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Mitochondrial energy from omega-3 protects immunity and cognition of mild Cognitive Impairment patients beyond Cholinesterase inhibitors

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Background: The innate immune system clears amyloid-β (Aβ) by phagocytosis but fails in Mild cognitive impairment patients (MCI) (reversibly) and in Alzheimer disease patients (AD) (irreversibly). Omega-3 fatty acids (omega-3), vitamin 1,25D3, and curcuminoids repair AD patients' phagocytic and transcriptional defects.

Objectives: Immune and cognitive effects of supplementation by a fish-derived lipid emulsion with omega-3 protected against oxidation.

Method: We measured Aβ phagocytosis by the flow cytometric Aβ blood test. We investigated the effects of omega-3 on mitochondrial respiration and glycolysis in immune cells using the Seahorse XF96 Extracellular Flux Analyzer (Agilent). We performed RNA-seq of macrophages using Illumina HiSeq 4000, aligned reads to the UCSC hg19 reference genome and obtained read counts using HT-Seq.

Result: Omega-3-supplemented MCI patients (MMSE >19) maintained cognitive status close to baseline 1.7 to 5.1 years beyond cholinesterase inhibitors. Omega-3 had no significant effects in patients with established Alzheimer – type dementia (MMSE < 19) or Lewy body disease. Omega-3 treatment of macrophages in vitro increased transcription of enzymes for glycolytic and ox-phos energies. Omega-3 increased basal oxygen consumption rate (OCR), ATP-linked OCR, and OCAR/ECAR ratio in peripheral blood mononuclear cells (PBMC's). In omega-3 supplemented subjects, Aβ phagocytosis was active even when glycolysis was inhibited by iodoacetate.

Conclusion: In a pilot study, cell signaling and increased energy from a fish-derived emulsion of omega-3 recovered the immune functions of MCI through increased mitochondrial energy and unfolded protein response. As the changes in immune and cognitive functions in MCI patients were correlated (r=0.77), the immune system may have a disease-modifying role in some MCI patients.

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