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Pulmonary fibrosis: Between a ROCK and a hard place

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Pulmonary fibrosis is a scarring or thickening of the lungs that causes shortness of breath, a dry cough, fatigue, chest discomfort, weight loss, a decrease in the ability of the lungs to transmit oxygen to the blood stream, and, eventually, heart failure. Cells known as myofibroblasts normally secrete materials that are required for wound healing; once the wound has closed, the cells disappear. In pulmonary fibrosis, the myofibroblasts stick around, continuing to secrete wound healing factors that cause fibrosis in the lungs.

In this issue of the Journal of Clinical Investigation, Yong Zhou and colleagues at the University of Alabama at Birmingham identified a mechanosensitive cellular signaling pathway in myofibroblasts that is activated by the hardening of tissue that has become fibrotic.

Activation of this pathway promotes myofibroblast survival and prevents the normal disappearance of these cells after completion of wound healing. The pathway is dependent on a protein known as ROCK.

Zhou and colleagues found that a drug that inhibits ROCK, fasudil, attenuates the pro-survival pathway and causes myofibroblasts to die. Further, fasudil treatment protected mice from injury-induced lung fibrosis.

These studies suggest that ROCK inhibitors could be used to treat pulmonary fibrosis. In a companion Attending Physician article, Dean Sheppard of the University of California, San Francisco, discusses the

feasibility of using ROCK inhibitors in a clinical setting.

More information: Inhibition of mechanosensitive signaling in myofibroblasts ameliorates experimental pulmonary fibrosis, *Journal of Clinical Investigation*, 2013. doi:10.1172/JCI66700
ROCKing pulmonary fibrosis, *Journal of Clinical Investigation*, 2013. doi:10.1172/JCI68417

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