Lung CD4+ T-cells in patients with lung fibrosis produce pro-fibrotic interleukin-13 together with interferon-γ

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To the Editor:

Progressive fibrosing interstitial lung diseases (PF-ILD) have poor prognosis and survival, and their pathogenesis is not well understood [1]. Mechanistically, lung fibrosis is thought to result from distorted wound-healing following tissue insults and inflammation, leading to scar formation by excess deposition of extracellular matrix proteins and destruction of lung architecture [2]. The fibrotic process is complex, and CD4+ T-cells are probably involved through their production of a wide range of cytokines and growth factors that promote fibroblast proliferation and differentiation, collagen production, and stimulate production of pro-fibrotic mediators by tissue macrophages [3]. However, CD4+ T-cells in PF-ILD are poorly characterised. To this end, we performed a detailed analysis of phenotype, cytokine production and clonality of T-cells from the lungs (bronchoalveolar lavage (BAL)) of PF-ILD patients. We found that BAL from PF-ILD lungs contained high numbers of clonally expanded CD4+ T-cells that produced an unusual combination of interferon (IFN)-γ and pro-fibrotic interleukin (IL)-13. Such cells were not found in patient blood or in control BAL samples.