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.

**Reviewer’s 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 premature birth or chronic cardiopulmonary 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. APT 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.

**Reviewer’s 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-
The Pattern of Atopic Sensitization is Associated with the Development of Asthma in Childhood
Anna Nowak-Wegrzyn

Pediatrics 2002;110:447

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Pediatrics 2002;110;447

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