What is IPF?

Pulmonary fibrosis (PF) is one of 200 interstitial lung diseases that cause scarring (fibrosis) in the lungs. The most common type of PF is idiopathic pulmonary fibrosis (IPF). Unlike other forms of PF that can be caused by immune-mediated diseases or the use of certain medications, the cause of IPF is unknown.

IPF causes lung tissue to become thickened, scarred and stiff, leading to lung damage and severe breathing difficulties. IPF is difficult to diagnose, associated with a poor prognosis and has limited treatment options that can only slow disease progression.

Researchers are working to develop a deeper understanding of IPF pathophysiology in order to develop innovative therapeutics that can halt or reverse lung fibrosis.

Lysophosphatidic acid (LPA) in IPF

Identifying the Culprits of Fibrosis

• Several biological targets have been implicated in the development of fibrosis in IPF including lysophosphatidic acid (LPA). LPA production is stimulated in response to lung cell injury, and can activate several receptors, including LPA. LPA receptor activation promotes pro-fibrotic processes, including the generation and accumulation of fibroblasts. Levels of LPA are elevated in the lungs of individuals with IPF. Preclinical studies antagonizing – or blocking – LPA receptors report reduced pulmonary fibrosis development, establishing the potential of LPA receptors as a clinical target for IPF.

Research Implications

• Therapeutics that block the LPA receptor have the potential to address the altered fibrotic processes observed in IPF.

Bristol-Myers Squibb is currently investigating a LPA receptor antagonist for the treatment of IPF. Learn more about our work in fibrosis by visiting: www.bms.com/researchers-and-partners/areas-of-focus.html