Targeting hyaluronan, CD44 and lung fibroblast invasion to block fibrogenesis

Duke File (IDF) Number

IDF #:T-003676

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Value Proposition

Idiopathic pulmonary fibrosis (IPF) is the most common type of pulmonary fibrosis. Scarring causes stiffness in the lungs and makes it difficult to breathe. Currently, more than 80,000 adults in the United States have IPF, and more than 30,000 new cases are diagnosed each year. The word "idiopathic" means it has no known cause, resulting in an unrelenting clinical treatment with characteristics reminiscent of cancer.

Technology

Researchers at Duke have revealed a new piece of the mechanism leading to severe lung fibrosis, elucidating a new potential therapeutic target for treating IPF. Specifically, the interaction between hyaluron synthase (HAS2) and cell-surface glycoprotein (CD44) has been revealed as having critical roles for regulating IPF. The inhibition of HAS2 abrogated the invasive fibroblast phenotype, impeded myofibroblast accumulation, and inhibited the development of lung fibrosis. Additionally, CD44 treated with a blocking antibody reduced lung fibrosis in mice *in vivo*. Finally, fibroblasts isolated from patients with IPF exhibited an invasive phenotype that was also dependent on both HAS2 and CD44. These revelations could lead to novel approaches to the treatment and diagnosis of disorders characterized by severe tissue fibrosis.

Advantages

- Offers new treatment strategies for idiopathic pulmonary fibrosis
- *In vivo* mouse treatment demonstrated