Airborne mouse allergen in the homes of inner-city children with asthma

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Background: Airborne mouse allergen has not previously been measured in inner-city homes, and its relationship to settled dust mouse allergen levels is unknown.
Objective: To quantify airborne and settled dust Mus m 1 levels in homes of inner-city patients with asthma and to identify risk factors for mouse allergen exposure.
Methods: One hundred inner-city school-age children with asthma in Baltimore underwent skin testing to a panel of aeroallergens, and their homes were inspected by a trained technician. Air and settled dust were sampled in the child's bedroom. Mus m 1, particulate matter smaller than 10 microns (PM_{10}), and particulate matter smaller than 2.5 microns were quantified in air samples, and Mus m 1 was quantified in settled dust samples.
Results: Mus m 1 was detected in settled dust samples from 100% of bedrooms. Airborne mouse allergen was detected in 48 of 57 (84%) bedrooms, and the median airborne mouse allergen concentration was 0.03 ng/m^3. The median PM_{10} concentration was 48 μg/m^3. Airborne and settled dust mouse allergen levels were moderately correlated ($r = .52$; $P < .0001$), and airborne Mus m 1 and PM_{10} levels were weakly correlated ($r = .29$; $P = .03$). Having cracks or holes in doors or walls, evidence of food remains in the kitchen, and mouse infestation were all independently associated with having detectable airborne mouse allergen.
Conclusion: Airborne mouse allergen concentrations in many inner-city homes may be similar to those found in animal facilities, where levels are sufficiently high to elicit symptoms in sensitized individuals. Exposed food remains, cracks and holes in doors or walls, and evidence of mouse infestation appear to be risk factors for having detectable airborne Mus m 1.
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Key words: *Mouse allergen, inner-city asthma, particulate matter, Mus m 1*

Abbreviations used
NCICAS: National Cooperative Inner City Asthma Study
OR: Odds ratio
PM_{10}: Particulate matter smaller than 10 microns
$PM_{2.5}$: Particulate matter smaller than 2.5 microns

Environmental and occupational respiratory disorders

Although mouse allergen is a well-recognized occupational allergen,[1,2] it has only recently been identified as a common household allergen. More than 3 quarters of US homes have detectable mouse allergen,[3] and the prevalence of mouse skin test sensitivity is 10% to 20%,[4,5] depending on the population studied. Mouse allergen is virtually ubiquitous in inner-city homes and has been detected in approximately 75% of middle-class suburban homes, but settled dust concentrations of mouse allergen are a log-fold higher in inner-city homes than in suburban homes.[5] However, settled dust concentrations have not been compared with airborne concentrations, so it is impossible to determine how household mouse allergen levels compare with levels in occupational settings, where levels are quantified in terms of airborne concentrations and median levels have been reported to be 0.13 ng/m^3.[2] If inner-city airborne mouse allergen levels are similar to those found in occupational settings where mouse allergy is a significant occupational health hazard, mouse allergen exposure may play a substantial role in asthma disease activity among inner-city inhabitants who are sensitized to mouse.

Although exposure to indoor allergens is through inhalation, exposure is typically assessed through reservoir dust sampling. The relationship between reservoir dust and air sampling has been examined for cat allergen, and no correlation was found between the settled dust and airborne cat allergen concentrations,[6] but this association has not been examined for mouse allergen. Because most nonoccupational studies of mouse allergen exposure have used settled dust allergen measures, we examined airborne and settled dust mouse allergen levels in homes of inner-city children with asthma to develop a better understanding of the relationship between dust and airborne measures of mouse allergen and to determine risk factors for domestic mouse allergen exposure.

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METHODS

Study population

Participants were recruited for an environmental intervention study from the Baltimore City public elementary schools and attended a school-based asthma education program. At the conclusion of all educational sessions, families who participated in the program were asked whether they were willing to participate in a study of environmental control measures. If the family expressed interest, a trained recruiter/interviewer contacted them and determined their willingness and eligibility. Eligibility requirements included an age between 6 and 12 years, doctor-diagnosed asthma, current asthma symptoms, and no other chronic lung disease.[7] If the families were willing and eligible, written informed consent was obtained. Three hundred eighty-seven children completed the asthma educational program, and 292 children who were potentially eligible for the study were identified. Of the 180 children successfully contacted by a recruiter, 100 completed the baseline home evaluation and clinic visit.[7] Institutional Review Boards for the Johns Hopkins University and Baltimore City Board of Education approved the study.

Baseline assessment

A trained interviewer administered a detailed questionnaire ascertaining demographic, medical, and environmental characteristics at the baseline visit. Eligible participants then received a home evaluation visit and a clinic evaluation. During the home environmental visit, environmental technicians completed an inspection checklist,[8] indoor air was collected for pollutant and mouse allergen analysis, and settled dust was collected for mouse allergen analysis. Air sampling in the child's bedroom was conducted over a 72-hour period. Samples for airborne particulate matter (particulate matter smaller than 10 microns [PM_{10}] and 2.5 microns [$PM_{2.5}$]) were collected by using 4 L/min MSP impactors (St Paul, Minn) loaded with 37-mm, 2-μm–pore PALL Teflo polytetrafluoroethylene membrane filters (Pall Corp, Ann Arbor, Mich). Air samples for mouse allergen analysis were collected on 25-mm, 0.3-μm–pore polytetrafluoroethylene membrane filters by using IOM Inhalable Dust Samplers (SKC, Eighty Four, Pa) at a flow rate of 2 L/min. Samples of duration <24 hours and with flow rates deviating by more than 25% from the 2 L/min set point were excluded from analysis. Household dust samples were collected from the child's bedroom, television-living room, and kitchen by using published methods.[9] Protein was extracted from the filters and dust samples by using a standardized protocol, and Mus m 1 was quantified by sandwich ELISA by using immunosorbant purified sheep anti–Mus m 1 (kindly supplied by Dr J. Ohman).[10] The dust samples were analyzed by using a sandwich ELISA, and the air samples were analyzed by using an amplified ELISA in which AMDEX streptavidin–horseradish peroxidase (Amersham Pharmacia Biotech, Buckinghamshire, United Kingdom) was used for the detection step.[11] The limit of detection for the unamplified Mus m 1 ELISA was 50 ng/g of dust, and for the amplified ELISA, 0.03 ng per air filter. The limit of detection for a typical 72-hour air sample of 8.6 m^3 air was therefore 0.003 ng/m^3.

During the clinic visit, each child underwent skin prick testing (Multi-Test II; Lincoln Diagnostics, Decatur, Ill) to 14 aeroallergens: American and German cockroach, dust mite mix, cat, dog, mouse, rat, 3 pollens, and 3 molds (Hollister-Stier Laboratories, Spokane, Wash; and Greer Laboratories, Lenoir, NC).

Statistical methods

All statistical analyses were performed with StataSE 8.0 (College Station, Tex). The correlations between airborne mouse allergen levels and settled dust mouse allergen, PM_{10}, and $PM_{2.5}$ levels were analyzed by using the Spearman correlation. Airborne mouse allergen levels were dichotomized to undetectable and detectable levels and low and high levels, with a high level defined as a level greater than the median, 0.03 ng/m^3. Similarly, dust levels of mouse allergen were dichotomized to low and high levels with a cutoff set at the median level of 3.8 μg/g. The relationships between sociodemographic and housing characteristics and mouse allergen levels were analyzed by using cross-tabulations, and odds ratios (ORs) were generated with simple logistic regression. Multivariable logistic regression was used to adjust for potential confounders.

RESULTS

Study population

Most of the participants were female (54.0%), and the mean age was 8.4 years (Table I). The participants were almost exclusively African American (99.0%) and had low annual incomes. The majority of participants lived in a home with a smoker (69.1%), and 31.0% of the participants were on a controller medication for asthma. Nine participants (9.2%; 95% CI, 4.2-16.6) were sensitized to mouse, and 69.7% had at least 1 positive skin test result. Twenty-two participants were sensitized to cat and 41 to cockroach. Five of the mouse-sensitized participants were also sensitized to cat, and 7 were also sensitized to cockroach.

Sixty-six percent of homes had cracks or holes in walls or doors, and seventy-six percent of homes had exposed food remains in the kitchen. Forty-one percent of homes had evidence of mouse infestation, and 33% had evidence of cockroach infestation. Approximately 1 quarter of homes had a cat.

Exposure characteristics

Ninety-eight families had valid baseline bedroom dust mouse allergen levels, and 57 families had valid airborne mouse allergen measurements. Among those with valid airborne Mus m 1 levels, the mean age was 8.3 years and 28 (49%) were female, and among participants without a valid measure, the mean age was 8.5 years and 60% were female ($P = .46$ and .26, respectively). The income of the group with valid airborne Mus m 1 levels was also similar to the subgroup without valid airborne measures: 28 (50%) had an income of <$15,000, 18 (32%) had an income of $15,000 to $24,999, and 7 (12%) had an income of ≥$25,000, compared with 56%, 35%, and 7% in each respective income stratum in the subgroup without valid airborne measures ($P = .68$). The median settled dust mouse allergen concentration was highest in the kitchen, and the levels from the television room, bedroom, and kitchen were highly correlated (kitchen and television room: $r = .71$; kitchen and bedroom: $r = .69$; television room and bedroom: $r = .82$; $P < .0001$ for all correlations). Every bedroom had detectable mouse allergen in settled dust, and 48 of 57 (84.2%) bedrooms had detectable mouse allergen in the air (Table II). The median bedroom Mus m 1 settled dust concentration was 3.8 μg/g, and the

TABLE I. Study population characteristics

Baseline characteristic	
Age, y (mean ± SD)	8.4 ± 1.4
Female, n (%)	54 (54.0)
African American, n (%)	99 (99)
Annual income,* n (%)	
<$15,000	52 (52.5)
$15,000-24,999	33 (33.3)
≥$25,000	10 (10.1)
Refused or don't know	4 (4.1)
Smoker in home,* n (%)	67 (69.1)
Mouse skin test sensitivity,* n (%)	9 (9.2)
Atopy,* n (%)	69 (69.7)
On controller medication, n (%)	31 (31.0)
Cracks or holes in walls/doors	66 (66.0)
Dog in household	20 (20.0)
Cat in household	26 (26.0)
Evidence of mice	41 (41.0)
Evidence of cockroaches	33 (33.0)
Food on kitchen countertops/floor	76 (76.0)
Food remains in television room or bedroom*	48 (48.5)

*Three or fewer missing.

TABLE II. Exposure characteristics

Exposure variable* (n)	
Airborne Mus m 1, ng/m^3 (57)	0.03 (0.01-0.10)
Settled dust Mus m 1, μg/g	
Kitchen (99)	14.7 (1.5-37.4)
Television room (97)	4.7 (1.4-13.4)
Bedroom (98)	3.8 (1.0-10.4)
Airborne particulate matter, μg/m^3	
PM_{10} (93)	48 (31-71)
$PM_{2.5}$ (91)	35 (21-57)
Mouse infestation (100)	41 (41)
Roach infestation (100)	33 (33)

*Presented as median (interquartile range).

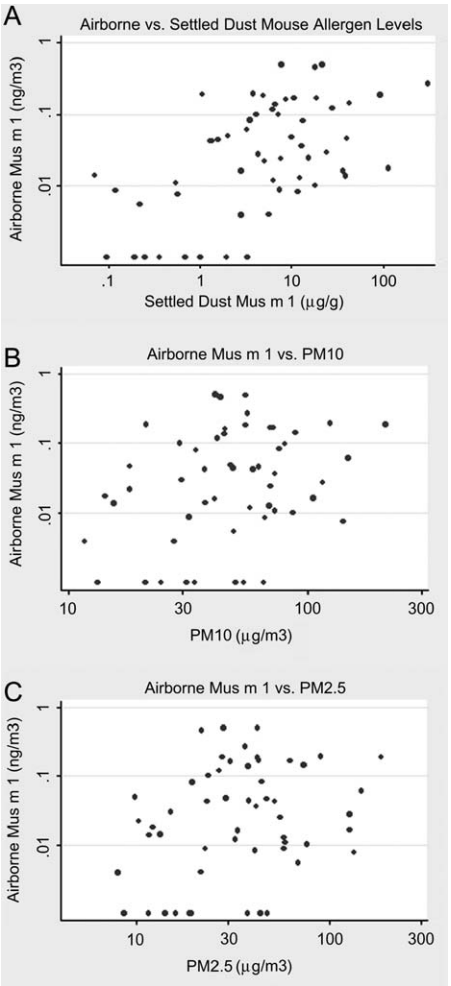

FIG 1. Scatter plots of baseline levels of airborne Mus m 1 and settled dust Mus m 1, PM_{10}, and $PM_{2.5}$. Airborne Mus m 1 concentrations (ng/m^3) are plotted in on the *y-axes*, and settled dust Mus m 1 (μg/g), PM_{10} (μg/m^3), and $PM_{2.5}$ (μg/m^3) are plotted on the *x-axes*. **A,** Settled dust Mus m 1 (n = 57); $r = .53$; $P < .0001$. **B,** PM_{10} (n = 57); $r = .29$; $P = .03$. **C,** $PM_{2.5}$ (n = 54); $r = .24$; $P = .09$.

median airborne concentration was 0.03 ng/m^3. Airborne and settled dust mouse allergen levels were moderately correlated ($r = .52$; $P < .0001$; Fig 1, *A*). Ninety percent of bedrooms with more than 0.5 μg/g of Mus m 1 in settled dust had detectable airborne Mus m 1. The median bedroom PM_{10} and $PM_{2.5}$ levels were 48 μg/m^3 and 35 μg/m^3, respectively. PM_{10} and airborne mouse allergen levels were weakly correlated ($r = .29$; $P = .03$), and $PM_{2.5}$ and airborne mouse allergen levels were not significantly correlated ($r = .24$; $P = .09$; Fig 1, *B and C*).

Demographic and home characteristics and airborne mouse allergen levels

Having cracks or holes in walls or doors was associated with a more than 5-fold increased risk of having detectable airborne mouse allergen (OR, 5.4; 95% CI, 1.2-24.8) but was not significantly associated with having high airborne mouse allergen levels (OR, 2.4; 95% CI, 0.8-7.3) or high settled dust mouse allergen levels (OR, 1.3; 95% CI, 0.6-3.1; Table III). Evidence of mouse infestation was associated with a more than 10-fold risk of having detectable airborne mouse allergen (OR, 10.3; 95% CI, 1.2-88.8) but was not significantly associated with having high levels of airborne or settled dust mouse allergen (OR, 1.6; 95% CI, 0.6-4.7; and OR, 2.0; 95% CI, 0.9-4.5, respectively). The presence of a cat was associated with a decreased risk of both detectable (OR, 0.4; 95% CI, 0.1-1.9) and high airborne mouse allergen levels (OR, 0.5; 95% CI, 0.1-1.9), but these point estimates were not statistically significant. However, the presence of a cat was associated with a substantially lower risk of having high levels of settled dust mouse allergen (OR, 0.2; 95% CI, 0.1-0.5). Neither cockroach infestation nor the presence of a dog was significantly associated with airborne or settled dust mouse allergen levels.

Exposed food in the kitchen, either on the countertops or the floor, was associated with a more than 5-fold

TABLE III. Predictors of airborne and settled dust mouse allergen levels, OR (95% CI)

Characteristic	Detectable air Mus m 1	High* air Mus m 1	High† dust Mus m 1
Cracks or holes in walls/doors	5.4 (1.2-24.8)	2.4 (0.8-7.3)	1.3 (0.6-3.1)
Dog in household	1.9 (0.2-16.7)	0.6 (0.2-2.4)	0.8 (0.3-2.1)
Cat in household	0.4 (0.1-1.9)	0.5 (0.1-1.9)	0.2 (0.1-0.5)
Evidence of mice	10.3 (1.2-88.8)	1.6 (0.6-4.7)	2.0 (0.9-4.5)
Evidence of cockroaches	3.6 (0.4-31.7)	1.4 (0.4-4.3)	0.8 (0.4-1.9)
Food on kitchen countertops/floor	5.4 (1.2-24.7)	3.3 (1.0-10.7)	1.0 (0.4-2.5)
Food remains in TV room or bedroom	2.3 (0.4-12.2)	1.5 (0.5-4.4)	1.5 (0.7-1.2)
Age	1.1 (0.7-1.8)	0.9 (0.6-1.3)	0.9 (0.7-1.2)
Male sex	0.70 (0.2-3.1)	0.4 (0.1-1.2)	0.9 (0.4-2.0)
Income			
<$15,000	1.0	1.0	1.0
$15,000-24,999	4.6 (0.5-42.3)	0.7 (0.2-2.3)	1.2 (0.5-2.8)
≥$25,000	0.7 (0.1-4.4)	0.7 (0.1-3.5)	1.8 (0.5-7.3)
Smoker in home	4.1 (0.9-17.9)	2.0 (0.6-6.7)	1.4 (0.6-3.3)
Mouse skin test sensitivity	0.4 (0.0-4.4)	0.5 (0.0-5.6)	0.5 (0.1-1.9)
On controller medication	0.7 (0.1-3.1)	1.7 (0.5-5.5)	0.8 (0.3-1.8)

*>0.03 ng/m^3.
†>3.8 μg/m^3.

TABLE IV. Multivariate analysis of predictors of mouse allergen levels, adjusted OR (95% CI), adjusted for income

Home characteristic	Detectable air Mus m 1	High* air Mus m 1	High† dust Mus m 1
Cracks or holes in walls/doors	6.2 (1.3-29.1)	1.9 (0.6-6.1)	1.5 (0.6-3.5)
Dog in household	1.9 (0.2-18.0)	0.6 (0.2-2.4)	0.9 (0.3-2.4)
Cat in household	0.4 (0.1-2.0)	0.5 (0.1-1.9)	0.2 (.05-0.5)
Evidence of mice	10.0 (1.1-87.4)	1.5 (0.5-4.4)	1.9 (0.8-4.3)
Evidence of cockroaches	3.4 (0.4-30.3)	1.2 (0.4-3.9)	0.8 (0.3-1.9)
Food on kitchen countertops/floor	6.5 (1.3-32.6)	3.3 (1.0-10.7)	1.1 (0.4-2.9)
Food remains in television room or bedroom	2.5 (0.5-13.4)	1.6 (0.5-4.7)	1.6 (0.7-3.5)

*>0.03 ng/m^3.
†>3.8 μg/m^3.

increased risk of having detectable airborne mouse allergen (OR, 5.4; 95% CI, 1.2-24.7) and a more than 3-fold increased risk of having high levels of airborne mouse allergen (OR, 3.3; 95% CI, 1.0-10.7). Age, sex, income, the presence of a smoker in the home, mouse skin test sensitivity, and taking a controller medication were not associated with having detectable airborne, high airborne, or high settled dust mouse allergen levels.

After adjusting for income, cracks or holes in walls or doors, mouse infestation, and food remains in the kitchen remained independent predictors of detectable airborne Mus m 1 (Table IV). Similarly, food remains on the kitchen counters or floor remained an independent predictor of high airborne Mus m 1 levels, and a cat in the household was associated with a decreased risk of high settled dust levels of Mus m 1 after adjusting for income.

DISCUSSION

This study is the first to describe airborne mouse allergen levels in inner-city homes, and our findings suggest that as many as a quarter of inner-city homes may contain airborne concentrations of mouse allergen of 0.1 ng/m^3 or greater, similar to levels found in mouse research facilities.[2] In addition, the presence of holes in walls or doors, exposed food in the kitchen, and evidence of mouse infestation were all strongly and independently associated with detectable airborne mouse allergen levels.

The fact that 90% of participants with >0.5 μg/g of Mus m 1 in bedroom settled dust samples had detectable airborne Mus m 1 suggests that a significant proportion of inner-city homes may have detectable airborne Mus m 1. In the National Cooperative Inner City Asthma Study (NCICAS), in which the median bedroom Mus m 1 level was 0.5 μg/g, as many as 45% of all participants may have had detectable airborne Mus m 1 in their bedrooms.[12] We have previously reported that the median airborne mouse allergen concentration from a survey of a mouse facility was 0.13 ng/m^3,[2] and allergic workers who enter mouse rooms at this facility report allergic symptoms. Therefore, the fact that a quarter of our study participants' homes had levels >0.1 ng/m^3 suggests that household mouse allergen

levels may be high enough to trigger asthma symptoms in mouse-sensitized inner-city populations. Although analysis of data from the NCICAS found no statistically significant relationship between settled dust mouse allergen levels and sensitization and measures of asthma morbidity, there was a trend suggesting that mouse sensitization may be associated with more days of wheezing, more nights of lost sleep, and more days of reduced activity.[4] An analysis of asthma morbidity in the current study was not performed because of the small number of participants with mouse skin test sensitivity, but the findings support the need for further investigation into the role of mouse allergen exposure in asthma morbidity.

We also found that having cracks in walls or doors and exposed food remains were both significant risk factors for detectable levels of airborne mouse allergen, but neither characteristic was associated with having high levels of settled dust mouse allergen. These findings suggest that sealing holes and vigilance in cleaning up food remains could reduce airborne mouse allergen levels. It is unclear, however, why these 2 characteristics were associated with airborne, but not settled dust, mouse allergen levels. This discrepancy needs further exploration in future studies to determine whether these risk factors are indeed specific for airborne, rather than settled dust, allergen. The presence of a cat was associated with a decreased risk of high dust levels of mouse allergen, and this finding corroborates findings published by Chew et al.[13] Although the relationship between having a cat and airborne mouse allergen levels was not statistically significant, the point estimates are similar to those found for settled dust mouse allergen, and the lack of statistical significance may reflect the smaller sample size for airborne mouse allergen. However, because many individuals allergic to mouse will also be allergic to cat, acquisition of a cat to control mouse allergen levels may not be a reasonable public health strategy to reduce mouse allergen levels.

Interestingly, settled dust and airborne concentrations of mouse allergen were correlated in this study. These findings contrast with those from previous studies in which no relationship was found between settled dust and airborne Fel d 1 levels.[6] Because both mouse and cat allergens are found in the particle size fraction from 0.5 to 10 μm[10,14] and therefore have a long airborne residence time once resuspended, some correlation between dust and airborne levels would be expected for both allergens. On the other hand, the discrepancy between mouse and cat allergen may be a result of differences in the chemical and physical characteristics of the allergen as well as the reservoir matrix and surface characteristics (eg, carpet vs linoleum).[15] This discrepancy between the 2 allergens, however, can most likely be attributed to differences in the sampling methods used in the studies. In the current study, indoor sampling was conducted by using an inspirable sampler with flow rates of 2 L/min over a 72-hour period, whereas personal samplers were used in the cat allergen study at flow rates of 3 L/min to 4 L/min over a 1-hour period. The longer sampling time in the current study may simply have compensated for any large fluctuations in airborne allergen levels that may occur with changes in the household activity level. Because airborne Mus m 1 is a component of PM_{10}, one might expect that the 2 measures would be correlated. Although homes with higher PM_{10} levels tended to have higher airborne Mus m 1 levels, the weak correlation between these 2 measures is a function of the fact that there are many factors, such as environmental tobacco smoke, that contribute to PM_{10} levels. Airborne Mus m 1 and $PM_{2.5}$ were not likely to be correlated for similar reasons. In addition, Mus m 1 is found primarily among particles from 3.3 to 10 microns[10] and therefore is less likely to be related to $PM_{2.5}$ than to PM_{10}.

Valid airborne samples were obtained for only 57% of study participants, so it is possible that the airborne Mus m 1 levels are not representative of levels in the study population as a whole. Obtaining airborne samples proved to be more difficult than obtaining dust samples because a monitor had to be left in the home for a period of days. Successful procurement of an air sample required access to the house to set up the monitor, functioning equipment for a period of 3 days, no manipulation of the pump by the family, and access to the home to retrieve the pump. However, there were no differences in age, sex, or income between participants with and without valid airborne measurements, suggesting that these airborne Mus m 1 levels are a reasonable estimate of levels in the study population as a whole. In addition, the study focused on a relatively small sample of homes of school-age children with asthma living in inner-city Baltimore, so the airborne mouse allergen levels may not be representative of levels found in homes of people without asthma or in other cities. However, these findings underscore the need for further examination of airborne Mus m 1 levels and their effect on asthma morbidity.

Although previous reports indicate that approximately 10% to 20% of children with asthma are sensitized to mouse,[4,5] only 9% of this study population demonstrated skin test sensitivity to mouse. The 70% prevalence rate of atopy in this study population is also slightly lower than that found in other inner-city study populations, so the prevalence rates of specific sensitivities may be lower than those reported for other inner-city school-age children with asthma. However, cat and cockroach sensitization rates are similar to those reported in the NCICAS study population.[16] The relatively low prevalence rate of mouse sensitization is more likely a function of random sampling, as suggested by an upper 95% confidence limit of 16.6% for the prevalence rate.

In summary, airborne mouse allergen concentrations in many inner-city homes may be similar to those found in animal facilities, where levels are sufficiently high to elicit symptoms in sensitized individuals. Further study of the role of mouse allergen exposure in asthma morbidity in nonoccupational settings is clearly needed. Interventions aimed at reducing airborne mouse allergen should focus on rodent extermination, sealing holes and cracks, and educating families to clean up all food remains.

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