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%) und 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 ANTIOXIENZYME 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 antoxienzyme 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 administrated to the culture medium with the concentrations of 0, 0.01, 0.02, and 0.05mM. The

experiments were performed for 72 hours. The oxygen was re-charged every 12 hours, and the medium was changed every 24 hours. Apoptosis and cell numbers were measured every 24 hours. After 72 hours, the cells were collected and lysed. Protein concentrations, Cu/Zn-SOD and cellular GPx activities were measured by spectrophotometer.

Results: The SOD activity was highest under 60% oxygen concentration. However, the activity range was very large. The cellular GPx activity was highest under room air. The activity decreased under 60% and 80% oxygen concentrations. Exogenous nitric oxide may decrease the apoptosis percentage of PAEC under 60% oxygen concentration. However, it didn't affect antioxienzyme activity of PAEC under hyperoxic condition.

Conclusion: Hyperoxia suppressed cellular GPx activity in PAEC. This effect might contribute to the development of BPD in preterm infants.

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PULMONARY OXYGEN UPTAKE CAPACITY IS REDUCED IN SCHOOL-AGED CHILDREN AFTER BRONCHOPULMONARY DYSPLASIA (BPD)

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Background: Disturbed pulmonary vascularization and alveolar development are hallmarks of BPD in the era of surfactant therapy and antenatal steroids. Data on long-term functional consequences of these changes are scarce. The objective of our study was to assess aerobic fitness of formerly very low birth weight infants (VLBW) with and without BPD compared with children born at term and to identify factors accounting for group differences.

Methods: Fourty children aged 7.9-12.9 years volunteered for this study. Ten children had BPD, 15 were VLBW without BPD (non-BPD) and 15 formerly term infants served as controls (CON). Aerobic fitness was assessed as peak oxygen uptake, allometrically adjusted for body mass (VO_2peak_{adj}), during an incremental cycling task to volitional fatigue. Physical activity (PA) was expressed as relative time in moderate-and-vigorous PA (MVPA).

Results: The 3 groups did not differ in anthropometric indices. Although heart rate and respiratory exchange ratio at the end of exercise were identical among groups, VO_2peak_{adj} was lower in the BPD group (1329±149 ml/min) compared with non-BPD (1526±152 ml/min) and CON (1536±197 ml/min). MVPA was lower in VLBW (BPD 1.7±1.1%, non-BPD 2.2.±0.9%) compared with CON (4.4±1.1%). The difference in aerobic fitness was not explained by differences in PA. Several lung function parameters were decreased in BPD. Only adjustment for diffusion capacity abrogated the association of BPD with decreased aerobic fitness.

Conclusions: The impaired diffusion capacity and aerobic fitness of school-aged children with BPD indicate a long-term functional consequence of disturbed pulmonary vascular and alveolar development in the disease.

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IMPAIRED SURFACTANT PROTEIN B SYNTHESIS IN INFANTS WITH CONGENITAL DIAPHRAGMATIC HERNIA

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Background: Whether congenital diaphragmatic hernia (CDH) is associated with surfactant deficiency or dysfunction is controversial.

Aims: To measure disaturated phosphatidylcholine (DSPC) and surfactant protein B (SP-B) synthesis and metabolism in infants with CDH.

Methods: DSPC and SP-B amounts and kinetics were studied in tracheal aspirates (TA) of 12 infants with CDH (BW 2978±447g, GA 38±2 wk) and in 8 GA-matched control infants (BW 3160±350g, GA 38±2 wk). Seventeen infants received a 24h infusion of ^{13}C-leucine, an i.v. bolus of ^2H_2O and 0.0625% of fluid intake as ^2H_2O every 12h over the next 48h. Three infants received only ^{13}C-leucine infusion.