chal hyperreactivity (BHR) as measured by methacholine inhalation challenge in infants with recurrent wheezing.

**Study Population.** Seventy-two children, 6 to 31 months old (median: 15 months) were studied. All of the children had previously suffered from recurrent wheezing defined as >3 separate wheezing episodes in the past 12 months. Excluded were infants born prematurely or small for gestational age or known cardiopulmonary abnormalities. All patients with atopic dermatitis were excluded. None of the children were treated with inhaled corticosteroids or cromolyn for 4 weeks before the tests were performed. Bronchodilator treatment was stopped >12 hours before testing.

**Methods.** All children were sedated with chloral hydrate before lung function testing. Compliance of the respiratory system (CRS) and resistance of the respiratory system (RRS) were measured from the passive tidal expiratory flow volume curve obtained by standard single breath method before and after methacholine challenge. Only children with normal baseline flow volume loops, CRS and RRS underwent methacholine provocation. Methacoline was inhaled during tidal breathing using a face mask with a leak-free seal. Starting dose consisted of 5 inhalations equivalent to 50 μg methacholine. Thereafter, the doses were increased by doubling the number of inhalations every 2 minutes until a positive reaction occurred defined as an increase of RRS by at least 50% or maximum dose of 900 μg methacholine was inhaled. Venous blood samples for ECP were performed after lung function testing. The subjects were divided into 3 groups based on serum ECP levels—low (<10 μg/L), medium (10–20 μg/L) and high (>20 μg/L).

**Results.** Provocative methacholine concentrations at which an increase of RRS of at least 50% was measured, ranged from 30 to 976 μg. There was no significant correlation between serum ECP levels and BHR as measured by methacholine reactivity. Also, in all patients analyzed, there was no apparent relationship of BHR or high level of ECP with the levels of total or specific immunoglobulin E (IgE), peripheral blood eosinophils, family history of atopic disease, or exposure to cigarette smoking.

**Conclusions.** No significant correlation between serum ECP levels and bronchial reactivity was found in 72 children under 31 months of age with recurrent wheezing. These parameters may reflect independent pathogenic mechanisms in the etiology of childhood asthma.

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**FREQUENCY, SEVERITY, AND DURATION OF RHINOVIRUS INFECTIONS IN ASTHMATIC AND NONASTHMATIC INDIVIDUALS: A LONGITUDINAL COHORT STUDY**


**Purpose of Study.** Rhinovirus infections cause a significant proportion of asthma exacerbations. The aim of this study was to determine if people with asthma are more susceptible to rhinovirus infections than people without asthma.

**Study Population.** The study participants were 76 cohabiting and nonsmoking couples, 1 who had asthma and at least 1 positive skin test, and 1 who was neither atopic nor asthmatic.

**Methods.** From September to December, each subject recorded peak expiratory flow twice daily and categorized any upper and lower respiratory tract symptoms as mild (1), moderate (2) or severe (3) once daily. Nasal aspirates were obtained every 2 weeks for rhinovirus reverse transcriptase-polymerase chain reaction (RT-PCR); each member of a couple had nasal aspirates collected within 24 hours of the other. Clinical illness was defined as a symptom score above the individual’s median score for at least 3 days, preceded and followed by symptom scores below the median score. Any illnesses beginning within 7 days before or 2 days after rhinovirus isolation was classified as being associated with that infection.

**Results.** After adjusting for the use of inhaled corticosteroids and sex, the 2 groups did not differ significantly with respect to risk of rhinovirus infection (odds ratio [OR]: 1.15; 95% confidence interval [CI]: 0.71–1.87) or severity (P = 0.38) or duration (P = 0.66) of upper respiratory tract symptoms. With their first rhinovirus infection, subjects with asthma had more lower respiratory tract symptoms (P = 0.051), more severe (P = 0.001) and longer (P = 0.005) duration of lower respiratory tract symptoms, and greater mean fall in peak flow (P = 0.03) than subjects without asthma. Risk of infection was slightly but not significantly higher in asthmatics using continuous inhaled corticosteroids (OR: 1.15; 95% CI: 0.60–2.19). Sex was not associated with susceptibility to rhinovirus infection (OR: 0.87; 95% CI: 0.53–1.42).

**Conclusions.** People with atopic asthma are not at greater risk of rhinovirus infection than those without atopy and asthma, but have more frequent, more severe, and longer lasting lower respiratory tract symptoms with rhinovirus infections.

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**OBESITY IS A RISK FACTOR FOR DYSPNEA BUT NOT FOR AIRFLOW OBSTRUCTION**

**Sin DD, Jones RL, Man SF.** *Arch Intern Med.* 2002;162: 1477–1481

**Purpose of the Study.** Previous research suggests that obesity is an important risk factor for asthma. However, because obesity can cause dyspnea through mechanisms other than airflow obstruction, diagnostic misclassification of asthma could partially account for this association. The purpose of the study was to determine if there is a relationship between obesity and airflow obstruction.

**Study Population and Methods.** A total of 16 171 participants (17 years or older) from the Third National Health and Nutrition Examination Survey (NHANES III) were divided into 5 quintiles based on their body mass index (BMI) to determine the association between BMI quintile and risk of self-reported asthma, bronchodilator use, exercise performance, and airflow obstruction. Significant airflow obstruction was defined as a ratio <80% the predicted
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