Effect of n-3 polyunsaturated fatty acid deficiency on fatty acid composition of brain phospholipid classes in rats

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A previous study found that n-3 PUFA deficiency will cause an increased blood pressure later in life in Sprague-Dawley rats (1), decreased 22:6n-3 in neural membranes associated with changes in Na-K-ATPase function, Na-channel action and prostaglandin production (2). The aim of this study was to investigate the effect of n-3 diet rich in salt and saturated fat, with and without polyunsaturated fatty acids on the fatty acid composition of brain phospholipid classes in rats.

Eighteen Sprague-Dawley dams were randomly divided into three groups of six animals at mating. Each group of the dams were put onto one of three different diets A, B and C, which were supplied by Glen Forrest Stockfeeders in Western Australia. Diet A was high saturated fat, high salt minus n-3 PUFA, diet B was high saturated fat, high salt plus n-3 PUFA, and diet C was a low saturated fat, low salt with n-3 PUFA. All diets were stored at 4°C. Pups were kept with their mothers till weaning at 21 days. All pups from each of the diets were put together for a few days and were fed the same diets and water ad libitum as their dams. A total number of 6 pups per diet were then separated according to sex and boxed into four groups of 3 animals with their body weight matched. At 6 weeks post-weaning, the pups were boxed into groups of 2. The pups aged six months were killed by decapitation and the brain frontal cortex was collected. Lipid was extracted by chloroform : methanol (2 : 1, v/v). Phospholipid classes was separated by thin liquid chromatography (20 × 20 cm, Silica Gel 60, Merck) with solvent system: chloroform : methanol : acetic acid : NH₄OH (28%) : H₂O (50 : 35 : 4 : 1 : 1). Individual phospholipid class was collected and fatty acid methyl esters were prepared by saponification of using KOH (0.68 mol/L in methanol) followed by transesterification in BF3 in methanol. Fatty acids were identified GLC.

There was no effect of high saturated fat on the 22:6n-3 levels in the brain, however the diet deficient in n-3 PUFA had significantly reduced levels of 22:6n-3 in PE, PC and PS and significantly increased levels of 22:4n-6 and 22:5n-6 in these fractions (P < 0.01). The magnitude of 22:6n-3 loss was approximately 20%, which indicates how difficult it is to deplete this tissue of n-3 PUFA.