

Original article

Cockroach allergen levels and associations with cockroach-specific IgE

Ginger L. Chew, ScD,^a Matthew S. Perzanowski, PhD,^a Stephen M. Canfield, MD, PhD,^d
Inge F. Goldstein, DrPH,^b Robert B. Mellins, MD,^e Lori A. Hoepner, MPH,^c
Maxine Ashby-Thompson, MPH,^b and Judith S. Jacobson, DrPH^b *New York, NY*

Background: Among inner-city children with asthma, cockroach allergen exposure has been associated with allergic sensitization.

Objective: We hypothesized that cockroach allergen levels in homes would be associated with sensitization to cockroach allergens in children.

Methods: From a low-income preschool program, 341 four-year-old children selected on the basis of the willingness of their caregivers to participate in the study were enrolled. Dust from their beds and kitchens were analyzed for cockroach (Bla g 2), mouse (mouse urinary proteins), and cat allergens (Fel d 1). Serum samples were analyzed for allergen-specific IgE antibodies by immunoassay.

Results: Bla g 2 levels >1 U/g in children's bed and kitchen dust samples were independently associated with cockroach-specific IgE (odds ratio [OR], 2.7; 95% CI, 1.1-6.4; and OR, 3.4; 95% CI, 1.2-9.4, respectively), adjusting for sex, ethnicity, asthma, pet ownership, mother's allergic sensitization, environmental tobacco smoke, and having lived in other homes. Bla g 2 was associated (OR, 3.6; 95% CI, 1.0-13.1) with cockroach-specific IgE among children with asthma. Among children without asthma, the ORs were similar (OR, 3.0; 95% CI, 0.9-10.3), but the association was not statistically significant.

Conclusion: Concentrations of the major cockroach allergen, Bla g 2, in settled dust were associated with cockroach-specific IgE independent of other factors in a cohort of 4-year-old inner-city children.

Clinical implications: These findings demonstrate the importance of domestic exposure to cockroach allergen to the development of sensitization and suggest a potential primary

prevention target in this community. (*J Allergy Clin Immunol* ■■■■;■■■;■■■-■■■.)

Key words: Cockroach, cat, mouse, allergen, IgE, inner-city, atopy, allergy, children

Although allergen exposure is necessary for the development of an allergic response, a dose-response relationship between allergen exposure and allergic sensitization or respiratory symptoms has been difficult to demonstrate, in part because susceptible individuals may respond to low levels of exposure, or because sensitized individuals may consciously or subconsciously avoid allergen exposure.^{1,2} Among young children, sensitization to indoor allergens takes time to develop and may not be evident even when exposure levels are high³; however, among sensitized children, allergen exposure is associated with risk of developing asthma and decreased lung function.⁴

The rates of pediatric asthma hospitalization in New York City (NYC) are among the highest reported for urban areas in the United States.⁵ In NYC and other inner-city communities, pediatric asthma has been shown to be strongly associated with sensitization to cockroach and mouse allergens.⁶⁻⁸ High levels of cockroach and mouse allergen have been observed in homes in these neighborhoods, and the combination of sensitization and exposure increases the risk of exacerbation of symptoms among children with asthma.^{6,8-12}

To date, early cross-sectional studies that included children without asthma or family history of atopy were hampered in their ability to show dose-response associations between cockroach and cat allergen concentrations in the homes and sensitization to these allergens because either the prevalence of sensitization was low or exposure was low.^{13,14} No comparable studies included assessment of mouse allergen and sensitization. Because sensitization to indoor allergens can develop early in life,¹⁵ a lack of an association in these previous studies may have been a consequence of exposure being assessed too far after the critical exposure window. Ideally, exposure would be assessed from birth and related to the development of sensitization; however, birth cohort studies can be expensive and have an inherently long time lag before associations with sensitization to many indoor allergens can be examined. In the current study, we have assessed the association between cockroach, mouse, and cat allergen exposure and

From ^athe Department of Environmental Health Sciences, ^bthe Department of Epidemiology, and ^cthe Data Coordinating Center, Mailman School of Public Health, Columbia University; and ^dthe Department of Medicine, Division of Pulmonary, Allergy, Critical Care Medicine, and ^ethe Pediatric Pulmonary Division, Columbia University College of Physicians and Surgeons.

Supported by grants from the National Institutes of Health (HL068236 and P30 ES009089). G.L.C. received funding as a National Institutes of Health National Center on Minority Health and Health Disparities fellow.

Disclosure of potential conflict of interest: The authors have declared that they have no conflict of interest.

Received for publication May 8, 2007; revised August 13, 2007; accepted for publication August 14, 2007.

Reprint requests: Ginger L. Chew, ScD, Department of Environmental Health Sciences, Mailman School of Public Health, Columbia University, 60 Haven Avenue, B-1, New York, NY 10032. E-mail: cg288@columbia.edu. 0091-6749/\$32.00

© 2007 American Academy of Allergy, Asthma & Immunology

doi:10.1016/j.jaci.2007.08.024

Abbreviations used

ETS: Environmental tobacco smoke
 ICAS: Inner-City Asthma Study
 MUP: Mouse urinary protein
 NCICAS: National Cooperative Inner-City Asthma Study
 NYC: New York City
 OR: Odds ratio

sensitization at age 4 years in a sample of children attending Head Start centers in NYC. These children live in communities with an asthma prevalence that is among the highest in the United States.⁵ We believed that allergen exposure assessed among 4-year-olds would be a better proxy for exposure in the critical sensitization window (ie, birth to 3 years) than exposure assessed among older children, like the participants in the large inner-city asthma studies.^{6,8} Our sample also allowed us to study the association of allergen exposure with sensitization among children with and without asthma.

METHODS

Subjects and recruitment

Because of the known high prevalence of asthma among children from low-income families in urban minority communities, we recruited children (mean age, 4 ± 0.6 years) at 50 Head Start centers in northern Manhattan, the Bronx, and Brooklyn, the New York City areas with the highest pediatric asthma prevalence and hospitalization rates. At the centers, bilingual English-speaking and Spanish-speaking interviewers obtained informed consent from the children's caregivers for participation in a study involving questionnaires and home visits, during which we collected serum samples and dust samples. The study was approved by the Institutional Review Board of the Columbia University Medical Center.

Questionnaire

Parents completed the questionnaire either at the Head Start center after the presentation or, if they preferred, by telephone or in the home. The survey instrument included questions about the child's history of asthma/wheeze/cough, personal and family medical history, home environment, and demographics. Children born in the United States who had at least 1 parent born in a Spanish-speaking country were categorized as having that national origin. Children whose parents were born in a Spanish-speaking country other than the Dominican Republic, Mexico, or Puerto Rico, or who were not born in the same Spanish-speaking country were categorized as other/mixed Hispanic. Children whose parents were not born in a Spanish-speaking country and who were not identified as Hispanic were categorized as non-Hispanic.

We also categorized children based on questionnaire responses indicating whether they lived with a cat or dog from birth to 12 months of age and whether they currently lived with ≥ 1 smoker (proxy for environmental tobacco smoke [ETS]). As an indicator of socioeconomic status, the mother's educational attainment was dichotomized (attended college vs did not attend college). The number of homes in which a child had lived during his or her lifetime was dichotomized (ie, 1 vs more than 1 home).

Making a definitive diagnosis of asthma in a 4-year-old child on the basis of questionnaire responses is difficult. For the purposes of

analyses of our cohort at this age, we categorized children as having asthma if they met the following criteria: (1) a history of physician-diagnosed asthma and any wheezing or difficulty in breathing in the past year, (2) wheezing or difficulty in breathing ≥ 3 times in the past year, (3) any urgent physician, emergency department, or hospital visit because of wheezing or difficulty in breathing, or (4) the use of any asthma medication in the past year unless the only symptom was cough.

Home visits

Settled dust samples in the homes of the participating children were collected using a Mitest dust collector (Indoor Biotechnologies, Charlottesville, Va) attached to a canister vacuum cleaner (Eureka Mighty Mite, Bloomington, Ill). Bed samples were collected by vacuuming the upper half of the bed near the pillows for 3 minutes. Kitchen dust was collected by vacuuming the floor adjacent to the baseboards for 3 minutes. When a rug was present in the kitchen, at least 1 minute was devoted to vacuuming that surface. Samples were frozen at -20°C after collection.

Allergen exposure assessment

Dust samples were extracted with PBS with 0.05% Tween 20 and assayed for mouse (mouse urinary protein [MUP]), cat (Fel d 1), and cockroach (Bla g 2) allergens as previously described.^{9,16,17} The MUP reagents were obtained from Greer Laboratories, Inc (Lenoir, NC), and the Fel d 1 and Bla g 2 reagents were obtained from Indoor Biotechnologies. Allergen levels were treated as continuous (natural log-transformed) and as dichotomous variables (Fel d 1 > 8 $\mu\text{g/g}$; Bla g 2 > 1 U/g [40 ng/g]; MUP > 1.6 $\mu\text{g/g}$). In published literature, these levels of have been associated with allergic sensitization.^{7,13,18} Allergen levels were additionally dichotomized according to the median levels we observed in this cohort. Dust mite allergen was not measured in dust samples because previous analysis had shown very low levels of Der f 1 and Der p 1 in multi-family buildings in the northeastern United States.^{11,19}

IgE analysis

Serum samples were obtained from each participating child and mother. Specific IgE antibodies against dust mite (*Dermatophagoides farinae*), cockroach (German cockroach whole body extract), mouse (MUP), and cat (cat dander) were measured by PharmaciaCAP (Phadia, Portage, Mich). Study participants were classified as seroatopic if they had specific IgE ≥ 0.35 IU/mL to any of the 4 allergens tested.

Statistical analysis

Seroatopic and nonseroatopic children were compared with respect to demographic characteristics, health indicators, and environmental factors using χ^2 tests and logistic regression for categorical variables and t tests for continuous variables. Logistic regression models were constructed with allergen levels in bed and kitchen dust as the predictors and allergen-specific IgE antibody levels (≥ 0.35 IU/mL) as the outcomes, with and without stratification by factors including sex, ethnicity, living with a cat or dog at ages 0 to 12 months, living with a smoker, mother's education, mother's seroatopy, number of homes in which the child has lived, and child's current asthma status. We also evaluated our data using the 3.50 IU/mL cutoff because it represents a high positive (ie, CAP class 3) response.

RESULTS

Dust samples were collected and analyzed from 388 homes. Corresponding to these homes, serum was

TABLE I. Demographic, health, and environmental factors by seroatopic status of the child

	Seroatopic		
	Total (n = 341)*	No (n = 224)*	Yes (n = 117)*
Boys	48%	44%	55%
Ethnicity			
Mexican	34%	35%	31%
Puerto Rican	9%	9%	9%
Dominican	26%	26%	27%
Mixed/other Hispanic	22%	20%	26%
Other	9%	10%	7%
Mother attended college	26%	25%	28%
No. of homes in child's lifetime is ≥ 2	54%	53%	54%
Children with asthma	31%	26%	41%†
Children with specific IgE ≥ 0.35 IU/mL to			
Cat	8%	—	22%
Mouse	10%	—	30%
Dust mite	16%	—	45%
Cockroach	22%	—	64%
Mothers with seroatopy	47%	38%	64%†
Children who live with smokers	21%	19%	26%
Children who lived with a cat or dog at age 0-12 mo	5%	5%	5%

*Sample size varies slightly within strata because of missing data for some questions, IgE, or allergen measurements.

† χ^2 Test for differences between seroatopic and nonseroatopic children; $P < .01$.

collected from 341 mother-child pairs. Demographic characteristics, health indicators, and environmental conditions in the homes of the study participants are listed in Table I. Thirty-four percent of the children had IgE antibodies to at least 1 of the tested allergens; 22% had anti-cockroach specific IgE. Almost half the mothers were seroatopic, and almost a third of the children had asthma (as defined). Allergen-specific IgE was more prevalent among children of seroatopic mothers ($P < .01$) and among children with asthma than among other children ($P < .01$). Although most types of allergen-specific sensitivity did not vary significantly by ethnicity (Table II), cat allergen sensitivity was much more prevalent among Puerto Rican children (23%) than children of all other ethnicities combined (6%; $P < .01$). However, cat ownership was low among all of our participants (5%).

Descriptive statistics for allergen levels in bed and kitchen dust samples are listed in Table II. Average Bla g 2 levels were much higher in kitchen dust than in bed dust, but MUP levels were not significantly different between locations. Bla g 2 levels in beds and kitchens were correlated ($n = 302$; $r = 0.50$; $P < .001$), as were MUP levels ($n = 316$; $r = 0.45$; $P < .001$). Mean levels of cockroach, mouse and cat allergens in beds were not significantly different for children who had lived in only 1 home compared with those who had ever moved (t test P values = .35, .90, and .60), and a similar lack of association was observed for Bla g 2 and MUP in kitchens (t test P values = .65 and

TABLE II. Allergen levels in homes

Type and location of allergen	Geometric mean (SD)	Median (25th to 75th percentile)
Beds ($n = 330$)		
Bla g 2 (U/g)*	4.8 (5.8)	4.9 (0.8-19.1)
Fel d 1 ($\mu\text{g/g}$)	0.2 (4.8)	0.1 (0.1-0.3)
MUP ($\mu\text{g/g}$)	1.2 (4.0)	0.8 (0.4-17.5)
Kitchens ($n = 334$)		
Bla g 2 (U/g)*	15.3 (9.2)	20.1 (2.9-92.0)
MUP ($\mu\text{g/g}$)	1.7 (6.6)	0.4 (0.4-7.5)

*1 U Bla g 2 = 40 ng.

.31). The percentage of homes with concentrations of Bla g 2 > 1 U/g in both beds and kitchens varied by ethnicity (Table III).

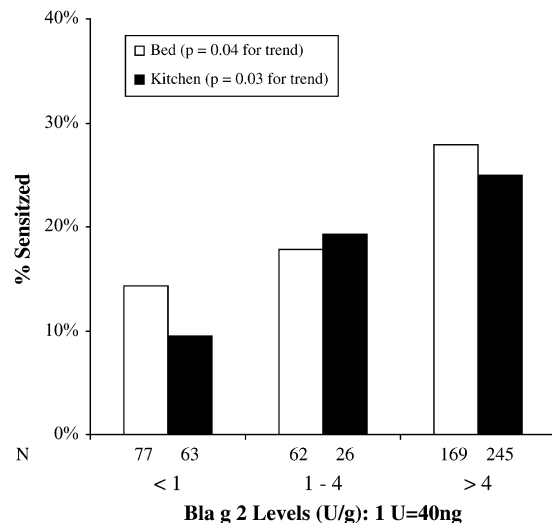
Cockroach allergen level in dust was associated with an increase in percentage of children who were cockroach seroatopic (Fig 1). Bla g 2 levels in children's bed and kitchen dust samples were positively associated with cockroach seroatopy with and without adjustment for ethnicity, sex, ETS, current asthma status, mother's education, cat or dog ownership in the first year of life, number of homes, and mother's seroatopy (Table IV) and within strata of those variables (data not shown). In most strata and in the multivariable model, the parameter estimates for the association of cockroach-specific IgE with Bla g 2 remained close to that in the crude model. For example, among the children with asthma, those with Bla g 2 > 1 U/g in their kitchen were more likely to be cockroach seroatopic than those with a lower Bla g 2 level (odds ratio [OR], 3.6; $P = .05$), and among those without asthma, the association was still high but not statistically significant (OR, 3.0; $P = .08$). Bla g 2 in dust was also associated with higher levels of cockroach-specific IgE (≥ 3.5 IU/mL). In the multivariable model, the association between Bla g 2 > 1 U/g and cockroach-specific IgE ≥ 3.5 IU/mL was strong whether exposure was considered in beds (OR, 6.8; $P = .02$) or kitchens (OR, 7.5; $P = .05$). Neither Fel d 1 nor MUP concentrations in dust samples were statistically significantly associated with cat or mouse sensitization in crude or adjusted models (Table IV). Using the higher cutpoint for mouse and cat-specific IgE did not result in statistically significant associations with the allergen level in dust.

The strongest associations between sensitization and dichotomized Bla g 2 exposure were observed using a cutpoint of 1 U/g in both kitchen and bedroom samples (Table V). The associations remained positive at higher cutpoints, and statistically significant at the median of 4.9 U/g for the bedroom samples and at all cutpoints in the kitchen samples, but as the cutpoints rose, the point estimates shifted toward the null. Changing the cutpoints for Fel d 1 or MUP to the median or other levels used in published literature (eg, Fel d 1 > 1 $\mu\text{g/g}$) did not result in significant relationships between allergen concentration and sensitization to cat or mouse allergen.

TABLE III. Prevalence of elevated allergen levels and allergic sensitivity by ethnicity

Variable	Ethnicity				
	Mexican (n = 115)	Puerto Rican (n = 31)	Dominican (n = 88)	Mixed/other Hispanic (n = 75)	Other (n = 30)
Allergen level (beds)					
Bla g 2 >1 U/g*†	90%	75%	65%	68%	70%
Fel d 1 >8 µg/g	6%	0%	7%	5%	5%
MUP >1.6 µg/g	39%	24%	27%	35%	26%
Allergen level (kitchens)					
Bla g 2 >1 U/g†	79%	60%	52%	61%	50%
MUP >1.6 µg/g	39%	27%	47%	39%	40%
Allergen-specific IgE ≥0.35 IU/mL					
Cockroach	24%	16%	22%	23%	20%
Mouse	8%	3%	9%	15%	17%
Cat†	4%	23%	3%	8%	17%
Dust mite	14%	19%	16%	13%	20%
Any of the above	31%	35%	35%	40%	27%

*1 U Bla g 2 = 40 ng.

† $\chi^2 P < .001$.**FIG 1.** Dose-response relationship between Bla g 2 and cockroach sensitization (≥ 0.35 IU/mL). Each bar indicates the percentage of cockroach-seroatopic children within each exposure category. The total number of children (regardless of seroatopy) in each exposure category is listed beneath each bar.

DISCUSSION

Among these 4-year-old children, cockroach allergen concentrations in dust samples were positively associated with cockroach-specific IgE in serum whether the cutpoint for sensitization was set at the level of detection for cockroach-specific IgE (≥ 0.35 IU/mL) or at a level associated with stronger allergic reactions (≥ 3.5 IU/mL). The association held when sex, early-life cat or dog exposure, ETS, child's current asthma status, and mother's seroatopy were taken into account. The association between Bla g 2 allergen levels and cockroach

seroatopy weakened slightly when, for comparability with other studies, we used higher cutpoints for Bla g 2 exposure (Table V).^{8,13,20} As the average exposure level in the referent group rose, the differences between the 2 groups became smaller. Associations between exposure and allergen specific seroatopy were not observed for mouse or cat allergens. Children with atopic predisposition have been found to develop allergy to agents in their environment.^{6,14,21-23} Furthermore, studies of children with asthma have shown associations between Bla g 1 and Bla g 2 concentrations in dust and sensitization to cockroaches.^{18,24} To facilitate comparison of our findings with those in studies of children with asthma, we stratified our logistic regression models by the children's current asthma status. We observed that Bla g 2 was associated with cockroach-specific IgE among children with asthma. Among children without asthma, the ORs were similar, but the association was not statistically significant, perhaps because of sample size, our definition of asthma at this early age, or the possibility that the subjects without asthma in our cohort may be a less susceptible group.

A possible reason for our finding regarding allergen exposure and IgE could be the high prevalence of cockroach sensitization and the wide range of Bla g 2 levels in our community. For example, 22% of our subjects, but only 2% of those in Detroit, were sensitized to cockroach allergen.²⁵ Also, a high percentage of our subjects (68%) but only 2% of the homes in Los Alamos had Bla g 2 >2 U/g.¹⁴ Compared with other inner-city populations of the United States, our study participants appear to have been exposed to higher levels of cockroach allergen (beds: geometric mean, 4.8 U/g or 192 ng/g; kitchens: geometric mean, 15.3 U/g or 612 ng/g).⁶ In the Inner-City Asthma Study (ICAS), the percentage of homes with Bla g 1 >2 U/g in beds or floors ranged from 8% in Seattle to 62% in NYC. Similar to the NYC sample in ICAS, 68% of our participants had bed levels ≥ 2 U/g. Although the cockroach allergens that were measured (Bla g 1 vs Bla

TABLE IV. Associations between allergen levels and cockroach sensitization: ORs and 95% CIs

Exposure variable	Bivariate model	Multivariable model†			
	Exposure variable OR (95% CI)	Exposure variable OR (95% CI)	Boys OR (95% CI)	Seroatopic mother OR (95% CI)	Child has asthma OR (95% CI)
Bla g 2 >1 U/g					
Bed sample	2.0 (1.0-4.1)	2.7 (1.1-6.4)*	1.9 (1.0-3.7)*	3.6 (1.8-7.0)**	1.8 (1.0-3.6)
Kitchen sample	3.1 (1.3-7.4)*	3.4 (1.2-9.4)*	1.9 (1.0-3.6)*	3.2 (1.7-6.2)**	2.4 (1.2-4.6)**
MUP >1.6 µg/g					
Bed sample	0.8 (0.4-1.7)	0.7 (0.3-1.6)	1.6 (0.7-3.7)	3.6 (1.5-8.6)**	2.6 (1.1-6.0)*
Kitchen sample	1.8 (0.9-3.7)	1.9 (0.9-4.2)	2.0 (0.9-4.6)	3.1 (1.3-7.3)**	2.3 (1.0-5.2)
Fel d 1 >8 µg/g					
Bed sample	1.5 (0.3-6.8)	1.6 (0.2-10.7)	1.6 (0.6-4.3)	4.0 (1.4-11.7)*	3.1 (1.1-8.6)*

* $P < .05$.** $P < .01$.

†In addition to the variables listed (eg, sex, mother's seroatopic status, and child's asthma), the multivariable model also included mother's education, number of homes where child had lived, ETS, early life ownership of a cat or dog, and indicator variables for ethnicity (Mexican was the referent group).

g 2) were different,⁶ there is a strong correlation between the 2 allergens ($r = 0.92$; $P < .01$).²⁶ Our measurements in bed dust suggest that many of our participants were exposed to a constant source of cockroach allergen near the breathing zone while sleeping. In addition, we consider cockroach allergen a marker of overall housing dilapidation¹² that could result in exposure to various other allergens and adjuvants (eg, fungi and lipopolysaccharides) that could influence the development allergic sensitization.²⁷⁻³⁰

We did not see significant associations between MUP levels and allergic sensitization. Concentrations of MUP in our homes were high (beds: median, 0.8 µg/g) compared with those of homes in the National Survey of Lead and Allergens in Housing (beds: median, 0.25 µg/g).³¹ However, the national survey included many suburban and rural homes and only 15 apartments in high-rise multifamily buildings. The National Inner City Asthma Study (NCICAS) participants, like ours, represented inner-city populations.³² Their median level of Mus m 1 (0.52 µg/g) was lower than ours, but the kitchen median (1.6 µg/g) was much higher. However, the mouse allergens that were measured were different in the 2 studies (Mus m 1 vs MUP). NCICAS also differed from our study in other ways: (1) asthma was one of its inclusion criteria; (2) NCICAS participants were ages 4 to 9 years; and (3) 77% of the NCICAS subjects with asthma were atopic (compared with only 45% of our subjects with asthma).³³ All of these differences along with the low prevalence of mouse sensitization among our study participants could explain why the NCICAS found an association between mouse allergen exposure and mouse sensitization and we did not.

The prevalence of cat sensitization among children in this study was 8%, and only 6% of their homes had high levels of Fel d 1, which could explain why an association between exposure and sensitization was not detectable in our study. ICAS showed that cat allergen varied by city and, like our study, did not find an association between cat allergen exposure and cat sensitivity.⁶ The prevalence of pet ownership in the child's infancy was much lower in

TABLE V. ORs for cockroach seroatopy using different cutpoints for Bla g 2 level in dust

Allergen cutpoints for Bla g 2 (U/g)	OR	P value
Bed		
≤1	Referent	—
>1	2.0	.05
≤2	Referent	—
>2	1.3	.29
≤4.9*	Referent	—
>4.9*	1.8	.04
≤8	Referent	—
>8	1.3	.32
Kitchen		
≤1	Referent	—
>1	3.4	.01
≤2	Referent	—
>2	2.4	.02
≤8	Referent	—
>8	2.2	.01
≤20.1*	Referent	—
>20.1*	1.8	.03

*Indicates median level in study homes.

our study (5%) than in others (53% in Detroit, 43% in New Zealand).^{25,34,35} Low prevalence may account for our failure to find an association of cat allergen with cat sensitization. Also, several studies have reported an inverse association between cat ownership and sensitization to cat, and this pet protective effect, although not observed with our small sample size of pet owners, could further complicate the dose response between Fel d 1 in the dust and sensitization to cat allergen in this cohort.³⁶⁻³⁸

The associations that we observed between ethnicity and both allergen exposure and specific IgE are somewhat paradoxical and may be caused by factors not captured in our study. For example, some neighborhoods in New York represent distinct ethnic enclaves, and therefore, in relation to exposures, ethnicity may serve mainly as a proxy for neighborhood and other housing factors.³⁹ Also, our one-time measurements may not have accurately

captured biologically relevant exposure with respect to the timing or location of exposure for some children (such as daycare centers),⁴⁰ especially those whose parents moved to the United States from other countries, such as Mexico.⁴¹ In addition, we have not gathered information on potential genetic markers of susceptibility to allergic sensitization.^{3,42} Other investigators have observed variations in asthma prevalence and differences in IgE within Latino populations.^{43,44} Their observations and ours may help to frame the questions that research on gene-environment interactions will need to answer.⁴⁵

In conclusion, we found that concentrations of the major cockroach allergen, Bla g 2, in both bed and kitchen dust were associated with cockroach-specific IgE independent of other factors in a cohort of 4-year-old inner-city children. As with previous studies, we observed an increase in allergen specific IgE with a maternal history of atopy,⁴⁴ male sex,⁴⁶ and ETS,⁴⁷ but adjustment for these covariates did not affect the association between cockroach allergen and sensitization. Although several previous studies of children with asthma have reported an association between cockroach allergen exposure and IgE to cockroach,^{6,8,24} we report an association in a population not restricted to subjects with atopy or asthma. We observed this association even among children without current probable asthma. Given the age of these children and previous studies demonstrating an increased risk for the development of asthma with early life sensitization to inhalant allergens, it is likely that although they do not have asthma symptoms at age 4 years, they are at increased risk for developing asthma as they age.⁴ These findings demonstrate the importance of domestic exposure to cockroach allergen to the development of sensitization and suggest a potential primary prevention target⁴⁸ in this community.

REFERENCES

- Verhoeff AP, Van Strien RT, Van Wijnen JH, Brunekreef B. House dust mite allergen (Der p I) and respiratory symptoms in children: a case-control study. *Clin Exp Allergy* 1994;24:1061-9.
- Almqvist C, Egmar AC, van Hage-Hamsten M, Berglund N, Pershagen G, Nordvall SL, et al. Heredity, pet ownership confounding control in a population-based birth cohort. *J Allergy Clin Immunol* 2003;111:800-6.
- Simpson A, John SL, Jury F, Niven R, Woodcock A, Ollier WE, et al. Endotoxin exposure CD14, and allergic disease: an interaction between genes and the environment. *Am J Respir Crit Care Med* 2006;174:386-92.
- Illi S, von Mutius E, Lau S, Niggemann B, Gruber C, Wahn U, et al. Perinatal allergen sensitization early in life and chronic asthma in children: a birth cohort study. *Lancet* 2006;368:763-70.
- Garg R, Karpati A, Leighton J, Perrin M, Shah M. Asthma facts. 2nd ed. New York: New York City Department of Health and Mental Hygiene; 2003.
- Gruchalla RS, Pongracic J, Plaut M, Evans R, Visness CM, Walter M, et al. Inner City Asthma Study: relationships among sensitivity, allergen exposure, and asthma morbidity. *J Allergy Clin Immunol* 2005;115:478-85.
- Phipatanakul W, Eggleston PA, Wright EC, Wood RA. 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* 2000;106:1075-80.
- Rosenstreich DL, Eggleston P, Kattan M, Baker D, Slavin RG, Gergen P, et al. The role of cockroach allergy and exposure to cockroach allergen in causing morbidity among inner-city children with asthma. *N Engl J Med* 1997;336:1356-63.
- Chew GL, Perzanowski MS, Miller RL, Correa JC, Hoepner LA, Jusino CM, et al. Distribution and determinants of mouse allergen exposure in low-income New York City apartments. *Environ Health Perspect* 2003;111:1348-51.
- Goldstein IF, Reed CE, Swanson MC, Jacobson JS. Aeroallergens in New York inner-city apartments of asthmatics. *Experientia Suppl* 1987;51:133-8.
- Miller RL, Chew GL, Bell CA, Biedermann SA, Aggarwal M, Kinney PL, et al. Prenatal antigen exposure, maternal sensitization, and sensitization in utero to indoor allergens in an inner-city cohort. *Am J Respir Crit Care Med* 2001;164:995-1001.
- Rauh VA, Chew GL, Garfinkel RS. Deteriorated housing contributes to high cockroach allergen levels in inner-city households. *Environ Health Perspect* 2002;110:323-7.
- Call RS, Smith TF, Morris E, Chapman MD, Platts-Mills TAE. Risk factors for asthma in inner city children. *J Pediatr* 1992;121:862-6.
- Ingram JM, Sporik R, Rose G, Honsigner R, Chapman MD, Platts-Mills TAE. Quantitative assessment of exposure to dog (*Can f 1*) and cat (*Fel d 1*) allergens: relation to sensitization and asthma among children living in Los Alamos, New Mexico. *J Allergy Clin Immunol* 1995;96:449-56.
- Holt PG, Thomas WR. Sensitization to airborne environmental allergens: unresolved issues. *Nat Immunol* 2005;6:957-60.
- Chapman MD, Aalberse RC, Brown MJ, Platts-Mills TAE. Monoclonal antibodies to the feline allergen, *Fel d 1*, II: single step affinity purification of *Fel d 1*, N-terminal sequence analysis and development of a sensitive two-site immunoassay to assess *Fel d 1* exposure. *J Immunol* 1988;140:812-8.
- Pollart SM, Mullins DE, Vailes LD, Hayden ML, Platts-Mills TAE, Sutherland WM, et al. Identification, quantitation, and purification of cockroach allergens using monoclonal antibodies. *J Allergy Clin Immunol* 1991;87:511-21.
- Sarpong SB, Hamilton RG, Eggleston PA, Adkinson NF. Socioeconomic status and race as risk factors for cockroach allergen exposure and sensitization in children with asthma. *J Allergy Clin Immunol* 1996;97:1393-401.
- Chew GL, Higgins KM, Muilenberg ML, Gold DR, Burge HA. Monthly measurements of indoor allergens and the influence of housing type in a northeastern US city. *Allergy* 1999;54:1058-66.
- Gelber LE, Seltzer L, Bouzoukis JK, Pollart SM, Chapman MD, Platts-Mills TAE. 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* 1993;147:573-8.
- Peat JK, Tovey E, Toelle BG, Haby MM, Gray EJ, Mahmic A, et al. House dust mite allergens: a major risk factor for childhood asthma in Australia. *Am J Respir Crit Care Med* 1996;153:141-6.
- Perzanowski MS, Ronmark E, Nold B, Lundback B, Platts-Mills TA. Relevance of allergens from cats and dogs to asthma in the northernmost province of Sweden: schools as a major site of exposure. *J Allergy Clin Immunol* 1999;103:1018-24.
- Matsui EC, Wood RA, Rand C, Kanchanaraks S, Swartz L, Eggleston PA. Mouse allergen exposure and mouse skin test sensitivity in suburban, middle-class children with asthma. *J Allergy Clin Immunol* 2004;113:910-5.
- Matsui EC, Wood RA, Rand C, Kanchanaraks S, Swartz L, Curtin-Brosnan J, et al. Cockroach allergen exposure and sensitization in suburban middle-class children with asthma. *J Allergy Clin Immunol* 2003;112:87-92.
- Cole Johnson C, Ownby DR, Havstad SL, Peterson EL. Family history, dust mite exposure in early childhood, and risk for pediatric atopy and asthma. *J Allergy Clin Immunol* 2004;114:105-10.
- Pollart SM, Smith TF, Morris EC, Gelber LE, Platts-Mills TAE, Chapman MD. Environmental exposure to cockroach allergens: Analysis with monoclonal antibody-based enzyme immunoassays. *J Allergy Clin Immunol* 1991;87:505-10.
- Holt PG. The role of genetic and environmental factors in the development of T-cell mediated allergic disease in early life. *Paediatr Respir Rev* 2004;5(suppl A):S27-30.
- Perzanowski MS, Miller RL, Thorne PS, Barr RG, Divjan A, Sheares BJ, et al. Endotoxin in inner-city homes: associations with wheeze and eczema in early childhood. *J Allergy Clin Immunol* 2006;117:1082-9.

29. Rylander R, Holt PG. (1→3)-beta-D-glucan and endotoxin modulate immune response to inhaled allergen. *Mediators Inflamm* 1998;7:105-10.
30. Savilahti R, Uitti J, Roto P, Laippala P, Husman T. Increased prevalence of atopy among children exposed to mold in a school building. *Allergy* 2001;56:175-9.
31. Cohn RD, Arbes SJ, Yin M, Jaramillo R, Zeldin DC. National prevalence and exposure risk for mouse allergen in US households. *J Allergy Clin Immunol* 2004;113:1167-71.
32. Phipatanakul W, Eggleston PA, Wright EC, Wood RA. Mouse allergen I: the prevalence of mouse allergen in inner-city homes. *J Allergy Clin Immunol* 2000;106:1070-4.
33. Kattan M, Mitchell H, Eggleston P, Gergen P, Crain E, Redline S, et al. Characteristics of inner-city children with asthma: the National Cooperative Inner-City Asthma Study. *Pediatr Pulmonol* 1997;24:253-62.
34. Erwin EA, Wickens K, Custis NJ, Siebers R, Woodfolk J, Barry D, et al. Cat and dust mite sensitivity and tolerance in relation to wheezing among children raised with high exposure to both allergens. *J Allergy Clin Immunol* 2005;115:74-9.
35. Ownby DR, Johnson CC, Peterson EL. Exposure to dogs and cats in the first year of life and risk of allergic sensitization at 6 to 7 years of age. *JAMA* 2002;288:963-72.
36. Hesselmar B, Aberg N, Aberg B, Eriksson B, Bjorksten B. Does early exposure to cat or dog protect against later allergy development? *Clin Exp Allergy* 1999;29:611-7.
37. Perzanowski MS, Ronmark E, Platts-Mills TA, Lundback B. Effect of cat and dog ownership on sensitization and development of asthma among preteenage children. *Am J Respir Crit Care Med* 2002;166:696-702.
38. Platts-Mills T, Vaughan J, Squillace S, Woodfolk J, Sporik R. Sensitisation, asthma, and a modified Th2 response in children exposed to cat allergen: a population-based cross-sectional study. *Lancet* 2001;357:752-6.
39. Corburn J, Osleeb J, Porter M. Urban asthma and the neighbourhood environment in New York City. *Health Place* 2006;12:167-79.
40. Domelas de Andrade AD, Charpin D, Birnbaum J, Lanteaume A, Chapman M, Vervloet D. Indoor allergen levels in day nurseries. *J Allergy Clin Immunol* 1995;95:1158-63.
41. Eldeirawi K, McConnell R, Freels S, Persky VW. Associations of place of birth with asthma and wheezing in Mexican American children. *J Allergy Clin Immunol* 2005;116:42-8.
42. Gem JE, Reardon CL, Hoffman S, Nicolae D, Li Z, Roberg KA, et al. Effects of dog ownership and genotype on immune development and atopy in infancy. *J Allergy Clin Immunol* 2004;113:307-14.
43. Lara M, Akinbami L, Flores G, Morgenstern H. Heterogeneity of childhood asthma among Hispanic children: Puerto Rican children bear a disproportionate burden. *Pediatrics* 2006;117:43-53.
44. Scirica CV, Gold DR, Ryan L, Abulkerim H, Celedon JC, Platts-Mills TA, et al. Predictors of cord blood IgE levels in children at risk for asthma and atopy. *J Allergy Clin Immunol* 2007;119:81-8.
45. Salari K, Choudhry S, Tang H, Naqvi M, Lind D, Avila PC, et al. Genetic admixture and asthma-related phenotypes in Mexican American and Puerto Rican asthmatics. *Genet Epidemiol* 2005;29:76-86.
46. Nickel R, Illi S, Lau S, Sommerfeld C, Bergmann R, Kamin W, et al. Variability of total serum immunoglobulin E levels from birth to the age of 10 years: a prospective evaluation in a large birth cohort (German Multicenter Allergy Study). *Clin Exp Allergy* 2005;35:619-23.
47. Kramer U, Lemmen CH, Behrendt H, Link E, Schafer T, Gostomzyk J, et al. The effect of environmental tobacco smoke on eczema and allergic sensitization in children. *Br J Dermatol* 2004;150:111-8.
48. Prevention of allergy and allergic asthma. Geneva: World Health Organization; 2003. Available at: http://whqlibdoc.who.int/hq/2003/WHO_NMH_MNC_CRA_03.2.pdf. Accessed July 31, 2007.