Involvement of the corpus callosum after neurodevelopmental and behaviour outcome in established in vitro models of neonatal hypoxic-ischaemic brain injury.

**Aim** To evaluate the effect of SN in established in vivo and in vitro models of neonatal hypoxic-ischaemic brain injury.

**Methods** Seven day old mice underwent unilateral common carotid artery ligation, followed by exposure to hypoxia (8% oxygen). Thereafter, mouse pups were randomly injected intraperitoneally with SN (0.25 mg/kg body weight) or vehicle. As endpoint we determined the histological injury score and the number of caspase-3 positive cells 24 h after the insult. Primary cultured hippocampal neurons were treated with oxygen glucose deprivation (OGD) on day 10. Neurons were assigned to the following groups: i) control ii) OGD iii) OGD+SN (1, 10 or 50 μg/l). As primary outcome parameter, cell death was evaluated via real time live confocal imaging using calcine-AM and propidium iodide (PI).

**Results** SN displayed a non-significant trend to lower mean values of histological injury score compared to control (n = 11–12, p > 0.05) and significantly reduced the number of cells stained positively for activated caspase-3 (n = 6, p < 0.05). In vitro SN application on hippocampal neurons (OGD+SN) significantly reduced the number of dead cells assessed by the PI/calcine ratio compared with the untreated OGD group (n = 8, p < 0.05).

**Conclusion** We provide first evidence that SN is neuroprotective in established in vitro and in vivo models of neonatal hypoxic-ischaemic brain injury and might therefore be considered a promising therapeutic option.

Involvement of the corpus callosum after perinatal asphyxia demonstrated using diffusion weighted MRI is related to neurodevelopmental outcome.

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Neurodevelopmental and behaviour outcome of preterms (GA <30wks) at 8 years by age appropriate psychometric evaluations to see whether tests used at younger ages could predict worst outcomes at older ages in relation to some neonatal factors.

**Method** Along with neurologic examinations, 33 infants were prospectively evaluated at 3, 6, 12, 18, 24 months of corrected age with Bayley Scales of Infant Development – II (BSID-II), at 3, 5 years with Stanford-Binet, at 8 years with WISC-R.

**Results** 72% of children had no (IQ >85), 24.3% had mild (IQ 74–84), 3% had major (IQ <70, blindness) impairments. 24.2% had special education, 15.2% ADHD, 6.1% autism, 9.1% learning/language, 6.1% anxiety disorders. The probability of neurodevelopmental test and IQ scores of VLBW infants <1000 gr being lower than healthy children at same age was 10.5 times higher (OR 4.7, 95%, CI 0.92–24.5) at 8 years of age. Oxygen treatment >30 days adversely affected the scores up to 18th month (OR 2.1, 95%, CI 0.44–9.8). Babies having low scores of the18th month-cognitive and motor sub-test of BSID-II had 16 times higher probability of having low WISC-R total IQ scores at 8 years. (p < 0.05). 19 children with sepsis at 8 years had lower performance and total IQ scores (p < 0.05).

**Conclusion** Prolonged oxygen therapy and having and sepsis are significant factors affecting later IQ of VLBW infants. Lower BSID-II scores at 18th month may predict future lower total IQ scores. Longitudinal follow up and early intervention is of paramount importance.

Neuroprotective effect of pentoxifylline in rat pups with hypoxic-ischaemic encephalopathy.

**Aim** To evaluate the effect of SN in established in vivo and in vitro models of neonatal hypoxic-ischaemic brain injury and might therefore be considered a promising therapeutic option.

**Methods** Seven day old mice underwent unilateral common carotid artery ligation, followed by exposure to hypoxia (8% oxygen). Thereafter, mouse pups were randomly injected intraperitoneally with SN (0.25 mg/kg body weight) or vehicle. As endpoint we determined the histological injury score and the number of caspase-3 positive cells 24 h after the insult. Primary cultured hippocampal neurons were treated with oxygen glucose deprivation (OGD) on day 10. Neurons were assigned to the following groups: i) control ii) OGD iii) OGD+SN (1, 10 or 50 μg/l). As primary outcome parameter, cell death was evaluated via real time live confocal imaging using calcine-AM and propidium iodide (PI).

**Results** SN displayed a non-significant trend to lower mean values of histological injury score compared to control (n = 11–12, p > 0.05) and significantly reduced the number of cells stained positively for activated caspase-3 (n = 6, p < 0.05). In vitro SN application on hippocampal neurons (OGD+SN) significantly reduced the number of dead cells assessed by the PI/calcine ratio compared with the untreated OGD group (n = 8, p < 0.05).

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