

Distribution and Determinants of Mouse Allergen Exposure in Low-Income New York City Apartments

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Previous studies of mouse allergens and laboratory-animal-worker-related allergy and asthma suggest that quantifying mouse allergen levels in homes could augment our understanding of inner-city asthma. We hypothesized that levels of mouse allergen in inner-city homes would be related to certain household characteristics. Dust samples were collected from the kitchens and beds of 221 mothers enrolled in a prospective birth cohort study, 92 of African American and 129 of Dominican ethnicity. Samples were analyzed for mouse urinary protein. The geometric mean for kitchen samples was 4.6 µg/g [95% confidence interval (95% CI), 3.2–6.5] and for bed samples was 0.9 µg/g (95% CI, 0.8–1.1). The variables associated with mouse allergen levels in the home were frequency of mouse sightings, use of traps or pesticides for mice, presence of holes in ceilings or walls, absence of a cat, and living in a building with fewer than eight floors. Statistically significant neighborhood differences in levels of mouse allergen and report of rodents in the home were also observed. In conclusion, mouse allergen was prevalent among inner-city apartments, and the positive predictive value of self-reported frequent mouse sightings was high (90% for kitchens). However, high levels of mouse allergen were also found in many homes where mothers reported never seeing mice. **Key words:** asthma, home characteristics, mouse allergen, *Mus m 1*. *Environ Health Perspect* 111:1348–1351 (2003). doi:10.1289/ehp.6124 available via <http://dx.doi.org/> [Online 7 May 2003]

Mouse allergen exposure and sensitization have been implicated in the development of laboratory-animal-worker-related allergy and asthma (Hollander et al. 1996; Renstrom et al. 2001; Schumacher et al. 1981). One Polish study reported increased sensitization to laboratory animals (mice, rats, and hamsters) in a cohort of children whose parents worked in laboratory settings (Krakowiak et al. 1999). The exposure was hypothesized to be caused by periodically bringing laboratory animals home and inadvertently carrying allergen on their clothes.

The urban landscape, replete with multi-family dwellings, a high density of grocery stores and restaurants, poor housing maintenance, and difficulties with litter and sanitation, provides a conducive environment for mice. In addition, several studies have shown that low-income urban areas have a disproportionate prevalence of childhood asthma (Carr et al. 1992; Litonjua et al. 1999). Several investigators have seen an association between asthma and sensitization to another common urban pest, cockroaches (Call et al. 1992; Gelber et al. 1993), and high exposure (Bla g 1 > 8 U/g) among those sensitized has been reported as a risk factor for asthma medication use and number of asthma-related hospitalizations (Rosenstreich et al. 1997). Recently, a high prevalence of mouse allergen in inner-city homes throughout the United States has been reported by investigators from

the National Cooperative Inner City Asthma Study (NCICAS) (Phipatanakul et al. 2000a). The NCICAS study further observed that significantly more asthmatic children were allergic to mice (assessed by skin prick test) when mouse allergen levels in house dust were > 1.6 µg/g (Phipatanakul et al. 2000b).

The Columbia Center for Children's Environmental Health (New York City, New York) is currently evaluating levels of indoor and outdoor pollutant exposures in the homes of pregnant women. The infants will undergo intensive clinical follow-up, and their homes will continue to be monitored through early childhood. The assessment of mouse allergen levels in the homes of the pregnant women is important for two main reasons: *a*) Early exposure to other indoor allergens has been shown to be important (whether negatively or positively associated) in the development of allergic sensitization and respiratory symptoms (Litonjua et al. 2001; Ownby et al. 2002; Sporik et al. 1990); and *b*) understanding the factors influencing this exposure could lead to intervention targets, resulting in decreased morbidity. The aim of this study is to describe the distribution of mouse allergen in the homes of pregnant women living in three inner-city neighborhoods of New York City and to identify household characteristics that are associated with high mouse allergen levels in settled dust samples.

Materials and Methods

Description of study cohort. Study subjects were Dominican ($n = 129$) and African-American ($n = 92$) women who delivered at Columbia (New York) Presbyterian Medical Center (NYPMC) or Harlem Hospital (HH). The classification of ethnicity was based on self-definition and group identification. Nonsmoking women, 18–35 years old, who registered at the obstetrics and gynecology clinics at NYPMC and HH by the 20th week of pregnancy; were free of diabetes, hypertension, or known HIV; and had resided in the area for at least 1 year were eligible. Women with a history of drug use or who answered yes to questions related to drug use were also excluded. Informed consent was obtained from all participants in accordance with the NYPMC Institutional Review Board.

Home characteristics questionnaire. A 45-min questionnaire was administered during the last trimester of pregnancy. It included demographic information and home characteristics such as type of building, problems with moisture, water leaks, or visible mold, pet ownership, and frequency of cockroach, mouse, and rat sightings inside the apartment.

Dust sample collection. Dust samples were collected separately from the kitchen and beds of 221 pregnant women during their third trimester and/or 12 months after delivery. Dust samples from 125 prenatal visits (109 beds and 112 kitchens, including 93 homes with both) and from 151 postnatal visits (131 beds and 122 kitchens) were available for allergen analyses. Forty-two mothers

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had both a prenatal and a postnatal kitchen sample, and 38 mothers had both a prenatal and postnatal bed sample.

Dust was collected onto 70-mm cellulose filters (Whatman International, Maidstone, UK) with a canister vacuum cleaner (Eureka Mighty Mite, Bloomington, IN) and a modified collection nozzle (ALK, Hørshølm, Denmark). In the kitchen, exposed areas of the floor were vacuumed for 4 min. For the bed sample, the pillows, upper half of the bed, and upper half of all bed layers were vacuumed for 4 min. Samples were returned to the laboratory for postweighing and then stored at -20°C. Dust samples were not sieved. Depending on the amount of dust, phosphate-buffered saline with 0.05% Tween 20 (PBS-T) was added in quantities sufficient to create a slurry. The exact extraction concentration was recorded. Next, the mixture was shaken at 200 rpm for 1 hr at 30°C. Samples were centrifuged at 11,000 rpm for 1 min, and the supernatant was removed and frozen at -20°C until assayed.

Analysis of mouse and cockroach allergens. Mouse urinary protein (MUP) contains several immunoglobulin E-binding proteins, including Mus m 1 (Hollander et al. 1996; Lorusso et al. 1986). MUP was assayed with a competitive enzyme-linked immunosorbent assay (ELISA) using MUP extract (Greer Laboratories, Inc., Lenoir, NC) (Hollander et al. 1996), a polyclonal rabbit anti-MUP antibody (Greer Laboratories), and a polyclonal goat anti-rabbit antibody (Sigma Chemical

Co., St Louis, MO). MUP (3 µg/mL of carbonate-bicarbonate buffer, pH = 9.6) was incubated in Immunlon 4 microtiter plate wells (Dynatech Laboratories, Inc., Chantilly, VA) overnight at 4°C. The plates were washed with PBS-T and blocked with bovine serum albumin-PBS-T. Three-fold dilutions of standards (from 0.004 to 3 µg/mL) and dust extracts (full strength, 1:3, 1:9, and 1:27) were added. A 1:2,500 solution of rabbit anti-MUP antibody was added to each well and incubated overnight at 4°C. After washing the plate, goat anti-rabbit antibody (1 µg/mL) was added to each well and incubated for 1 hr at 25°C. The plate was washed and developed with 2,2'-azino-bis-3-ethylbenzthiazoline-6-sulfonic acid and H₂O₂ (1 µg/mL) and optical density was read kinetically. The lower limit of detection (LOD) of MUP was 0.004 µg/mL, but the LOD for MUP per gram of dust varied slightly because of differences in extraction concentrations. For validation, 20 samples ranging from 0.1 µg/g to 184.6 µg/g were assayed with the commercially available Mus m 1 assay (Indoor Biotechnologies, Charlottesville, VA). The results from both mouse allergen assays were highly correlated (Spearman rank correlation coefficient = 0.96; $p < 0.0001$), and for these samples, the median

concentration of MUP was 3-fold higher than that of Mus m 1. Antigens (Bla g 2) from German cockroaches were analyzed by ELISA (Indoor Biotechnologies) (Luczynska et al. 1989; Pollart et al. 1991).

Data analysis. We analyzed allergen concentrations as continuous and as categorical variables. Allergen measurements below the LOD were assigned the LOD for the specific allergen and collection location. Allergen concentrations underwent a natural log transformation to stabilize the variance and approximate a normal distribution. Dichotomous categories were created for Bla g 2 (cut point for kitchens = 0.03 µg/g and for beds = 0.02 µg/g) and MUP (cut point for kitchens = 1.98 µg/g and for beds = 0.5 µg/g) based upon the median concentrations observed in our cohort. We treated the questionnaire outcomes as categorical variables. For example, a composite index of food available for pests in the kitchen was developed by using questions about how often various conditions occurred overnight in the home in the past month (e.g., food in an uncovered garbage can, dirty dishes in the sink). Seasons were categorized as winter (January–March), spring (April–June), summer (July–September), and fall (October–December). Three neighborhoods were defined by zip codes (Washington

Table 1. Household characteristics (n = 221).

Characteristic	Frequency (%)
Frequency of mouse sightings	
Never	29
Rarely	32
Weekly	5
Daily	8
No response	26
Frequency of rat sightings	
Never	67
Rarely	5
Weekly	0
Daily	1
No response	26
Frequency of cockroach sightings	
Never	12
Rarely	41
Weekly	4
Daily	18
No response	26
Presence of cat in home	
No	79
Yes	8
No response	14
Use of mouse traps or rodenticides	
No	76
Yes	24
Presence of holes in ceilings/walls in home	
No	66
Yes	33
No response	1

Table 2. Descriptive statistics of MUP and Bla g 2 allergens measured prenatally and 12 months postnatally in kitchens and beds.

Dust sample	No. ^a	Samples below LOD (%) ^b	GM (95% CI)	Maximum
Prenatal				
MUP in bed (µg/g)	107	57	0.9 (0.8–1.1)	20
MUP in kitchen (µg/g)	112	43	4.6 (3.2–6.5)	1,478
Bla g 2 in bed (µg/g)	109	41	0.06 (0.04–0.07)	6
Bla g 2 in kitchen (µg/g)	104	23	0.3 (0.2–0.5)	32
Postnatal				
MUP in bed (µg/g)	128	31	1.7 (1.4–2.1)	175
MUP in kitchen (µg/g)	119	34	6.6 (4.6–9.5)	2,373
Bla g 2 in bed (µg/g)	124	30	0.07 (0.05–0.09)	16
Bla g 2 in kitchen (µg/g)	121	29	0.3 (0.2–0.4)	16

GM, geometric mean.

^aDue to the study design and loss to follow-up, the prenatal and postnatal samples were not necessarily collected from the same participants. ^bSamples below the LOD were assigned the value of the LOD for calculating the GM. For these samples, Bla g 2 LOD was 0.02 µg/g for beds and 0.03 µg/g for kitchens, and MUP LOD was 0.5 µg/g for beds and 1.0 µg/g for kitchens.

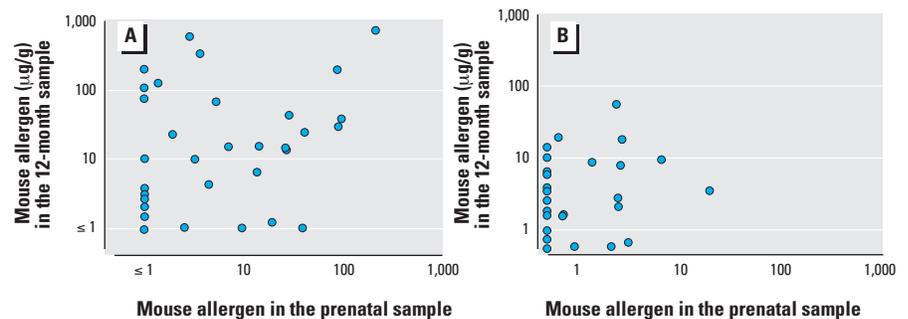


Figure 1. MUP (µg/g) in the prenatal sample versus the 12 postnatal samples in the kitchen (A) and bed (B) samples (n = 42 and 38, respectively). Samples below the LOD were assigned the value of the LOD. Nine and 10 samples were below LOD for the prenatal and 12-month samples from the kitchen and bed, respectively. There was a significant correlation between kitchen samples ($r = 0.60$, $p < 0.001$) but not bed samples ($r = 0.06$, $p = 0.73$).

Heights, 10032, 10033, 10040; Harlem, 10027, 10029, 10035; middle neighborhood, 10031).

We calculated Pearson correlation coefficients to quantify the linear relationships among allergen concentrations. Mouse allergen levels collected prenatally were compared by a Student's *t*-test with those collected 12 months postnatally (natural logged values). The negative predictive value (NPV) of a question was reported as the probability of a dust sample having a low allergen concentration (i.e., below a given cut point), given the absence of a certain home characteristic. The positive predictive value (PPV) of a question was reported as the probability or risk of a dust sample having a high allergen concentration (i.e., above a given cut point), given the presence of a certain home characteristic. Odds ratios (ORs) were calculated with 95% confidence interval (95% CI) for determinants of high mouse allergen levels. Multiple logistic regression models included all variables that were significant in the bivariate analysis. All data were analyzed with SPSS statistical software (SPSS Inc., Chicago, IL).

Results

Of the 221 participating women, 74% reported an annual household income less than \$20,000, and 20% were between \$20,000 and \$80,000 (6% did not answer the question). Some mothers also did not answer the question regarding frequency of mice, cockroach, or rat sightings. Slightly more kitchens had MUP levels above the median in the whole group from whom dust was collected (50%) compared with those who did not answer the question about frequency of mouse sightings, 50% versus 46%, but this association was not significant. The housing characteristics of the study cohort are described in Table 1.

Table 2 lists the descriptive statistics for the mouse and cockroach allergens recovered from the kitchens and beds during the prenatal and postnatal home visits. In the prenatal samples, the mouse allergen levels in beds and kitchens were significantly correlated ($r = 0.63$, $p < 0.001$). However, kitchen levels tended to be higher ($p < 0.001$) and more

variable. Thirty-eight homes had a matching 12-month postnatal visit bed sample, and 42 homes had a matching 12-month postnatal visit kitchen sample. The pre- and postnatal measurements were significantly correlated (Figure 1), but means were significantly higher for the later visit (bed, $p = 0.001$; kitchen, $p = 0.018$).

Based upon logistic regression (Table 3), the statistically significant variables associated with high mouse allergen in bed and kitchens included the following: frequency of mouse sightings, use of traps or pesticides for mice, no report of cat in the home, living in a building with fewer than eight floors, and presence of holes in ceilings or walls. The NPV and PPV of these questions were poor (Table 3). However, when reports of weekly or daily sightings of mice were compared with responses of "never," the predictive values exceeded 90% for kitchen samples and 70% for bed samples.

Because of results from a previous study of mouse allergen in inner-city homes, we adjusted for high levels of Bla g 2 when conducting multiple logistic regression, even though this variable was not a significant covariate in our bivariate model. No significant seasonal variation of median mouse allergen levels was observed, and the aggregate index of food available for mice (e.g., food in uncovered garbage can, dirty dishes in sink) was not significant in regression models. In the multiple logistic regression models (adjusted for Bla g 2 levels and other variables significant in the bivariate models), only one variable remained significant for MUP in the kitchen: frequency of mouse sightings (OR = 4.9; 95% CI, 1.7–13.8).

Differences in report of rodent sightings and mouse allergen in the homes were observed among three neighborhoods: Harlem, Washington Heights, and a neighborhood located between the two (Figure 2). Significantly more homes in Washington Heights than in Harlem had high MUP in the bed ($p = 0.02$) and in the kitchen ($p = 0.01$) and reported seeing rodents ($p = 0.02$). Similar trends were observed between the middle neighborhood and Washington Heights, but this association was only

significant for kitchen allergen ($p = 0.003$) and report of rodents ($p = 0.002$). The neighborhoods varied in the composition of building type (e.g., buildings with eight or more floors), ethnicity (African American or Dominican), and report of heavy bus and traffic on the street nearest to the home. In our cohort, Harlem had the most high-rise homes (39%), followed by the middle neighborhood (17%), and Washington Heights (14%). Harlem contained mainly African Americans (80%), Washington Heights contained mainly Dominicans (88%), and the middle neighborhood contained a mixture of both African Americans (47%) and Dominicans (43%). More residents in Harlem reported heavy traffic near their home (47%) compared with the middle neighborhood (36%) and with Washington Heights (27%).

Discussion

Although most studies of mouse allergen exposure have focused on laboratory animal workers (Hollander et al. 1996; Ohman et al. 1994; Renstrom et al. 1997), our study revealed the presence of a potentially sensitizing allergen in many homes of women who do not work with laboratory mice. Our study of prenatal mouse allergen exposure revealed that *a*) mouse allergens were commonly recovered in low-income New York City apartments and at levels similar to other urban areas of the United States;

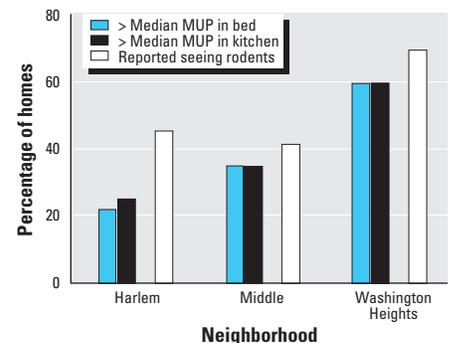


Figure 2. Mouse allergen levels and reports of mice sightings by neighborhood. For analysis, 76, 75 and 157 mothers lived in one of these three neighborhoods and had a bed sample analyzed, kitchen sample analyzed, or answered a question about seeing rodents, respectively.

Table 3. Bivariate analyses for mouse allergen in prenatal kitchen and bed dust samples.

Predictors	Kitchen samples			Bed samples		
	Predictive value (%)		> Median level ^a	Predictive value (%)		> Median level ^a
	NPV	PPV		NPV	PPV	
Frequency of mouse sightings	62	65	5.3 ^b (2.6–10.9)	62	52	1.6 ^b (1.1–2.4)
Use of traps or pesticides for mice	21	42	7.8 (2.5–24.1)	37	36	4.5 (1.7–12.1)
Bla g 2 > median level (0.3 µg/g in kitchen and 0.03 µg/g in bed)	56	56	1.2 (0.5–2.9)	50	60	1.5 (0.7–3.4)
Building has eight or more floors	40	13	0.10 (0.03–0.37)	51	14	0.16 (0.04–0.59)
Presence of cat in home	46	25	0.3 (0.1–0.9)	52	20	0.3 (0.1–1.0)
Presence of holes in ceilings/walls in home	41	45	1.8 (0.8–4.1)	34	32	4.0 (1.7–9.6)

^aMedian level of mouse allergen in kitchens and beds was 1.98 µg/g and 0.5 µg/g (lower LOD), respectively. ^bAlthough the PPV and NPV were calculated by using the dichotomous variable, never versus any (rarely, weekly, or daily) sightings, the ORs were calculated by using the ordinal variable (i.e., never, rarely, weekly, daily).

b) frequency of mouse sightings, use of mouse extermination methods, absence of a cat, type of building, and presence of holes in ceilings or walls were associated with dust-borne mouse allergens; and c) mouse allergen concentrations varied by neighborhood.

The NCICAS study was the first report that examined mouse allergen in residential environments (Phipatanakul et al. 2000a, 2000b). In this multicity U.S. study, the highest median mouse allergen level was observed in kitchens (1.60 µg/g), followed by living rooms (0.57 µg/g) and bedrooms (0.52 µg/g). Our levels were comparable (Table 2), with mouse allergen levels in the kitchen higher than those in bedrooms. Furthermore, our maximum level of mouse allergen in the kitchen was 2,373 µg/g, compared with their maximum of 618 µg/g.

Clinicians and public health researchers require some estimate of the representativeness of a sample in order to assess exposure. Although no seasonal variation was observed, mouse allergen levels tended to be higher in the 12-month postnatal samples (compared with the prenatal sample). Reasons for this difference could include changes in cleaning activities after delivery of a baby; however, the sample size ($n = 42$) did not permit further analyses at this time. Also, levels of mouse allergen in kitchen samples were higher than those of bed samples, but the two samples were correlated. The observation that mouse allergen levels in bed and kitchen samples were highly correlated may apply only to this urban population, where the distance between the two areas is rather small compared with that in larger homes. Also, the correlation between mouse allergen measurements 1 year apart suggests that in this urban environment, homes with high mouse allergen levels tend to retain those allergens in the dust samples.

The frequency of mouse sightings, the use of mouse extermination methods, absence of a cat, living in a building with fewer than eight floors, and the presence of holes in ceilings or walls were all significantly associated with higher levels of mouse allergen in kitchens or beds. There appeared to be collinearity among several of the variables, because only frequency of mouse sightings was independently associated with high mouse allergen in the kitchen. Incidentally, many homes without a report of mice also had high levels of mouse allergen, so clinicians and public health researchers alike should be cognizant that allergen exposure can occur in the absence of known or reported home characteristics (Chew et al. 1998).

Compared with the other two neighborhoods, more homes located in Washington Heights contained mouse allergen levels above the median. The three distinct neighborhoods possibly have different community-level characteristics of the built environment that might not have been captured by our study. Although these differences might not exist for other inner-city neighborhoods with different neighborhood characteristics, we speculate that some of the variables such as frequency of mouse sightings and use of mouse traps will be strong predictors of mouse allergen in a home regardless of where it is located.

In contrast to the NCICAS study, we did not observe an association between cockroach allergen and mouse allergen in the bedroom, although their level of correlation was moderate ($r = 0.18$) (Phipatanakul et al. 2000a). Factors such as building type and level of disrepair influence infestations both of cockroaches and mice, and the effect of these factors might vary between cities.

The prevalence of mouse allergen in these homes along with previous reports of an association between sensitization to MUP and allergic symptoms (Renstrom et al. 1994) suggests that the inner-city home should be an important target for intervention. Future studies should include other sources of exposure such as schools and subways. Assessment of the other locations would be necessary to determine when and where the children of these women might become sensitized to mice. The present study design will enable exposure assessment of the homes as the children mature, and a prospective assessment of allergy status.

Compared with laboratory animal workers, children with mouse allergen in their homes endure exposures for a longer proportion of each day. Although the levels of mouse allergens in many of the homes are not as high as those observed in laboratories, the effects of constant inhalation exposure in early life remains unknown. Half of our participating household kitchens contained mouse allergen levels greater than 1.98 µg/g. The NCICAS reported that those children living in homes with Mus m 1 (a component of MUP) greater than 1.6 µg/g had mouse allergy as defined by skin prick test (Phipatanakul et al. 2000b). Further, 2 µg/g has been proposed as a threshold for sensitization to dust mite allergen (Sporik et al. 1990). Our results stress the need for further examination of the health effects of residential mouse allergen exposure and the development of avoidance strategies in this community.

REFERENCES

- Call RS, Smith TF, Morris E, Chapman MD, Platts-Mills TAE. 1992. Risk factors for asthma in inner city children. *J Pediatr* 121:862–866.
- Carr W, Zeitel L, Weiss KB. 1992. Variations in asthma hospitalizations and deaths in New York City. *Am J Public Health* 82:59–65.
- Chew GL, Burge HB, Dockery DW, Muilenberg ML, Weiss ST, Gold DR. 1998. Limitations of a home characteristics questionnaire as a predictor of indoor allergen levels. *Am J Respir Crit Care Med* 157:1536–1541.
- Gelber LE, Seltzer L, Bouzoukis JK, Pollart SM, Chapman MD, Platts-Mills TAE. 1993. Sensitization and exposure to indoor allergens (dust mite, cat, and cockroach) as risk factors for asthma among patients presenting to hospital. *Am Rev Respir Dis* 147:573–578.
- Hollander A, van Run P, Spithoven J, Heederik D, Doekes G. 1996. Exposure of laboratory animal workers to airborne rat and mouse urinary allergens. *Clin Exp Allergy* 27:617–626.
- Krakowiak A, Szulc B, Gorski P. 1999. Allergy to laboratory animals in children of parents occupationally exposed to mice, rats, and hamsters. *Eur Respir J* 14:352–356.
- Litonjua AA, Carey VJ, Burge HA, Weiss ST, Gold DR. 2001. Exposure to cockroach allergen in the home is associated with incident doctor-diagnosed asthma and recurrent wheezing. *J Allergy Clin Immunol* 107:41–47.
- Litonjua AA, Carey VJ, Weiss ST, Gold DR. 1999. Race, socioeconomic factors, and area of residence are associated with asthma prevalence. *Pediatr Pulmonol* 28:394–401.
- Lorusso JR, Moffat S, Ohman JL. 1986. Immunologic and biochemical properties of the major mouse urinary allergen (Mus m 1). *J Allergy Clin Immunol* 78:928–937.
- Luczynska CM, Yin L, Chapman MD, Platts-Mills TAE. 1989. A two-site ELISA for the quantification of the major *Dermatophagoides* spp. allergens, *Der p 1* and *Der f 1*. *J Immunol Methods* 118:227–235.
- Ohman JL, Hagberg K, MacDonald MR, Jones RR, Paigen BJ, Kaceris JB. 1994. Distribution of airborne mouse allergen in a major breeding facility. *J Allergy Clin Immunol* 94:810–817.
- Owby DR, Johnson CC, Peterson EL. 2002. Exposure to dogs and cats in the first year of life and risk of allergic sensitization at 6 to 7 years of age. *JAMA* 288:963–972.
- Phipatanakul W, Eggleston PA, Wright EC, Wood RA. 2000a. Mouse allergen. I. The prevalence of mouse allergen in inner-city homes. The National Cooperative Inner-City Asthma Study. *J Allergy Clin Immunol* 106:1070–1074.
- . 2000b. Mouse allergen. II. The relationship of mouse allergen exposure to mouse sensitization and asthma morbidity in inner-city children with asthma. *J Allergy Clin Immunol* 106:1075–1080.
- Pollart SM, Smith TF, Morris EC, Gelber LE, Platts-Mills TAE, Chapman MD. 1991. Environmental exposure to cockroach allergens: analysis with monoclonal antibody-based enzyme immunoassays. *J Allergy Clin Immunol* 87:505–510.
- Renstrom A, Gordon S, Larsson PH, Tee RD, Newman-Taylor AJ, Malmberg P. 1997. Comparison of a radioallergen sorbent (RAST) inhibition method and a monoclonal enzyme linked immunosorbent assay (ELISA) for aeroallergen measurement. *Clin Exp Allergy* 27:1314–1321.
- Renstrom A, Karlsson AS, Malmberg P, Larsson PH, van Hage-Hamsten M. 2001. Working with male rodents may increase risk of allergy to laboratory animals. *Allergy* 56:964–970.
- Renstrom A, Malmberg P, Larsson K, Sundblad BM, Larsson PH. 1994. Prospective study of laboratory-animal allergy: factors predisposing to sensitization and development of allergic symptoms. *Allergy* 49:548–552.
- Rosenstreich DL, Eggleston P, Kattan M, Baker D, Slavin RG, Gergen P, et al. 1997. The role of cockroach allergy and exposure to cockroach allergen in causing morbidity among inner-city children with asthma. *N Engl J Med* 336:1356–1363.
- Schumacher MJ, Tait BD, Holmes MC. 1981. Allergy to murine antigens in a biological research institute. *J Allergy Clin Immunol* 68:310–318.
- Sporik R, Holgate ST, Platts-Mills TAE, Cogswell JJ. 1990. Exposure to house-dust mite allergen (*Der p 1*) and the development of asthma in childhood: a prospective study. *N Engl J Med* 323:502–507.