

which evaluated indoor and outdoor allergens, sensitization to cockroach, cat, dog, and mite allergens was higher in women of low socioeconomic status and was independently associated with asthma.^{9,10}

Our finding of the interaction between mouse sensitization and asthma by race/ethnicity was independent of measured indices of poverty (which are correlated with race/ethnicity). Black and Hispanic women are sensitized to more allergens than white women in our cohort. Because sensitization to multiple allergens is a risk factor for asthma morbidity, the unique contribution of mouse allergen sensitization to the risk of morbidity among minority women may consequently be smaller for minority women than for white women.

This study is limited in that it may be generalizable only to women of childbearing age from the urban United States. It is also limited by the absence of mouse allergen data and by the absence of additional information to confirm the diagnosis of asthma. However, this study is relevant to a large, at-risk portion of the population of women of childbearing age.

In summary, regardless of race, ethnicity, or socioeconomic status, women sensitized to mouse allergen have higher rates of a physician's diagnosis of asthma and asthma morbidity. Although the effect of mouse allergen sensitization on asthma and asthma morbidity was somewhat lower in minorities, mouse sensitization was present in a higher proportion of the minority population. Thus, the population at risk for mouse allergy-associated asthma may be greater among the African American and Hispanic compared with the non-Hispanic white women of childbearing age. Exposure patterns and other factors associated with the sensitization to mouse allergen should be examined more closely in the future.

Wanda Phipatanakul, MD, MS^{a,b,c}

Augusto A. Litonjua, MD, MPH^{b,c}

Thomas A. E. Platts-Mills, MD, PhD^d

Lisa M. Naccara, BA^d

Juan C. Celedon, MD, DrPH^{b,c}

Hassen Abdulkarim, MS^{b,c}

Elaine B. Hoffman, PhD^e

Diane R. Gold, MD, MPH^{b,c}

From ^athe Department of Pediatrics, Division of Allergy and Immunology, Children's Hospital, Boston, Mass; ^bthe Channing Laboratory, Department of Medicine, Brigham and Women's Hospital, Boston, Mass; ^cHarvard Medical School, Boston, Mass; ^dthe Asthma and Allergic Diseases Center, University of Virginia, Charlottesville, Va; and ^ethe Department of Biostatistics, Harvard School of Public Health, Boston, Mass. E-mail: wanda.phipatanakul@childrens.harvard.edu.

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Mouse allergen levels vary over time in inner-city homes

To the Editor:

Mouse allergen is almost ubiquitous in inner-city US homes¹⁻³ and can be found in the majority of suburban US homes.^{4,5} Exposure to mouse allergen has been linked to allergic sensitization to mouse⁶ and asthma morbidity among inner-city children with asthma who are sensitized to mouse.² Even though there is growing evidence that home exposure to mouse allergen is associated with adverse health outcomes, little is known about the temporal variability of mouse allergen levels. A better understanding of the temporal variability of mouse allergen levels is needed to understand how changing exposure may impact asthma symptoms over time and to optimize abatement strategies. The primary objective of this study was to describe the variability of mouse allergen levels over time within a home, as well as to identify time-dependent predictors of home mouse allergen levels. We assessed settled dust and airborne Mus m 1 levels in the bedroom, along with household and environmental characteristics, at baseline and 3 and 6 months in a population of 127 inner-city preschool children with asthma. Air samples for mouse allergen analysis were collected on 25-mm, 0.3- μ m-pore polytetrafluoroethylene membrane filters using IOM Inhalable Dust Samplers (SKC, Inc, Eighty Four, Pa) at a flow rate of 2 L/min for 72 hours. Home dust

samples were collected from the child's bedroom, television-living room, and kitchen at baseline and 3 and 6 months by using published methods.⁷ 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 immunosorbent purified sheep anti-Mus m 1 (kindly supplied by Dr J Ohman).⁸ 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. The study was approved by the Johns Hopkins Institutional Review Board. A more detailed description of the methods and characteristics of the study population is available elsewhere.²

The distributions of settled dust and airborne Mus m 1 levels among the population as a whole were similar at the baseline, 3-month, and 6-month study visits (Table I). However, mouse allergen levels within any given home did not necessarily remain stable over time. Within homes, there was only a modest correlation between baseline mouse allergen levels and levels at future study visits. Specifically, both bedroom settled dust and airborne Mus m 1 levels at the baseline visit were moderately correlated with the respective levels at the 3 month visit (Spearman correlation coefficient [r_s] = 0.63, P < .0001; r_s = 0.60, P < .0001, respectively). However, the correlations between baseline and 6-month settled dust and airborne Mus m 1 levels were less strong (r_s = 0.48, P < .0001; r_s = 0.37, P = .008, respectively).

Because correlations between mouse allergen levels at different study visits were moderate rather than strong, almost 50% of homes had substantial variability in mouse allergen levels over a 3-month to 6-month period. For example, when ranked by tertiles, homes frequently moved from one tertile of mouse allergen level to another from one study visit to the next. By the 6-month study visit, more than 40% of the homes in the lowest tertile had increases in settled dust and airborne mouse allergen levels, resulting in a shift to either the middle or highest tertile of mouse allergen (Fig 1). Similarly, more than 40% of homes in the highest tertile at baseline had decreases in airborne and settled dust mouse allergen levels. Moreover, a substantial proportion of homes that had a bedroom settled dust mouse allergen level at baseline below the threshold that has been associated with asthma morbidity in this same population (0.5 $\mu\text{g/g}$) had an increase in mouse allergen to a concentration above 0.5 $\mu\text{g/g}$. By the 6-month study visit, 37% of the homes with a mouse allergen level below this threshold had a level above this threshold. The variability in home mouse allergen levels over time was similar when participants who had relocated were excluded from the analysis (data not shown). This marked variability in mouse allergen levels may explain some of the lability in asthma symptoms in mouse-sensitized patients.

Seasonal differences in mouse allergen levels were also observed. Settled dust mouse allergen levels were highest in the spring, whereas airborne levels were highest in fall

TABLE I. Distribution of mouse allergen levels in the study population

	Baseline, median (IQR)	3 Months, median (IQR)	6 Months, median (IQR)
Kitchen ($\mu\text{g/g}$)	7.4 (0.9-32.9)	3.5 (0.3-20.3)	6.5 (0.9-31.9)
TV room	2.3 (0.5-8.3)	1.9 (0.4-7.7)	2.2 (0.5-8.9)
Bedroom settled dust ($\mu\text{g/g}$)	2.5 (0.6-8.8)	2.3 (0.4-10.2)	2.9 (0.4-12.2)
Airborne (ng/m^3)	.017 (.003-.046)	.014 (BD to .050)	.017 (.004-.054)

BD, Below detection.

and winter. These seasonal differences remained in a multivariate model that used generalized estimating equations to account for repeated measures of mouse allergen, even after adjusting for humidity, acquisition of a cat, cracks/holes in walls or doors, and relocation (Fig 2). Although it is not entirely clear why the seasonal patterns of airborne versus settled dust Mus m 1 levels differ from one another, we speculate that this finding may be a result of characteristics of the allergen compartment being sampled. The settled dust allergen compartment functions as a reservoir, and reservoir allergen levels likely reflect the accumulation of mouse allergen over months to years. As a result, an increase in mouse infestation that may occur in fall and winter months may not manifest as an increase in settled dust allergen levels for several months. On the other hand, airborne allergen levels may fluctuate more rapidly over a period of hours to days, and may be more acutely sensitive to changes in mouse infestation than settled dust levels. In addition, other unmeasured factors such as ventilation, which is likely to be lower in fall and winter months, would have a greater effect on airborne particles than settled dust particles. The lower ventilation in colder months may translate into a measurable increase in airborne mouse allergen levels with minimal effect on settled dust allergen levels.

Even though only 8 homes acquired a cat between the baseline and 6-month study visits, acquisition of a pet cat was independently associated with a decrease in settled dust mouse allergen levels. Homes without a cat for the entire 6-month period ($n = 75$) had an increase in settled dust mouse allergen levels (absolute change in Mus m 1 [$\mu\text{g/g}$, median (interquartile range, IQR)]: 0.47 [−3.9 to 9.4]), whereas homes that acquired a cat had a 61% reduction in mouse allergen levels (absolute change [median (IQR)]: −1.39 [−92.8 to −0.03]; $P = .05$). In a multivariate model that accounted for potential confounders as well as repeated mouse allergen measures, the presence of a cat remained associated with significantly lower levels of both settled dust and airborne Mus m 1. Although similar trends were observed for airborne mouse allergen levels, the changes in airborne levels were not statistically significant.

Indoor relative humidity was measured using a HOBO temperature and humidity data logger (Onset Corp, Pocasset, Mass), and the mean relative humidity was

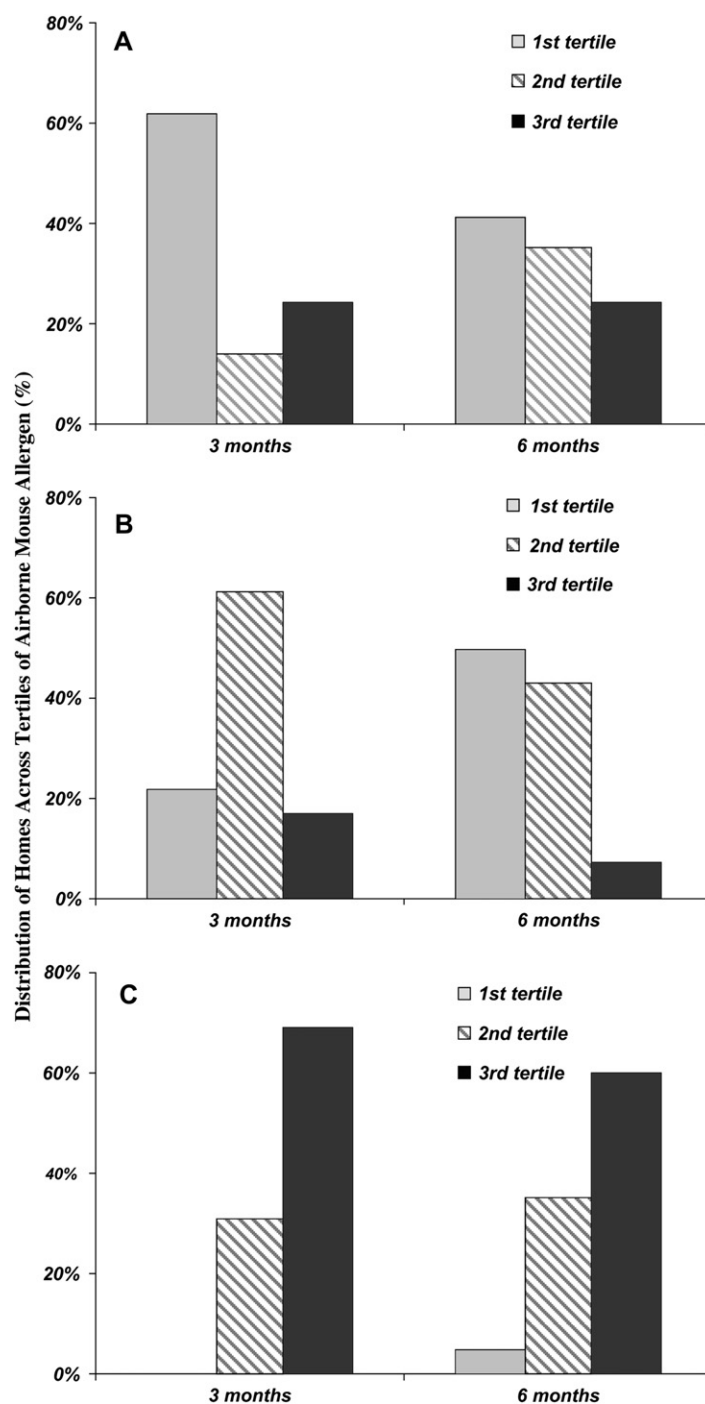


FIG 1. Changes in airborne mouse allergen levels over time among homes that were in the lowest mouse allergen tertile (A), the middle tertile (B), and the highest tertile (C) at the baseline study visit. The figure depicts the distribution of these homes across allergen tertiles at subsequent visits. The cutpoints for the tertiles were as follows: 1st tertile, below detection to 0.005 ng/m³; 2nd tertile, 0.006 to 0.035 ng/m³; 3rd tertile, 0.036-5.68 ng/m³. There were 85, 84, and 74 homes at baseline, 3 months, and 6 months, respectively.

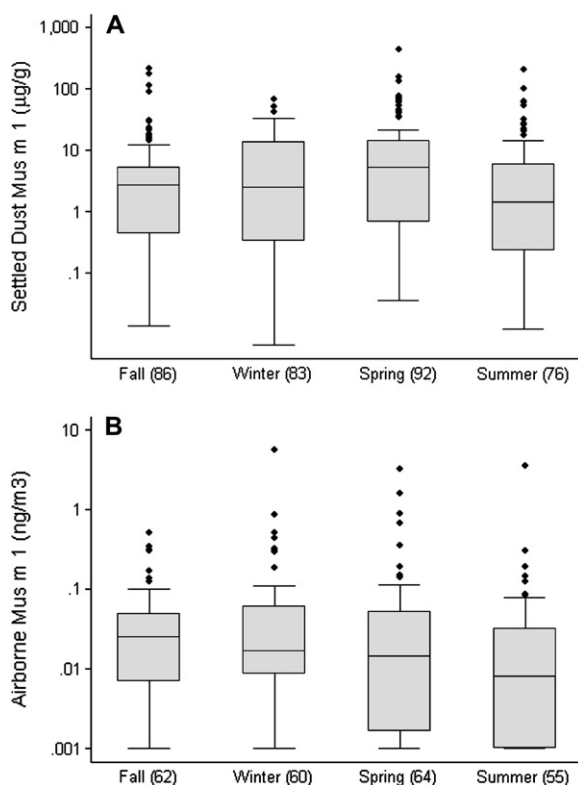


FIG 2. Bedroom settled dust (**A**) and airborne (**B**) mouse allergen levels by season. One hundred twenty-seven bedrooms were evaluated at baseline and 3 and 6 months, for a total of 337 settled dust measurements and 242 airborne measurements. The total number of measurements in each season is indicated below the x-axis. In a final multivariate model adjusting for presence of a cat, humidity, relocation, and cracks and holes in walls and doors, bedroom settled dust levels remained higher in the spring than the other seasons ($P = .05$), and airborne levels remained higher in the fall and winter than in spring ($P < .001$) and summer ($P = .04$).

37.8% $\pm$ 10.8%. At all study visit time points, homes with lower relative humidity ($\leq 45\%$) had higher airborne mouse allergen levels than homes with higher relative humidity ($>45\%$), although none of the comparisons at each of the time points was statistically significant. Median airborne Mus m 1 levels at each study visit in lower humidity versus higher humidity homes were as follows: baseline, 0.020 versus 0.011 ng/m³; 3 months, 0.014 versus 0.008 ng/m³; and 6 months, 0.038 versus 0.026 ng/m³. After adjusting for potential confounders and repeated measures in a multivariate model, an indoor relative humidity of 45% or lower was associated with higher airborne Mus m 1 levels ($P = .003$). The association between lower relative humidity and higher airborne Mus m 1 levels has been reported in an occupational setting as well,⁹ and may be related to the fact that lower humidity increases the aerosolization of particles from surfaces by reducing the adhesive forces between particles and surfaces. Consistent with this hypothesis, we found no relationship between indoor relative humidity and settled dust Mus m 1 levels.

In this population of inner-city preschool children with asthma, both airborne and settled dust mouse allergen levels vary substantially over time within homes. This degree of variability suggests that mouse-sensitized inner-city children with asthma may shift from low exposure to high exposure environments, or vice versa, over a course of months—potentially resulting in lability in asthma symptoms and changes in risk of morbidity. In addition, this temporal variability underscores the importance of obtaining repeated exposure measures in epidemiologic studies of mouse allergen exposure and health effects. Last, time-dependent factors, including acquisition of a cat, indoor relative humidity, and season, significantly affect home mouse allergen levels. Knowledge of these predictors lends insight into which environmental conditions are most likely to be associated with high mouse allergen levels, and how environmental interventions may be optimized to reduce home mouse allergen levels.

Elizabeth C. Matsui, MD, MHS
Peyton A. Eggleston, MD
Patrick Breysse, PhD
Gregory B. Diette, MD, MHS

From Johns Hopkins University, Baltimore, Md. E-mail: ematsui@jhmi.edu. Supported by grants from the US Environmental Protection Agency (R82672401), the National Institute of Environmental Health Sciences (ES009606), the National Heart, Lung, and Blood Institute (HL04266, HL67850), the National Institute of Allergy and Infectious Diseases (AI060955, AI070630), and the US Environmental Protection Agency's Science to Achieve Results program.

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