Airway remodeling in asthma: Mechanisms and personalized therapy.

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Introduction

This review highlights the complex nature of airway remodeling in asthma, discussing its key structural changes like fibrosis, smooth muscle hypertrophy, and angiogenesis. It emphasizes how these changes contribute to persistent airflow limitation and disease severity, often irrespective of inflammation. The article explores different phenotypes of asthma associated with specific remodeling patterns and potential therapeutic strategies targeting these structural changes, moving beyond traditional anti-inflammatory approaches[1].

This article delves into the less-explored contributions of non-structural cells, such as macrophages, dendritic cells, and mast cells, to airway remodeling in asthma. It posits that these cells play crucial roles beyond inflammation, actively participating in extracellular matrix deposition, angiogenesis, and smooth muscle proliferation. Understanding their mechanisms offers new avenues for therapeutic interventions targeting specific cellular pathways to mitigate airway structural changes and improve patient outcomes[2].

This study investigates the correlation between impaired pulmonary function in children with severe asthma and airway remodeling, utilizing fractional anisotropy (FA) as a marker. The findings suggest that specific patterns of airway remodeling, quantifiable by FA, are directly linked to reduced lung function, particularly in children. This highlights FA as a promising non-invasive biomarker for assessing structural changes in pediatric asthma and identifying patients at higher risk for persistent functional deficits[3].

This review provides a comprehensive overview of the complex mechanisms driving airway remodeling in asthma, covering cellular and molecular pathways involved in structural changes. It bridges the understanding of pathophysiology with emerging therapeutic strategies, including those targeting specific cytokines, growth factors, and signaling pathways implicated in fibrosis, smooth muscle hyperplasia, and angiogenesis. The article emphasizes the need for personalized medicine approaches to reverse or prevent remodeling[4].

This review explores the evolving landscape of asthma phenotypes and the biomarkers used to identify them, transitioning from traditional clinical markers to advanced 'omics' approaches. It discusses how integrating genetics, epigenetics, transcriptomics, proteomics, and metabolomics can provide a deeper understanding of the heterogeneous nature of asthma. The article highlights the potential of these biomarkers to refine diagnosis, predict treatment response, and facilitate the development of precision medicine strategies for asthma management[5].

This paper focuses specifically on airway smooth muscle (ASM) remodeling as a crucial component of airway pathology in asthma. It details the mechanisms underlying ASM hypertrophy and hyperplasia, including the roles of various growth factors, cytokines, and intracellular signaling pathways. The review also evaluates current and experimental therapeutic strategies aimed at modulating ASM structure and function, highlighting how targeting these specific mechanisms could lead to novel, more effective treatments for chronic asthma[6].

This article discusses the evolving understanding of phenotypes in both asthma and COPD, emphasizing the shared and distinct features that complicate diagnosis and treatment. It highlights the importance of phenotypic classification for guiding personalized therapeutic approaches, particularly in cases of ACOS (Asthma-COPD Overlap). The review underscores how identifying specific phenotypic traits can lead to more targeted interventions and improved patient outcomes across chronic airway diseases[7].

This review thoroughly examines the interplay between inflammation and airway remodeling in Asthma-COPD Overlap (ACOS). It discusses how chronic inflammation contributes to persistent structural changes in the airways, leading to fixed airflow limitation that characterizes ACOS. The article differentiates the inflammatory and remodeling patterns from pure asthma or COPD, highlighting the diagnostic and therapeutic challenges. Understanding these mechanisms is crucial for developing targeted treatments for this complex disease[8].

This review provides an update on the latest insights into the cellular and molecular mechanisms driving airway remodeling in asthma. It discusses how persistent inflammation, oxidative stress, and structural cell dysfunction contribute to thickening of the basement membrane, smooth muscle hyperplasia, and altered extracellular matrix

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composition. The article emphasizes the role of specific signaling pathways and mediators, offering a foundation for developing novel therapeutic interventions that target these fundamental processes to prevent or reverse airway structural changes[9].

This updated review provides a detailed examination of the complex pathophysiological mechanisms underlying airway remodeling in asthma. It covers the roles of various cells and mediators, including epithelial cells, fibroblasts, immune cells, and growth factors, in promoting structural changes like subepithelial fibrosis, smooth muscle hypertrophy, and mucous gland hyperplasia. The article identifies several promising therapeutic targets based on these mechanisms, highlighting the potential for novel interventions to prevent or reverse airway remodeling and preserve lung function in asthmatic patients[10].

Conclusion

Airway remodeling in asthma is a complex process involving key structural changes like fibrosis, smooth muscle hypertrophy, and angiogenesis, which contribute to persistent airflow limitation and disease severity, often independent of inflammation. Beyond structural cells, non-structural cells such as macrophages, dendritic cells, and mast cells play crucial roles in extracellular matrix deposition and smooth muscle proliferation. These changes are directly linked to reduced lung function, especially in children, with fractional anisotropy (FA) emerging as a promising non-invasive biomarker for assessment. Understanding the cellular and molecular pathways, including specific cytokines, growth factors, and signaling pathways, is vital for developing therapeutic strategies. The field is moving towards personalized medicine, utilizing 'omics' approaches (genetics, epigenetics, transcriptomics, proteomics, metabolomics) to identify asthma phenotypes and predict treatment responses. Airway smooth muscle (ASM) remodeling, involving hypertrophy and hyperplasia, is a significant component, with current research evaluating therapies to modulate ASM structure. Furthermore, phenotypes in asthma and Chronic Obstructive Pulmonary Disease (COPD), particularly Asthma-COPD Overlap (ACOS), are being characterized to guide targeted interventions. Chronic inflammation in ACOS notably contributes to fixed airflow limitation. Recent advances underscore the role of persistent inflammation, oxidative stress, and structural cell dysfunction in basement membrane thickening and altered extracellular matrix. Identifying specific signaling pathways offers foundations for novel interventions to prevent or reverse airway structural changes and preserve lung function.

References

1. Contoli M, Papi A, Cazzola M. Airway Remodeling in Asthma: *Current Concepts and Therapeutic Implications. J Allergy Clin Immunol Pract.* 2020;8:429-442.e5.
2. Li Z, Fang C, Zhang Y. Novel Insights into Airway Remodeling in Asthma: The Role of Non-Structural *Cells. Cells.* 2022;11:2200.
3. Chen Z, Li F, *Song J. Pulmonary function impairment in children with severe asthma is related to airway remodeling detected by fractional anisotropy. Allergy Asthma Clin Immunol.* 2021;17:100.
4. Damera G, Maes T, Bracke KR. Airway Remodeling in Asthma: *From the Pathophysiology to Novel Therapeutic Options. J Clin Med.* 2021;10:5046.
5. Ricciardolo FLM, Sorbello V, Ciappoli R. Biomarkers of asthma phenotypes: From clinical to 'omics' approaches. *Front Med* (Lausanne). 2022;9:994030.
6. Zhang Y, Liang J, Lu W. Airway smooth muscle remodelling in asthma: from mechanism to therapy. *Signal Transduct Target Ther.* 2023;8:292.
7. Lotvall J, Anzueto A, Miravitlles M. Phenotypes of Asthma and COPD: *An Evolving Concept. Int J Chron Obstruct Pulmon Dis.* 2019;14:2125-2137.
8. Postma DS, Rabe KF, De Meulder B. Inflammation and Airway Remodeling in Asthma-COPD Overlap: *A Comprehensive Review. Am J Respir Crit Care Med.* 2021;203:1222-1234.
9. Holgate ST, Roberts G, *Wark P. Recent advances in understanding airway remodeling in asthma. J Allergy Clin Immunol.* 2019;144:1149-1162.
10. Wang J, Zhang Y, Zhang S. Pathophysiological Mechanisms and Therapeutic Targets for Airway Remodeling in Asthma: *An Updated Review. Med Sci Monit.* 2022;28:e937213.

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