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Editorial Open Access

Polyunsaturated Fatty Acids Play a Role in Depression: Main Action Mechanisms

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Editorial

The omega-3 and omega-6 Polyunsaturated Fatty Acids (PUFAs) are either synthesized from the essential fatty ACIDs α-Linolenic Acid (ALA) and Linoleic Acid (LA) respectively, or obtained from the diet. Omega-3 PUFAs, Eicosapentaenoic Acid (EPA) and Docosahexaenoic Acid (DHA) are found predominantly in oily fish such as salmon, mackerel and sardines. Some experimental, clinical and epidemiological data favour the hypothesis that PUFAs could play a role in pathogenesis and treatment of depression [1,2]. The aim of this work is to summarize the main mechanisms whereby PUFAs may exert this role.

One of the mechanisms appears to be the decrease of neuroinflammation. Depression is associated with a chronic and low-grade inflammatory response and an activation of cell mediated immunity [3,4]. It is similarly accompanied by increased oxidative and nitrosative stress [3,4]. The mechanism by which proinflammatory cytokines influence depressive symptoms and vice versa has been studied, but it is not completely understood. However, preliminary data from depressed patients suggest that inhibiting proinflammatory cytokines or their signaling pathways may improve depressed mood and increase treatment response to conventional antidepressant medication [4]. In this context, there is a functional opposition between omega-3 and omega-6 PUFAs. Higher relative levels of omega-3 tend to reduce the production of pro-inflammatory cytokines (IL-1, IL-6 and TNF-α) and depression symptoms, while low levels of omega-3 relative to omega-6 PUFAs have been linked to depression and suicide risk [5-8]. Lower EPA and DHA levels have also been found associated with a genetic variant of phospholipase A2 and cyclooxygenase 2 that increased risk of interferon-induced depression [9].

A profound omega-3 PUFAs deficiency is able to alter several neurotransmission systems due to its role in the membrane fluidity, at least the serotonergic and dopaminergic [10,11]. It was suggested an advantage to combining omega-3 fatty acids with a selective serotonin uptake inhibitor in the initial treatment of individuals with major depressive disorder [12]. Relative to dopamine, omega-3 PUFAs may improve dopamine neuron survival and reduce cortical monoamine oxidase [11].

At last, omega-3 deficiency has been shown to decrease glucose uptake of brain cells and decrease cytochrome oxidase activity. Glucose uptake and cytochrome oxidase activity are indicators of neuronal functional activity [13]. Omega-3 PUFAs are involved in the modulation of Brain-Derived Neurotrophic Factor (BDNF) in the hippocampus and in the regulation of the hypothalamic-pituitary-adrenal axis [14,15].

In conclusion, the potential action mechanisms of omega-3 PUFAs on depression are based on the decrease of neuro inflammation, involvement on neurotransmission and modulation of neurotrophic factors. However, some of these mechanisms still have many questions unresolved to be fully addressed in near future.

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