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# A community-based participatory research study of multifaceted in-home environmental interventions for pediatric asthmatics in public housing

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## Abstract

Pest infestation is a major problem in urban, low-income housing and may contribute to elevated asthma prevalence and exacerbation rates in such communities. However, there is poor understanding of the effectiveness of integrated pest management (IPM) efforts in controlling pediatric asthma, or of the interactions among various interventions and risk factors in these settings. As part of the Boston-based Healthy Public Housing Initiative, we conducted a longitudinal, single-cohort community-based participatory research intervention study. Fifty asthmatic children aged 4–17 from three public housing developments in Boston, Massachusetts, USA successfully completed interventions and detailed environmental, medical, social, and health outcome data collection. Interventions primarily consisted of IPM and related cleaning and educational efforts, but also included limited case management and support from trained community health advocates. In pre-post analyses, we found significant reductions in a 2-week recall respiratory symptom score (from 2.6 to 1.5 on an 8-point scale, $p = 0.0002$) and in the frequency of wheeze/cough, slowing down or stopping play, and waking at night. Longitudinal analyses of asthma-related quality of life similarly document significant improvements, with a suggestion of some improvements prior to environmental interventions with an increased rate of improvement subsequent to pest management activities. Analyses of potential explanatory factors demonstrated significant between-development differences in symptom improvements and suggested some potential contributions of allergen reductions, increased peak flow meter usage, and improved social support, but not medication changes. In spite of limitations with pre-post comparisons, our results are consistent with aggressive pest management and other allergen reduction efforts having a positive impact on clinical health outcomes associated with asthma. Our findings reinforce the multifactorial nature of urban asthma and suggest a need for further study of the relative contributions of and possible synergies between environmental and social factors in asthma intervention programs.

*Keywords:* United States; Asthma; Cockroach; Community-based participatory research; Integrated pest management; Public housing

## Introduction

Asthma prevalence in the US increased from 1980 to the late 1990s (Mannino et al., 2002), with

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continued increases until 2002 among children (National Institutes of Health, 2004). Low income and minority populations suffer greater asthma prevalence and more severe health outcomes (National Institutes of Health, 2004). In New England, lower income is strongly associated with higher asthma prevalence and severity (New England Asthma Regional Council, 2004).

Asthma is potentially responsive to both clinical interventions through medication optimization and educational efforts, as well as to environmental interventions that remove exposure to factors that exacerbate asthma. Clinical trials of environmental interventions have included physical and chemical control of dust mites (Gotzsche, Johansen, Burr, & Hammarquist, 2003), air filtration (Francis et al., 2003; McDonald et al., 2002), HEPA vacuums (Popplewell et al., 2000), and multiple environmental interventions applied together (Carter, Perzanowski, Raymond, & Platts-Mills, 2001; Evans et al., 1999; Krieger, Takaro, Song, & Weaver, 2005; Morgan et al., 2004). However, past studies of pest management and antigen reduction efforts demonstrated difficulties in reducing exposures below exacerbation levels on a long-term basis (Arbes et al., 2003; Carter et al., 2001; Gergen et al., 1999).

There are multiple important design decisions in implementing an asthma intervention study. A crucial but often unacknowledged question is whether to use a community-based participatory research (CBPR) approach (Baker, Homan, Schonhoff, & Kreuter, 1999; Brugge & Hynes, 2005; Israel, Eng, Schulz, Parker, & Satcher, 2005; Minkler & Wallerstein, 2003). Since asthma is widespread in low-income urban areas, members of these communities may be interested in finding effective interventions and participating in relevant research projects. From a research perspective, the value of CBPR lies in its potential to encourage high-risk communities to participate in research, in part by providing tangible benefits to the participants and community and by promoting local changes in policy and practice that remain after the research is completed. Importantly, CBPR may engage communities in such a way that researchers learn from the community about their problems and about potentially effective interventions.

However, there are some tradeoffs. For example, one of the only CBPR asthma intervention studies to date (Krieger et al., 2005) found only modest differences between low-intensity and high-intensity interventions (with social and environmental dimensions of both), suggesting the potential for a Hawthorne effect due to engagement in the study. As control populations may be unacceptable to the community, it is challenging to design CBPR asthma intervention studies that are both acceptable to community partners and that lead to meaningful and useful findings.

An additional design decision is whether to focus on individual or bundled interventions. Bundled interventions complicate attribution of benefits but have often proven more effective (O'Connor, 2005), fitting the complex and multifactorial nature of asthma. Moreover, bundled interventions may be more likely to succeed in high-risk populations with elevated prevalence of multiple risk factors.

In CBPR studies evaluating bundled interventions without a control population, researchers must adopt multiple strategies to evaluate the interventions. One way that causality can be inferred from such studies is by evaluating time trends for health outcomes and multiple hypothesized risk factors. These types of analyses are most interpretable with numerous repeated measures but can be informed by more simple pre-post comparisons.

The Healthy Public Housing Initiative (HPHI) was a CBPR project involving Boston city agencies for housing and health, three universities, several public housing community groups and expert consultants (Hynes et al., 2003). Key CBPR dimensions included involvement of community representatives in the project conception, design and implementation, a decision-making committee that included representation of all partners, and participation of residents in data collection, instrument design, and other key research steps. Our focus on public housing residents was motivated by community concerns and earlier surveys, which suggested that children in public housing may have significantly elevated prevalence of asthma (Brugge, Melly, et al., 2003), potentially associated with a number of environmental, medical, and social risk factors prevalent in low-income urban housing.

The core study of HPHI was a longitudinal, single-cohort pre-post study of integrated pest management (IPM) and related environmental interventions in the homes of asthmatic children. As in many CBPR settings, use of a standard control group was complicated by a number of factors, including hesitation from the community partners, the city, and residents. In addition, our focus on unit-level interventions in multi-family dwellings made it implausible to mask the intervention and avoid spill-over effects. We addressed these limitations with dense longitudinal data collection on both environmental factors and health outcomes, with explicit consideration of a number of medical and social factors. This allowed us to address a significant public health problem in a setting where there are both design and analysis limitations driven in part by the complexity of CBPR.

The goal of this investigation is to evaluate the impact of our interventions on respiratory symptoms. We evaluate whether any health benefits were observed and use questionnaire and environmental sampling data to help determine whether any observed improvements could be attributed to the environmental interventions, concurrent changes in asthma management, or social support-related factors.

## Methods

We recruited asthmatic children from three midrise walk-up family public housing developments in Boston—West Broadway (WeB), Franklin Hill (FH), and Washington Beech (WaB). The primary structural difference among the developments is that WeB has undergone renovations in recent decades (roof and interior work in the late 1980s and a new heating system in 2000), while FH and WaB have not undergone any major renovations since they were built in the 1950s (Brugge, Melly, et al., 2003).

Any children between 4 and 17 who lived in the developments and had self-reported doctor-diagnosed asthma were eligible. Asthma status and severity were corroborated by a pediatric pulmonologist (Levy et al., 2004). Recruitment and enrollment occurred between April 2002 and January 2003, coordinated by Community Health Advocates (CHAs). CHAs were residents of the developments or surrounding neighborhoods who received training about asthma and interviewing techniques, and who were involved in both recruitment/retention activities and primary data collection. Recruitment methods included a survey, advertised enrollment open houses, community meetings, mailbox drops for flyer circulation, and door knocking.

Participants were followed for a period of time before and after interventions, with a minimum of 3 months of data required in both periods. Health outcomes data were collected using multiple approaches over different time periods to best understand the effects of our interventions. Below, we describe the various health instruments and additional data that inform our analyses. All questionnaires were administered in both English and Spanish, and bilingual/bicultural CHAs were involved in pilot testing and question modification to ensure interpretability and cultural competency.

### *Enrollment questionnaire*

Upon enrollment, the caregiver of the asthmatic child was interviewed about family demographics, child and family asthma history, access to health care, and asthma medications and management activities. Caregivers were also asked about multiple risk factors, including cigarette smoking, individual social support (Wade et al., 1997), perceived stress (Cohen, Kamarck, & Mermelstein, 1983), and neighborhood social cohesion and exposure to violence (Sampson, Raudenbush, & Earls, 1997). We also asked about the frequency in the last 2 weeks of various asthma symptoms (wheeze/cough, slowing down or stopping play due to asthma, waking up at night due to asthma, severe asthma attacks) and about hospitalizations in the last 2 months. These questions are our primary health outcome measures.

The enrollment questionnaire was administered again at the end of the study (in the fall of 2003). While we focus primarily on the pre-post comparison of the enrollment questionnaire in this manuscript, results from other data streams inform our interpretation of these findings.

### *Longitudinal health data collection*

Each month, we collected data on caregiver (Juniper et al., 1996b) and child asthma-related quality of life (Juniper et al., 1996a), which included respiratory symptoms, emotional function and activity limitation. These data allowed for longitudinal analyses with individuals serving as their own controls. Although these questionnaires have generally been applied to children age 6 and older, we included younger children as well, and tested the sensitivity of our findings to their inclusion. Questionnaires are answered by the caregiver for children under 8.

### *Allergy testing and allergen sampling*

Allergy testing was conducted with skin testing using the prick puncture method (Levy et al., 2004).

Allergens included an 11-tree mix, a 7-grass mix, ragweed, dog, cat, mouse, cockroach (Bla g 1 and Bla g 2), Der f 1, Der p 1, and multiple fungi (*Alternaria*, *Aspergillus fumigatus*, *Cladosporium*, and *Penicillium*). In addition, Bla g 1 and Bla g 2, Der f 1 and Der p 1, Fel d 1, Can f 1, mouse urinary protein (MUP), and *Alternaria* were measured in dust once pre-intervention and 1–4 times post-intervention (at approximately 1–6 weeks, 10–20 weeks, 19–44 weeks, and 60–66 weeks post-intervention).

All allergens were measured in the air and beds, with Bla g 1, Bla g 2, and MUP measured in the kitchen. Dust samples were collected by vacuum in a 19-mm × 90-mm Whatman cellulose extraction thimble placed in a retrofitted wand extension and capped with a crevice tool, following the protocol from the Inner City Asthma Study (Chew et al., 1998). Air samples were collected using a commercially available electrostatic precipitator (Ionic Breeze Quadra, The Sharper Image, San Francisco, CA) (Custis, Woodfolk, Vaughan, & Platts-Mills, 2003), run for 2-week periods. For all allergens but MUP and *Alternaria*, a sandwich enzyme link immunosorbent assay (ELISA) was used for detection and quantification. For MUP and *Alternaria*, a competitive inhibition ELISA was used. More detailed information about the environmental sampling protocol is available elsewhere (Brugge, Vallarino, et al., 2003; Peters, 2005).

## *Medication evaluation*

We evaluated the degree to which children were properly medicated throughout the study, to determine if medication changes might explain our health outcomes findings. This was based on predefined criteria given available information (Table 1), using an asthma severity classification conducted upon enrollment (Levy et al., 2004).

## *Environmental interventions*

Our environmental interventions included four major components, three of which were primarily designed to address cockroach infestation.

- Assessment by visual inspection and trap placement, sealing cracks and small holes using caulking and expandable spray foam, and targeted crack and crevice treatment using hydromethylnon gel (MaxForce), Abamectin gel granules, and boric acid after intensive cleaning. Participants received 1–5 treatment visits depending on the severity of the infestation.
- One-time intensive cleaning using HEPA vacuum cleaners, cleaning radiators, removing grease and food debris from surfaces, and removing cockroach droppings.
- In-home education about pest reductions, presented in three modules: (1) identification and mitigation of asthma triggers and conditions in the home that support pest infestation; (2) hazards associated with broadcast pesticide applications; (3) resident knowledge about IPM, current practices, and ways to monitor for roaches to facilitate communication with the housing authority and pest contractors. Residents were also given tools to reduce clutter and access to food, including storage bins, covered garbage cans, mops and brooms. In addition, residents were encouraged to use the work order system to get more extensive repairs (i.e., repairs of holes too large to be sealed through caulking

Table 1
Criteria for evaluation of the appropriateness of asthma medication regimens

| | Persistent asthma[a] (mild, moderate, or severe) | Intermittent asthma[a] |
|---|---|---|
| No medications | Improper | Improper |
| No rescue medications | Improper | Improper |
| Rescue medications only | Improper | Proper (unless being used more than 2 times/week) |
| Rescue and controller medications | Proper (unless not using controller medications daily or dosage too low)[b] | Proper[c] |

[a] As determined by a pediatric pulmonologist upon enrollment in the study.

[b] Presumed proper if dosage information not available.

[c] Presumes that asthma was persistent at the time of prescription, even though intermittent symptoms were reported in the 2-week recall questionnaire upon enrollment.

and spray foam), both for documentation purposes and as an important step to follow after the end of our study.

- Replacement of mattresses in the asthmatic child's room with new mattresses with microfiber technology.

The electrostatic precipitators, used for allergen sampling, additionally contributed to the intervention through potential reductions in airborne allergens. These environmental interventions were implemented in two phases given rolling recruitment. Thirteen households at FH received environmental interventions in the fall of 2002, with the remainder in the spring of 2003.

### *Asthma education/case management*

Along with the environmental interventions, we implemented limited asthma education/case management. A Community Health Nurse worked with caregivers on asthma action plans and provided peak flow meters. In addition, she facilitated linkages with health care providers for children in acute distress. CHAs also provided ongoing support, largely as part of data collection visits occurring 1–2 times per month for the duration of the study. The primary goal of the case management was to help isolate the influence of environmental interventions by stabilizing health care before the environmental interventions, as well as to provide ancillary benefits in a CBPR study.

### *Statistical analyses*

The primary objectives of our statistical analyses were to determine whether there were significant reductions in the frequency of asthma symptoms and health care utilization, and if so, what factors might explain these reductions. We primarily focus on the enrollment questionnaire but also consider time trends in quality of life.

For our pre-post enrollment questionnaire analyses, to avoid issues of multiple comparisons, we focused our statistical analyses on an eight-point respiratory symptom score based on four 2-week recall symptom questions, as done previously (Levy et al., 2004), but evaluated individual questions to determine the robustness of our findings. Paired two-sample Wilcoxon tests were used on all continuous outcomes, with McNemar's test on paired proportions used to determine if the frequency of symptoms or other risk factors changed significantly.

While we address issues of causality by evaluating time trends for health outcomes and risk factors, we also explore regression analyses to evaluate whether health improvements are associated with changes in specified risk factors. We consider both logistic regression models (evaluating whether the symptom score increased or decreased) and linear regression models on the magnitude of the symptom score change.

Predictive covariates included a number of demographic, medical, and social factors, along with allergen levels. We focused on Bla g 1 and Bla g 2 as the targets of IPM, but also considered other allergens given the potential for ancillary benefits from intensive cleaning. Because the shape of the dose–response function for asthma exacerbation is not well defined, there are multiple ways in which a covariate representing reductions in allergen loading can be constructed. First, we considered whether concentrations were reduced from above estimated exacerbation cut-points to below those levels, using 8 U/g for cockroaches (Eggleston & Arruda, 2001; Rosenstreich et al., 1997) and 10 μg/g for dust mite (Platts-Mills, Thomas, Aalberse, Vervloet, & Champman, 1992), dog and cat (Ingram et al., 1995). For mouse (Phipatanakul, Eggleston, Wright, & Wood, 2000) and *Alternaria*, the median value was used as the cut-point.

We also created dummy variables for reductions above or below the median reduction for each allergen in air, bed, and kitchen. In addition, we considered the number of allergens for which concentrations were reduced below the cut-point/median or where reductions were above the median reduction, and the same covariates restricted to allergens to which the child was allergic. Within our regression models, given our limited sample size, we included only terms for Bla g 1 and Bla g 2 and the multi-allergen reduction variables. In all cases, we compared the pre-intervention levels with the measurements taken closest to the end of the study.

We also evaluated monthly longitudinal quality of life data to examine time trends that may provide insight about causal factors, using mixed models with random intercepts that correct for autocorrelation of within-subject responses (Clougherty, Levy, Hynes, & Spengler, 2006). Time trends were sectioned (or divided) by optimal knot points, which were selected using the AIC information criteria. This allowed us to identify critical periods for health improvements, both before and after environmental interventions. All statistical analyses were conducted in SAS version 9.

## Results

### Participant demographics

Fifty asthmatic children from 41 households received interventions and had adequate data capture. This was from 78 children who ever enrolled in the study, of which 58 received environmental interventions, with the remainder lost to follow-up or with missing questionnaires. All subsequent statistics are based on these 50 children. A majority of participating children were Hispanic (70%) with most of the remainder self-reporting as African-American (28%) (Table 2).

These participants had 8–17 months of health data (median = 11 months), with 3–10 months of post-intervention follow-up (median = 6 months). The distributions were bimodal, corresponding to the two intervention phases.

### Health outcomes

Reported respiratory symptoms decreased significantly between the start and end of the study. The mean respiratory symptom score was reduced from 2.6 to 1.5 ($p = 0.0002$ in Wilcoxon two-sample test), with 71% of children having either a reduced score or no reported symptoms either pre- or post-intervention. In the pre-post comparison, 95% of children at WeB exhibited symptom improvements (mean symptom score reduction from 2.7 to 0.8, $p = 0.0002$), versus 59% at FH (reduction from 2.5 to 1.6, $p = 0.03$) and 43% at WaB (no change). There was no significant difference in the degree of symptom improvement as a function of season of enrollment.

Looking at the individual questions that comprise the symptom score (Fig. 1), while 76% of children reported wheeze or tightness in the chest or cough in the 2 weeks prior to enrollment, this was reduced to 40% by the end of the study. Similar reductions were seen for slowing down or stopping play (from 64% to 26%) and for waking up at night (from 64% to 30%). There were no significant changes in severe asthma attacks or asthma hospitalizations, possibly related to our small sample size and the rarity of those events.

Conclusions were similar within our longitudinal analysis of the child's asthma-related quality of life, as reported in more detail elsewhere (Clougherty et al., 2006). We found a clinically significant total improvement of 1.32 points on average (on a 7-point scale). In addition, we found significant improvements in the months leading up to intervention (mean improvement = 0.09 points/month), an increased rate of improvement in the first 5 months post-intervention (0.16 points/month), and a slower rate of improvement in the later stage post-intervention (0.04 points/month). Results were unaffected by the exclusion of children under 6 years of age.

### Changes in risk factors

We explore various factors that might explain the reductions in respiratory symptoms, including

Table 2
Demographics of children in intervention study

| | Franklin Hill (FH) | West Broadway (WeB) | Washington Beech (WaB) | Total |
|---|---|---|---|---|
| Number of children | 22 | 21 | 7 | 50 |
| Number of households | 17 | 19 | 5 | 41 |
| *Age at enrollment* (%) | | | | |
| 4–5 | 14 | 19 | 14 | 16 |
| 6–9 | 32 | 24 | 29 | 28 |
| 10–12 | 27 | 38 | 29 | 32 |
| 13–17 | 27 | 19 | 29 | 24 |
| *Race/ethnicity* (%) | | | | |
| Hispanic | 55 | 81 | 86 | 70 |
| African-American | 45 | 14 | 14 | 28 |
| Caucasian | 0 | 5 | 0 | 2 |

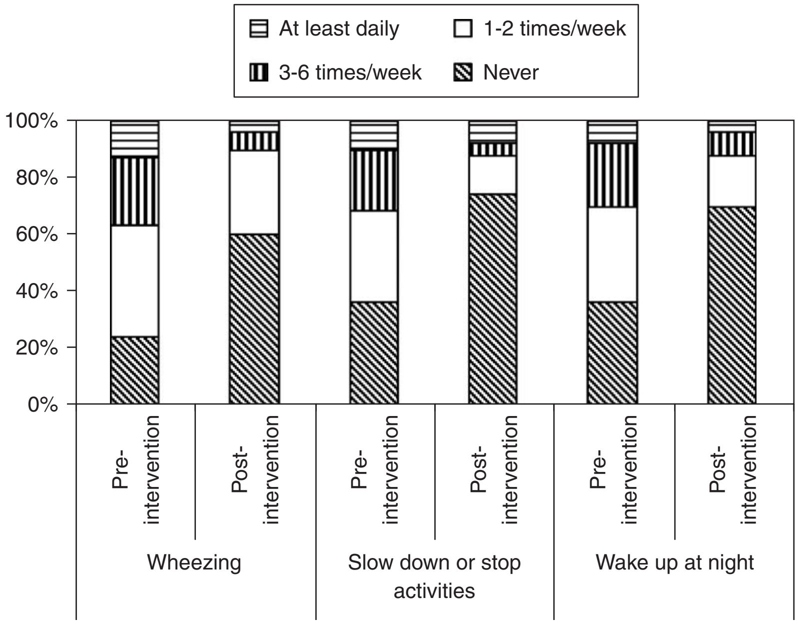

Fig. 1. Respiratory symptoms in the 2 weeks prior to enrollment versus the 2 weeks prior to the end of the study.

environmental, medical, and social factors. We initially consider time trends in these factors and then construct exploratory regression models.

First considering environmental measures, cockroach allergen concentrations were reduced significantly by IPM activities, although median concentrations increased after about six months post-intervention while remaining below baseline levels (Table 3). The percentage of homes with reductions in other allergens varied by allergen and sampling location/medium from 37 to 65%, with the highest percentages other than for cockroach seen for dust mites and cat allergen in the bed (Table 3).

Allergy testing indicated that 77% of children had allergies to one of the allergens evaluated, with 58% allergic to cockroaches and 60% to dust mites (47% to both and 72% to either). At the start of the study, 46% of children were reported by their caregivers to have allergies, with only 6% reported to have allergies to cockroaches, dust mites, or other indoor allergens. By the end of the study, these figures were increased to 69% and 40%, respectively, indicating that information from the allergy testing and other aspects of our intervention had influenced knowledge about allergies.

Looking at asthma management and health care, there was little change during the study. There were no statistically significant increases in having a doctor to call other than the emergency room for asthma care (from 70% to 74%, $p = 0.80$) or having a visit in the last year other than for an acute attack (from 77% to 86%, $p = 0.21$). The percentage of children with asthma action plans did not increase significantly (from 40% to 46%, $p = 0.67$).

One dimension of asthma management that did change significantly was the prevalence and usage of peak flow meters, which was expected given that peak flow meters were provided to all children as part of our intervention. Not only did the prevalence increase from 29% to 93%, but usage among those with a peak flow meter became more frequent (from 25% usage at least once a month to 86%), consistent with our attempts to have children record peak flow measures for asthma action plans and therapeutic purposes.

Another dimension of asthma management is adequacy of prescribed medications and corresponding perceptions of caregivers. Based on criteria in Table 1, only 14 of 44 children (32%) evaluated at the start of the study were adequately medicated. In many cases, persistent asthmatics were not prescribed any controller medications. In spite of this, caregivers perceived that the medication was adequate, with 89% of children reported by their caregivers to be receiving the right type and level of medication. Few children had significant shifts in their prescribed medications during the study, with only five children changing from improperly medicated to properly medicated by

Table 3
Median allergen concentrations before environmental interventions, at the lowest level post-intervention, and at the final post-intervention measurement

| Allergen | Site | Baseline | Low, post-intervention | Final, post-intervention | % with concentration decreases (baseline–final) |
|---|---|---|---|---|---|
| Bla g 1 | Air | 0.22 | 0.057 | 0.10 | 57 |
| (U/g) | Bed | 0.23 | 0.06 | 0.09 | 58 |
| | Kitchen | 31 | 3.0 | 14 | 61 |
| Bla g 2 | Air | 1.2 | 0.16 | 0.34 | 62 |
| (U/g) | Bed | 1.6 | 0.63 | 0.88 | 56 |
| | Kitchen | 56 | 9.6 | 35 | 65 |
| MUP | Air | 0.11 | 0.067 | 0.11 | 51 |
| (μg/g) | Bed | 0.094 | 0.085 | 0.12 | 46 |
| | Kitchen | 0.26 | 0.08 | 0.48 | 42 |
| Der f 1 | Air | 0.015 | 0.0078 | 0.014 | 43 |
| (μg/g) | Bed | 0.18 | 0.011 | 0.030 | 61 |
| Der p 1 | Air | 0.011 | 0.011 | 0.012 | 49 |
| (μg/g) | Bed | 0.010 | 0.0066 | 0.0096 | 52 |
| Fel d 1 | Air | 0.28 | 0.14 | 0.30 | 49 |
| (μg/g) | Bed | 0.48 | 0.15 | 0.34 | 62 |
| Can f 1 | Air | 0.14 | 0.10 | 0.20 | 42 |
| (μg/g) | Bed | 0.15 | 0.097 | 0.19 | 37 |
| *Alternaria* | Air | 2.9 | 2.6 | 3.5 | 49 |
| (μg/g) | Bed | 2.8 | 2.0 | 2.8 | 38 |

the end of the study. Perceptions of caregivers regarding medication adequacy did not change significantly by the end of the study. Of note, we were unable to assess the degree to which prescribed medications were used properly.

Social conditions could also explain the health improvements if they changed during the study (although our interventions did not directly target these factors). There were no significant changes in the prevalence of fear of violence or the percentage of caregivers who let their children play outside, with only five caregivers (11%) reporting reduced fear of violence. Reported violence and perceived fears were lower at WeB throughout the study (i.e., 10% of WeB caregivers would not allow their children to play outside post-intervention due to fear of violence, versus 45% at FH and 14% at WaB). There were no significant changes in mean social support or social cohesion overall or within developments, although 48% and 43% of caregivers reported improvements, respectively. There were greater reductions in the four-item Cohen Perceived Stress Scale, with 56% of caregivers improving and statistically significant reductions at WeB (from a mean of 6.6 to 4.5, $p = 0.009$).

### *Predictors of respiratory symptom improvements*

We focus our statistical analyses on the respiratory symptom score, considering a number of the environmental, medical, and social factors mentioned above (see Table 4 for a complete list), while controlling for season and time between intervention and the post-intervention questionnaire. We consider candidates for multivariate models any covariates with $p < 0.1$ in univariate regressions.

In univariate logistic regressions, improvements in the respiratory symptom score are most significantly associated with living at WeB or having cat allergies (Table 4). Reductions in Bla g 2 in the bed above the median reduction and the number of allergens in the bed to which the child was allergic with reductions above the median reduction were moderately associated ($p = 0.052$ and $0.053$) with improvements in respiratory symptoms. In multivariate models using stepwise regression on the significant terms in Table 4, only living at WeB and having cat allergies enter into the final model.

Following a similar approach but using linear regressions on the magnitude of the symptom score change, decreases in the symptom score are

Table 4
Univariate and multivariate logistic regressions of improvements in respiratory symptom scores on demographic, environmental, medical, and social covariates. Only those covariates for which $p<0.1$ in the univariate regressions are listed[a]

| | Univariate regression | | Multivariate regression | |
|---|---|---|---|---|
| | Odds ratio (95% CI) | N | Odds ratio (95% CI) | N |
| West Broadway | 10.0 (2.0, 50)** | 49 | 6.7 (1.4, 32)** | 41 |
| Cat allergy | 9.2 (1.0, 85)** | 42 | 10.1 (1.0, 99)** | |
| Bla g 2 reduction in bed >median | 4.5 (1.0, 20)* | 44 | — | |
| # of allergens in bed to which child is allergic with reductions >median | 68 (0.9, 4900)* | 44 | — | |

Additional covariates tested include Hispanic status, age, tree/grass pollen allergy, roach allergy, dust mite allergy, reductions in smoking in the home, improvements in having a doctor to call for asthma, improvements in asthma action plans, reductions in perceived stress, reductions in fear of violence, reductions in Bla g 1 or Bla g 2 below 8 U/g (in kitchen), reductions in Bla g 1 above the median reduction (in bed), reductions in Bla g 2 above the median reduction (air or bed), number of allergens reduced below exacerbation cut-points/median reductions (air or bed), and number of allergens to which the child is allergic reduced below exacerbation cut-points/median reductions (air or bed).

**$p<0.05$.

*$p<0.1$.

[a] All models controlled for season and time between intervention and post-intervention questionnaire.

significantly associated with living at WeB and having improvements in social capital or individual social support. In a multivariate model using stepwise regression, improvement in individual social support is the only term that enters into the final model, with a statistically significant ($p = 0.04$) 1.2 point decrease among those with improved social support.

## Discussion

There is a clear indication from both 2-week recall questionnaires administered at the start and end of the study and from monthly quality of life questionnaires that respiratory symptoms were reduced significantly during our intervention study. By itself, this evidence suggests the ability to improve respiratory health among pediatric asthmatics in public housing with a home-based intervention. However, this conclusion is tempered by the fact that it is difficult to determine the cause of the improvement, given that we lack a control population. In addition, we had multiple simultaneous interventions, and cannot discount the possibility that participation in the study itself led to either reported or actual improvements, independent of our interventions, or that we have observed regression toward the mean. While we cannot resolve these questions definitively, we can narrow the possibilities and inform future efforts.

Many dimensions of health care did not change during our intervention study. In spite of the work of our Community Health Nurse and CHAs, many children still relied on the emergency room rather than primary care physician, there were no gains in the prevalence of asthma action plans, and many children remained incorrectly medicated with their caregivers perceiving otherwise. No medical care covariates were significant in regression models, and all reported findings are robust to their inclusion.

This may point toward the limitations of a home-based intervention, in which we did not have direct access to the child's pediatrician, and toward the need for a more systematic and substantial effort to improve asthma management among high-risk children. Regardless of the underlying limitations of our efforts, these findings indicate that changes in medical management are not likely responsible for the observed improvements in respiratory symptoms. The one factor that may merit further attention is the usage of peak flow meters, which increased significantly over the course of our study and was positively (but not significantly) associated with symptom improvements. It seems unlikely that this factor alone could have led to such a significant reduction in the frequency of asthma exacerbation, but it is a potential contributor, as it may lead to better asthma management.

For environmental conditions, there were clear reductions in cockroach allergen levels, although patterns varied and levels started to increase after about 6 months post-intervention. There were smaller absolute and percentage reductions in levels of other allergens. In addition, the time period with the greatest reductions in cockroach allergen concentrations corresponded precisely to the time period with the greatest improvement in asthma-related quality of life.

Although we found positive associations between multiple allergen reduction terms and symptom improvements, these associations were not statistically significant at the $p<0.05$ level, and many other allergen terms were highly insignificant. This may be in part a statistical power issue (both given our sample size and the fact that most children improved or exhibited no symptoms), but multiple other factors may be influential.

For example, relatively few homes that began above 8 U/g ended up below 8 U/g, and only a subset of homes with such reductions had a child allergic to cockroaches. Of our 41 participants with allergy testing and allergen sampling data, only 5 were both allergic to cockroaches and had a reduction from above to below 8 U/g in either Bla g 1 or Bla g 2. This clearly limits our power for analyzing this subpopulation, although all of these children either had less-frequent symptoms or no change in symptoms. The story is similar if we consider those with cockroach allergies and reductions above the median reduction in kitchen concentrations of either Bla g 1 or Bla g 2. Of the 12 children who fulfill these criteria, 10 had improved symptoms or had no symptoms either pre- or post-intervention, and the other two exhibited no change. In general, there are issues in assessing allergen exposures from measurements at limited time points, as well as in developing covariates given a lack of knowledge about the shape of the allergen dose–response curve.

In spite of these limitations, there is some evidence (informed by the longitudinal models) that allergen reductions are associated with improvements in respiratory symptoms among pediatric asthmatics in this cohort. While this does not prove that IPM and intensive cleaning were the causal agents in symptom improvements, it indicates that this factor is worthy of further study, with evidence from allergen concentration patterns that the benefits of IPM were largely realized within the 5–6 months following interventions.

The prevalence of most social factors did not change significantly over the course of the study, but there was some evidence (from the linear regression models) that improvements in individual social support or social capital were associated with respiratory symptom improvements. This may indicate that the social support mechanisms related to our study had a direct or indirect health benefit. Direct health benefits were illustrated in a study of asthmatic adolescents, in which life stress and degree of control over one's health were associated with higher levels of Th-2 cytokines (IL-5) and cytokines with a Th-1 response (IFN-$\gamma$), and marginally lower morning cortisol (Chen, Fisher, Bacharier, & Strunk, 2003). Increased social support may also allow for better preventative asthma management, information sharing about asthma care, caretaking assistance, awareness of asthma triggers, and confidence in pursuing unit-level structural repairs from the housing authority.

An intriguing finding is the much greater improvement for children at West Broadway as compared with the other developments. Looking across the risk factors evaluated (Table 4), only reductions in perceived stress significantly differed ($p<0.05$) between WeB and the other two developments and could plausibly explain the symptom improvements, along with a near-significant difference in improvements in social support (62% improving at WeB vs. 38% in other developments, $p = 0.1$). Many other risk factors did not significantly differ, and children at WeB had significantly fewer gains in having a doctor to call for asthma, reductions of Bla g 1 in the kitchen above the median reduction, and allergens in the air with reductions above the median reduction.

It is impossible to infer causality for any single term in this cross-development comparison, given differences in the physical infrastructure and renovation status of the developments, the initial magnitude of pest infestation, the ways in which IPM was implemented, the social environment of the surrounding community, the nature of any resident education/interaction (given different CHAs), and numerous other dimensions. Regardless, this substantial difference between developments within a single housing authority illustrates the complexity of implementing and interpreting home-based interventions for asthma, and shows that the potential interplay of social factors is important to evaluate explicitly.

A similarly intriguing finding is the significant association with cat allergies. While this could be causal, it more likely represents a proxy for other factors—children allergic to cats were more likely to also have roach and dust mite allergies, had more substantial reductions in cockroach allergens and total allergen loading, and had caregivers who exhibited greater reductions in social stress and increases in social support.

Clearly, there are limitations in the conclusions we can draw from our analysis. Given a small sample size and numerous correlated risk factors, any findings (both significant and insignificant terms) must be interpreted with caution. In addition, our primary statistical analyses are based on a simple pre-post comparison of two points in time. Given substantial variability in asthma symptoms as a function of season and other factors, this complicates interpretation of our findings. However, the conclusions from the pre-post analysis and longitudinal analysis are similar, and our findings were not affected by controlling for season. Another limitation is the fact that our pre-post analyses treat each child as an independent observation, although multiple children were enrolled from some households. Although our sample size and design do not allow for more formal analyses of within-household correlations, we re-ran our analyses restricted to only the oldest child in each home, and our conclusions were unchanged. The longitudinal model was also run random by household, accounting for within-household correlations.

We have also based our conclusions on self-reported symptoms, which could result in reporting bias given the implicit social support dimensions of our project. It could also be argued that involvement of community members as data collectors could introduce bias (a common concern about CBPR studies). While this is theoretically possible, the complex and multi-factorial nature of our questionnaires and the fact that CHAs worked with multiple families using multiple data collection instruments makes it unlikely that systematic bias could have been introduced. More generally, self-reported symptoms have been commonly used in previous environmental intervention studies and have yielded consistent and reproducible findings. In general, our findings are broadly consistent with the literature on home interventions and asthma (Carter et al., 2001; Krieger et al., 2005; Morgan et al., 2004), which found that multifactorial interventions including cockroach allergen reductions were correlated with improvements in respiratory health, but could not assign exact proportions of the benefits to specific social/environmental intervention measures.

Our experience and findings suggest several lessons for future CBPR studies that involve environmental or housing interventions. Our CBPR design involved some tradeoffs, as we were able to gain access to a high-risk population who would have been difficult to engage without the involvement of the community, but we were unable to implement a control population. Similarly, more intensive interventions were necessary for this setting but limited our sample size given budgetary constraints. While we were able to make useful associational observations in the absence of a control group, the inclusion of a control group would clearly strengthen the causal interpretation of any intervention study. In the absence of a control group, longitudinal collection of dense data on environment, social factors, and health are advisable.

In general, much CBPR research to date has been observational or otherwise fallen short of achieving clinical trial standards, which has potentially limited the impact of these studies on public health policy and programs. This may arise partly from the complexity of managing and sustaining equitable partnerships, as well as from resistance from community or city partners to aspects of scientific methodology that may not directly benefit the community in the short term (since a more rigorous methodology may conflict with addressing immediate needs). However, CBPR is arguably the only approach that can both address research questions about urban asthma and provide the knowledge base within the community to promote local changes in policy and practice, so it is important to find approaches to resolve these issues. An additional benefit of the CBPR approach in this study is that it effectively engaged the city and helped with plans to expand IPM in Boston public housing. Open discussions at the outset among researchers and community members about the strengths and weaknesses of various study designs may best address these issues. Further refining the CBPR approach to strengthen its scientific basis while not losing sight of the benefits for the participating community adds to the challenge of such efforts, but is necessary to ensure broader and more meaningful impacts.

## Conclusion

In conclusion, our intervention study documented significant decreases in respiratory symptoms among asthmatic children in public housing. It is possible that our environmental interventions were a primary causative agent, given the increased rate of symptom improvement immediately subsequent to our environmental interventions. Given some improvements before environmental interventions and between-development differences not easily explained by allergen exposures, the benefits of our intervention may be attributable at least in part to social support effects. It is likely that being involved in our study provided an expanded network of social support and options for asthma and environmental management. Without a control population, it is impossible to rule this possibility out. In a population of asthmatic children with allergies to multiple indoor allergens, including cockroaches, IPM and intensive cleaning appear to be beneficial for respiratory health, potentially through both environmental and social pathways. Future studies should evaluate environmental interventions in the context of other medical/social factors, and explicitly assess the impact of social support on asthma outcomes.

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