Early-life risk factors and allergic rhinitis: Comparing European and US data

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Allergic rhinitis has been shown to affect 3% to 19% of subjects in various epidemiologic studies of different populations. Eighty percent of those with allergic rhinitis experience symptoms by the age of 20 years, and 40% have symptoms by the age of 6 years. Allergic rhinitis is a substantial burden on both adult and pediatric patients, not only because of the bothersome physical symptoms but also because of the emotional and social effects. It is also closely linked with the development of asthma. Investigation into early-childhood factors influencing the development of allergic rhinitis raises the possibility of altering its natural history and decreasing this significant negative effect.

In this issue of the Journal, Matheson et al present data from the European Community Respiratory Health Study II (ECRHS II) regarding the association between the incidence of rhinitis and early-life exposures. The most striking findings of this study were that a number of early-life exposures seem to be protective against rhinitis. The incidence of rhinitis decreased with increased number of siblings, bedroom sharing with older children before the age of 5 years, exposure to pets before the age of 5 years, and farm upbringing, regardless of atopy status. Risk factors for rhinitis included maternal smoking in pregnancy and childhood and, as one would expect, a parental history of allergic disease. Focusing on atopic subjects only, with respect to the incidence in childhood, adolescence, and adult life, the authors found that having any siblings was associated with a reduced incidence of rhinitis. Pet ownership in the first 5 years of life reduced the incidence of rhinitis in adolescence but not adulthood. Farm upbringing was also associated with a reduced incidence of rhinitis in adolescence. The authors created a combined early-life variable, which included siblings, pets, and farm upbringing, and found that each factor had an additive protective effect. Maternal smoking during pregnancy and childhood was again a risk factor for rhinitis in atopic subjects and remained so in all stages of life.

Comparing these ECRHS II data with US data on early exposures and rhinitis reveals striking parallels but also some divergence. The large size of the ECRHS II cohort, which spans many countries, ethnic backgrounds, and socioeconomic levels, is a major strength of this study. Such large studies regarding rhinitis in the US do not exist, which must be taken into account for any data comparison.

In the US, as regards number of siblings, a study of African American and Dominican children living in low-income neighborhoods in New York City found no association between birth order and allergic rhinitis prevalence from birth to age 3 years. The authors of this study postulated that there might be other environmental or social factors masking a birth-order effect. They also postulated that follow-up until only the age of 3 years might not have been enough time for a birth-order effect to manifest. A follow-up study examined the association between birth order, atopy, and respiratory symptoms in 4-year-old children from inner-city New York City and did not find a significant decrease in the prevalence of rhinitis among children determined to be atopic by means of serology with or without siblings. Certainly there are differences between these 2 studies and the study by Matheson et al: the cohorts are much smaller, and the data examine prevalence and not incidence. Despite these differences, the contrast in results raises questions about what factors might differ in the
inner-city US populations compared with the European populations. These questions deserve more study.

US data regarding pet exposure and allergic rhinitis are limited, and therefore drawing firm conclusions from data comparisons is difficult. A study of the epidemiology of physician-diagnosed allergic rhinitis in childhood from the Tucson Children's Respiratory Study, which had a study population of 747 children, concluded that the presence of dogs was associated with allergic rhinitis by age 6 years. The presence of dogs remained a significant risk factor for atopic children when compared with nonatopic children in their multivariate analysis. This study is also limited by a much smaller cohort size and follow up duration than the ECRHS study.

The US Childhood Allergy Study looked, in part, at allergic sensitization and dog and cat exposure in the first year of life in a prospective birth cohort study of 474 healthy, full-term infants in Detroit, Michigan, to a mean age of 6.7 years. Although this study did not directly address the prevalence of allergic rhinitis, it found that exposure to 2 or more dogs or cats in the first year of life might reduce the subsequent risk of allergic sensitization to multiple allergens in childhood. An implication of these findings is that this decreased risk of sensitization would result in a decreased risk of allergic rhinitis in the population.

Unlike the ECRHS II and other European data, US data examining the relationship between rhinitis and a farm upbringing are limited. The mixed data on pet exposure suggests that there are other environmental factors influencing the development of rhinitis or that the study discrepancies are a function of study size and design.

In terms of risk factors for rhinitis, the ECRHS II data on smoking in pregnancy and childhood agree with the US data from the Cincinnati Childhood Allergen and Air Pollution Study. This study of 633 infants found that exposure to more than 20 cigarettes daily was associated with an increased risk of allergic rhinitis at age 1 year and rhinitis symptoms during the first year of life. There are numerous reasons to avoid smoke exposure in pregnancy and childhood, and preventing rhinitis should be counted among them.

The comparison between the data on early-childhood exposure and the development of allergic rhinitis from the ECRHS II study with the US data shows areas of concurrence and areas of divergence. The protective effect against rhinitis by an increasing number of siblings shown in the ECRHS II study is convincing given the large sample size and follow-up period. It is worthy to consider whether the same finding would be proved in larger US studies with a more diverse patient population. A similar statement can also be made about the comparison of the ECRHS II and US pet exposure data. Further study is needed, particularly in the US, to determine whether discrepancies are due to population differences, environmental differences, or study differences. That smoking is a risk factor for rhinitis seems to be a clearer area of agreement between the European and US data. Additional convincing and larger datasets, whether concordant or discordant, would open up promising avenues for mechanistic study. This would, in turn, provide potential new targets for altering the natural history of allergic rhinitis, with the hope of decreasing its considerable burden.

References


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