

**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 nitropruside (SNP), the NO-independent stimulator of soluble guanylate cyclase (sGC) BAY 41–2272, the  $\beta$ -adrenoceptor agonist isoproterenol, the adenylate cyclase activator forskolin, and acute hypoxia (PO<sub>2</sub> 3 kPa). The relaxations induced by ACh, isoproterenol, the  $\beta_2$ -adrenoceptor agonist salbutamol, the  $\beta_3$ -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.

#### PS-341 LEARNING DIFFICULTIES AND UNDERNUTRITION. IS THERE A PROBLEM IN THE SYNAPSES? STUDYING IT WITH AN ANIMAL MODEL

<sup>1</sup>C Durán, <sup>2</sup>C Carrasco, <sup>2</sup>J Rodríguez, <sup>3</sup>N Villalanzo, <sup>3</sup>JC Jiménez-Chillarón, <sup>2</sup>M Camprubí. <sup>1</sup>Neonatal Unit, Complejo Hospitalario Universitario de Vigo, Vigo, Spain; <sup>2</sup>Neonatal Unit, Hospital Sant Joan de Deu, Barcelona, Spain; <sup>3</sup>Fundación Sant Joan de Deu, Hospital Sant Joan de Deu, Barcelona, Spain

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**Backgrounds and aims** Intrauterine growth restriction (IUGR) and rapid postnatal weight gain increase susceptibility to metabolic syndrome during adult life. Longitudinal studies have also revealed high incidence of learning difficulties in children with IUGR.

The aim of the present study was to investigate the effect of nutrition on learning memory in an IUGR animal model.

**Methods** We use a mouse model of IUGR induced by caloric maternal undernutrition during late gestation. During the suckling period, dams were either fed *ad libitum* or food restricted. Pups were divided into: control-control (CC), undernutrition-control (UC), control-undernutrition (CU) and undernutrition-undernutrition (UU), indicating the prenatal-postnatal experimental conditions.

At 4 weeks of age, memory was assessed via water maze test. Finally, rats were anaesthetised and sacrificed. To assess possible alterations of the hippocampal synaptic network, 3 specific synaptic proteins (PSD95, SNAP25, synaptophysin) were tested by Western Blot.

**Results** CC, UC, CU exhibited shorter escape latencies (EL) along the days. UU hardly changed its EL, indicating a poor spatial memory performance. Learning differences between CC and UU were statistical significant ( $p < 0,01$ ). CC animals had the higher protein synaptic levels in the hippocampus compared to all other groups ( $p < 0,05$ ).

**Conclusions** Nutrition plays an important role in learning. A poor pre and postnatal nutrition is associated with learning and memory alterations. Catch-up growth group showed an improvement in learning compared to UU. A decreased level of synaptic proteins in animals with a deficient nutrition (pre,

postnatal or both), suggests that malnutrition results in less functional or efficient synapses.

## Preterm Brain Injury – Experimental

#### PS-341a NEW GENERATION LIPID EMULSION PROTECTS AGAINST LPS-INDUCED BRAIN INFLAMMATION IN PREMATURE PIGLETS

<sup>1</sup>G Guthrie, <sup>1</sup>B Hodges, <sup>2</sup>C Martin, <sup>1</sup>B Stoll, <sup>3</sup>O Olutoye, <sup>4</sup>S Freedman, <sup>1</sup>D Burrin. <sup>1</sup>Pediatrics, USDA/ARS Children's Nutrition Research Center/Baylor College of Medicine, Houston, USA; <sup>2</sup>Neonatology, Beth Israel Deaconess Medical Center/Harvard Medical School, Boston, USA; <sup>3</sup>Surgery, Texas Children's Hospital, Houston, USA; <sup>4</sup>Medicine, Beth Israel Deaconess Medical Center/Harvard Medical School, Boston, USA

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**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 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<sup>-1</sup>\*d<sup>-1</sup> 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.

## Primary Care General I

#### PS-342 MOTHER'S DEPRESSIVE SYMPTOMS AND THEIR COMMUNITIES' SOCIAL CAPITAL

J Pascoe, R Rapp, M Perry, M Pesce. Pediatrics, Wright State University, Dayton, USA

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**Background** Previous studies have documented the association between mothers' personal social support and mothers' depressive symptoms. Maternal depressive symptoms have a pernicious effect on women's ability to function effectively as a mother. This study expands the concept of mothers' 'social connectedness' to include mothers' perception of their communities' support capital.