

## “ Research & Commentary ”

contains information for healthcare professionals about subjects of interest, we understand how important it is to your practice to be informed about latest clinical research and information. We also appreciate that your time is precious commodity, which makes it essential to get you the most update information in the most efficient manner.

### Ethyl-eicosapentaenoate modulates changes in neurochemistry and brain lipids induced by parkinsonian neurotoxin 1-methyl-4-phenylpyridinium in mouse brain slices

#### Abstract

Evidence suggests a link between Parkinson's disease and the dietary intake of omega (n)-3 and n-6 polyunsaturated fatty acids (PUFAs). Presently, we investigated whether an acute dose of parkinsonian neurotoxin 1-methyl-4-phenylpyridinium (MPP(+)) affects brain n-3 and n-6 PUFA content and expression of fatty acid metabolic enzymes cytosolic phospholipase A2 (cPLA2) and cyclooxygenase-2 (COX-2) in brain slices from C57Bl/6 mice. Furthermore, we investigated whether feeding a diet of n-3 PUFA ethyl-eicosapentaenoate (E-EPA) to these mice can attenuate the MPP(+) induced changes in brain PUFA content and expression of cPLA2 and COX-2, and attenuate MPP(+) induced changes in neurotransmitters and metabolites and apoptotic markers, bax, bcl-2 and caspase-3. MPP(+) increased brain content of n-6 PUFAs linoleic acid and arachidonic acid, and increased the mRNA expression of cPLA2. MPP(+) also depleted striatal dopamine levels and increased dopamine turnover, and depleted noradrenaline levels in the frontal cortex. The neurotoxin induced increases in bax, bcl-2 and caspase-3 mRNA expression that approached significance. E-EPA by itself increased brain n-3 content, including EPA and docosapentaenoic acid (C22:5, n-3), and increased cortical dopamine. More importantly, E-EPA attenuated the MPP(+) induced increase in n-6 fatty acids content, partially attenuated the striatal dopaminergic turnover, and prevented the increases of pro-apoptotic bax and caspase-3 mRNAs. In conclusion, increases in n-6 PUFAs in the acute stage of exposure to parkinsonian neurotoxins may promote pro-inflammatory conditions. **EPA may provide modest beneficial effects in Parkinson's disease, but further investigation is warranted.**

#### References :

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Meng Q', Luchtman DW, El Bahh B, Zidichouski JA, Yang J, Song C.

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**Reduction in cerebral atrophy associated with ethyl-eicosapentaenoic acid treatment in patients with Huntington's disease.****Abstract**

**Ultra-pure ethyl-eicosapentaenoic acid (ethyl-EPA), a semi-synthetic ethyl ester of eicosapentaenoic acid, is associated with clinical improvement in motor functioning in Huntington's disease.** The aim was to determine the extent to which it might reduce the rate of progress of cerebral atrophy. **High-resolution cerebral magnetic resonance imaging scans were acquired at baseline, 6 months and 1 year in up to 34 patients with stage I or II Huntington's disease who took part in a randomized, double-blind, placebo-controlled trial of ethyl-EPA.** For each subject and each pair of structural images, the two-timepoint brain volume change was calculated in a double-blind manner. Significant group-level reductions in brain atrophy were observed in the head of the caudate nucleus and the posterior thalamus. **These findings show that treatment with ethyl-EPA is associated with significant reduction in brain atrophy, particularly in the caudate and thalamus. No other drug tested in Huntington's disease has shown this effect.**

## References :

J Int Med Res. 2008 Sep-Oct;36(5):896-905.

Puri BK<sup>1</sup>, Bydder GM, Manku MS, Clarke A, Waldman AD, Beckmann CF.**Provided by the maker's of****LACTOMEGA™****Natural Omega 3 (DHA, EPA) & Anti-oxidants**

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