Background and aims Acute kidney injury (AKI) is a common consequence of perinatal asphyxia (PA). Therapeutic hypothermia (TH) used for neuroprotection in hypoxic-ischaemicencephalopathy (HIE) may also ameliorate kidney injury. AKI can be associated with more severe PA, but association with worse neurologic outcome remains to be proven. We aim to evaluate the incidence of AKI in neonates under TH and the association with HIE grades and outcome.

Methods A total of 51 cooled infants were reviewed using a prospectively collected database. Modified AKIN criteria were used to classify AKI. We studied perinatal, resuscitation and clinical data during admission to the NICU. Outcome of interest was duration of NICU stay, predictable neurologic outcome and mortality.

Results AKI was found in 17 of 51 neonates (33%). Based on the modified AKIN criteria, 13, 1, and 3 patients had stages I, II, and III, respectively. AKI was more frequent in HIE 2/3 than in the rare HIE 1 infants that were cooled (p < 0.03) and in those with worse base deficit at birth (p < 0.05). A significant association was found between vancomycin and gentamicin use and AKI (p < 0.01). Renal replacement therapy was needed in 2/3 of AKIN stage III infants. NICU stay, mortality and predicted adverse neurologic outcomes were not associated with AKI.

Conclusions AKI was less frequent in our cohort than the one previously described in non-cooled newborns. More severely asphyxiated neonates were more likely to experience AKI, but AKI was not related to worse outcomes.

**Background** Prematurity is a major determinant of neonatal mortality and morbidity. The number of preterm birth is still on the rise. Recently we and others could demonstrate neuroprotective effects of sigma-1 receptor ligands in adult and newborn animal models of brain injury. Since sigma-1 receptor agonists are already undergoing clinical trials in adult neurological disease, they might be considered a promising therapeutic option also in preterm brain injury. We have previously shown that the selective sigma-1 receptor agonist PRE-084 (2-(4-morpholino-nethyl)-1-phenylcyclohexane-carboxylate) protects against neonatal excitotoxic brain injury in vivo. The aim of the present study was to investigate whether PRE-084 is able to prevent neurotoxicity following glutamate exposure in vitro.

**Methods** Cultured primary hippocampal neurons (day in vitro 10) were pre-treated with PRE-084 before glutamate was applied. Subsequently cell death was quantified by means of PI/calcine – AM staining using live confocal microscopy. Neurons were randomly assigned to one of the following groups: i) control, ii) glutamate or iii) glutamate+PRE-084. PRE-084 was applied in two dosages (10 and 100 μM) prior to glutamate.

**Results** The application of PRE-084 significantly reduced the percentage of dead cells (PRE-084 10 μM: 22.09 (20.50;28.84) and 100 μM: 25.87 (18.77;33.40)) compared to the untreated glutamate control group 43.56 (39.86;46.02).

**Conclusion** Our data show that administration of PRE-084 protects against glutamate induced cell death in primary hippocampal neurons. PRE-084 shows considerable promise as a therapeutic strategy in preterm brain injury and might provide an adequate means of combating this major cause of neurological disability in infancy.

**Poster abstracts**