NIH-PA Author Manuscript

NIH Public Access
Author Manuscript
Ann Allergy Asthma Immunol. Author manuscript; available in PMC 2010 January 28.

Published in final edited form as:
Ann Allergy Asthma Immunol. 2009 December ; 103(6): 480.

The effect of low-cost modification of the home environment on the development of respiratory symptoms in the first year of life

Victoria Persky, MD[*], **Julie Piorkowski, MPH**[*], **Eva Hernandez, RN, MSN**[*], **Noel Chavez, PhD, RD, LDN**[†], **Cynthia Wagner-Cassanova, RN, BSN**[*], **Sally Freels, PhD**[*], **Carmen Vergara, RN, BSN**[*], **Darlene Pelzel, RN, BSN**[*], **Rachel Hayes, RN, PhD**[‡], **Silvia Gutierrez**[§], **Adela Busso**[||], **Lenore Coover, RN, MSN, AE-C**[¶], **Peter S. Thorne, PhD**[#], and **Dennis Ownby, MD**[**]

[*] Division of Epidemiology and Biostatistics, The University of Illinois at Chicago School of Public Health

[†] Division of Community Health Sciences, The University of Illinois School of Public Health, Chicago

[‡] Vanderbilt University, Informatics Center, Nashville, Tennessee

[§] Nuestra Familia (Family Focus), Chicago, Illinois

[||] Chicago Commons, Chicago, Illinois

[¶] Pediatric Case Management Services, Highland, Indiana

[#] College of Public Health Environmental Health Science Research Center and Department of Occupational and Environmental Health, University of Iowa, Iowa City

[**] Department of Pediatrics, Medical College of Georgia, Augusta

Abstract

Background—Previous studies have suggested that environmental exposures may be related to the development of respiratory symptoms in early life. Intervention studies, however, have not produced consistent findings.

Objective—The Peer Education in Pregnancy Study examined the effect of home environment intervention with pregnant women at risk for having children with asthma on the development of respiratory symptoms in their infants.

Methods—A total of 383 pregnant women whose unborn child had a first-degree relative with an allergic history were randomized to 1 of 2 intervention groups, both of whom received general health education, smoking cessation advice, and encouragement to breastfeed. In addition, the intensive education group received 3 home visits focused on home environment modification. Home assessment was performed at baseline and after 1 year of follow-up. Respiratory symptoms were identified during the first year of life.

Results—Families in both intervention groups showed significant changes in several environmental factors, with significant differences between the 2 groups in insects other than cockroaches, use of mattress covers, and washing in hot water. Children in the intensive education group had slightly lower incidence rates of respiratory symptoms, but few differences were statistically significant.

Requests for reprints should be addressed to: Victoria Persky, MD, Division of Epidemiology and Biostatistics, University of Illinois at Chicago School of Public Health, 1603 Taylor St, Room 878a, Chicago, IL 60612, vwpersky@uic.edu.

Disclosures: Authors have nothing to disclose.

Publisher's Disclaimer: Disclaimer: The contents of this article are solely the responsibility of the authors and do not necessarily represent the official views of the National Institutes of Health.

Conclusions—The results of this study do not provide strong support for a primary intervention focused on general modification of the home environment during pregnancy for high-risk children. It does not address the effects of more aggressive approaches or of interventions targeting individual environmental factors.

INTRODUCTION

The prevalence of asthma increased substantially between the mid-1970s and the mid-1990s, with rates currently high throughout much of the Western world.[1,2] Morbidity and mortality rates are disproportionately greater in low-income underserved communities.[1,3] Although a variety of environmental factors are known to exacerbate asthma, it is not clear that these same variables increase the risk of developing the disease. Previous studies have suggested that in utero and early life exposure to tobacco smoke,[4–6] cockroaches,[6–8] dust mites,[9–11] mice,[12] *Penicillium* mold,[13,14] and endotoxin[15,16] may increase the risk of developing allergic symptoms. Others have suggested that early life exposures to infections from children in day care settings,[17] farm animals,[18,19] endotoxins,[20] and dogs and cats[7,21,22] may protect against the development of asthma later in life.

Primary intervention studies investigating the effects of reducing environmental allergens have been few and have mostly focused on mite reduction alone.[23,24] Few of these studies have shown important effects; the more aggressive measures of the Manchester, England, study[25] showed reduction in some, but not all, respiratory symptoms in the first year. Other combined intervention studies that included dietary allergen avoidance,[26,27] breastfeeding,[28,29] smoking,[28,29] or supplementation with omega-3 fatty acids[30] showed some reduction in allergic disease later in life. Most of the previous studies were performed in populations with high mite exposure. None were in the United States, and none studied an underserved inner-city population.

The Peer Education in Pregnancy Study was undertaken to examine the effect of community educators working with low-income pregnant women at risk of having a child with asthma on modification of factors in the home known to exacerbate the disease. This article presents the results of that intervention on the home environment and on respiratory symptoms during the first year of life.

METHODS

Population

The Peer Education in Pregnancy Study is a randomized controlled trial testing the effect of home environmental modifications during pregnancy on the development of asthma and allergic symptoms in children at risk for developing the disease. Recruitment for the study was staggered over several years (September 24, 1998, through October 4, 2004). A pregnant woman was eligible if her unborn child had a first-degree relative with asthma, eczema, or hay fever; she lived in a selected community area on the west side of Chicago; she was in the first 4 months of pregnancy; and she did not intend to move in the next year. Most of the women were recruited from 1 of the local health centers. The rest of the women were identified in a variety of places, including health care and social service agencies. A total of 483 women signed consent forms and underwent the baseline evaluation. Of those women, 383 were stratified by place of recruitment (clinic 1, clinic 2, and other) and randomized into the study. Reasons for dropouts before randomization were miscarriage (n = 24), moved out of the study area (n = 2), lived with another participant (n = 1), unable to contact (n = 49), and not interested (n = 24). Reasons for dropouts after randomization were miscarriage (n = 1), moved out of the study area (n = 1), stillbirth (n = 1), hysterical pregnancy (n = 1), newborn deceased (n = 1), and not interested (n = 6). To date, all infants have reached the age of 1 year: 11 families

NIH-PA Author Manuscript NIH-PA Author Manuscript NIH-PA Author Manuscript

NIH-PA Author Manuscript

NIH-PA Author Manuscript

NIH-PA Author Manuscript

withdrew before year 1 (2.9% of the 383 women randomized). We have observed 353 (92.2%) mother/child pairs to the age of 1 year. Six women were excluded from multivariable analyses because of missing data on critical variables, leaving 347 (90.6%) with follow-up symptom information available for this report. A total of 328 homes were evaluated at baseline and 1 year later, with comparable baseline and follow-up dust allergen measurements available for 152 of the homes. There was no difference in percentage recruited in the fall (September 1 through November 30) between the 2 intervention groups (21.8% vs 27.6%). Demographic information for women at baseline and observed for 1 year is given in Table 1. At baseline, the mean age was 25.6 years; 240 women, or 62.7%, were of Mexican heritage and, of those, 80.0% were born in Mexico. For 37.9% of the women, this was their first pregnancy.

Intervention

All women in the study received basic health education, advice for smoking cessation, general nutrition, and encouragement to breastfeed. In addition, the intensive education group received 3 home intervention visits by a community health educator focused on modification of the environment. Two of these visits were during pregnancy (at 5–6 and 7–8 months of gestation) and 1 was when the child was aged 4 months. The intervention included advice about dust control, removal of pets from the house, washing bed linens in hot water, cost-effective means of controlling water leaks, pest and rodent control through Integrated Pest Management, identification of cockroach droppings and use of gel baits, removal of carpets when feasible, use of clothes hampers, and use of wet mopping. Mothers received dust mite–impermeable mattress covers for their beds. They did not receive covers for pillows or box springs.

Data Collection

Baseline examinations in pregnancy included visual inspection of the home environment and health and lifestyle of the mother and immediate family. Follow-up inspections of the home were done 1 year after baseline. When the child was aged 4 to 6 weeks, 6 months, and 1 year, medical and lifestyle questionnaires were administered in English or Spanish depending on the woman's preference. Nurse telephone follow-ups at 3 and 9 months were also performed to obtain interim medical and symptom histories.

Home Environment Evaluation

Perceived exposure to passive smoke at home was determined from nurse questions regarding any exposure to smoke at home. A summary dichotomous variable was created from all pregnancy nurse surveys and when the newborn was aged 4 to 6 weeks to represent any perceived exposure to passive smoke at home at anytime during pregnancy.

Other home smoke and environmental variables were evaluated through the Coover tool, developed by 1 of the investigators (L.C.), which was administered by a community health educator during home inspections at baseline and 1 year later. Educators asked about the presence of household members who smoke, any smoking inside the home, cockroaches, rodents, other insects or pests, home improvement, mattress covers, washing clothes in hot water, food eaten in the mother's room, and presence of pets. Additional environmental factors were visually evaluated through the Coover tool during educator home walk-throughs. This standardized visual evaluation does not include recording of humidity, but notes presence of peeling wallpaper, paint, or plaster; holes in the walls or floors; signs of leaks in ceilings or walls; mold growth in the kitchen or baths; signs of moisture around the window ledges; live plants in the home; wall-to-wall carpeting; area rugs; air conditioning; and number of knickknacks or figurines.

Development of respiratory end points was determined by any positive response at visit 4 (child aged 4–6 weeks), visit 5 (child aged 6 months), or visit 6 (child aged 12 months) or telephone

NIH-PA Author Manuscript NIH-PA Author Manuscript NIH-PA Author Manuscript

calls at 3 and 9 months concerning any wheezing (whistling in the chest), wheezing ever disturbing sleep at night, any coughing frequently throughout the day or night, coughing ever disturbing sleep at night, treatment in the emergency department for breathing problems (coughing, congestion, runny nose, or wheezing), admission to the hospital for breathing problems, physician-diagnosed asthma, or physician-diagnosed eczema.

Potential Confounders Related to Respiratory Symptoms

Active smoking in pregnancy was determined from nurse questions when the mother was 4 to 5 months pregnant and 7 to 8 months pregnant and when the newborn was aged 4 to 6 weeks. A summary smoking variable was created to express the presence of any smoking by the mother in mid to late pregnancy (between the first enrollment and the end of pregnancy). Exposure to passive smoke in pregnancy was determined by questions at each nurse visit addressing both smoking in the home and elsewhere.

Breastfeeding was determined on the first visit after delivery (when the newborn was aged 4–6 weeks). Other potential confounders included age when formula was introduced (categorized by child's birth, <4 weeks, 4–12 weeks, and >12 weeks); family history of asthma, evaluated at baseline by questionnaire history of a first-degree relative of the unborn child having a history of asthma; antibiotic use in pregnancy, evaluated by questionnaire after 4–5 months' gestation; low birth weight, evaluated by questionnaire when the child was aged 4 to 6 weeks; child's sex; maternal age; and maternal Mexican ethnicity.

During the home environment evaluation, reservoir dust samples were collected at baseline and 1 year later by vacuuming 1 m^2 of living room floor area for 2 minutes using portable vacuums (Dirt Devil). After sampling, the vacuum bags were sealed and stored at 4°C. Dust was extracted at 50 mg/mL using sterile phosphate-buffered saline with 0.05% polysorbate 20 (Tween 20) (1 hour of shaking and 20 minutes of centrifugation at 600*g*). Cockroach, cat, and mite allergens were analyzed by enzyme immunoassay using monoclonal capture antibodies (anti–Bla g 1, anti–Fel d 1, and anti–Der p1) and labeling antibodies (rabbit anti–recombinant Bla g 1 and biotinylated anti–Fel d 1 monoclonal antibodies [mAb], and biotinylated anti–Der group 1 mAb) (Indoor Biotechnologies Inc, Charlottesville, Virginia). Bla g 1, Fel d 1, and Der p 1 standards were also from Indoor Biotechnologies Inc. The Bla g 1 assay used peroxidase-labeled goat anti–rabbit IgG (Biosource, Camarillo, California). Absorbance was read at 405 nm (SpectraMax Plus 384; Molecular Devices, Sunnyvale, California). Two different laboratories were used for these measurements. Although the methods used were comparable, standardization of values between the laboratories suggested that there were important differences. For this article, therefore, only measurements from the laboratory responsible for 74% of the results are presented.

Statistical Analysis

Statistical analyses were performed using commercially available software (SAS version 9.1; SAS Institute Inc, Cary, North Carolina). The McNemar test for paired data was used to analyze the difference between home environment exposures at baseline and at the 1-year follow-up. The McNemar *P* value tested the significance of changes for the overall sample and individually for the intensive education and nonintensive education groups. Proportional odds logistic regression models were run to provide a comparison of baseline to follow-up home exposure variables across the intervention groups using change scores (follow-up minus baseline, coded as −1, 0, and 1) as dependent variables. Odds ratios and *P* values are presented for each home exposure to show the effect of intervention on the change score. Models control for the effect of moving between baseline and follow-up. Additional analyses used a sub-sample of homes that were the same during both assessments (the participants did not move).

NIH-PA Author Manuscript

NIH-PA Author Manuscript

NIH-PA Author Manuscript

The χ^2 statistic tested the significance of relationships of potential confounders with respiratory end points. Logistic regression models were used to estimate the effect of being in the intervention group on the child's respiratory symptoms in the first year of life. Odds ratios and 95% confidence intervals are presented. Multiple logistic regression was used to obtain odds ratios adjusted for maternal age, child's sex, maternal Mexican ethnicity, child breastfed for 4 or more weeks, active smoking in mid to late pregnancy, exposure to passive smoke during pregnancy, low birth weight (<2,500 g), antibiotic use in late pregnancy, age when formula was introduced (categorized by birth, <4 weeks, 4–12 weeks, and > 12 weeks), and family history of asthma.

The study was approved by the University of Illinois at Chicago Human Subjects Institutional Review Board.

RESULTS

Women who initially consented were similar to those subsequently randomized and observed until the child was aged 1 year (Table 1). Demographic variables and potential confounders showed somewhat fewer Mexican women and slightly more women in other ethnic/race groups with a higher percentage speaking Spanish and a lower percentage speaking English and Spanish in the intensive education group. There were no other substantial differences by intervention status (Table 2).

Environmental factors were similar at baseline between the 2 intervention groups, with only holes in the wall, being overrun with cockroaches, not having a rug, and presence of knickknacks being associated with the subsequent development of more than 1 respiratory end point (data not shown). Between baseline and 1 year later, there were significant improvements in the home environment overall (Table 3). Families in the intensive education group had significant reductions in smokers living in the home, holes in the wall, leaks in the ceiling or walls, being overrun with cockroaches, having insects other than cockroaches, having furry pets, and food eaten in the mother's room. Mothers also increased the use of mattress covers and were more likely to wash linens with hot water. Families in the nonintensive education group also had significant reductions in household members or visitors smoking inside the home, presence of furry pets, window moisture, and any moisture. Differences in changes in exposure between the 2 intervention groups, however, were significant only for insects other than cockroaches, use of mattress covers, home improvement in the last year, and washing in hot water. Dust measurements in the subsample of 152 homes with measurements before and after intervention showed a trend toward decreases in levels in both intervention groups with no significant differences seen within or between groups (Table 4).

Respiratory symptoms were common in the first year of life, with 33.4% reporting some wheezing, 21.6% reporting wheezing that disturbed sleep, 65.6% reporting coughing that disturbed sleep, 34.3% going to the emergency department for a respiratory problem, 10.1% being hospitalized for a respiratory problem, and 4.6% being diagnosed as having asthma (data not shown).

Active smoking in pregnancy was rare (10.1% in early in pregnancy and 3.8% at baseline). Among Mexican women born in Mexico, it was particularly rare, with 17.6% of those born in the United States and 5.6% of those born in Mexico smoking early in pregnancy.

Most women breastfed their infants (87.9%), with 68.6% breastfeeding for 4 weeks or longer. Early use of formula was also common, with 70.0% starting at birth and another 14.7% beginning in the first month of life. Thus, most women fed their infants with a combination of breast and formula feeding (data not shown).

NIH-PA Author Manuscript NIH-PA Author Manuscript NIH-PA Author Manuscript

Potential confounders that were significantly associated with respiratory symptoms when the child was aged 1 year included the mother not being of Mexican ethnicity or the mother being exposed to smoke or antibiotics during pregnancy and the child having a first-degree relative with asthma, having a low birth weight, being breastfed for less than 4 weeks, or having formula started when younger than 3 months (data not shown).

The percentage of children in each intervention group with symptoms in the first year of life is given in Table 5. In general, those in the intensive education group had a slightly lower incidence of respiratory symptoms than those in the usual care group, although the only difference to reach significance was physician visit for respiratory symptoms after control for potential confounders.

DISCUSSION

The results of this study do not provide strong support for a primary intervention focused on low-cost general modification of the home environment in pregnancy for children at high risk of allergic disease up to the age of 1 year. These findings are consistent with the previous literature that did not find significant effects of low-level environmental interventions on incidence of respiratory symptoms.

The Prevention and Incidence of Asthma and Mite Allergy (PIAMA) study, testing the effect of allergen-impermeable mattress covers in pregnancy, found a significant difference in night cough without a cold in children at the age of 2 years,[23] with no difference in symptoms at the age of 4 years.[31] Similarly, the Study of Prevention of Allergy in Children in Europe of mite-impermeable mattress covers in 696 newborns at high risk of developing allergies found no significant differences at the age of 24 months in sensitization to house dust mites, symptoms, or allergic diseases.[24] On the other hand, more aggressive environmental manipulation (including provision of a high-filtration vacuum cleaner, removal of carpets in the infant's room, new custom-made cot, and carry-cot–encased mattresses) in the Manchester study was associated with significantly less prescribed medication for wheezing attacks, wheezing after playing or exertion, and attacks of severe wheezing with shortness of breath.[25]

Other studies that have combined manipulation of the home environment with other interventions have had more impressive results. The first of these,[26] using mattress covers, acaricide, and breastfeeding on a low-allergen diet, noted significantly less wheezing, nocturnal cough, and atopy after 8 years of follow-up. The Canadian Childhood Asthma Primary Prevention Study,[29,32] combining in utero avoidance of house dust, pets, and environmental tobacco smoke, encouragement of breastfeeding, and delayed introduction of solid foods, found significantly lower allergist-diagnosed asthma by the age of 7 years.[29] Finally, a trial of 616 children at high risk for asthma, enrolled before birth, testing house dust mite allergen reduction and/or supplementation with omega-3 fatty acids[30] noted the following results at the age of 3 years: (1) a significant 10% reduction in the prevalence of cough, but not wheezing or asthma, in atopic, but not in nonatopic, children in the active diet group; and (2) no reduction in symptoms in the dust mite reduction group.

The lack of effectiveness of the intervention on symptoms in the children through the age of 1 year in the current study is consistent with the results of previously mentioned studies. Our intervention was designed as low cost and portable because the inner-city population addressed in the project is highly mobile and lacks resources to duplicate high-cost procedures in other settings. A small percentage of our mothers smoked and most breastfed, which was encouraged for both groups of participants. Although there were some differences between the 2 groups in modification of the home environment, these differences were not substantial, with few reaching statistical significance. The previous studies[23,24,30] in high-risk populations have not

NIH-PA Author Manuscript

NIH-PA Author Manuscript

NIH-PA Author Manuscript

prevented allergic symptoms with low-cost mite prevention. It is impossible to know whether our intervention would have been more effective if more families had been able to make all the changes recommended by the peer educators. There are also many potentially important factors, such as outdoor air pollution and proximity of traffic pollution, not measured in this study that are beyond the ability of individual families to change.

An alternative explanation for our findings relates to concurrent changes in the home environment of the nonintensive education group. General health and smoking information was part of the intervention for both groups. This is supported by the decreases in overall passive smoke exposure. Our data also suggest that, although modification of the home environment was not part of the intervention for the nonintensive group, at follow-up there were modest decreases in exposure to pets and moisture in both groups, as well as trends in the percentage of homes with detectable allergens, that could bias the findings toward the null hypothesis. This explanation, if confirmed by future studies, would suggest that minimal exposure to educators and/or nurses in 1 home visit might be effective as an intervention strategy.

A third explanation relates to the hygiene hypothesis. If early exposure to animals protects against asthma, decreases in that exposure could negate protective effects of decreased exposure to passive smoke, cockroaches, and moisture. However, pet exposure at baseline and follow-up did not relate to respiratory symptoms during the first year of life and significant decreases in furry pets were seen in both intervention groups.

Limitations of the study include lack of measurements of atopy on the entire cohort, the relatively small numbers examined, the possibility that environmental changes occurred in both intervention groups, and the mobility of the population; these factors limit our ability to test specific hypotheses. In addition, the inclusion of only high-risk children precludes generalization to other populations. Follow-up to the age of 1 year does not allow for the diagnosis of asthma in sufficient numbers to allow for hypothesis testing. Strengths, however, are the high percentage observed, the uniqueness of this immigrant population, and the completeness of the data collection during pregnancy; these factors will allow exploration of other factors that relate to early development of respiratory symptoms.

In summary, this study does not provide support for the effectiveness of environmental intervention during pregnancy for the prevention of allergic symptoms in high-risk children up to the age of 1 year. Also, it does not address effects in low-risk children. Finally, it does not examine the effectiveness of more aggressive or individual interventions, such as reduction in mold or cockroach exposure. Additional term follow-up will be necessary to examine longer-term effects.

Acknowledgments

Funding Sources: This study was supported by grants R21ES08716 and R01ES011377 from National Institute of Environmental Health Sciences.

We thank Anamaria Sanchez and Alicia Contreras for their help with the data collection in the early years of the study; Lee Francis, Erie Family Health Center, for his support throughout the study; and Eduardo Anguiano, from Chicago Commons, and Amanda Caballero, Mariana Osoria, and Blanca Almonte, from Nuestra Familia, for their ongoing collaboration.

References

1. Moorman JE, Rudd RA, Johnson CA, et al. National surveillance for asthma—United States, 1980–2004. MMWR Surveill Summ 2007;56:1–54. [PubMed: 17947969]

NIH-PA Author Manuscript
NIH-PA Author Manuscript
NIH-PA Author Manuscript

2. The International Study of Asthma and Allergies in Childhood (ISAAC) Steering Committee. Worldwide variation in prevalence of symptoms of asthma, allergic rhinoconjunctivitis, and atopic eczema: ISAAC. Lancet 1998;351:1225–1232. [PubMed: 9643741]
3. Marder D, Targonski P, Orris P, Persky V, Addington W. Effect of racial and socioeconomic factors on asthma mortality in Chicago. Chest 1992;101:426S–429S. [PubMed: 1591943]
4. Lannerö E, Wickman M, Pershagen G, Nordvall L. Maternal smoking during pregnancy increases the risk of recurrent wheezing during the first years of life (BAMSE). Respir Res 2006;7:3. [PubMed: 16396689]
5. Zlotkowska R, Zejda JE. Fetal and postnatal exposure to tobacco smoke and respiratory health in children. Eur J Epidemiol 2005;20:719–727. [PubMed: 16151886]
6. Gold DR, Burge HA, Carey V, Milton DK, Platts-Mills T, Weiss ST. Predictors of repeated wheeze in the first year of life: the relative roles of cockroach, birth weight, acute lower respiratory illness, and maternal smoking. Am J Respir Crit Care Med 1999;160:227–236. [PubMed: 10390405]
7. Belanger K, Beckett W, Triche E, et al. Symptoms of wheeze and persistent cough in the first year of life: associations with indoor allergens, air contaminants, and maternal history of asthma. Am J Epidemiol 2003;158:195–202. [PubMed: 12882940]
8. Litonjua AA, Carey VJ, Burge HA, Weiss ST, Gold DR. Exposure to cockroach allergen in the home is associated with incident doctor-diagnosed asthma and recurrent wheezing. J Allergy Clin Immunol 2001;107:41–47. [PubMed: 11149989]
9. Schonberger HJ, Dompeling E, Knottnerus JA, Kuiper S, van Weel C, van Schayek CP. Prenatal exposure to mite and pet allergens and total serum IgE at birth in high-risk children. Pediatr Allergy Immunol 2005;16:27–31. [PubMed: 15693908]
10. Sporik R, Holgate ST, Platts-Mills TAE, Cogswell JJ. Exposure to house-dust mite allergen (Der p I) and the development of asthma in childhood. N Engl J Med 1990;323:502–507. [PubMed: 2377175]
11. Johnson CC, Ownby DR, Havstad SL, Peterson EL. Family history, dust mite exposure in early childhood, and risk for pediatric atopy and asthma. J Allergy Clin Immunol 2004;114:105–110. [PubMed: 15241351]
12. Phipatanakul W, Celedon JC, Sredl DL, Weiss ST, Gold DR. Mouse exposure and wheeze in the first year of life. Ann Allergy Asthma Immunol 2005;94:593–599. [PubMed: 15948302]
13. Stark PC, Burge HA, Ryan LM, Milton DK, Gold DR. Fungal levels in the home and lower respiratory tract illnesses in the first year of life. Am J Respir Crit Care Med 2003;168:232–237. [PubMed: 12724122]
14. Gent JF, Ren P, Belanger K, et al. Levels of household mold associated with respiratory symptoms in the first year of life in a cohort at risk for asthma. Environ Health Perspect 2002;101:A781–A786. [PubMed: 12460818]
15. Park JH, Gold DR, Spiegelman DL, Burge HA, Milton DK. House dust endotoxin and wheeze in the first year of life. Am J Respir Crit Care Med 2001;163:322–328. [PubMed: 11179100]
16. Perzanowski MS, Miller RL, Thorne PS, et al. Endotoxin in inner-city homes: associations with wheeze and eczema in early childhood. J Allergy Clin Immunol 2006;117:1082–1089. [PubMed: 16675336]
17. Ball TM, Castro-Rodriquez JA, Griffith KA, Holberg CJ, Martinez FD, Wright AL. Siblings, day-care attendance, and the risk of asthma and wheezing during childhood. N Engl J Med 2000;343:538–543. [PubMed: 10954761]
18. Riedler J, Braun-Fahrlander C, Eder W, et al. Exposure to farming in early life and development of asthma and allergy: a cross-sectional survey. Lancet 2001;358:1129–1133. [PubMed: 11597666]
19. Ege MJ, Bieli C, Frei R, et al. Prenatal farm exposure is related to the expression of receptors of the innate immunity and to atopic sensitization in school-age children. J Allergy Clin Immunol 2006;117:817–823. [PubMed: 16630939]
20. Douwes J, van Strien R, Doekes G, et al. Does early indoor microbial exposure reduce the risk of asthma? the Prevention and Incidence of Asthma and Mite Allergy birth cohort. J Allergy Clin Immunol 2006;117:1067–1073. [PubMed: 16675334]
21. Ownby DR, Johnson CC, Peterson EL. Exposure to dogs and cats in the first year of life and risk of allergic sensitization at 6 to 7 years of age. JAMA 2002;288:963–972. [PubMed: 12190366]

22. Remes ST, Castro-Rodrigquez JA, Holberg CJ, Martinez FD, Wright AL. Dog exposure in infancy decreases the subsequent risk of frequent wheeze but not of atopy. J Allergy Clin Immunol 2001;108:509–515. [PubMed: 11590373]
23. Koopman LP, van Strien RT, Kerkhof M, et al. Placebo-controlled trial of house dust mite-impermeable mattress covers. Am J Respir Crit Care Med 2002;166:307–313. [PubMed: 12153962]
24. Horak F Jr, Matthews S, Ihorst G, et al. Effect of mite-impermeable mattress encasings and an educational package on the development of allergies in a multinational randomized, controlled birth-cohort study: 24 months results of the Study of Prevention of Allergy in Children in Europe. Clin Exp Allergy 2004;34:1220–1225. [PubMed: 15298561]
25. Custovic A, Simpson BM, Kissen P, Woodcock A. for the NAC Manchester Asthma and Allergy Study Group. Effect of environmental manipulation in pregnancy and early life on respiratory symptoms and atopy during first year of life: a randomized trial. Lancet 2001;258:188–193. [PubMed: 11476835]
26. Arshad SH, Bateman B, Matthews SM. Primary prevention of asthma and atopy during childhood by allergen avoidance in infancy: a randomized controlled study. Thorax 2003;58:489–493. [PubMed: 12775858]
27. Arshad SH. Primary prevention of asthma and allergy. J Allergy Clin Immunol 2005;116:3–14. [PubMed: 15990764]
28. Chan-Yeung M, Manfreda J, Dimich-Ward H, Ferguson A, Watson W, Becker A. A randomized controlled study on the effectiveness of a multifaceted intervention program in the primary prevention of asthma in high-risk infants. Arch Pediatr Adolesc Med 2000;154:657–663. [PubMed: 10891016]
29. Chan-Yeung M, Ferguson A, Watson W, et al. The Canadian Childhood Asthma Primary Prevention Study: outcomes at 7 years of age. J Allergy Clin Immunol 2005;116:49–55. [PubMed: 15990772]
30. Peat JK, Mihrshahi S, Kemp AS, et al. Three-year outcomes of dietary fatty acid modification and house dust mite reduction in the Childhood Asthma Prevention Study. J Allergy Clin Immunol 2004;114:807–813. [PubMed: 15480319]
31. Corver K, Kerkhof M, Brussee JE, et al. House dust mite allergen reduction and allergy at 4 yr: follow up of the PIAMA-study. Pediatr Allergy Immunol 2006;17:329–336. [PubMed: 16846450]
32. Becker A, Watson W, Ferguson A, Dimich-Ward H, Chan-Yeung M. The Canadian asthma primary prevention study: outcomes at 2 years of age. J Allergy Clin Immunol 2004;113:650–656. [PubMed: 15100668]

NIH-PA Author Manuscript
NIH-PA Author Manuscript
NIH-PA Author Manuscript

NIH-PA Author Manuscript
NIH-PA Author Manuscript
NIH-PA Author Manuscript

Table 1

Demographic Variables by Enrollment and Follow-up[a]

Variable	Women who initially consented (n = 483)	Women who enrolled (n = 383)	Women observed to 1 y (n = 347)
Maternal age, y[b]	25.6 (14–45)	25.6 (15–43)	25.7 (15–43)
Race/ethnicity			
Mexican	60.0	62.7	65.1
Puerto Rican	16.8	15.4	14.4
Other/mixed Hispanic	10.6	11.0	11.0
African American	6.4	6.0	5.8
Other	6.2	5.0	3.8
Born in the United States	41.7	39.2	37.8
Years in the United States for those not born in the United States[b]	8.6 (0.2–39.0)	8.4 (0.2–39.0)	8.4 (0.2–39.0)
Language			
English	42.7	39.7	38.0
Spanish	46.2	48.8	50.7
Both English and Spanish	11.2	11.5	11.2
Education completed			
< High school	42.0	41.2	42.1
High school graduate	32.9	33.9	34.0
Some college	20.4	20.1	18.7
College graduate	4.8	4.7	5.2
Works outside the home	32.6	31.3	30.0
Married	44.0	46.5	47.3
No. of previous live births			
0	38.5	37.9	37.8
1	26.2	27.2	27.1
2	21.9	22.4	23.1
≥3	13.4	12.8	12.1
Health care site			
1	56.9	57.4	57.6
2	25.3	26.9	27.7
3	17.8	15.7	14.7

[a]Data are given as percentage of patients in each group unless otherwise indicated. Percentages may not total 100 because of rounding.

[b]Data are given as mean (range).

NIH-PA Author Manuscript
NIH-PA Author Manuscript
NIH-PA Author Manuscript

Table 2

Demographic Variables by Intervention Group[a]

Variable	Intensive education group (n = 192)	Nonintensive education group (n = 191)
Maternal age, y[b]	25.5 (15–43)	25.7 (15–40)
Race		
Mexican	57.3	68.1
Puerto Rican	16.7	14.1
Other/mixed Hispanic	12.0	10.0
African American	7.3	4.7
Other	6.8	3.1[c]
Born in the United States	38.5	39.8
Years in the United States for those not born in the United States[b]	7.7 (0.2–23.0)	9.0 (0.2–39.0)
Language		
English	40.1	39.3
Spanish	52.6	45.0
Both English and Spanish	7.3	15.7[c]
Education completed		
<High school	41.2	41.4
High school graduate	33.9	34.0
Some college graduate	22.4	17.8
College graduate	2.6	6.8
Works outside the home	28.7	34.0
Married	49.0	44.0
No. of previous live births		
0	35.9	39.8
1	30.2	24.1
2	22.9	22.0
≥3	10.9	14.1
Health care site		
1	57.3	57.6
2	26.0	27.8
3	16.7	14.7

[a]Data are given as percentage of each group unless otherwise indicated. Percentages may not total 100 because of rounding.

[b]Data are given as mean (range).

[c]$P < .05$ by χ^2 test.

NIH-PA Author Manuscript NIH-PA Author Manuscript NIH-PA Author Manuscript

Table 3

Housing Variables at Baseline and 1-Year Follow-up for the Intensive Education and Nonintensive Education Groups

Variable from home environment[a]	Overall (n = 328)		Intensive education group (n = 165)[b]		Nonintensive education group (n = 163)[b]		P value for comparison of baseline with follow-up across intervention groups[c]
	Baseline	Follow-up	Baseline	Follow-up	Baseline	Follow-up	
Smokers live in the home	36.8	30.4[d]	40.0	29.7[d]	33.4	31.1	.10
Any smoking inside the home by those who live there or by a visitor	25.3	19.0[d]	24.8	22.2	25.6	16.0[d]	.19
Mother smoking[e]	4.1	5.4	4.4	4.4	3.8	6.3	.29
Mother perceived ≥5 h of exposure at home[f]	11.1	6.7[d]	12.4	6.8	9.8	6.5	.56
Peeling wallpaper or paint	24.1	21.0	24.4	23.2	23.8	18.8	.46
Holes in the wall	19.4	12.5[d]	20.9	11.7[d]	18.0	13.5	.38
Leaks in the ceiling or walls	12.7	7.3[d]	14.9	5.0[d]	10.4	9.7	.05
Mold in the kitchen/bath	29.3	30.8	32.1	28.4	26.3	33.3	.10
Window moisture	17.8	11.5[d]	16.7	11.1	19.1	11.8[d]	.77
Any moisture	19.8	14.2[d]	17.1	14.6	22.6	13.8[d]	.24
Any leaks or moisture	35.4	27.7[d]	37.0	27.9	33.8	27.5	.64
Any mold/leaks or moisture	53.4	46.5[d]	55.2	46.7	51.9	46.3	.62
Live plants	49.1	49.4	43.3	43.3	55.0	55.6	.92
Furry pets	25.0	17.1[d]	24.2	14.6[d]	25.8	19.6[d]	.43
Any cockroaches	29.0	25.9	29.5	25.8	28.6	26.1	.82
Cockroaches, many/overrun	6.5	4.6	8.6	3.7[d]	4.4	5.6	.07
Rodents	18.3	16.1	19.8	14.2	16.8	18.0	.18
Other insects or pests	22.1	18.1	24.1	14.2[d]	20.1	22.0	.049
Wall-to-wall carpet	50.5	50.5	50.1	54.6	50.0	46.3	.18
Area rugs	42.9	36.8[d]	42.1	34.2	43.8	39.5	.58
Home improvement in the past year	32.2	30.7	25.9	31.5	38.5	29.8	.04
Many knickknacks	20.6	16.2	16.6	14.1	24.7	18.4	.43
No mattress cover on mother's bed	89.4	78.2[d]	87.0	68.3[d]	91.9	88.1	.003

NIH-PA Author Manuscript
NIH-PA Author Manuscript
NIH-PA Author Manuscript

Variable from home environment[a]	Overall (n = 328)		Intensive education group (n = 165)[b]		Nonintensive education group (n = 163)[b]		*P* value for comparison of baseline with follow-up across intervention groups[c]
	Baseline	Follow-up	Baseline	Follow-up	Baseline	Follow-up	
Not washing in hot water	62.3	47.6[d]	58.6	35.0[d]	66.0	60.3	.006
Food eaten in mother's room	34.6	29.2	37.7	27.3[d]	31.7	31.0	.13
Air conditioning	56.1	57.7	50.6	55.5	61.7	59.9	.29

[a]From the Coover tool, administered by a community health educator at baseline and 1-year follow-up, except for mother smoking and perceived 5 or more hours of exposure at home.

[b]Data are given as percentage of participants in each group.

[c]Comparisons of baseline with follow-up across intervention groups were examined using proportional odds logistic regression models with change scores coded as −1, 0, or 1 (calculated for each exposure and with control for whether family moved between baseline and follow-up).

[d]$P < .05$ for the difference between baseline and follow-up, using the McNemar test for paired data.

[e]From survey questions administered by a nurse at first nurse visit and when the child was 1-year of age.

[f]From survey questions administered by a nurse at first and third nurse visits.

NIH-PA Author Manuscript

Table 4

Living Room Dust Allergen Levels at Baseline and 1-Year Follow-up for the Intensive Education and Nonintensive Education Groups[a]

Variable	Overall (n = 152)		Intensive education group (n = 74)[a]		Nonintensive education group (n = 78)[a]		*P* value for comparison of baseline with follow-up across intervention groups[b]
	Baseline	Follow-up	Baseline	Follow-up	Baseline	Follow-up	
Detectable mite Der p	20.3	16.9	22.2	15.3	18.4	18.4	.40
Detectable cat allergen Fel d 1	61.2	53.3	62.2	56.8	60.3	50.0	.63
Detectable cockroach Bla g 1	47.4	42.1	50.0	47.3	44.9	37.2	.64

[a] Data are given as percentage of each group unless otherwise indicated.

[b] Comparisons of baseline with follow-up across intervention groups were examined using proportional odds logistic regression models with change scores coded as −1, 0, or 1 (calculated for each exposure and with control for whether family moved between baseline and follow-up).

NIH-PA Author Manuscript NIH-PA Author Manuscript NIH-PA Author Manuscript

Table 5

Associations of Respiratory and Allergic Symptoms With 347 Intervention Group Participants

Variable	Intensive education group, %	Nonintensive education group, %	Odds ratio (95% confidence interval)	
			Unadjusted	Adjusted[a]
Any respiratory symptom	90.8	93.1	0.73 (0.33–1.59)	0.69 (0.30–1.58)
Any congestion	87.3	88.4	0.90 (0.47–1.71)	0.98 (0.49–1.94)
Any wheezing	31.2	35.6	0.82 (0.52–1.28)	0.86 (0.53–1.40)
Sleep-disturbed wheezing	19.7	23.6	0.79 (0.48–1.33)	0.79 (0.46–1.37)
Any cough	76.3	83.3	0.64 (0.38–1.10)	0.65 (0.37–1.15)
Sleep-disturbed coughing	63.0	68.2	0.79 (0.51–1.24)	0.82 (0.50–1.32)
Coughing for ≥7 d	45.9	48.0	0.92 (0.60–1.41)	0.98 (0.62–1.54)
Emergency department visit for respiratory symptoms	33.5	35.1	0.93 (0.60–1.46)	0.94 (0.58–1.51)
Hospitalization for respiratory symptoms	8.7	11.5	0.73 (0.36–1.48)	0.78 (0.37–1.65)
Physician visit for respiratory symptoms	63.4	74.7[b]	0.59 (0.37–0.93)	0.60 (0.37–0.98)
Mother had to change plans	35.8	34.5	1.06 (0.68–1.65)	0.99 (0.62–1.60)
Asthma diagnosis	3.5	6.3	0.53 (0.19–1.47)	0.45 (0.15–1.33)
Eczema diagnosis	10.4	8.6	1.23 (0.60–2.53)	1.15 (0.54–2.45)

[a]The odds ratio and confidence interval from logistic regression after control for maternal age, child's sex, maternal Mexican ethnicity, child breastfed for at least 4 weeks, active smoking in mid/late pregnancy, exposure to passive smoke during pregnancy, low birth weight (<2,500 g), antibiotic use in late pregnancy, age when formula was introduced (categorized by birth, <4 weeks, 4–12 weeks, and >12 weeks), and family history of asthma.

[b]$P < .05$ by χ^2 test.