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 antioxidant 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: Forty 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 ($\text{VO}_{2\text{peak}_{\text{adj}}}$), 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, $\text{VO}_{2\text{peak}_{\text{adj}}}$ 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 $1.7 \pm 1.1\%$, non-BPD $2.2 \pm 0.9\%$) compared with CON ($4.4 \pm 1.1\%$). 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 ± 447 g, GA 38 ± 2 wk) and in 8 GA-matched control infants (BW 3160 ± 350 g, GA 38 ± 2 wk). Seventeen infants received a 24h infusion of ¹³C-leucine, an i.v. bolus of ²H₂O and 0.0625% of fluid intake as ²H₂O every 12h over the next 48h. Three infants received only ¹³C-leucine infusion.

DSPC and SP-B were isolated from TA. DSPC amounts, ^2H -palmitate and ^{13}C -leucine enrichments were measured by gas chromatography mass spectrometry.

Results: DSPC amount and kinetics have been measured in 17 newborns, and SP-B kinetics in 18. DSPC median (interquartile range) fractional synthesis rate (FSR) was 5.0 %/day (3.5-8.5) in CDH patients vs 6.1 %/day (5.8-7.0) in controls ($p=0.31$). DSPC secretion time (ST) was 10.7h (6.9-11.5) vs 7.9h (5.2-14.0) ($p=0.65$).

SP-B FSR was 33.4 %/day (28.4-42.9) vs 52.0 %/day (40.0-83.4) ($p=0.03$); SP-B peak time was 26h (24-32) vs 18h (15-21) ($p=0.02$), both significantly lower in the CDH group. SP-B ST was 6.1h (4.0-9.9) vs 2.7h (1.1-7.1) ($p=0.24$), and half life 7.5h (5.2-9.1) vs 11.1h (5.8-17.3) ($p=0.33$).

Conclusions: Decreased and delayed SP-B synthesis was found in newborns with CDH compared to controls while DSPC kinetics was similar in both groups, which may contribute to severe respiratory failure in these patients.

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MUTATIONS OF SURFACTANT-RELATED GENES SP-B, SP-C AND ABCA3 IN UNEXPLAINED ALVEOLAR/INTERSTITIAL LUNG DISEASE IN NEWBORN AND INFANTS

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Aims: To determine the role of surfactant-related gene anomalies in neonatal/infantile interstitial lung disease.

Methods: 22 infants >32weeks/< 1year with unexplained progressive alveolar/interstitial lung disease. SP-B, SP-C and ABCA3 sequencing. Optical (OM) and electronical (EM) microscopy of biopsy/autopsy lung tissue. Surfactant proteins immunofluorescence.

Results: 13/22 infants (59.1%) had lamellar bodies (LB) ultrastructural abnormalities suggesting genetic surfactant deficiency. Eleven infants (50.0%) carried ABCA3 mutations.

-Five (22.7%) had bi-allelic mutations (2 homozygote frameshift, 3 double heterozygote missense),

showing small, dense LB with 1-2 denser cores and no normal LB on EM.

-Five (22.7%) had a single mutation (four missense, one intron), showing a mix of small and normal LB plus multivesicular bodies (MVB) on EM. On immunofluorescence, one had cytoplasmic mislocation of TTF-1 despite normal TTF-1 gene sequence, one had altered cleavage of proSP-B into SP-B, and one had reduced/misplaced alveolar capillaries.

-One infant (4.5%) carried a heterozygote SP-C mutation, plus a synonymous ABCA3 mutation, showing large, empty LB lacking normal surfactant layers on EM.

-One infant (4.5%) with normal LB + MVB had a heterozygote SP-B mutation.

-One infant (4.5%) with small dense LB (similar to homozygote ABCA3 mutants) had no mutation identified.

In the 9 remaining infants (40.9%), one had alveolar proteinosis, one had COX-deficiency, one had Niemann-Pick C disease, two had idiopathic pulmonary fibrosis, four remained cryptogenetic.

Conclusions: ABCA3 mutations are the most frequent abnormality. Although EM is indicative in most (92%) cases, molecular mechanisms are elusive in >50% of cases, likely involving complex, multiple surfactant-related gene interactions.

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ROLE OF CORD STRESS HORMONE LEVELS FOR DEVELOPING TRANSIENT TACHYPNEA OF NEWBORN IN CESAREAN DELIVERED LATE PRETERM AND TERM INFANTS

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Background and aims: Failure of adequate and timely clearance of fetal lung fluid results in transient tachypnea of newborn (TTN). Of the several factors that have been proposed to effect sodium reabsorption some have been investigated including glucocorticoids, oxygen, catecholamines, thyroid hormones, surfactant. The aim of this study was to investigate the role of cord stress hormones;