**Abstracts**

**306 A NOVEL ANTI-OXIDANT TO COUNTERACT OXIDATIVE STRESS DURING RESUSCITATION AFTER BIRTH ASPHYXIA**

doi:10.1136/archdischild-2012-302724.0306

1E Henckel, 1R Solberg, 1E Calisch, 1S Norgren, K Bohlin, 2OD Saugstad. 1Department of Clinical Science, Intervention and Technology, Karolinska Institutet, Stockholm, Sweden; 2Department of Pediatric Research, Oslo University Hospital - Rikshospitalet, Oslo, Norway

**Background and Aims** Oxidative stress contributes to tissue damage after perinatal asphyxia. The thiol-containing free radical scavenger N-acetylcysteine amide (NACA) is a promising new anti-oxidant with good penetration into the mitochondria. The objective was to investigate the protective effect of NACA in a piglet model of birth asphyxia.

**Methods** Anesthetized newborn piglets (n=51) were subjected to global hypoxemia and block-randomized to either

1. intravenous administration of NACA 300 mg/kg and resuscitated with 21% oxygen for 30 min,
2. saline and 21% oxygen,
3. NACA and 100% oxygen or
4. saline and 100% oxygen.

After resuscitation, the piglets were followed for 9 hours and samples for several markers of injury and oxidative stress were collected. Reported here are clinical parameters and measurements of reduced to oxidized glutathione (GSH/GSSG).

**Results** Thirty minutes after end-resuscitation metabolic acidosis was less pronounced in the 100%-NACA group compared to 100%-oxygen-alone (lactate 8.1±2.6 vs 10.9±3.4, p<0.05). This difference was not shown for the 21%-oxygen groups. Mean arterial blood pressure and hemoglobin levels remained similar between the groups. The GSSG values were generally low. At end-resuscitation GSH was lower in 100%-NACA compared to 100%-oxygen-alone group (164±111 vs 255±113 μmol, p<0.05) and delta-GSH during resuscitation greater (143±49 vs 32±66 μmol p<0.001).

**Conclusions** The data indicate that NACA may enhance immediate recovery, improve mitochondrial glutathione metabolism and restore the cell to a normal metabolism following asphyxia and resuscitation. Upcoming analyses of histopathology and injury markers will further elucidate neuroprotective effect of NACA treatment following birth asphyxia.

**309 TUMOR NECROSIS FACTOR-INDUCIBLE GENE 6 PROTEIN: A NOVEL NEUROPROTECTIVE FACTOR AGAINST INFLAMMATORY DEVELOPMENTAL BRAIN INJURY**

doi:10.1136/archdischild-2012-302724.0309

1F Bertling, S Prager, R Hermann, 1Bendix, HG Wisniewski, U Felderhoff-Müser, FM Keller. 1Dept. of Paediatrics I - Neonatology, University Hospital Essen, Essen, Germany; 2Dept. of Microbiology, NYU School of Medicine, New York, NY, USA; 3Children’s Hospital Passau, Passau, Germany

**Background and Aims** An important factor of developmental brain injury is inflammation. It has been shown that tumor necrosis factor-inducible gene 6 protein (TSG-6) has anti-inflammatory effects in several inflammatory conditions. Nothing is known so far about the role of TSG-6 in the developing brain, its impact on inflammation and its therapeutic potential.

**Methods** PCR, Western Blotting and Immunohistochemistry was performed according to standard protocols. Brain hemispheres of untreated Wistar rats (p1-p15) were evaluated under developmental aspects of TSG-6. LPS-treated rats (0.25mg/kg LPS i.p. on p8) were evaluated under pathological aspects of TSG-6. To evaluate whether exogenous rhTSG-6 reduces inflammatory-induced brain injury,