We investigated the contribution of genetic variations in CDH13 to adiponectin level in patients with statin treatment. A variation of CDH13 (rs3865188) was genotyped in 345 Korean patients. We divided them into two groups; statin-free group versus statin group. There were differences in the distributions of alleles and genotypes between each group. The genetic variation of CDH13 gene showed a significant correlation with several clinical factors, such as adiponectin, diastolic blood pressure (DBP), triglyceride (TG), and insulin. Patients with the TT had significantly lower adiponectin level than those with AA ($p = 0.046$). The fasting blood sugar (FBS) was lower in patients with TT than those with AA ($p= 0.026$). The highest TG was observed in the patients with TT compared with AA in statin-free group ($p = 0.025$). In statin-free group, we observed a significant difference of adiponectin level between patients with A allele and those with T allele ($p = 0.007$), while there was no difference of adiponectin level in statin group. In linear regression analysis, BMI and insulin were negatively correlated with adiponectin level in patients with A allele only regardless of statin group ($p=0.011$ vs, $p=0.003$, respectively). Whereas, triglyceride was associated with adiponectin in patients with T allele of statin-free group ($p<0.001$). In statin group, BMI, TG, and insulin could explain the usual decrease in adiponectin in patients with A allele ($p=0.002$, $p=0.009$, and $p=0.003$, respectively) not T allele. We identified an association between variants of CDH13 rs3865188 and adiponectin level in Korean patients with statin treatment.

Maternal-fetal evaluation of oxidized lipid products of polyunsaturated fatty acid induced by environmental contaminant perfluorooctane sulfonate
(TUESDAY, N9.06)

Presenter Last Name: Leung

Perfluorooctane sulfonate (PFOS) is synthetic fluorinated hydrocarbons. However the carbon-fluoride bonds render these compounds to be non-biodegradable, leading to their persistence in the environment and lengthy elimination half-life in vivo. PFOS could also penetrate the placental barrier and the blood brain barrier, and produce neurotoxic effect. High dose of PFOS leads to neonatal mortality and neurologic delays. It is known PFOS generate a dose-dependent ROS production, but the effect in PUFA lipid peroxidation, especially adrenic, arachidonic, docosahexaenoic and eicosapentaenoic acids that are important for cerebral development is not well investigated. In this study, we evaluated oxidised lipid products (F2- and F3-isoprostanes, F4-neuroprostanes, F2-dihomo-isoprostanes, dihomo-isofuran, HETEs, RvD1) of arachidonic, eicosapentaenoic, docosahexaenoic and adrenal acids in liver of maternal CD-1 mice treated with vehicle (control), 0.3 or 3 mg/kg/day PFOS for 21 days and after 7 days washout period. Fetal liver of male and female pups were also assessed upon in utero PFOS exposure to the maternal mice. Exposure of PFOS reduced liver arachidonic, eicosapentaenoic, docosahexaenoic and adrenic acids level in maternal and pup. Oxidized lipid products, in particular F2-

isoprostanes, F4-neuroprostanes F2-dihomo-isoprostanes and dihomo-isofuran elevated in liver of maternal mice exposed to PPFO at both concentrations compared to control. The levels did not recede after 7 days washout period and instead increased further. Concentration of the oxidized lipid products, notably F2- and F3-isoprostanes were also high in pup livers compared to control. Concentration of liver of female pups were higher than male. This study indicates exposure of PFOS altered PUFA metabolism and induced liver lipid peroxidation in maternal mice. Such effect can be transferred to fetus and cause toxicity that may lead to detrimental effect on neurological development.

Omega-3s in TBI and Post-Concussive Syndrome (TUESDAY, N12.07)

Presenter Last Name: Lewis

Background. Traumatic brain injury (TBI), with its diverse heterogeneity and prolonged secondary pathogenesis, remains a clinical challenge. Post-concussive syndrome (PCS) remains an enigma as well. Currently, there are no effective treatments for either. Management focuses on acute surgical and intensive care, long-term rehabilitation, and treatment of symptoms. Promising treatments have failed to translate clinically as they typically target single pharmacologic targets rather than considering the multiple mechanisms of the injury and a more holistic approach to the brain itself. A combination therapy influencing multiple aspects of neuroprotection, neuroinflammation, and neuroregeneration is needed. Omega-3 fatty acids (ω-3FA) offer the advantage of a poly-target approach. Eicosanoid, decosanoid, and resolvin biochemical pathways positively influence neuronal cell survival and neuroinflammation and promote remyelination, neurogenesis, and synaptogenesis. Methods. An unpublished pilot study of PCS patients using EEG brain-mapping and neurocognitive testing pre- and post-therapy with five weeks of high dose ω-3FA are detailed. Results. Brainmapping and neurocognitive testing demonstrate significant objective improvement in PCS subjects post- ω-3FA therapy as compared to baseline. Based on these results and experience, proposed clinical protocols are presented for TBI and PCS. Conclusions. The brain needs to be saturated with high doses of ω-3FA in order to have the best opportunity to heal following injury. This is substantiated with EEG brain-mapping and neurocognitive testing. Although further clinical research is needed, there is a growing body of experience suggesting ω-3FA are beneficial in TBI as well as for those who continue to suffer the longer term symptomatic consequences.

N-3 polyunsaturated fatty acids modulate homocysteine metabolism (SUNDAY, M4.05)

Presenter Last Name: Li