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Lipid oxidation and Carotenoid supplementation

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Oxidative stress has been considered as important in the pathogenesis of Alzheimer's disease, AD. Post-mortem investigations of affected brain regions in AD have shown the accumulation of oxidative damage to protein, DNA and lipids. Carotenoids have the ability to quench singlet oxygen and scavenge other reactive oxygen species (ROS) without being consumed in the process. We have previously shown increased concentrations of the novel oxidized phospholipid biomarker, 1-palmitoyl-2(5-oxo-valeroyl)-sn-glycero-3-phosphocholine (POVPC) and lower carotenoid plasma concentrations in AD. POVPC was analysed using electrospray ionisation tandem mass spectrometry (MS) with multiple reaction monitoring (MRM), 8-isoprostane (IsoP) was measured by ELISA and ferric reducing antioxidant potential (FRAP) was measured by a colorimetric assay in AD patients and healthy age-matched control subjects. The developed MRM-MS method was used to analyse POVPC as a measure of peroxidative damage to phospholipids in serum. Using this method, the peroxidised phospholipid POVPC was found to be higher in AD patients and was correlated with cognitive performance but not reduced by carotenoid supplementation. We also investigated the protective role for carotenoids against mitochondrial dysfunction induced by POVPC (1-20μM) in differentiated (d)SH-SY5Y neuronal cells. POVPC, lutein (0.1-1μM) and zeaxanthin (0.05-5μM) were recovered in dSH-SY5Y cells after 24 hours of treatment. Glutathione (GSH) levels, mitosox oxidation and mitochondrial function were analysed in cells treated with POVPC (1-20μM) and co-incubation with carotenoids (lutein and zeaxanthin). We found pathophysiological concentration-induced damage can be protected with appropriate dose of carotenoid.

The talk will highlight some of our main findings on the effects of carotenoid supplementation on lipid oxidation.

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