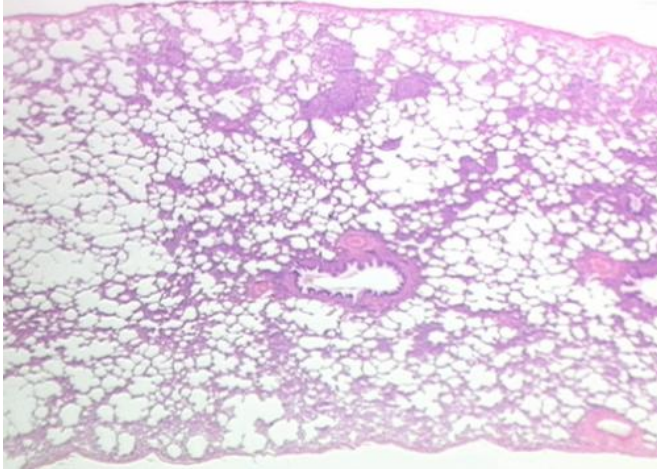


Clues to lung injury in preterm babies

6 March 2020, by Leigh MacMillan



More information: Jennifer M.S. Sucre et al. Hyperoxia Injury in the Developing Lung is Mediated by Mesenchymal Expression of Wnt5A, *American Journal of Respiratory and Critical Care Medicine* (2020). [DOI: 10.1164/rccm.201908-1513OC](https://doi.org/10.1164/rccm.201908-1513OC)

Provided by Vanderbilt University

Lung tissue. Credit: Rutgers University

Bronchopulmonary dysplasia (BPD)—a form of chronic lung disease—is a leading complication of preterm birth affecting infants born before 32 weeks gestation. Exposure to high levels of oxygen (hyperoxia) plays a role in BPD pathogenesis, but the precise molecular mechanisms remain uncertain.

Jennifer Sucre, MD, and colleagues [previously](#) demonstrated a pattern of increased Wnt signaling in human BPD tissue and hyperoxia models of BPD. They have now used three different model systems—3-D human organoids, mouse lung slices and a mouse in vivo model—to define mediators of activated Wnt signaling after hyperoxia injury.

They discovered that increased expression of Wnt5A in lung [connective tissue cells](#) contributes to the impaired alveolarization (alveoli are the sites of gas exchange) and septal thickening observed in BPD.

The findings, reported in the *American Journal of Respiratory and Critical Care Medicine*, suggest that precise targeting of Wnt5A in the lungs of preterm infants may prevent or reverse BPD.

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