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Mouse sensitivity is an independent risk factor for rhinitis in children with asthma

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Abstract

Background—Although mouse and cockroach allergy are known to be important in urban children with asthma, the independent association of mouse and cockroach sensitization with rhinitis in these children is unknown.

Objective—To determine the association of mouse and cockroach sensitization with rhinitis in urban children with asthma.

Methods—As part of the Mouse Allergen and Asthma Intervention Trial, 499 urban children (5–17yr) with persistent asthma underwent spirometry, skin prick testing to 14 common environmental allergens, serology for mouse-specific IgE. In 269 subjects, cockroach-specific IgE serology was also obtained. Patient/parent-reported rhinitis in the last two weeks and one year were the primary outcome measures. Mouse/cockroach exposure was measured by reported

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frequency of sightings. Mouse allergen settled bedroom dust samples were also measured in mouse-sensitized children.

Results—Rhinitis was reported in 49.9% and 70.2% of participants within the last 2 weeks and last one year, respectively. Serum mouse IgE level ≥0.35IU/mL was associated with rhinitis in the past two weeks (OR_{adj}=2.15, 95%CI= 1.02–4.54, P=0.04) and the past year (OR_{adj}=2.40, 95%CI =1.12–5.1, P=0.02) after controlling for age, race, gender, the presence of any smokers at home, primary caregiver education level, number of allergen sensitivities, cockroach IgE level ≥0.35IU/mL and study site (Boston or Baltimore). Measures of home mouse exposure were not associated with rhinitis, regardless of mouse sensitivity. Cockroach sensitivity was not associated with rhinitis regardless of sensitization to other allergens.

Conclusions—In urban asthmatic children, increased mouse-, but not cockroach-, IgE in the sera (mouse IgE ≥0.35IU/mL) may be associated independently with rhinitis.

Keywords

mouse; hypersensitivity; rhinitis; asthma; pediatric; immunoglobulin E; risk factor

Introduction

There exists a close epidemiologic link between allergic rhinitis (AR) and asthma.[1] Approximately 40% of children with AR carry a concurrent diagnosis of asthma and 60 to 80% of children with asthma may have AR.[2–5] In addition to the direct impact on a child's health and quality of life,[2,6] AR is associated with poorly controlled asthma in children.[7–9] Asthma exacerbations and asthma-related emergency department (ED) visits also are associated with AR[10] and treatment of AR in individuals with asthma is associated with reductions in asthmarelated hospital admissions and ED visits.[11–13]

Low-income, urban minority children with asthma have a disproportionately high burden of asthma morbidity, which may be related to factors associated with poverty such as poor housing conditions and air pollution.[14] It is possible that rhinitis plays a role in asthma-related morbidity in low-income, urban minority children as well. Greater insight into the poorly understood prevalence and triggers of rhinitis in this population could provide an important opportunity to target rhinitis as an approach to improve asthma control. Previous studies showed that allergies to mouse and cockroach, two of the most common indoor allergens,[15] are associated with worse asthma outcomes and are most likely major contributors to asthma morbidity in the urban pediatric population.[16,17] No study has assessed the relationship that sensitization to mouse and cockroach has with rhinitis in urban children with poorly controlled asthma. In this study, we hypothesized that sensitivity to mouse would be associated with the presence of rhinitis and that rhinitis may serve to mediate the association of mouse sensitivity with poor asthma outcomes in urban children. In this study, our primary objectives were to estimate the prevalence of rhinitis in a population of urban children and adolescents with uncontrolled asthma and to examine whether mouse allergen sensitization and exposure are associated with rhinitis, while controlling for cockroach sensitization and exposure, in our study population. Our secondary

objective was to examine whether rhinitis may serve as a mediator between mouse sensitivity and asthma outcomes in these children.

Materials and methods

Study population

Four hundred and ninety-nine children and adolescents with persistent asthma living in Boston and Baltimore (age 5–17) eligible for the Mouse Allergy and Asthma Intervention Trial (MAAIT) and screened for the trial were included in this study. MAAIT was approved by the institutional review boards of Boston Children's Hospital and Johns Hopkins Hospital. Assent and informed consent obtained from the participating children and their parents/guardians, respectively. MAAIT's primary purpose was to evaluate the role of a home-based mouse-allergen targeted environmental reduction strategy and reducing asthma morbidity in mouse allergen and home mouse-allergen exposed children. Major eligibility criteria for screening to be considered for randomization in MAAIT included age between 5 and 17 years, persistent asthma [defined by 1) utilization of a long-term controller medication or 2) asthma symptoms for 3 or more days per week over the past 2 weeks or 3) nocturnal asthma symptoms at least 3 times in the past month], which was considered to be uncontrolled [defined by 1) at least one asthma-related unscheduled visit to an ED, clinic or urgent care facility in the last year, 2) at least one asthma-related overnight hospitalization in the last year, or 3) one of more bursts of oral corticosteroids in the last year].[18] Among children who were screened for the parent study MAAIT, those who completed the home environmental questionnaire, skin testing, and measurement of mouse-specific IgE in the serum were included as the analysis population for this paper. 605 participants completed the home environmental questionnaire and 499 of these completed skin testing and had mouse-specific IgE measured in the serum. Exclusion criteria included pregnancy; lung disease (other than asthma) requiring daily medication; cardiovascular disease (not hypertension) requiring daily medication; current use of a beta blocker, omalizumab (anti-IgE therapy), allergen immunotherapy; and active smoking. Participant data including demographics, as well as clinical and exposure assessments from the baseline, screening visit were utilized for this study.

Clinical assessments

A questionnaire capturing medication use, asthma symptoms, and health care utilization was administered to the primary caregiver of children aged 5–11 and to both the study participant and the primary caregiver for adolescents aged 12–17. The questionnaire included questions used in many previous studies of low-income urban children with asthma and included whether the participant was currently using an asthma controller medication or required an asthma-related ED visit in the preceding year.[17,19] Asthma control was assessed using the Asthma Control Test (ACT). Rhinitis was assessed with questions derived from the International Study of Asthma and Allergies in Childhood (http://isaac.auckland.ac.nz/) questionnaire. Specifically, rhinitis was queried by asking whether the child had problems with sneezing, or runny or blocked nose when the child did not have a cold or flu in the last two weeks and in the last year.

Allergy testing by skin prick testing (SPT) was performed to 14 allergens in all children including dog, cat, dust mites (*Dermatophagoides pteronyssinus* and *Dermatophagoides farinae*), rat epithelia, German cockroach, American cockroach, mouse epithelia, tree mix (including American Elm, American Beech, Eastern Cottonwood, Red Oak, River Birch, Hickory Shagbark, and White Ash trees), grass mix (Kentucky Blue/June, Orchard, and Timothy grasses), Alternaria, Aspergillus, common ragweed, and Cladosporium (Greer Laboratories, Lenoir, NC). Positive histamine and negative saline controls were used in all cases. Wheal diameter was measured 15 minutes after the SPT was placed and a wheal diameter ≥3 mm larger than the negative control was considered positive. All children also had serum mouse urine-specific IgE levels with the mouse urine CAP (e72; Phadia ImmunoCAP, Thermo Fisher Scientific) measured, with a mouse urine-specific IgE level ≥0.35 IU/mL considered to be positive for sensitivity. Mouse sensitization was determined by either positive skin or serological testing. Children who tested positive for mouse sensitivity had serum IgE levels to German cockroach allergens determined as well (i6; Phadia ImmunoCAP, Thermo Fisher Scientific), with a serum specific IgE level ≥0.35 IU/mL considered to be positive. Pulmonary function testing was performed as well at the baseline visit according to American Thoracic Society guidelines.[20,21]

Exposure assessments

The frequency of mouse and cockroach sightings was queried on the administered questionnaires as 1) less than once per week, 2) once to three times per week, 3) four to six times per week, 4) once a day, or 5) more than once per day. In children with mouse sensitivity, defined as either a mouse SPT net wheal of ≥3mm or mouse urine-specific IgE of ≥0.35kU/L, Mus m 1 was measured by ELISA in dust collected from the participant's bed and bedroom floor.

Statistical analyses

All statistical analyses were performed with the statistical computing program R.[22] Univariate and multivariate logistic regression was performed with the Regression Modeling Strategies package[23] to identify association of mouse and cockroach sensitivity and exposure as independent variables with rhinitis as the dependent variable. Final multivariate models controlled for covariates of age, race, gender, the presence of any smokers at home, education level of primary caregiver, number of aeroallergen sensitivities, and study site. Multivariate models for association between rhinitis and the independent variable serological sensitivity also controlled for both mouse and cockroach IgE levels ≥0.35 IU/mL. Univariate and multivariate logistic regressions were likewise performed to identify association between mouse and cockroach sightings as the independent variables and rhinitis as the dependent variable. The final multivariate models for this analysis controlled for covariates age, race, gender, the presence of any smokers at home, education level of primary caregiver, number of aeroallergen sensitivities, mouse or cockroach sensitivity (corresponding to the type of sighting being analyzed) and study site. Associations were evaluated by performing regression modeling of the relationship between the independent variable of mouse sensitivity by serology and clinical features of asthma as the dependent variables. Linear regression was used for the following dependent, or outcome, variables: FEV1/FVC, percent predicted FEV1, percent predicted FEF25–75, and logistic regression

was performed for the following dichotomous dependent, or outcome, variables: ACT score ≤ 19, use of a controller medication (yes vs. no), and use of a long-acting beta-agonist (yes vs. no). Mediation was tested for using the Sobel-Goodman test from the *bda* package.[24] A P-value < 0.05 is considered statistically significant.

Results

Participant characteristics

A total of 605 participants in MAAIT completed the home environmental questionnaire and 499 of these participants met inclusion criteria for our study by completing skin testing and having mouse-specific IgE measured in the serum. The demographic characteristics of these 499 children with asthma, comprising our study population, are summarized in Table 1. Over three quarters of our study participants were black, 68.8% of children lived in a family with annual income less than $30,000, 83.0% of children had public health insurance (e.g. Medicaid) and 52.1% of children's primary guardians reported an educational attainment of high school or less. Although the minority of children had reported exposure to either tobacco smoke (37.4%) or furred pets (38.4%) at home, 72.1% of primary caregivers reported seeing mice in their home at least once in the previous month. In contrast, only 34.0% of primary caregivers reported seeing cockroaches in their home at least once in the previous month. Of those children living in homes where either mice or cockroaches were seen within the previous month, 23.4% and 20.6% reported seeing mice and cockroaches, respectively, at least once per day (Table 2).

The atopic and asthma characteristics of the study population are described in Table 3. Most participants had uncontrolled asthma as reflected by the ACT scores (52.4% with ACT score ≤ 19) and 77.7% reporting ED use for asthma in the last year. The majority (88.6%) of these children were also atopic, with a mean of approximately 5 aeroallergen sensitivities. Mouse sensitivity was detected in 65.7% of children and cockroach sensitivity was detected in 68.3% of children. Of note, almost half of these children (51.3%) experienced rhinitis within the last two weeks and 71.3% experienced rhinitis within the last year. In both cases, the vast majority of the children had detectable aeroallergen sensitivities (Table 3). In comparing the 499 MAAIT participants who were included in our study to the 106 MAAIT participants who were excluded, there were no statistically significant differences with respect to age (P=0.34), gender (P=0.51), race (P=0.07), ethnicity (P=0.80), income (P=0.14), insurance (P=0.79), education level of the guardian (P=0.76), prevalence of rhinitis in the past 2 weeks (P=0.16), and prevalence of rhinitis in the past one year (P=0.20) (data not shown).

Associations with rhinitis

We assessed whether mouse or cockroach exposure and sensitivity were associated with rhinitis in the study population. Sensitivities detected by SPT were considered separately from sensitivities detected by serology. Although a negative association was detected between cockroach sensitivity by SPT and rhinitis within the past year on univariate regression (odds ratio [OR] =0.64, 95%CI: 0.43–0.94, P=0.02), this association was no longer statistically significant after adjusting for potential confounders (OR_{adj}=0.60, 95%CI:

0.37–1.01, P=0.05). There were likewise no other statistically significant associations between mouse or cockroach sensitivities by SPT and rhinitis (Table 4). In contrast, mouse sensitivity by serology (IgE+, mouse-specific IgE ≥0.35 IU/mL) was associated with rhinitis in the past two weeks (OR_{adj}=2.15, 95%CI: 1.02– 4.54, P=0.04) and within the past year (OR_{adj}=2.40, 95%CI: 1.12–5.12, P=0.02) (Table 4). Adjusting for cockroach skin test sensitivity yielded similar results as those shown in Table 4, which adjusted for cockroach-specific IgE (rhinitis past two weeks: OR_{adj}=1.43, 95%CI: 0.92– 2.22, P = 0.11; rhinitis in the past year: OR_{adj}=1.82, 95%CI: 1.12–2.95, P=0.02).

In order to better understand the association between rhinitis and mouse sensitivity, we derived point estimates for the associations between rhinitis and mouse sensitivity defined as (1) SPT+IgE-, (2) SPT+IgE+ or (3) SPT-IgE+. Consistent with the results in Table 4, point estimates for the association between rhinitis in the last two weeks and mouse sensitivity defined as SPT+IgE-(unadjusted OR=0.62, 95%CI: 0.37–1.05, P=0.07) did not indicate a positive association. This was similarly the case for the association between rhinitis in the last year and SPT+IgE- mouse sensitivity (unadjusted OR=0.56, 95%CI: 0.33–0.95, P=0.03). For mouse-specific IgE+ children, the point estimate for the association between rhinitis in the last two weeks was stronger for mouse sensitivity defined as SPT+IgE+ (unadjusted OR=1.40, 95%CI: 0.98–1.99, P=0.06) than SPT-IgE+ (unadjusted OR=1.25, 95%CI: 0.54–2.90, P=0.61). This was similarly true for the association between rhinitis in the last year and mouse sensitivity (unadjusted OR=1.48, 95%CI: 1.00–2.20, P=0.05 for SPT+IgE+; unadjusted OR=1.14, 95%CI: 0.44–2.97, P=0.78 for SPT-IgE+). These results suggest that the association between mouse sensitivity by serology is driven in large part by study subjects who are positive for mouse sensitivity by both SPT and serology.

Cockroach sensitivity by serology, however, was not associated with rhinitis (Table 4). In order to determine whether a dose-response relationship existed between mouse-specific IgE level and the prevalence of rhinitis, the association with rhinitis was also examined with mouse-specific IgE level (mean: 15.4 IU/mL, range: 0.05–101.0 IU/mL) as a continuous variable. However, no statistically significant association was found between mouse IgE level and rhinitis within the last two weeks (OR=1.00, 95%CI: 0.99–1.00, P=0.41) and rhinitis within the last one year (OR=1.00, 95%CI: 1.00–1.01, P=0.41). Similar results were found for the association between rhinitis and log_{10}-transformed mouse IgE levels.

Exposures to mouse and cockroach were first assessed as home sightings within the past month. The frequencies of mouse and cockroach home sightings within the past month, considered as a binary variable (yes/no), were not significantly associated with rhinitis within either the past two weeks or the past year (Table 5). The frequencies of mouse and cockroach home sightings, considered as ordered or categorical variables (as previously shown in Table 2), were likewise not significantly associated with rhinitis. Study participants were stratified by mouse sensitivity and no statistically significant association was found between mouse home sightings and rhinitis among either participants with mouse sensitivity or those without mouse sensitivity (data not shown). Study participants were likewise stratified by cockroach sensitivity and no statistically significant association was found between cockroach home sightings and rhinitis among either participants with cockroach sensitivity or those without cockroach sensitivity (data not shown).

Mus m 1 levels in bed and bedroom floor dust were measured in children with mouse sensitivity. In those children, Mus m 1 levels from neither the bed nor the bedroom floor dust were associated with rhinitis (P≥0.56, Table E1 in the online repository). This was also the case when considering Mus m 1 and $\log_{10}$-transformed Mus m 1 levels, as well as using cut-offs of Mus m 1 levels to test for associations with rhinitis (P≥0.09, Table E1 in the online repository).

Relationship between mouse sensitivity, rhinitis and clinical features of asthma

In our cohort, serologic mouse sensitivity was associated with worse pulmonary function, which reached significance for overall FEV1/FVC spirometry values (unadjusted β = −0.017, 95%CI: −0.002 – −0.033, P = 0.02) but not for having FEV1/FVC less than 80% (unadjusted OR = 1.37, 95%CI: 0.94 – 1.99, P = 0.10). Mouse sensitivity by serology was also not significantly associated with percent predicted FEV1 (unadjusted β = −0.024, 95%CI: −0.055 – 0.008, P = 0.14) or percent predicted $FEF_{25–75}$ (unadjusted β = −0.056, 95%CI: −0.114 – 0.003, P = 0.06). Although no statistically significant association was found between serologic mouse sensitivity and poor asthma control, defined by ACT score ≤ 19 (OR = 1.30, 95%CI: 0.91 – 1.86, P = 0.15), serologic mouse sensitivity was associated with the use of a controller medication (OR = 1.93, 95%CI: 1.21 – 3.08, P = 0.01) or a long-acting beta-agonist (OR = 1.77, 95%CI: 1.13 – 2.77, P = 0.01), alone or in combination with an inhaled corticosteroid, in the last two weeks. We checked for mediation by rhinitis within the last two weeks in the significant associations between serologic mouse sensitivity and FEV1/FVC spirometry values, use of controller medication and use of long-acting beta-agonist medication but found no statistically significant evidence of mediation (P = 0.75 for FEV1/FVC, P = 0.23 for use of controller medication, P = 0.33 for use of long-acting beta-agonists). There was likewise no significant evidence of mediation by rhinitis in the last year in the association of serologic mouse sensitivity and FEV1/FVC (P = 0.51), use of controller medication (P = 0.97) and use of long-acting beta-agonists (P = 0.15).

Discussion

Mouse allergen exposure and sensitivity have been described as major determinants of poor asthma outcomes for children living in urban settings.[17,25] Although AR has been shown to be associated with increased morbidity in children with asthma,[7–10] the relationship between mouse allergy and rhinitis in urban children with asthma has not been previously described. Here we show that mouse sensitization by positive serological testing, a marker for mouse exposure and sensitization,[26] is associated with rhinitis in a cohort of urban children with comorbid asthma. This effect was independent of cockroach sensitivity, which was not associated with rhinitis in these children.

The prevalence of AR in urban asthmatics is not well characterized. In our cohort of asthmatic children, whose demographic characteristics largely reflect that of lower socioeconomic classes, we find a prevalence of rhinitis of 51.3% within the last 2 weeks and 71.3% in the last year, which is consistent with previous studies of asthmatic children.[2–5] Previous work has highlighted a similarly high prevalence of AR in the urban pediatric

population, with underserved children at higher risk for undiagnosed AR[27] and up to two thirds of these children reportedly receiving inadequate or no treatment for AR.[28]

Mouse and cockroach allergens have been previously reported to be the two most abundant aeroallergens in many urban communities[15] and our results here, identifying mouse sensitivity by serology as the dominant association with active rhinitis is also consistent with previous work reporting the dominance of mouse over cockroach allergy in asthma outcomes in a similar cohort of urban children.[17] The association between rhinitis and serologic mouse sensitivity was driven largely by children who were positive for mouse sensitivity by both serology and SPT. In contrast, we did not find association between mouse sensitivity defined only by SPT and rhinitis in our cohort of urban children with asthma. In fact, we found a negative association between patient-reported rhinitis within the past year and mouse sensitivity defined as having a positive SPT but not a positive specific IgE test. Although it is unclear why we found this negative association, it is possible that this finding reflects differences between the source of the extract for skin testing and IgE testing, respectively. The skin testing was performed with mouse epithelial extract and the specific IgE test was performed with the mouse urine CAP, so it is possible that those with a positive skin test but a negative specific IgE test were sensitized to mouse epithelial proteins and not mouse urinary proteins and that sensitization to mouse epithelial proteins are less clinically relevant than sensitization to mouse urinary proteins. In fact, the major mouse allergen, Mus m 1, is a mouse urinary protein. Although these differences in the extracts used for skin vs. serum IgE testing might explain an absence of a relationship between mouse epithelial sensitization and rhinitis, they would not explain a protective association as we observed.

Our results are also consistent with previous work showing association between serologic mouse sensitization and development of rhinitis by three years of age in urban children.[29] Mouse sensitivity by serology, which we have found to be associated with rhinitis, has previously been shown to be a reflection of both sensitivity and exposure.[26] Previous work has also found that mouse allergen exposure was associated with a decreased likelihood for development of AR in a cohort of school-aged Puerto Rican children but that this effect was primarily derived from children who were not sensitized to mouse.[30] It has been hypothesized that exposure to either mouse allergen or associated microbial elements may be protective against development of AR in children who are not sensitized to mouse.[30] In our study, we found no association between parent-reported frequency of home mouse sightings with rhinitis. This was true in both children who were and were not sensitized to mouse. We found a similar lack of association between levels of Mus m 1 in bedroom dust and rhinitis in mouse-sensitized children. That we have not found a relationship between mouse exposure and rhinitis in mouse-sensitized patient may be due to several reasons. It is possible that our method for detecting rhinitis is not sensitive enough. Additionally, it is possible that major sources of mouse exposure for these children also may be in environments outside of the home. Previous work has shown that allergen levels of both mouse and cockroach are elevated not only in urban homes but also in other urban locales such as schools.[31]

We also did not find any association between cockroach sensitivity or exposure and rhinitis in our study population. Previous multi-center research has highlighted the association between cockroach sensitivity and exposure with the worst asthma outcomes of urban children; cockroach allergen levels were found to be highest in Boston and Baltimore, with both of these cities comprising our study sites.[16] That we did not find an association between rhinitis and cockroach sensitivity or exposure in these children mirrors previous work showing the significance of mouse over cockroach allergy in determining asthma outcomes in children at one of our sites (Baltimore, MD, USA).[17]

Finally, as expected[17] we did find association between mouse sensitivity by serology and measures indicative of asthma severity, including FEV1/FVC, use of a controller medication, and use of a long-acting beta agonist medication (alone or in combination with an inhaled corticosteroid). However, we did not find rhinitis to serve as a mediator for the associations between mouse sensitivity by serology and these clinical features of asthma reflecting severity. The “unified airway hypothesis”, which references the common pathophysiologic processes linking upper and lower airway disease,[32–35] suggests that rhinitis may mediate comorbid asthma. That we did not find a significant mediator effect for rhinitis between mouse sensitivity and clinical features of asthma may be related to limitations of our study described below. Moreover, our study cohort represents the subset of asthmatic children with uncontrolled asthma; it is also possible that in these children, rhinitis does not modify already uncontrolled asthma. As rhinitis has been shown to have a substantial quality of life impact on asthmatic children,[36] our results nonetheless elucidate novel associations with rhinitis that may lead to quality of life detriments in these urban children with uncontrolled asthma.

This study was limited in several respects. We are unable take into account the severity of rhinitis (e.g. frequency and severity of symptoms or quality of life impact) but rather consider rhinitis as a binary variable (yes or no). It is possible that more subtle relationships exist between sensitivity and exposure to mouse and cockroach and rhinitis. That we are not able to more specifically classify rhinitis by severity in these children prevents us from gaining greater insight into the relationship between sensitivities and exposure with rhinitis. We likewise are unable to control for use of AR medications, such as anti-histamines or intranasal corticosteroids, in our study participants. Because use of these medications may modify the reported presence of rhinitis symptoms, the lack of AR medication usage limits interpretation of our results. However, we expect that this confounder would mostly affect reported rhinitis symptoms within the prior two weeks. We expect that children with AR would likely have some AR symptomatology within the last year, regardless of medication use.

Our study identifies the dominance of mouse over cockroach sensitization for rhinitis in our urban population of children with uncontrolled asthma. The National Allergy Education and Prevention Program guidelines recommend evaluation for potential allergen triggers in children with asthma and our results support the importance of testing for allergen sensitization. Since serologic testing for environmental allergens is readily available, general healthcare providers should consider screening their patients with asthma to a limited panel including mouse allergen (in particular for patients living in urban settings). Referral to an

allergy specialist for additional testing and management should be considered for those with uncontrolled asthma as the guidelines suggest.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Abbreviations

AR	allergic rhinitis
ED	emergency department
MAAIT	Mouse Allergen and Asthma Intervention Trial
IgE	immunoglobulin E
SPT	skin prick testing
IU/mL	international unit per milliliter
FEV1	forced expiratory volume in one second
FVC	forced vital capacity
$FEF_{25–75}$	forced expiratory flow at 25 – 75%
ACT	asthma control test
OR	odds ratio
OR_{adj}	adjusted odds ratio
95%CI	95% confidence interval

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Highlights

What is already known about this topic?

Rhinitis has a significant impact on the quality of life of asthmatic children. Mouse and cockroach sensitivity are important modifiers of asthma in urban children.

What does this article add to our knowledge?

Mouse sensitivity as detected by serology is independently predictive of rhinitis in urban children with poorly-controlled asthma. In contrast, cockroach sensitivity is not associated with rhinitis in these children.

How does this study affect current management guidelines?

Assessment of mouse sensitization and implementation of environmental controls may have a positive impact in urban children with poorly controlled asthma, particularly in those with co-morbid rhinitis.

Table 1

Demographic characteristics of study subjects (n = 499)

Characteristic	
Age, mean (SD)	10.2 years (3.1 years)
Gender	
Male	61.3%
Female	38.7%
Race	
Black	78.2%
Non-black	21.8%
Ethnicity	
Hispanic	21.7%
Non-hispanic	78.3%
Annual family income	
<$30,000	68.8%
≥$30,000	31.2%
Insurance	
Public	83.0%
Private	17.0%
Highest education of primary guardian	
High school graduate or less	52.1%
Some college or 2-year college/technical school graduate	37.3%
4-year college graduate and beyond	10.6%
Home environment	
Smoker in the home	37.4%
Primary guardian is a smoker	25.3%
Other smokers in the home	24.2%
Furred pets	38.4%
Cat	20.6%
Dog	21.2%
Mice seen in the last month	72.1%
Cockroaches seen in the last month	34.0%

Table 2

Frequency of home mouse and cockroach sightings, when seen in the last month

Frequency	Mouse	Cockroach
Less than once per week (rarely)	24.2%	29.7%
Once to three times per week (some days)	24.6%	27.3%
Four to six times per week (most days)	7.0%	4.8%
Once a day	20.7%	17.6%
More than once per day	23.4%	20.6%

Table 3

Asthma and atopic characteristics

Characteristics	
Asthma parameters	
Uncontrolled[1]	52.4%
FEV1 percent predicted, mean (SD)[2]	90.5% (15.9%)
FEV1/FVC, mean (SD)	81.3% (8.5%)
$FEF_{25–75}$ percent predicted, mean (SD)	85.0% (32.1%)
Used ED for asthma in the last year	77.7%
On controller medication	82.0%
Aeroallergen skin prick test (SPT) sensitivity	
One or more positive SPT	88.6%
Grass	53.1%
Cat	48.1%
Tree	44.9%
Mold	43.0%
Dust mite	41.9%
Ragweed	30.3%
Dog	20.4%
Mouse sensitivity	
SPT positivity	61.1%
Specific IgE positivity[3]	52.1%
SPT or specific IgE	65.7%
Cockroach sensitivity	
SPT positivity	42.5%
Specific IgE positivity[3]	56.8%
SPT or specific IgE	68.3%
Total number of sensitivities[4], mean (SD)	5.1 (3.5)
Reported rhinitis prevalence	
In the last 2 weeks	51.3%
Fraction of the above with at least one sensitivity	89.8%
In the last 1 year	71.3%
Fraction of the above with at least one sensitivity	89.9%

[1] Asthma Control Test (ACT) score ≤ 19

[2] For participants age 8 years and older

[3] specific IgE level ≥ 0.35 IU/mL

[4] Based on SPT

Table 4

Associations between sensitization and rhinitis

	Univariate OR for rhinitis	P value	Multivariate OR for rhinitis	P value
Mouse SPT positive (N = 499)				
Rhinitis in last 2 weeks	1.13 (0.79 – 1.62)	0.52	1.05[1] (0.65 – 1.70)	0.83
Rhinitis in last year	1.10 (0.74 – 1.64)	0.63	1.22[1] (0.72 – 2.07)	0.46
Roach SPT positive (N = 499)				
Rhinitis in last 2 weeks	0.85 (0.60 – 1.22)	0.39	0.82[2] (0.52 – 1.29)	0.38
Rhinitis in last year	**0.64 (0.43 – 0.94)**	**0.02**	0.60[2] (0.37 – 1.01)	0.05
Mouse IgE ≥ 0.35 (N = 499)				
Rhinitis in last 2 weeks	**1.45 (1.02 – 2.06)**	**0.04**	**2.15[3] (1.02 – 4.54)**	**0.04**
Rhinitis in last year	**1.51 (1.02 – 2.23)**	**0.04**	**2.40[3] (1.12 – 5.12)**	**0.02**
Roach IgE ≥ 0.35 (N = 269)				
Rhinitis in last 2 weeks	0.93 (0.55 – 1.57)	0.78	1.25[4] (0.68 – 2.31)	0.48
Rhinitis in last year	0.61 (0.34 – 1.11)	0.11	0.72[4] (0.36 – 1.43)	0.34

[1]Controlling for age, race, gender, the presence of any smokers at home, education level of primary caregiver, number of sensitivities (excluding mouse), roach sensitivity by SPT, and study site

[2]Controlling for age, race, gender, the presence of any smokers at home, education level of primary caregiver, number of sensitivities (excluding roach), mouse sensitivity by SPT,and study site

Abbreviations: SPT = skin prick testing, OR = odds ratio

[3]Controlling for age, race, gender, the presence of any smokers at home, education level of primary caregiver, number of sensitivities (excluding mouse), roach IgE level ≥ 0.35IU/mL (for serological associations), and study site

[4]Controlling for age, race, gender, the presence of any smokers at home, education level of primary caregiver, number of sensitivities (excluding roach), mouse IgE level ≥ 0.35IU/mL (for serological associations),and study site

Abbreviations: SPT = skin prick testing, OR = odds ratio

Table 5

Associations between home mouse and cockroach sightings in the last month and rhinitis

	Univariate OR for rhinitis	P value	Multivariate OR for rhinitis[1]	P value
Mouse home sightings (yes or no)				
Rhinitis in last 2 weeks	0.75 (0.47 – 1.19)	0.22	0.80 (0.46 – 1.37)	0.41
Rhinitis in last year	0.87 (0.52 – 1.46)	0.60	1.16 (0.63 – 2.14)	0.64
Cockroach home sightings (yes or no)				
Rhinitis in last 2 weeks	1.05 (0.72 – 1.53)	0.81	1.14 (0.73 – 1.77)	0.56
Rhinitis in last year	0.97 (0.63 – 1.48)	0.88	1.11 (0.69 – 1.80)	0.66

[1] Controlling for age, race, gender, the presence of any smokers at home, education level of primary caregiver, number of sensitivities, mouse sensitivity (by SPT or serology), roach sensitivity (by SPT or serology), and study site