ASSOCIATION BETWEEN RHINITIS AND ASTHMA
DEFINITION

Rhinitis is defined as the presence of sneezing, rhinorrhea nasal congestion, and/or nasal itching arising from irritation and inflammation of the nasal passages. Rhinosinusitis describes disorders affecting both the nasal passages and paranasal sinuses. (1)

Asthma is heterogeneous disease, usually characterized by chronic airway inflammation. It is defined by the history of respiratory symptoms, such as wheeze, shortness of breath, chest tightness and cough, that vary over time and in intensity, together with variable expiratory airflow limitation. (2)
Asthma and allergic rhinitis often coexist and may represent similar disease entities (one airway hypothesis).

There is also a strong link between asthma and bacterial rhinosinusitis, viral upper respiratory infections and nasal polyposis.

Rhinitis occurs in 75 to 90 percent of adult subjects with allergic asthma and 80 percent of those with nonallergic asthma. Conversely, asthma occurs in 25 to 50 percent of individuals with rhinitis.

Adults with perennial rhinitis are more likely to have asthma than those without rhinitis.
The same agents can trigger both rhinitis and asthma. These include:

The structure of the airway mucosa is similar in the nose and bronchi. Histologically similar respiratory epithelium extends posteriorly from the nasal septum and lateral walls of the nasal fossa to the nasopharynx, larynx, trachea, bronchi, and bronchioles.

Postulated mechanisms through which the upper and lower airway may interact include:

● **Neural interaction (nasal bronchial reflex)**

  – Animal studies demonstrate reflexes arising from receptor sites in the nose and nasopharynx. These reflexes are mediated by the afferent sensory components of the trigeminal and glossopharyngeal nerves and the efferent bronchoconstrictor fibers of the vagus nerve.

**Disturbance of nasal mucosa conditioning of the air entering the respiratory tree**

  – Air warming and humidification are the basic functions of the nasal airway and sinuses. Nasal breathing appears to have a protective effect on exercise-induced bronchospasm. Exercise treadmill tests were conducted with 12 children with mild-to-moderate asthma who were instructed to breathe only through their nose, only through their mouth, or breathe "naturally" (resulted in oral breathing in most patients). Spontaneous breathing during the exercise challenge resulted in bronchoconstriction, with a decrease in FEV₁. Oral breathing exaggerated the airway hyperreactivity, and nasal breathing abrogated the bronchoconstrictive response. (4,5,6)
A study of eight adult females with asymptomatic mild asthma demonstrated a slight, progressive decrease in FEV$_1$ over a one-hour period during exclusive oral breathing.

Patients also perceived increased difficulty in breathing, and three patients experienced coughing/wheezing after oral breathing.

Similar findings were not seen with enforced nasal breathing in the same subjects.(4,5,6)
INTERACTIONS BETWEEN THE UPPER AND LOWER AIRWAYS

- **Effects of nitric oxide on both upper and lower airways**
  - Nitric oxide is formed in multiple cell types by various mechanisms. It serves a protective function. It has strong antiviral and bacteriostatic activity, bronchodilatory effects, and modulatory effects on lower airway responsiveness. In addition, it improves oxygenation. Decreased nitric oxide levels are found in patients with inflammatory conditions, such as chronic rhinosinusitis with or without nasal polyposis.

- **Irritant and inflammatory effects of nasal secretions directly draining into the lower airway, including particles and irritants entrapped in the mucociliary blanket**
  - Studies in a rabbit model of acute rhinosinusitis provided strong evidence that drainage of nasal inflammatory mediators into the lung may precipitate asthma. There is evidence that pharyngeal aspiration occurs in healthy humans as well as in those with decreased consciousness. (4,5,6)
INTERACTIONS BETWEEN THE UPPER AND LOWER AIRWAYS

- **Systemic propagation of inflammation via effects of mediators and inflammatory cells**

  Systemic propagation of inflammation may be a principal connection between the upper and lower airways in patients with allergic respiratory disease.

Studies suggest that allergic airway inflammation leads to recruitment of eosinophils into the systemic circulation, which then leads to inflammation of respiratory mucosa not exposed to the culprit allergen. Specifically, allergen provocation can lead to inflammatory cell production in the bone marrow.

The systemic increase in inflammatory cells can also be detected outside of the airway. Patients with asthma and allergic rhinitis have increased numbers of eosinophils, mast cells, and T cells in duodenal biopsies. (4,5,6)
The mucosal cellular infiltrates that characterize rhinosinusitis and asthma are similar (eg, eosinophils, mast cells, macrophages, and T cells). In addition, the same proinflammatory mediators are present in both nasal and bronchial mucosa (eg, histamine; leukotrienes; interleukin [IL] 4; IL-5; IL-13; granulocyte-macrophage colony-stimulating factor; regulated on activation, normal T cell expressed and secreted [RANTES]; and adhesion molecules). (7)
Longitudinal studies suggest that both allergic rhinitis with positive allergy skin tests and nonallergic rhinitis are risk factors for development and persistence of asthma.

*One series surveyed 690 individuals as college freshmen and then reevaluated them 23 years later. None of these individuals had a diagnosis of asthma or symptoms compatible with asthma at the time of their first evaluation. However, 162 had a diagnosis of rhinitis.

The incidence of asthma over 23 years was 10.5 percent in subjects with rhinitis and 3.6 percent in those without rhinitis. This study suggests that subjects with allergic rhinitis are three times more likely to develop asthma than those without allergic rhinitis.*
The Tucson Epidemiologic Study of Obstructive Lung Diseases assessed rhinitis as a potential risk factor for asthma. This study compared 173 adults who developed asthma over 10 years with 2177 control subjects who, during the same approximate period, did not have any chronic lower respiratory tract symptoms. They controlled for several confounding variables, including smoking status and concomitant chronic obstructive pulmonary disease. The odds ratio (OR) for developing asthma was 2.59 (95% CI 1.54-4.34) if rhinitis was present and was 6.28 (95% CI 4.01-9.82) if both rhinitis and rhinosinusitis were present. The risk for asthma increased with the persistence or severity of rhinitis.\

A birth cohort study examined risk factors for persistent wheezing at six years of age. Persistent asthma at six years of age was associated with inhalant allergy, with positive skin tests to aeroallergens and rhinitis independent of colds. These continue to be markers of asthma persistence through adolescence and adult life.
CONCLUSION

- **One airway hypothesis** – Asthma and rhinosinusitis often coexist and likely represent a spectrum of the same disease entity. Rhinitis occurs in 75 to 90 percent of adult subjects with asthma, and asthma occurs in 25 to 50 percent of individuals with rhinitis. The upper and lower airways are contiguous and have anatomic and physiologic similarities. In addition, the cellular infiltrates and inflammatory mediators present in rhinitis and asthma are similar, and both disorders have the same triggers.

- **Rhinitis is a risk factor for asthma** – Longitudinal studies suggest that both allergic rhinitis with positive allergy skin tests and nonallergic rhinitis are risk factors for development and persistence of asthma in children and adults.

- **Interactions between the upper and lower airway** – Postulated mechanisms through which the upper and lower airway may interact include the nasal bronchial reflex, disturbance of nasal mucosa conditioning, nitric oxide effects, drainage of irritant and inflammatory material, and systemic propagation of inflammation.

- **Clinical implications** – Patients with persistent asthma should be evaluated for rhinitis and rhinosinusitis because detection and treatment of nasal disease improves asthma.
REFERENCES


THANK YOU FOR YOUR ATTENTION!