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Allergens, germs and asthma

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Abstract

Objective: To explore asthma pathogenesis using data from upper and lower airways.

Data Source: English-language papers on human asthma and nasal polyp subjects from 1990 onwards.

Study Selection: High-quality studies in established journals.

Results: The recognition of its inflammatory nature led to a quantum leap in the understanding and treatment of asthma, with lives saved by inhaled corticosteroids. Further work at genetic, molecular, histological and clinical levels has shown that asthma is polymorphic and rarely involves isolated Th2 bronchial inflammation.

Viral infections may act as an initiating event in children and adults, showing synergy with atopy. Chronic staphylococcal colonization of the mucosa may act as a promoter, as in atopic dermatitis. These two observations may be linked, with viruses providing an entry for bacteria into the mucosal epithelium.

Conclusions: Most asthma begins in the nose and involves allergy and infection: both viral and bacterial. The combination of atopy and infection suggests new possibilities for therapy.

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Key words
airway epithelium – asthma – asthma mechanisms – bacterial infection – viral infection

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The relationship between allergy and asthma has long been the subject of debate. It is obvious that allergen exposure in Immunoglobulin E (IgE)-sensitized asthmatic subjects can provoke wheezing, shortness of breath and falls in forced expiratory volume in 1 second (1). However, allergen avoidance and allergen-specific immunotherapy have not proven consistently effective in asthma (2, 3), which involves mechanisms other than Th2 inflammation, such as Th1 inflammation and remodeling (4, 5). Anti-IgE therapy has reopened the issue: it is undoubtedly effective in severe asthma (6), but it appears that eosinophils, rather than IgE, may be a better biomarker for those likely to benefit, since non-atopic severe asthmatics also respond (7), as do nasal polyps (8). Local IgE formation, without evidence of sensitization at systemic level, i.e. negative skin prick and blood IgE tests, may provide an answer (9–12), since the immunopathology of allergic and intrinsic asthma is similar (13).

An interaction between infection and allergy has been noted: children with allergic rhinitis suffer more and for longer with viral colds (14). Most acute asthma exacerbations start in the nose with a viral cold (15, 16). In allergic asthmatic children who are exposed to the relevant allergen and who then catch a cold, the odds ratio for hospital admission for asthma is 19 (17).

In fact, asthma itself usually begins with nasal disease. The European Community Respiratory Health Survey data show that both allergic and non-allergic rhinitis are risk factors for subsequent asthma development (18). The combination of recurrent viral colds and allergic rhinitis in children carries a high odds