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Evidence that Fgf10 offers therapeutic opportunities after hyperoxic lung injury in mice

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

Aims

To investigate the role of *Fgf10* in alveologenesis and during/after hyperoxic lung injury.

Methods

1) 10 weeks old $Fgf10^{+/-}$ mice (50% *Fgf10* expression compared to WT) in normoxic condition: Lung function and morphometric analysis.

2) BPD model:

a) $Fgf10^{+/-}$ and $Fgf10^{+/+}$ *mice* were exposed to 85% O_2 from P0-P8. Morphometric analysis and α-Actin/vWF staining were performed at P3.

b) $Rosa26^{rtTA/+}$;*tet(O)Fgf10* (gain-of-function) mice were exposed to 85% O_2 from P0-P8. From P9-P45 the pups were exposed to normoxia and fed either with normal food (control) or doxycycline food (experimental) to activate the transgene *Fgf10.* Morphometric analysis was carried out at P45.

3) Tolerance study: $Rosa26^{rtTA/+}$;*tet(O)Fgf10* and WT mice (both 10 weeks old) were exposed to doxycycline for 2 weeks. Then survival rate, histology, Ki67 and TUNEL staining were performed.

Results

1) $Fgf10^{+/-}$ mice under normoxic condition have worse lung function and lung structure compared to WT mice.

2) All $Fgf10^{+/-}$ mice die from hyperoxic injury due to increased lung injury and vascular malformation.

3) Overexpression of *Fgf10* after hyperoxic injury leads to improvement of lung structure compared to control group without overexpression.

4) *Fgf10* overexpression after hyperoxic injury does not increase mortality and side effects (weight loss, mucosal proliferation due to hypercellularity with no impact on apoptosis) are reversible.

Conclusion

Fgf10 attenuates hyperoxic lung injury, is well tolerated and should be further studied as a potential therapeutic for BPD.

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