neurogenesis pathway related genes (including Bdnf and Shh) on cerebellar and prefrontal cortical tissue.

Results Overall BDNF analysis showed no differences between term and preterm brains but levels were significantly different between day 5 and 26 in preterms only. SHH appeared to be lower in preterms compared to terms, but only significantly on Day 26.

Conclusions The reduced levels of SHH, specifically at day 26, suggest that SSH may be a useful biomarker for delayed brain development and indicate that the pig may provide a relevant model to study the premature brain.

PS-335 PRETERM AND TERM PIGLETS SHOW SIMILAR POSTNATAL ELECTROENCEPHALOGRAPHY (EEG)

¹AM Plomgaard, ²AD Andersen, ³TH Petersen, ²T Thymann, ²PT Sangild, ¹G Greisen. ¹Department of Neonatology, Copenhagen University Hospital Rigshospitalet, Copenhagen, Denmark; ²Department of Nutrition Exercise and Sports Faculty of Science, University of Copenhagen, Frederiksberg, Denmark; ³Research Unit on Brain Injury Neurorehabilitation Copenhagen. Department of Neurorehabilitation TBI Unit, University of Copenhagen, Glostrup, Denmark

10.1136/archdischild-2014-307384.634

Background Electroencephalography (EEG) changes rapidly with maturation of the brain in preterm infants. Amplitude-integrated EEG (aEEG) becomes more continuous, bandwidth narrows, and frequencies switch towards faster rhythms, as the child grows older. In preterm infants with brain damage this maturation is typically delayed. We are in the process of developing a preterm piglet brain model. Here we present the results of the EEG as a measurement of brain development in 1–11-day-old preterm and term piglets.

Methods One hour of EEG was recorded in 31 preterm piglets aged 1, 2, 4 and 11 days and in 10 term piglets aged 2 and 11 days. All piglets were delivered by C-section at either 90% or 100% gestation. Upper and lower margins of the aEEG band were visually identified and bandwidth calculated as the difference between the two values. Spectral analysis of the raw EEG was used to determine the relative power in the delta-(0.5–3 Hz), theta-(4–7.5 Hz), alfa-(8–12.5 Hz), and beta-(13–30 Hz) bands. General linear models were used with term vs. preterm, and age as predictors.

Results All aEEGs were continuous. The overall means (SD) of upper and lower margin, and bandwidth were 7 mcV (1.7), 15 mcV (5.7), and 8 mcV (4.3). Upper-, lower margin, bandwidth, alpha, beta and delta bands were unaffected by the predictors whereas the theta band was negatively correlated to age.

Conclusions The preterm piglet EEG was continuous already 10 days prior to term. Thus, maturation of EEG was neither seen in preterm nor term piglets and EEG may not be useful for studying perinatal brain maturation.

PS-336 INFLUENCE OF N-ACETYL CYSTEINE AMIDE (NACA) ON THE INFLAMMASOME PATHWAY. A STUDY ON NEONATAL PIGS

¹T Benterud, ¹M Ystgard, ¹L Pankratov, ²S Norgren, ¹R Solberg, ¹OD Saugstad. ¹Department of Pediatric Research, Oslo University Hospital, Oslo, Norway; ²Department of Pediatric Medicine, Karolinska University Hospital, Stockholm, Sweden

10.1136/archdischild-2014-307384.635

Abstract PS-336 Table 1 Study design: Fifty-four newborn piglets, age 12–36h, were included. Invasive blood pressure, EEG and ECG were measured continuously. One control group (n = 6) and 4 experimental groups (n = 12), exposed to global hypoxia, until BE was either -15 or -20 mmol/l (moderate/severe asphyxia with or without NACA) The pigs were observed for 9.5 h

Background and aims Severe perinatal hypoxia contributes to approximately 6% of spastic cerebral palsy (CP). Studies have indicated an association between elevation of IL-1beta after perinatal asphyxia and the development of CP. The NLRP3 Inflammasome complex may lead to release of the cytokines IL-1beta and IL-18 and cell death. Reactive oxygen species (ROS) have been proposed to be an upstream inducer of this complex and the anti-oxidant N-Acetylcysteine amide (NACA) may provide organ protection after hypoxia.

Objectives To map inflammasome activation in specific brain regions of the pig after neonatal hypoxia-reoxygenation and to investigate if the expression of different proteins in this pathway are modulated by NACA.

Methods

Study design (Table 1).

ELISA was used to measure IL-1b protein in cerebral cortex and Realtime PCR for mRNA expression of NLRP3, ASC, IL-1b and IL18 in cortex, cerebellum, hippocampus and striatum.

Results After severe hypoxia the protein expression of IL-1b in cerebral cortex was reduced for the NACA treated pigs vs. saline, $p < 0.05$.

Abstract PS-336 Table 2 Fold change for NLRP3, ASC, IL-18 and IL-1b measured in cerebral cortex. Significant difference in Fold Change of ASC (*), $p < 0.05$. All values are Median values