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Note: The file you are trying to download: Microsoft Office Word Viewer-129.exe has been replaced by a new version, click the Download button below.Idiopathic Pulmonary Fibrosis (IPF) is a progressive and lethal fibroblastic interstitial lung disease (ILD) with high mortality. This disease has a typical clinical course where it rapidly progresses with respiratory failure and death within two years of diagnosis. Although the pathogenesis of the disease is poorly understood, it is known that an epithelial-myofibroblast transition plays a major role in the progression of this disease. The most commonly affected cells in the IPF lung are alveolar epithelial type II (AE2) cells, which play a key role in the control of the alveolar architecture. Epithelial senescence has been shown to play an important role in the pathogenesis of IPF. The central hypothesis of this proposal is that pulmonary fibrosis in IPF is induced by AE2 cell dysfunction and subsequent altered interactions between these cells and the surrounding myofibroblasts. We will pursue this goal through the following specific aims. Aim 1. To determine whether activation of the Notch signaling pathway and/or epithelial senescence are involved in the pathogenesis of pulmonary fibrosis in IPF. We will investigate a) whether the activation of Notch signaling is associated with increased ILD lung disease severity in human IPF; and b) whether the activity of the Notch pathway is altered during AE2 cell senescence; and c) whether AE2 cell dysfunction induces myofibroblast senescence and subsequent apoptosis via Notch pathway activation. Aim 2. To determine whether TGF-? is involved in the induction of pulmonary fibrosis in IPF. We will investigate a) whether TGF-? is produced by AE2 cells and myofibroblasts in IPF; and b) whether TGF-? promotes the epithelial-myofibroblast transition and IPF disease severity. Aim 3. To determine whether the chemokine IL-33 is involved in the induction of pulmonary fibrosis in IPF. We will investigate a) whether IL-33 is produced by AE2 cells and by myofibroblasts in IPF; and b) whether IL-33 promotes the epithelial-myofibroblast transition and IPF disease severity. PUBLIC HEALTH RELEVANCE: Idiopathic pulmonary fib

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