Allergic Rhinitis and Asthma: Role of Environmental Determinants

ABSTRACT: Environmental exposures both cause and exacerbate allergic rhinitis and asthma, which are allergy-mediated diseases. The critical period for allergen sensitization is the first 2 to 3 years of life or earlier. Environmental interventions initiated during the third trimester for pregnant women with a personal or family history of allergies or asthma may reduce allergen sensitization and the development of allergic rhinitis and asthma in children. Most persons with allergies and almost three quarters of persons with asthma are sensitized to dust mites. Exposure to house dust mites in early childhood is an important determinant for the later development of asthma. Reduction of dust mite allergen to very low levels may prevent dust mite sensitization and asthma. Cat and dog allergen sensitization is strongly associated with the development of asthma. The combination of cockroach sensitization and high levels of cockroach exposure may explain the increased frequency of allergic asthma among inner-city children.

Key words: rhinitis, asthma, allergens, dust mites, cockroaches

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Approximately 56 million Americans (20% of the population) suffer from allergic rhinitis, and about 5% have asthma.1 Recent epidemiologic studies indicate that the prevalence of allergic rhinitis and asthma is increasing in the United States and throughout the world.1,2 Indoor environmental allergen exposure is widely believed to be a major contributing factor to the rising prevalence of these disorders. This hypothesis is supported by studies that have compared the prevalence of atopy and asthma among rural and urban populations and found that these conditions were more prevalent among rural inhabitants who moved to urban dwellings.1,3 This striking finding highlights the importance of domestic exposures in creating susceptibility to atopy and asthma among urban dwellers.

In this article, I review the environmental determinants of allergic rhinitis and asthma. In a second article, page 508, I address the importance of allergen avoidance measures in patients with these conditions and review interventions directed at reducing exposure to dust mite, pet, and cockroach allergens.

ALLERGIC DISEASE

The annual cost of treating allergic rhinitis in the United States exceeds $17 billion. The annual cost of asthma treatment is estimated at nearly $17 billion per year.1,4 This economic burden is even more substantial if one factors in the costs of treating sinusitis and otitis media, which are common complications of inadequately treated allergic rhinitis.1,5 The significant morbidity and rising health care costs associated with allergic rhinitis and asthma mandate that physicians become more proficient in the evaluation and treatment of these conditions. The triggers and symptoms of rhinitis and asthma are reviewed in the Table.

Allergic rhinitis. This is the most prevalent chronic illness diagnosed in children younger than 18 years and the fifth most common chronic illness diagnosed overall. Persons with allergic rhinitis are at increased risk for asthma.1,5 Seasonal allergic rhinitis refers to the presence of allergy symptoms triggered by pollen or mold spore allergens during the spring, summer, or fall. These pollen and mold spore seasons vary geographically throughout the United States.

Symptoms may include sneezing fits (5 to 10 sneezes in succession); itchiness of the eyes, ears, nose, throat, and palate; runny nose; watery/puffy eyes; nasal congestion; postnasal drip; sinus pressure; and fatigue. Symptoms are typically worse when patients are outdoors during pollen seasons and improve when they are indoors in an air-conditioned environment.1,6 Patients usually can distinguish whether
symptoms occur seasonally, perennially, or both.

Perennial allergic rhinitis refers to year-round symptoms that are triggered by indoor allergens, such as those from dust mites, cockroaches, mold spores, feathers, and animals. Symptoms include nasal congestion, postnasal drip, sinus pressure/headaches, and ear plugging/popping and may include any or all of the above seasonal allergy symptoms.

Patients with both perennial symptoms and seasonal exacerbations are considered to have perennial allergic rhinitis with a seasonal component.\textsuperscript{1,6} Triggers may include outdoor pollens and indoor allergens such as dust mites; mold spores; and exposures to cats, dogs, birds, and cockroaches. Perennial allergens, such as those from dust mites, molds, animals, and cockroaches, may be difficult to identify by the history alone. Skin testing is necessary to confirm sensitization to these allergens but does not indicate that the person is currently being exposed.\textsuperscript{1,4}

Conditions such as nonallergic rhinitis (vasomotor rhinitis or nonallergic rhinitis with eosinophil syndrome) can mimic allergic rhinitis. Patients with nonallergic rhinitis experience nasal congestion, postnasal drip, sinus pressure/headaches, and ear plugging. Results of skin testing for seasonal and perennial allergens are negative.

The triggers for nonallergic rhinitis include weather changes (both temperature and barometric pressure changes); postural changes; and irritants, such as smoke, potpourri, perfumes, cleaning agents, solvents, incense, and soaps or detergents.\textsuperscript{1,6} Patients who have allergic and nonallergic rhinitis components are said to have mixed rhinitis, which may occur in up to 50% of patients presenting with rhinitis symptoms. More recently, patients who do not exhibit positive skin prick testing or specific IgE serologic testing to aeroallergens but who have localized intranasal specific IgE to aeroallergens have been described and are referred to as having “entopic” rhinitis.\textsuperscript{7}

**Asthma.** This chronic obstructive inflammatory lung disease is characterized by airway inflammation, bronchial hyperresponsiveness, and at least partial improvement of lung function after treatment with bronchodilator medication.\textsuperscript{1,8} Untreated asthma can lead to airway remodeling or scarring of the airways that results in permanent loss of lung function.

Typical asthma symptoms include wheezing, coughing, chest tightness, and shortness of breath. However, many other conditions, such as gastroesophageal reflux dis-

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### Table – Triggers and symptoms of allergic disease

<table>
<thead>
<tr>
<th>Condition*</th>
<th>Triggers</th>
<th>Symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Seasonal allergic rhinitis</strong></td>
<td>Pollen or mold spores</td>
<td>Sneezing fits; itchiness of eyes, ears, nose, throat, palate; runny nose; watery/puffy eyes; nasal congestion; postnasal drip; sinus pressure; fatigue</td>
</tr>
<tr>
<td><strong>Perennial allergic rhinitis</strong></td>
<td>Indoor allergens such as those from dust mites, cockroaches, mold spores, feathers, and animals</td>
<td>Nasal congestion, postnasal drip, sinus pressure/headaches, ear plugging/popping plus above symptoms of seasonal allergic rhinitis</td>
</tr>
<tr>
<td><strong>Perennial allergic rhinitis with a seasonal component</strong></td>
<td>Outdoor pollens and indoor allergens such as dust mites; mold spores; exposures to cats, dogs, birds, and cockroaches</td>
<td>Symptoms of seasonal and perennial allergic rhinitis</td>
</tr>
<tr>
<td><strong>Nonallergic rhinitis</strong></td>
<td>Weather changes (both temperature and barometric pressure changes); postural changes; irritants, such as smoke, potpourri, perfumes, cleaning agents, solvents, incense, and soaps or detergents</td>
<td>Nasal congestion, postnasal drip, headaches/sinus pressure, ear plugging</td>
</tr>
<tr>
<td><strong>Allergic asthma</strong></td>
<td>Animals, dust mites, cockroaches, mold spores, pollen</td>
<td>Wheezing, cough, chest tightness, dyspnea</td>
</tr>
<tr>
<td><strong>Nonallergic asthma</strong></td>
<td>Viral upper respiratory tract infections, exercise, extreme temperatures, irritants</td>
<td>Wheezing, cough, chest tightness, dyspnea</td>
</tr>
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</table>

*Mixed rhinitis and mixed asthma have both allergic and nonallergic triggers.*
Evidence also indicates that asthma is primarily an allergy-mediated disease. In 1989, Burrows and colleagues reported a strong correlation between total IgE levels, positive allergen skin test responses, and asthma findings; this has subsequently been confirmed by other investigators. It is now well established that patients with extrinsic (allergic) and intrinsic (nonallergic) asthma both express TH2 cytokines that play a role in the development of asthma and a number of other allergic diseases. 

Atopy is now considered a risk factor for asthma. The critical period for allergen sensitization appears to be the first 2 to 3 years of life. Studies investigating whether environmental interventions initiated during the third trimester in pregnant women with a personal or family history of allergies or asthma would prevent or delay allergen sensitization and asthama in their infants have found that specific interventions successfully reduced allergen exposures and allergen sensitization and the development of asthma in children whose homes had such interventions early on, but eventually children predisposed to asthma and allergic rhinitis became sensitized to indoor and outdoor aeroallergens.

Most of the longitudinal birth cohort studies indicate that avoidance measures over several years prevent allergen sensitization and the progression of asthma and allergic rhinitis when multiple interventions are implemented. For example, one study reported that strict allergen avoidance reduced the risk of dust mite sensitization and the development of some cases of childhood asthma. The Canadian Childhood Asthma Primary Prevention Study implemented a multifaceted intervention program to prevent asthma in high-risk infants (ie, infants with an immediate family history of asthma and allergies). Researchers found that the prevalence of pediatric allergist–diagnosed asthma was lower in the intervention group at 7 years of age. However, studies that implemented single interventions to reduce allergen exposure (bedding encasements only) did not demonstrate prevention of dust mite sensitization or the prevention of asthma and allergic rhinitis.

Dust mite allergen. A number of studies have sought to determine the role of allergenic stimuli in the subsequent development of allergic asthma. To date, the dust mite allergen has been the most extensively studied allergen. The major allergens of house dust mites come from their fecal waste particles and glandular secretions.

Dust mite allergens are primarily enzyme proteins, which may explain why they are highly sensitizing. About 90% of persons with allergies and more than 70% of asthmatic patients are sensitized to dust mites. These organisms thrive in humid environments and feed on human skin scales. They are most commonly found in bedding (pillows, mattresses, box springs, comforters), carpets, and upholstered furniture.

Many studies have confirmed the relationship between dust mites and the development of asthma and allergic rhinitis. Sporik and associates reported that exposure to house dust mites in early childhood...
is an important determinant in the later development of asthma. Peat and associates\textsuperscript{22} found that house dust mites are an important cause of childhood asthma and that reducing young children's exposure to these organisms may help prevent asthma. Subsequent studies have shown that reduction of dust mite allergen to very low levels (less than 2 µg/g of dust) can prevent dust mite sensitization and asthma.\textsuperscript{20} As mentioned, single interventions directed at reducing dust mite allergen (such as bedding encasements over the pillow and mattress) effectively reduced allergen exposure but did not reduce asthma and allergy symptoms.\textsuperscript{18,21}

**Cat and dog allergens.** Animal allergens are also an important cause of and exacerbating factor in asthma and allergic rhinitis. It is estimated that more than a third of Americans have 1 or more cats in their home. Cat allergen is a very light allergen (3 µm in diameter) that is primarily released from the sebaceous glands in the skin. Cat allergen can stay suspended in the air for 5 to 6 hours at a time and become airborne with minimal disturbance in the room.\textsuperscript{20} It sticks to walls, clothing, shoes, carpets, bedding, and furniture, and it is difficult to get rid of completely. Even after the cat has been removed from the home, measurable amounts of cat allergen can be detected for several months.\textsuperscript{24}

Lindfors and colleagues\textsuperscript{25} found a strong relationship between cat exposure and sensitization. A combination of dampness, passive smoke exposure, and cat exposure with cat sensitization was strongly associated with subsequent development of asthma. Studies have also reported significant levels of cat allergen in homes where there are no cats, which suggests that cat allergen can be brought in from outside sources on shoes and clothing.

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Lindfors and colleagues\textsuperscript{25} found that early exposure to dog or cat allergen may decrease the risk of sensitization and asthma.\textsuperscript{30,34} This observation raises important questions about the dose-response relationship between allergen exposure and sensitization.

**Cockroach allergens.** Cockroaches are present in most homes even though they usually cannot be readily observed. The sighting of a cockroach during the daytime often suggests that there is major overcrowding of cockroach populations and significant infestation in the home. The 2 major species of cockroaches in the United States are German and American.\textsuperscript{35} A significant amount of evidence supports a relationship between cockroach sensitization and asthma.

Rosenstreich and associates\textsuperscript{36} found that inner-city children with asthma are allergic to dust mites, cockroaches, and cats. However, they found that cockroach allergen levels in the bedrooms of these children were approximately 5 times higher than dust mite and cat allergen levels. Children with cockroach allergy and high cockroach bedroom exposure levels were at much greater risk for hospitalization and unscheduled medical visits for asthma and had more asthma symptoms overall compared with other children with asthma not sensitized to cockroach allergen.

The combination of cockroach sensitization and high levels of cockroach exposure may explain the increased frequency of asthma reported among inner-city children. Sarpong and colleagues\textsuperscript{37} reported that African American race and low socioeconomic status were significant independent risk factors for cockroach allergen sensitization in children with atopic asthma.

**Other risk factors.** Many other factors have been found to be potentially important for the later development of asthma and allergies. These include a maternal history of asthma, maternal smoking during pregnancy, lack of prenatal care, low birth weight, long duration of breastfeeding, firstborn status, moldy or humid living environments, passive
smoke exposure, and recurrent viral infections or bronchiolitis in the first few years of life.30

REFERENCES:

CLINICAL HIGHLIGHTS
- Persons with allergic rhinitis are at increased risk for asthma.
- Nonallergic rhinitis may mimic allergic rhinitis. Patients with nonallergic rhinitis experience nasal congestion, postnasal drip, headaches, sinus pressure, and ear plugging. Results of skin testing for seasonal and perennial allergens in these patients are negative. Up to 50% of patients with rhinitis symptoms may have allergic and nonallergic rhinitis components.
- Seasonal allergic rhinitis is triggered by pollen or mold spore allergens. Patients with perennial allergic rhinitis have year-round symptoms that are triggered by indoor allergens, such as those from dust mites, cockroaches, mold spores, feathers, and animals.
- Patients with both perennial symptoms and seasonal exacerbations may be sensitive to dust mites, mold spores, and exposures to cats, dogs, birds, and cockroaches. Skin testing is necessary to confirm sensitization to these allergens.
- Triggers of allergic asthma include animals, dust mites, cockroaches, mold spores, and pollen. Nonallergic triggers include viral upper respiratory tract infections, exercise, extreme temperatures, and irritants.