
Effect of mouse allergen and rodent environmental intervention on asthma in inner-city children

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Background: Mouse allergens are prevalent in inner-city households, and increasing levels of exposure are associated with sensitization in children with asthma.

Objectives: To examine mouse allergen sensitization and exposure in inner-city children, mouse allergen as an independent risk factor for asthma morbidity, and the efficacy of a rodent environmental intervention.

Methods: We conducted a subanalysis of children with asthma aged 5 to 11 years enrolled in the Inner-City Asthma Study. After randomization, 150 participants received a home rodent-specific environmental intervention. Asthma morbidity measures were obtained bimonthly. Bedroom dust was collected and analyzed for *Mus m 1* at baseline and every 6 months for 2 years.

Results: Twenty-two percent of children tested positive to mouse. Most bedrooms (80%) had detectable mouse allergen. Sensitization occurred at low levels of exposure. Sensitization and exposure were associated with increased asthma morbidity, including hospitalizations. Mouse allergen levels on the bedroom floor decreased 27.3% (95% confidence interval, −46.1% to −1.9%) in intervention homes. Mouse allergen reduction was associated with less missed school, sleep disruption, and caretaker burden but not symptoms or medical utilization.

Conclusions: Mouse allergen is prevalent in inner-city homes. Sensitization seems to occur at low levels of exposure. Mouse allergen is an independent risk factor for asthma morbidity. The described environmental intervention reduced mouse allergen levels and asthma-related sleep and activity disturbance.

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INTRODUCTION

Asthma is disproportionately prevalent in low-income urban neighborhoods, and morbidity is exceptionally high in children living in these areas.¹ Potential explanations include exposure to high levels of indoor allergens, such as cockroach and mouse allergens. The National Cooperative Inner-City Asthma Study (NCICAS) demonstrated that mouse allergens are prevalent in inner-city households² and that increasing levels of exposure are associated with sensitization.³ Other studies have also shown that mouse allergen is widely distributed in inner-city homes in Europe⁴ and in urban and nonurban homes across the United States.^{5–7} Mouse sensi-

zation has been associated with greater than 1.6 $\mu\text{g/g}$ of mouse allergen in the kitchen² and 2.2 ng/g (0.0022 $\mu\text{g/g}$) in the bedroom.⁶ The NCICAS did not find a relationship with asthma morbidity.³ However, a recent study⁷ of inner-city preschoolers found that those sensitized and exposed to 0.5 $\mu\text{g/g}$ of *Mus m 1* or greater had more asthma morbidity than other children.

An environmental intervention was previously conducted as part of the Inner-City Asthma Study (ICAS).⁸ This tailored, multifaceted, home-based intervention successfully decreased indoor cockroach and dust mite allergen concentrations, and there was a significant relationship between the decline in allergen levels and the reduction in asthma symptoms. The effect on morbidity observed during the intervention was sustained throughout 1-year follow-up. Until recently, no studies have focused on mouse allergen interventions. A small, controlled trial⁹ of environmental intervention reduced mouse allergen levels but did not affect asthma symptoms.

This article presents the ICAS findings regarding mouse allergen. Exposure to other rodents was not measured in this study. The distribution of mouse allergen across cities is described. Mouse allergen is examined as an independent risk factor for asthma morbidity by studying the relationships among mouse sensitization, exposure, and asthma morbidity. We also present the efficacy of a rodent environmental intervention and its effect on asthma morbidity.

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METHODS

The ICAS included 937 asthmatic children aged 5 to 11 years and their caretakers from 7 US inner-city areas. All caregivers provided written informed consent. Eligibility required at least 1 hospitalization or 2 emergency department (ED) visits for asthma during the 6 months before screening and a positive skin test reaction (wheal size ≥ 2 mm greater than saline control) to at least 1 common indoor allergen. Children had to sleep in the intervention home at least 5 nights per week. Specific recruitment methods and baseline clinical information have been previously reported.¹⁰ The study was conducted between August 1, 1998, and August 31, 2001, and was approved by the institutional review board of each participating institution. The sample size for the entire study was chosen on the basis of being able to detect a 0.6-day difference in maximum symptom days with 90% power.

Groups were randomly preassigned to study identification numbers using a random number generator with a uniform distribution and blocks of size 8 and 12 within site. Group assignments were supplied to sites in opaque envelopes labeled with sequential study identification numbers, which were opened by site interviewers on determination of the child's eligibility. Neither study staff nor participants were blinded to group assignment.

The environmental intervention was tailored in that specific modules for cockroach, dust mite, mold, and rodent were administered based on participant skin test results (using a 2-mm wheal). Details of the environmental intervention modules are described elsewhere.¹⁰ The rodent module consisted of (1) providing a high-efficiency particulate air (HEPA) vacuum cleaner and placing a HEPA air filter in the child's bedroom, (2) filling rodent access points and setting traps throughout the home, and (3) educating the family about kitchen cleaning and proper food storage. Of 157 families with a child with mouse or rat skin test positive results who qualified for the rodent module, 150 received it. Of these, 97% also received at least 1 other module.

Trained interviewers administered a baseline clinical interview to the primary caretaker that included demographics, asthma morbidity, home characteristics, and the child's exposure to environmental tobacco smoke. Morbidity was measured at baseline and at 2-month intervals for 24 months. Ninety-two percent of these follow-up morbidity assessments were completed. The primary outcome, maximum symptom days per 2 weeks, is the largest value among (1) number of days in the past 2 weeks that the child experienced wheezing, chest tightness, or cough; (2) number of nights that the child awoke because of asthma; and (3) number of days that the child had to slow down or discontinue play activities because of asthma. In addition, the caretaker reported the number of school days missed due to asthma, caretaker days of lost sleep, and having to change plans due to the child's asthma in the past 2 weeks and the number of hospitalizations, ED visits, and scheduled and unscheduled clinic visits for asthma

in the past 2 months. Total unscheduled visits were calculated as the sum of unscheduled clinic visits and ED visits.

During the baseline clinical evaluation, children underwent skin testing (Multi-Test II; Lincoln Diagnostics, Decatur, Illinois) to histamine and saline controls and 11 allergens¹¹: *Dermatophagoides farinae*, *Dermatophagoides pteronyssinus*, cockroach (German and American mix), rat, mouse, *Alternaria*, *Cladosporium*, *Aspergillus*, *Penicillium*, cat (standardized 10,000 BAU/mL), and dog (mixed breeds). Cockroach extract was from Bayer Corp (Spokane, Washington). All other extracts were from Greer Laboratories (Lenoir, North Carolina).

One to 3 weeks after the baseline clinical evaluation, and every 6 months thereafter for 2 years, research assistants performed home evaluations involving visual inspection and questions.¹⁰ Eighty-eight percent of all home visits were completed. Data on the entire home, including the child's sleeping area, family/television room, kitchen, and bathroom, were collected. Using a standardized protocol and equipment, research assistants collected separate vacuumed dust samples from the child's bedroom floor and bed, which were placed in separate sealed bags and shipped to the Dermatology, Allergy, and Clinical Immunology Reference Laboratory at The Johns Hopkins University, Baltimore, Maryland, or the laboratory at Harvard School of Public Health, Boston, Massachusetts, for evaluation of Der p 1, Der f 1, Bla g 1, Fel d 1, Can f 1, and Mus m 1 levels using enzyme-linked immunosorbent assay. Rat allergen (Rat n 1) was not assayed in these dust samples. Data were collected on paper forms and subsequently entered by local site staff into a Web-based data management system. Data entry screens resembled the paper forms and provided immediate error checking and validations.

Mus m 1 was measured in house dust using an enzyme-linked immunosorbent assay method described by Phipatanakul et al.^{2,3} House dust specimens were sieved (50 mesh) and extracted at 1:20 wt/vol. Mus m 1 was captured with purified mouse anti-Mus m 1. The detection antibody was a purified and biotinylated rabbit polyclonal anti-Mus m 1 (Indoor Biotechnologies, Charlottesville, Virginia). The assay was standardized with Mus m 1 in mouse urine substandardized to MUP E428 reference preparation. The working range of the assay was 0.5 to 11.25 ng/mL. The detection limit after correction for extraction was 10 ng/g of dust. The assay separates bound and free components with a buffer wash after incubation of the extract. The wells are coated with capture antibody and are blocked with irrelevant protein (bovine serum albumin) to reduce nonspecific binding. The consequence is that Mus m 1-negative dust specimen extracts produce a response signal that is statistically not different from the assay buffer. Also, dusts from different environments are heterogeneous, limiting the ability to produce a single "reference" dust. Values below the limit of detection were assigned a value of half the limit of detection.

Although a 2-mm wheal (to any indoor allergen) was required for study entry and for rodent module eligibility

(when positive to mouse or rat), we analyzed risk of sensitivity and morbidity based on sensitivity using a 3-mm wheal. The risk of sensitivity to mouse according to exposure was calculated using contingency tables and a Mantel-Haenszel adjustment for city where indicated.

The effect of sensitivity and exposure on 2-year morbidity was examined using a longitudinal model with a generalized estimating equation adjustment for correlated measurements within individuals.¹² Bimonthly morbidity assessments were averaged to the closest semiannual allergen evaluation. Average symptoms were analyzed using a linear model, and a Poisson model was used to examine rates of hospitalizations and unscheduled visits.^{13,14} Children with allergen levels higher than the first quartile for *Mus m 1* were considered exposed. An *a priori* test was used to compare children sensitive and exposed with those exposed but not sensitive, sensitive but not exposed, and neither sensitive nor exposed.^{15,16} Morbidity outcomes were calculated for each group controlling for visit number, season of baseline visit, age, sex, race, inhaled anti-inflammatory medications, cockroach sensitivity, cockroach and dust mite allergen levels in the bed, household smoking, any relatives with asthma, and environmental intervention group. We did not control for city in this model because of substantial collinearity with mouse allergen levels.

We used a linear mixed model to evaluate whether mouse allergen level reductions in the intervention group ($n = 150$) were associated with decreased asthma morbidity.¹² Analyses on the effect of the intervention on mouse allergen levels used the change in allergen levels from baseline to the average of the 6- and 12-month evaluations to provide a more stable measure across time. The control group consisted of 155 children who were sensitive to mouse or rat and had follow-up home visits and asthma morbidity measures during the intervention year but who did not receive the environmental intervention. Analyses were generated using a statistical software program (SAS for Windows Version 9.1; SAS Institute Inc, Cary, North Carolina).

RESULTS

The population was primarily black and Hispanic, and most were boys (Table 1). Sixty percent of families had an annual household income less than \$15,000 (data not shown). Twenty-two percent of the children were skin test positive to mouse (≥ 3 -mm wheal), and 23% of caretakers reported problems with mice or rats (Table 1). Detailed characteristics of participants and their homes are available elsewhere.¹⁵

Of the 823 homes with sufficient dust for analysis, 74% had detectable mouse allergen in bedroom floor dust (median concentration, $0.04 \mu\text{g/g}$; range, 0.0 – $184.8 \mu\text{g/g}$). Most beds (80%) had detectable mouse allergen, and levels were similar to those from the floor (data not shown). Mouse allergen levels varied considerably by city (Fig 1). Mouse allergen was detected in nearly all homes in Boston, the Bronx, Chicago, and Manhattan, whereas it was much less common in Dallas, Seattle, and Tucson.

Exposure to mouse allergen levels higher than the first quartile level was strongly associated with skin test sensitivity (bed or bedroom floor risk ratio, 3.00; 95% confidence interval [CI], 1.92 to 4.70) (Table 2). This relationship holds, except in the case of bed allergen levels, when adjusting for city.

Morbidity results are given in Table 3. Children sensitive and exposed to mouse allergen are compared with those not sensitive but exposed, exposed but not sensitive, and neither sensitive nor exposed, controlling for covariates. The hospitalization rate was significantly higher for children sensitized and exposed than for the other groups (rate ratio, 1.65; 95% CI, 1.09 to 2.50). There was a significant difference between those sensitized and exposed vs the other groups for maximum symptom days, nights of lost sleep, and days caretaker had to change plans, even when controlling for cockroach sensitivity and exposure.

In the ICAS, 469 of 937 participants were randomized to receive the environmental intervention. Of these participants, 157 qualified for the rodent module based on rat or mouse skin test sensitivity, of which 150 (96%) completed the rodent environmental intervention and had at least 1 home visit and morbidity measure during the intervention year. Allergen levels in these homes are compared with those in 155 homes where the child was rat or mouse skin test positive but no intervention was conducted. Mouse allergen levels on the bedroom floor were reduced by 27.3% (95% CI, -46.1% to -1.9%) in intervention homes but increased by 28.0% (95% CI, -6.10% to 74.5% ; $P = .01$) in control homes. Bed allergen levels did not change for either group. For households that received other (nonrodent) environmental interventions, mouse allergen levels were not affected.

To directly test the hypothesis that the observed reductions in mouse allergen in mouse-sensitive children were related to a corresponding reduction in asthma morbidity, the estimated effects of a 50% reduction in allergen levels from baseline were calculated (Table 4). Twenty-nine percent of intervention families achieved 50% reduction on the floor, and 26% did so on the bed. After controlling for cockroach allergen exposure and sensitivity, we did not find a decrease in asthma symptoms or health care utilization to be associated with mouse allergen reduction during the intervention year. We did, however, observe significantly reduced school absenteeism, nights of child and caretaker waking, and days caretakers had to change plans.

DISCUSSION

This article provides important information about mouse allergy and environmental intervention in inner-city children with asthma. The overall prevalence of mouse allergen sensitization was 22% in this cohort, but the rates varied among cities (8.6% to 46.2%). In the NCICAS, 18% of children were sensitized to mouse.³ The reason for the higher rate in the ICAS was probably related to different recruitment criteria that yielded an atopic group of children. Other studies^{4,6,7,17} have reported mouse sensitization rates of 12% to 36%.

Table 1. Characteristics of Participants and Their Homes, Inner-City Asthma Study (ICAS)

Characteristics	Eligible for rodent intervention ^a		Total ICAS population (N = 937)
	Intervention group (n = 157) ^b	Control group (n = 155)	
Race/ethnicity, % ^c			
Black	40.1	50.0	39.7
Puerto Rican	19.1	14.3	15.3
Mexican American	9.6	6.5	13.8
Other Hispanic	10.8	15.6	11.1
White	4.5	4.6	7.0
Asian	1.3	0.6	1.2
American Indian	2.6	0	3.1
Mixed/other	12.1	8.4	8.8
Male sex, %	58.0	67.1	62.6
Age of child, mean, y	7.5	7.6	7.7
Type of dwelling, % ^c			
Detached house	25.5	25.2	29.1
Duplex/triplex	10.2	8.4	9.2
Row house	3.8	1.9	2.4
Low-rise apartment (1–3 floors)	24.2	32.3	29.8
High-rise apartment (>3 floors)	35.7	31.0	27.5
Mobile home/trailer	0.6	1.3	2.0
Evidence of cockroaches in the home, %	61.9	53.6	61.9
Evidence of mice/rats in the home, %	25.3	26.0	23.3
≥3-mm wheal to rat allergen, % ^c	38.8	41.9	13.4
≥3-mm wheal to cockroach allergen, % ^c	66.9	63.9	59.9
≥3-mm wheal to mouse allergen by research city, % ^c			
Boston	84.4	80.0	46.2
Bronx	89.3	88.5	35.8
Chicago	47.1	57.1	11.4
Dallas	68.8	67.7	20.0
New York	63.0	63.3	25.5
Seattle	31.8	17.6	7.9
Tucson	46.7	55.6	8.6
All	65.0	65.8	21.8

^a Having at least a 2-mm skin test wheal to mouse or rat allergen. There were no significant differences by intervention group.

^b Seven of these children did not actually receive their intervention.

^c $P < .05$ for the difference between children eligible for the rodent module vs all children.

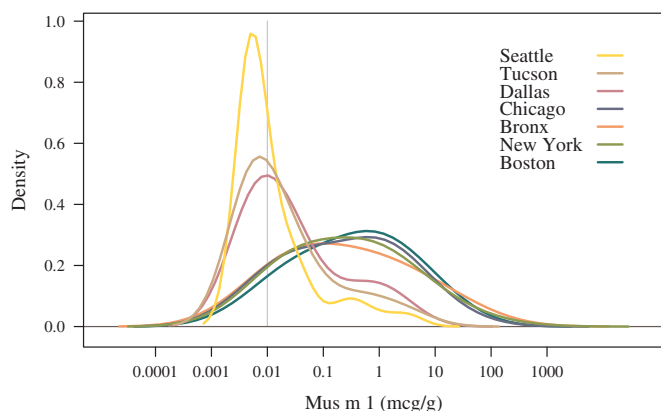


Figure 1. Kernel density estimate of the probability density function for Mus m 1 by city.

Mouse allergen was detected in 80% of ICAS bedrooms, similar to prevalence rates reported in other studies^{5–7} but somewhat lower than the 95% prevalence in the NCICAS² and the 100% prevalence in inner-city Baltimore.^{7,18} This may be related to regional differences. Although little variation was noted in a sample of 75 locations across the country,⁵ the ICAS found variability among cities, with lower prevalences in Seattle (37.4%), Tucson (52.2%), and Dallas (63.4%). Less variability was seen in the NCICAS, possibly due to the lack of southern and western cities in that study.

We found an overall median level of Mus m 1 in bedrooms one-tenth of that found in the NCICAS. This is not due to differences in assay methods because the same assay was used in both studies. It may be explained by geographic variation in the ICAS or by housing differences across cities. When the same cities are considered, minimal differences are seen between the NCICAS and the ICAS. The magnitude of

Table 2. Relationship of Mouse Allergen Exposure to Mouse Sensitization, Inner-City Asthma Study

Location	Positive skin test response (≥ 3 -mm wheal), %		Risk ratio (95% CI)	Risk ratio (95% CI) adjusted for city ^a
	Mus m 1 below quartile 1	Mus m 1 above quartile 1		
Bed	11	26	2.35 (1.64–3.37)	1.10 (0.78–1.54)
Bedroom floor	11	27	2.46 (1.75–3.48)	1.58 (1.08–2.28)
Either bed or floor	9	26	3.00 (1.92–4.70)	1.60 (0.99–2.57)

Abbreviation: CI, confidence interval.

^a Cochran-Mantel-Haenszel adjustment.Table 3. Relationship of Mouse Sensitivity and Exposure to Asthma Morbidity^a

	Skin test positive		Skin test negative		P value or rate ratio (95% CI)
	Exposed	Not exposed	Exposed	Not exposed	
Maximum symptom days in past 2 wk, No.	3.7	3.3	3.3	3.0	.02 ^b
Days of wheeze in past 2 wk, No.	3.0	2.9	2.7	2.6	.07 ^b
Nights child lost sleep in past 2 wk, No.	1.8	1.6	1.6	1.3	.04 ^b
Days child's activity reduced in past 2 wk, No.	2.4	2.1	2.2	1.9	.08 ^b
School days missed in past 2 wk, No.	0.7	0.6	0.7	0.6	.18 ^b
Nights caretaker lost sleep in past 2 wk, No.	1.9	1.6	1.7	1.3	.06 ^b
Days caretaker changed plans in past 2 wk, No.	1.1	0.7	0.8	0.7	.02 ^b
Rate of hospitalizations per 1,000 person-days	0.63	0.43	0.47	0.27	1.65 (1.09–2.50) ^c
Rate of unscheduled asthma visits per 1,000 person-days	5.1	4.5	5.6	4.5	1.06 (0.88–1.27) ^c

Abbreviation: CI, confidence interval.

^a Longitudinal generalized estimating equation model controlling for visit, season of baseline visit, age, race, sex, cockroach allergen levels on the bed, cockroach skin test sensitivity, dust mite allergen levels on the bed, any relatives with asthma, household smoking, inhaled anti-inflammatory drug use, and environmental intervention group.^b A priori test comparing children who are skin test positive to mouse allergen and exposed to levels of at least 0.02 $\mu\text{g/g}$ compared with the other 3 groups.^c Rate ratio comparing children who are skin test positive to mouse allergen and exposed to levels of at least 0.02 $\mu\text{g/g}$ compared with the other 3 groups.

variation in Mus m 1 levels is also similar in these 2 studies (ICAS: 0.0 to 184.8 $\mu\text{g/g}$ vs NCICAS: 0.0 to 294.4 $\mu\text{g/g}$). Other studies^{5,19} demonstrated higher median levels of mouse allergen, but their limit of detection was higher than that of the ICAS, presumably due to differences in assay methods.

We found an association between mouse exposure and sensitization. This analysis focused only on current bedroom exposure. Sensitization was evident at very low levels of exposure. Similar findings were reported in the NCICAS for bedroom exposure to cockroach allergen.²⁰ The NCICAS found associations between sensitization and mouse allergen exposure in the kitchen and living room but not in the bedroom.³

We found a relationship among mouse allergen sensitivity, exposure, and asthma morbidity, even after controlling for cockroach sensitivity and exposure because this was shown to have a significant impact on asthma morbidity in the NCICAS.¹⁶ Asthma hospitalization rates, maximum asthma symptom days, nights of child sleep disruption, and days of caretaker change in plans were significantly higher in mouse-allergic children exposed to mouse allergen. Other

asthma-related outcomes, specifically, days of wheeze and nights of caretaker sleep disruption, approached significance in the same children. A recent study⁷ of inner-city preschool children found more asthma symptom days, β -agonist use, unscheduled physician visits, ED visits, and hospitalizations among those sensitized and exposed to high levels (>0.5 $\mu\text{g/g}$) of mouse allergen. The NCICAS did not find a significant effect of mouse exposure and sensitization on asthma morbidity, although trends were seen for days of wheezing, child's sleep disruption, and reduced activity.³ Although median bedroom levels of mouse allergen were similar in the NCICAS and the ICAS, the NCICAS demonstrated higher median levels (1.60 $\mu\text{g/g}$) in the kitchen,² and this source of exposure was used in analyses of asthma morbidity.³ This approach may account for why ICAS (bedroom) and NCICAS findings differ.

The ICAS is also the largest controlled trial of an environmental intervention to reduce mouse allergen levels in inner-city homes. Whereas allergen levels increased by 28% in control homes, there was a 27% reduction in mouse allergen levels on the bedroom floor but not in the bed in intervention

Table 4. Relationship Between Mus m 1 Reduction and Change in Morbidity Among Rodent Intervention Group Participants

	Morbidity change, mean (SE) ^a	P value
Maximum symptom days in past 2 wk	−0.0108 (0.0891)	.90
Days of wheeze in past 2 wk	−0.0372 (0.0771)	.63
Nights child lost sleep in past 2 wk	−0.0708 (0.0756)	.35
Days child's activity reduced in past 2 wk	−0.1664 (0.0600)	.006
School days missed in past 2 wk	−0.0653 (0.0326)	.05
Nights caretaker lost sleep in past 2 wk	−0.1535 (0.0641)	.02
Days caretaker changed plans in past 2 wk	−0.0989 (0.0468)	.03
Hospitalizations in past 2 mo	−0.0104 (0.0068)	.13
Unscheduled asthma visits in past 2 mo	−0.0311 (0.0205)	.13

^a Morbidity change for a 50% reduction from the baseline allergen level. Results are controlled for sensitivity and exposure to cockroach allergen.

homes. Because the intervention focused on limiting food sources, vacuuming, and rodent access, the preferential effect on floor Mus m 1 is not surprising. This finding regarding increased allergen levels in control homes is interesting because Mus m 1 is a urinary allergen, and the ongoing or growing presence of mice might be expected to increase allergen deposition over time.

Decreased mouse allergen levels were associated with significant reductions in some measures of impairment but not risk, specifically, asthma-related outcomes, including child sleep disruption and slow play, but not wheeze or health care utilization. Although the interventions to cockroach and mouse were similar, these morbidity effects were observed after controlling for cockroach exposure and sensitivity, so we do not believe that these results are due to reduced cockroach exposure. Phipatanakul et al⁹ conducted an environmental intervention in 18 inner-city homes in Boston. Their strategy incorporated integrated pest management using pesticides and professional extermination 4 times during 5 months. They achieved a 79% reduction in mouse allergen levels, whereas levels in control homes increased more than 300%. No effect on asthma symptoms or pulmonary function was detected, but the sample size was rather modest to detect such effects. It is not known whether environmental interventions affect airborne Mus m 1, but such a determination would be useful in assessing the potential for impacting asthma morbidity. A recent study¹⁸ found that 90% of households with greater than 0.5 µg/g of Mus m 1 in bedroom settled dust had detectable airborne mouse allergen.

The strengths of this study include the large number of participants and the wide geographic distribution. A limitation is that ongoing allergen exposure was evaluated only in

the bedroom. Previous exposure or other sources of exposure could not be taken into account. Also, inner-city families frequently relocate. Consequently, current exposure may not relate to the previous development of sensitization. In addition, although we used a 3-mm wheal diameter for morbidity analyses, the appropriate cutoff value for the Multi-Test device may be different from that of other devices. Symptom outcomes were based on self-report rather than on objective assessments or medical record review. Because we did not measure rat allergen, we cannot evaluate whether these findings are due to reduction in rat allergen levels. Finally, because most homes received a HEPA air purifier it is unclear to what extent decreased exposure to particulates and environmental tobacco smoke contributed to these results.

Asthma is generally accepted to be a multifactorial disease in which allergen sensitization and exposure play a critical role. Previous studies have demonstrated that cockroach allergen is an independent risk factor for asthma morbidity in inner-city children.¹⁶ This article demonstrates that mouse allergen sensitization and exposure are also important and that sensitization may occur at low levels of exposure. Mouse allergen affects asthma symptoms and hospitalization rates independent of cockroach allergen. Furthermore, we presented the results of the largest controlled trial of a mouse allergen environmental intervention to date. After controlling for cockroach allergen exposure and sensitivity, the reduction in mouse allergen levels was associated with significantly decreased caregiver burden, child sleep disruption, and missed school. These findings suggest that mouse allergen is important in inner-city children with asthma and that tailored environmental interventions can be effective in reducing mouse allergen concentrations and in improving asthma-related outcomes.

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