Editorial

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FGF-1 Acts as an Antifibrogenic Mediator in Human Lung Fibroblasts by Activating Several Signaling Pathways

Alekhya Thirunahari*

Department of Biotechnology, Osmania University, Hyderabad, Telangana, India

***Corresponding author:**
Alekhya Thirunahari

thirunaharialekhya151315@gmail.com

Department of Biotechnology, Osmania University, Telangana, India.

Tel: 551832291000

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Editorial

Fibroblasts are the most common mesenchymal cells seen in connective tissues, and they synthesise, secrete, and turnover a variety of ECM components, including structural proteins, adhesion proteins, glycosaminoglycans, and proteoglycans. Fibroblasts contribute actively in idiopathic pulmonary fibrosis (IPF), a common interstitial lung disease, and are involved in all ECM physiological and pathophysiological operations (ILD). IPF is a progressive, irreversible, and fatal lung disease defined by the accumulation of activated fibroblast and myofibroblast subpopulations in localised areas. These cells undergo active and aberrant ECM remodelling and participate in their growth, eventually resulting in an excessive accumulation of ECM and irreversibly modifying lung histo-architecture.

The downregulation of matrix metalloproteinase-1 (MMP-1) and the overexpression of collagen and alpha-smooth muscle actin (-SMA), a hallmark of myofibroblast development, are all mediated primarily by TGF-1 stimulation, which is known as the main profibrogenic cytokine in IPF. As a result, fibroblast and myofibroblast subpopulations play a key role in fibrotic disorders like IPF, because these cells are involved in ECM turnover and may have fibrogenic activity. In fibroblasts derived from human lung fibroblasts, however, we discovered that the acidic fibroblast growth factor (FGF-1) coupled with heparin (FGF-1/H) chains have antifibrogenic effects, antagonising TGF-1's profibrogenic action (HLF).

Upregulation of MMP-1, downregulation of collagen I, and TGF-1-induced -SMA are some of the effects observed in HLF in vitro. Similar results were seen in a pulmonary fibrosis model in female Sprague-Dawley rats in vivo, where lung fibrosis was reduced when adenoviral vectors were employed to deliver extended transient overexpression of FGF-1 (AdFGF-1) and TGF-1 (AdTGF-1). In this paradigm, FGF-1 prevented and treated TGF-1-induced lung fibrosis by decreasing myofibroblast development and alveolar epithelial cell proliferation, downregulating TGF-1, and degrading transforming growth factor receptor type 1 (TBRI) via suppression of Smad2/3 phosphorylation.

Furthermore, the signalling pathways activated by FGF-1/H to upregulate MMP-1 and downregulate collagen I and -SMA are still poorly understood. As a result, we looked into the signalling pathways activated by FGF-1/H in HLF that cause MMP-1 to increase while collagen I and TGF-1-induced -SMA production decreases. We predicted that FGF-1/H stimulates signalling pathways that regulate MMP-1 and collagen I production or block the increase of -SMA in HLF generated by TGF-1.

The development of the current work took into account a variety of factors. The fact that the physiopathogenetic mechanisms underlying IPF are complicated, including multiple physiological mediators such as cytokines, chemokines, interleukins, and growth factors, which in turn activate multiple signalling pathways such as MEK, JNK, p38, pI3K, and TGF-/Smad. The latter stimulates the subpopulations of fibroblasts, myofibroblasts, and type II cells that are found in IPF, a non-inflammatory condition. Furthermore, FGF-1 can activate all of these pathways regardless of cellular type by engaging with all of the FGF family's receptors (FGFR1-4), thereby modulating the ECM's metabolic turnover.

In addition, FGF-1/anti-fibrous H's properties, which have been demonstrated both in vitro and in vivo, can reduce collagen expression, particularly type I collagen, which is the most abundant protein in fibrous lung tissue. It also raises the expression of MMP-1, which is the first enzyme to degrade type I fibrillar collagen in its natural state. It inhibits the expression of -SMA, which is required for the transformation of fibroblasts into myofibroblasts. As a result, we wanted to know which of these signalling pathways in fibroblasts were triggered to boost the expression of these three proteins. To accomplish this, we employed a cellular model to learn more about the involvement of FGF-1 in the pathophysiology of IPF. Patients

with this illness typically die 3 to 5 years following the onset of respiratory symptoms, emphasising the importance of this study. We anticipate that fresh research will be created as a result of combining the findings presented here with existing information on FGF-1's anti-fiber potential in order to establish a viable treatment for this disease.