

A MATHEMATICAL MODEL FOR THE PROGRESSION OF IDIOPATHIC PULMONARY FIBROSIS AND ITS POTENTIAL TREATMENTS

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ABSTRACT. Idiopathic Pulmonary Fibrosis (IPF) is a chronic lung disease marked by accumulation of scar tissue along the alveolar wall. The excessive buildup of extracellular matrix (ECM) components impedes gas exchange between the external environment and the capillary bed, thus creating a shortage of oxygen supply to the body. The prevailing theory behind the onset of IPF points to aberrant reepithelialization and abnormal myofibroblast behavior. The persistence of myofibroblasts at the fibroblast foci is the main source of collagen, proteoglycans, and other proteins that form the scar tissue characteristic of IPF. We present a mathematical model to describe the process by which collagen deposits in the microenvironment of a wound site in an alveolus. Our model consists of a system of coupled, non-linear partial differential equations that describe the interplay between fibroblasts, myofibroblasts, and chemical species important in the progression of IPF. The results of numerical simulations exhibit essential features of developing IPF such as high fibroblast concentrations at the wound site, imbalance of matrix metalloproteinases (MMPs) and tissue inhibitors of proteinases (TIMPs), and the buildup of collagen in the alveolar space. Finally, we performed simulations to emulate the effects of proposed treatments such as Pirfenidone, Interferon- γ 1b, and HMG-CoA.