

DEVELOPMENT MECHANISM AND TREATMENT METHODS OF ALLERGIC RHINITIS AND BRONCHIAL ASTHMA

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<https://doi.org/10.5281/zenodo.17197682>

Abstract. Allergic rhinitis is an IgE-mediated inflammatory response of the nasal mucosa to allergens such as pollen, dust mites, mold, or animal dander. Bronchial asthma is a chronic inflammatory disease of the airways characterized by hyperresponsiveness, reversible airflow obstruction, and recurrent episodes of wheezing, breathlessness, and coughing.

Keywords: APCs, Mast cell activation, Chronic inflammation, Airway hyperreactivity, β 2-agonists.

Introduction

Allergic rhinitis (AR) and bronchial asthma (BA) are chronic inflammatory diseases of the respiratory system that often coexist and share common immunopathological mechanisms.

Both conditions significantly affect the quality of life, productivity, and overall health of patients worldwide. Understanding their development mechanisms and modern treatment strategies is crucial for effective management.

Development Mechanism

Allergic Rhinitis

Allergic rhinitis is an IgE-mediated inflammatory response of the nasal mucosa to allergens such as pollen, dust mites, mold, or animal dander.

Pathogenesis:

Sensitization phase – Initial exposure to allergens activates antigen-presenting cells (APCs), leading to T-helper 2 (Th2) cell activation.

IgE production – Th2 cells stimulate B lymphocytes to produce allergen-specific IgE antibodies.

Mast cell activation – IgE binds to mast cells, and upon re-exposure, allergen cross-links IgE receptors, leading to mast cell degranulation.

Release of mediators – Histamine, leukotrienes, prostaglandins, and cytokines cause sneezing, nasal congestion, rhinorrhea, and itching.

Chronic inflammation – Eosinophils and other inflammatory cells infiltrate the mucosa, leading to persistent symptoms.

Bronchial Asthma

Bronchial asthma is a chronic inflammatory disease of the airways characterized by hyperresponsiveness, reversible airflow obstruction, and recurrent episodes of wheezing, breathlessness, and coughing.

Pathogenesis:

Sensitization and IgE response – Similar to AR, allergens induce IgE production and mast cell activation.

Acute phase reaction – Histamine and leukotrienes cause bronchoconstriction, increased vascular permeability, and mucus secretion.

Chronic inflammation – Persistent recruitment of eosinophils, Th2 cells, and mast cells contributes to airway remodeling.

Airway hyperreactivity – Smooth muscle hypertrophy, goblet cell hyperplasia, and basement membrane thickening lead to exaggerated bronchial responses to triggers.

Treatment Methods

Allergic Rhinitis

1. Allergen avoidance – Reducing exposure to dust mites, pollen, pets, and irritants.

2. Pharmacotherapy:

Antihistamines (e.g., loratadine, cetirizine) – reduce sneezing and itching.

Intranasal corticosteroids (e.g., fluticasone, mometasone) – most effective for nasal inflammation.

Decongestants – provide temporary relief of nasal obstruction.

Leukotriene receptor antagonists (e.g., montelukast) – especially useful in patients with concomitant asthma.

3. Allergen immunotherapy (AIT): Subcutaneous or sublingual administration of allergens to induce tolerance and modify disease progression.

Bronchial Asthma

1. Trigger control – Avoiding allergens, smoke, pollutants, and occupational irritants.

2. Pharmacotherapy:

Short-acting β_2 -agonists (SABA) – rescue therapy for acute attacks.

Inhaled corticosteroids (ICS) – cornerstone of long-term control.

Long-acting β_2 -agonists (LABA) – used with ICS for moderate-to-severe asthma.

Leukotriene receptor antagonists – for mild asthma or add-on therapy.

Biological agents – anti-IgE (omalizumab), anti-IL-5 (mepolizumab), anti-IL-4/13 (dupilumab) for severe refractory asthma.

3. Non-pharmacological approaches:

Pulmonary rehabilitation and breathing exercises.

Patient education and self-management plans.

Conclusion

Allergic rhinitis and bronchial asthma are closely related conditions sharing similar immunological pathways. Both are driven by IgE-mediated hypersensitivity, mast cell degranulation, and chronic airway inflammation. Effective management requires a multifaceted approach including allergen avoidance, pharmacological treatment, and immunotherapy. Recent advances in biologics offer promising options for patients with severe, uncontrolled asthma and persistent allergic rhinitis. An integrated treatment strategy not only relieves symptoms but also improves long-term prognosis and quality of life.

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