LetPub 结果：

具体实验结论直接对应Citation [2]

【段落撰写结果】

Current evidence indicates that macrophage responses in pulmonary fibrosis are heterogeneous but consistently implicate a shift toward profibrotic programs enriched in alternatively activated, M2-like populations rather than a simple expansion of resident macrophages alone [1] [2] [3]. In bleomycin models, depletion of lung macrophages or Ly6C^hi monocytes attenuates collagen accumulation and fibrosis scores, while reducing markers of alternative activation such as Ym1 and arginase-1, supporting a functional link between recruited monocyte/macrophage compartments and fibrogenesis [2]. Concordantly, monocyte-derived alveolar macrophages acquire profibrotic transcriptional programs during fibrotic phases and their selective deletion ameliorates lung fibrosis, whereas tissue-resident alveolar macrophages appear less directly pathogenic in this context [1]. Several stud at M2-polarized macrophages contribute to tissue rem g myofibroblast differentiation and fibroblast activation; for example, recruited macrophages in fibrotic lungs were predominantly M2, M2 cells enhanced LR-MSC-to-myofibroblast differentiation through Wnt/β-catenin signaling, and C1q^+ interstitial macrophages localized around fibrotic nodules with profibrotic gene expression [4] [5]. Mechanistically, extracellular matrix–integrin β4β1–Rac2 signaling and CCL1-driven CCR8/AMFR signaling have each been shown to promote M2 polarization and fibrosis, underscoring that macrophage polarization is actively regulated during disease progression rather than merely reflecting injury [6] [7].

不同研究发现分别匹配具体Citation

【参考文献】

1. Monocyte-derived alveolar macrophages drive lung fibrosis and persist in the lung over the life span

作者：Alexander V. Misharin; Luisa Morales-Nebreda; Paul A. Reyfman; Carla M. Cuda; James M. Walter; Alexandra C. McQuattie-Pimentel; Ching-I Chen; Kishore R. Anekalla; Nikita Joshi; Kinola J.N. Williams; Hiam Abdala-Valencia; Tyrone J. Yacoub; Monica Chi; Stephen Chiu; Francisco J. Gonz á lez-Gonz á lez; Khalilah L. Gates; Anna P. Lam; Trevor T. Nicholson; Philip Homan; Saul Soberanes; Salina Dominguez; Vince K. Morgan; Rana Saber; Alexander Shaffer; Monique Hinchcliff; Stacy A. Marshall; Ankit Bharat; Sergejs Berdnikovs; Sangeeta Bhorade; Elizabeth T. Bartom; Richard I. Morimoto; William E. Balch; Jacob I. Sznajder; Navdeep S. Chandel; G ö khan M. Mutlu; Manu Jain; Cara J. Gottardi; Benjamin D. Singer; Karen M. Ridge; Neda Bagheri; Ali Shilatifard; G. R. Scott Budinger; Harris Perlman

期刊：The Journal of Experimental Medicine (2017)

DOI：https://doi.org/10.1084/jem.20162152

文章要点：Little is known about the relative importance of monocyte and tissue-resident macrophages in the development of lung fibrosis. We show that specific genetic deletion of monocyte-derived alveolar macro...

正文Citation与Reference编号对应，文献来源清晰可追溯

2. Ly6Chi Monocytes Direct Alternatively Activated Profibrotic Macrophage Regulation of Lung Fibrosis
作者：Michael Gibbons; Alison C. MacKinnon; Prakash Ramachandran; Kevin Dhaliwal; Rodger Duffin; Alexander Phythian-Adams; Nico van Rooijen; Christopher Haslett; Sarah Howie; A. John Simpson; Nikhil Hirani; Jack Gauldie; John P. Iredale; Tariq Sethi; Stuart J. Forbes
期刊：American Journal of Respiratory and Critical Care Medicine (2011)
DOI：https://doi.org/10.1164/rccm.201010-1719oc

文章要点：RATIONALE: Idiopathic pulmonary fibrosis (IPF) is a devastating disease. Antiinflammatory therapies, including corticosteroids, are of no benefit. The role of monocytes and macrophages is therefore co...

3. Dynamic atlas of immune cells reveals multiple functional features of macrophages associated with progression of pulmonary fibrosis
作者：Jiaoyan Lv; Haoxiang Gao; Jie Ma; Jiachen Liu; Yujie Tian; Chunyuan Yang; Mansheng Li; Yue Zhao; Zhimin Li; Xuegong Zhang; Yunping Zhu; Jianhong Zhang; Li Wu
期刊：Frontiers in Immunology (2023)
DOI：https://doi.org/10.3389/fimmu.2023.1230266

文章要点：This study investigates the role of immune regulation in the progression of idiopathic pulmonary fibrosis (IPF). Through single-cell profiling, the study reveals the distinct components of immune cell...

4. M2 macrophages promote myofibroblast differentiation of LR-MSCs and are associated with pulmonary fibrogenesis
作者：Jiwei Hou; Jingyan Shi; Ling Chen; Zhongyang Lv; Xiang Chen; Honghui Cao; Zou Xiang; Xiaodong Han
期刊：Cell Communication and Signaling (2018)
DOI：https://doi.org/10.1186/s12964-018-0300-8

文章要点：BACKGROUND: Idiopathic pulmonary fibrosis (IPF) is a devastating disease characterized by the histopathological pattern of usual interstitial pneumonia and is associated with a high mortality rate. Re...

提供DOI及文章要点，
可直接定位原文核查

5. Profibrotic properties of C1q+ interstitial macrophages in silica-induced pulmonary fibrosis in mice
作者：Tatsuro Ogawa; Shigeyuki Shichino; Satoshi Ueha; Kunia Bando; Kouji Matsushima
期刊：Biochemical and Biophysical Research Communications (2022)

DOI：https://doi.org/10.1016/j.bbrc.2022.02.037

文章要点：This study revealed the role of interstitial macrophages (IMs) in pulmonary fibrosis (PF) and their potential as a therapeutic target. IMs were found to have pro-fibrotic and anti-inflammatory propert...

6. Rac2 is required for alternative macrophage activation and bleomycin induced pulmonary fibrosis; a macrophage autonomous phenotype
作者：Shweta Joshi; Alok R. Singh; Simon S. Wong; Muamera Zulcic; Min Jiang; Annie Pardo; Moisés Selman; James S. Hagood; Donald L. Durden
期刊：PLoS ONE (2017)
DOI：https://doi.org/10.1371/journal.pone.0182851

文章要点：Idiopathic pulmonary fibrosis (IPF) is a chronic lung disease characterized by cellular phenotype alterations and deposition of extracellular matrix proteins. The alternative activation of macrophages...

7. The chemokine CCL1 facilitates pulmonary fibrosis by promoting macrophage migration and M2 polarization
作者：Suosi Liu; Suosi Liu; Ziying Zhang; Yu Wang; Yu Zhang; Jiali Min; Xia Li; Shanshan Liu; Shanshan Liu
期刊：International Immunopharmacology (2023)
DOI：https://doi.org/10.1016/j.intimp.2023.110343

文章要点：This study demonstrates that CCL1 plays a crucial role in macrophage M2 polarization in pulmonary fibrosis. It interacts with AMFR and CCR8 receptors to promote the formation of macrophage M2 phenotyp...