# Study finds key regulator in pulmonary fibrosis

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A Yale-led research team has identified an important enzyme that could lead to new therapies for a chronic fatal lung disease that affects hundreds of thousands in the United States each year.

Idiopathic pulmonary fibrosis (IPF) causes scar tissue inside the lungs, and patients may experience coughing and progressive shortness of breath. There is no cure for IPF. In a new study, the research team led by Yale Professor of Medicine Naftali Kaminski and by Marie Curie Fellow Dr. Argyrios Tzouvelekis explored the role of SHP2, an enzyme that removes phosphates from proteins and thus renders them inactive. The researchers found that the enzyme was low in lungs of humans with the disease.

In both cell culture and animal models of pulmonary fibrosis, the researchers found that suppression of the enzyme triggered pro-fibrotic changes in the lung, while increased enzyme activity prevented fibrosis. The findings suggest that SHP2 is an essential regulator and that potentially enhancing the activity or expression of it could provide a new therapeutic strategy for this deadly disease.

**More information:** Argyrios Tzouvelekis et al. SH2 Domain-containing Phosphatase-SHP-2 is a Novel Anti-fibrotic Regulator in Pulmonary Fibrosis, *American Journal of Respiratory and Critical Care Medicine* (2016). DOI: 10.1164/rccm.201602-0329OC