Results. Analysis performed on the larger, unverified study population showed that subjects with asthma were more likely to be female and younger and less likely to engage in exercise at least 3 times per week. When BMI was examined, enrollees with asthma were more likely to have BMIs 25.0 to 29.9 kg/m² than enrollees without asthma (odds ratio [OR]: 1.2; 95% confidence interval [CI]: 1.1–1.4). Enrollees with asthma were also more likely to be obese (BMI ≥30 kg/m²). These findings held after adjustment for age and sex and when the analysis was performed on the verified sample. The OR for asthma increased with increasing BMI in both the larger study population and the verified sample. These findings remained in the final multivariate regression model for the larger study population and the verified sample, with a maximal asthma risk with BMI between 35 to 39.9 kg/m² in the verified sample (OR: 3.8; 95% CI: 2.0–7.2).

Conclusion. BMI >25.0 is associated with asthma and increasing BMI is associated with increasing odds of asthma.

Reviewers’ Comments. This large study confirms findings of previously published smaller studies and suggests that obesity is a risk factor for the general adult population. Although selection bias may be a weakness of this study, its strength lies in the large study population. Whether obesity plays a causal role in the development of asthma or vice versa remains unclear.

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IMPACT OF LOW BIRTH WEIGHT ON EARLY CHILDHOOD ASTHMA IN THE UNITED STATES


Purpose of the Study. To study the contribution of low birth weight to the prevalence of asthma in children under 4 years old in the United States.

Study Population. A total of 8071 children on whom data was collected in the 1988 National Maternal-Infant Health Survey (NMIHS) and the 1991 Longitudinal Follow-up Survey. The NMIHS collected data from the primary caretakers of a set of children born in the United States in 1988, and then follow-up information was collected in their third year of life. African American and low birth weight infants were oversampled, increasing their representation in the database. Data were weighted to be nationally representative.

Methods. The primary endpoint was whether a health care provider had ever told the primary caretaker that the patient had asthma. Data on birth weight, sex, race, maternal age, maternal education and socioeconomic status, maternal smoking, and poverty were also collected. Birth weight was stratified to very low birth weight (VLBW) (<1500 g), moderately low birth weight (1500–2499 g), and not low birth weight (LBW). Data were then analyzed to determine relative contributions of birth weight and other factors to development of asthma.

Results. The prevalence of asthma was higher at lower birth weights: 6.7% in children weighing ≥2500 g at birth, 10.9% in children weighing 1500 to 2499 g at birth, and 21.9% in children weighing <1500 g at birth. Birth weight was independently associated with prevalence of asthma, as was African American race. Although LBW and VLBW infants had similar risks of developing asthma regardless of race, the prevalence of VLBW was tripled in African Americans.

Conclusions. These data identify a strong association between LBW and asthma. A total of 4000 excess asthma cases were attributable to LBW. The substantially increased prevalence of VLBW in the African American community may contribute to the higher prevalence of asthma in this community.

Reviewers’ Comments. The major weakness of this study is that it relies on retrospective data reported by the primary caretaker. The only measure of asthma used was the answer to a single question on whether the child had ever been diagnosed with asthma by a health care professional. This measure may simultaneously miss some patients in whom the caretaker failed to recall the diagnosis of asthma and overcount some patients who do not actually have asthma. Nonetheless, the data is compelling and supportive of other studies. These results provide another reason for improving prenatal care to prevent LBW and VLBW births and also suggest we should be targeting the LBW and VLBW children for asthma screening and early intervention with asthma therapies.

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DETECTION OF IgA AND IgG BUT NOT IgE ANTIBODY TO RESPIRATORY SYNCYTIAL VIRUS IN NASAL WASHES AND SERA FROM INFANTS WITH WHEEZING


Purpose of the Study. The role of respiratory syncytial virus (RSV) in stimulating an immunoglobulin E (IgE) antibody response and enhancing the development of asthma remains controversial. The aim of this study was to measure IgE, immunoglobulin A (IgA), and immunoglobulin G (IgG) antibody responses to immunodominant RSV antigens in nasal washes and serum samples from infants with and without respiratory symptoms.

Study Population. Forty infants aged 6 weeks to 2 years (20 with wheezing, 9 with rhinitis, and 11 without respiratory tract symptoms) were included in the investigation.

Methods. The children were enrolled in an emergency department during the mid-winter months and seen again at follow-up when they were asymptomatic. Nasal washes were obtained by standard methods and were evaluated for RSV antigen. Moreover, determination of antibody isotypes (IgE, IgA, and IgG) to RSV antigens was performed in nasal washes and serum samples by using an enzyme-linked immunosorbent assay. In a subset of nasal washes, IgE to RSV was also evaluated by using a monoclonal anti-FcE antibody-based assay.

Results. At enrollment, 15 patients with wheezing, 2 with rhinitis, and 1 control subject tested positive for RSV antigen. Thirteen patients with wheezing were <6 months old, and most (77%) were experiencing their first attack. Among the children with positive test results for RSV antigen, an increase in both nasal wash and serum IgA antibody to RSV-Fa and Ga was observed at the follow-up visit. There was no evidence for an IgE antibody response to either antigen.

Conclusions. Both IgA and IgG antibodies to the immunodominant RSV-Fa and Ga antigens were readily detected in the nasal washes and serum samples from patients in this study. The investigators were unable to demonstrate specific IgE antibody to these antigens and concluded that the production of IgE as a manifestation of a Th2 lymphocyte response to RSV is unlikely.

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AIRWAY EOSINOPHILIA IS ASSOCIATED WITH WHEEZE BUT IS UNCOMMON IN CHILDREN WITH PERSISTENT COUGH AND FREQUENT CHEST Colds


Purpose of the Study. To evaluate sputum eosinophils in children with wheeze versus cough versus chest colds.

Study Population. A total of 83 children 8 to 11 years old recruited from primary schools in Australia based on a questionnaire regarding respiratory symptoms. Four groups: wheezed more than twice in last 12 months with or without colds (n = 28), night cough (but no wheeze) for at least 2 weeks in the last 12 months without colds (n = 12), chest colds (cough, but no wheeze) more than twice in last 12 months (n = 27) and control (no cough or wheeze) (n = 26).


Results. Median sputum eosinophil percentages were higher for children with wheeze (3.1%) than for children with cough (0.5%), chest colds (0%) or controls (0%) (P = .03). The percentage in each group with “eosinophilic bronchitis” (defined as sputum eosinophilia >2.5%) was 45%, 20%, 15% and 9.4%, respectively.

Conclusion. The authors conclude that “wheeze is a good discriminator for the presence of eosinophilic bronchitis, and that persistent cough and recurrent chest colds without wheeze should not be considered a variant of asthma.”

Reviewer’s Comments. It is interesting to note the greater likelihood of finding sputum eosinophilia with wheeze as opposed to cough, however, I think it would be a mistake to dismiss the idea that chronic or recurrent cough, with or without viral respiratory infections, can be the sole manifestation of asthma. Sputum eosinophilia on a single induced sputum sample would not seem to be a gold standard for the diagnosis of asthma as fewer than half of those with recurrent wheeze had “eosinophilic bronchitis” and 15% to 20% of those with “variant asthma” did. Although other causes of chronic cough, such as tobacco exposure should be evaluated as the authors suggest, I believe a trial of asthma medication is often warranted in such children.

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EFFECTS OF MATERNAL SMOKING DURING PREGNANCY AND ENVIRONMENTAL TOBACCO SMOKE ON ASTHMA AND WHEEZING IN CHILDREN


Purpose of the Study. Asthma is becoming more prevalent in the industrialized regions of the world. The rapid rise in childhood asthma suggests an environmental etiology. In this study, the authors evaluate the effects of maternal smoking during pregnancy and childhood environmental tobacco smoking (ETS) exposure on asthma and wheezing in school-aged children in southern California.

Methods. Self-administered questionnaires were sent to parents of 4th, 7th, and 10th grade students in 12 southern California communities. The responses to 5762 of these questionnaires completed by parents were used to ascertain children with wheezing or physician-diagnosed asthma and to gather information on lifetime household exposures to tobacco smoke and history of maternal smoking during pregnancy. Logistic regression models were fitted to cross-sectional data to estimate the effect of in utero exposure to maternal smoking and previous and current household exposure to tobacco smoke on the prevalence of wheezing and physician-diagnosed asthma.

Results. In utero exposure to any maternal smoking without subsequent postnatal environmental tobacco smoking exposure was associated with increased prevalence of physician-diagnosed asthma (odds ratio [OR]: 1.8), asthma with current symptoms (OR: 2.3), asthma requiring medication use in the previous 12 months (OR: 2.1), lifetime history of wheezing (OR: 1.6), current wheezing with colds (OR: 2.1) and without colds (OR: 2.5), persistent wheezing (OR: 3.1), wheezing with exercise (OR: 2.4), attacks of wheezing causing shortness of breath (OR: 2.4) or awakening at night in the previous 12 months (OR: 3.2), and wheezing requiring medication (OR: 2.1), or emergency room visit(s) during previous year (OR: 3.4).

Current and previous ETS exposure was not associated with asthma prevalence, but was associated with subcategories of wheezing. Current ETS exposure was associated with lifetime wheezing (OR: 1.3), current wheezing with colds (OR: 1.6) and without colds (OR: 1.5), wheezing with exercise (OR: 1.7), attacks of wheezing causing shortness of breath (OR: 1.6) or awakening at night (OR: 1.5), and wheezing requiring medication (OR: 1.4), or emergency room visit(s) in the previous year (OR: 1.9). The effect of current ETS exposure on subcategories of wheezing were most pronounced among children exposed to 2 or more smokers.

Conclusions. Authors conclude that in utero exposure to smoking increases the risk of physician-diagnosed asthma and wheezing during childhood. However, current ETS exposure is associated with wheezing, but not physician-diagnosed asthma. The authors further hypothesized that ETS acts as a cofactor with other insults such as recurrent infections as a trigger of wheezing attacks, rather than as a factor that induces asthma.

Reviewers’ Comments. This was a large study involving 5672 school-aged children in 12 different communities in southern California. The most striking finding in this study is the association between in utero exposure to tobacco smoke alone and childhood physician-diagnosed asthma. This is consistent with growing evidence that in utero exposure to tobacco smoke increases the risk of abnormal lung function at birth and in childhood, bronchial hyperactivity, wheezing, and asthma in childhood. The results suggest that in utero exposure to tobacco smoke may alter...
Detection of IgA and IgG but Not IgE Antibody to Respiratory Syncytial Virus in Nasal Washes and Sera from Infants with Wheezing

John M. James

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