Editorial

International Journal of Clinical Rheumatology

Lung Fibrosis Fibroblast Subsets: Unraveling Cellular Diversity in Pulmonary Remodeling

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Received: 01-Oct-2025, Manuscript No. fmijcr-26-188476; **Editor assigned:** 03-Oct-2025, Pre- fmijcr-26-188476 (PQ); **Reviewed:** 16-Oct-2025, QC No. fmijcr-26-188476; **Revised:** 22-Oct-2025, Manuscript No. fmijcr-26-188476 (R); **Published:** 30-Oct-2025, **DOI:** 10.37532/1758-4272.2025.20(10).477-477

Introduction

Lung fibrosis is a chronic and progressive disease characterized by excessive extracellular matrix deposition, alveolar remodeling, and impaired respiratory function. Fibroblasts are central mediators of this process, yet recent studies reveal that they are not a uniform population. Distinct fibroblast subsets with specialized functions contribute differently to tissue repair, inflammation, and pathological remodeling. Understanding these subsets is essential for developing targeted therapies for pulmonary fibrosis.

Fibroblast Subsets in Lung Fibrosis

Fibroblast heterogeneity in fibrotic lungs arises from differences in developmental origin, gene expression, and microenvironmental cues. Key subsets include:

Myofibroblasts, which produce high levels of collagen and α-smooth muscle actin (α-SMA), driving tissue stiffening and scarring.

Lipofibroblasts, storing lipids and supporting alveolar epithelial cell function, which may lose their regenerative capacity during fibrosis.

Inflammatory fibroblasts, secreting cytokines and chemokines that perpetuate immune cell recruitment and chronic inflammation.

Single-cell RNA sequencing has been pivotal in identifying these subsets, revealing unique transcriptional profiles and signaling pathways that regulate their activation and function.

Implications for Therapy

Recognizing fibroblast heterogeneity provides opportunities for precision medicine in lung fibrosis. Therapeutic strategies could selectively inhibit pathogenic myofibroblasts while preserving beneficial subsets such as lipofibroblasts. Anti-fibrotic drugs targeting key signaling pathways, including TGF-β and Wnt, are under investigation to modulate fibroblast activity. Furthermore, fibroblast subset-specific biomarkers may improve disease stratification and monitor treatment response.

Conclusion

Fibroblast subsets play distinct and critical roles in the pathogenesis of lung fibrosis. Deciphering their heterogeneity enhances our understanding of disease mechanisms and informs the development of targeted therapies. Advances in single-cell and multi-omics profiling are expected to accelerate the identification of pathogenic and protective fibroblast populations, ultimately improving treatment strategies and patient outcomes in pulmonary fibrosis.