Gastrin-releasing peptide drives pulmonary fibrosis through effects on lung fibroblasts

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Video Byte

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Abstract

Idiopathic pulmonary fibrosis (IPF) is a complex lung disease that results in scarring of the lungs, making it hard to breathe. Many signaling molecules are involved in the pathogenesis of IPF, including growth factors, chemokines, and cytokines. Unfortunately, current treatments cannot completely reverse scarring, suggesting that unknown pathways may be involved. One potential pathway is through gastrin-releasing peptide (GRP) released from cells in the lungs. A new study evaluated the role of GRP in pulmonary fibrosis development using two different human cell lines. In MRC5 lung fibroblasts, GRP signaling promoted differentiation into myofibroblasts, while in A549 adenocarcinoma cells, it primarily caused proliferation, both of which could drive scarring in pulmonary fibrosis. More research will investigate the role of GRP in vivo, but the results suggest that GRP-mediated signaling may be an ideal treatment target to prevent fibrosis, giving new hope to patients struggling with fibrotic lung disease.