LEARNING DIFFICULTIES AND UNDERNUTRITION. IS THERE A PROBLEM IN THE SYNAPSES? STUDYING IT WITH AN ANIMAL MODEL

Methods Carotid artery rings from 2–3 d-old and 9–10 d-old rats were mounted in myographs and studied at 33 and 37°C. Results Hypothermia did not significantly affect the contractions induced by KCl and U46619, nor the relaxations induced by acetylcholine (ACh), the nitric oxide (NO) donor sodium nitroprusside (SNP), the NO-independent stimulator of soluble guanylate cyclase (sGC) BAY 41–2272, the β-adrenoceptor agonist isoproterenol, the adenylyl cyclase activator forskolin, and acute hypoxia (PO2 3 kPa). The relaxations induced by ACh, isoproterenol, the β2-adrenoceptor agonist salbutamol, the β1-adrenoceptor agonist CL-316243 and hypoxia increased with postnatal age and were impaired by endothelium removal or by inhibition of NO synthase (L-NAME) or sGC (ODQ). By contrast, the relaxations induced by SNP, BAY 41–2272 and forskolin were endothelium-independent and did not change with age.

Conclusions Mild hypothermia (33°C) does not affect the reactivity of neonatal rat carotid arteries. Our data suggest a reduced NO bioavailability in the carotid artery during the first days of life. This transient reduction in endothelium-dependent relaxation might play a role in the adaptation of the circulatory system to birth and in the neonatal vascular response to insults such as hypoxia.

Preterm Brain Injury – Experimental

Background Premature infants provided parenteral nutrition (PN) high in n-6 polyunsaturated fatty acids (PUFA) have increased risk of inflammatory disease, such as nosocomial sepsis. The pro-inflammatory insult can also contribute to injury and delayed neuronal growth in the perinatal brain. Provision of high long chain n-3 PUFA in parenteral lipids is associated with decreased inflammation and incidence of sepsis. The provision of n-3 PUFA, especially docosahexaenoic acid (DHA) also is critical for neurodevelopment in premature infants.

Aim To determine whether a new generation lipid emulsion (PN) high in n-3 PUFA (SMOFlipid) protects against inflammation and improves neuroprotection in response to lipopolysaccharide (LPS) compared to a lipid emulsion high in n-6 PUFA (Intralipid).

Methods Preterm piglets delivered 7 d preterm were assigned into two groups receiving complete TPN containing either Intralipid or SMOFlipid at 10 g*kg-1*d-1 for 10 d. On day 10, subgroups of piglets were assigned to receive either an 8-hr infusion of lipopolysaccharide (2 mg/kg) or control saline and target gene expression in brain tissue was analysed.

Results LPS increased brain gene expression of pro-inflammatory cytokines IL-6, IL-8, and TNF in the Intralipid group, but not the SMOFlipid group. The gene expression of the anti-inflammatory cytokine IL-10 was increased in both LPS-treated lipid groups. Brain-derived neuronal growth factor, a marker of neuronal proliferation, was decreased in the LPS-treated SMOFlipid group, but not the LPS-treated Intralipid group.

Conclusions SMOFlipid protected against LPS-induced inflammation, but did not acutely increase the expression of the neuroprotective protein, BDNF, in the presence of LPS.