Background Recently bronchopulmonary dysplasia (BPD) occurs not infrequently without an exposure to hyperoxia and/or mechanical ventilation in very preterm infants. To simulate the recent pattern of BPD, novel animal model of BPD induced by inflammation alone is needed. We were to establish a rat model of BPD induced by postnatal systemic lipopolysaccharide (LPS) administration alone.

Methods Two days before delivery, 1 µg of LPS or vehicle (V) was injected into each amniotic sac, and after birth 0.25 mg/kg of LPS or V was injected into peritoneum of pups at P1, P3, and P5. At P7 and P14, bronchoalveolar lavage (BAL) and lung harvest were performed. BAL fluid (BALF) and peripheral blood (PB) were examined for blood cell counts and lung tissue was examined for morphometry, vascular density, neutrophil infiltration, and expressions of pro-inflammatory cytokines and angiogenic growth factors.

Results Postnatal systemic LPS significantly increased the neutrophil counts in PB, BALF and within the alveoli and expressions of pro-inflammatory cytokines and angiogenic growth factors at P7. When postnatal systemic LPS was preceded by intra-amniotic LPS administration, these findings were not observed. Postnatal systemic LPS led to significant disruption of alveolar and pulmonary vascular developments at P14.

Conclusions Early postnatal systemic LPS induced systemic and pulmonary pro-inflammatory responses and disrupted alveolar and pulmonary vascular developments. This rat model of new BPD induced by early postnatal inflammation *per se* without an exposure to hyperoxia can be used to test the effects of anti-inflammatory agents for BPD.

PS-211a TOTAL OXIDANT STATUS AND TOTAL ANTIOXIDANT CAPACITY IN AMNIOTIC FLUID OF LATE PRETERM INFANTS

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Background and aims Pregnancy represents a complex state in which the mother and the fetus both contribute to the oxidative stress and production of reactive oxygen species. The ability to buffer these is called total antioxidant capacity (TAC). We aimed to evaluate whether amniotic total oxidant status (TOS) and TAC can have a role in late preterm births after premature rupture of membranes (ROM).

Methods The study was a prospective observational trial. Group 1 (n = 30): late preterms with premature ROM, Group 2 (n = 30): term infants with prolonged ROM, Group 3 (n = 30): term infants born without prolonged ROM. Amniotic fluid and cord blood samples were collected during delivery and TOS (µmolH₂O₂ Eq/L) and TAC(mmol Trolox Eq/L) levels were measured.

Results Mean gestational ages were 35,3 ± 1,1; 39,29 ± 1,25; 38,7 ± 0,63 weeks respectively. Cord blood samples of the groups revealed no difference between any of the parameters checked (pH, pCO₂, HCO₃, lactate, Methb, TAC and TOS) (p > 0.05, for each). TAC levels of amniotic fluids (2,05 ± 0,60; 2,04 ± 0,5 and 21,89 ± 0,36) were also similar between groups (p > 0.05). But TOS levels of Group1 was significantly higher than Group2 (p < 0.05) and Group3 (p < 0.01). No significant difference was detected between Group 2 and 3.

Conclusion Similar cord blood levels of all parameters reflect the similar perinatal conditions of infants at birth. Similar TOS levels of term groups show that there is no close relation between TOS and infectious status. But on the other hand; significantly higher TOS levels of Group1 can be related with premature birth; which can lead us to antioxidant therapies to avoid premature birth.

Neonatal Sepsis

PS-212 EPIDEMIOLOGY OF NEONATAL SEPSIS IN SWITZERLAND – RESULTS FROM THE SWISS PAEDIATRIC SEPSIS STUDY

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Background and aims Neonatal infection is a major cause of morbidity and mortality. The ongoing Swiss Paediatric Sepsis Study prospectively evaluates the epidemiology of blood culture-proven sepsis in newborns and children in Switzerland.

Methods Newborn infants admitted to nine Swiss neonatal intensive care units (NICUs) and presenting with culture-proven sepsis between 9.2011–2.2014 were prospectively enrolled. Early-onset sepsis (EOS) was defined as infection occurring.

Results We identified 189 episodes of blood culture-proven sepsis in 186 patients. Thirty seven episodes were classified as EOS and 152 episodes were classified as LOS. Median gestational age at birth was 34 weeks for EOS, and 28 weeks for LOS. Mortality was 14% in EOS and 9% in LOS. Forty six percent of patients required mechanical ventilation during the sepsis episode, and 4% required catecholamine treatment for arterial hypotension.

Group B Streptococcus (GBS) and Escherichia coli were the most frequently isolated pathogens in EOS, accounting for 35% and 16% of episodes. Coagulase-negative staphylococci were the leading pathogens in LOS (36%), followed by Staphylococcus aureus (18%), Escherichia coli (16%), and GBS (11%). The proportion of hospital-acquired LOS due to Coagulase-negative staphylococci, Staphylococcus aureus and Escherichia coli varied between 10–68%, 0–45% and 0–30% in different NICUs.

Conclusions This national study confirms that neonatal sepsis continues to cause high morbidity and significant mortality. GBS is the most common cause of EOS. There are important differences in the aetiology of LOS in different institutions.

PS-213 EPIDEMIOLOGY AND OUTCOME OF NEONATAL SEPTIC SHOCK IN A PICU OF TUNISIA

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