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Indoor Environmental Differences between Inner City and Suburban Homes of Children with Asthma

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ABSTRACT *We conducted this study to compare environmental exposures in suburban homes of children with asthma to exposures in inner city homes of children with asthma, to better understand important differences of indoor pollutant exposure that might contribute to increased asthma morbidity in the inner city. Indoor PM_{10}, $PM_{2.5}$, NO_2, O_3, and airborne and dust allergen levels were measured in the homes of 120 children with asthma, 100 living in inner city Baltimore and 20 living in the surrounding counties. Home conditions and health outcome measures were also compared. The inner city and suburban homes differed in ways that might affect airborne environmental exposures. The inner city homes had more cigarette smoking (67% vs. 5%, p<.001), signs of disrepair (77% vs. 5%, p<.001), and cockroach (64% vs. 0%, p<.001) and mouse (80% vs. 5%, p<.001) infestation. The inner city homes had higher geometric mean (GM) levels (p<.001) of PM_{10} (47 vs. 18 μg/m^3), $PM_{2.5}$ (34 vs. 8.7 μg/m^3), NO_2 [19 ppb vs. below detection (BD)], and O_3 (1.9 vs. .015 ppb) than suburban homes. The inner city homes had lower GM bedroom dust allergen levels of dust mite (.29 vs. 1.2 μg/g, p=.022), dog (.38 vs. 5.5 μg/g, p<.001) and cat (.75 vs. 2.4 μg/g, p=.039), but higher levels of mouse (3.2 vs. .013 μg/g, p<.001) and cockroach (4.5 vs. .42 U/g, p<.001). The inner city homes also had higher GM airborne mouse allergen levels (.055 vs. .016 ng/m^3, p=.002). Compared with the homes of suburban children with asthma, the homes of inner city Baltimore children with asthma had higher levels of airborne pollutants and home characteristics that predispose to greater asthma morbidity.*

KEYWORDS *Indoor air, Inner city asthma, Particulate matter, Air pollution, Allergens*

Abbreviations: PM_{10} – Particulate matter with aerodynamic diameter less than 10 μm; $PM_{2.5}$ – Particulate matter with aerodynamic diameter less than 2.5 μm; PM – Particulate matter; Ppb – Parts per billion; BD – Below detection; GM – Geometric mean; GSD – Geometric standard deviation; AM – Arithmetic mean; ASD – Arithmetic standard deviation; OR – Odds ratio; SEM – Standard error of the mean

INTRODUCTION

Children living in urban environments, especially in inner cities, have a higher prevalence of asthma[1] and greater asthma morbidity than the general population.[2] The reasons for this disparity are complex and include unhealthy home environ-

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ments.[3–5] Home environmental surveys in the inner city have documented that homes are frequently old and in poor repair, with leaky roofs, water damage, and mold contamination. In addition, cigarette smoking rates are high, cockroach and rodent infestation is common,[3,5–8] and indoor NO_2 and particulate levels have been reported to be high.[3,9]

All of these factors, especially indoor dampness and mold, airborne particulates, and indoor allergens, have been shown to be associated with increased morbidity among children with asthma.[10–12] Although the quality of inner city housing and indoor environment are often cited as partially responsible for the observed higher asthma morbidity, there is little quantitative data establishing the differences between relative exposures in inner city and suburban homes. Surveys from the US Census suggest that 10–14% of homes have had water leakage in the last 12 months[13] and only limited data are available regarding pollutants in middle class homes. The home indoor environment is crucial to children's exposure to allergens and pollutants because concentrations are often amplified indoors and children spend the bulk (i.e. >60%) of their time at home.[14]

For these reasons, we conducted the present study to provide a direct, quantitative comparison of environmental exposures in suburban homes of children with asthma to exposures in inner city homes of children with asthma, to better understand important differences of indoor pollutant exposure that might contribute to asthma morbidity.

METHODS

We compared the home environments of 100 inner city children with asthma, who had participated in a clinical trial that included a detailed environmental evaluation, to the home environments of 20 children with asthma who lived in counties surrounding Baltimore.

Study Population The inner city household data were collected from participants in a clinical trial of environmental intervention.[8,15] In that trial, children and their families were recruited from six inner city Baltimore elementary schools with an estimated total population of approximately 3,000 students. Recruitment began in the schools with school-based asthma self-management classes; 387 children participated. At the conclusion of the program, 317 families agreed to speak with a recruiter, 180 completed a baseline eligibility questionnaire, 125 consented, and 100 were randomized into the study; the data from these 100 families are the basis for the present report. Eligibility criteria included age 6–12 years, reported doctor-diagnosed asthma, current asthma symptoms, and no other chronic lung diseases. Baseline data on asthma symptoms and management, home characteristics, air pollutants, and allergens were collected before the interventions.[8,15]

The suburban comparison group was recruited from families who had completed another trial of environmental modification for asthma management, the Home Environmental Adherence Trial (Home Study). Home study participants were recruited from the records of four pediatric practices in the Baltimore area. Of the 3,796 children who had diagnostic codes consistent with asthma symptoms in their chart, invitation letters were mailed to 3,185 children who were between ages 6–17 years. After telephone screening, 382 children completed a recruitment visit, including skin prick testing to common indoor and outdoor allergens, and 241

families agreed to be in the study and were randomized. At the time of recruitment for the current home environmental evaluation and air monitoring, 176 families had completed the Home Study environmental modifications and 103 had given written permission to be contacted about a future air monitoring study. Sixty-seven of the children were eligible to participate, because they lived outside of the Baltimore city limits and were still less than age 18 years old at the time of the air monitoring study. Subjects were randomly selected from this pool of 67 families. The study and consent procedures were approved by the Johns Hopkins Institutional Review Board.

Study Procedures After obtaining a written informed consent, a questionnaire regarding demographics, asthma symptoms, medications and provider use was administered by a trained research technician. The child performed spirometry before and after inhalation of albuterol by metered-dose inhaler. A home inspection was performed to document home location, general state of repair, sources and signs of moisture, pets, smoking, gas stove use, and carpeting. "Arterial" streets included urban or suburban streets with high, continuous traffic flow during the daytime. Environmental assessments were conducted using identical protocols and cross-trained environmental technicians to ensure the consistency of data collection between the two populations.

Over a 72-hour period, indoor air was collected from the child's bedroom for pollutant analysis. Particulate matter with an aerodynamic size less than 10 μm (PM_{10}) and particulate matter with an aerodynamic size less than 2.5 μm ($PM_{2.5}$) samples were collected using 4 L/min MSP™ impactors (St. Paul, MN) loaded with 37-mm, 2.0-μm pore, PALL Teflo™ PTFE membrane filters with polypropylene support rings (Pall Corporation, Ann Arbor, MI). Inlet flow rates were checked at the beginning and end of each sampling period using primary standards (BIOS DryCal™, Bios International Corporation, Butler, NJ). PM gravimetric analysis was conducted on a Mettler T5 microbalance, after filters were preequilibrated for 24 hours at a constant temperature and humidity.

Nitrogen dioxide (NO_2) and ozone (O_3) were sampled passively using Ogawa badges (Pompano Beach, Florida). NO_2 was measured using screens coated with triethanolamine (TEA).[16] In the presence of a color reagent, NO_2 and TEA form a highly colored azo dye that was measured spectrophotometrically at 540 nm. O_3 was measured using screens coated with nitrite; O_3 and nitrite react to yield nitrate, which was quantified by ion chromatography. The detection limits were calculated based on the analysis of field blanks. The median detection limit for O_3 was 3.1 parts per million for a 72-h sample and the median detection limit for NO_2 was 6.8 parts per billion (ppb).[15]

Settled dust samples were collected by vacuum (DataVac, Metropolitan Vacuum Cleaner Corp, Suffern, NY) from the child's bed and bedroom floor using an unwoven fabric sleeve inserted into the nozzle.[4] The mattress, bedding, and a 1-m^2 area near and underneath the bed were vacuumed for 2 minutes. An aqueous extract of sieved dust (sieve size 300 μm) was prepared in PBS-Tween (1:20 weight/volume). Der f 1, Der p 1, Bla g 1, Mus m 1, Fel d 1, and Can f 1 were quantified using monoclonal antibody-based sandwich ELISA assays with a peroxidase detection system.[17,18,19] Results were expressed per gram of sieved dust and detection limits were 36 ng/g for Der f 1, 110 ng/g for Der p 1, .4 U/g for Bla g 1, 4.4 ng/g for Mus m 1, 5.6 ng/g for Fel d 1, and 20 ng/g for Can f 1.

Mus m 1 was also collected in air samples using 2 L/min IOM dust samplers (SKC Inc., Eighty-four, PA) loaded with 25-mm, .3-μm pore Teflon filters (Pall

TABLE 1 Housing characteristics on inspection

Housing characteristics	Suburbs % (*N*=20)	City % (*N*=100)	*p*
Detached/duplex house	80	4	<.001
Home on arterial street	0	45	<.001
Signs of disrepair	5	77	<.001
Leaky roof	0	24	.12
Broken windows	0	4	1.0
Broken plaster	0	69	<.001
Peeling paint	0	53	<.001
Peeling wallpaper	5	14	.46
Cracks/holes in plaster	0	66	<.001
Smoker	5	67	<.001
Allergen sources			
Dog	50	20	.009
Cat	15	26	.40
Dog or cat	55	39	.22
Cockroach infestation	0	64	<.001
Mouse infestation	5	80	<.001
Wall to wall carpet			
Kitchen	5	2	.42
Living room	60	53	.63
Bedroom	75	43	.015
Large rug			
Kitchen	0	3	1.0
Living room	35	19	.14
Bedroom	25	5	.013
Scatter rug			
Kitchen	55	23	.006
Living room	45	14	.03
Bedroom	25	6	.021
Bedroom any carpet	95	48	<.001
Wall to wall any room	80	57	.08
Dust mite covers			
Pillow	55	5	<.001
Mattress	55	1	.083
Gas stove	40	84	<.001
Unvented dryer	0	22	.023
Signs of moisture			
Damp or leaks, any room	5	31	.013
Kitchen	0	22	.031
Kitchen plumbing	0	8	.35
Family room	5	8	.69
Bedroom	0	18	.49
Musty smell, any room	25	61	.006
Family room	10	32	.57
Bedroom	5	34	.007
Mildew, any room	20	32	.30
Kitchen ceiling	0	4	1.0
Kitchen walls	5	3	.52
Kitchen windows	5	20	.19
Bedroom ceiling	10	3	.20
Bedroom walls	0	2	1.0
Bedroom windows	20	16	.075

Corporation, Ann Arbor, MI). As described above, inlet flow rates were calibrated against a primary flow device (BIOS DryCal™, Bios International Corporation, Butler, NJ) and checked after sampling. Mus m 1 was extracted in 1.5-mL phosphate-buffered saline with .1% Tween, analyzed as described below, and expressed as ng/m^3. The detection limit was .005 ng/m^3 over the 3-day collection.

Statistical Analysis The primary variables of interest were the differences in the levels of airborne pollutants between suburban and inner city homes. Secondary variables included differences in symptoms in the past 2 weeks, medication and provider use, home characteristics, and dust allergen levels. Analysis was conducted on categorical or log-transformed continuous data. Dichotomous variables were compared by Fisher's exact test and continuous variables were compared by Student's *t* test or Kruskal–Wallis test. Linear regression was performed to model the relationship between inner city home exposures and PM_{10} and $PM_{2.5}$ levels. Analyses were performed using SPSS version 12, STATA version 8.0, and SAS version 9.1.

We estimated that the suburban comparison group size of 20 would allow us 80% power to detect a difference of 35 μg/m^3 PM_{10} compared with the 100 inner-city homes, with an α of .05.

RESULTS

Inner city and suburban homes differed in ways that might affect airborne environmental exposures (Table 1). None of the suburban homes were on arterial streets, compared with almost half of the inner city homes ($p<.001$). Inner city homes were more commonly row houses or apartments, whereas 80% of suburban homes were detached houses or duplexes ($p<.001$). Inner city homes were more likely to show signs of neglect. Over 75% of inner city homes had signs of disrepair, most commonly damaged walls and ceilings or peeling paint, but also including serious damage such as leaky roofs or broken windows. More of the inner city homes had a cigarette smoker (67% vs. 5%, $p<.001$) and a gas stove (84% vs. 40%, $p<.001$), and fewer inner city homes had carpeting in the child's bedroom (48% vs. 95%, $p<.001$) and central air conditioning (17% vs. 95%, $p<.001$).

Thirty-one percent of inner city homes had signs of moisture damage. The only suburban home with water damage had a plumbing leak in the family room, whereas 15% of the inner city homes had moisture in one room, 11% had moisture in two rooms and 3% had moisture in all three rooms. Only inner city homes had evidence of leaky roofs, suggesting that home structural damage may have been a greater cause of leaks from the outdoors in the inner city. Sixty-one percent of inner city homes had a musty smell in some room, compared to 25% of suburban homes ($p=.006$). Of the inner city homes, 21% had a musty smell in one room, 12% in two rooms, and 19% in three rooms. In the suburbs, 2/20 homes had a musty smell in two of the three rooms. Visible mold and mildew were not significantly different in the inner city homes, either in the rate in a given room or in the number of rooms with mold growth. Of the inner city kitchens, 19% had mildew at one site and 4% at two sites, whereas of the suburban kitchens, only 10% had mildew at one site ($p=.17$).

All airborne pollutant levels were higher in inner city homes (Table 2). Median and GM PM_{10} levels were almost three times as high in the inner city homes as in the suburban homes (GM 47 vs. 18 μg/m^3, $p<.001$). Median and GM $PM_{2.5}$ levels were more than three times higher in the inner city homes (GM 34 vs. 8.7 μg/m^3, $p<.001$). Median and GM NO_2 levels were 19 ppb in the inner city and BD in the

TABLE 2 Indoor pollutant levels

	Suburbs								City								
	N	25th	Median	75th	AM	ASD	GM	GSD	N	25th	Median	75th	AM	ASD	GM	GSD	P^a
PM_{10} ($\mu g/m^3$)	20	14	18	23	23	17	18	1.9	93	31	48	71	57	41	47	1.9	<.001
$PM_{2.5}$ ($\mu g/m^3$)	18	4.6	9.8	15	12	8.6	8.7	2.3	91	21	35	57	45	37	34	2.1	<.001
O_3 (ppb)	20	BD	BD	BD	.027	.040	.015	2.5	88	.99	1.6	2.5	2.7	3.2	1.9	2.1	<.001
NO_2 (ppb)	20	BD	BD	BD	BD	4.6	BD	BD	95	10	19	37	32	40	19	2.7	<.001

[a]Student's *t* test of log data

TABLE 3 Relationship of housing characteristics and particulate levels in the inner city[a]

		N	Median PM_{10} μg/m^3	*p* (GM)	*N*	Median $PM_{2.5}$ μg/m^3	*p* (GM)
Current smoking	Yes	60	53		61	39	
	No	33	34	.015	30	23	.016
House on arterial street	Yes	43	52		40	39	
	No	50	44	.35	51	35	.35
Carpet in bedroom	Yes	44	50		42	36	
	No	47	45	.61	47	35	.77
Dog	Yes	19	59		18	47	
	No	72	44	.21	71	35	.11
Cat	Yes	30	52		30	44	
	No	62	45	.25	60	30	.04
Cat or dog	Yes	41	54		40	44	
	No	50	42	.07	49	29	.009
Gas stove	Yes	77	49		75	38	
	No	16	21	.02	16	18	.07
Moisture in any room	Yes	27	52		27	39	
	No	65	44	.23	63	35	.43
Musty smell	Yes	55	52		53	38	
	No	38	42	.09	38	31	.28
Mildew	Yes	29	48		29	35	
	No	63	48	.95	61	38	1.00
Drier vented to outside	Yes	15	37		15	25	
	No	78	50	.21	76	37	.38
Leaky roof	Yes	22	50		20	39	
	No	71	45	.63	71	35	.32
Cracks in inside walls	Yes	59	48		58	37	
	No	34	46	.45	33	33	.83

[a]Numbers may not add to N=93 for PM_{10} and N=91 for $PM_{2.5}$ because of missing home inspection data

suburban homes (p<.001). GM O_3 levels were 1.9 ppb in the inner city and .015 ppb in the suburban homes (p<.001).

We sought possible explanations for the higher inner city pollutant levels. The higher particulate levels in the inner city related in part to the higher rate of cigarette smoking in inner city homes (67%). Within inner city homes with a smoker, PM_{10} levels were almost twice as high as in inner city homes without a smoker (53 vs. 34 μg/m^3, p=.015 for GM); $PM_{2.5}$ levels were also higher in homes with a smoker (39 vs. 23 μg/m^3, p=.016 for GM). At the same time, the PM_{10} level was 34 μg/m^3 in inner city homes without smokers compared to 18 μg/m^3 in the 19 suburban homes without a smoker (p<.001 for GM), so there are likely additional explanations for the difference. More inner city homes were located on an arterial street, which may have contributed to the elevated inner city PM levels; among the 45% of inner city homes on arterial streets, PM_{10} levels were higher than in homes not on arterial streets (52 vs. 44 μg/m^3, p=.035 for GM).

Particulate levels were higher in inner city homes with a gas stove (PM_{10} 49 vs. 21 μg/m^3, p=.02 for GM). $PM_{2.5}$ levels were higher in homes with cats or dogs (Table 3). Multivariate modeling suggested that inner city PM_{10} levels were influenced by the presence of a cigarette smoker or a gas stove and that $PM_{2.5}$ levels were influenced by the presence of a cigarette smoker (Table 4). Open combustion contributes to NO_2 production and the high proportion of gas stoves in inner city homes was associated with higher NO_2 levels (19 ppb vs. BD, p=.001 for GM). No

TABLE 4 Linear regression model for the prediction of particulate matter in the inner city (log_{10} transformed, N=91 for PM_{10}, N=89 for $PM_{2.5}$)

	Log PM_{10}			Log $PM_{2.5}$		
Independent variables	Coefficient	SEM	*p*	Coefficient	SEM	*p*
Intercept	1.429	.073	<.0001	1.290	.091	<.0001
Cigarette smoke	.131	.058	.025	.159	.073	.035
Gas stove	.155	.077	.048	.102	.096	.289
Cat or dog	.044	.059	.465	.138	.074	.066
Musty smell	.021	.059	.728	−.012	.074	.874
Arterial street	.011	.056	.843	.005	.071	.942

other home characteristics, including kitchen ventilation or use of auxiliary heating sources were associated with a higher NO_2 level. No home characteristics were associated with higher ozone levels. There was no significant seasonal variation of PM_{10}, $PM_{2.5}$, and NO_2, although O_3 was significantly higher in the warmer months (July median 3.3 ppb [range BD–9.5] vs. February median BD [range BD–.96], $p<.001$ for GM).

We compared bedroom dust allergen levels in the inner city and suburban homes. The pattern of dust allergen concentration differed in inner city and suburban homes (Table 5). Inner city homes had higher concentrations of cockroach and mouse allergen than suburban homes, reflecting the higher rate of reported infestation (Table 1). Suburban homes contained higher levels of dust mite, cat and dog allergen, reflecting the higher rate of pet ownership in these homes. Homes with dogs had substantially higher Can f 1 levels than homes without dogs (13.4 vs. .17 μg/g, $p<.001$ for GM). Cat allergen levels were lower in the inner city; however, cat ownership was similar (26% vs. 15%, $p=.40$). Homes with cats had significantly higher Fel d 1 levels than homes without cats (16.9 vs. .43 μg/g, $p<.001$ for GM).

The children recruited from the inner city and suburbs were similar with respect to gender, but different with respect to age, ethnic background, socioeconomic background, and allergen sensitization (Table 6). Markers of asthma disease severity, including baseline FEV_1 and asthma symptoms in the 2-week period before the study, were also similar in the two groups, as was medication use. Children in the inner city tended to take less inhaled steroid medications or other medications capable of controlling asthma. In the 3 months before entering the study, eight inner city children had been hospitalized for asthma compared to none of the suburban children, and 34 inner city children had ER visits, compared with three suburban children ($p=.048$). In the 2 weeks before the study, seven inner city children had exacerbations requiring oral steroids compared with none of the suburban children. These differences were consistent with previous publications.[3,4]

DISCUSSION

Inner city homes had higher levels of particulate matter, NO_2, and O_3 than found in suburban homes, and a different pattern of allergen in settled dust. Our findings document the differences in inner city Baltimore and suburban homes of children with asthma, with respect to type of home, location, state of repair, moisture, carpeting, and cigarette smoke exposure. Airborne mouse allergen levels and

TABLE 5 Dust allergen and airborne mouse allergen levels

	Suburbs *N*=19							City *N*=98							
	25th	Median	75th	AM	ASD	GM	GSD	25th	Median	75th	AM	ASD	GM	GSD	*P*[a]
Dust Mite (μg/g)	.20	.39	28	16	29	1.2	11	.15	.18	.43	.63	1.4	.29	2.7	.022
Bla g 1 (U/g)	BD	BD	BD	.46	.30	.42	1.4	.99	4.7	16	19	37	4.5	6.2	<.001
Mus m 1 (μg/g)	.006	.009	.023	.023	.034	.013	2.7	1.0	4.0	11	13	35	3.2	6.1	<.001
Fel d 1 (μg/g)	.48	1.3	7.5	39	141	2.4	8.5	.19	.49	3.3	9.1	29	.75	9.1	.039
Can f 1 (μg/g)	1.6	7.8	36	23	37	5.5	8.5	.039	.26	1.3	16	60	.38	13	<.001
Airborne Mus m 1 (ng/m^3)	BD	BD	BD	.12	.42	.016	4.0	.01	.060	.18	.17	.31	.055	4.6	.002

[a]Student's *t* test of log data

TABLE 6 Characteristics of suburban and inner-city children with asthma[a]

	Suburbs % (*N*=20)	City % (*N*=100)	*p*
Male gender	55%	46%	.48
Age (mean ± SD)	13.2±3.0	8.8±1.5	<.001
Ethnic background			<.001
African American	20	99	
Asian	5	0	
White	75	0	
Unanswered		1	
Income			.026
<$50,000	0	95	
>$50,000	60	0	
Unanswered	40	5	
FEV 1 % predicted (mean ± SD)	94 ± 12	97 ± 21	.34
Skin prick test		*N*=99	
Dust mite	100	28	<.001
Bla g 1	35	40	.80
Mus m 1	25	9	.059
Fel d 1	80	20	<.001
Can f 1	30	3	.001
Any positive	100	70	.003
Asthma severity[b]			.53
Mild intermittent	35	34	
Mild persistent	30	25	
Moderate persistent	35	33	
Severe persistent	0	8	
Current asthma provider	100	96	1.0
Hospitalizations (3 months)	0	8	.35
ER visits (3 months)	15	34	.048
Medications (2 weeks)			
Quick reliever	55	54	1.0
Controller	55	31	.070
Inhaled steroids	45	17	.014
Steroid bursts	0	7	.60

[a]Percent of children with characteristics
[b]Based on NAEPP grading [NIH]

bedroom dust levels of mouse and cockroach allergen were higher in the inner city, whereas dust mite, dog and cat allergen levels were higher in suburban homes.

Our finding that particulate levels in inner city Baltimore homes were more than twice as high as those in suburban homes may be compared to earlier reports. In urban homes, some of which contained smokers, Pellizzari and colleagues[20] reported median PM_{10} levels of 28 μg/m^3 in Indianapolis and 23 μg/m^3 in Toronto; $PM_{2.5}$ levels were 18 and 15 μg/m^3, respectively. Liu and colleagues measured indoor particulate levels in 19 nonsmoking homes of 6- to 13-year-old children with asthma living in Seattle and found geometric mean PM_{10} levels of 16.8±1.7 μg/m^3 and $PM_{2.5}$ levels of 7.9±1.7 μg/m^3.[21] Williams and colleagues[22] reported levels of PM_{10} (10 μg/m^3) and $PM_{2.5}$ (8.5 μg/m^3) in homes in a retirement center; they commented that these unusually low levels were probably related to decreased cooking and other activities in the retirement site that they studied. There are few inner city comparators. Using laser nephelometry that approximated $PM_{2.5}$ levels,

Wallace and colleagues[9] examined 292 inner city homes in eight U.S. cities and found geometric mean levels of 17.2±2.6 μg/m^3, substantially lower than our levels. Using methods similar to the ones used in the present study, Keeler and colleagues[23] found arithmetic mean PM_{10} levels of 52.2 μg/m^3 and $PM_{2.5}$ levels of 34.4 μg/m^3 in inner city Detroit. Thus, using various methodologies, earlier studies found similar levels to those we report in suburban homes, but variable levels in inner city homes.

It was tempting to explain the higher PM levels in inner city homes by the large proportion of these homes containing a smoker (67%). Environmental tobacco smoke is the major contributor to indoor PM. Wallace and colleagues[9] reported that $PM_{2.5}$ levels were 37 μg/m^3 higher in homes with at least one smoker. Keeler and colleagues[23] reported a PM increase in homes with a cigarette smoker comparable to our results (33%), and found that these differences were similar in winter, when windows were closed, and in summer. In our study, inner city homes with at least one smoker had PM_{10} levels of 53 μg/m^3 compared to 34 μg/m^3 in nonsmoking homes. At the same time, when we compared PM levels in homes in the suburbs and inner city in which smoking was not reported, PM levels remained significantly higher in the inner city homes, so other urban sources of airborne particles, such as traffic, may be contributing.[9]

Other home characteristics associated with particulate levels in bivariate analysis and linear regression included the presence of a cat or dog and a gas stove. Although the multivariate model was likely more valid, there was no good explanation for an association of higher particulate levels with cat or dog ownership or the presence of a gas stove. Although frying and burning during cooking are important contributors to indoor particulate levels[9], both gas and electric stoves have been associated with elevated particulate levels.[9,24]

In the suburban homes, the range of O_3 levels was small. In both the city and suburban homes, air monitoring was conducted in more than one season and included data collected during the warmer seasons, when ambient O_3 levels are expected to be higher. Although O_3 levels are expected to be higher in urban areas than in neighboring regions[25], O_3 exposure in suburban areas is becoming an increasing concern,[26] and our study showed remarkably low levels of O_3 in the suburbs. The lower O_3 levels in the suburban homes may reflect a greater distance from arterial streets or windows being left closed on days of high ambient O_3 levels, compared with the inner city homes.

In this study, the suburban homes of children with asthma contained a lower percentage of smokers than that of the general Maryland population reported in the Maryland Tobacco Statistics.[27] In that survey, the percentages of households with minor children and an adult who smoked cigarettes were 49.6% in Baltimore City, 35.1% in Baltimore County, 31.8% in Anne Arundel County, and 27.1% in Howard County. In our study, a higher percentage of homes had a smoker in the inner city (67%), than had been reported in the earlier survey of Baltimore city as a whole. This increase may explain the higher airborne particulate levels found in this study, compared with the levels in other studies, where inner city homes were not specifically selected. The lower percentage of smokers in the suburban homes of this study (5%) compared to all homes with a child in counties surrounding Baltimore (27–35%) may reflect a volunteer bias or changes in practice as a result of participation in a previous asthma trial.

Although standards specific for home indoor air quality have not been established, we found that inner city children were exposed to home pollutant levels in excess of the Environmental Protection Agency's National Ambient Air

Quality Standards.[28] We found that 53% of the inner city children studied were exposed to $PM_{2.5}$ levels that exceeded the 24-hour $PM_{2.5}$ standard (35 $\mu g/m^3$), whereas none of the suburban homes reached this level. Eighty-five percent of inner city homes and 22% of suburban homes had $PM_{2.5}$ levels higher than the annual standard (15 $\mu g/m^3$). Fourteen percent of inner city homes had NO_2 levels above the EPA recommended limit (53 ppb). No suburban home had NO_2 levels greater than the EPA standard.

We found that 31% of inner city homes had evidence of water damage, compared to only one suburban home, and 32% of inner city homes had evidence of mildew. By comparison, census data for the entire United States describe exterior leaks found in 11% of urban homes and 12% of suburban homes. Excess residential moisture has been shown to affect asthma symptoms, so these differences are likely to be significant health risks. A survey of nearly 6,000 Russian children in nine cities reported water damage and mold in 10% of living areas in the past 12 months. Compared to homes without water damage and molds, the odds ratios (OR) of current asthma-like symptoms were 1.77 (95% CI 1.36–2.03) for water damage and 1.98 (1.53–2.55) for presence of molds.[29] In a survey of nearly 15,000 Canadian children in 30 cities, molds were reported in 32% of homes, flooding in 24%, and moisture in 14%. Compared to homes without mold, the OR for cough with mold at one site in a home was 1.61 (95% CI 1.36–1.89) and the OR for cough with mold at two sites in a home was 2.26 (1.80–2.83). Similar effects were seen for wheeze, dyspnea, and a diagnosis of asthma.[10] In a case-control study of Dutch children with asthma, home dampness was shown to be strongly associated with sensitization to dust mites or mold, in combination with respiratory symptoms.[30]

More inner city homes had signs of mouse and cockroach infestations, and higher measured dust Bla g 1 and Mus m 1 allergen levels were seen in inner city homes with signs of infestation. Although previous studies have detected Bla g 1 in the kitchens of suburban homes with no history or signs of cockroach infestation[31], we did not detect Bla g 1 in the child's bedroom of suburban homes. Cat ownership was not significantly different between inner city and suburban homes, and we speculated that the higher bedroom dust allergen levels of Fel d 1 in the suburbs might reflect urban residents keeping cats outdoors.

The strength of this study is that we used the same questionnaires and techniques to compare home characteristics and air pollutants in inner city and suburban homes. The format for the questionnaires and home inspection checklists of the suburban comparison group was dictated by those used for the inner city group. In addition, participation in the suburban group was restricted to children with a positive skin test to an indoor allergen. However, within the limitations of the study design, the inner city and suburban groups were a representative sample of the large numbers of children with doctor-diagnosed asthma from which they were selected.

This study had several weaknesses that affected its interpretation. A small number of homes were studied in the suburban setting. In addition, the suburban homes were selected from allergic children with asthma who had recently completed a clinical trial that selected participants based on allergic asthma and had tested an intervention intended to modify families' allergen reduction strategies. However, the inner city participants included both allergic and non-allergic children with asthma. It is entirely possible that suburban homes had been modified by the intervention in ways that would have decreased environmental

conditions that we have examined. Given the differences that we describe here, it will be important to confirm these findings.

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