Mouse allergen exposure and mouse skin test sensitivity in suburban, middle-class children with asthma

Elizabeth C. Matsui, MD,[a] Robert A. Wood, MD,[a] Cynthia Rand, PhD,[b] Sukon Kanchanaraksa, PhD,[c] Lee Swartz, MBA,[a] and Peyton A. Eggleston, MD[a] *Baltimore, Md*

Background: Exposure to mouse allergen is prevalent in inner-city homes and is associated with an increased risk of mouse skin test sensitivity in inner-city children with asthma.
Objective: To determine the distribution of mouse allergen and its relationship to mouse skin test sensitivity in a primarily suburban, middle-class population of asthmatic children.
Methods: Children with asthma, 6 to 17 years old, were recruited from 3 pediatric practices located in counties surrounding the city of Baltimore and from 1 practice located within the city limits. Participants underwent skin prick testing and completed a baseline questionnaire. Their homes were inspected, and settled dust samples were collected for allergen analysis.
Results: Two hundred fifty-seven of 335 (76.7%) participants resided outside the city, and 53.7% had annual incomes >$50,000. Mouse allergen was detected in 74.9% of bedrooms, and 13.1% were sensitized to mouse. Lower maternal education (odds ratio [OR], 2.17; 95% CI, 1.28-3.67), city residence (OR, 5.39; 95% CI, 2.23-13.02), and higher bedroom cockroach allergen levels (OR, 9.61; 95% CI, 1.17-79.03) were independent predictors of high bedroom mouse allergen. The risk of mouse skin test sensitivity increased with increasing bedroom Mus m 1 exposure (OR, 1.43; 95% CI, 1.04-1.96, with each increase in quartile), and dog skin test sensitivity was a strong independent predictor of mouse skin test sensitivity (OR, 7.23; 95% CI, 3.03-17.22).
Conclusion: Mouse allergen exposure is common among suburban, middle-class asthmatic children. Increasing bedroom levels of Mus m 1 and dog skin test sensitivity are risk factors for mouse skin test sensitivity. (J Allergy Clin Immunol 2004;113:910-5.)

Key words: *Mouse allergen, pediatric asthma, mouse allergy, indoor allergen exposure*

Mouse allergen exposure is common among laboratory animal workers and is associated with an increased risk of occupational mouse allergy.[1-3] In fact, workers with mouse allergy frequently have substantial symptoms and may develop occupational asthma.[4,5] Although several studies have been published examining the relationship between mouse exposure and clinical mouse allergy in the occupational setting,[3,5,6] less is known about domestic mouse allergen exposure and its relationship to mouse allergy and asthma.

Abbreviations used
NCICAS: National Cooperative Inner-City Asthma Study
OR: Odds ratio

Recently, mouse allergen was found to be almost ubiquitous in the homes of children who participated in the National Cooperative Inner-City Asthma Study (NCICAS).[7] Risk factors for high levels of mouse allergen in this population included both mouse and cockroach infestations. In addition, 18% of this study population demonstrated skin test sensitivity to mouse, and exposure to kitchen mouse allergen levels >1.6 μg/g was associated with an increased risk of IgE-mediated mouse sensitization.[8] The distribution of mouse allergen in homes outside the inner city and its relationship to allergic sensitization, however, remain unknown. We therefore examined mouse allergen exposure and allergic sensitization in a population of primarily middle-class, suburban children with asthma to determine the prevalence of mouse allergen exposure and examine the relationship between exposure and allergic sensitization to mouse.

METHODS

Study population

Participants were recruited for a randomized controlled trial of environmental allergen control from 3 pediatric practices in the counties surrounding Baltimore and 1 practice located within the Baltimore city limit. Data collected during the baseline visit of the study were analyzed for the current study. Children between 6 and 17 years old who had doctor-diagnosed asthma were eligible if they had currently active asthma, defined as at least 1 symptomatic day in the previous week, and families were willing to have home visits for dust collection. The Johns Hopkins University Joint Commission on Clinical Investigation approved the study, and all parents and children provided written informed consent and assent, respectively.

From the Departments of [a]Pediatrics and [b]Medicine and the [c]Bloomberg School of Public Health, Johns Hopkins University, Baltimore, Md.
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Reprint requests: Elizabeth C. Matsui, MD, Johns Hopkins Hospital, CMSC 1102, 600 N Wolfe St, Baltimore, MD 21287; E-mail: ematsui@jhmi.edu.
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Skin testing

Skin tests to cat, dog, *Dermatophagoides pteronyssinus*, *Dermatophagoides farinae*, mouse, German cockroach, ragweed, grass, oak, maple, *Alternaria*, *Penicillium*, *Aspergillus*, and *Helminthosporium* were performed by using full-strength glycerinated extracts with the MultiTest device (Lincoln Diagnostics, Decatur, Ill). The skin test panel was considered valid if the positive control was at least 3 mm, and a particular skin test was considered positive if the orthogonal wheal diameter was at least 3 mm greater than the negative control and at least ½ of the histamine control.

Questionnaire

As part of the baseline evaluation, parents completed a questionnaire that included sociodemographic information such as maternal education, family income and race, and details about the home environment. The questionnaire was adapted from one used in the Childhood Asthma Management Program study.[9]

Environmental assessment

A trained environmental technician conducted a home inspection as a part of the baseline evaluation by using a home evaluation checklist to collect data regarding structural aspects of the home and the location of the home (city, suburban, rural). The home evaluation checklist was originally developed and tested in the NCICAS.[10] A dust sample was also collected during this visit.

Dust sample collection, extraction, and ELISAs

Baseline dust samples were collected by trained research technicians by using a hand-held vacuum (Oreck, New Orleans, La) and collection sleeve according to published methods. Samples were collected from the bed and adjacent floor, television room furniture and the adjacent floor, and the kitchen floor. After sieving, samples were extracted in borate buffered saline with 5% BSA, and extracts were analyzed for Mus m 1, Bla g 1, Can f 1, Der p 1, Der f 1, and Fel d 1 by using 2-site monoclonal-based ELISAs.[11-13]

Statistical analysis

All analyses were performed by using Stata SE 8.0 (College Station, Tex). The study population included 335 participants who completed the baseline evaluation and had valid skin test results and adequate bedroom settled dust samples for mouse allergen analysis. Because 71% of participants resided in the suburbs and only 6% resided in rural areas and the characteristics of these 2 groups were similar, the suburban and rural participants were combined into 1 group. A previous analysis of zip codes of the combined rural and suburban subgroups confirmed that 93% of the study population lived outside the Baltimore city limit.[14] The correlations between room levels of Mus m 1 were examined by using the Spearman rank correlation test. Mus m 1 levels were compared between the city and suburban subgroups by using the Mann-Whitney *U* test. An elevated bedroom Mus m 1 level was defined as a level above the median (22 ng/g), and the distributions of covariates among those with low and high bedroom levels were examined. The relationships between indoor allergen levels and Mus m 1 were also examined, and Bla g 1 and Mus m 1 levels were significantly correlated. Bedroom Bla g 1 was then analyzed as an ordinal variable by using the following 4 categories: below detection, 1 to 2 U/g, 2.01 to 7.99 U/g, and ≥8 U/g. Results of analyses examining the relationships between high bedroom Mus m 1 and sociodemographic and environmental variables were expressed as odds ratios (ORs) and 95% CIs. Multivariable logistic regression was used to adjust for potential confounders.

Bedroom Mus m 1 level was stratified by quartiles, and the Cuzick test for trend was used to analyze prevalence rates of mouse skin test sensitivity by quartile of Mus m 1 exposure.[15] Atopy was defined as an ordinal variable by using the following 3 categories: 0, 1 to 3, or ≥4 positive skin prick tests. Results of analyses examining the relationships between mouse skin test sensitivity and the study variables, including atopy and exposure, were expressed as ORs and 95% CIs. The association between quartile of bedroom Mus m 1 exposure and mouse skin test sensitivity was modeled by using multivariate logistic regression to control for the potential confounders of age, sex, atopy, and skin test sensitivity to dog.

TABLE I. Study population characteristics

Characteristic	Total population (n = 335)	Suburban (n = 257)	City (n = 78)
Age, y; mean ± SD	10.8 ± 3.1	10.3 ± 3.2	11.1 ± 3.0
Sex, n (%)			
Male	184 (54.9)	142 (55.3)	42 (53.9)
Female	151 (45.1)	115 (44.7)	36 (46.1)
Race			
African American	128 (38.4)	61 (23.8)	67 (87.0)
White	164 (49.3)	156 (60.9)	8 (10.4)
Asian	5 (1.5)	5 (2.0)	0
Hispanic	3 (0.9)	3 (1.2)	0
Multiracial	30 (9.0)	28 (10.9)	2 (2.6)
Other	3 (0.9)	2 (1.2)	0
Maternal education*			
Some high school	19 (5.7)	4 (1.6)	15 (19.5)
High school graduate/ General Educational Development	57 (17.2)	30 (11.8)	27 (35.1)
Some college	97 (29.3)	74 (29.1)	23 (29.9)
College degree	97 (29.3)	91 (35.8)	6 (7.8)
Postgraduate studies	61 (18.5)	55 (21.7)	6 (7.8)
Annual income, $			
<15,000	31 (9.3)	8 (3.1)	23 (29.5)
15,000-29,999	34 (10.2)	12 (4.7)	22 (28.2)
30,000-49,999	41 (12.2)	28 (10.9)	13 (16.7)
50,000-74,999	57 (17.0)	46 (17.9)	11 (14.1)
≥75,000	123 (36.7)	115 (44.7)	8 (10.2)
Declined to answer	49 (14.6)	48 (18.7)	1 (1.3)

*n = 254 for suburban group, 77 for city group.

RESULTS

Study population

The study population characteristics have been described previously and are summarized in Table I. The mean age was 10.8 years, and 54.9% were male. Almost 50% were white, and 38.4% were African American. Approximately 77% resided in the suburbs, and the remainder resided in the city. More than 75% of mothers had completed some college coursework, and more than 50% of families had annual incomes >$50,000.

Distribution of mouse allergen

Mus m 1 was detectable in 74.9% of bedrooms, 79.5% of television rooms, and 63.1% of kitchens. The median bedroom, television room, and kitchen Mus m 1 levels were 22 ng/g, 28 ng/g, and 17 ng/g, respectively. Specific room Mus m 1 levels were correlated with one another (bedroom and television room, $r = 0.67$; $P < .0001$;

TABLE II. Household Mus m 1 levels and home location

Room	City Mus m 1, ng/g median (IQR)	n	Suburban Mus m 1, ng/g median (IQR)	n	*P* value
Bedroom	757 (160-3209)	78	12 (BD-48)	257	<.0001
Television room	996 (177-5588)	77	16 (BD-44)	250	<.0001
Kitchen	2483 (274-18946)	75	6.6 (BD-50)	250	<.0001

BD, Below detection; *IQR,* interquartile range.

TABLE III. Risk factors for high levels of bedroom Mus m 1

Characteristic	Crude OR (95% CI)
Age, y	1.02 (0.96-1.09)
Race, African American vs non−African American	4.13 (2.58-6.62)
Sex, male vs female	0.69 (0.45-1.07)
Annual income, $	
<15,000	10.79 (3.86-30.19)
15,000-29,999	12.04 (4.33-33.42)
30,000-49,999	3.24 (1.56-6.74)
50,000-74,999	2.00 (1.05-3.81)
≥75,000	1.0
Maternal education	
Some high school	7.43 (2.35-23.49)
High school graduate	3.27 (2.06-5.22)
College graduate	1.0
Location, city vs suburban	12.86 (6.14-26.93)
Bedroom Bla g 1 (U/g)	
Below detection	1.0
1-2	1.90 (0.64-5.62)
2.01-7.99	7.40 (2.76-19.87)
≥8	28.47 (3.77-215.24)

bedroom and kitchen, $r = 0.59$; $P < .0001$; kitchen and television room, $r = 0.72$; $P < .0001$). After stratifying by home location, Mus m 1 was detected in 69.3% of suburban bedrooms and 93.6% of city bedrooms ($P < .0001$), 74.4% of suburban television rooms and 96.1% of city television rooms ($P < .0001$), and 55.6% of suburban kitchens versus 88.0% of city kitchens ($P < .0001$). Levels of Mus m 1 in city homes were substantially higher than those in suburban homes in all 3 rooms sampled (Table II). For example, the median bedroom Mus m 1 level was 12 ng/g for suburban homes and 757 ng/g for city homes ($P < .0001$). Bla g 1 levels correlated with bedroom Mus m 1 levels ($r = 0.37$; $P < .0001$), but other indoor allergens, including Can f 1, did not.

In bivariate analyses, African American race (OR, 4.13; 95% CI, 2.58-6.62), lower annual income (<$15,000 vs ≥$75,000: OR, 10.79; 95% CI, 3.86-30.19), lower maternal education (high school degree vs college degree: OR, 3.28; 95% CI, 2.06-5.22), city location (OR, 12.86; 95% CI, 6.14-26.93), and higher bedroom Bla g 1 level (≥8 U/g vs undetectable: OR, 28.46; 95% CI, 3.77-215.24) were all associated with high levels of bedroom Mus m 1 (Table III). In multivariate analyses, lower maternal education (high school degree vs college degree: OR, 2.17; 95% CI, 1.28-3.67), city location (OR, 5.39; 95% CI, 2.23-13.02), and bedroom Bla g 1 (≥8 U/g vs undetectable: OR, 9.61; 95% CI, 1.17-79.03) remained independent predictors of having a high bedroom Mus m 1 (Table IV).

TABLE IV. Multivariable analysis: Risk factors for high levels of bedroom Mus m 1 (n = 331)

Characteristic	Adjusted* OR (95% CI)
Age, y	0.99 (0.91-1.07)
Race, African American vs non−African American	1.46 (0.81-2.66)
Sex, male vs female	0.73 (0.44-1.23)
Maternal education	
Some high school	1.54 (0.35-6.73)
High school graduate	2.17 (1.28-3.67)
College graduate	1.0
Location, city vs suburban	5.39 (2.23-13.02)
Bedroom Bla g 1 (U/g)	
Below detection	1.0
1-2	0.83 (0.24-2.88)
2.01-7.99	3.44 (1.16-10.22)
≥8	9.61 (1.17-79.03)

*Adjusted for all variables included in the table.

TABLE V. Prevalence rates of skin test sensitivities

Allergen, n (%)	City (n = 78)	Suburban (n = 257)	Total population (n = 335)
Mouse	14 (18.0)	30 (11.7)	44 (13.1)
Cat	35 (44.9)	128 (49.8)	163 (48.7)
Cockroach	28 (34.2)*	54 (21.0)*	82 (24.5)
Dog	5 (6.4)	27 (10.5)	32 (9.6)
Dust mite	54 (69.2)	177 (68.9)	231 (69.0)

*$P = .007$.

Skin test sensitivity

Overall, 13.1% of study participants had a positive mouse skin prick test, 18.0% in the city group and 11.7% in the suburban group ($P = .15$; Table V). Prevalence rates of mouse, cat, dog, and dust mite skin test sensitivities were not significantly different between city and suburban groups. Prevalence rates of cockroach skin test sensitivity were significantly higher in the city group (34.2%) than in the suburban group (21.0%; $P = .007$). In bivariate analyses, maternal education, atopy, and positive cockroach, cat, and dust mite skin prick tests were associated with mouse skin test sensitivity (Table VI). Dog skin test sensitivity was most strongly associated with mouse skin test sensitivity (OR, 8.34; 95% CI, 3.77-18.42). Specifically, 15 of 32 (46.9%) of subjects with a positive skin test to dog also had a positive mouse skin test, whereas only 29

TABLE VI. Risk factors for a positive mouse skin prick test

Characteristic	Crude OR (95% CI)
Age, y	1.06 (0.96-1.17)
Race, African American vs non−African American	1.23 (0.65-2.34)
Sex, male vs female	1.36 (0.71-2.59)
Annual income, $	
<15,000	0.86 (0.27-2.77)
15,000-29,999	0.78 (0.24-2.47)
30,000-49,999	1.00 (0.37-2.72)
50,000-74,999	0.82 (0.32-2.08)
≥75,000	1.0
Maternal education	
Some high school	2.74 (0.80-9.40)
High school graduate	2.09 (1.05-4.17)
College graduate	1.0
Location, city vs suburban	1.66 (0.83-3.31)
Atopy*	3.74 (1.75-7.99)
Specific sensitizations	
Cockroach	3.08 (1.60-5.93)
Dog	8.34 (3.77-18.42)
Cat	2.87 (1.44-5.71)
Dust mite	2.64 (1.13-6.15)

*Atopy defined categorically as 0, 1 to 3, or ≥4 positive skin prick tests.

of 303 (9.6%) of those with a negative skin test to dog had a positive mouse skin test ($P < .0001$).

Bedroom Mus m 1 levels were higher among those with positive mouse skin prick tests than those with negative mouse skin prick tests (medians, 43.5 ng/g vs 19.6 ng/g, respectively; $P = .13$), but the difference was not statistically significant. Mus m 1 levels in other rooms of the home did not differ significantly between the sensitized and unsensitized groups (television room: 40 ng/g vs 27 ng/g; $P = .62$; kitchen: 15 ng/g vs 17 ng/g; $P = .88$).

When bedroom mouse exposure was broken into quartiles, prevalence rates of mouse skin test sensitivity trended upward with increasing quartiles of exposure in both the total study population and the suburban subgroup (Fig 1, *A* and *B*). In the total study population, 8.3% with undetectable bedroom Mus m 1 demonstrated skin test sensitivity to mouse, and the prevalence rates increased to 17.9% among participants in the highest quartile who were exposed to >146 ng/g of bedroom Mus m 1 ($P = .06$). In the suburban group, prevalence rates of mouse skin test sensitivity ranged from 8.9% among those with undetectable bedroom Mus m 1 to 16.0% among those exposed to more than 146 ng/g Mus m 1 in the bedroom ($P = .25$). In the city group, 88% of participants fell into the highest 2 quartiles and had similar prevalence rates of mouse sensitization (20.0% and 18.6%, respectively; $P = .61$; Fig 1, *C*).

After controlling for age, gender, and atopy, quartile of bedroom Mus m 1 and having a positive skin prick test to dog remained independent predictors of mouse skin test sensitivity (Table VII). Each quartile increase of bedroom Mus m 1 was associated with an increased risk of mouse skin test sensitivity (OR, 1.43; 95% CI, 1.04-1.96), and

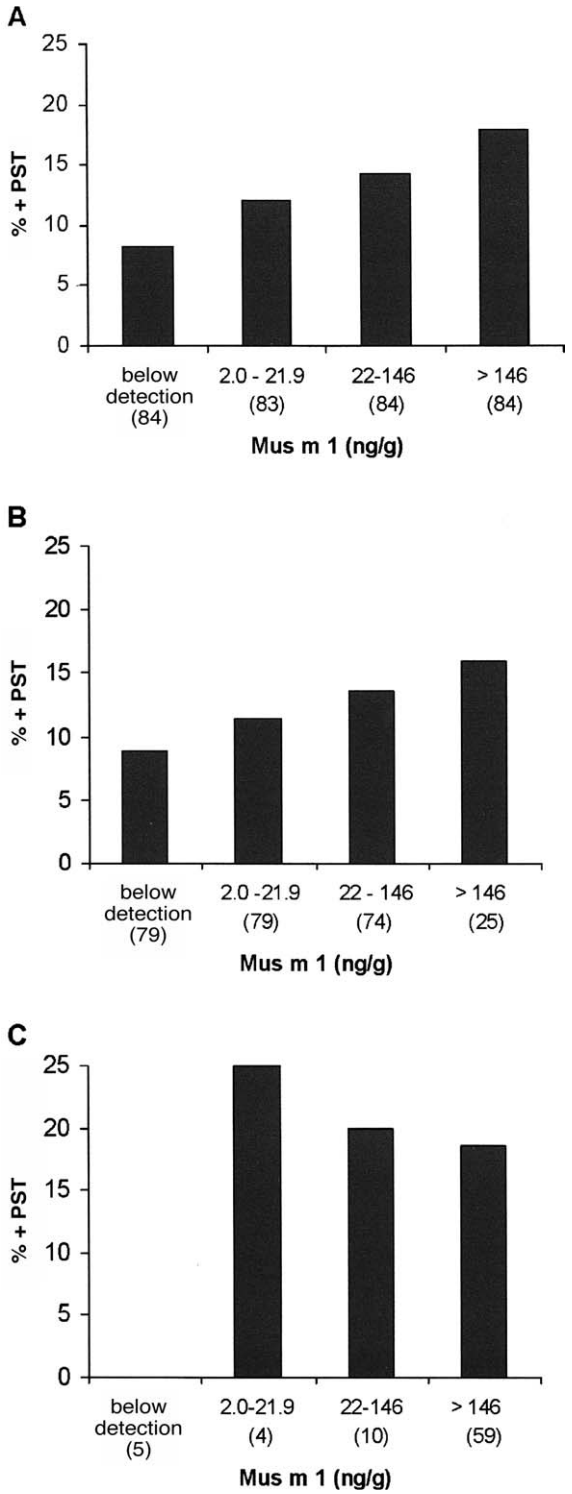

FIG 1. Relationship between quartile of bedroom Mus m 1 and mouse skin test sensitivity. Exposure is expressed in nanograms per gram, and skin test sensitivity is expressed as a prevalence rate within each quartile of Mus m 1. The number of participants in each group is indicated in parentheses along the abscissa. **A,** Total study population ($P = .06$). **B,** Suburban group ($P = .25$). **C,** City group ($P = .51$).

TABLE VII. Bedroom Mus m 1 exposure and mouse sensitization

Characteristic	Adjusted* OR (95% CI)
Bedroom Mus m 1, ng/g, quartiles	1.43 (1.04-1.96)
Age, y	1.05 (0.94-1.18)
Sex, male vs female	1.60 (0.75-3.43)
Atopy	2.66 (1.20-5.91)
Positive skin test to dog	7.23 (3.03-17.22)

*Adjusted for all variables in the table.

a positive dog skin prick test was associated with a more than 7-fold increase in risk of mouse skin test sensitivity (OR, 7.23; 95% CI, 3.03-17.22).

DISCUSSION

Three quarters of the homes in this study had detectable mouse allergen, and even when city homes were excluded from the analysis, mouse allergen was still detectable in almost 70% of the suburban homes. In addition, approximately 13% of participants had a positive skin prick test to mouse, and the risk of mouse skin test sensitivity increased with increasing levels of bedroom Mus m 1 exposure. These findings suggest that mouse allergen exposure may be more common than previously thought for suburban, middle-class asthmatic children, and that the risk of mouse skin test sensitivity increases as exposure increases.

Prevalence rates of mouse allergen exposure among NCICAS participants ranged from 74% in Cleveland to 100% in Baltimore.[7] These prevalence rates are higher than those found in our overall study but are consistent with those in city homes. In fact, living in the city was a strong independent risk factor for having a high bedroom Mus m 1 level. In addition, although the median Mus m 1 levels in our total study population were orders of magnitude less than the range of 0.3 to 7.9 μg/g reported for the NCICAS population,[7] the median levels of Mus m 1 among the city subgroup, which ranged from 0.76 to 2.5 μg/g, were consistent with those found in the NCICAS population.

Bedroom Bla g 1 was also an independent predictor of having a high level of bedroom Mus m 1. Participants with bedroom Bla g 1 levels between 2 and 7.99 U/g had a more than 3-fold greater risk of having high levels of bedroom Mus m 1 than those with undetectable bedroom Bla g 1. Those with >8 U/g of bedroom Bla g 1 were at an almost 10-fold greater risk of having high levels of bedroom Mus m 1. These findings are consistent with those of the NCICAS study, in which cockroach infestation was a strong risk factor for having a high kitchen level of Mus m 1.[7] Although it is likely that the association between cockroach and mouse allergens is explained by certain home characteristics, socioeconomic status, housekeeping practices, or a combination of all 3, the precise reasons for this relationship remain unclear.

This study also suggests that the risk of mouse skin test sensitivity increases with increasing levels of bedroom Mus m 1. Mouse skin test sensitivity was also associated with exposure in the NCICAS, but the increased risk was seen with kitchen, rather than bedroom, exposure.[8] In addition, mouse skin test sensitivity in the previous study was associated with exposure to kitchen Mus m 1 levels above the median level of 1.6 μg/g, whereas in the current study, the risk appeared to increase at exposure levels as low as 2.2 ng/g. The precise nature of the exposure-response relationship could have differed between the 2 studies for several reasons. For example, allergic sensitization may occur at low levels of exposure, so that exposure-response relationships may be more difficult to detect in study populations, such as NCICAS, with relatively uniform and high levels of exposure. In the current study population, exposure was both highly variable and relatively low compared with that in the NCICAS population, thus providing better conditions for discerning an exposure-response relationship. Bedroom Mus m 1 exposure may have been important in the current study population for similar reasons: exposure is relatively low, and exposure in the room where participants spend the most time may affect the risk of allergic sensitization the most. Despite some differences in the details of the exposure-response relationship, both studies found an increased risk of mouse skin test sensitivity associated with exposure.

A similar increase in prevalence rates of mouse skin test sensitivity with increasing bedroom Mus m 1 exposure was seen in the suburban group, although the trend was not statistically significant. On the other hand, no dose-dependent increase in prevalence of mouse skin test sensitivity was found in the city subgroup, likely because of the skewed distribution of bedroom Mus m 1 levels. The lowest exposure group, for example, consisted only of 5 participants, none of whom had a positive mouse skin prick test. Only 4 participants were exposed to 2.2 to 21.99 ng/g Mus m 1, and 1 had a positive mouse skin prick test. It is therefore difficult to draw any firm conclusions regarding the exposure-response relationship for the city group.

Although these findings suggest an increasing dose-response relationship between mouse allergen exposure and risk of mouse skin test sensitivity, it remains unknown whether this quantity of exposure is related to allergic symptoms among subjects who demonstrate skin test sensitivity. Certainly among those with mouse skin test sensitivity in the NCICAS, measures of asthma morbidity were not significantly related to settled dust concentrations of Mus m 1, which were, in general, a log higher than those measured in the current study.[8] Further studies are needed to develop a better understanding of the relationship between mouse allergen exposure and allergic symptoms.

Another striking finding was the strong association between mouse and dog skin test sensitivities. Having a positive skin prick test to dog carried a >7-fold increase in the risk of having a positive mouse skin prick test, even after adjusting for atopy. A positive skin prick test to other

indoor allergens, including cockroach, cat, and dust mite, was not independently associated with mouse skin test sensitivity. Furthermore, the association between dog and mouse skin test sensitivities did not appear to be explained by a relationship between mouse and dog exposure, because these respective allergen levels were not correlated. Interestingly, pet allergy has been identified as a risk factor for occupational rodent allergy, and furthermore, dog skin test sensitivity in particular was associated with mouse skin test sensitivity.[2] In this cross-sectional study of laboratory animal workers, 60% of those with mouse skin test sensitivity also had a positive skin prick test to dog, compared with 35% who also had a positive skin prick test to cat. In the final analysis, however, dog and cat skin test sensitivity were combined and evaluated as 1 risk factor, making it difficult to interpret the risk attributable to dog skin test sensitivity alone.

The reasons for the association between mouse and dog skin test sensitivity remain unclear, but there are several possible explanations. For example, cross-reactivity between dog and mouse albumins has been demonstrated, and an estimated 35% of persons with dog allergy have IgE specific for dog albumin, suggesting that as many as 35% of persons with dog allergy may have IgE that cross-reacts with mouse albumin, potentially resulting in a positive skin prick test to mouse epithelial extract.[16] However, it is less clear what proportion of mouse allergic persons have IgE specific for mouse albumin. In addition, Can f 1, Can f 2, and Mus m 1 are all lipocalins, and cross-reactivity between these proteins cannot be excluded as a possible explanation. However, cross-reactivity between these proteins has not been demonstrated, and there is less homology between these major allergens than between dog and mouse albumins,[17,18] making this explanation less likely. Or perhaps the association between dog and mouse allergic sensitization reflects a genetic predisposition to mount an allergic response to these particular allergens, but risk factors such as HLA associations have not yet been examined.

In conclusion, mouse allergen exposure and mouse sensitivity are surprisingly common even outside the inner city. Mouse allergen exposure and sensitivity should therefore be considered in the evaluation of all asthmatic children. Furthermore, dog skin test sensitivity appears to be a very strong and independent risk factor for mouse skin test sensitivity. Further studies are needed to elucidate the immunologic and/or genetic mechanisms behind this association. The high prevalence rates of exposure and common occurrence of mouse skin test sensitivity are compelling reasons to conduct further studies to examine the effect of mouse allergen exposure on asthma morbidity.

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