



Immune Mechanisms and Clinical Management of Bronchial Asthma

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DESCRIPTION

Bronchial asthma is a chronic inflammatory disease of the airways characterized by reversible airway obstruction, bronchial hyper responsiveness and respiratory symptoms such as wheezing, coughing, chest tightness and shortness of breath. The condition affects individuals of all ages and remains a major global health concern due to its high prevalence, morbidity and potential for severe exacerbations. The pathophysiology of asthma is complex and involves a dynamic interaction between genetic predisposition, environmental exposures and immune system dysregulation. In susceptible individuals, inhaled allergens, pollutants, respiratory infections or occupational irritants can trigger an exaggerated immune response, leading to airway inflammation, mucus production and smooth muscle contraction. Understanding the mechanisms that drive these responses is critical for the development of effective treatment strategies and for improving patient outcomes. Asthma is now recognized as a heterogeneous condition with multiple phenotypes and endotypes, which reflect differences in underlying immunological pathways, clinical presentation and response to therapy. The immune response in bronchial asthma is dominated by T helper type 2 lymphocytes, which produce cytokines such as interleukin 4, 5 and 13. These cytokines orchestrate a cascade of events, including the recruitment of eosinophils, activation of mast cells and stimulation of immunoglobulin E production by B cells. The resulting inflammatory milieu contributes to airway edema, increased mucus secretion and structural changes in the airway known as remodelling. Non-eosinophilic forms of asthma, including neutrophilic and mixed granulocytic phenotypes, have been increasingly recognized, particularly in adults with severe disease or those resistant to conventional therapy. These variations highlight the importance of personalized approaches in diagnosis and treatment. Genetic studies have identified multiple susceptibility loci associated with immune regulation, epithelial barrier function and airway hyperreactivity, further emphasizing the complex interplay between inherited and environmental factors in disease development.

The prevalence of bronchial asthma has risen significantly over the past few decades, affecting an estimated 300 million people worldwide. Urbanization, increased exposure to air pollutants, lifestyle changes and alterations in microbial exposure are considered key contributors to this trend. The disease places a substantial burden on healthcare systems due to frequent hospitalizations, emergency visits and the need for long-term medication. Moreover, uncontrolled asthma significantly impairs quality of life, limiting physical activity, sleep and daily functioning. The association of asthma with other allergic conditions, such as rhinitis and atopic dermatitis, underscores the systemic nature of immune dysregulation and the need for integrated management strategies. Accurate diagnosis of bronchial asthma requires careful clinical evaluation, including detailed history-taking and physical examination. Pulmonary function tests, such as spirometry, are essential to assess airflow limitation and reversibility following bronchodilator administration. Additional assessments, including peak flow monitoring, exhaled nitric oxide measurement and sputum eosinophil counts, provide further insight into airway inflammation and disease phenotype. Early identification of triggers and phenotypic characterization allow clinicians to tailor treatment and prevent exacerbations. Education and self-management strategies, including the use of asthma action plans and regular monitoring, are critical components of effective long-term care. Pharmacological management of bronchial asthma has evolved significantly, with inhaled corticosteroids forming the cornerstone of therapy for persistent disease. These medications reduce airway inflammation and prevent exacerbations, while short-acting beta agonists provide rapid symptom relief during acute episodes. For patients with moderate to severe disease, long-acting bronchodilators, leukotriene receptor antagonists and biologic therapies targeting specific inflammatory pathways offer additional control. Biologics, such as monoclonal antibodies against immunoglobulin E or interleukin 5, have demonstrated substantial efficacy in reducing exacerbations and improving lung function in carefully selected patient populations. The integration of these therapies with environmental control

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Received: 24-Nov-2025, Manuscript No. JAT-25-31331; **Editor assigned:** 26-Nov-2025, Pre QC No. JAT-25-31331 (PQ); **Reviewed:** 10-Dec-2025, QC No JAT-25-31331; **Revised:** 17-Dec-2025, Manuscript No. JAT-25-31331 (R); **Published:** 24-Dec-2025, DOI: 10.35248/2155-6121.25.16.445

Citation: Thompson M (2025) Immune Mechanisms and Clinical Management of Bronchial Asthma. *J Allergy Ther.*16:445.

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measures, patient education and regular follow-up provides a comprehensive approach to disease management.

In conclusion, bronchial asthma is a multifaceted chronic disease driven by immune dysregulation, environmental factors and genetic susceptibility. Effective management requires an integrated approach that includes accurate diagnosis, pharmacological therapy, patient education and lifestyle modification. Advances in immunology and personalized medicine have provided new insights into disease phenotypes

and innovative treatment options, particularly biologic therapies for severe asthma. Continued research, combined with public health strategies and patient-centered care, is essential for controlling disease progression, reducing exacerbations and improving quality of life for individuals affected by this pervasive condition. Bronchial asthma exemplifies the complex interaction between immunity, environment and genetics, highlighting the importance of multidisciplinary efforts in addressing chronic respiratory diseases.