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DIFFERENT CHANGES IN THE SMALL INTESTINE IN PULMONARY FIBROSIS. THE BODY'S RESPONSE TO EXPERIMENTAL PULMONARY FIBROSIS

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Abstract
Idiopathic pulmonary fibrosis (IPF) is a devastating disease characterized by severe and progressive scar formation in the gas-exchange regions of the lung. Despite years of research, therapeutic treatments remain elusive and there is a pressing need for deeper mechanistic insights into the pathogenesis of the disease. In this article, we review our current knowledge of the triggers and/or perpetuators of pulmonary fibrosis with special emphasis on the alveolar epithelium and the underlying mesenchyme. In doing so, we raise a number of questions highlighting critical voids and limitations in our current understanding and study of this disease.

Keywords: epithelium, lung fibrosis, myofibroblast.

PULMONARY FIBROSIS IS A GENERAL term used to describe an increased accumulation of extracellular matrix in the distal lung, rendering the lung stiff and compromising its ability to facilitate normal gas exchange. Patients typically present with the insidious onset of shortness of breath with exertion as the disease often goes unnoticed in its early stages. Pulmonary fibrosis can be associated with a number of underlying diseases (such as connective tissue/rheumatologic disease) or environmental exposures (asbestosis), or it can be idiopathic in nature. Idiopathic pulmonary fibrosis (IPF) is the most common form of fibrotic lung disease with a prevalence of 14.0–42.7 cases per 100,000 individuals in the United States (depending on the case definition used) and a median survival of 2.5–3.5 yr . It is viewed as a disease of aging, with the median age at diagnosis being in the mid-60s. There are few effective therapies for IPF short of lung transplant . Because a pharmacologic therapy capable of halting or at least slowing the progression of the disease has been elusive, there are intense efforts to better understand the factors that trigger and perpetuate this disease. As **The goal** - we begin to unravel this mystery, it is becoming clear that important clues lie in the complex cross talk that exists between the alveolar epithelium and the many cell types in the neighboring mesenchyme.

Conclusions

In this article, we review our current knowledge of triggers and/or perpetuators of pulmonary fibrosis with special emphasis on the alveolar epithelium. We present new ideas from mouse models and lineage-tracing studies that address the potential cell types responsible for generating the histology characteristic of IPF. We review current knowledge of the origins of

pathologic myofibroblasts in the lung and provide some additional hypotheses. Throughout the article, we raise a number of questions to highlight critical voids in our current understanding of the pathogenesis of this disease

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