豆包结果：

Current Evidence

Accumulating evidence demonstrates that macrophage M1/M2 polarization dynamics and M2 macrophage infiltration play central roles in the pathogenesis of pulmonary fibrosis by driving aberrant tissue remodeling and extracellular matrix (ECM) deposition. In the early inflammatory phase following alveolar epithelial injury, classically activated M1 macrophages predominate, secreting pro-inflammatory cytokines (TNF-α, IL-1β, IFN-γ) that initiate innate immune responses and tissue damage; however, as injury persists, a phenotypic shift toward alternatively activated M2 macrophages occurs, and these M2-polarized cells accumulate preferentially in fibrotic foci where they secrete potent profibrotic mediators including TGF-β, PDGF, IL-4, IL-13, and CTGF, which directly stimulate fibroblast proliferation, myofibroblast differentiation, and excessive collagen production, thereby perpetuating pathological tissue remodeling rather than physiological wound resolution PMC. Single-cell transcriptomic and histopathological analyses of human idiopathic pulmonary fibrosis (IPF) lungs and murine bleomycin-induced fibrosis models have consistently confirmed that M2 macrophages, identified by markers such as CD206, Arg1, and Ym1, are the predominant macrophage subset in advanced fibrotic lesions, and their density correlates with disease severity and decline in pulmonary functionPMC. Notably, the M1-to-M2 transition is not merely a marker of disease progression but a pathogenic driver: experimental depletion of M2 macrophages or reprogrammi ation toward an M1 phenotype ameliorates fibrotic tissue ereas adoptive transfer of M2 macrophages or factors that promote M2 polarization exacerbate fibrosisPMC. This body of work establishes dysregulated macrophage polarization as a critical mechanistic node linking chronic inflammation to progressive fibrotic tissue remodeling in the lung, positioning macrophage phenotypic modulation as a promising ther for pulmonary fibrosis.

具体科学论述仅标注“PMC”，未注明对应文献

正文仅以“PMC”标注来源，与文末8篇References未建立明确对应

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Reference信息错配：该题名对应论文实际发表于Cell & Bioscience 14:140，作者、期刊及DOI等关键信息不符

Reference信息错误：该DOI对应Jiang et al.，Cell Death Discovery 10:487，并非Wang et al., 10:234

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