

Infection, inflammation, remodeling drive respiratory diseases.

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Introduction

This systematic review and meta-analysis examined airway remodeling in non-Cystic Fibrosis (CF) bronchiectasis, revealing significant structural changes like increased bronchial wall thickness. The review underscores the importance of understanding these architectural alterations, which are central to disease progression and impaired lung function, suggesting that addressing remodeling pathways could be a key therapeutic target [1].

A systematic review and meta-analysis on the role of respiratory viruses in bronchiectasis exacerbations found that various viral pathogens are frequently associated with acute worsening of the condition. This highlights how viral infections directly contribute to poor asthma control and increased disease burden in patients with bronchiectasis, emphasizing the need for robust antiviral strategies and infection prevention [2].

This review explores airway inflammation and remodeling in asthma, contrasting it with COPD and discussing current and future therapeutic perspectives. It highlights how chronic inflammation drives structural changes in the airways, contributing to reduced lung function and suboptimal asthma control, offering insights into potential targets for intervention in similar airway diseases [3].

This study delves into the critical role of eosinophilic inflammation in bronchiectasis, particularly in cases co-occurring with Allergic Bronchopulmonary Aspergillosis (ABPA). It suggests that eosinophils contribute significantly to airway damage and remodeling, impacting disease severity and potentially influencing asthma-like symptoms and control strategies in this patient subgroup [4].

This review highlights the substantial impact of viral respiratory infections on asthma exacerbations and overall control. It discusses how viruses trigger immune responses that worsen airway inflammation and contribute to the remodeling processes, making asthma harder to manage. The insights emphasize the importance of identifying and mitigating viral triggers for better asthma outcomes [5].

A prospective cohort study investigating the airway bacterial microbiome in bronchiectasis revealed that specific bacterial communities are associated with an increased risk of exacerbations. This

underscores the crucial link between chronic bacterial infections, recurrent inflammatory cycles, and the potential for progressive airway damage, impacting overall disease stability and long-term control [6].

This systematic review explores the complex relationship between bronchiectasis and asthma, questioning if there's a direct link. It identifies a significant overlap in clinical presentation and underlying inflammatory mechanisms, suggesting that coexistence or shared pathways might impact disease progression, airway remodeling, and overall treatment responsiveness for both conditions [7].

This article focuses on airway remodeling in chronic lung diseases, specifically highlighting the roles of Epithelial-Mesenchymal Transition (EMT) and fibroblasts. It explains how these cellular processes contribute to the structural changes seen in airways, providing fundamental insights into the mechanisms driving irreversible damage in conditions like bronchiectasis and severe asthma [8].

This review elucidates the role of inflammation and infection in airway epithelial dysfunction and remodeling in asthma. It details how recurrent infections and chronic inflammatory responses impair the epithelial barrier, leading to structural changes that perpetuate disease and complicate asthma control. Understanding these interactions is crucial for developing targeted therapies [9].

This systematic review and meta-analysis evaluated the clinical impact of fungal respiratory infections on asthma severity and control. It found that fungal infections are significant contributors to poorly controlled and severe asthma, suggesting they can exacerbate airway inflammation and potentially influence remodeling, which demands careful diagnostic and therapeutic attention for improved asthma management [10].

Conclusion

Research highlights the critical role of airway remodeling and chronic inflammation in respiratory diseases like bronchiectasis and asthma. In non-Cystic Fibrosis (CF) bronchiectasis, significant structural changes, such as increased bronchial wall thickness, are central to disease progression and impaired lung function, suggest-

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ing remodeling pathways as therapeutic targets. Viral respiratory infections are frequently linked to acute worsening of bronchiectasis exacerbations, directly contributing to poor disease control and increased burden, which points to the need for effective antiviral strategies. Similarly, these infections trigger immune responses that exacerbate airway inflammation and remodeling, making asthma harder to manage. The interplay of inflammation and infection is crucial in airway epithelial dysfunction in asthma, where recurrent infections and chronic inflammatory responses impair the epithelial barrier, leading to structural changes that complicate asthma control. Eosinophilic inflammation also plays a significant role in bronchiectasis, especially when Allergic Bronchopulmonary Aspergillosis (ABPA) is present, contributing to airway damage and influencing asthma-like symptoms. Specific bacterial communities in the airway microbiome are associated with increased exacerbation risk in bronchiectasis, emphasizing the link between chronic bacterial infections and progressive airway damage. There's a notable overlap between bronchiectasis and asthma in clinical presentation and inflammatory mechanisms, indicating that shared pathways impact disease progression and treatment. Finally, fungal respiratory infections notably contribute to severe and poorly controlled asthma, exacerbating inflammation and influencing remodeling, requiring precise diagnostic and therapeutic focus for better management.

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