persons/room).²⁷ Housing age was categorized by reported year of construction and relates to 3 distinct eras in housing construction (<1946 [pre-World War II]; 1946-1973 [post-World War II], ≥ 1974 [post-energy crisis]; and unknown [n = 545]).

The final NHANES III data set does not allow for differentiation of persons residing in central cities from those who live in greater metropolitan areas. However, during phase I of NHANES III (1988-1991), the following categories were used: (1) central city of the standardized metropolitan statistical area (SMSA); (2) in the SMSA but not in the central city; and (3) not in the SMSA. We conducted separate analyses with only the phase I sample population (n = 2149) to describe the association between urbanicity and allergen sensitivity.

We limited our assessment of possible interactions between covariates to testing 3 hypotheses: (1) the relation of race to allergen sensitivity varies by poverty status; (2) the relation of poverty to allergen sensitivity varies by urban residence; and (3) the relation of race to allergen sensitivity varies by urban residence. Terms representing these interactions were tested in models for each allergen. Only the interaction for hypothesis 3 in the model for cockroach sensitivity was significant and is presented.

RESULTS

Overall, 43% of children were sensitive to at least 1 of 4 indoor allergens (Table I). Almost 20% were sensitive to cockroach, 25% to dust mite, and 15% to 16% to cat or *A alternata*. Boys, older children, African Americans, and residents of the Northeast had higher prevalence of sensitivity to at least one allergen. Additionally, 21% of children were sensitive to only 1 allergen, 14% were sensitive to 2, 6% were sensitive to 3, and 2% were sensitive to all 4 (data not shown). Multivariate models for each allergen are discussed below. Covariates are discussed in the order of their significance in each model.

Cockroach

Race-ethnicity was a significant independent correlate of cockroach sensitivity (Table II). Among African American children, the odds of cockroach sensitivity were more than twice those of white children (adjusted odds ratio [OR], 2.5; 95% CI, 1.9-3.2). Similarly, Mexican American children had significantly higher odds of sensitivity (adjusted OR, 1.9; 95% CI, 1.3-2.8). Family history of allergy-asthma, older age, and male sex were each independently associated with cockroach sensitivity in multivariate analysis. No other covariates were significant in the adjusted model.

In the model using data only from phase I of NHANES III, we observed a significant interaction between race and urban residence ($P < .001$). Including this term in a model with all other covariates indicated that racial disparities in cockroach sensitivity were strongest in the inner city (Table III). In this model crowding was also significantly ($P < .01$) associated with sensitivity. Children living in homes with 1.51 or more persons per room had odds of cockroach sensitivity 2.4 times (95% CI, 1.24-4.72) those of children in the least crowded conditions. Parameter estimates for other covariates did not change meaningfully in this model (data not shown).

TABLE I. Sample characteristics and prevalence of allergen sensitivity

	Sample (n)	Weighted (%)	Sensitive (%)				
			Any allergen	Cockroach	Dust mite	Cat	<i>A alternata</i>
All children	4164	100.0	43 (1.4)	20 (0.9)	25 (1.4)	15 (1.1)	16 (1.2)
Sex							
Male	2057	52	48 (1.8)	22 (1.5)	29 (1.9)	17 (1.8)	19 (1.8)
Female	2107	48	37 (1.6)	18 (1.0)	21 (1.3)	13 (1.1)	12 (1.0)
Age (y)							
6-11	2520	53	38 (1.6)	17 (1.1)	22 (1.6)	11 (1.1)	13 (1.5)
12-16	1644	47	48 (1.8)	22 (1.5)	29 (1.9)	20 (1.5)	18 (1.9)
Race-ethnicity							
White NH	1116	73	41 (1.9)	16 (1.1)	24 (1.8)	16 (1.4)	14 (1.4)
Black NH	1502	17	52 (2.0)	33 (1.8)	29 (2.0)	15 (1.5)	22 (1.5)
Mexican American	1546	10	43 (1.9)	25 (2.1)	24 (1.7)	12 (0.9)	14 (2.3)
PIR							
<1.3	1929	29	42 (2.0)	25 (1.5)	22 (1.7)	13 (2.1)	16 (2.1)
1.3-3	1329	38	44 (2.4)	19 (1.2)	28 (2.2)	16 (1.8)	15 (2.1)
≥3.0	590	28	40 (2.4)	15 (1.9)	22 (2.4)	16 (2.1)	16 (2.1)
Unknown	316	5	53 (5.6)	24 (4.0)	35 (6.5)	16 (3.6)	16 (5.4)
Education (y)							
<12	1746	24	43 (1.9)	25 (2.0)	24 (1.8)	10 (1.8)	13 (2.0)
≥12	2418	77	43 (1.6)	18 (1.1)	25 (1.8)	17 (1.4)	16 (1.4)
Region							
Northeast	404	19	47 (3.1)	18 (0.9)	33 (3.9)	19 (3.9)	13 (1.3)
Midwest	750	22	40 (3.8)	22 (2.6)	23 (2.2)	13 (2.0)	17 (2.6)
South	1847	37	37 (2.0)	21 (1.3)	27 (2.0)	13 (1.4)	15 (1.7)
West	1163	22	41 (2.3)	16 (1.4)	17 (1.9)	18 (2.1)	18 (3.5)
Family history							
No	3413	74	41 (1.4)	18 (0.9)	24 (1.4)	13 (1.0)	13 (1.2)
Yes	751	26	49 (2.6)	23 (1.9)	27 (2.5)	21 (2.5)	22 (2.4)
Crowding (person/room)							
>1.51	407	3	41 (3.0)	32 (2.5)	18 (3.2)	7 (2.2)	13 (3.2)
1.01-1.5	3200	74	42 (1.6)	20 (0.9)	25 (1.5)	15 (1.3)	14 (1.0)
<1.0	557	23	47 (3.1)	18 (2.0)	27 (2.9)	18 (2.1)	19 (3.2)
Year house was built							
<1946	665	20	42 (2.9)	22 (1.8)	25 (2.1)	13 (2.4)	12 (2.0)
1946-1973	1687	34	43 (2.3)	19 (1.5)	25 (2.6)	16 (1.6)	17 (1.4)
≥1973	1267	37	43 (2.3)	17 (1.8)	24 (1.9)	15 (1.8)	16 (2.6)
Unknown	545	10	45 (3.2)	26 (1.5)	26 (3.0)	16 (3.7)	15 (2.9)

NH, Non-Hispanic; PIR, poverty/income ratio.

Dust mite

Male sex, residence outside of the West, and older age were significantly associated with dust mite sensitivity in multivariate analysis. There was also a strong independent association between nonwhite race-ethnicity and dust mite sensitivity (Table II), and compared with white children, both African American (OR, 1.3; 95% CI, 1.0-1.7) and Mexican American (OR, 1.6; 95% CI, 1.2-2.2) children had higher odds of dust mite sensitivity.

Cat

Being in the older age group (OR, 2.0; 95% CI, 1.5-2.5) was the strongest correlate of cat sensitivity after adjusting for all other covariates (Table II). Children with a positive family history of asthma or allergy also had higher odds of cat sensitivity. The odds of cat sensitivity were significantly decreased among children in households where the adult household respondent had less than

a high-school education (OR, 0.6; 95% CI, 0.4-0.9) and among children residing in the Midwest (OR, 0.6; 95% CI, 0.4-0.9) or the South (OR, 0.6; 95% CI, 0.4-1.00).

A alternata

In adjusted analysis the odds for *A alternata* sensitivity among African American children were more than twice those of white children (OR, 2.1; 95% CI, 1.6-2.8; Table II). Positive family history and male sex were also independently associated with sensitivity to *A alternata*.

DISCUSSION

We have estimated that more than 2 in 5 children in a nationally representative sample were sensitive to at least 1 of 4 major indoor allergens. These analyses provide the first evidence that the racial differences in cockroach allergen sensitivity noted in local studies of asthmatic subjects²⁸ exist in the general US population, independent

TABLE II. ORs for indoor allergen sensitivity

Covariate (referent)	German cockroach		Dust mite		Cat		<i>A alternata</i>	
	Adjusted OR	95% CI	Adjusted OR	95% CI	Adjusted OR	95% CI	Adjusted OR	95% CI
Sex (female)	*		†		*		†	
Male	1.3	1.0-1.7	1.5	1.3-1.9	1.3	1.0-1.7	1.8	1.3-2.3
Age, y (6-11)	†		†		†			
12-16	1.4	1.1-1.8	1.5	1.2-1.8	2.0	1.5-2.5	1.4	1.0-2.1
Race-ethnicity (white NH)	†		†				†	
Black NH	2.5	1.9-3.2	1.3	1.0-1.7	1.2	0.9-1.6	2.1	1.6-2.8
Mexican American	1.9	1.3-2.8	1.6	1.2-2.2	0.9	0.6-1.3	1.1	0.7-1.7
PIR (≥ 3.0)								
<1.3	1.3	0.9-2.0	1.1	0.8-1.5	1.2	0.7-2.0	1.2	0.8-1.7
1.3-3	1.1	0.8-1.6	1.4	1.0-1.9	1.2	0.9-1.8	1.1	0.7-1.7
Unknown	1.8	0.7-2.4	2.0	0.9-4.2	1.5	0.7-3.0	1.1	0.5-2.5
Education, y (≥ 12)					*			
<12 y	1.2	0.8-1.6	0.9	0.7-1.3	0.6	0.4-0.9	0.8	0.5-1.3
Region (West)			†		*			
Northeast	1.3	0.9-1.9	2.8	1.8-4.5	1.0	0.7-1.5	0.7	0.4-1.2
Midwest	1.6	1.1-2.3	1.6	1.1-2.4	0.6	0.4-0.9	0.9	0.5-1.6
South	1.4	1.1-1.9	1.9	1.3-2.8	0.6	0.4-1.0	0.7	0.4-1.3
Family history (no history)	†				†		†	
Yes	1.6	1.2-2.1	1.2	0.9-1.5	1.5	1.1-2.1	2.1	1.5-2.9
Crowding, persons/room (<1.0)								
>1.51	1.4	1.0-1.9	0.6	0.3-1.1	0.5	0.2-1.2	0.7	0.3-1.3
1.01-1.5	0.9	0.7-1.3	0.8	0.6-1.1	0.9	0.6-1.4	0.7	0.5-1.0
Year house was built (≥ 1974)								
<1946	1.3	0.9-1.8	0.9	0.7-1.2	0.8	0.5-1.3	0.6	0.4-1.2
1946-1973	1.0	0.6-1.4	1.0	0.7-1.5	1.0	0.7-1.6	1.0	0.6-1.5
Unknown	1.2	0.8-1.8	0.9	0.6-1.4	1.1	0.6-2.1	0.7	0.5-1.2

ORs are adjusted for all covariates in the Table.

NH, Non-Hispanic.

* $P < .05$.† $P < .01$.**TABLE III.** ORs for cockroach sensitivity by race and urbanicity

	Central city Of SMSA		Not central city of SMSA		Not in SMSA	
	City	95% CI	Suburb	95% CI	Rural	95% CI
White	1		1.9	0.9-3.7	2.2	1.0-4.9
African American	4.6	2.0-10.7	3.2	1.6-6.5	3.2	1.6-6.6
Mexican American	3.4	1.3-8.9	2.5	1.0-6.3	2.5	0.9-6.8

Adjusted for age, sex, poverty/income ratio (PIR), education, family history, region, housing age, and crowding.

of other socioeconomic factors. We also found that racial differences in cockroach sensitivity are especially marked in central cities. Importantly, we have revealed significant racial disparities in sensitivity to 2 other indoor allergens, dust mite and *A alternata*, that (to our knowledge) have not been reported elsewhere. Because sensitivity to each of these allergens has been linked to increased morbidity from asthma,³⁻⁹ and *A alternata* in particular has been associated with increased risk of death from asthma,⁶ these findings are particularly noteworthy.

These findings are consistent with other studies showing that age, sex, and family history are associated with allergen sensitivity.^{21,22} These data also reinforce the importance of regional climate in predicting dust mite sensitivity or exposure¹⁹ and concur with observations of a lower prevalence of cat sensitivity or cat allergen exposure among low-socioeconomic-status populations.^{8,15}

The consistency of our observations of significant independent associations between nonwhite race and sensitivity to 3 of 4 indoor allergens raises important questions as to their origins. It seems unlikely that the strong racial differences in allergen sensitivity we observed are solely accounted for by genetic differences, particularly because our models for sensitivity adjust for family history of allergy. Other intrinsic factors, such as birth order or family size, have also been linked to sensitivity.²⁹ However, we think it unlikely that the racial patterns we describe here are due to these factors because a term for family size added to these models yielded results similar to those presented. Likewise, it does not appear that poverty can explain these racial differences because we modeled poverty in several ways, and our findings were unchanged. Consequently, on the basis of other studies, we think it more likely that race is associated

with factors in the housing and community environment,^{8,15,30} which in turn predispose to indoor allergen exposure, mediate sensitivity to indoor allergens, or both.

National evidence of significant racial gradients in housing environmental characteristics is found in recent data from the US Census Bureau/American Housing Survey (AHS),³¹ suggesting a basis for the racial disparities in allergen sensitivity observed here. Compared with white respondents, African American respondents to the AHS were significantly more likely to report housing characteristics that are known to promote proliferation of indoor allergens, such as interior water leaks, evidence of rodents, and residence in multiple dwellings.³¹ Home dampness or high relative indoor humidity has been associated with increased dust mite allergen levels and sensitivity,³² as well as with higher levels of indoor mold and fungi³³ and sensitivity to *A alternata*.³⁴ In addition, factors promoting mouse infestation also promote cockroach infestation.³⁵ Finally, residence in a multiple dwelling is a strong independent predictor of cockroach allergen exposure in the Northeast.¹⁶

In contrast, dust mite allergen levels are higher in single-family homes,^{16,36} suggesting that there are other important factors influencing sensitivity to dust mite among these minority populations nationally. For example, a growing body of literature shows combustion byproducts, particularly diesel exhaust particles, act as adjuvants enhancing IgE production in response to inhaled allergens.^{37,38} There is some evidence of racial disparities in exposure to diesel exhaust particles and other ambient air pollutants.³⁰ If such disparities exist nationally, they could play an important role in establishing the pattern of racial differences in sensitivity documented here.

Our analyses suggest that racial disparities in cockroach sensitivity are greatest in central cities, which may help explain why the social gradient in asthma morbidity is especially pronounced in such areas.^{13,39} However, somewhat surprisingly, our data also suggest that central-city white children are less likely to be sensitive to cockroach than white children elsewhere. We are not aware of data to corroborate this finding, and this result needs replication in other studies.

Although among African Americans there is considerable correspondence between our data for sensitivity and national patterns of asthma morbidity, our data are not entirely consistent with apparently lower rates of asthma morbidity among Mexican Americans noted by some,^{40,41} but not all,^{42,43} researchers. The relationship of allergic sensitization to asthma development is complex.⁴⁴ Clearly, allergic sensitization alone is not sufficient for asthma development.⁴⁵ These inconsistencies suggest the presence of factors protective for asthma (eg, larger airway size) in the Mexican American population.^{46,47}

Limitations to the analyses we present here are primarily related to the cross-sectional nature of NHANES III. Because allergen sensitivity is a consequence of past cumulative exposure, some social or demographic factors may have changed between the time a child was sensitized and tested. In addition, like estimates of allergen

sensitivity from NHANES II,²¹ the NHANES III probably represents an underestimate of the actual prevalence of allergic sensitization among US children because medical exclusions targeted children with preexisting breathing or allergic problems. This would have a small effect, at worst, on overall prevalence estimates. Because the final sample weights take into account the marginal differences in response to the examination component by sex, age, race, and ethnicity,²³ nonparticipation was unlikely to have affected the overall pattern of findings observed here. In addition, although we have shown that racial differences in allergen sensitivity parallel the large social gradients in national asthma morbidity, undoubtedly several other factors also contribute to this gradient.⁴⁸

NHANES III data show that indoor allergen sensitivity is common among US children but reveal significant racial differences in sensitivity among children nationally. These differences closely parallel racial disparities in asthma morbidity. In conjunction with evidence showing housing and community environmental conditions among minorities may be substantially worse than those among whites, these findings suggest that racial disparities in housing and community environmental quality play a role in determining national patterns of asthma morbidity.

Even if the relative contribution or interaction of biological factors and environmental factors to the development of sensitivity is not wholly understood, our data support the development and adoption of national public health and housing policies designed to reduce indoor allergen exposure among all children. These policies may be especially important for those in minority communities, where the burden of indoor allergen sensitivity is the greatest.

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