Indoor Air Pollution and Childhood Asthma: Effective Environmental Interventions

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Exposure to indoor air pollutants such as tobacco smoke and dust mites may exacerbate childhood asthma. Environmental interventions to reduce exposures to these pollutants can help prevent exacerbations of the disease. Among the most important interventions is the elimination of environmental tobacco smoke from the environments of children with asthma. However, the effectiveness of reducing asthmatic children's exposure to environmental tobacco smoke on the severity of their symptoms has not yet been systematically evaluated. Dust mite reduction is another helpful environmental intervention. This can be achieved by enclosing the child's mattresses, blankets, and pillows in zippered polyurethane-coated casings. Primary prevention of asthma is not as well understood. It is anticipated that efforts to reduce smoking during pregnancy could reduce the incidence of asthma in children. European studies have suggested that reducing exposure to food and house dust mite antigens during lactation and for the first 12 months of life diminishes the development of allergic disorders in infants with high total IgE in the cord blood and a family history of atopy. Many children with asthma and their families are not receiving adequate counseling about environmental interventions from health care providers or other sources. — Environ Health Perspect 103(Suppl 6):55–58 (1995)

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Introduction

The environment in which a child lives may contribute to a child's risk of having ongoing exacerbations of asthma. There is also some evidence that the environment contributes to a child's risk of developing asthma. Much of the early evidence that there was a significant environmental component to asthma came from studies of twins. Edfors-Lubs studied 6996 twin pairs in the Swedish twin registry and found that when one monozygotic twin had asthma, the other twin had it 19% of the time (*1*). She concluded that the remaining 81% were due to environmental factors. Among dizygotic twins, if one twin had asthma, the other twin had it only 4.8% of the time. Thus, she reasoned, although there are certainly genetic factors involved, there is clearly an important environmental component to developing asthma.

Asthma prevalence is higher among African–American children than among white children (*2,3*). Furthermore, African–American children have higher rates of hospitalization for asthma, but much of this increase is thought to be due to poverty rather than to race (*4,5*). It is not known whether children who live in poverty are more heavily exposed to indoor air pollutants than children who do not live in poverty.

In the United States, children spend most of their time (average, 20 hr) indoors (*6*). Therefore, in addition to considering the effects of outdoor air pollution (*7*), it is important to take into account the effects that exposure to indoor air pollutants may have on childhood asthma. The dominant allergens associated with asthma are found indoors, i.e., mite, cat, dog, and cockroach (*8,9*). Some have hypothesized that the increased prevalence of asthma among children may be a manifestation of an increase in children's sensitization to inhaled allergens (*10,11*). Higher concentrations of tobacco smoke and pesticides are also found indoors (*12,13*). This article reviews primary and secondary prevention of asthma, focusing on interventions for reducing tobacco smoke and indoor allergens, and provides some information about the possible role of pesticides.

Prevention of Asthma

To date, most of the asthma interventions have been designed to prevent asthma exacerbations (known as secondary prevention); few efforts to prevent the development of asthma (known as primary prevention) have been undertaken. Because it is the more common approach, secondary prevention will be discussed first, followed by primary prevention.

Secondary Prevention: Passive Smoking

Secondary prevention includes efforts to prevent asthma exacerbations, such as household interventions to eliminate cigarette smoke from the child's environment. Children with asthma who have a parent who smokes have more frequent exacerbations and more severe symptoms (*14–27*). Table 1 summarizes recent epidemiologic studies of the effects of passive smoking on asthma in childhood. Despite the importance of exposure to environmental tobacco smoke for asthmatic children, the effectiveness of reducing children's exposure to environmental tobacco smoke has not been systematically evaluated. In the only intervention among asthmatic children reported in the literature, Murray and Morrison reported that if parents expose their asthmatic children to less cigarette smoke, their asthmatic symptoms will be less severe (*21*). However, this study's conclusions are limited by the investigators' inability to measure exposure to environmental tobacco smoke and the subjective assessment of severity of symptoms. Additional studies of the effectiveness of reducing asthmatic children's exposure to environmental tobacco smoke are needed.

Secondary Prevention: Dust Mite Elimination

Secondary prevention also includes household interventions to eliminate dust mites. Among the most effective measures to decrease mite infestation are plastic mattress covers. Ehnert and her colleagues performed

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Table 1. Recent epidemiologic studies of effects of passive smoking on asthma in childhood.[a]

Study	Population studied	ETS exposure assessment	Outcome variable	Results[b]	Observations
Burchfiel et al. (*14*)	3482 nonsmoking children 0 to 19 years of age, in Tecumseh, MI	Questionnaire answered by subjects or parents	Prevalence of asthma	OR = 1.7 (1.2, 2.5) for boys; OR = 1.2 (0.8, 1.9) for girls	Independent of parental respiratory illness, age, parental education, family size, and allergies
Evans et al. (*17*)	191 children 4 to 17 years of age in New York, NY	Parental questionnaire	Emergency room visits and hospitalizations for asthma (from medical records)	3.1±0.4 vs 1.8±0.3 (p=0.008)	No distinction made between maternal and paternal smoking; independent of race and parental employment status
O'Connor et al. (*22*)	292 subjects 6 to 21 years of age in Boston, MA	Parental questionnaire	Bronchial response to cold air	Significantly increased response in asthmatics whose mothers smoked	No increase in nonasthmatics whose mothers smoked
Murray and Morrison (*19*)	415 children 1 to 17 years of age with asthma in Vancouver, Canada	Parental questionnaire	Asthma symptom score for severity of asthma	Higher scores (p<0.01) in children of smoking mothers	Stronger effect in boys and older children
Krzyzanowski et al. (*18*)	298 children 5 to 15 years of age in Tucson, AZ	Parental questionnaire	Parental reports of asthma in their children	OR = 9.0 (2.4, 34.0) for children exposed to ETS and formaldehyde vs. nonexposed	Small sample
Sherman et al. (*24*)	770 children 5 to 9 years of age followed for 11 years in Boston, MA	Parental and subject questionnaire	Physician diagnosis of asthma	No effect of parental smoking on prevalence or incidence of asthma	No effort to assess effect of heavy smoking by parents; no control for socioeconomic status
Weitzman et al. (*25*)	4331 children 0 to 5 years of age (U.S. National Health Interview Survey)	Maternal questionnaire	Asthma for at least 3 months at time of questionnaire	OR = 2.1 (1.3, 3.3) for children whose mothers smoked 10 cigarettes/day	Independent of race, sex, family size, presence of both parents, and number of rooms
Oldigs et al. (*23*)	11 asthmatic children	Direct exposure to ETS for 1 hr	Changes in lung function	No effect	No assessment of effect of chronic exposure
Martinez et al. (*26*)	774 children 0 to 5 years of age followed for several years in Tucson, AZ	Parental questionnaire	Physician diagnosis of asthma	OR = 2.5 (1.4, 4.6) for children of low maternal education whose mothers smoked 10 cigarettes/day	No effect among children of better educated mothers
Ehrlich et al. (*16*)	228 children; 72 with acute asthma; 35 with nonacute asthma and 121 controls	Cotinine levels in urine of children; smoking by maternal caregiver	Emergency room and asthma clinic visits	Higher levels of cotinine in asthmatics OR = 1.9 (1.0, 3.4)	Similar cotinine levels in acute and nonacute asthmatics
Chilmonczyk et al. (*15*)	199 children 8 months to 13 years of age and with asthma in Portland, ME	Cotinine levels in urine of children	Number of acute exacerbations of asthma in the in the previous year	RR = 1.7 (1.4, 2.1)	Similar results using parental reports of smoking

Abbreviations: ETS, environmental tobacco smoke; OR, odds ratio; RR, relative risk. [a]Adapted from U.S. Environmental Protection Agency report (*26*). [b]95% confidence intervals in parentheses.

a randomized controlled trial of two dust-mite elimination procedures in the bedrooms of 24 children with asthma (*28*). In group one, mattresses, blankets, and pillows were enclosed in polyurethane-coated casings and bedroom carpets were treated with tannic acid at the start of the study and at months 4 and 8. In group two, mattresses and carpets were treated with the acaricide, benzyl benzoate (BB) at the start of the study and at months 4 and 8. A third group received treatment of mattresses and carpets with placebo foam at the start of the study and at months 4 and 8. There was a significant decrease in concentrations of mite allergen on mattresses covered by polyurethane casings, while there was no significant reduction of mite allergen on mattresses treated with BB or placebo foam. In addition, the children whose mattresses were enclosed in polyurethane casings showed a significant reduction of bronchial hyperreactivity after 8 months. Unfortunately, this study did not test the effectiveness of polyurethane casings alone, but in combination with other measures.

Murray and Ferguson did a controlled trial among 20 asthmatic children with positive skin-prick tests to either house dust or *Dermatophagoides farinae* (*29*). The experimental group was given zippered vinyl covers for pillows, mattresses, and box springs and instructed to launder the curtains and wash blankets and mattress pads every 2 weeks. The control group was not instructed to make any changes in the child's bedroom. After 1 month, children in the experimental group had fewer days on which wheezing was observed, medication was administered, or an abnormally low PEFR was recorded. In the experimental group, the bronchial responsiveness to inhaled histamine decreased 4-fold, while it increased 2-fold in those in the control group.

Comprehensive reviews of environmental controls in the management of lung disease have been published by Samet (*30*) and Ingram and Heymann (*31*).

Primary Prevention

Primary prevention of asthma is the prevention of the development of asthma in young children. *In utero* exposure to tobacco-smoke products has been shown to be an important determinant of wheezing in the first year of life (*20*). Thus, primary prevention of asthma should include efforts to reduce smoking during pregnancy. No studies to date have investigated whether asthma incidence can be reduced by decreasing smoking during pregnancy.

Swedish investigators have studied the usefulness of screening cord blood for IgE at birth. They report that 75% of infants with a high cord blood IgE will develop atopic diseases by 1 year of age and 82% of newborn infants with high cord blood IgE will develop atopic symptoms before 7 years of age (*32,33*). Under the assumptions that preventive measures can delay the onset of atopic allergies by an average of 8 months and reduce existing treatment costs within the 6-year period by about 10% (and assuming total patient compliance) then IgE screening of all newborn infants or infants with a family history of atopic disease in Sweden is cost effective (*34*). They found that using family history of atopic disease alone as a screening test is not cost effective. The investigators estimated that an IgE screening program in Sweden would result in a total cost savings for atopic allergies before 6 years of age of approximately 20 million Swedish crowns or 3 million U.S. dollars per year. Because these assumptions and results may be different in the United States, similar studies should be undertaken in this country.

Hide and his colleagues did a randomized controlled trial of infants with a family history of atopy and high cord total IgE. In the experimental group, lactating mothers avoided milk, eggs, fish, and nuts; and infants avoided soya, wheat, and orange for the first 12 months of life (*35*). All infants in the experimental group slept on polyvinyl-covered mattresses, and acaricide foam and powder were applied to the infants' bedroom carpet, living room carpet, and upholstered furniture every three months. At age 12 months, 4 of 58 infants in the experimental group had signs of asthma compared to 12 of 62 infants in the control group. At age 2 years, 4 of 43 infants in the experimental group had asthma, compared with 9 of 41 infants in the control group (*36*). The authors concluded that reducing the exposure to food and house-dust mite antigens diminished the development of allergic disorders in high-risk infants in the first 2 years of life. It would be useful to test each of these interventions separately to find out which has a more important effect.

Future Research Needs: Pesticides and Asthma

There is currently no evidence of a link between indoor exposure to pesticides and exacerbations of childhood asthma. Evidence of a link between pesticides and asthma is largely limited to anecdotal reports of asthma among adults. Bryant (*36*) described two patients in whom asthma was reportedly precipitated by exposure to synthetic organophosphates. Occupational asthma has been reported in a worker producing the fungicide, captafol (*37*); in a woman cleaner working with a carpet fungicide, tributyl tin oxide (*38*); in a farmer using the fungicide tetrachloroisophthalonitrile in his plastic greenhouse (*39*); and among farm workers spraying crops with organophosphate pesticides containing carbamates and phosphorodithioates (*40*).

A cross-sectional study was conducted in Saskatchewan, Canada, to investigate the association of self-reported asthma and pesticide use in 1939 crop farmers (*41*). Each farmer participating in the study completed a questionnaire about history of employment; respiratory health; working conditions; cigarette smoking; and use of pesticides, herbicides, and fertilizers. The prevalence of asthma was associated with the use of carbamate insecticides (OR = 1.8; 95% CI: 1.1 to 3.1, $p = 0.02$).

Shim and Williams (*42*) reported that 51 of 60 adult patients with asthma claimed that exposure to pesticides worsened their asthma. The most frequently mentioned source of the insecticide exposure was fumigation of the house or apartment or roach sprays. One limitation of this study is that the histories of patients' exposures were not verified.

Newton and Breslin studied seven patients with histories of worsening asthma due to exposure to a widely used aerosol insecticide spray. Under controlled conditions, they were exposed to concentrations up to 6.7 mg/l, and lung functions were measured after the exposure. Chest tightness was reported by all of them after being exposed to the insecticide, but objective evidence of airway obstruction was present in only one, who showed a greater than 20% fall in FEV_1, compared to his baseline value; two showed a very small decline in the maximum midexpiratory flow rate (*43*).

Does indoor exposure to pesticides cause exacerbation of asthma among children? Although there is little objective evidence, this exposure may deserve further investigation.

Summary

Exposure to indoor air pollutants such as tobacco smoke and dust mites may exacerbate asthma, and effective measures to prevent exposures to these pollutants are available. However, it appears that many children and their families, particularly those who rely on emergency rooms as their primary source of care, are not receiving adequate counseling from health care providers or other sources. Improvement of the health status of children with asthma requires a public health approach that emphasizes disease prevention rather than the traditional medical approach. Success in improving the health status of children with asthma will not be possible without addressing the predominant social, cultural, and environmental conditions in which children live.

REFERENCES

1. Edfors-Lubs M-L. Allergy in 7000 twin pairs. Acta Allergol 26:249–285 (1971).
2. Gergen PJ, Mullally DI, Evans R. National survey of prevalence of asthma among children in the United States, 1976 to 1980. Pediatrics 81:1–7 (1988).
3. Weitzman M, Gortmaker SL, Sobol AM, Perrin JM. Recent trends in the prevalence and severity of childhood asthma. JAMA 268:2673–2677 (1992).
4. Gergen PJ, Weiss KB. Changing patterns of asthma hospitalization among children: 1979 to 1987. JAMA 264:1688–1692 (1990).
5. Wissow LS, Gittelsohn AM, Szklo M, Starfield B, Mussman M. Poverty, race, and hospitalization for childhood asthma. Am J Public Health 78:777–782 (1988).
6. Schwab M, McDermott A, Spengler JD. Using longitudinal data to understand children's activity patterns in an exposure

context: data from the Kanawha County Health Study. Environ Health Perspect 18:173–189 (1992).
7. Bates DV. The effects of air pollution on children. Environ Health Perspect 103(Suppl 6):49–53(1995).
8. Platts-Mills TAE. Allergen-specific treatment for asthma: 3. Am Rev Respir Dis 148:553–555 (1993).
9. Pope AM, Patterson R, Burge H (eds). Indoor Allergens: Assessing and Controlling Adverse Health Effects. Washington:National Academy Press, 1993.
10. Cullinan P, Newman Taylor AJ. Asthma in children: environmental factors. Br Med J 308:1585–6 (1994).
11. Peat JK, van den Berg RH, Green WF, Mellis CM, Leeder SR, Woolcock AJ. Changing prevalence of asthma in Australian children. Br Med J 308:1591–6 (1994).
12. DHHS. The health consequences of involuntary smoking. A report of the Surgeon General. DHHS Publ No (CDC) 87-8398. Washington:U.S. Department of Health and Human Services, 1987.
13. U.S. EPA. Nonoccupational pesticide exposure study (NOPES). Publ No 600/3-90/003. Research Triangle Park, NC:U.S. Environmental Protection Agency, 1990.
14. Burchfiel CM, Higgins MW, Keller JB, Howatt WF, Butler WJ, Higgins IT. Passive smoking in childhood: respiratory conditions and pulmonary function in Tecumseh, Michigan. Am Rev Respir Dis 133:966–973 (1986).
15. Chilmonczyk BA, Salmun LM, Megathlin KN, Neveux LM, Palomaki GE, Knight GJ, Pulkkinen AJ, Haddow JE. Association between exposure to environmental tobacco smoke and exacerbations of asthma in children. N Engl J Med 328:1665–9 (1993).
16. Ehrlich R, Kattan M, Godbold J, Saltzberg DS, Grimm KT, Landrigan PJ, Lilienfield DE. Childhood asthma and passive smoking. Urinary cotinine as a biomarker of exposure. Am Rev Respir Dis 145:594–599 (1992).
17. Evans D, Levison J, Feldman CH, Clark NM, Wasilewski Y, Levin B, Melins RB. The impact of passive smoking on emergency room visits of urban children with asthma. Am Rev Respir Dis 135:567–572 (1987).
18. Krzyzanowski M, Quackenboss JJ, Lebowitz MD. Chronic respiratory effects of indoor formaldehyde exposure. Environ Res 52:117–125 (1990).
19. Murray AB, Morrison BJ. Passive smoking by asthmatics: its greater effect on boys than on girls and on older than on younger children. Pediatrics 84:451–459 (1989).
20. Tager IB, Hanrahan JP, Tosteson TD, Castile RG, Brown RW, Weiss ST, Speizer FE. Lung function, pre- and post-natal smoke exposure, and wheezing in the first year of life. Am Rev Respir Dis 147:811–817 (1993).
21. Murray AB, Morrison BJ. The decrease in severity of asthma in children of parents who smoke since the parents have been exposing them to less cigarette smoke. J Allergy Clin Immunol 91:102–110 (1993).
22. O'Connor GT, Weiss ST, Tager IB, Speizer FE. The effect of passive smoking on pulmonary function and non-specific bronchial responsiveness in a population based sample of children and young adults. Am Rev Respir Dis 135:800–804 (1987).
23. Oldigs M, Jorres R, Magnussen H. Acute effects of passive smoking on lung function and airway responsiveness in asthmatic children. Pediatr Pulmonol 10:123–131 (1991).
24. Sherman CB, Tosteson TD, Tager IB, Speizer FE, Weiss ST. Early childhood predictors of asthma. Am J Epidemiol 132:83–95 (1990).
25. Weitzman M, Gortmaker S, Klein Walker D, Sobol A. Maternal smoking and childhood asthma. Pediatrics 85:505–511 (1990).
26. Martinez FD, Cline M, Burrows B. Increased incidence of asthma in children of smoking mothers. Pediatrics 89:21–26 (1992).
27. U.S. EPA. Respiratory health effects of passive smoking: lung cancer and other disorders. Publ No 600/6-90/006F. Washington:U.S. Environmental Protection Agency, 1992.
28. Ehnert B, Lau-Schadendorf S, Weber A, Buettner P, Schou C, Wahn U. Reducing domestic exposure to dust mite allergen reduces bronchial hyperreactivity in sensitive children with asthma. J Allergy Clin Immunol 90:135–138 (1992).
29. Murray AB, Ferguson AC. Dust-free bedrooms in the treatment of asthmatic children with house dust or house dust mite allergy: a controlled trial. Pediatrics 71:418–422 (1983).
30. Samet J. Environmental controls and lung disease. Report of the American Thoracic Society Workshop on Environmental Controls and Lung Disease, Santa Fe, NM, March 24–25, 1988. Am Rev Respir Dis 142:915–939 (1990).
31. Ingram JM, Heymann PW. Environmental controls in the management of asthma. Immunol Allergy Clin N Am 13:785–801 (1993).
32. Kjellman N-I. Predictive value of high IgE levels in children. Acta Paediatr Scand 65:465–471 (1976).
33. Hjalte K, Croner S, Max Kjellman N-I. Cost-effectiveness of neonatal IgE-screening for atopic allergy before 7 years of age. Allergy 42:97–103 (1987).
34. Arshad SH, Matthews S, Gant C, Hide DW. Effect of allergen avoidance on development of allergic disorders in infancy. Lancet 339:1493–1497 (1992).
35. Hide DW, Matthews S, Gant C, Arshad SH. Effect of allergen avoidance in infancy on allergic manifestations at age two years. J Allergy Clin Immunol 91:249 (1993).
36. Bryant DH. Asthma due to insecticide sensitivity. Aust NZ J Med 15:66–8 (1985).
37. Royce S, Wald P, Sheppard D, Balmes J. Occupational asthma in a pesticides manufacturing worker. Chest 103:295–6 (1993).
38. Shelton D, Urch B, Tarlo SM. Occupational asthma induced by a carpet fungicide—tributyl tin oxide. J Allergy Clin Immunol 90:274–5 (1992).
39. Honda I, Kohrogi H, Ando M, Araki S, Ueno T, Fatatsuka M, Ueda A. Occupational asthma induced by the fungicide tetrachloroisophthalonitrile. Thorax 47:760–1 (1992).
40. Underner M, Cazenave F, Patte F. Occupational asthma in the rural environment. Rev Pneumol Clin 43:26–35 (1987).
41. Senthilselvan A, McDuffie HH, Dosman JA. Association of asthma with use of pesticides. Result of a cross-sectional survey of farmers. Am Rev Respir Disease 146:884–7 (1992).
42. Shim C, Williams MH Jr. Effect of odors in asthma. Am J Med 80:18–22 (1986).
43. Newton JG, Breslin AB. Asthmatic reactions to a commonly used aerosol insect killer. Med J Aust 1:378–80 (1983).