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Indoor exposures to air pollutants and allergens in the homes of asthmatic children in inner-city Baltimore

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Abstract

This paper presents indoor air pollutant concentrations and allergen levels collected from the homes of 100 Baltimore city asthmatic children participating in an asthma intervention trial. Particulate matter (PM), NO_2, and O_3 samples were collected over 72 h in the child's sleeping room. Time-resolved PM was also assessed using a portable direct-reading nephelometer. Dust allergen samples were collected from the child's bedroom, the family room, and the kitchen. The mean PM_{10} concentration, $56.5 \pm 40.7 \,\mu g/m^3$, is 25% higher than the $PM_{2.5}$ concentration ($N = 90$), $45.1 \pm 37.5 \,\mu g/m^3$. PM concentrations measured using a nephelometer are consistent and highly correlated with gravimetric estimates. Smoking households' average $PM_{2.5}$ and PM_{10} concentrations are $33–54 \,\mu g/m^3$ greater than those of nonsmoking houses, with each cigarette smoked adding $1.0 \,\mu m/m^3$ to indoor $PM_{2.5}$ and PM_{10} concentrations. Large percentages of NO_2 and O_3 samples, 25% and 75%, respectively, were below the limit of detection. The mean NO_2 indoor concentration is $31.6 \pm 40.2 \,ppb$, while the mean indoor O_3 concentration in the ozone season was $3.3 \pm 7.7 \,ppb$. The levels of allergens are similar to those found in other inner cities. Results presented in this paper indicate that asthmatic children in Baltimore are exposed to elevated allergens and indoor air pollutants. Understanding this combined insult may help to explain the differential asthma burden between inner-city and non-inner-city children.
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1. Introduction

Asthma is the most common chronic health problem in children and is one of the most common health complaints in the US (Institute of Medicine (IOM), 2000). Unlike many other chronic diseases, asthma rates appear to be increasing particularly in disadvantaged inner-city minority children. From 1975 to 1993–1995, the estimated annual number of office visits for asthma more than doubled, from 4.6 to 10.4 million (Mannino et al., 1998). The number of children dying from asthma has increased by a factor of three, 93–266, from 1979 to 1996 (Centers for Disease Control (CDC), 2003a, b). Disease rates are highest among African-Americans, Hispanics, and populations in urban inner cities. The

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emergency department visit rate for blacks in 2000 was 125% higher than that for whites (CDC, 2003a, b). A recent analysis of asthma hospitalizations in the state of Maryland indicates that Baltimore City has the highest asthma hospitalization rates of any jurisdiction in the state and that African-American children in Baltimore have an annual asthma hospitalization rate (110/10,000) that is three times higher than that of Caucasian children (33.2/10,000) (Mankarious and Quinn, 2001).

Allergens in the home are an established risk factor for asthma. After exposure to an allergen, the immune system can become sensitized and produces antibodies to allergen-specific proteins, creating an inflammatory response that leads to airway hyperreactivity. Children with atopy, a genetic predisposition to allergen sensitivity, are at higher risk of developing asthma. In a sensitized individual, small amounts of allergen can result in a large inflammatory response. It has been estimated that approximately 80% of asthma in children is allergic asthma (IOM, 2000). Asthma rates are higher in children who are sensitized to allergens (Lau et al., 2000; Kattan et al., 1997; Nelson et al., 1999; Eggleston, 2000).

The association between air pollution and asthma has also been well established. Outdoor air pollution has been associated with emergency room visits for asthma in numerous cities around the world (Schwartz et al., 1993; Chew et al., 2000; Garty et al., 1998; Lipsett et al., 1997). Ambient air pollution has also been associated with increased symptoms and decreased lung function among asthmatics (Yu et al., 2000; Peters et al., 1997). The role of indoor air pollutants and asthma has not been as extensively investigated. When studied, indoor air pollutants associated with damp homes (mold), environmental tobacco smoke, and combustion products have been linked to increased asthma symptoms (Jaakkola et al., 2002; Thorn et al., 2001; Zock et al., 2002). Indoor pollutant concentrations in inner-city homes, particularly for particulate matter (PM), have not been well characterized.

To evaluate the effect of a comprehensive strategy to reduce indoor air pollution and allergen levels on the health of children with asthma, The Johns Hopkins Center for Childhood Asthma in the Urban Environment is conducting a randomized clinical trial. The purpose of this paper is to summarize indoor air pollutant (ozone, nitrogen dioxide, PM) concentrations and reservoir dust allergens levels in the homes of 100 participating Baltimore inner-city asthmatic children as part of a baseline assessment for this trial.

2. Methods

2.1. Study design and recruitment

Descriptions of the intervention study design including rationale and recruitment have been previously published (Eggleston et al., 1999; Swartz et al., 2004). Briefly, participants were recruited from a school-based asthma education program (A+ Asthma Program) conducted at six elementary schools in east Baltimore. To be eligible for the study, families met the following eligibility criteria: (1) child was aged 6–12 years, (2) child had doctor-diagnosed asthma, current asthma symptoms, or medication use at least once in the previous 3 months, (3) child had no other chronic illnesses, (4) the home had electricity and was within a defined geographic area of east Baltimore, MD, and (5) the child completed the school-based asthma education program or its equivalent.

A total of 387 children completed the A+ program. The families of 292 potentially eligible children were given information about the study and 180 were referred by the A+ Asthma Program to the study recruiter. The most common reason for not referring a family to the recruiter was inability to contact the family. One hundred children were recruited from this pool and enrolled in the study.

The study was approved by the Institutional Review Board of the Johns Hopkins Medical Institutions. Before randomization into the trial, each volunteer signed an informed consent, completed a questionnaire, and had a home environmental evaluation to inspect the home and measure allergens, NO_2, O_3, and airborne PM.

2.2. Air sampling procedures

Air pollution sampling was conducted over a 72-h period in the sleeping room of the asthmatic child. PM_{10} (PM with an aerodynamic size less than 10 μm) and $PM_{2.5}$ (PM with an aerodynamic size less than 2.5 μm) samples were collected using 4 L/min MSP impactors (St. Paul, MN) loaded with 37-mm, 2.0-μm pore size, PALL Teflo PTFE membrane filters with polypropylene support rings (Pall Corp. Ann Arbor, MI). Inlet flows were checked at the beginning and end of each sampling period using primary standards (BIOS DryCal; Bios International Corp., NJ). Samples were collected using battery-operated pumps plugged into house electrical service to assure 72-h of operation. Ozone and nitrogen dioxide were sampled passively using Ogawa badges. All sampling heads and passive badges were attached to the outside of a sampling frame that was placed in a convenient place in the child's bedroom. In most cases the sampling frame was placed on the dresser or a nightstand. In some cases, the sample frame was placed on portable stand constructed out of PVC pipe.

PM gravimetric analysis was conducted according to 40CFR50 appendix L using a Metler T5 microbalance. Before analysis, filters were placed in petri dishes and stored for 24 h in a weighing room equipped with temperature and humidity control.

Time-resolved PM was also assessed using a portable direct-reading nephelometer with data-logging capability (MIE pDR1000s; ThermoElectron, Franklin, MA). The instrument incorporates a pulsed, high-output, near-infrared light-emitting diode source (880 nm). The intensity of the light scattered over the forward angle inside the inlet by the particles passing through the sensing chamber is linearly proportional to the airborne PM concentration. The instrument's optical configuration produces response to particles in the size range of 0.1–10 μm, although empirical evidence suggests that there is a differential response such that particles in the size range of 0.3–2 μm are more efficiently detected relative to the size fraction from 2 to 10 μm (Liu et al., 2002; Howard-Reed et al., 2000; Quintana et al., 2000). The instrument was operated in the passive sampling mode and has a measurement range of 1.0–400,000 μg/m^3.

Ozone was monitored using a validated passive technique which was shown to be in close agreement ($r = 0.95$, $P < 0.01$) with EPA's ambient equivalent method based on UV photometry (Koutrakis et al., 1993). The sampler is small (2×3 cm) and light weight (7 g). The method is based on ozone's reaction with a nitrite-coated filter to yield nitrate which is then quantified by ion chromatography. Samplers and coated filters were purchased from Ogawa, Inc. (Pompano Beach, FL). Air is effectively sampled at a rate of 22.8 cm^3/min. The limit of detection (LOD) was calculated based on the analysis of field blanks. The median LOD was 3.1 ppm for a 72-h sample with a batch-specific LOD ranging from 0.9 to 8.6 ppm.

Nitrogen dioxide was measured using the same passive monitors used for ozone monitoring (Palmes tubes) but loaded with filters coated with triethanolamine (TEA) (Palmes et al., 1976). In the presence of a color reagent, nitrogen dioxide and TEA form a highly colored azo dye that is measured spectrophotometrically at 540 nm. The median LOD calculated from the analysis of field blanks was 6.8 ppb for a 72-h sample. The batch-specific LOD ranged from 1.4 to 77 ppb.

2.3. Allergen collection and analysis

Household dust samples were collected on an unwoven fabric collector inserted into the nozzle of a standard portable vacuum. Samples were collected from three sites (the child's bedroom, the family or television room, and the kitchen) using standard methods (Platts Mills et al., 1992). The bedroom sample was collected by vacuuming a 1-m^2 area near and underneath the bed for 2 min combined with a 2-min sample from the mattress and bedding. In the television/living room samples were collected for 2 min each from any upholstered furniture and from a 1-m^2 area next to the furniture. In the kitchen, the entire floor was vacuumed for 2 min with

particular attention to the base of the counters and the interior of under-sink cabinets. After each room sampling, the fabric collector was removed from the vacuum and sealed in a plastic bag.

An aqueous extract of 100 mg of the sieved dust (sieve size 300 μm) specimen was prepared in 2 mL of borate-buffered saline. The extracts were stored at −30 °C until they were assayed for Der p 1, Der f 1, Fel d 1 Bla g 1, and Mus m 1 using mAb-based enzyme-linked immunosorbent assays (Indoor Biotechnologies, Charlottesville, VA) (Chapman et al., 1987, 1988; Pollart et al., 1991; Ohman et al., 1994). Results were expressed as mass of allergen per gram of settled dust when appropriated standards were available or as units per gram in the case of Bla g 1. Detection limits were 100 ng/g for the group 1 mite allergen, 50 ng/g for Fel d 1, Can f 1, and Mus m, and 1 U/g for Bla g 1.

2.4. Household inspection and activity assessment

Household activity information was collected from study participants for each day of the sampling period. The purpose of collecting household activity information was to evaluate potential exposure determinants. Information collected included smoking activity, cleaning activities such as vacuuming and sweeping, combustion-related activities, e.g., burning food or incense, and gas cooking activities.

Prior to sampling, each home was inspected by a trained environmental health technician to document the type, salient features (e.g., HVAC system), and condition of the house. In addition to general housing conditions, the home inspection focused on the living/family room, the kitchen, and the child's bedroom.

3. Results

The study population is a group of school-aged inner-city children with asthma living in east Baltimore, MD. The mean age of the 100 children enrolled in the study is 8.4 years and the proportions of female and African-Americans are 54% and 99%, respectively. Eighty-nine percent of the children are cared for by a single mother or another single female (grandmother, guardian, or aunt) and 74% of the households had reported incomes of < $20,000.

Home characteristics are summarized in Table 1. The predominant housing type was a row house. All houses cooked with gas stoves. Most houses had some evidence of general deterioration with a quarter of the houses having leaking roofs. Broken plaster (69%), peeling paint (53%), and cracks or holes in the walls or doors (66%) were highly prevalent. Evidence of smoking was common in the study subject homes (46%) and the inspection of kitchen and bedroom revealed evidence of

poor housekeeping. Evidence of cockroach rodent infestation was also commonly observed.

Table 2 summarizes the 72-h time-weighted average (TWA) indoor air pollutant concentrations. The number of homes sampled does not equal 100 because a few samples were lost due to equipment failure and tampering. The mean PM_{10} concentration ($N = 91$), $56.5 \pm 40.7 \, \mu g/m^3$, is 25% higher than the $PM_{2.5}$ concentration ($N = 90$), $45.1 \pm 37.5 \, \mu g/m^3$, suggesting that roughly 75% of the indoor PM mass is less than $2.5 \, \mu m$ in aerodynamic diameter. The standard deviations of the two means are large, indicating a considerable amount of variability in measured 72-h TWAs. Maximum measured PM_{10} and $PM_{2.5}$ concentrations are 275.7 and $191.7 \, \mu g/m^3$, respectively. The percentage of samples with $PM_{2.5}$ concentrations above EPA's proposed 24-h average National Ambient Air Quality Standard (NAAQS) of $65 \, \mu g/m^3$ is 17%, while 2% of the PM_{10} samples are above the $150\text{-}\mu g/m^3$ 24-h NAAQS for PM_{10}. PM_{10} and $PM_{2.5}$ concentrations within children's bedrooms are highly correlated (Fig. 1) with a Pearson's correlation coefficient of 0.91. MIE TWA PM concentrations are consistent and highly correlated with gravimetric estimates. Correlation coefficients for PM_{10} and $PM_{2.5}$ with MIE-based gravimetric estimates are 0.72 and 0.81, respectively.

MIE PM data provide an additional opportunity for evaluating exposure trends. Fig. 2 depicts the PM concentration profile represented by median values from approximately 300 person-days of monitoring from Baseline homes of the Intervention study. These results illustrate the cyclic nature of PM levels within a home and suggest that increased nighttime symptoms may be associated with increased PM exposures late in the day. Median PM concentrations in the evening hours were roughly $10 \, \mu g/m^3$ higher than midday concentrations. The temporal increase in evening PM concentration is even more evident if the 75% of the distribution is considered.

Based on household activity information, we were able to estimate the numbers of cigarettes smoked in the home during each 72-h sampling period. Average $PM_{2.5}$, PM_{10}, and MIE concentrations in nonsmoking households are 25.8 ± 14.9, 37.7 ± 18.8, and $35.1 \pm 24.9 \, \mu g/m^3$, respectively. By comparison, smoking households' average $PM_{2.5}$, PM_{10}, and MIE concentrations are 59.1 ± 42.5, 71.2 ± 46.7, and $89.5 \pm 76.0 \, \mu g/m^3$, respectively. This represents a 33–$54\text{-}\mu g/m^3$ increase in PM concentrations in smoking houses. Regression analysis of number of cigarettes smoked versus the natural logarithm of the concentration indicates that on

Table 1
Summary of home characteristics based on the home inspection

Home characteristic	% of homes
Type of home	
Row house/townhouse	91
Apartment	5
Detached	4
General condition of dwelling	
Leaks in roof	24
Broken plaster	69
Peeling paint	53
Peeling wallpaper	14
Cracks or holes on wall and doors	66
Pets in household	
Cat	26
Dog	20
Current smoking in the home	46
Kitchen	
Dishes in sink	69
Moisture or leaks	22
Food on countertops	70
Living cockroaches	31
Mouse droppings	38
Child's bedroom	
Leaks in bedroom	18
Food or food remains	29
Mess on the floor	61
Living cockroaches	8
Mouse droppings	7
Floor covering type	
Wall to wall carpet	43
Linoleum/tile	29
Hardwood	25

Table 2
Summary of 72-h time-weighted average indoor area air pollutant levels collected in bedrooms of asthmatic children in Baltimore, MD

Pollutant	N	% detect	Mean ± SD[a]	Median	Min	25th %	75th %	Max
$PM_{2.5}$	90	100	$45.1 \pm 37.5 \, \mu g/m^3$	$35.1 \, \mu g/m^3$	$4.4 \, \mu g/m^3$	$20.8 \, \mu g/m^3$	$57.5 \, \mu g/m^3$	$191.7 \, \mu g/m^3$
PM_{10}	91	100	$56.5 \pm 40.7 \, \mu g/m^3$	$45.2 \, \mu g/m^3$	$11.7 \, \mu g/m^3$	$29.9 \, \mu g/m^3$	$70.3 \, \mu g/m^3$	$275.7 \, \mu g/m^3$
MIE[b]	97	100	$62.8 \pm 61.0 \, \mu g/m^3$	$44.1 \, \mu g/m^3$	$1.5 \, \mu g/m^3$	$21.7 \, \mu g/m^3$	$76.7 \, \mu g/m^3$	$310.8 \, \mu g/m^3$
NO_2	95	75	$31.6 \pm 40.2 \, ppb$	$19.1 \, ppb$	$4.1 \, ppb$	$9.7 \, ppb$	$36.5 \, ppb$	$259.7 \, ppb$
O_3	88	28	—	$1.6 \, ppb$	$0.9 \, ppb$	$1.1 \, ppb$	$2.5 \, ppb$	$20.7 \, ppb$
O_3 In season[c]	56	41	—	$1.7 \, ppb$	$0.8 \, ppb$	$1.0 \, ppb$	$3.9 \, ppb$	$55.2 \, ppb$

[a]SD, standard deviation.
[b]MIE, pDR1000 nephelometer.
[c]In season, May–September.

average each cigarette smoked adds $1.0 \, \mu g/m^3$ to indoor $PM_{2.5}$ and PM_{10} concentrations (Fig. 3).

The frequencies of candle and incense burning, cooking events, and other potential dust-producing events were also compiled as a part of the household activity survey. When these values were added to a regression model either individually or in combination, they were not significantly predictive of indoor PM concentration (result not shown). In addition, the street in front of each house was classified based on the

amount of traffic passing in front of the house as being major arterial, minor arterial, or side street. The street classification in front of the house was not predictive of indoor PM concentration.

Twenty-five percent of NO_2 samples were below the LOD. Mean NO_2 concentration ($N = 95$) is 31.6 ppb. The median NO_2 indoor concentration is 19.1 ppb with a maximum concentration of 259.7 ppb. The predominant sources of NO_2 in the homes are cooking and smoking since all study houses used gas stoves and many homes had smokers.

A large percentage (72%) of the 88 indoor O_3 samples collected were below the LOD. This is due to the fact that many of the samples were collected during the winter months when ambient ozone levels are low. The overall median ozone concentration ($N = 88$) is 1.6 ppb. Given the large number of samples below the LOD, the overall mean O_3 concentration is not presented. Fifty-six samples were collected during Baltimore's ozone season (May–September). Of these samples, 41% were above the LOD, with a median of 1.7 ppb.

The seasonal variation in indoor pollutant concentrations is presented in Fig. 4. Ozone is the only pollutant with significant seasonal variability. Since there is no indoor source of ozone, the increase in the summer is due to increased ambient ozone during warmer months. The highest indoor ozone concentrations were measured during the months of June, July, and August with median ozone concentrations equal to 2.5, 4.1, and 2.3 ppb, respectively.
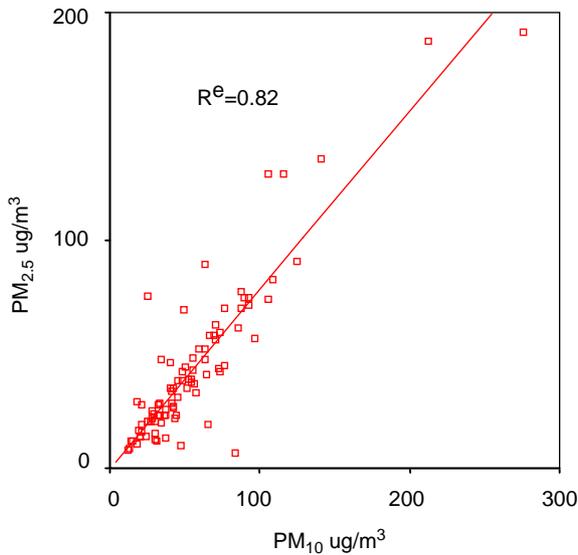

Fig. 1. Correlation of PM_{10} and $PM_{2.5}$ airborne concentrations in the bedrooms of asthmatic children in Baltimore, MD.
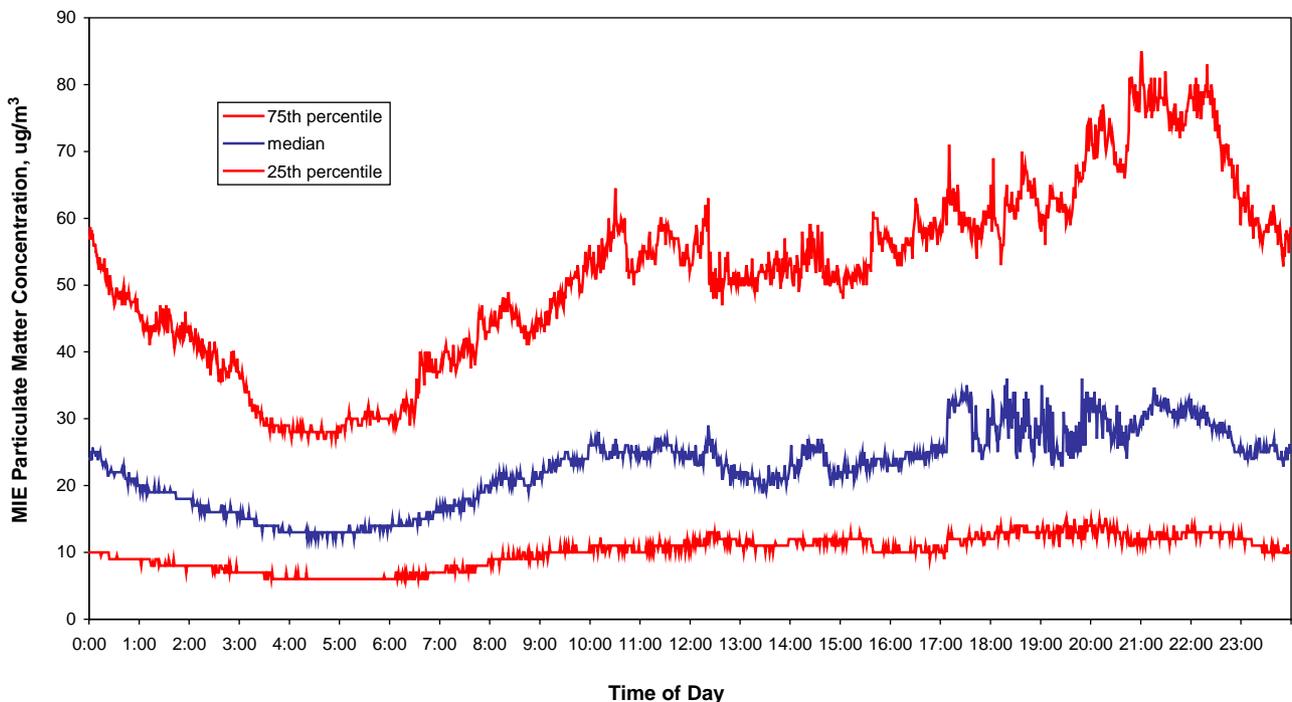

Fig. 2. MIE PM concentration profile represented by median values from approximately 300 person-days of monitoring.
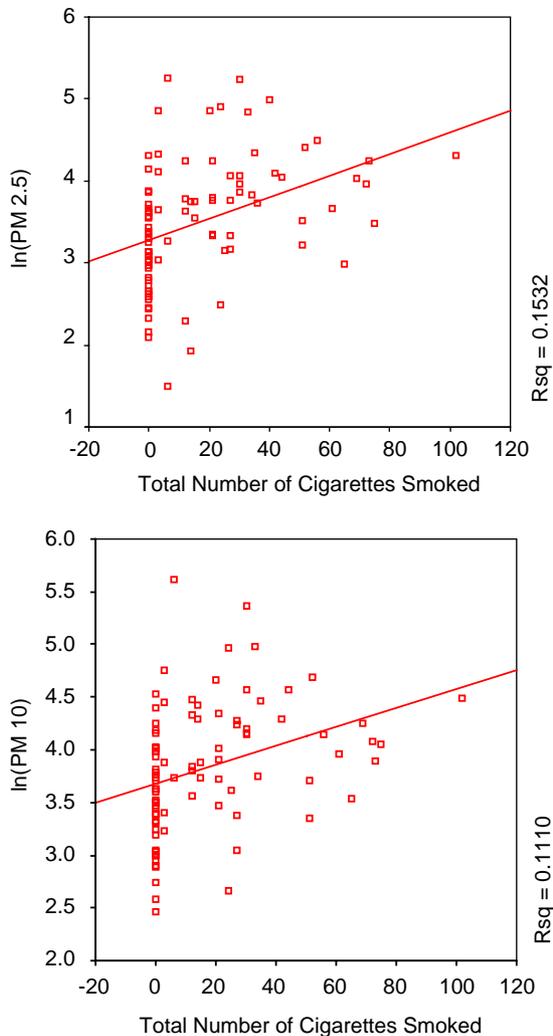
Fig. 3. Regression analysis of the natural logarithms of $PM_{2.5}$ and PM_{10} concentrations versus the number of cigarettes smoked.

The distribution of allergens in settled dust is summarized in Table 3. The most commonly detected allergen was mouse, found in all the bedroom samples and in 99% of kitchen samples. Cockroach, dog, and cat allergens were also common, while house dust mite was less common. The predominant house dust mite allergen, Der f 1, was detected in 59% of bedroom samples, but the median concentration was only 66 ng/g. Mouse allergen was also found in large quantities, with a median concentration in bedroom dust (3659 ng/g) that was an order of magnitude higher than cat or dog allergen. Median concentrations of cockroach allergen were highest in the kitchen (22 U/g), followed by the living room (median 5.2 U/g) and bedroom (4.5 U/g).

4. Discussion

A direct comparison of indoor PM concentrations with other studies is difficult due to differences in collection methods and strategies. In general, however, the indoor PM concentrations presented in this paper are consistent with results of other studies but tend to be on the higher side of reported ranges, particularly with regard to $PM_{2.5}$ (Wallace, 1996). Indoor 72-h $PM_{2.5}$ concentrations in this study ranged from 4.4 to 191.7 μg/m³ with an average of 45.1 μg/m³. These results are higher than indoor $PM_{2.5}$ concentrations measured in inner-city homes in Detroit, MI, which found an average 34.4 ± 21.7 μg/m³ (Keeler et al., 2002). Indoor $PM_{2.5}$ concentrations in Baltimore City are also on the upper end of the distribution of home measurements collected in upstate New York. Results reported by Sheldon et al. (1989) indicate mean indoor $PM_{2.5}$ concentrations between 36 and 46 μg/m³. Indoor PM_{10} concentrations in the Detroit study, which found an average indoor PM_{10} concentration of 52.2 ± 30.6 μg/m³, are in closer agreement with the results presented in this study $(56.5 \pm 40.7$ μg/m³).

As has been reported elsewhere, smoking is a major determinant of indoor PM concentration (Ozkaynak et al., 1996; Wallace, 1996). Previous studies have consistently reported increased PM concentrations in homes with smokers, indicating that the smoking contribution to indoor $PM_{2.5}$ ranges from 25 to 45 μg/m³, with the contribution from a single cigarette ranging from approximately 1 to 2 μg/m³. Results presented in this paper are consistent with these observations.

Nitrogen dioxide is a by-product of high-temperature combustion inside the home. Sources include unvented gas appliances (stoves, hot water heaters, and furnaces) and portable kerosene heaters. Previous investigations have documented a roughly two–three fold increase in indoor NO_2 concentrations in homes with combustion sources (Samet et al., 1992; Neas et al., 1991). The NO_2 concentrations reported in this paper are similar to those reported by Samet et al. (1992) for homes with gas cooking in New Mexico and on the high end of exposures reported by Neas et al. (1991) for homes with gas stoves sampled as a part of the six-cities study. Mean household NO_2 concentrations measured in homes with gas stoves and/or kerosene heaters in the six-cites study ranged from 11.0 to 31.3 ppb.

Since there are no indoor sources of ozone, there are few published studies on indoor O_3 exposures. Ozone is a pollutant of ambient origin. Indoor ozone concentrations are typically significantly attenuated from outside concentrations. The results published in this investigation suggest that for inner-city asthmatic children indoor ozone concentrations are low compared to outdoor levels and are well below the EPA's 80-ppb 8-h criteria level.

The levels of allergens and their distribution are similar to those found in samples from other inner cities such as the seven cities in the NCICAS study (Kattan et al., 1997; Eggleston et al., 1998; Phipatanakul et al., 2000). The levels of cockroach allergen and mouse allergen that we
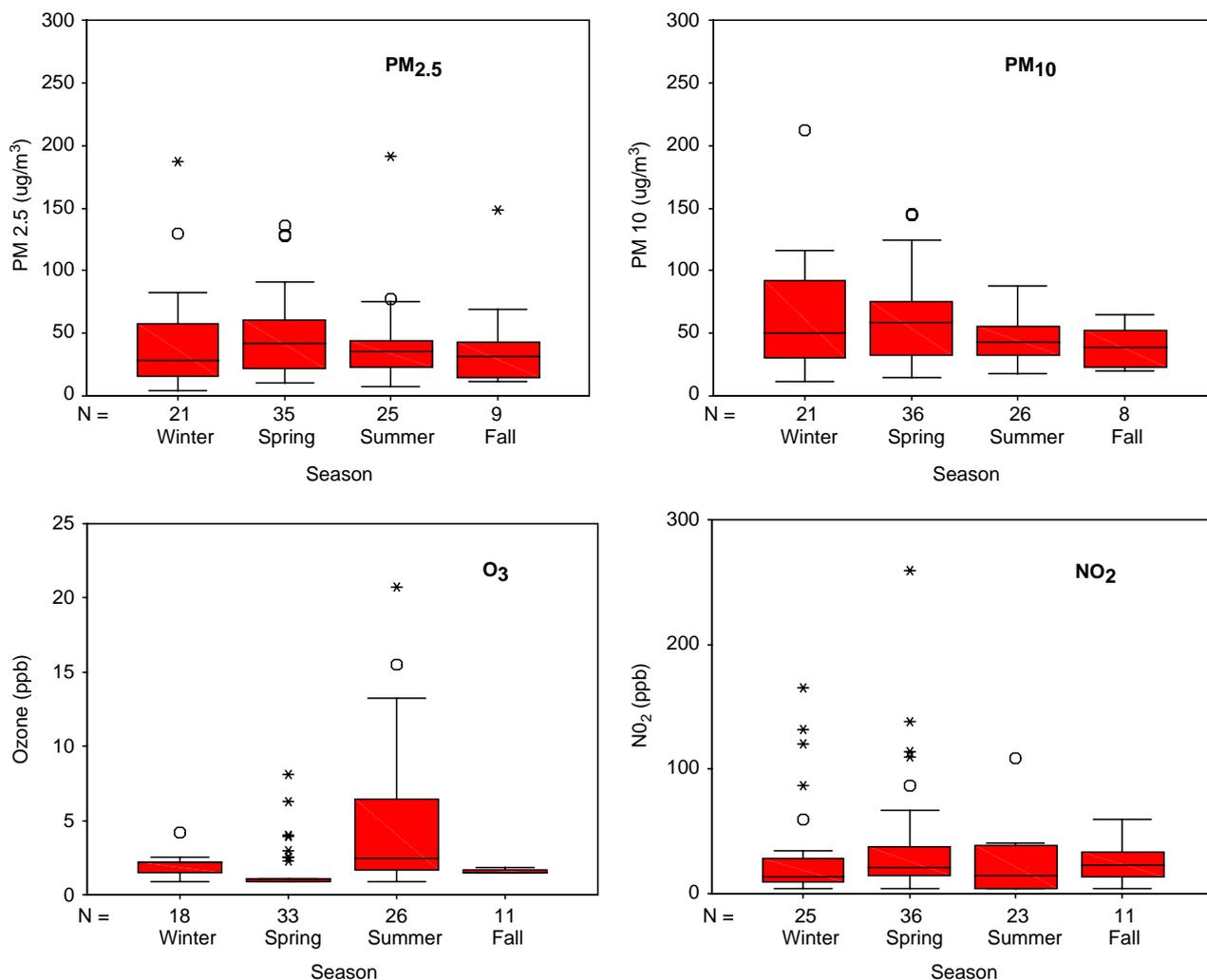
Fig. 4. Boxplots of 72-h indoor air concentrations by season.

found in the bedroom have been associated with sensitization to these allergens in asthmatic children (Eggleston et al., 1998; Phipatanakul et al., 2000) and in the case of cockroach allergen the median values are near 8 U/g, the level associated with increased morbidity in sensitized children (Rosenstreich et al., 1997).

Not surprisingly, none of the allergen loadings were correlated with air pollutant concentrations. The only significant correlations among allergen or pollutant concentrations were for the three estimates of indoor PM exposure.

5. Conclusion

The combined evaluation of residential allergen and air pollution exposure among asthmatic children living in the urban environment is important in understanding its contribution to the health risk to a susceptible population. This is particularly true for inner-city disadvantaged children where air pollution and allergen exposures tend to be the highest. Although indoor allergen levels for asthmatic children are generally well characterized, there are few studies reporting indoor air pollution exposures for asthmatic children in inner-city environments and there are few studies that have reported on the combined exposures to allergens and indoor air pollutants in asthmatic children. Airborne PM, ozone, nitrogen dioxide, and allergens have all been identified as risk factors for asthma morbidity. Results presented in this paper suggest that children in inner-city Baltimore are exposed to elevated allergens and indoor air pollutants, particularly $PM_{2.5}$. Although the epidemiology and basic science literature indicates that both air pollutants and allergens are independently associated with increased asthma risk, the effects of the combined insult have not been elucidated. This combined insult may help to explain the differential asthma burden between inner-city and non-inner-city children. Recent studies suggest that PM exposure can promote

Table 3
Summary of allergens measured in reservoir dust in homes of asthmatic children in Baltimore, MD

Allergen	Room	N	% detect	Mean (SD)[a,b]	Median	Min	25th %	75th %	Max
Bla g1	BR	96	78	19.3±37.5	4.5	BD	1.0	18.5	245
	LR	96	82	34.5±80.1	5.2	BD	1.3	26.0	565
	KT	95	85	191.3±579.1	22.0	BD	4.0	99.0	4787
Der p	BR	96	7	—	BD	BD	BD	BD	6314
	LR	96	9	—	BD	BD	BD	BD	2143
	KT	97	2	—	BD	BD	BD	BD	582
Der f	BR	96	59	416.5±978.1	66.0	BD	BD	270.2	5356
	LR	96	50	583.5±256.0	12.0	BD	BD	250.5	17,632
	KT	97	8	—	BD	BD	BD	BD	6380
Feld	BR	96	96	9279±29,583	498.0	BD	164.0	3331.2	209,780
	LR	96	97	25,058±77,613	525.0	BD	214.0	8748.2	553,939
	KT	97	77	4658±14,841	104.0	BD	24.5	660.0	119,508
Can f	BR	96	83	15,698±60821	288.0	BD	41.2	1097.2	455,487
	LR	95	89	6214±25112	327.0	BD	123.0	1199.0	194,371
	KT	97	53	9303±59,125	37.0	BD	BD	234.0	568,369
Mup	BR	96	100	9628±17298	3659	36	939.5	10,374	116,050
	LR	95	100	12396±20610	4000	40	1257.0	13,352	119,478
	KT	97	99	108839±716195	14674	BD	1479.5	37,094	7,058,040

[a] SD, standard deviation.
[b] All units in ng/g except Bla g1 which is in U/g.

sensitization to aeroallergens (Diaz-Sanchez et al., 1996). Outdoor PM collected in urban Baltimore has been shown to enhance specific IgE antibody responses to house dust mite exposure in a genetically susceptible murine strain (Walters et al., 2001). In addition, recent evidence suggests that PM may induce the allergic phenotype by the induction of airway hyperresponsiveness which occurs via induction of a component of the innate immune system, the complement system (Walters et al., 2002).

Much of what is known about the impact of PM on asthma is based on studying outdoor PM. By focusing on indoor PM and on allergen exposures, the data presented in this paper will form the basis for testing the hypothesis that the combination of indoor air pollutants and allergen exposures is an important contributor to the increased asthma burden in the inner-city.

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