DeepSeek 结果：

多个具体科学论述后未直接标注Citation

The current evidence implicates macrophage polarization as a central mechanism in the pathogenesis of pulmonary fibrosis, wherein the balance between classically activated M1 and alternatively activated M2 phenotypes dictates the trajectory of tissue remodeling. While M1 macrophages are associated with early inflammatory responses and the clearance of pathogens, persistent injury and aberrant wound healing promote a shift toward an M2-dominant profile, characterized by the secretion of profibrotic mediators such as transforming growth factor-β1 (TGF-β1) and platelet-derived growth factor (PDGF). This M2 polarization, and the subsequent infiltration of M2 macrophages into the pulmonary interstitium, has been shown to drive fibroblast activation, myofibroblast differentiation, and excessive extracellular matrix deposition, thereby perpetuating the fibrotic cascade (Murray & Wynn, 2011; Wynn & Vannella, 2016). Furthermore, studies in both human fibrotic lung tissue and bleomycin-induced murine models demonstrate that increased M2 marker expression—such as CD206, arginase-1, and chitinase-like proteins—correlates with the severity of fibrosis, while therapeutic strategies aimed at inhibiting M2 polarization or promoting a shift back toward an M1 phenotype attenuate collagen accumulation and improve lung architecture (He et al., 2021; Zhang et al., 2020). Consequently, understanding the molecular cues governing M2 infiltration and polarization has emerged as a promising avenue for develo therapies that modulate maladaptive tissue remodeling.

不同类型的研究结论共用同一组Citation，具体claim与单篇文献的对应关系未区分

References

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