proliferation was detected using bromodeoxyuridine incorporation. Expression of SP-A to -D, adipocyte triglyceride lipase (ATGL), lyso-PC-acyltransferase (LPCAT) and the ATGL activator CGI-58 were measured with PCR.

Results: RhKGF, betamethasone and combination treatment increased SP-B expression throughout by +97-117%, +42-51% and +93-309% ($P < 0.001$), respectively. SP-C was increased by +35-48%, +30-44% and +74-108% ($P < 0.001$) after the respective treatments, while SP-A & -D were unchanged in immature lungs. All treatments increased the expression of ATGL (+50-75%), LPCAT (+15-20%) and CGI-58 (+5-10%) ($P < 0.05$). However, whereas rhKGF effects were accompanied by increased pneumocyte proliferation, betamethasone blocked both basic (d7) and rhKGF-induced (d7+d21) proliferation.

Conclusion: Short term medication of neonatal rats with rhKGF/Palifermin® increased the expression of genes relevant for surfactant function and metabolism. Actions were identical or superior to those of betamethasone, with combination treatment exerting maximal effects. However, while betamethasone effects were at the expense of lung anabolism/pneumocyte proliferation, rhKGF enhanced proliferation.

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FEMALE SEX STEROIDS MODULATE ALVEOLAR EPITHELIAL SODIUM TRANSPORT

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Background and aims: Sodium transport plays a crucial role in alveolar fluid clearance (AFC), where sodium enters the alveolar epithelial cells through apical sodium channels (ENaC) and is extruded by basolateral Na,K-ATPases. Women with acute respiratory distress syndrome have higher AFC and higher survival than males. Female sex steroids are supposedly responsible for these gender-related differences. Therefore, the aim of this study was to analyze the effects of estrogen (E2) and progesterone (P) on epithelial sodium transport.

Methods: Isolated alveolar epithelial cells from 18-19-day gestational age rat fetuses, grown in serum-free media supplemented with E2 and P were studied, involving Real-Time PCR analysis,

single-channel patch clamp, Ussing chamber measurements and Western blotting.

Results: rtPCR revealed an increase of α - and β -ENaC in media supplemented with E2 and P, where the effect on α -ENaC was most pronounced in P-rich media. The mRNA-level of γ -ENaC was not altered, but the Na-K-ATPase- β_1 subunit and the CFTR-mRNA expression were increased by E2 and P supplementation. Single-channel patch clamp analysis showed highly-selective and non-selective ENaC in the examined cells. The percentage of responsive patches increased from 45% in non-supplemented media to 91% in the presence of E2 and P. Short-circuit current (I_{sc}) measurement showed that the baseline, amiloride- and ouabain-sensitive I_{sc} were elevated by E2 and P in a dose-dependent manner.

Conclusions: These results demonstrate increased expression of epithelial transport proteins by female sex steroids. Furthermore, vectorial sodium transport was increased. The findings may explain the observed survival advantage.

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HYPEROXIA SUPPRESS ANTIOXIDANT ACTIVITY IN CULTURED PULMONARY ARTERIAL ENDOTHELIAL CELLS

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Background: Hyperoxia is one of the risks to cause BPD or ROP in preterm infants. It has been known to suppress the angiogenesis of vascular endothelial cells. However, the effect of hyperoxia to the antioxidant activity of vascular endothelial cells is not very clear.

Objective: To evaluate the effects of hyperoxia to the superoxide dismutase (SOD) and glutathione peroxidase (GPx) activities of cultured pulmonary arterial endothelial cells (PAEC).

Materials and methods: Primary fetal sheep (GA 125 to 140 days) pulmonary arterial endothelial cells (PAEC) were cultured under room air, 60%, and 80% oxygen. NO donor (DETA NONOate) was administered to the culture medium with the concentrations of 0, 0.01, 0.02, and 0.05mM. The