Home environmental intervention in inner-city asthma: a randomized controlled clinical trial

Peyton A. Eggleston, MD*; Arlene Butz, RN, ScD*; Cynthia Rand, PhD*; Jean Curtin-Brosnan, MA†; Sukon Kanchanaraksa, PhD†; Lee Swartz, MBA*; Patrick Breysse, PhD†; Timothy Buckley, PhD†; Gregory Diette, MD, MHS*; Barry Merriman, MA*; and Jerry A. Krishnan, MD, PhD*

Background: Airborne pollutants and indoor allergens increase asthma morbidity in inner-city children; therefore, reducing exposure, if feasible, should improve asthma morbidity.

Objective: To conduct a randomized controlled trial of methods to reduce environmental pollutant and allergen exposure in the homes of asthmatic children living in the inner city.

Methods: After the completion of questionnaires, spirometry and allergen skin tests, home inspection, and measurement of home air pollutant and allergen levels, 100 asthmatic children aged 6 to 12 years were randomized to the treatment group (home-based education, cockroach and rodent extermination, mattress and pillow encasings, and high-efficiency particulate air cleaner) or to the control group (treated at the end of the 1-year trial). Outcomes were evaluated by home evaluations at 6 and 12 months, clinic evaluation at 12 months, and multiple telephone interviews.

Results: In the treatment group, 84% received cockroach extermination and 75% used the air cleaner. Levels of particulate matter 10 μm or smaller declined by up to 39% in the treatment group but increased in the control group ($P < .001$). Cockroach allergen levels decreased by 51% in the treatment group. Daytime symptoms increased in the control group and decreased in the treatment group ($P = .04$). Other measures of morbidity, such as spirometry findings, nighttime symptoms, and emergency department use, were not significantly changed.

Conclusions: A tailored, multifaceted environmental treatment reduced airborne particulate matter and indoor allergen levels in inner-city homes, which, in turn, had a modest effect on morbidity.

Ann Allergy Asthma Immunol. 2005;95:518–524.

INTRODUCTION

Although many factors contribute to the excessive asthma morbidity in children in US inner cities, indoor environmental allergen and pollutant exposures have been shown to be important contributors in cross-sectional epidemiologic studies.[1–3] The association of asthma with indoor exposures suggests that environmental interventions would be a sensible public health measure to reduce asthma morbidity in this population, but the few clinical trials[4–6] reported to date either have included environmental controls in global interventions or have not used effective interventions. A notable exception is the Inner-City Asthma Study, which recently reported the successful treatment of asthma using an environmental intervention.[6] Based on evidence from inhalation challenges and animal models that airborne particulates and allergens affect asthma synergistically,[7,8] we created an intervention that combines strategies to reduce allergen and particulate exposures. The purpose of this study was to test the efficacy of a home-based intervention in reducing allergen and particulate exposure in a randomized controlled clinical trial. In addition, we hypothesized that the successful reduction of levels of particulates, allergens, or both could improve the health of asthmatic children living in the home.

METHODS

The methods used have been published previously.[9,10] In summary, we recruited participants from graduates of a school-based asthma education program in Baltimore public schools. A recruiter/interviewer visited the families to determine eligibility (6–12 years old, physician-diagnosed asthma, current asthma symptoms, and no other chronic lung disease) and to obtain informed consent. Institutional review boards for The Johns Hopkins University and the city of Baltimore approved the study.

Questionnaires detailing demographic, medical, psychosocial, and environmental characteristics were administered at the baseline home visit. The home environmental evaluation visit included an inspection using a checklist that detailed the housing condition, water damage, evidence of infestation, and bedding condition.[11] Household dust samples were collected on an unwoven fabric sleeve inserted into the nozzle of a standard portable vacuum. Samples were collected from 3

* Departments of Pediatrics and Internal Medicine, the Johns Hopkins University School of Medicine, Baltimore, Maryland.
† Departments of Environmental Health Science and Epidemiology, the Johns Hopkins University Bloomberg School of Public Health, Baltimore, Maryland.

This study was supported by grant R82672401 from the US Environmental Protection Agency, grant ES09606 from the National Institute of Environmental Health Sciences, grant HL058942 from the National Heart, Lung, and Blood Institute, and a grant from the US Environmental Protection Agency's Science to Achieve Results (STAR) program.
Received for publication February 28, 2005.
Accepted for publication in revised form April 2, 2005.

sites (the child's bedroom, the television/living room, and the kitchen) using standard methods.[12] The bedroom sample was collected by vacuuming a 1-m^2 area near and under the bed for 2 minutes combined with a 2-minute sample from the mattress and bedding. 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 sieved dust specimen (sieve size, 300 μm) was prepared in borate-buffered saline. The extracts were stored at −30°C until assayed for house dust mite allergens (Der p 1 and Der f 1), cat allergen (Fel d 1), dog allergen (Can f 1), mouse allergen (Mus m 1), and cockroach allergen (Bla g 1) using sandwich enzyme-linked immunosorbent assays.[12–16] Air pollution sampling was conducted during a 72-hour period in the child's bedroom. Levels of particulate matter 10 μm or smaller (PM_{10}) and 2.5 μm or smaller ($PM_{2.5}$) were collected at a flow rate of 4 L/min on Marple-Miller impactors (MSP Corp, Shoreview, Minn) loaded with a 37-mm filter with a 2.0-μm pore size. Ozone and nitric oxide were sampled passively using Ogawa badges. Time-resolved PM was also evaluated using a portable direct-reading nephelometer with data logging capabilities (MIEpDR100s, Thermo Electron Corp, Franklin, MA).[9,17,18] At a clinic visit, parents and children were administered psychosocial and quality-of-life questionnaires.[10,11,19,20] The children underwent prick-puncture skin testing to 14 aeroallergens and spirometry.

After evaluation, families were randomized to the treatment group, which received intervention immediately, or to the control group, which received the intervention at the end of the study. Follow-up evaluations were conducted quarterly by telephone to administer questionnaires detailing the home environment, asthma-related symptoms, quality of life, health care utilization, and adherence to environmental control recommendations. Home environmental evaluations were conducted at 6 and 12 months, and the clinic visit was repeated at the conclusion of the study. All follow-up contacts were conducted by staff unaware of the participant's study assignment.

The intervention consisted of a combination of physical and behavioral interventions.[10] Each family was given a room-sized high-efficiency particulate air (HEPA) filter (Holmes Products Inc, Milford, MA), which was placed in the child's bedroom. The on/off switch was locked, and the unit was fitted with an electrical field measuring device (Onset Computer, Bourne, MA) to monitor compliance with its use. Allergen-proof mattress and pillow encasings (Mission: Allergy, Hawleyville, CT) were fitted to the child's bed. Families with evidence of cockroach infestation or with a child who was allergic to cockroach were provided free professional extermination (American Pest Management, Takoma Park, MD). Cockroach extermination followed the principles of integrated pest management, with placement of MaxForce FC bait gel containing 0.01% fipronil (The Clorox Corp, Oakland, CA) in the kitchen and bathroom and in other rooms as needed. Mouse extermination was provided through bait traps containing 0.005% bromdialone (Protecta RTU; Bell Laboratories Inc, Madison, WI). Obvious mouse entry points were closed with wire mesh, and the family was provided with plastic food containers to reduce cockroach and mouse reinfestation. A second application was placed a week later for heavy infestations. An additional professional exterminator visit was provided for families that reported continuing infestation at 6 months.

The behavioral interventions were provided by a trained environmental educator at 3 home visits and during a telephone follow-up during a 5-month period. The educator provided the family with the results of their child's allergy testing, the results of their home inspection, and pollutant and allergen levels. Information was provided about avoiding environmental tobacco smoke and indoor allergen sources, and behavior was modeled to reduce exposure. The air filter maintenance and pest control were explained. The content and sequence of the interventions were tailored to increase awareness, establish goals, and increase self-efficacy.

The primary outcome variables were household levels of airborne particulates (PM_{10} and $PM_{2.5}$) and cockroach allergen Bla g 1 levels in settled dust. Secondary outcomes included symptoms in the past 2 weeks. Outcomes were analyzed for differences between the treatment and control groups at 6 and 12 months and for the proportional change from baseline. The absolute reduction in the proportion of children with symptoms by treatment group was compared at each follow-up visit (3, 6, 9, and 12 months). Overall differences during follow-up between groups were also compared using generalized estimating equations to account for possible correlations between repeated measurements within participants and the presence of symptoms at baseline.[21] Forced expiratory volume in 1 second (FEV_1) and quality-of-life scores between groups at baseline and 12 months were compared using *t* tests. Statistical significance was assumed at $P < .05$.

We estimated that a sample size of 50 per group was necessary for 80% power to be able to detect a 50% reduction in airborne particulate levels and a 38% reduction in settled dust allergen levels. Based on the same assumptions, the trial was powered at a 74% level to detect a clinically significant change in the quality-of-life scale. Analyses were performed using Stata version 8.0 (StataCorp, College Station, TX) and SPSS version 11 (SPSS Inc, Chicago, IL).

RESULTS

As shown in Figure 1, 387 children enrolled in the school-based asthma education program, and 180 families were referred to the study recruiter. Of these 180 families, 100 (56%) were randomized in the study. Most children were female (54%), with a mean age of 8.4 years (age range, 6.1–11.9 years). Eighty-nine percent of the caretakers were single women, 46% had less than a high school education, and 74% had symptoms of depression on a standard interview.[20] Seventy-three percent of families lived below the 2000 poverty level.[22] Twenty-one percent of the children

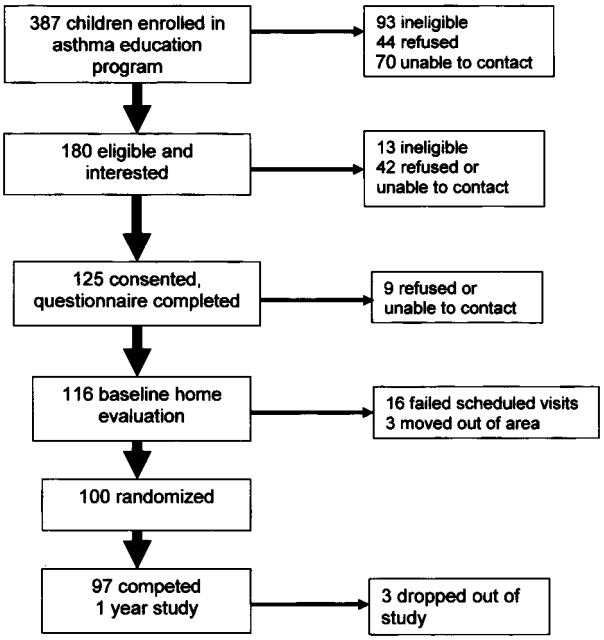

Figure 1. Study flowchart.

were born at least 3 weeks prematurely, and 18% received ventilator support after birth.

At baseline, 54% of the children reported daytime asthma symptoms in the previous 2 weeks (mean, 2.1 of 14 days), and 39% reported nighttime symptoms. Thirty-four percent of the children had a health care visit for acute asthma in the past 3 months, and 30% took daily controller medications. Mean FEV_1 was 97% of predicted. Sixty-eight percent of the children had at least 1 positive skin test reaction; 42% had a positive reaction to cockroach, 47% to pollen, and 29% to house dust mite. Sixty-nine percent of the homes contained at least 1 smoker, and 43% of the mothers smoked. Children in the treatment and control groups were similar regarding sociodemographic and health characteristics (Table 1).

Ninety-one percent of the children lived in row homes. These homes generally were in poor repair, with 24% having a leaky roof and 18% having evident leaks in the child's bedroom. Forty-three percent of the children's bedrooms were carpeted. Mouse allergen Mus m 1 was found in dust samples from all the children's bedrooms, and cockroach allergen Bla g 1 was found in most bedrooms (Table 2). House dust mite allergen was commonly undetectable, and median levels were near the detection limits. Cat allergen levels were relatively low. Although the median PM concentrations were lower than Environmental Protection Agency–recommended 24-hour standards for ambient air (PM_{10}: 150 $\mu g/m^3$ and $PM_{2.5}$: 65 $\mu g/m^3$),[23] PM_{10} and $PM_{2.5}$ levels exceeded these levels in 2% and 18% of homes, respectively. Particulate concentrations were higher in the 69% of homes that contained a smoker: mean ± SD $PM_{2.5}$ concentrations were 58.8 ± 42.2 $\mu g/m^3$ in smokers' homes compared with 25.8 ± 14.9 $\mu g/m^3$ in nonsmokers' homes, and mean ± SD PM_{10} concentrations were 72.3 ± 47.2 $\mu g/m^3$ in smokers' homes compared with 37.7 ± 18.8 $\mu g/m^3$ in nonsmokers' homes.

During the 1-year trial, 3 families left the study. Twenty-three participants moved at least once during the study, and 49 changed their telephone numbers. Despite these obstacles, 91% of the follow-up contacts were completed. Compliance

Table 1. Baseline Characteristics of the Treatment and Control Groups

	Control group (n = 50)	Treatment group (n = 50)
Age, mean ± SD, y	8.3 ± 1.4	8.5 ± 1.5
Female sex, %	60	48
African American, %	98	100
Income, %		
<$10,000	33	45
$10,000–20,000	42	34
>$20,000	25	21
Single caretaker, %	68	82
Smoker in home, %	65	73
Cat or dog in home, %	40	38
Reported cockroach infestation, %	66	62
Reported mouse infestation, %	76	84
FEV_1, mean ± SD, % of predicted	94 ± 21	101 ± 20
ED visit for asthma (3 mo), %	36	32
Hospitalization (3 mo), %	2	14
Moderate-severe persistent symptoms, %	20	28
Controller medications, %	34	28
Positive skin test result, %		
House dust mite	36	23
Cockroach	44	40
Mouse	8	10
Cat	26	19
≥1 positive result	65	75

Abbreviations: ED, emergency department; FEV_1, forced expiratory volume in 1 second.

Table 2. Bedroom Pollutant and Allergen Levels at Baseline

	Children, No.	% Detectable	Median (IQR)
PM_{10}, $\mu g/m^3$	93	100	47.8 (30.6–71.0)
$PM_{2.5}$, $\mu g/m^3$	91	100	35.1 (20.8–57.1)
Nitric oxide, ppb	95	75	19.1 (9.7–36.5)
Ozone, ppb	88	28	1.6 (1.1–2.5)
Bedroom allergen levels			
Bla g 1, U/g	98	78	4.7 (1.0–15.5)
Der p 1 and Der f 1, ng/g	98	59	62 (BD-324)
Fel d 1, $\mu g/g$	98	96	0.5 (0.2–3.4)
Mus m 1, $\mu g/g$	98	100	3.7 (0.9–10.3)

Abbreviations: BD, below detection; IQR, interquartile range; PM_{10}, particulate matter 10 μm or smaller; $PM_{2.5}$, particulate matter 2.5 μm or smaller.

with cockroach intervention was excellent. Forty-two families (84%) received an initial cockroach extermination, and 9 received a second application within a month. Thirty-nine families (78%) received additional treatment at 6 months. Thirty-five families (70%) received an initial mouse extermination, and 10 received a second application. Thirty-nine families received additional treatment at 6 months. Only 2 parents stopped smoking, although 40% (20 in the treatment group and 18 controls) reported smoking outside. Mattress and pillow encasings were reported to be in place in 78% of the families at 3 months, 53% at 6 months, 39% at 9 months, and 27% at 1 year. At 6 months, 96% of the families reported having the air cleaner in the home, and 75% reported that they used it all or nearly all the time. Records from the electronic monitor of air cleaner activity showed that the unit was operated at least half of the time in 48% (21/44) of the families in the treatment group.

Particulate concentrations were significantly lower at 6 and 12 months in the treatment homes (Table 3). Levels of PM_{10} decreased 30% at 6 months and 39% at 12 months in the treatment group compared with increases of 8% and 5% in the control group ($P < .001$ at 12 months) (Fig 2). Changes in $PM_{2.5}$ concentrations were also significant. In the treatment group, cockroach allergen Bla g 1 levels in the bedroom decreased by 51% at 6 months and remained at 42% of baseline values at 12 months ($P = .08$).

At baseline, almost half the children reported no symptoms in the previous 2 weeks (Table 4); only 5 children (2 in the treatment group and 3 controls) reported daily symptoms. During the trial, the proportion of symptomatic children increased in the control group and decreased in the treatment group, with significant differences between groups at 6 months and later. There was a significantly greater absolute reduction in the proportion of children with daytime symptoms in the intervention group compared with the control group at 6, 9, and 12 months (Fig 3). Using the generalized estimating equation model, the treatment group was, on average, significantly less likely to report daytime symptoms during the first 9 months compared with the control group

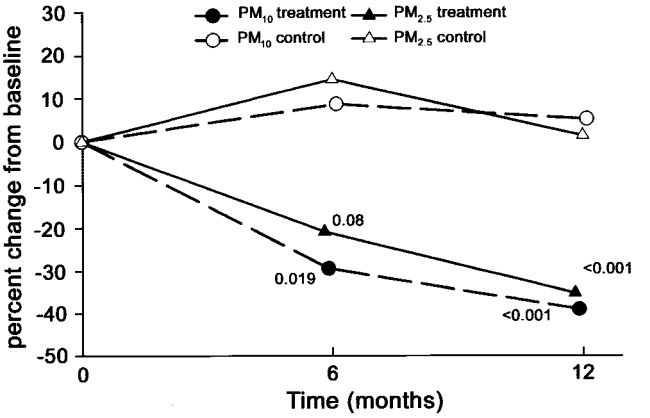

Figure 2. Median change in levels of particulate matter 10 μm or smaller (PM_{10}) and 2.5 μm or smaller ($PM_{2.5}$) measured in the child's bedroom.

after adjusting for baseline symptoms (odds ratio, 0.55; 95% confidence interval, 0.31–0.97; $P = .04$). However, the mean difference between groups was marginal comparing daytime symptoms across all 12 months of follow-up (odds ratio, 0.62; 95% confidence interval, 0.36–1.05; $P = .07$). The mean differences in other symptomatic outcomes given in Table 4 were not significant.

Other health outcome measures were not different in the 2 groups. At baseline, 32% of the children in the treatment group and 36% in the control group had a visit for acute asthma in the past 3 months. During the trial, both groups had fewer visits (15% and 13% per 3 months, respectively), but the changes were not statistically significantly different. Hospitalizations were reduced in both groups as well (data not shown). The mean ± SD quality-of-life scores were similar at baseline (3.69 ± 1.28 vs 4.01 ± 1.29) and at 12 months (4.70 ± 1.22 vs 5.00 ± 1.39). The mean ± SD FEV_1 did not change across the study in the treatment group (101% ± 20% of predicted at baseline vs 94% ± 21% of predicted at 12 months) or in the control group (100% ± 21% of predicted at baseline vs 101% ± 20% of predicted at 12 months).

Table 3. Home Pollutant Exposures During the Intervention*

	Control group			Treatment group		
	Baseline	6 mo	12 mo	Baseline	6 mo	12 mo
PM_{10}, $\mu g/m^3$	42 (32–64)	41 (31–65)	40 (29–74)	55 (30–83)	33 (15–67)	31 (18–55)†
$PM_{2.5}$, $\mu g/m^3$	30 (20–45)	29 (22–50)	31 (20–53)	38 (23–70)	23 (10–60)	24 (10–43)†
Nitric oxide, ppb	19 (10–83)	18 (10–43)	20 (13–52)	20 (10–87)	14 (10–136)	23 (9–37)
Bedroom allergen levels						
Bla g 1, U/g	2.8 (0.8–18)	5.3 (1.2–20)	7.1 (1.0–24)	4.9 (1.1–14)	2.2 (0.4–8.9)	2.9 (BD-11)‡
Fel d 1, $\mu g/g$	0.5 (0.15–3.4)	1.4 (0.2–10)	2.4 (0.4–26)	0.5 (0.2–3.0)	0.4 (0.1–1.7)	0.5 (0.2–2.2)
Der p1 and Der f 1, ng/g	0.07 (BD-0.3)	BD (BD-0.2)	0.07 (BD-0.2)	0.05 (BD-0.4)	BD (BD-0.1)	BD (BD-0.2)
Mus m 1, $\mu g/g$	3.7 (0.67–1.3)	3.5 (1.1–14.3)	2.5 (0.68–14)	4.3 (1.2–10)	5.1 (1.1–14)	4.5 (2.6–10)

Abbreviations: BD, below detection; PM_{10}, particulate matter 10 μm or smaller; $PM_{2.5}$, particulate matter 2.5 μm or smaller.
* Data are given as median (interquartile range).
† $P < .001$, difference from baseline compared with control.
‡ Difference from baseline compared with control was not significant.

Table 4. Children Reporting Asthma Symptoms in the Past 2 Weeks*

	Control group					Treatment group				
	Baseline	3 mo	6 mo	9 mo	12 mo	Baseline	3 mo	6 mo	9 mo	12 mo
Daytime symptoms	50	55	66	60	59	58	59	50†	38†	55‡
Symptoms with exercise	51	47	52	42	38	52	41	33†	31	33
Nighttime symptoms	36	41	42	32	31	42	41	36‡	40	30
Interfere with child's activity	60	49	46	42	41	71	57	40‡	31‡	43

* Data are given as percentages.
† $P < .01$, difference from baseline compared with control.
‡ $P < .05$, difference from baseline compared with control.

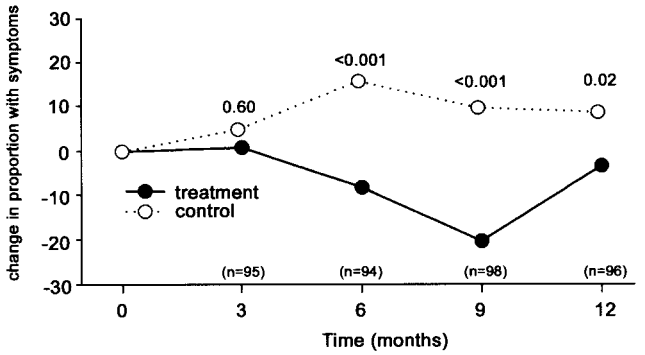

Figure 3. Change in the proportion of children with daytime asthma symptoms. The proportion was significantly lower at 6, 9, and 12 months in the treatment group (*t* test), and the average difference was significant during the first 9 months (generalized estimating equation).

We considered whether children with more severe asthma responded differently. Of the 30 children whose baseline FEV_1 was less than 90% of predicted, changes across the study were similar in the treatment group (mean ± SD, 78% ± 9% of predicted at baseline vs 90% ± 22% of predicted at 12 months) and in the control group (mean ± SD, 73% ± 17% of predicted at baseline vs 89% ± 22% of predicted at 12 months). The change in the proportion of children who reported symptoms was similar in those who reported symptoms at baseline. Among the 54 children who reported symptoms at baseline, the proportion who reported symptoms during the study was similar in the treatment group (69% at 3 months, 60% at 6 months, 50% at 9 months, and 46% at 12 months) and in the control group (68% at 3 months, 84% at 6 months, 68% at 9 months, and 64% at 12 months). Thus, the response to treatment in the more symptomatic children was similar to that seen in the entire study population.

DISCUSSION

The results of this study demonstrate that an intervention that combines strategies to reduce indoor pollutant and allergen exposure in inner-city homes substantially reduces exposures to PM and cockroach allergen. Furthermore, there were modest reductions in asthma symptoms in asthmatic children living in the treated homes.

We reduced the concentrations of airborne PM in the bedrooms of children in the treatment group. Indoor PM levels are the result of a dynamic series of generation and clearance processes. Evidence is consistent implicating smoking as the major indoor contributor, on average adding 30 $\mu g/m^3$ to $PM_{2.5}$. Cooking is also an important indoor PM source. In addition, it has been estimated that 30% to 60% of indoor PM is of outdoor origin.[24,25] Because the intervention was not successful in reducing smoking in the home, the observed indoor PM reduction in the treatment homes was most likely attributable to the room HEPA filter. The PM concentration in untreated homes was consistent throughout the year, whereas the reduction in the treated homes was sustained at 6 and 12 months, suggesting that the filters removed particles at a steady rate and established a new equilibrium that was maintained throughout the year. We are aware of only 1 other published test of the effects of a room air filtration device on airborne PM concentrations in homes,[26] and that trial showed a 73% reduction in suspended particles measured using a light-scattering device for more than 4 weeks. Several studies[27–29] examining effects on airborne allergen levels have not shown large effects on either allergen levels or symptoms.

Cockroach allergen levels were reduced by 51% at 6 months, a value that is comparable with earlier results with intensive interventions[6,13,30] and much better than those with less intensive interventions.[4,5] Although these changes were sustained at 12 months, it was surprising that they did not decrease further given the second extermination elected by most families at 6 months. It was disappointing that we did not reduce levels of mouse allergen despite the application of an effective rodenticide, but different cleaning strategies may be required.

We considered why health effects were not more striking. First, it is possible that methodological problems biased the outcomes. The study treatment assignment was not blinded because it included a large behavioral component that could not be masked. At the same time, the study staff that performed the periodic clinic visits and telephone questionnaires were not involved in the treatment and were blinded to group assignment. Because participants in the active and control groups were informed about their child's sensitization status

and the study's purpose, it is possible that both groups undertook appropriate changes to minimize environmental exposures ('Hawthorne effect'). However, we found significantly greater reductions in PM and Bla g 1 levels in the active treatment group, making this explanation unlikely.

It was also possible that the modest observed health effects could be attributed to our inclusion of study participants with mild disease activity or with no evidence of atopy. Because environmental exposure change in the homes of asthmatic children was the primary outcome, we recruited children who had physician-diagnosed asthma and who reported symptoms in the past year. They did not have to have poor asthma control, daily medication requirements, or an abnormal FEV_1, criteria that are commonly used in clinical trials of asthmatic children, including the recently reported Inner-City Asthma Study.[6,31,32] As a result, approximately half of the children were asymptomatic at enrollment, so that the usual outcomes, ie, FEV_1 and symptom frequency, could not be analyzed, and the health effect was difficult to demonstrate. However, when we analyzed subgroups that reported symptoms at baseline or that had an abnormal FEV_1 at baseline, we did not find that their changes were greater than those of the entire randomized group. We included asthmatic children without positive skin test reactions because pollutant reduction might affect their asthma symptoms. The outcomes in the 55 children with at least 1 positive skin test reaction did not differ from those in the entire study group (data not shown). Thus, we found no evidence that including children with mild asthma or without positive skin test reactions interfered with detecting an effect on their asthma.

We were left with the possibility that the environmental changes were not adequate to affect health. However, the magnitude of the decrease in PM_{10} concentration in the treatment group (22 $\mu g/m^3$) is in the range that has been associated with significant changes in respiratory symptoms in panel studies of asthmatic patients[33] and in cross-sectional studies.[34] In the only previous study[26] of HEPA filters in homes, PM greater than or equal to 0.3 μm was reduced by 73% during 4 weeks and was associated with reduced upper airway but not asthma symptoms. The Inner-City Asthma Study found a similar reduction in bedroom cockroach allergen levels and showed that days with symptoms were statistically significantly reduced.[6] We can also extrapolate from house dust mites, where a reduction in bedroom exposure during an intervention trial of less than 50% is frequently not associated with clinical improvement[35]; the 55% decrease at 6 months and the 40% decrease at 12 months in the present study are in this range. Thus, it is possible that the decreased environmental exposure accomplished by this intervention was not enough to produce a more striking clinical effect. It is not clear whether greater reductions are feasible in the homes or whether it will also be necessary to reduce exposure in other indoor spaces, such as school or friends' homes, but this is a subject for future research.

In summary, a tailored intervention that combined behavioral and physical interventions reduced indoor PM and relevant allergen levels in low-income, inner-city homes. The children enrolled in this trial had relatively mild asthma, with normal pulmonary function test results and many days during the trial with few symptoms. The number of days with various symptoms decreased significantly in the treatment group, although a change in lung function was not seen. The cost of the intervention was approximately $492 per child, including mattress and pillow encasings, the room HEPA cleaner, pest control visits, and educator visits. Adapted as part of comprehensive treatment of asthma in inner-city children, this strategy could contribute to symptom reduction in this vulnerable population and should be feasible in a public health setting.

REFERENCES

1. Rosenstreich DL, Eggleston P, Kattan M, et al. The role of cockroach allergy and exposure to cockroach allergen in causing morbidity among inner-city children with asthma. *N Engl J Med*. 1997;336:1356–1363.
2. Strachan DP, Cook DG. Parental smoking and childhood asthma: longitudinal and case-control studies. *Thorax*. 1998;58:204–212.
3. Mortimer KM, Neas LM, Dockery DW, et al. The effect of air pollution on inner city children with asthma. *Eur Respir J*. 2002;19:699–705.
4. Evans R III, Gergen PJ, Mitchell H, et al. A randomized clinical trial to reduce asthma morbidity among inner-city children: results of the National Cooperative Inner-City Asthma Study. *J Pediatr*. 1999;135:332–338.
5. Carter MC, Perzanowski MS, Raymond A, Platts-Mills TA. Home intervention in the treatment of asthma among inner-city children. *J Allergy Clin Immunol*. 2001;108:732–737.
6. Morgan WJ, Crain EF, Gruchalla RS, et al. Results of a home-based environmental intervention among urban children with asthma. *N Engl J Med*. 2004;351:1068–1080.
7. Diaz-Sanchez A. Tsien A, Casillas A, et al. Enhanced nasal cytokine production in human beings after in vivo challenge with diesel exhaust particles. *J Allergy Clin Immunol*. 1996;98:114–123.
8. Gavett SH, Madison SL, Stevens MA, Costa DL. Residual oil fly ash amplifies allergic cytokines, airway responsiveness and inflammation in mice. *Am J Respir Crit Care Med*. 1999;160:1897–1904.
9. Breysse PN, Buckley TJ, Williams D, et al. Indoor exposures to air pollutants and allergens in the homes of asthmatic children in inner-city Baltimore. *Environ Res.* 2005;98:167–176.
10. Swartz L, Callahan KA, Butz AM, et al. Methods and issues in conducting a community based environmental randomized trial. *Environ Res*. 2004;95:156–165.
11. Mitchell H, Senturia Y, Gergen P, et al. Design and methods of the methods of the National Cooperative Inner-City Asthma Study. *Pediatr Pulmonol*. 1997;24:237–252.
12. Platts-Mills TA, Thomas WR, Aalberse RC, et al. Dust mite allergens and asthma: report of a second international workshop. *J Allergy Clin Immunol*. 1992;89:1046–1060.
13. Wood RA, Eggleston PA, Rand C, et al. Cockroach allergen abatement with sodium hypochlorite in inner-city homes. *Ann Allergy Asthma Immunol*. 2001;87:60–64.
14. Chapman MD, Aalberse RC, Brown MJ, Platts-Mills TAE. Monoclonal antibodies to the major cat allergen Fel d I, II:

single step affinity purification of Fel d I, N terminal sequence analysis and development of a sensitive two-site immunoassay to assess Fel d I exposure. *J Immunol*. 1988;140:812–821.
15. Pollart SM, Smith TF, Morris EC, et al. Environmental exposure to cockroach allergens: analysis with monoclonal antibody–based enzyme immunoassay. *J Allergy Clin Immunol*. 1991;87:505–510.
16. Ohman JL, Hagbeerd K, MacDonald MR, et al. Distribution of mouse allergen in a major mouse breeding facility. *J Allergy Clin Immunol*. 1991;94:810–817.
17. Koutrakis P, Wolfson JM, Bunyaviroch A, et al. Measurement of ambient ozone using a nitrite-coated filter. *Anal Chem*. 1993; 65:209–214.
18. Palmes ED, Gunnison AF, Dimattio J, Tomczyk C. Personal sampler for nitrogen dioxide. *Am Ind Hyg Assoc J*. 1976;6: 570–577.
19. Juniper EF, Guyatt GH, Feeny DH, et al. Measuring quality of life in children with asthma. *Qual Life Res*. 1996;5:35–46.
20. Radloff LS. The CES-D scale: a self-report depression scale for research in the general population. *Appl Psychol Meas*. 1977; 1:385–401.
21. Liang K-Y, Zeger SL. Longitudinal data analysis using generalized linear models. *Biometrica*. 1986;73:13–22.
22. US Department of Health and Human Services. US HHS poverty guidelines. Available at: http://aspe.hhs.gov/poverty. Accessed February 5, 2003.
23. US EPA. National ambient air quality standards for particulate matter; final rule, 62 *Federal Register* 130 (1997) (codified at 40 CFR §50).
24. Wallace LA, Mitchell H, O'Connor GT, et al. Particle concentrations in inner city homes of children with asthma: the effect of smoking, cooking and outdoor pollution. *Environ Health Perspect*. 2003;111:1265–1267.
25. Wallace LA. Indoor particles: a review. *J Air Waste Manage Assoc*. 1996;46:98–126.
26. Reisman RE, Mauriello PM, Davis GB, et al. A double-blind study of the effectiveness of a high-efficiency particulate (HEPA) filter in the treatment of patients with perennial allergic rhinitis and asthma. *J Allergy Clin Immunol*. 1990;85: 1050–1057.
27. Francis H, Fletcher G, Anthony C, et al. Clinical effects of air filters in homes of asthmatic adults sensitized and exposed to pet allergens. *Clin Exp Allergy*. 2003;33:101–105.
28. Van der Heide S, Kauffman HF, Dubois AE, de Monchy JG. Allergen reduction measures in houses of allergic asthmatic patients: effects of air-cleaners and allergen-impermeable mattress covers. *J Allergy Clin Immunol Clin Immunol*. 1997;132: 1217–1223.
29. Wood RA, Johnson EF, Van Natta ML, et al. A placebo-controlled trial of a HEPA air cleaner in the treatment of cat allergy. *Am J Respir Crit Care Med*. 1998;158:115–120.
30. Arbes SJ Jr, Sever M, Archer J, et al. Abatement of cockroach allergen (Bla g 1) in low income, urban housing: a randomized clinical trial. *J Allergy Clin Immunol*. 2003;112:339–345.
31. Peden DP, Berger WE, Noonan MJ, et al. Inhaled fluticasone propionate delivered by means of two different multidose powder inhalers is effective and safe in a large pediatric population with persistent asthma. *J Allergy Clin Immunol*. 1998;102: 32–38.
32. The Childhood Asthma Management Program Research Group. Long term effects of budesonide or nedocromil in children with asthma. *N Engl J Med*. 2001;343:1054–1063.
33. Romieu I, Meneses F, Ruiz S, et al. Effects of air pollution on the respiratory health of asthmatic children living in Mexico City. *Am J Respir Crit Care Med*. 1996;154:300–307.
34. Schwartz J, Dockery DW, Neas LM, et al. Acute effects of summer air pollution on respiratory symptoms reported in children. *Am J Respir Crit Care Med*. 1994;150:1234–1242.
35. Custovic A, Simpson A, Chapman MD, Woodcock A. Allergen avoidance in the treatment of asthma and atopic disorders. *Thorax*. 1998;53:63–72.

Requests for reprints should be addressed to:
Peyton A. Eggleston, MD
Department of Pediatrics
Johns Hopkins Hospital
600 N Wolfe St, CMSC 1102
Baltimore, MD 21287
E-mail: pegglest@jhmi.edu