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

Elizabeth C. Matsui, MD, MHS,[a] Elinor Simons, MD,[a] Cynthia Rand, PhD,[a] Arlene Butz, RN, ScD,[a] Timothy J. Buckley, PhD,[b] Patrick Breysse, PhD,[b] and Peyton A. Eggleston, MD[a] *Baltimore, Md*

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.
(J Allergy Clin Immunol 2005;115:358-63.)

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.

From [a]the Johns Hopkins University School of Medicine, and the [b]Johns Hopkins University Bloomberg School of Public Health.
Supported by grants from the US Environmental Protection Agency (R82672401), the National Institute of Environmental Health Sciences (ES09606), and the National Heart, Lung, and Blood Institute (HL058942). This research has been supported by a grant from the US Environmental Protection Agency's Science to Achieve Results program.
Received for publication August 25, 2004; revised October 27, 2004; accepted for publication November 1, 2004.
Available online January 10, 2005.
Reprint requests: Elizabeth C. Matsui, MD, MHS, Department of Pediatrics, Johns Hopkins Hospital, 600 N Wolfe St, CMSC 1102, Baltimore, MD 21287. E-mail: ematsui@jhmi.edu.
0091-6749/$30.00

doi:10.1016/j.jaci.2004.11.007

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