CORD BLOOD STRESS HORMONES ASSOCIATE WITH GENE EXPRESSION OF AIRWAY EPITHELIAL SODIUM TRANSPORT-RELATED MOLECULES DURING THE EARLY POSTNATAL PERIOD

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Background. Lung fluid clearance is essential for the aeration and gas exchange during postnatal pulmonary adaptation. Key-players in the switch from fetal lung fluid secretion to permanent reabsorption are the apical epithelial sodium channel ENaC and basolateral Na-K-ATPase. Serum- and glucocorticoid-inducible kinase SGK1 mediates hormonal stimuli on ion transport and may thereby play a facilitatory role in lung fluid balance.
Objective. Our aim was to investigate whether the postnatal gene expression of molecules vital to airway epithelial sodium transport associates with the level of birth stress defined as concentrations of noradrenaline and cortisol in cord blood.
Methods. We included 71 term newborns born from normal pregnancies, 28 born by vaginal delivery and 43 by elective cesarean section. Cord blood samples were taken at birth and hormone concentrations were measured by liquid chromatography tandem mass spectrometry. We gathered nasal epithelial cell samples at the median age of 2 min, 1.3 h and 26 h for the quantitation of alpha-, beta-, gamma-ENaC, alpha1-, beta1-Na-K-ATPase and SGK1 mRNAs with RT-PCR.
Results. alpha-, beta-ENaC and alpha1-, beta1-Na-K-ATPase subunits correlated with noradrenaline concentrations in cord blood at 2 min of age ($r=0.332$, $p=0.012$; $r=0.399$, $p=0.002$ and $r=0.344$, $p=0.009$; $r=0.385$, $p=0.003$) and at 1.3 h of age ($r=0.581$, $p<0.001$; $r=0.515$, $p=0.002$ and $r=0.419$, $p=0.015$; $r=0.696$, $p<0.001$). At both timepoints, SGK1 correlated with noradrenaline and cortisol ($r=0.324$, $p=0.015$; $r=0.473$, $p=0.005$ and $r=0.329$, $p=0.010$; $r=0.406$, $p=0.014$). Na-K-ATPase beta1-subunit also correlated with cortisol concentrations at 2 min and 1.3 h ($r=0.292$, $p=0.024$ and $r=0.447$, $p=0.006$).
Conclusions. The association of ENaC, Na-K-ATPase, and SGK1 with cord blood noradrenaline and cortisol indicates that birth stress induces molecular mechanisms of lung fluid clearance essential for successful postnatal pulmonary adaptation in the newborn infant.