

Letter to the Editor

Sensitization to mouse allergen and asthma and asthma morbidity among women in Boston

To the Editor:

Recent studies have shown that mouse allergen is prevalent and potentially important in both urban and suburban environments, particularly in homes of subjects with asthma sensitized to this allergen.^{1,4} However, little is known about the relation of sensitization to mouse allergen to asthma morbidity in adults with asthma outside the occupational laboratory setting. In this study, we evaluated whether sensitization to mouse allergen is a cause of morbidity in women with asthma from a range of socioeconomic backgrounds from a large metropolitan area in the United States.

This study involved women who were screened for participation in the Epidemiology of Home Allergens and Asthma Study, which has previously been described in detail⁵⁻⁷ (see this article's [Online Repository](#) at [www.jacionline.org](#)). This analysis includes 853 women for whom serum IgE to mouse allergen was measured. The study was approved by the Human Research Committee of the Brigham and Women's Hospital. Information on race was determined from the woman's response to questions asking her in which ethnic or racial groups she would classify herself (white, black, Hispanic, Asian, or other) as previously described⁵ (see this article's [Online Repository](#) at [www.jacionline.org](#)). Other demographic and socioeconomic variables were obtained from screening questionnaires and have been previously described.⁷

A serum sample drawn at screening was analyzed for total IgE and IgE specific to a panel of allergens using the UNICAP system (Pharmacia, Uppsala, Sweden).⁷ Furthermore, sera were assayed for IgE to mouse urinary allergen (Pharmacia ImmunoCAP e88) and to recombinant Mus m 1 (*Mus musculus*) using the streptavidin technique.⁸ Results for recombinant Mus m 1 showed a strong quantitative correlation with IgE antibody to mouse allergen ($r = 0.92$; $P < .001$). We defined sensitization to each allergen as CAP class level 1 or above (≥ 0.35 IU/mL).

Each woman was asked detailed questions regarding her current and past history of respiratory disease, as previously described.⁹ In the screening questionnaire, women were asked, "Has a doctor ever said that you have asthma?" These women were asked questions regarding asthma morbidity in the past 12 months. A bout of asthma lasting 1 week or more was defined as "prolonged illness," treatment in a hospital emergency room (ER) for asthma was defined as "use of a hospital ER," use of an oral steroid for asthma exacerbation was defined as "use of steroid," and having wheezing or whistling in the absence of a cold (upper respiratory viral infection) was defined as "wheeze without a cold." A positive response to any of the above defined asthma morbidity indicators ("prolonged illness," "use of hospital ER," "use of steroid," and "wheeze without a cold") was defined as "overall asthma morbidity."

The univariable analysis was conducted using χ^2 for categorical variables and 2-tailed t tests for pairs of continuous and categorical variables. Multivariable models were created by using stepwise logistic regression while adjusting for potential confounders and examining interactions. For the interaction between mouse allergen sensitization and race, we dichotomized the data into those who were white and non-Hispanic versus black or Hispanic. The interaction between race and asthma and asthma morbidity was examined by using adjusted logistic regression models to compute odds ratios (ORs; see this article's [Online Repository](#) at [www.jacionline.org](#) for details on statistical methods).

Of the 853 women in this analysis, 60 (7.0%) were sensitized to mouse allergen, 134 (16.3%) were sensitized to cockroach allergen, and 325 (38.1%) had been diagnosed with asthma by a physician. Of the 325 women with asthma, 49 (15.1%) were sensitized to mouse allergen. Characteristics of the population are in [Table E1](#) in this article's [Online Repository](#) at [www.jacionline.org](#). Most of the women were white and non-Hispanic (519 or 60.8%). Sensitization to mouse allergen was more common among nonwhites (11.9% for Blacks, 14.4% for Hispanics, 14.2% for Asians/others, and 3.3% for white non-Hispanics), among subjects living within the city of Boston, in areas of poverty, and in areas where the population had lower household incomes.

In univariable analyses, sensitization to mouse allergen was associated with 8-fold increased odds of doctor-diagnosed asthma (OR, 8.3; 95% CI, 4.3-16.3; $P < .0001$). After adjustment for potential confounders, including sensitization to cockroach and other allergens, race, poverty, health insurance status, and living within Boston, sensitization to mouse allergen was associated with more than twice the odds of physician-diagnosed asthma (95% CI for OR, 1.2-5.7; $P = .02$). In addition, we created multivariable models for each of the measured indicators of socioeconomic status and did not find them to be significant confounders of the effect of mouse allergen sensitization on asthma.

[Table I](#) shows the results of unadjusted and adjusted analyses of the relation between mouse allergen sensitization and indicators of asthma morbidity. In these analyses, mouse allergen sensitization was strongly associated with use of steroids, use of hospital ER, wheeze without a cold, prolonged illness, and overall asthma morbidity. Similar results were seen in the association between mouse allergen sensitization and asthma morbidity after adjustment for sensitization to all of the allergens other than mouse (see this article's [Table E2 and E3](#) in the [Online Repository](#) at [www.jacionline.org](#)).

Although mouse allergen sensitization was a risk factor for asthma prevalence and morbidity regardless of race or ethnicity, the magnitude of the relation of mouse sensitization with asthma morbidity risk was modified by race. The interaction between race and overall asthma morbidity was significant in the unadjusted and adjusted models

TABLE I. Mouse sensitization as a predictor of disease morbidity* among women with asthma (N = 325)^{†‡}

Mouse allergy	Steroid use (n = 37) [†]		ER visit (n = 53) [†]		Wheeze without a cold (n = 148) [†]		Prolonged illness (n = 50) [†]		Overall morbidity (n = 175) [†]	
	OR (95% CI)	OR (95% CI)	OR (95% CI)	OR (95% CI)	OR (95% CI)	OR (95% CI)	OR (95% CI)	OR (95% CI)	OR (95% CI)	OR (95% CI)
	Unadjusted	Adjusted	Unadjusted	Adjusted	Unadjusted	Adjusted	Unadjusted	Adjusted	Unadjusted	Adjusted
Negative	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0
Positive	4.4 (2.1-9.4)	3.2 (1.2-8.4)	3.7 (1.9-7.3)	3.3 (1.4-7.8)	2.6 (1.4-4.9)	2.5 (1.2-5.3)	3.0 (1.5-6.1)	3.7 (1.5-8.9)	3.1 (1.5-6.2)	3.0 (1.3-6.6)

*Definitions of asthma morbidity: steroid use, use of oral steroids during worse bout of illness; ER visit, treatment in a hospital ER for asthma in the past 12 months; wheeze without a cold, wheezing or whistling in the chest in the absence of a cold (upper respiratory viral infection) in the past 12 months; prolonged illness, a bout of asthma during the past 12 months lasting 1 week or more; overall morbidity, a positive response to any of these indicators.

[†]n = Number of women who had these symptoms of asthma morbidity in the previous year of the 325 women who were diagnosed with asthma. Total population was 853 women.

[‡]All multivariate models were adjusted for age, race, area poverty, living in inner-city Boston, cockroach allergen sensitization, cat allergen sensitization, and health insurance.

(for details, see this article's [Online Repository](#) at www.jacionline.org; unadjusted OR for asthma morbidity in non-Hispanic whites, 16.3; 95% CI, 2.1-127.8; unadjusted OR for blacks or Hispanics, 1.8; 95% CI, 0.8-3.9; adjusted OR for non-Hispanic whites, 16.4; 95% CI, 2.0-132.3; adjusted OR for blacks or Hispanics, 1.6; 95% CI, 0.6-3.9; *P* for interaction, .04.

Among 853 women in Boston, sensitization to mouse allergen was associated with physician-diagnosed asthma and several markers of asthma morbidity and overall asthma morbidity, regardless of race/ethnicity. This finding remained significant after adjusting for indicators of socioeconomic status at the personal and community level and for sensitization to other allergens. We therefore report the unique contribution of mouse allergy to asthma prevalence and morbidity, adjusting for other allergies. Mouse allergy was more prevalent in women from economically disadvantaged areas and ethnic minority groups. Mouse allergy was also associated with asthma prevalence and asthma morbidity, regardless of race/ethnicity. However, white women who were sensitized to mouse allergen had a higher rate of asthma morbidity compared with women from minority ethnic groups who were sensitized to mouse allergen, suggesting that the relation of mouse sensitization with asthma morbidity risk was modified by race.

To our knowledge, this is the first report of a significant association between mouse sensitization and asthma and asthma morbidity in adults outside the laboratory setting. In a previous study,⁷ we showed that black and Hispanic women had an increased risk of having an elevated total serum IgE and of being sensitized to multiple allergens (other than mouse, which was not included). In that study, which evaluated indoor and outdoor allergens, sensitization to cockroach, cat, dog, and mite allergens was higher in women of low socioeconomic status and was independently associated with asthma.^{9,10}

Our finding of the interaction between mouse sensitization and asthma by race/ethnicity was independent of measured indices of poverty (which are correlated with race/ethnicity). Black and Hispanic women are sensitized to more allergens than white women in our cohort. Because sensitization to multiple allergens is a risk factor

for asthma morbidity, the unique contribution of mouse allergen sensitization to the risk of morbidity among minority women may consequently be smaller for minority women than for white women.

This study is limited in that it may be generalizable only to women of childbearing age from the urban United States. It is also limited by the absence of mouse allergen data and by the absence of additional information to confirm the diagnosis of asthma. However, this study is relevant to a large, at-risk portion of the population of women of childbearing age.

In summary, regardless of race, ethnicity, or socioeconomic status, women sensitized to mouse allergen have higher rates of a physician's diagnosis of asthma and asthma morbidity. Although the effect of mouse allergen sensitization on asthma and asthma morbidity was somewhat lower in minorities, mouse sensitization was present in a higher proportion of the minority population. Thus, the population at risk for mouse allergy-associated asthma may be greater among the African American and Hispanic compared with the non-Hispanic white women of childbearing age. Exposure patterns and other factors associated with the sensitization to mouse allergen should be examined more closely in the future.

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METHODS

Enrollment of the cohort

Subjects were women who delivered a child at a large tertiary hospital in Boston, Mass (Brigham and Women's Hospital). Between September 1994 and June 1996, women were approached for enrollment in a birth cohort study, the Epidemiology of Home Allergens and Asthma Study, designed to examine the effects of early life allergen exposure on the development of childhood asthma. On weekdays (Monday through Friday), all mothers who delivered their babies at the Brigham and Women's Hospital were approached for screening 24 to 48 hours after delivery if they lived within Route 128 (which roughly encircles the greater metropolitan area), were ≥ 18 years old, and were able to speak English or Spanish. Women were not screened if they were identified by the floor nurse as unlikely to be comfortable with an interview, or if the index child was premature (< 36 weeks), had a major congenital abnormality, or was in the neonatal intensive care unit. Mothers were asked the following simple screening questions: "Have you ever had a doctor's diagnosis of asthma, hay fever, or allergies? Has the biological father of your child ever had a doctor's diagnosis of asthma, hay fever, or allergies?" If the mothers answered yes to one or both of these questions, they were asked to complete a more detailed screening questionnaire.

Over the enrollment period, we identified 7657 women who lived within the Route 128 area and were 18 years of age or older; 5973 were approached and asked about their history of asthma or allergy and the index child's biological father's history of asthma or allergy. Most of the women (5904) agreed to answer the simple screening questions. Of these, 1539 had a personal history or a partner's history of asthma or allergy, and 1405 women agreed to complete a more detailed screening questionnaire. A total of 906 of the 1405 were excluded from the study. The reasons for exclusion included plans to move within the next year (39% of those screened), reluctance to participate in a longitudinal study (45%), loss to follow-up (14%), and other (2%). This left 499 women and their families who were enrolled in the longitudinal cohort.

Subjects for the current analysis

Subjects for the current analysis are the 415 (83.1%) mothers enrolled in the longitudinal study who had blood available for mouse IgE measurements. These subjects were the basis of previous reports.^{E1-E3} An additional 438 women who completed the detailed screening questionnaire and had blood available for mouse IgE measurements but were not enrolled in the study are included in the current analysis, leaving a total of 853 women. The study was approved by the Human Research Committee of the Brigham and Women's Hospital.

In the screening questionnaire, women were asked, "Has a doctor ever said that you have (1) asthma (2) hay fever, or (3) eczema?" Positive responses were defined as physician-diagnosed asthma, physician-diagnosed hay fever, and physician-diagnosed eczema. Information on race was determined from the woman's response to questions asking her in which ethnic or racial groups she would classify herself, as previously described.^{E1} She was classified as black if she classified herself as black irrespective of other groups identified. She was classified as Hispanic if she classified herself as Hispanic but not black. She was classified as Asian if she classified herself as Asian but neither Black nor Hispanic. She was classified as other if she classified herself as not white, black, Hispanic, or Asian. The remaining individuals were classified as white. Other variables obtained from the screening questionnaires included age (categorized as 18 to < 30 , 30 to < 33 , 33 to < 36 , and 36-46 years of age), current and former smoking (categorized as yes or no), and insurance status (defined as private/self-pay vs Medicaid). Subjects' addresses were sent to a commercial firm (Mapping Analytics, Rochester,

NY) to be geocoded, whereby the address was used to assign block group number on the basis of the geographic areas defined in the 1990 US Census of Population and Housing.^{E4} Three census-based geographic area socioeconomic status variables were derived from block group information and other variables from the 1990 Census as previously reported.^{E3,E5} A marker of poverty in the women's area of residence was established in terms of the proportion of the population in that block group living below the poverty level (composite variable based on family income and family size, according to 1990 US Census data) and categorized for analysis into 3 categories: $< 5\%$, 5% to 20% , and $> 20\%$ of the population in the block group living below the poverty level. Area educational level was categorized into $> 60\%$, 25% to 60% , and $< 25\%$ of the population in the block group having completed at least a college education. Area household income was categorized into $> 50\%$, 25% to 50% , and $< 25\%$ of the population in the block group with a household income of $> \$50,000$.

A serum sample drawn at screening was analyzed for total IgE and IgE specific to a panel of allergens, as measured with the UNICAP system (Pharmacia, Uppsala, Sweden). Specific IgE antibodies were assayed for dust mite, dog, cat, cockroach, *Alternaria* species, *Aspergillus* species, rye grass, and ragweed, with each allergen attached to a cellulose sponge (ImmunoCAP), as previously described.^{E3,E6,E7} Furthermore, sera were assayed for IgE to mouse urinary allergen (Pharmacia ImmunoCAP e88) and to recombinant Mus m 1 (*Mus musculus*) using the streptavidin technique. Results for recombinant Mus m 1 showed a strong quantitative correlation with IgE antibody to mouse allergen ($r = 0.92$; $P < .001$). In the CAP system, CAP classes correspond to the following units: 0, 0 to < 0.35 IU/mL; 1, 0.35 to < 0.7 IU/mL; 2, 0.7 to < 3.5 IU/mL; 3, 3.5 to < 17.5 IU/mL; 4, 17.4 to < 50 IU/mL; 5, 50 to < 100 IU/mL; and 6, ≥ 100 IU/mL. We defined sensitization to each allergen as CAP class level 1 or above (≥ 0.35 IU/mL).

Main outcome measures

Each woman was asked detailed questions regarding her current and past history of respiratory disease, as has been previously described.^{E5-E7} Women with physician-diagnosed asthma were defined as those with asthma. These women were asked questions regarding current and past morbidity. A bout of asthma during the past 12 months lasting 1 week or more was defined as "prolonged illness," treatment in a hospital emergency room (ER) for asthma in the past 12 months was defined as "use of a hospital ER," and use of an oral steroid for asthma exacerbation was defined as "use of steroid." Women whose chest had sounded wheezy or whistling in the absence of a cold (upper respiratory viral infection) in the past 12 months were defined as having "wheeze without a cold." A positive response to any one of these indicators was defined as "overall asthma morbidity."

Statistical analysis

The univariable analysis was conducted by using χ^2 for categorical variables and 2-tailed t tests for pairs of continuous and categorical variables. Multivariable models were created using stepwise logistic regression to study the relation between sensitization to mouse allergen and asthma and asthma morbidity variables while adjusting for potential confounders and examining interactions. Variables were kept in the final models if they were significantly associated with the outcome ($P < .05$), if they were clinically relevant (such as measures of socioeconomic status), or if they satisfied a change in estimate criteria ($\geq 10\%$ change in the β coefficient variable for mouse allergen sensitization).

For the interaction between mouse allergen sensitization and race, we dichotomized the data into those who were white and non-Hispanic versus black or Hispanic. Because there were very few Asian/others in the entire cohort ($n = 18$), we removed Asians/others since it did not change the results of our analysis. For physician's

diagnosis of asthma ($n = 835$) and asthma morbidity among women with asthma ($n = 316$) were examined this interaction using adjusted logistic regression models were to compute the ORs. All covariates in the adjusted logistic regression models were centered (if race = 0 for white and non-Hispanic and race = 1 for black or Hispanic, then centered race = $-1/2$ for white and centered race = $1/2$ for black or Hispanic) so that the ORs of interest were at an averaged value of the other covariates in the model.

RESULTS

Although mouse allergen sensitization was a risk factor for asthma prevalence and morbidity regardless of race or ethnicity, the magnitude of the relation of mouse sensitization with asthma morbidity risk was modified by race. However, the evidence for effect modification was weaker after adjusting for the measured potential confounders of the relation of mouse sensitization to asthma. Stratifying by race, the unadjusted odds of asthma for those with mouse sensitization versus those without sensitization was greater for the small group of white non-Hispanic women than for the larger group of black or Hispanic women (unadjusted OR for asthma in white non-Hispanics, 21.5; 95% CI, 4.8-95.0; unadjusted OR, blacks or Hispanics, 4.9; 95% CI, 2.1-11.4). Adjusting for socioeconomic factors with an interaction term for race, the data more weakly suggested that race/ethnicity modified the relation of mouse sensitization to asthma (adjusted OR, white non-Hispanics, 14.8; 95% CI, 1.8-121.8; adjusted OR, blacks or Hispanics, 1.8; 95% CI, 0.7-4.8; P for effect modification, .07). The evidence for effect modification by race/ethnicity in adjusted models was somewhat stronger for

the outcome of asthma morbidity (unadjusted OR for asthma morbidity in white non-Hispanics, 16.3; 95% CI, 2.1-127.8; unadjusted OR, blacks or Hispanics, 1.8; 95% CI, 0.8-3.9; adjusted OR, white non-Hispanics, 16.4; 95% CI, 2.0-132.3; adjusted OR, blacks or Hispanics, 1.6; 95% CI, 0.6-3.9; P for effect modification, .04). This modification by race/ethnicity on the relation of mouse sensitization to asthma morbidity risk was also found for components of overall asthma morbidity (eg, for wheeze without a cold and prolonged illness in women with asthma).

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TABLE E1. Characteristics of study population (percentages are percent of total in each column)*

Characteristic	Total sample (n = 853)		Women without asthma (n = 528)		Women with asthma (n = 325)	
	N	Percentage	N	Percentage	N	Percentage
Mouse IgE						
Negative	793	93.0	517	97.9	276	84.9
Positive	60	7.03	11	2.1	49	15.1
Cockroach IgE						
Negative	690	83.7	471	91.2	219	71.1
Positive	134	16.3	45	8.7	89	28.9
Cat IgE						
Negative	548	66.5	399	77.3	149	43.4
Positive	276	33.5	117	22.7	159	51.6
Age group						
18 to <30 y	318	37.3	147	27.8	171	52.6
30 to <33 y	193	22.6	127	24.1	66	20.3
33 to <36 y	177	20.8	136	25.8	41	12.6
36 to <46 y	165	19.3	118	22.3	47	14.5
Race						
White	519	60.8	282	53.4	145	44.6
Black	177	20.8	85	16.1	92	28.3
Hispanic	139	16.3	60	11.3	79	24.3
Asian/other	18	2.1	9	1.7	9	2.8
Poverty, percentage of population living below poverty line						
<5%	301	36.6	204	38.6	97	29.8
5% to 20%	361	42.3	222	42.0	139	42.8
>20%	161	18.9	72	13.6	89	27.4
Residence within city of Boston						
No	484	56.7	335	63.4	149	45.8
Yes	369	43.3	193	36.6	176	54.2
Health insurance						
Private/self-pay	727	86.7	477	91.6	250	79.8
Medicaid	112	13.4	44	8.4	68	20.2
Household income (percentage of population with income $\geq$ \$50,000)						
>50%	261	30.6	177	33.5	84	25.8
25% to 50%	331	38.8	225	42.6	106	32.6
<25%	261	30.6	126	23.9	135	41.5

*In the population of 853 women, there are 29 missing values for cat and cockroach allergen-specific IgE and 14 missing values for health insurance. In the 325 women with asthma, there are 17 missing values for cat and cockroach allergen-specific IgE and 7 missing values for health insurance.

TABLE E2. Multivariable model of predictors of physician's diagnosis of asthma*

Variable	Multivariable model	
	OR (95% CI)	P value
Mouse IgE		
Negative	1.00	
Positive	2.6 (1.2-5.7)	.02
Cockroach IgE		
Negative	1.00	
Positive	1.9 (1.2-3.2)	.01
Cat IgE		
Negative	1.00	
Positive	2.4 (1.6-3.7)	<.0001
Dog IgE		
Negative	1.00	
Positive	2.1 (1.2-3.8)	.01
Dust mite IgE		
Negative	1.00	
Positive	1.3 (0.9-1.9)	.15
<i>Alternaria</i> IgE		
Negative	1.00	
Positive	1.7 (1.0-2.9)	.07
<i>Aspergillus</i> IgE		
Negative	1.00	
Positive	0.7 (0.3-1.4)	.3
Rye grass IgE		
Negative	1.00	
Positive	1.2 (0.8-1.8)	.5
Ragweed IgE		
Negative	1.00	
Positive	0.6 (0.4-1.0)	.05

*Model also adjusts for age, race, poverty, living within Boston city limits, and health insurance.

TABLE E3. Multivariable model* of predictors of overall asthma morbidity†

Variable	Multivariable model	
	OR (95% CI)	P value
Mouse IgE		
Negative	1.00	
Positive	3.4 (1.7-6.8)	.0005
Cockroach IgE		
Negative	1.00	
Positive	1.7 (1.0-2.8)	.04
Cat IgE		
Negative	1.00	
Positive	1.7 (1.1-2.8)	.02
Dog IgE		
Negative	1.00	
Positive	1.9 (1.1-3.4)	.03
Dust mite IgE		
Negative	1.00	
Positive	0.9 (0.6-1.3)	.5
<i>Alternaria</i> IgE		
Negative	1.00	
Positive	1.2 (0.7-2.2)	.5
<i>Aspergillus</i> IgE		
Negative	1.00	
Positive	0.6 (0.3-1.4)	.3
Rye grass IgE		
Negative	1.00	
Positive	1.1 (0.7-1.7)	.7
Ragweed IgE		
Negative	1.00	
Positive	1.0 (0.6-1.6)	.9

*Model also adjusts for age, race, poverty, living within Boston city limits, and health insurance.

†Overall morbidity, a positive response to any of the following asthma morbidity measures: steroid, use of oral or inhaled steroid during worse bout of illness; ER visit, treatment in a hospital ER for asthma in the past 12 months; wheeze no cold, women whose chest had ever sounded wheezy or whistling in the absence of a cold (upper respiratory viral infection) in the past 12 months; prolonged illness, a bout of asthma during the past 12 months of 1 week or more duration.