EXPRESSION OF α -, β -, AND γ ENaC IN HUMAN FETAL AND NEWBORN LUNG

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Background: Airway epithelial sodium channel ENaC is critical to perinatal lung fluid clearance. Data on ENaC protein expression in developing human lung are limited and absent for infants with RDS or BPD.

Objective: To characterize the expression of α -, β -, and γ ENaC expression in lung tissue from human fetuses and newborns with and without RDS or BPD.

Methods: Lung tissue was obtained from autopsies (n = 23) from fetuses (GA 12 - 21 wks), preterm infants with (GA 25 - 28 wks) or without RDS (GA 23 - 25 wks), and infants with BPD (GA 24 - 30 wks at birth). The primary antibodies were rabbit polyclonal α ENaC (1:60 Sigma-Aldrich Inc, MO, US), β ENaC (1:2250 Aviva Systems Biology, CA, US) or γ ENaC (1:200 Lifespan Biosciences Inc, WA, US).

Results: In fetal lung developing respiratory, glandular epithelium stained with α -, β -, and γ ENaC from 12+3 wks GA. In preterm infants without RDS expression of α -, β -, and γ ENaC was apparent in single type II pneumocytes, while type I pneumocytes remained unstained. In RDS and BPD the expression of ENaC subunits in alveolar epithelium was largely absent but reactive, hyperplastic type II pneumocytes stained strongly for α - and β ENaC. Throughout development, and in infants with RDS or BPD α -, β -, and γ ENaC stained the bronchiolar epithelium.

Conclusions: In human fetal lung all three ENaC subunits were expressed during the pseudoglandular stage of lung development. Expression of ENaC subunits in bronchioles may indicate a role for conductive airways in perinatal lung liquid clearance.