6 months of age. The formulas contained the same protein amount (13.1g/L) but different concentrations of α -lactalbumin and casein-glycomacropeptide: (1) standard formula (11% α -lactalbumin/14% casein-glycomacropeptide; n=10), (2) α -lactalbumin-enriched formula (25% α -lactalbumin/15% casein-glycomacropeptide; n=11), and (3) α -lactalbumin-enriched/casein-glycomacropeptide-reduced formula (25% α -lactalbumin/10% casein-glycomacropeptide; n=11). Ten exclusively breastfed infants were recruited as a reference. At 5.5 months of age, infants were given ^{58}Fe with one of their feeds. Blood samples were collected 14 days later for fractional iron absorption and serum ferritin determination.

Results: Mean ($\pm$ SD) iron absorption was $10.3\pm 7.0\%$ from standard formula, $8.6\pm 3.8\%$ from α -lactalbumin-enriched formula, and $9.2\pm 6.5\%$ from α -lactalbumin-enriched/casein-glycomacropeptide-reduced formula, with no difference among the three formula groups ($P=0.92$). Iron absorption from all three formulas was $9.4\pm 5.8\%$ compared to $12.9\pm 6.5\%$ from breastmilk ($P=0.73$). In the formula-fed infants, but not the breastfed infants ($P=0.51$), iron absorption was negatively correlated with serum ferritin ($P=0.01$), and was higher ($P=0.01$) in infants with serum ferritin $< 12\mu\text{g/L}$ ($16.4\pm 12.4\%$) compared to those with serum ferritin $\geq 12\mu\text{g/L}$ ($8.6\pm 4.4\%$).

Conclusions: α -Lactalbumin and casein-glycomacropeptide do not affect iron absorption from low-iron formula in healthy term infants. Low serum ferritin concentrations enhance iron absorption from infant formula.

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HYPOTHERMIA-TREATED INFANTS WITH HIE: MRI FINDINGS IN RELATION TO SHORT-TERM OUTCOME.

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Background and aims: Induced moderate hypothermia (HT) reduces neurodevelopmental disabilities in full-term infants with moderate/severe HIE. Pathological findings on MRI correlate to outcome.

We investigated whether pathological findings on MRI after HT treatment predicted short-term neuromotor outcome in HIE infants.

Methods: Between May 2006 and December 2009, 69 infants were treated with HT (33.5° C for 72 hours). MRI was performed on day 5-15 (median 8) in surviving infants. Patterns of injury were defined as: watershed (WS), basal ganglia/thalamus (BGT), or global(G) predominant pattern on MRI by two blinded readers. Motor functions were assessed at 4,12 and 24 months of age. All included infants had reached the age of 4-months.

Results: Out of 69 HT-treated infants 61 had a MRI examination; 3 parents did not consent and 5 infants died. Two infants were lost for the 4-months follow up. WS-pattern was seen in 5 (9%), BGT pattern in 11 (19%), and global lesions in 2 (3 %). Pathological 4-months outcome was found in 10/11 infants with BGT-pattern. None of the infants with WS or normal MRI had abnormal score on the Alberta Infant Motor Scale (AIMS) at 4 months of age. In the infants with global lesions, care was discontinued after the MRI examination.

Conclusion: Among asphyxiated infants treated with HT, only those with abnormalities in basal ganglia/thalamus on MRI showed pathologic short-term neuro-motor outcome.

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SURFACTANT DIPALMITOYL PHOSPHATIDYLCHOLINE KINETIC IS MARKEDLY IMPAIRED IN TERM NEWBORNS WITH PNEUMONIA

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Background and aims: The efficacy of exogenous surfactant treatment in term newborns with pneumonia is still undetermined. Aim of the study is to assess surfactant dipalmitoyl phosphatidylcholine (DPPC) kinetics in newborns with pneumonia using U^{13}C -DPPC as tracer.

Methods: Nineteen full-term newborns requiring mechanical ventilation were enrolled: 9 had severe

pneumonia and 10 no lung disease. Gestational age and birth weights were similar (38.3 ± 2.0 wks and 2782 ± 724 g). All newborns received intra-tracheally 2 mg kg^{-1} U^{13}C -DPPC mixed with 2 to 5 mg kg^{-1} of exogenous surfactant. We collected sequential tracheal aspirates from the start of the study (before the tracer administration) and isotopic enrichments of DPPC palmitate moieties were measured from serial tracheal aspirates by gas chromatography mass spectrometry. Data are expressed as median (interquartile range) and comparisons are performed by Mann-Whitney test. $p < 0.05$ was regarded as statistically significant.

Results: Median and interquartile range of DPPC pool size in newborns with pneumonia was 3 mg kg^{-1} (1.5-14.0) and 26.7 mg kg^{-1} (18.3-44.0) in control group. The difference was statistically significant ($p=0.041$). Median DPPC half life was 10.1 h (7.6-13.9) in pneumonia and 22.5 h (18.2-36.1) in controls. The difference was highly significant ($p=0.004$).

Conclusions: Term newborns with pneumonia exhibited a smaller pool size and shorter DPPC half life than controls suggesting a markedly faster surfactant turnover presumably triggered by neonatal pneumonia.

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NEW ANTI-INFLAMMATORY THERAPY FOR NEONATAL ACUTE LUNG INJURY: VARESPDLADIB (A-001) INHIBITS EX-VIVO SECRETORY PHOSPHOLIPASE

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Background: Secretory phospholipase A2(sPLA2) is crucial for surfactant catabolism and inflammation. We demonstrated high sPLA2 activity during infection related respiratory failure (IRRF), hyaline membrane disease (iRDS), RSV-triggered ARDS. sPLA2 is also responsible for lung damage during

meconium aspiration syndrome (MAS). Varespladib is the first new anti-inflammatory drug able to directly inhibit sPLA2 and it resulted efficacious in animals and promising in adults.

Aim: To test varespladib efficacy in an ex-vivo model of direct endotracheal administration in various types of neonatal acute lung injury.

Methods: 24 neonates affected by iRDS, 12 with IRRF, 5 with MAS have been subjected to non-bronchoscopic broncho-alveolar lavage according to European Respiratory Society advices. Supernatant has been subdivided in aliquots which have been added with various concentrations of varespladib or equivalent volumes of saline. Real time sPLA2 activity and total protein content have been measured. Enzyme activity has been corrected for dilutional factors and varespladib absorbance. Clinical data were registered up to the intensive care unit discharge.

Results: Varespladib reduces sPLA2 during iRDS(-20%; $p < 0.0001$), MAS(-23%; $p=0.04$), IRRF(-15%; $p=0.003$). Varespladib efficacy is higher when a lower capillary extravasation is present (ratio sPLA2 activity reduction/total proteins = 2.17 in iRDS;0.59 in IRRF,0.53 in MAS). IC50 has been calculated and varespladib efficacy correlates with the basal enzyme activity ($\rho=0.375$; $p=0.017$). No correlations with gestational age, birth weight, surfactant cumulative need, mechanical ventilation and oxygen therapy duration were found.

Conclusions: Varespladib efficaciously inhibits sPLA2 in an ex-vivo model of direct administration. This might have important anti-inflammatory effects and spare surfactant from its catabolism.

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REGULATION OF AGE-DEPENDENT TYPE II CELL EPITHELIAL MESENCHYMAL TRANSITION BEHAVIOR

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Background and aims: Epithelial- Mesenchymal Transition (EMT) is a process where epithelial cells acquire a mesenchymal cell phenotype. EMT is activated in association with tissue repair and remodeling after injury in multiple organs including