Published in final edited form as:
Ann Allergy Asthma Immunol. 2005 May ; 94(5): 593–599.

Mouse exposure and wheeze in the first year of life

Wanda Phipatanakul, MD, MS[*,†,‡], **Juan C. Celedón, MD, DrPH**[†,‡], **Diane L. Sredl, MPH**[†,‡], **Scott T. Weiss, MD, MS**[†,‡], and **Diane R. Gold, MD, MPH**[†,‡]

* *Division of Allergy and Immunology, Department of Pediatrics, Children's Hospital Boston, Boston, Massachusetts.*

† *Channing Laboratory, Department of Medicine, Brigham and Women's Hospital, Boston, Massachusetts.*

‡ *Harvard Medical School, Boston, Massachusetts.*

Abstract

Background—Studies have found that exposure to mice is highly prevalent among children with asthma living in urban areas.

Objective—To examine the relationship between exposure to mice and wheeze in the first year of life.

Methods—We conducted an ongoing prospective birth cohort study of 498 children with a history of allergy or asthma in at least 1 parent living in metropolitan Boston (the Home Allergens and Asthma Study).

Results—In a multivariate analysis, infants whose parents reported exposure to mice in the household had nearly twice the odds of developing any wheeze in the first year of life as children without exposure (odds ratio [OR], 1.83; 95% confidence interval [CI], 1.14–2.95; $P = .01$). Other variables associated with wheeze in the first year of life included low birth weight (OR, 1.77; 95% CI, 1.06–2.95; $P = .03$), having at least 1 lower respiratory tract illness (OR, 5.59; 95% CI, 3.46–9.04; $P < .001$), exposure to high levels of endotoxin at age 2 to 3 months (fourth quartile compared with first quartile: OR, 2.32; 95% CI, 1.19–4.54; $P = .01$), and exposure to cockroach allergen of 0.05 U/g of dust or more at age 2 to 3 months (OR, 1.83; 95% CI, 1.09–3.08; $P = .02$).

Conclusion—Among children with a parental history of asthma or allergies, exposure to mice is associated with wheeze in the first year of life, independent of other factors.

INTRODUCTION

More than 80% of children with asthma are allergic to 1 or more inhaled allergens,[1,2] and many studies[3–6] have investigated the role of exposure to indoor allergens, such as dust mites, pets, and cockroaches, in early childhood wheeze or asthma. Several studies[7–9] have suggested that mouse allergen is highly prevalent in urban home environments of school-aged children with asthma. Although mouse allergens are well-documented causes of allergic symptoms and wheeze in occupational settings,[10–12] there are no published studies, to our knowledge, that evaluate the relationship between exposure to mice and wheeze in early life.

The Home Allergens and Asthma Study is an ongoing prospective birth cohort study of children with a parental history of asthma or allergies in the Boston metropolitan area. The primary

Requests for reprints should be addressed to: Wanda Phipatanakul, MD, MS, Division of Immunology, Children's Hospital Boston, Harvard Medical School, 300 Longwood Ave, Fegan 6, Boston, MA 02115, E-mail: wanda.phipatanakul@childrens.harvard.edu.

Supported by grants AI/EHS 35785 and ES 07036 from the National Institutes of Health. Dr Phipatanakul is supported by K-23 grant AI 054972 from the National Institutes of Health.

purpose of this study is to evaluate the relationship between exposure to indoor allergens in early childhood and the subsequent development of asthma and allergic disease. In this article, we examine the relationship between mouse exposure and wheeze in the first year of life in children in the Home Allergens and Asthma Study with a parental history of asthma or allergies.

METHODS

Study Participants

A total of 505 infants (including 6 sets of twins) from 499 families with a history of allergy or asthma in at least 1 parent were recruited between September 1, 1994, and August 31, 1996. The screening and recruitment of families have been described elsewhere.[3] In brief, the eligibility criteria included residence inside Route 128 (a highway that encircles the Boston metropolitan area); maternal age of 18 years or older; a history of hay fever, bronchial asthma, or allergies in either parent; and maternal ability to speak English or Spanish. Families were not screened if the newborn was hospitalized in the intensive care unit, if his or her gestational age was younger than 36 weeks, or if he or she had a congenital anomaly. Of the 1,405 families initially screened, 906 were excluded from the study before the first home visit. Reasons for exclusion were reluctance to participate in a longitudinal study (51% of those refusing), plans to move within 1 year (39%), early loss to follow-up (9%), and other (1%).

After obtaining written informed consent from the child's parents, a home visit was made when the child was aged 2 to 3 months, and a questionnaire regarding home characteristics, environmental exposures, cigarette smoking, and demographics was administered by trained research assistants. Every 2 months, beginning when the child was aged 2 months, a follow-up telephone questionnaire was administered to the child's primary caretaker. Of the 505 children, 7 were excluded from analysis because they were followed up for 4 months or less during their first year of life. The study was approved by the institutional review board of the Brigham and Women's Hospital in Boston.

Definition of the Primary Predictor Variable

Parental report of exposure to mice in the home was evaluated every 2 months during the first year of life. At age 2 to 3 months, the primary caretaker was asked, "In the past 12 months, have you seen or noticed signs of mice?" Subsequently, every 2 months the primary caretaker was asked, "In the past 2 months, have you been troubled by any of the following pests (mice)?" Children who lived in homes where the primary caretaker answered "yes" to these questions during the first year of the child's life were categorized as having reported mouse exposure.

Definition of Other Predictor Variables

Potential confounders of the relationship between exposure to mice and wheeze were considered for inclusion in the multivariate models. Sociodemographic variables included the child's race or ethnicity (according to parental report)[13] and the annual household income, classified as less than $30,000 vs $30,000 or more. Perinatal and familial factors included birth weight (classified as 1.84 to <3.79 kg vs 3.79 to <4.91 kg), season of birth (winter, spring, summer, and fall), maternal age (in quartiles), maternal cigarette smoking during pregnancy (yes or no), exclusive and supplemented breast-feeding (yes or no), number of months of breastfeeding (never, <4 months, or ≥4 months), and parental history of asthma, allergic rhinitis, atopic dermatitis, or food allergy (ever or active asthma in the mother, father, or both). Variables related to the home environment included type of dwelling (single-family home vs other), the presence of any pets in the home when the child was 2 to 3 months old, total cigarette smoking (average number of cigarettes per day smoked by all adults in the household), and number of older siblings (aged ≤14 years). Day care attendance in the first year of life was also evaluated (yes or no).[14] Other variables examined in the first year of life included 3 or more

vs fewer than 3 reports of a runny or stuffed nose (recurrent nasal catarrh), any vs no upper respiratory tract illnesses (ear infection or physician-diagnosed sinus infection), and any vs no lower respiratory tract illnesses (croup, bronchitis, bronchiolitis, or pneumonia).

Analysis of House Dust Samples

Methods for the collection of dust samples and the processing and assay of allergens and endotoxin have been detailed previously.[3,15–17] Briefly, when the infant was 2 to 3 months old, 5 separate dust samples were collected in a standard manner during the home visit by vacuuming the following areas: (1) the baby's bedroom floor, (2) the baby's bed, (3) the parent's bed, (4) the family living room floor, and (5) the kitchen floor. Within 24 hours of collection, the dust samples were weighed and then sifted through a 425-μm mesh sieve, and the fine dust was weighed again and divided into aliquots for various analyses—allergens (dust mite, cat, dog, and cockroach) and endotoxin. Allergen concentrations were grouped in categories consistent with potential relevance to sensitization, and endotoxin exposure was categorized into quartiles as previously reported in this cohort.[3,17] Endotoxin was assayed only if there was sufficient dust after all the other assays had been performed. We used data from the living room because this room had the most available dust for analysis.

Analysis of House Dust Samples for Mouse Allergen

Dust samples were removed from the vacuum collection thimble, sieved, extracted, and then stored at −30°C until they were assayed for mouse allergen (mouse urinary protein [MUP]). A competitive enzyme-linked immunosorbent assay with high-titer anti-MUP antibody was used to determine the concentration of MUP in micrograms per gram of dust (Greer Laboratories, Lenoir, NC). The lower limit of detection for the assay was 0.25 μg/g of dust. Because of limited funding and dust sample availability, only dust samples from the kitchen and living room were analyzed for MUP. These dust samples were analyzed for MUP after all the other allergens and endotoxin were analyzed, limiting analyses for mouse allergen to homes with adequate dust samples. Of the 498 homes, 376 (75.5%) had dust samples from the kitchen and 421 (84.5%) had dust samples from the living room that were adequate for analysis.

Definition of the Outcome Variable

Every 2 months during the first year of the child's life, the primary caretaker was asked, "Since we last spoke with you on [date given], has your child had wheezing or whistling in the chest?" The outcome variable of interest was no vs any report of wheeze (any wheeze) in the first year of life. Children whose primary caretakers answered "yes" to this question in the child's first year of life were categorized as having any wheeze in the first year of life.

Statistical Methods

The univariate analysis of the relationship between predictor variables and wheeze in the first year of life used 2 × 2 contingency tables for categorical variables and *t* tests for continuous variables. We used logistic regression analysis to study the relationship between report of mouse exposure and wheeze while controlling for potential confounders and examining interactions. Stepwise logistic regression analysis was used to develop the multivariate models. An indicator variable for missing values was included in the multivariate analysis to allow us to control for the missing values in the models. In the final models, we included variables that satisfied a change-in-estimate criterion (≥10% in the odds ratio estimate) or that were significant at $P < .05$.

RESULTS

Table 1 summarizes the primary characteristics of the 498 children followed up throughout the first year of life. Most of the participating children were white and came from households with annual incomes of at least $30,000. Of the 498 participating children, 132 (26.5%) lived in homes where there was a report of exposure to mice, and 197 children (39.6%) developed wheeze in the first year of life. Of the 132 children who lived in households that reported mouse exposure, parental report of mouse exposure preceded parental report of any wheeze in 126 children (95.5%). Of the 376 kitchen dust samples adequate for analysis, mouse allergen was detectable in 119 (31.6%). Of the 421 living room dust samples adequate for analysis, mouse allergen was detectable in 140 (33.3%). Because values for mouse allergen in the kitchen were missing for 122 infants, we compared the characteristics of these infants with those of the 376 infants for whom dust samples were adequate for analysis. The characteristics of children with and without complete data on mouse allergen levels in the kitchen were similar, without any statistically significant differences except for spring season of birth (Table 1).

Table 2 summarizes the results of the univariate analysis of the relationship between selected variables and any wheeze in the first year of life. Parental report of exposure to mice was significantly associated with increased odds of wheeze in the first year of life ($P = .004$). For predictor variables other than mouse exposure, previous studies from this cohort examined repeated wheeze[3] and any wheeze[15] in the first year of life, with similar results. In brief, variables associated with increased odds of any wheeze in the first year of life included household income less than $30,000, birth weight less than 3.79 kg, being born in the spring, day care attendance, recurrent nasal catarrh, having at least 1 upper respiratory tract illness, having at least 1 lower respiratory tract illness, and exposure to cockroach allergen levels of 0.05 U/g of dust or more. Exposure to dog allergen levels of 20 to less than 200 μg/g of dust and exposure to dust mite allergen of 2 μg/g of dust or greater were associated with decreased odds of wheeze in the first year of life.

Table 3 provides the results of the multivariate analysis of the relationship between mouse exposure and wheeze in the first year of life. Model 1 shows that infants who lived in households that reported exposure to mice had nearly twice the odds of having wheeze in the first year of life as those who reported no such exposure ($P = .01$). In model 1, variables associated with increased odds of wheeze in the first year of life included birth weight less than 3.79 kg ($P = .03$), having at least 1 lower respiratory tract illness ($P < .001$), exposure to high levels of endotoxin at age 2 to 3 months ($P = .01$), and exposure to cockroach allergen of 0.05 U/g of dust or more at age 2 to 3 months ($P = .02$). In this analysis, exposure to dog allergen levels ranging from 20 to less than 200 μg/g of dust was inversely associated with wheeze in year 1. Model 2 shows the multivariate analysis with detectable mouse allergen in the kitchen and wheeze in the first year of life. In this model, detectable mouse allergen in the kitchen was positively, but only weakly, associated with wheeze in the first year of life ($P = .08$). Detectable mouse allergen in the living room was also only weakly associated with wheeze in the first year of life (data not shown).

Because there was a linear relationship between increases in endotoxin levels by quartiles and wheeze, we repeated the multivariate analysis including endotoxin as an ordinal variable and obtained similar results (data not shown). We also repeated the analysis after excluding children whose parents reported exposure to mice after reporting any wheeze and obtained similar results (data not shown). We found no significant modification of the effect of exposure to mice on wheeze in the first year of life by any of the variables included in the multivariate analysis.

DISCUSSION

Among children with a parental history of asthma or allergies, we found that reported exposure to mice was associated with wheeze in the first year of life. To our knowledge, this is the first study of an association between exposure to mice and wheeze in early childhood.

Exposure to mice is a well-documented cause of symptoms of rhinitis and asthma in adults in occupational settings.[11,18] Previous studies[19–23] have suggested that mouse-induced occupational asthma is caused by sensitization to high-molecular-weight antigens found in MUPs. However, Gautrin and associates[24] found that many laboratory workers report symptoms of rhinitis and asthma without mouse allergen sensitization, suggesting that mouse exposure may have an irritant effect on the airways. In our study, wheeze in the first year of life may not represent allergic wheeze because these infants may not necessarily develop persistent wheeze or asthma later in life.[25] It is thus possible that infant mouse exposure increases the risk of airway inflammation through nonallergic and allergic mechanisms.

The role of exposure to mice in the home environments of children with asthma has only recently been evaluated. These studies[7,9,26,27] have primarily evaluated urban environments and suggest that exposure to mice is highly prevalent in these homes. In the National Cooperative Inner-City Asthma Study (NCICAS),[7] 49% of the homes had reported problems with mice, and this was highly associated with mouse allergen levels greater than the median level for each room. Similar associations were seen in 221 urban New York City apartments, in which Chew et al[26] found that the positive predictive value for detectable mouse allergen levels was 90% in households that reported exposure to mice. In 209 rural Appalachian school-aged children with asthma, Welch and associates[28] found that 33% of parents reported mice in the home.

Although we found an association between reported exposure to mice and wheeze in early life, this association was weaker with respect to mouse allergen levels in house dust samples collected at age 2 to 3 months. However, we had limited statistical power to detect an association between mouse allergen level in house dust and wheezing because of the lack of measurements of mouse allergen in many of the samples. In addition, we obtained data on reported mouse exposure every 2 months during the first year of life, whereas our dust samples were collected only once, at age 2 to 3 months. Therefore, our reported exposure may have been a more accurate evaluation of current exposure during the first year of life rather than past exposure. Perhaps current exposure to mice has a more significant association with current symptoms. Although not as clinically relevant, we analyzed detectable mouse allergen with report of wheeze by 4 months of age, and the relationship was stronger but still not statistically significant, with a very wide confidence interval. This supports our theory that current exposure may be more significantly associated with current symptoms, but we likely had limited statistical power from less outcomes of wheeze by age 4 months and available data from dust samples. Furthermore, findings from previous studies[7,26] in urban environments suggest that reported exposure to mice is a good marker of exposure to mouse allergen.

Among 167 mothers of an urban New York birth cohort, Miller and colleagues[29] found that infants of mothers exposed to mouse allergen may develop allergy in utero as detected by cord blood allergen-induced cellular proliferation. In the NCICAS,[8] mouse allergen exposure was associated with mouse allergen sensitization in children allergic to other allergens. Although data from the NCICAS[8] suggested a possible relationship between mouse exposure and asthma symptoms, the high correlation between mouse and cockroach in the urban environment limited the certainty of these relationships. Our study of infants from urban and suburban environments suggests a relationship between mouse exposure and wheeze even after adjustment for

socioeconomic factors commonly found in urban environments, such as cockroach allergen and household income.

We recognize several limitations of this study. Report of mouse exposure may be biased because some parents of infants in homes with low mouse allergen levels may not report mouse exposure. We also did not obtain mouse allergen measurements every 2 months as we did in parental report of exposure to mice, and the power obtained from our 1-time dust sample measurement at age 2 to 3 months was limited. Previous studies in urban home environments have shown that families that report mouse exposure have high mouse allergen levels. Therefore, we believe that we may have underestimated rather than overestimated the strength of the association between mouse exposure and wheeze. In addition, parental report of wheeze may reflect parental reporting bias. However, parental reports of wheeze in our cohort and others[30] have been shown to be reliable in determining risk factors for wheeze or asthma. In addition, we did not have mouse allergen sensitization data available for this analysis of early life exposure. Because exposure is often related to sensitization, future analysis of sensitization is desirable. Finally, this study was not designed to be a random sample of the population in the greater Boston area because we selected a stable population with a parental history of allergy or asthma, which limits the generalizability of our study. However, our study is representative of those most at risk of developing asthma.

In conclusion, we found that exposure to mice is a risk factor for wheeze in the first year of life among children with a parental history of asthma or allergies, independent of other factors. Further study of this cohort will help us examine whether early exposure to mice predicts allergen sensitization or the development of asthma later in childhood.

Acknowledgements

We thank the study participants and Jaylyn Olivo for her editorial assistance.

References

1. Niemeijer NR, de Monchy JG. Age-dependency of sensitization to aero-allergens in asthmatics. Allergy 1992;47(pt 2):431–435. [PubMed: 1456415]
2. Schwartz J, Weiss ST. Relationship of skin test reactivity to decrements in pulmonary function in children with asthma or frequent wheezing. Am J Respir Crit Care Med 1995;152(pt 1):2176–2180. [PubMed: 8520794]
3. Gold DR, Burge HA, Carey V, Milton DK, Platts-Mills T, Weiss ST. Predictors of repeated wheeze in the first year of life: the relative roles of cockroach, birth weight, acute lower respiratory illness, and maternal smoking. Am J Respir Crit Care Med 1999;160:227–236. [PubMed: 10390405]
4. Celedon JC, Litonjua AA, Ryan L, Platts-Mills T, Weiss ST, Gold DR. Exposure to cat allergen, maternal history of asthma, and wheezing in first 5 years of life. Lancet 2002;360:781–782. [PubMed: 12241839]
5. Platts-Mills T. Major risk factors according to age: the relevance of indoor allergens to the increase in asthma. In: Neffen HE, Baena-Cagnani CE, Fabbri L, Holgate ST, O'Byrne PO, eds. *Asthma: A Link Between Environment, Immunology and the Airways.* Seattle, WA: Hogrefe & Huber Publishers; 1999:200–208.
6. Platts-Mills TA, Blumenthal K, Perzanowski M, Woodfolk JA. Determinants of clinical allergic disease: the relevance of indoor allergens to the increase in asthma. Am J Respir Crit Care Med 2000;162(pt 2):S128–S133. [PubMed: 10988167]
7. Phipatanakul W, Eggleston PA, Wright EC, Wood RA. Mouse allergen, I: the prevalence of mouse allergen in inner-city homes: the National Cooperative Inner-City Asthma Study. J Allergy Clin Immunol 2000;106:1070–1074. [PubMed: 11112888]
8. 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: the

National Cooperative Inner-City Asthma Study. J Allergy Clin Immunol 2000;106:1075–1080. [PubMed: 11112889]

9. Stelmach I, Jerzynska J, Stelmach W, Majak P, Chew G, Kuna P. The prevalence of mouse allergen in inner-city homes. Pediatr Allergy Immunol 2002;13:299–302. [PubMed: 12390447]
10. Bush RK, Wood RA, Eggleston PA. Laboratory animal allergy. J Allergy Clin Immunol 1998;102:99–112. [PubMed: 9679853]
11. Bush RK. Mechanism and epidemiology of laboratory animal allergy. ILAR J 2001;42:4–11. [PubMed: 11123184]
12. Phipatanakul W. Rodent allergens. Curr Allergy Asthma Rep 2002;2:412–416. [PubMed: 12165208]
13. Litonjua AA, Carey VJ, Burge HA, Weiss ST, Gold DR. Parental history and the risk for childhood asthma: does mother confer more risk than father? Am J Respir Crit Care Med 1998;158:176–181. [PubMed: 9655726]
14. Celedon JC, Litonjua AA, Weiss ST, Gold DR. Day care attendance in the first year of life and illnesses of the upper and lower respiratory tract in children with a familial history of atopy. Pediatrics 1999;104(pt 1):495–500. [PubMed: 10469775]
15. Park JH, Gold DR, Spiegelman DL, Burge HA, Milton DK. House dust endotoxin and wheeze in the first year of life.[comment]. Am J Respir Crit Care Med 2001;163:322–328. [PubMed: 11179100]
16. Litonjua AA, Milton DK, Celedon JC, Ryan L, Weiss ST, Gold DR. A longitudinal analysis of wheezing in young children: the independent effects of early life exposure to house dust endo-toxin, allergens, and pets. J Allergy Clin Immunol 2002;110:736–742. [PubMed: 12417882]
17. Phipatanakul W, Celedon JC, Raby BA, et al. Endotoxin exposure and eczema in the first year of life. Pediatrics 2004;114:13–18. [PubMed: 15231902]
18. Cullinan P, Lowson D, Nieuwenhuijsen MJ, et al. Work related symptoms, sensitisation, and estimated exposure in workers not previously exposed to laboratory rats. Occup Environ Med 1994;51:589–592. [PubMed: 7951789]
19. Schumacher MJ. Clinically relevant allergens from laboratory and domestic small animals. N Engl Reg Allergy Proc 1987;8:225–231. [PubMed: 3118159]
20. Taylor AN, Longbottom JL, Pepys J. Respiratory allergy to urine proteins of rats and mice. Lancet 1977;2:847–849. [PubMed: 72196]
21. Twiggs JT, Agarwal MK, Dahlberg MJ, Yunginger JW. Immunochemical measurement of airborne mouse allergens in a laboratory animal facility. J Allergy Clin Immunol 1982;69:522–526. [PubMed: 7076993]
22. Gautrin D, Ghezzo H, Infante-Rivard C, Malo JL. Host determinants for the development of allergy in apprentices exposed to laboratory animals. Eur Respir J 2002;19:96–103. [PubMed: 11843334]
23. Gautrin D, Infante-Rivard C, Ghezzo H, Malo JL. Incidence and host determinants of probable occupational asthma in apprentices exposed to laboratory animals. Am J Respir Crit Care Med 2001;163:899–904. [PubMed: 11282763]
24. Gautrin D, Ghezzo H, Infante-Rivard C, Malo JL. Natural history of sensitization, symptoms and occupational diseases in apprentices exposed to laboratory animals. Eur Respir J 2001;17:904–908. [PubMed: 11488324]
25. Martinez FD, Wright AL, Taussig LM, Holberg CJ, Halonen M, Morgan WJ. Asthma and wheezing in the first six years of life: the Group Health Medical Associates. N Engl J Med 1995;332:133–138. [PubMed: 7800004]
26. Chew GL, Perzanowski MS, Miller RL, et al. Distribution and determinants of mouse allergen exposure in low-income New York City apartments. Environ Health Perspect 2003;111:1348–1351. [PubMed: 12896857]
27. Brugge D, Vallarino J, Ascolillo L, Osgood ND, Steinbach S, Spengler J. Comparison of multiple environmental factors for asthmatic children in public housing. Indoor Air 2003;13:18–27. [PubMed: 12608922]
28. Welch JE, Hogan MB, Wilson NW. Mouse allergy among asthmatic children from rural Appalachia. Ann Allergy Asthma Immunol 2003;90:223–225. [PubMed: 12602670]
29. Miller RL, Chew GL, Bell CA, et al. Prenatal 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. [PubMed: 11587985]

30. Castro-Rodriguez JA, Holberg CJ, Wright AL, Martinez FD. A clinical index to define risk of asthma in young children with recurrent wheezing. Am J Respir Crit Care Med 2000;162(pt 1):1403–1406. [PubMed: 11029352]

Table 1

Characteristics of Children in the Cohort

Covariate	Children, No. (%)		Total (N = 498)
	With measurement of mouse allergen in kitchen (N = 376)	Without measurement of mouse allergen in kitchen (N = 122)	
Report of mouse exposure			
Yes	96 (25.5)	36 (29.5)	**132** (26.5)
No	280 (74.5)	86 (70.5)	**366** (73.5)
Detectable mouse allergen (kitchen)			
Yes	119 (31.6)	NA	**119** (23.9)
No	257 (68.4)	NA	**379** (76.1)
Sex			
M	203 (54.0)	65 (53.3)	**268** (53.8)
F	173 (46.0)	57 (46.7)	**230** (46.2
Race			
White	281 (74.7)	94 (77.1)	**375** (75.3)
Black	48 (12.8)	12 (9.8)	**60** (12.1)
Hispanic	21 (5.6)	7 (5.7)	**28** (5.6)
Asian/others	26 (6.9)	9 (7.4)	**35** (7.0)
Household income, $[*]			
<30,000	37 (10.1)	8 (6.8)	**45** (9.3)
≥30,000	329 (89.9)	110 (93.2)	**439** (90.7)
Type of housing			
Single family, detached	201 (53.5)	65 (53.3)	**266** (53.4)
Single family, attached	20 (5.3)	5 (4.1)	**25** (5.0)
2 family	69 (18.4)	26 (21.3)	**95** (19.1)
Apartment/other	86 (22.9)	26 (21.3)	**112** (22.5)
Maternal history of asthma			
Yes	117 (31.1)	35 (28.7)	**152** (30.5)
No	259 (68.9)	87 (71.3)	**346** (69.5)
Paternal history of asthma[*]			
Yes	93 (25.2)	23 (19.0)	**116** (23.7)
No	276 (74.8)	98 (80.1)	**374** (76.3)
Maternal smoking during pregnancy			
Yes	28 (7.4)	5 (4.1)	**33** (6.6)
No	348 (92.6)	117 (95.9)	**465** (93.4)
Season of birth			
Winter (Dec–Feb)	100 (26.6)	24 (19.7)	**124** (24.9)
Spring (Mar–May)[†]	98 (26.1)	46 (37.7)	**144** (28.9)
Summer (Jun–Aug)	89 (23.7)	21 (17.2)	**110** (22.1)
Fall (Sep–Nov)	89 (23.7)	31 (25.4)	**120** (24.1)
Breastfed			
Yes	276 (73.4)	96 (78.7)	**372** (74.7)
No	100 (26.6)	26 (21.3)	**126** (25.3)
Day care attendance			
Yes	183 (48.7)	55 (45.1)	**238** (47.8)
No	193 (51.3)	67 (54.9)	**260** (52.2)

Abbreviation: NA, not applicable.

[*] There was missing information on paternal history of asthma (n = 8) and household income (n = 14).

[†] $P < .05$ with vs without measurement of mouse allergen.

Table 2

Univariate Analysis of Predictors of Wheeze in the First Year of Life

Covariate	Children, No. (%)		OR (95% CI)
	Wheeze (n = 197)	No wheeze (n = 301)	
Report of mouse exposure			
Yes†	66 (33.5)	66 (21.9)	1.79 (1.20–2.68)
Detectable mouse allergen (kitchen)			
Yes	49 (34.5)	70 (29.9)	1.23 (0.79–1.93)
Sex			
M	99 (50.2)	169 (56.2)	1.00
F	98 (49.8)	132 (43.8)	1.27 (0.88–1.82)
Household income*			
<$30,000†	24 (12.6)	21 (7.2)	1.86 (1.01–3.44)
Paternal history of asthma*			
Yes	41 (21.1)	75 (25.3)	0.79 (0.51–1.22)
Maternal cigarette smoking in pregnancy			
Yes	17 (8.6)	16 (5.3)	1.68 (0.83–3.41)
Birth weight, kg*			
3.79 to <4.91	37 (18.8)	87 (29.0)	1.00
<3.79†	160 (81.2)	213 (71.0)	1.77 (1.14–2.73)
Season of birth			
Winter (Dec–Feb)	39 (19.8)	85 (28.2)	1.00
Spring (Mar–May)†	63 (32.0)	81 (26.9)	1.70 (1.03–2.80)
Summer (Jun–Aug)	43 (21.8)	67 (22.3)	1.40 (0.82–2.40)
Fall (Sep–Nov)	52 (26.4)	68 (22.6)	1.67 (0.99–2.81)
Day care attendance			
Yes†	105 (53.3)	133 (44.2)	1.44 (1.01–2.06)
≥3 episodes of nasal catarrh			
Yes†	179 (90.9)	242 (80.4)	2.42 (1.38–4.25)
≥1 upper respiratory tract illness‡			
Yes†	149 (75.6)	175 (58.1)	2.24 (1.50–3.33)
≤1 lower respiratory tract illness§			
Yes†	85 (43.2)	47 (15.6)	4.10 (2.69–6.24)
Endotoxin levels, EU/mg*			
First quartile (range, 2.14–52.48)	34 (21.4)	66 (27.3)	1.00
Second quartile (range, 52.50–80.00)	36 (22.6)	64 (26.5)	1.09 (0.61–1.95)
Third quartile (range, 80.48–123.19)	43 (27.0)	58 (24.0)	1.44 (0.81–2.55)
Fourth quartile (range, 125.61–713.20)	46 (28.9)	54 (22.3)	1.65 (0.93–2.93)
Cockroach allergen levels, U/g of dust*			
<0.05	129 (66.5)	231 (76.7)	1.00
≥0.05†	52 (26.8)	57 (18.9)	1.63 (1.06–2.52)
Dog allergen levels, μg/g*			
<20	134 (82.2)	208 (82.9)	1.00
≥20 to <200†	5 (3.1)	22 (8.8)	0.35 (0.13–0.95)
≥200	24 (14.7)	21 (8.4)	1.77 (0.95–3.31)
Cat allergen levels, μg/g of dust*			
<1	72 (36.7)	87 (28.9)	1.00
≥1 to <8	75 (38.3)	131 (43.5)	0.69 (0.45–1.05)
≥8	49 (25.0)	83 (27.6)	0.71 (0.44–1.14)
Dust mite allergen levels, μg/g of dust*			
<0.05	19 (9.8)	14 (4.6)	1.00
≥0.05 to <2	87 (44.8)	132 (43.8)	0.49 (0.23–1.02)
≥2 to <10†	40 (20.6)	73 (24.2)	0.40 (0.18–0.89)
≥10†	48 (24.7)	82 (27.2)	0.43 (0.10–0.94)

Abbreviations: CI, confidence interval; OR, odds ratio.

*There was missing information on paternal history of asthma (n = 8), household income (n = 14), birth weight (n = 1), mouse allergen (n = 122), endotoxin levels (n = 97), dog allergen (n = 84), cockroach allergen (n = 3), dust mite allergen (n = 3), and cat allergen (n = 1). Allergen levels and endotoxin levels are from living room house dust samples collected when the child was aged 2 to 3 months.

†$P < .05$.

‡Defined as ear infection or physician-diagnosed sinus infection.

§Defined as croup, bronchitis, bronchiolitis, or pneumonia.

Table 3

Multivariate Analysis of the Relationship Between Mouse Exposure and Wheeze in the First Year of Life*

Covariate	OR (95% CI)	
	Model 1	Model 2
Report of mouse exposure (no)		
Yes	1.83 (1.14–2.95)	NA
Detectable mouse allergen (kitchen)		
Yes	NA	1.34 (0.80–2.27)
Birth weight (3.79 to <4.91 kg)		
<3.79 kg	1.77 (1.06–2.95)	1.77 (1.06–2.94)
≥1 lower respiratory tract illness (no)		
Yes	5.59 (3.46–9.04)	5.66 (3.51–9.15)
Endotoxin levels (first quartile)		
Second quartile	1.19 (0.61–2.33)	1.20 (0.61–2.36)
Third quartile	1.86 (0.95–3.63)	1.80 (0.92–3.52)
Fourth quartile	2.32 (1.19–4.52)	2.39 (1.22–4.68)
Cockroach allergen (<0.05 U/g of dust)		
≥0.05 U/g of dust	1.83 (1.09–3.08)	1.96 (1.17–3.31)
Dog allergen (<20 μg/g of dust)		
≥20 to <200 μg/g of dust	0.14 (0.03–0.64)	0.15 (0.03–0.69)
≥200 μg/g of dust	1.52 (0.75–3.11)	1.55 (0.76–3.16)

Abbreviations: CI, confidence interval; NA, not applicable; OR, odds ratio.

* Models also adjust for sex, household income, and paternal history of asthma.