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 SHH may be a useful biomarker for delayed brain development and indicate that the pig may provide a relevant model to study the premature brain.

Preterm and term piglets show similar postnatal electroencephalography (EEG)

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), alpha-(8–12.5 Hz), and beta-(13–30 Hz) bands. General linear models were used with term vs. preterm, 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.

Influence of N-acetylcysteine amide (NACA) on the inflammasome pathway. A study on neonatal pigs

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.
When comparing all the pigs treated with NACA vs. saline after hypoxia Fold Change of ASC in cortex was significantly reduced, p (Table 2).

In hippocampus, cortex and Striatum Fold Change of IL-1b was elevated in all the hypoxia groups compared with the control group, p

**Conclusion** NACA reduces the protein expression of IL-1beta and mRNA-expression of ASC in cortex after hypoxia. This may indicate that NACA has some neuroprotective abilities after perinatal asphyxia.

Upcoming analyses of histopathology and injury markers will elucidate possible neuroprotective effects of NACA treatment following birth asphyxia.

**PS-337** WITHDRAWN

**PS-338** CONCURRENT ALLOPURINOL AND HYPOTHERMIA TREATMENT IN A TERM NONHUMAN RAT MODEL OF HYPOXIC ISCHAEMIC ENCEPHALOPATHY

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Background and aims Hypoxic-ischaemic encephalopathy (HIE) has been associated with long-term disabilities. Hypothermia is effective but does not provide complete neuroprotection, adjunctive therapies are necessary. Allopurinol has been proved as a good neuroprotector, but it has never been tested associated with hypothermia. The aim of the present study was to examine therapeutically effectiveness of dual therapy (hypothermia + allopurinol) versus hypothermia, in a neonatal rat model of HIE.

**Methods** 120 Wistar pups at postnatal day 10 were used and divided into 5 groups: Sham-Operated, Hypoxic-ischaemic (HI) aggression, HI aggression + Allopurinol, HI aggression + Hypothermia, HI aggression + Hypothermia + Allopurinol.

At 25 day of life, spatial memory was assessed via water maze test. Finally, rats were anaesthetised and sacrified. In order to assess possible alterations in the hippocampal synaptic network, 3 specific synaptic proteins (PSD95, SNAP25, synaptophysin) were tested by Western Blot.

**Results** There were differences in the learning outcomes among hypoxic, hypoxic + allopurinol, hypothermia, hypoxia + allopurinol and sham operated (p < 0.05). The worst group was the hypoxic one.

Synaptophysin and SNAP25 levels were higher in controls and treatment groups compared with hypoxic untreated animals. However, the highest level of PSD95 corresponded to the hypoxic group.

**Conclusions** Hypothermia and allopurinol seem to improve learning in HIE pups.

Increased levels of presynaptic proteins in the treatment groups suggest that hypothermia and allopurinol improve synaptic plasticity compared with untreated group.

PSD95 was also described in the literature as a suppressor of dendritic arbour development, so this could explain our results in the hypoxic group.