Methods. A 21-item questionnaire, which assessed food allergy awareness, avoidance measures, and treatment strategies, was mailed to the 273 schools. Multiple-choice questions were derived from suggested school guidelines for anaphylaxis.

Results. A total of 104 responses were received representing 109 schools (40% response rate). A total of 39% characterized their school district as urban, 37% as rural, and 28% as suburban. Based on a school-reported estimate of 66,958 children, there was a 1.7% self-reported prevalence rate of food allergies. A total of 95 schools reported having at least 1 food-allergic student and 55% of those reported 10 or more food-allergic children. The most common food allergies were milk (81%), peanut (62%), tree nuts (32%), shellfish (28%), egg (23%), wheat (22%), and soy (7%). A total of 31 schools reported “other” food allergens including fruit, chocolate, red dye, tomato, fish, orange juice, spices, and cheese. Food-allergic children were identified primarily through official school records, and only 16% of school had written individual emergency plans. For education on food allergies, schools relied mainly on parents (52%) and in-services (47%) conducted most commonly by school nurses or principals. Avoidance measures to aid in preventing accidental ingestions included food substitution and special meal requests, non-sharing food policies, and instruction for food handlers on techniques to prevent cross-contamination. However, only 21% of schools reported instructions on reading food labels for hidden allergens. In the event of a serious allergic reaction or on administration of epinephrine, 94% of the schools reported that they would transport the student to medical facilities. The most common site for storage of epinephrine was the main office or the nurse’s office. Principals, nurses, and teachers were most often trained to administer epinephrine. No training of staff was reported by 10% of the schools.

Conclusions. Schools need to formally educate their personnel on a school-wide basis. Important prevention measures such as reading labels, written treatment plans, immediate accessibility to epinephrine, and staff training on administration of epinephrine are areas that need to be emphasized.

Reviewer’s Comments. This study demonstrates that most schools have at least 1, if not several, food-allergic children. It also revealed a large number of deficiencies in school policies regarding food-allergic children, such as lack of school-wide staff education, lack of avoidance measures (instructions on food labeling for cafeteria workers as well as knowledge on who has food allergies), lack of written emergency plans, lack of accessibility to epinephrine, and lack of personnel who can administer epinephrine. Previous studies have shown that even those who are responsible for administering self-injectable epinephrine often are not familiar with the correct technique for administration. Schools need help from physicians on proper policies and programs to keep food-allergic children safe from harm.

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AN ETIOLOGICAL ROLE FOR AEROALLERGENS AND EOSINOPHILS IN EXPERIMENTAL ESOPHAGITIS


Purpose of Study. An experimental model was established to test the hypothesis that eosinophilic esophagitis is mechanistically linked to eosinophilic allergic responses in the lung.

Study Population. Eight- to 10-week-old BALB/c mice, interleukin (IL)-5 gene-targeted mice, and eotaxin-deficient inbred mice were maintained with age- and sex-matched controls.

Methods. Using previously published protocols, mice were exposed to repeated inoculations of Aspergillus fumigatus antigens by oral, intragastric, and intranasal routes. Eosinophils levels in the esophagus were analyzed by anti-major basic protein immunostaining. The tissue distribution of eosinophils after intranasal allergen was examined in the blood, bronchoalveolar lavage fluid, stomach, and small intestine. Pathologic changes were defined using histologic examination of the esophagi and electron microscope analysis of tissue eosinophil morphology. Experimental eosinophilic esophagitis was induced in eotaxin gene-targeted mice and in IL-5 gene-targeted mice.

Results. Allergen-challenged mice developed marked levels of esophageal eosinophils, free eosinophil granules, and epithelial cell hyperplasia, which mimic pathophysiologic changes observed in humans with eosinophilic inflammation of the esophagus. Of note, eosinophil levels in the stomach and small intestine did not significantly increase after allergen challenge. As opposed to the intranasal route, exposure of mice to oral or intragastric allergen does not promote eosinophilic esophagitis, indicating that hypersensitivity in the esophagus occurs with simultaneous development of pulmonary inflammation. In the absence of eotaxin, eosinophil recruitment is attenuated, and furthermore, in the absence of IL-5, eosinophil accumulation and epithelial hyperplasia were ablated.

Conclusions. These results establish a pathophysiologic connection between allergic hypersensitivity responses in the lung and esophagus and demonstrate an etiologic role for inhaled allergens and eosinophils in gastrointestinal inflammation. Moreover, these investigations dissect the cellular and molecular mechanisms involved in eosinophil homing into the esophagus. Aeroallergens may be contributing to the pathogenesis of eosophageal inflammation in a subset of patients with primary eosinophilic esophagitis and gastroesophageal reflux disorders.

Reviewer’s Comments. Just when you thought you had heard of the last potential trigger for gastroesophageal reflux disorders, this very provocative investigative model of experimental eosinophilic esophagitis was published. These data suggest that eosinophilic esophagitis can be mediated by extrinsic allergens and establish a causal link between the development of allergic hypersensitivity in the respiratory tract and in the esophagus. This model not only implicates a role for aeroallergens in the pathogenesis of eosagitis, but also provides a novel system to evaluate the treatment of eosinophilic esophageal disorders, which include gastroesophageal reflux, allergic eosinophilic esophagitis, eosinophilic gastroenteritis, primary eosinophilic esophagitis, and drug reactions.

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ANAPHYLAXIS

CAN EPINEPHRINE INHALATIONS BE SUBSTITUTED FOR EPINEPHRINE INJECTION IN CHILDREN AT RISK FOR SYSTEMIC ANAPHYLAXIS?

An Etiological Role for Aeroallergens and Eosinophils in Experimental Esophagitis
John M. James

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