Lung diseases share molecular signature

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The fibrotic lung diseases BPD (bronchopulmonary dysplasia) and IPF (idiopathic pulmonary fibrosis) affect preterm infants and older adults, respectively. Evidence suggests that these diseases involve dysregulation of lung repair processes, including Wnt signaling.

Jennifer Sucre, MD, and colleagues previously showed that a certain modification (phosphorylation) of the protein beta-catenin—part of the Wnt signaling pathway—was associated with fibrotic changes in BPD.

Now, using a library of normal and diseased human lung samples, they have demonstrated that two forms of phosphorylated beta-catenin have the same cellular pattern in early normal lung development and in BPD and IPF. The signature was associated with increased expression of a Wnt target gene.

The findings, reported in the April issue of the *American Journal of Pathology*, support the concept that repair of lung injury involves activation of signaling pathways that are important during development. They also suggest that targeting beta-catenin phosphorylation may be useful for the treatment and prevention of both BPD and IPF.

More information: Jennifer M.S. Sucre et al. A Shared Pattern of ?-Catenin Activation in Bronchopulmonary Dysplasia and Idiopathic Pulmonary Fibrosis, *The American Journal of Pathology* (2018). DOI: 10.1016/j.ajpath.2017.12.004

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