# Impact of embryonic *Fgf10* expression deficiency on embryonic mouse lung development and repair following hyperoxia injury

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## Background

Bronchopulmonary dysplasia (BPD), a chronic lung disease of preterm infants, is characterized by impaired alveolar growth and pathologic vascularization.

## Aims

To investigate the impact of *Fgf10* expression deficiency on embryonic lung development and hyperoxia lung injury (BPD mouse model).

## Methods

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| 1) | Embryonic lungs (*Fgf10*$^{+/-}$ and *Fgf10*$^{+/+}$[WT]) were harvested at E12.5 and E18.5. Lung branches were quantified and H&E staining was performed. |
| 2) | After BrdU labeling lungs were harvest from embryos at E12.5. BrdU staining was performed on paraffin-embedded sections. |
| 3) | Transcriptomic analyses were performed by using whole lung RNA isolated at E18.5. |
| 4) | BPD mouse model: |

*Fgf10*$^{+/-}$ and *Fgf10*$^{+/+}$ *mice* were exposed to 85% $O_2$ from P0-P8. Lung morphometric analysis, IHC staining (α-Actin/vWF staining, SPC, E-cadherin, Ki67, TUNEL), gene expression analysis (RNA isolated from type II alveolar epithelial cells [AEC II]), FACS (epithelial/ mesenchymal progenitor cells, AEC I/ II) were performed at P3.

## Results

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| 1) | Embryonic *Fgf10* heterozygous lungs exhibit epithelial branching defects and decreased Fibroblast growth factor signaling. |
| 2) | Embryonic *Fgf10*$^{+/-}$ lungs show decreased epithelial proliferation. |
| 3) | Lungs of E18.5 *Fgf10*$^{+/-}$ embryos display structural defects and abnormal gene expression. |
| 4) | *Fgf10* heterozygous pups display increased sensitivity to hyperoxia exposure associated with significant structural lung defects. Transcriptomic analyses show epithelial defects linked to cell cycle dysregulation and increased Tgfb signaling. *Fgf10* heterozygous vessels are more sensitive to hyperoxia injury and exhibit a less muscularized phenotype. SPC staining shows significant decrease in SPC+ cells after hyperoxia injury. FACS analysis revealed an increase of AEC I on the expenses of AEC II (progenitor cell). |

## Conclusion

*Fgf10* deficiency leads to impaired embryonic lung development and death upon postnatal hyperoxia lung injury due to vulnerability of the epithelium.