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Omega-3 polyunsaturated fatty acid contents of processed fish products
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Background – Seafood is the major dietary source of long chain omega-3 polyunsaturated fatty acids (LC n-3 PUFA) including mainly EPA (eicosapentaenoic acid, 20:5n-3), DHA (docosahexaenoic acid, 22:6n-3) and DPA (docosapentaenoic acid, 22:5n-3). The health benefits of these n-3 PUFA have been well documented. However little information is available on the n-3 PUFA content of processed seafood.

Objective – To investigate n-3 PUFA content of commonly available processed fish products including Coles tuna cakes and fish fillets; Bird’s Eye tuna cakes, fish cakes and fish fillets; and Pacific West fish fillets.

Design – A total of six types of processed uncooked seafood were purchased from Coles supermarket in Melbourne. The total lipid was extracted with methanol-chloroform containing butylated hydroxytoluene. The fatty acid methyl esters were prepared by saponification of 20mg lipid plus 2mg of methyl tricosanoate using KOH followed by transesterification in BF₃ in methanol. The fatty acids methyl esters were separated by capillary GLC. One-way ANOVA and Tukey multiple comparisons were performed to determine differences in individual fatty acid level between different samples.

Outcomes – LC n-3 PUFA contents varied significantly between the six types of processed fish products, ranged from 97.2 mg/100g to 581.8mg/100g. Total n-6 PUFA contents also showed a marked difference between different products, and they were significantly higher than in fresh fish samples. As a consequence a relatively lower n-3: n-6 ratio was found in all six products, ranged from 0.5:1 to 1:1. No significant difference in n-3/n-6 PUFA ratio was found between the different products. In addition, contents of monounsaturated fatty acids (MUFA) and saturated fatty acids (SFA) differed significantly between the six products.

Conclusion – n-3 LC-PUFA content of processed fish products varies significantly between the products. Higher n-6 PUFA content and lower n-3: n-6 ratio in these processed fish products is more likely due to the addition of cooking oil during processing.

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Neuroprotective effect of epigallocatechin gallate against glutamate toxicity
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Background – Glutamate is a natural neurotransmitter widely distributed in the Central Nervous System (CNS). Its concentration is maintained at a low level through a rapid metabolic clearance mechanism. Accumulation of glutamate in CNS following a neurological disease or injury (e.g., stroke) can lead to neuronal death and cause further damage. It is believed that glutamate toxicity involves the blockage of the cellular glutathione antioxidant system.

Objective – To test whether epigallocatechin gallate (EGCG), a potent free radical scavenger abundant in green tea, can protect neurons from glutamate toxicity.

Design – Fetal rat hippocampal neuron cell line was cultured in Dulbecco’s modified Eagle’s medium containing 0 – 20mM glutamate for up to 24 hours with or without presence of Vitamin E or EGCG. The effects of various treatments on neuronal cell viability were assessed by MTT test, which is based on the reduction of MTT by mitochondrial hydrogenases in active cells. The treatment effects on cellular expression of various genes related to apoptosis were determined by polymerase chain reaction (PCR).

Outcomes – It was found that glutamate exposure caused significant reduction of neuronal cell viability in a time and dose dependent manner with the 50% reduction at 8mM after 12 hours exposure. Addition of 1 uM Vitamin E or 0.1 mM EGCG provided over 90% neuronal protection to glutamate toxicity. PCR analysis revealed that glutamate exposure significantly increased the expression of several genes related to apoptosis and addition of Vitamin E or EGCG restored the gene expression to the control levels.

Conclusions – The results of the present study demonstrate that EGCG, a natural compound from green tea, can protect neurons from glutamate toxicity in vitro and the compound may have a neuronal protective effect and reduce secondary neuronal damage in patients suffering a stroke.