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ORIGINAL ARTICLE

Environmental Allergens and Asthma Morbidity in Low-Income Children

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Asthma morbidity is high in inner-city children in the United States, which may be related in part to increased allergens in poorly maintained housing. This study examined asthma morbidity in relation to mold, cockroach, dust mite, and cat allergens in the homes of 61 low-income Chicago children with asthma. Children exposed to higher levels of *Penicillium* in the bedroom had more frequent asthma symptoms, whereas those exposed to higher levels of cockroach allergen in the bedroom had a higher number of asthma symptoms. Respiratory infections confounded the association of cockroach allergen with number of asthma symptoms.

Keywords allergens, asthma, mold, cockroach, *Penicillium*

INTRODUCTION

In the United States, asthma morbidity and mortality are highest in low-income minority populations. Previous studies have suggested that asthma morbidity is affected by the levels of indoor allergens, such as dust mites, cockroaches, cats, and fungi (1–10). The relationships of allergens to triggering of asthma symptoms are complex for a number of reasons including sensitization and exposure of individuals to more than one allergen, differing allergen reactivity levels between individuals, and variations in other factors related to asthma morbidity, such as respiratory infections, air pollution, dampness, weather, and medication use. Clinical trial data suggest that avoidance of dust mite allergens results in improvement of asthma symptoms (11, 12). Multifaceted intervention trials have also been shown to reduce morbidity (13, 14), possibly due to the reduction of multiple asthma triggers.

The purpose of this study was to examine associations of respiratory symptoms and allergens collected in selected rooms of the homes of low-income children with asthma. The data were collected at the baseline of an intervention trial that was designed to test the effectiveness of peer education on modifying levels of indoor allergens in a low-income Chicago population previously shown by our group to have high rates of asthma prevalence, morbidity, and mortality (15, 16). Overall methods, as well as a portion of the baseline data and compliance in the trial, have been described in two previous publications (17, 18). This article examines associations of asthma symptoms and allergens at baseline.

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METHODS

The project was designed as a randomized trial of a home intervention in 61 families who had children 3 to 13 years of age with asthma. Families who reported having a child with diagnosed asthma were recruited from Head Start sites in West Town and Humboldt Park as well as from Erie Family Health Center, a community-based health center serving the area. Recruitment occurred over a 21-month period in 1996 to 1998 as previously described (17). This study was approved by the Office for the Protection of Research Subjects at the University of Illinois at Chicago.

Before randomization, families met with the educator at the site of recruitment to establish rapport before going into the home, the study was explained, and they signed informed consent. Subsequent visits by the educator were in the family's home. Educators collected asthma symptom data, a detailed home assessment, including presence of smokers in the home, and environmental samples of dust and air during the home visits, but only baseline data are used in this analysis to avoid potential bias from changes in the outcomes induced by the multifaceted intervention.

Dust samples were collected at baseline from the child's mattress, bedroom floor, and living room floor using standardized collection and analysis methods that have been previously described (17). Dust was screened with a 650 μm filter, extracted in phosphate buffered saline, and analyzed for dust mite (Der p1 and Der f1), cockroach (Bla g1), and cat allergen (Fel d1) with the sandwich enzyme-linked immunosorbent assay (ELISA) method of Chapman et al. (19–21). Airborne fungi were collected in the kitchen and child's bedroom and at an outdoor site at the University of Illinois at Chicago with Andersen (Atlanta, Georgia) one-stage bioaerosol samplers (N-6) loaded with malt extract agar media plates as previously described (17). Plates were incubated at 25°C for 5 to 7 days; viable fungi colonies were counted and classified to genera. The colony-forming units per cubic meter of air (CFU/m^3) were corrected for undercounting, when necessary (22).

The child's primary caregiver was questioned about the occurrence of asthma and respiratory symptoms during the prior month including (1) attacks of wheezing; (2) sleep disturbed due to wheezing; (3) speech limited to one or two words at a time between breaths by wheezing; (4) wheezing during or after exercise; (5) dry cough at night apart from a cough associated with a cold or chest infection; (6) asthma symptoms interfering with daily activities; and (7) missing Head Start, day care, or school due to asthma. The morbidity questions were similar to those used in the International Study of Allergies and Asthma in Childhood (23) but referred to a time period of a month rather than a year. Two ordinal composite symptom variables were created from the reported symptoms. The first summed the total number of symptoms reported of the seven symptoms listed above (0–1, 2–3, 4–5, and 6–7 symptoms). The second quantified frequent symptoms into three categories: none, frequent night or day time symptoms, and frequent night and day time symptoms. Frequent night time symptoms were defined as one or more nights per week with dry cough or sleep disturbed by wheezing, and frequent day symptoms were either four or more wheezing attacks or four or more days of missed school for asthma in the last month.

To select covariates for the analysis, the associations of asthma symptoms and allergens with demographic and environmental factors were tested using non-parametric analytical methods, Chi-square tests, Wilcoxon rank sum tests, Kruskal-Wallis tests, and Spearman correlation coefficients as appropriate. Factors considered as possible covariates included: age, ethnicity, gender, Spanish spoken at home, family history of asthma, age at asthma diagnosis, number of people living in home, and sampling season (fall = September–November, winter = December–February, spring = March–May, and summer = June–August). Of these, ethnicity, age, gender, and fall season were selected as covariates. Ethnicity was coded as African American or Puerto Rican heritage versus other (predominantly Hispanic) because asthma morbidity was similar in African American and Puerto Rican children and higher in these two groups compared to the "other" ethnicity group.

Associations of asthma measures with allergens were evaluated in proportional odds logistic models, controlling for age, gender, ethnicity, and fall season. Three types of predictor variables were used in different models: individual allergen tertiles, the linear trend over allergen tertiles, and the linear trend using the natural log transformed allergen variable. Models were further adjusted for potential confounders of asthma symptom/allergen associations: parentally-reported respiratory infections in the last 30 days, measured levels of outdoor fungi, average daily dew point temperature in the 30 days before sampling, observed mold and dampness in the home, humidifier in the home, smoking in the home, and use of asthma controller medication (inhaled steroids or mast cell stabilizer). Daily dew point temperatures, which incorporate both temperature and humidity, for O'Hare airport in Chicago were obtained from the National Climatic Data Center. Allergen data from the living area (kitchen or living room) and from the bedroom were analyzed separately. Dust allergen data from the bedroom floor and mattress averaged before analysis, but similar results were obtained in analyses using the highest dust allergen level from the bedroom.

TABLE 1.—Demographics and environmental factors for 61 children with asthma.

Characteristic	Percent Yes or median minimum maximum
Male gender, %	63.9
Black or Puerto Rican race, %	55.7
Language spoken in home	
English, %	30
Spanish, %	35
Both, %	35
Age in years, median, minimum, maximum	5, 3, 13
Age at asthma diagnosis in years, median, minimum, maximum	1.5, 0, 10
History of asthma in parent or sibling, %	65.6
Sampling season	
Fall, %	29.5
Winter, %	19.7
Spring, %	39.3
Summer, %	16.4
Number of residents in home, median, minimum, maximum	5, 2, 9 5, 2, 9
Respiratory infection in the last month, %	44.3
Smoker in home, %	42.6
Inhaled asthma controller medication, %	54.1
30-day average dew point temperature, °C, median, minimum, maximum	32, 13, 62
Dampness in home, %	57.4
Humidifier in home, %	41.0
Outdoor total fungi, CFU/m^3, median, minimum, maximum	540, 30, 2840
Outdoor *Penicillium*, CFU/m^3, median, minimum, maximum	129, <LOD, 1452

LOD = limit of detection; CFU = colony forming units.

RESULTS

Demographic characteristics of the children in the study group are shown in Table 1. Composite asthma morbidity measures are shown in Table 2. A high proportion of caretakers reported asthma symptoms in the children during the month before sampling, with 87% of caretakers reporting at least one symptom. Respiratory infections and number of symptoms were significantly higher in children with black/Puerto Rican ethnicity and in the fall season, whereas frequency of symptoms was significantly higher in children with black/Puerto Rican ethnicity (not shown). Distribution and concentration of dust-borne house dust mite, cockroach, and cat allergens as well as air borne total fungi and

TABLE 2.—Composite asthma morbidity measures for in the month before sampling in 61 children with asthma.

Morbidity measure	Percent
Number of asthma symptoms, %*	
0–1	36.1
2–3	18.0
4–5	29.5
6–7	16.4
Frequent asthma symptoms, %**	
None	42.6
Day or night time	29.5
Day and night time	27.9

*Asthma symptoms include attacks of wheezing, speech limited to one or two words at a time between breaths by wheezing, wheezing during or after exercise, sleep disturbed due to wheezing, dry cough at night apart from a cough associated with a cold or chest infection, asthma symptoms interfered with daily activities, and missed school due to asthma.

**Frequent asthma symptoms were defined as: day time symptoms = 4 or more wheezing episodes/month or days of missed school/month; night time symptoms = 1 or more nights/week dry cough or sleep disturbed by wheezing.

TABLE 3.—Allergens in the bedrooms of 61 children with asthma.

Allergen	Median	Tertiles		
		First	Second	Third
Cat (Fel d1, μg/g)	0.43	<LOD–0.10	0.11–2.18	2.19–579.7
Cockroach (Bla g1, U/g)	38.0	1.9–14.3	15.8–72.6	85.8–2285.2
House dust mite (sum of Der p1 & Der f1, μg/g)	0.35	<LOD–0.13	0.14–0.96	1.11–86.1
Total fungi (CFU/m^3)	540	<LOD–264	330–704	797–6732
Penicillium (CFU/m^3)	154	<LOD–44	50–308	330–6606

LOD = limit of detection; CFU = colony-forming units.

Penicillium are shown in Table 3. Lower levels of *Penicillium* and total fungi were found in the fall season, and lower levels of *Penicillium* and higher levels of cat allergen were found in homes of children of black/Puerto Rican ethnicity (not shown). Indoor and outdoor total fungi and *Penicillium* levels were not significantly correlated (not shown).

Frequent asthma symptoms were associated with bedroom *Penicillium* and the association of number of asthma symptoms with *Penicillium* neared significance, adjusting for age, gender, ethnicity, and fall season (Table 4). The number of asthma symptoms, but not frequent symptoms, was associated with cockroach allergens from the bedroom, adjusting for age, gender, ethnicity, and fall season (Table 4). Cat allergen, house dust mite allergen, and total fungi from the bedroom were not associated with number or frequency of asthma symptoms (Table 4). Cockroach, cat, and house dust mite dust allergens from the living room and *Penicillium* and total fungi from the kitchen were not significantly associated with the number and frequency of asthma symptoms (not shown).

The associations of asthma morbidity with allergens were further adjusted for potential confounding factors that may also trigger asthma symptoms, including respiratory infections, tobacco smoke, outdoor fungi, and home dampness. Respiratory infections were significantly associated with cockroach allergen levels and with the number of asthma symptoms (not shown). After control for respiratory infection, the association of the number of asthma symptoms with bedroom cockroach allergen did not remain significant (Table 4). Cigarette smoking by household members and outdoor fungi levels were not significantly associated with asthma symptoms, and adjusting for these factors individually did not alter the significance of any symptom/exposure relationship (Table 4). Adjusting models individually for measures of home dampness (leaks or mold in the home, having a humidifier in the home, and the average daily dew point temperature for 30 days before sampling) did not attenuate significant associations of asthma morbidity with allergens (Table 4), although having a humidifier was significantly associated with the number and frequency of asthma symptoms (not shown). Adjusting for child's use of asthma controller mediation did not alter the significance of any symptom/exposure relationship (Table 4).

TABLE 4.—Associations of asthma morbidity with bedroom allergens in inner-city children with asthma.

Allergen	Number of symptoms*	Frequent symptoms†	
Cat	Tertile 1, OR‡	1.0	1.0
	Tertile 2, OR (95% CI)‡	2.4 (0.7, 8.7)	1.7 (0.5, 6.2)
	Tertile 3, OR (95% CI)‡	1.5 (0.4, 5.4)	1.8 (0.5, 6.8)
	Trend over Tertiles§	$p = 0.59$	$p = 0.38$
	Linear Trend‡	$p = 0.38$	$p = 0.35$
Dust Mites	Tertile 1, OR‡	1.0	1.0
	Tertile 2, OR (95% CI)‡	1.2 (0.4, 3.8)	1.0 (0.3, 3.3)
	Tertile 3, OR (95% CI)‡	1.0 (0.3, 3.2)	1.9 (0.6, 6.3)
	Trend over Tertiles§	$p = 0.99$	$p = 0.31$
	Linear Trend#	$p = 0.57$	$p = 0.83$
Cockroach	Tertile 1, OR‡	1.0	1.0
	Tertile 2, OR (95% CI)‡	4.4 (1.3, 15.8)	2.8 (0.8, 9.9)
	Tertile 3, OR (95% CI)‡	5.8 (1.5, 22.3)	2.0 (0.5, 7.1)
	Trend over Tertiles§	$p = 0.008$¶	$p = 0.27$
	Linear Trend#	$p = 0.03$¶	$p = 0.37$
Penicillium	Tertile 1, OR‡	1.0	1.0
	Tertile 2, OR (95% CI)‡	0.8 (0.2, 2.8)	1.2 (0.4, 4.4)
	Tertile 3, OR (95% CI)‡	3.8 (1.0, 14.4)	4.9 (1.2, 20.2)
	Trend over Tertiles§	$p = 0.09$	$p = 0.04$¶¶
	Linear Trend#	$p = 0.06$	$p = 0.01$
Total Fungi	Tertile 1, OR‡	1.0	1.0
	Tertile 2, OR (95% CI)‡	1.1 (0.3, 3.7)	0.8 (0.2, 2.8)
	Tertile 3, OR (95% CI)‡	1.1 (0.3, 3.6)	1.3 (0.4, 4.5)
	Trend over Tertiles§	$p = 0.92$	$p = 0.71$
	Linear Trend#	$p = 0.48$	$p = 0.16$

*Number of asthma symptoms (0–1, 2–3, 4–5, 6–7).

†Frequent asthma symptoms (none, frequent day or night time symptoms, frequent day and night time symptoms).

Associations were estimated with proportional odds logistic models adjusted for age, race, gender, and sampling in fall season:

‡Odds ratio and 95% confidence interval is from model with two indicator variables for allergen tertile:

§p value is from model with an ordinal variable for allergen tertile: and

#p value is from model with natural log transformed allergen variable.

Adjusting models for outdoor fungi, outdoor *Penicillium*, average dew point temperature for 30 days before sample, leaks or mold in home, humidifier in home, smoking family members, or child's use of asthma controller medication did not alter the significance of any symptom/exposure relationship.

¶With further adjustment for respiratory infection, $p > 0.05$.

¶¶With further adjustment for respiratory infection, $p = 0.056$.

DISCUSSION

In this study, we found an effect of bedroom cockroach and *Penicillium* allergens on asthma morbidity in low-income Chicago children with asthma. The bedroom may be the most important site of indoor allergen exposure because of the high level of exposure during sleep, large proportion of time spent in the area, and the proximity to the source. Sensitivity to cockroach allergen has been shown to be most closely related to bedroom concentrations of cockroach allergens (24). Similar to some other studies, we found significant associations of asthma symptoms with cockroach allergens limited exclusively to samples obtained from the bedroom (1, 2).

Dampness has been associated with respiratory morbidity, although the biologic organisms responsible for this effect have not been consistently identified (5, 25). Since humidity is the primary factor influencing fungal growth (26), it is possible that humidity in general was responsible for the relationship we found between *Penicillium* and asthma morbidity. However, the association of *Penicillium* with frequent asthma symptoms was not attenuated with control for dampness in the home, a humidifier in the home, or recent humid weather, suggesting that the relationship of *Penicillium* with asthma morbidity was independent of dampness.

The well-reported increase in asthma morbidity and hospitalizations in the fall may be related to increased infections and/or allergen exposure (27). In this study, respiratory

infections and total number of symptoms increased in autumn. Since we did not have repeated measures in this study, we adjusted for measurement in fall season, although this may not be sufficient. Respiratory infection may obscure the relationship of allergens to triggering of symptoms. Exposure to allergens in combination with respiratory infection has been reported to be a risk for severe asthma (28). We found that parent-reported respiratory infection was associated both with increased asthma morbidity and with increased bedroom cockroach allergens, but because of small numbers, we were not able to test for synergistic effects of respiratory infections and cockroach allergens on symptoms. Nevertheless, the results suggest that respiratory infection may confound the association we noted between number of asthma symptoms and cockroach allergens. Conversely, since there may be substantial misclassification bias in parental report of respiratory infection, the analysis of associations of asthma symptoms and allergens, without adjusting for respiratory infections, may be more appropriate.

There have been a number of studies of the relationship of allergens to asthma morbidity (1–10), and many, but not all studies (6, 8), restricted analyses to subjects who were sensitized to specific allergens or stratified analyses by sensitization status. An important limitation of this study is the lack of data on the atopic status of the children, and thus we can not determine if the lack of association of symptoms with some allergens, such as dust mites and cats, is real or due to lack of sensitivity to the allergen. Estimates of cockroach allergen sensitization in children with asthma range from 17 to 41% (29), with sensitivity in up to 69% of inner-city children with moderate to severe asthma (2). A similarly large proportion of the children in this study may be sensitive to cockroach allergens, and associations of symptoms with cockroach allergens are likely to be real. In addition, molds and house dust mites may also trigger asthma symptoms through nonatopic mechanisms (30), thus supporting the inclusion of both atopic and nonatopic subjects in this analysis.

Issues in measurement of allergen exposure also limit our conclusions. In this study and others, homes in high-poverty areas are likely to have higher levels of cockroach and lower levels of dust mite allergens (1, 2), and the low prevalence of house dust mite allergens restricts our ability to draw a conclusion about the role of this allergen in triggering respiratory symptoms. Also, we did not measure exposure to allergens in places other than the home where the children spent a significant amount of time, such as school. The method we used to estimate mold exposure measures only viable fungi and may underestimate non-viable, allergenic mold spores and fungal fragments. While indoor and outdoor air borne molds are generally correlated (26), we did not find this to be true in this study, perhaps because outdoor samples were collected at the University of Illinois at Chicago, not outside of the participant's home. Thus, our findings that outdoor fungi levels were not related to asthma morbidity and did not attenuate the effect of bedroom *Penicillium* on asthma morbidity could be attributed to the collection location rather than a lack of effect.

This study is limited by a number of other factors including lack of data on other important indoor allergens, such as dog and mouse; collection of symptom data over a short time period, precluding analysis of the rarer outcomes of emergency department encounters and hospitalizations; and lack of longitudinal, concurrent measurements of symptoms and allergens over several meteorological seasons. The small sample size also restricts our power to draw conclusions about allergens that did not show associations with asthma morbidity.

This study has several strengths. One is the close temporal relationship of the exposure and symptom measurements, which may have allowed us to find associations that may not be found in study designs where exposure and symptoms are measured over longer time periods and averaged for analysis (1, 6) or those with longer times between exposure and symptom measurement (4, 5). Our risk estimates for symptoms increased with increasing exposure to allergens, suggesting a dose-response relationship. Finally, the consistency of our findings with previous studies and coherence with biological and animal data supports the validity of these findings.

In summary, in low-income Chicago children with asthma, number of asthma symptoms was related to bedroom cockroach allergens, and frequency of asthma symptoms was related to bedroom *Penicillium* allergens. Respiratory infections confounded the association between asthma morbidity and cockroach allergens. These results support other literature suggesting that bedroom levels of mold and cockroach allergens may have important effects on asthma morbidity in children living in inner-city homes.

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