critical developmental pathways of the lung predisposing these children to wheezing and asthma. ETS also appears to be important in childhood wheezing (probably asthma), but less significant compared with in utero exposure.

The main limitation of this study results from the use of questionnaires and reliance on the retrospective recall of parents for data collection. Exposure to tobacco smoking was assessed through questionnaire responses and not validated by more objective measurements such as cotinine levels. The study lacks information on the actual duration and intensity of exposure to tobacco smoke, because children and parents may have altered their time-activity pattern to avoid exposure to tobacco smoke. The study also lacks information on confounding factors such as maternal nutritional status and alcohol or other toxic substance intake during pregnancy. Although one can also argue that questionnaire data collections are prone to errors in reporting, given the stigma associated with smoking, parents are more likely to underreport and bias the data toward the null hypothesis.

**ENVIRONMENTAL TOBACCO SMOKE EXPOSURE DURING CHILDHOOD IS ASSOCIATED WITH INCREASED PREVALENCE OF ASTHMA IN ADULTS**


**Purpose of the Study.** This study was conducted to determine if environmental tobacco smoke (ETS) exposure during childhood had an impact on the prevalence of asthma in adults.

**Study Population.** A random sample of 8008 residents aged 15 to 69 of Orebro, Sweden (age matched from a total population of 80 569).

**Methods.** A questionnaire was sent to the randomly selected study group during the winter from 1995–1996. It included questions about respiratory symptoms and disease, use of asthma drugs, symptoms with various exposures, and questions about smoking history, occupation, and family history of respiratory diseases. They were also asked about childhood ETS exposure with the question, “Do or did any of your parents/relatives smoke at home when you grew up?” Current and former smokers were excluded from the evaluation of effects of childhood ETS exposure on asthma because of the confounding effects of active smoking on respiratory disease.

**Results.** The questionnaire return rate was 84%. The total sample included 3556 never-smokers (52.8%), 1676 smokers (24.9%) and 1257 ex-smokers (18.7%); 243 did not respond to the question. Subjects 30 to 49 years old were most likely to have childhood ETS exposure and those that had ETS exposure were significantly more likely to be ever-smokers (54.5%) versus those without childhood ETS exposure (33.8%). In subjects with childhood ETS exposure, almost all airway-related symptoms were more prevalent compared with nonexposed subjects. Subjects with physician-diagnosed asthma were significantly more likely to have had childhood ETS exposure (7.6% asthma prevalence) versus the nonexposed group (5.8% asthma prevalence). The difference in asthma prevalence between ETS exposed and nonexposed subjects was most pronounced in the younger age group (age: 15–39), 8.8% prevalence for exposed versus 6.3% nonexposed. Subjects aged 15 to 19 with ETS exposure were more likely to be smokers themselves (36.9%) versus nonexposed (13.1% smokers). ETS exposure was a significant risk factor for physician-diagnosed asthma (odds ratio [OR]: 1.82), breathing difficulties with exercise (OR: 1.45), breathing difficulties from cigarette smoke (OR: 1.25), and breathing difficulties from pets (OR: 1.41).

**Conclusions.** Childhood ETS exposure increases the likelihood of asthma in adulthood. The risk of an individual to take up smoking is increased by 60% if they had childhood ETS exposure.

**Reviewer’s Comments.** Childhood ETS exposure was strongly associated with asthma in nonsmoking adults. This was especially true of nonsmoking adults without a family history of asthma. The authors note the limitations of this study in that details of the ETS exposure (duration and amount) were not obtained and possible bias issues that asthmatics may be more likely to report childhood ETS exposure than nonasthmatics. This article adds to the existing literature demonstrating the detrimental effects of childhood ETS exposure. It should again encourage us as child advocates to continue to address this important issue with our patients and their families.

**RESIDENTIAL EXPOSURES ASSOCIATED WITH ASTHMA IN US CHILDREN**


**Objective.** To determine the risks associated with residential exposures for childhood asthma.

**Study Population.** Participants included 8257 children <6 years old who were enrolled in the Third National Health and Nutrition Examination Survey. This was a survey of the health and nutritional status of children and adults in the United States.

**Methods.** The study was a cross-sectional survey that was conducted from 1988 to 1994. The main outcome measure was doctor-diagnosed asthma, as reported by the parents.

**Results.** Six percent of children in the survey were reported by their parents to have doctor-diagnosed asthma. The prevalence of asthma was higher among boys (6.7%) than girls (5.1%) and among black children (8.9%) compared with white children (5.2%). Risk factors for doctor-diagnosed asthma included a family history of atopy (odds ratio [OR]: 2.2), child’s history of allergy to a pet (OR: 24.2), exposure to environmental tobacco smoke (OR: 1.8), use of gas stove or oven for heat (OR: 1.8), and the presence of a dog in the household (OR: 1.6). The population attributable risk of ≥1 residential exposure for doctor-diagnosed asthma in US children ≤6 years old was 39.2%, or an estimated 533 000 excess cases, while a positive family history of atopy accounted for 300 000. The attributable cost of asthma as a result of these residential exposures for children ≤6 years old was $402 million annually.

**Conclusion.** The elimination these identified residential risk factors could result in a 39% decline in doctor-diagnosed asthma in US children ≤6 years old.

**Reviewer’s Comments.** There has been a dramatic increase in asthma prevalence in the past 20 to 30 years. Although many factors certainly contributed to this increase, this study suggests that specific exposures in the homes of our patients may play a major role. Please note also that the presence of a dog in this study has a deleterious effects, contrary to the finding in the next study. The answer to the question of indoor pets is still far from clear,
although this study and others clearly show that evidence of pet sensitivity are major risk factors for the diagnosis of asthma.

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THE PATTERN OF ATOPIC SENSITIZATION IS ASSOCIATED WITH THE DEVELOPMENT OF ASTHMA IN CHILDHOOD


Purpose of the Study. Asthma eventually develops in only one third of atopic children. The aim of this study was to prospectively investigate the pattern of atopic sensitization typically associated with the development of asthma in childhood.

Study Population. A cohort of 1314 children followed from birth to the age of 7 years in the German Multicenter Allergy Study.

Methods. Parental questionnaires on asthma and asthmatic symptoms were completed 6 times up to the age of 2 years and from then on yearly. Determination of specific immunoglobulin E to 9 food and inhalant allergens was performed yearly, and at the age of 7 years, a bronchial histamine challenge was conducted.

Results. Onset of atopic sensitization in children with current asthma at the age of 7 years was significantly earlier than in children without current asthma (39.4% before age 1 year vs 21.0%; P = .015). Early atopic sensitization without any sensitization to inhalant allergens at the age of 7 years conferred no increased risk for asthma at this age. Only those children sensitized to inhalant allergens by the age of 7 years were at a significantly increased risk of being asthmatic at this age (odds ratio 10.12; 95% confidence interval [CI]: 3.81–26.88). However, even in this group of persistently sensitized children, the risk of being asthmatic at the age of 7 years was only increased if a positive parental history of asthma or atopy was present (OR: 15.56; 95% CI: 5.78–41.83), with the effect being strongest for maternal asthma.

Conclusion. The results indicate that an underlying factor pertaining to asthma and maternal transmission may determine both a certain pattern of sensitization and the expression of asthma.

Reviewers’ Comments. This study challenges our current understanding of the natural history of childhood asthma. The notion of a progressive atopic march assumes that early atopic sensitization to food allergens is a risk factor for subsequent inhalant sensitization, which, in turn, is regarded as a risk factor for the development of asthma. However, recent epidemiologic studies showed no consistent protective effect of reduced food or inhalant allergens, as well as no positive effect of increased inhalant allergen levels on the development of asthma. This study supports the hypothesis that the development of childhood asthma and atopy run in parallel if certain perinatal or hereditary influences prevail, rather than being subsequent stepping stones in a progressive atopic march.

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PREDICTORS OF ASTHMA 3 YEARS AFTER HOSPITAL ADMISSION FOR WHEEZING IN INFANCY


Purpose of the Study. To evaluate whether early antiinflammatory therapy after a wheezing episode in infancy can prevent the development of asthma.

Study Population. A total of 89 infants <2 years old without a history of prematurity birth or chronic cardiorespiratory disease who had been hospitalized for wheezing.

Methods. This was a randomized, controlled study in which wheezing infants were divided into 3 treatment groups: cromolyn sodium 20 mg nebulized qid for 8 weeks followed by 20 mg nebulized tid for 8 weeks; budesonide 500 μg nebulized bid and 250 μg nebulized bid for 8 week successive periods; or no therapy. If clinically indicated, maintenance therapy was begun after the initial 16 weeks of antiinflammatory therapy. The presence of virus was assayed in nasal lavage specimens using standard techniques. The children were followed for 3 years, and at the conclusion of the study, asthma (total of 3 episodes of physician-diagnosed wheezing) was assessed, and skin prick tests (SPTs) were performed.

Results. Administration of antiinflammatory medication for 4 months after the initial wheezing episode had no significant effect on the development of asthma. SPT reactivity to indoor allergens, particularly cat or dog epithelial danders, was predictive of developing asthma. Conversely, a decreased risk of asthma was seen both in patients with a furred pet at home during infancy and in patients in whom the original wheezing episode was caused by respiratory syncytial virus (RSV) infection.

Conclusions. Early antiinflammatory therapy for 4 months after bronchiolitis does not prevent the development of asthma. In this study, the presence of RSV bronchiolitis was associated with a decreased incidence of asthma. Although the authors comment that the prospective design is a strength of the study, they note that the study was not blinded. The focus only on hospitalized children with wheezing also limits the scope to episodes of severe wheezing.

Reviewers’ Comments. Although this study demonstrates that early antiinflammatory therapy does not prevent later development of asthma, it does not answer the question of how such therapy might modulate airway remodeling or severity of asthma. In addition, the findings of this study are limited by the fact that their definition of asthma (3 wheezing episodes by age 3 years) includes both “transient” and “persistent” wheezers. The incidence of asthma after RSV bronchiolitis was high (22%); however, infants who wheezed without RSV infection were at greatest risk (61%) of developing asthma by age 3 years. Additional studies are needed to confirm whether RSV-negative wheezing in infancy is in fact a major risk factor for asthma.

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EARLY CHILDHOOD INFECTIOUS DISEASES AND THE DEVELOPMENT OF ASTHMA UP TO SCHOOL AGE: A BIRTH COHORT STUDY


Purpose of the Study. To investigate the association between early childhood infections and subsequent development of asthma.

Study Population. A total of 1314 children born in 1990 followed from birth to the age of 7 years.

Methods. A total of 499 newborn infants were recruited with risk factors for atopy (elevated cord blood immuno-
Residential Exposures Associated with Asthma in US Children
Christopher Randolph
*Pediatrics* 2002;110:446

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