ChatGPT 结果：

Accumulating evidence indicates that macrophage polarization is closely associated with the initiation and progression of pulmonary fibrosis. Although the conventional M1/M2 classification represents a simplified framework for the spectrum of macrophage activation states observed in vivo, classically activated M1 macrophages are generally associated with pro-inflammatory responses, whereas alternatively activated M2 macrophages exhibit reparative and, under persistent injury, profibrotic properties [1,2]. In fibrotic lungs, macrophage populations progressively acquire M2-like features, characterized by increased expression of markers such as CD206 and arginase-1 (Arg1), and experimental studies have demonstrated prominent infiltration and accumulation of M2 macrophages during the fibrotic phase following bleomycin-induced lung injury [3–5]. Consistently, alveolar macrophages from patients with fibrotic interstitial lung diseases display increased CD206 expression and enhanced production of M2-associated chemokines, including CCL17, CCL18, and CCL22 [6]. Functionally, M2-polarized macrophages can promote fibroblast activation, myofibroblast differentiation, extracellular matrix deposition, and tissue remodeling through the release of profibrotic mediators such as transforming growth factor-β1 (TGF-β1), interleukin-13 (IL-13), and other growth factors [3–5,7]. Moreover, single-cell transcriptomic analyses of human idiopathic pulmonary fibrosis (IPF) lungs have revealed distinct profibrotic macrophage populations enriched for SPP1 and extracellular-matrix-remodeling gen fibrosis-associated macrophages encompass he ding beyond the classical M1/M2 dichotomy [8]. Collectively, these findings support a close relationship between sustained M2-like macrophage infiltration, aberrant macrophage–stromal cell communication, and pathological tissue remodeling during pulmonary fibrosis.

多个不同研究结论合并引用，具体claim与单篇文献的对应关系未进一步区分

References

1. Murray PJ, Allen JE, Biswas SK, et al. Macrophage activation and polarization: nomenclature and experimental guidelines. *Immunity*. 2014;41(1):14–20. doi:10.1016/j.immuni.2014.06.008.
2. Kishore A, Petrek M. Roles of macrophage polarization and macrophage-derived miRNAs in pulmonary fibrosis. *Front Immunol*. 2021;12:678457. doi:10.3389/fimmu.2021.678457.
3. He C, Ryan AJ, Murthy S, Carter AB. Accelerated development of pulmonary fibrosis via Cu,Zn-superoxide dismutase-induced alternative activation of macrophages. *J Biol Chem*. 2013;288(28):20745–20757. [This mechanistic literature is discussed in the pulmonary-fibrosis macrophage evidence base; for strict bibliography verification before submission, I recommend checking the journal record.]

Reference仍需用户自行核验，生成后未闭合文献核查链路

4. Hou J, Shi J, Chen L, et al. M2 macrophages promote myofibroblast differentiation of LR-MSCs and are associated with pulmonary fibrogenesis. *Cell Commun Signal*. 2018;16:89.
5. Ballinger MN, Newstead MW, Zeng X, et al. IRAK-M promotes alternative macrophage activation and fibroproliferation in bleomycin-induced lung injury. *J Immunol*. 2015;194(4):1894–1904.
6. Pechkovsky DV, Prasse A, Kollert F, et al. Alternatively activated alveolar macrophages in pulmonary fibrosis—mediator production and signal transduction. *Clin Immunol*. 2010;137(1):89–101. doi:10.1016/j.clim.2010.06.017.
7. Li D, Guabiraba R, Besnard A-G, et al. IL-33 promotes ST2-dependent lung fibrosis by the induction of alternatively activated macrophages and innate lymphoid cells in mice. *J Allergy Clin Immunol*. 2014;134(6):1422–1432.e11.
8. Habermann AC, Gutierrez AJ, Bui LT, et al. Single-cell RNA sequencing reveals profibrotic roles of distinct epithelial and mesenchymal lineages in pulmonary fibrosis. *Sci Adv*. 2020;6(28):eaba1972. The associated single-cell analysis identifies an IPF-associated macrophage archetype with increasing **SPP1** expression and enrichment of ECM-remodeling genes including *SPARC*, *CTSK*, and *MMP9*.

部分Reference未提供DOI，文献定位仍需额外检索

以上结果来自 **GPT instant 5.6-Sol**