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Omega Fatty Acid: Role on it's Brain Developmental Activity

Csaba Pleh*

Department of Cognitive Science, Budapest University of Technology, Hungary

***Corresponding author:** Pleh C Department of Cognitive Science, Budapest University of Technology, Hungary, E-mail: vispleh@ceu.edu

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Abstract

A number of neuropsychiatric disorders, including Parkinson's disease, schizophrenia, attention deficit hyperactivity disorder, and, to some extent, depression, involve dysregulation of the brain dopamine systems. The etiology of these diseases is multifactorial, involving genetic and environmental factors. Evidence suggests that inadequate levels of n-3 (omega-3) polyunsaturated fatty acids (PUFA) in the brain may represent a risk factor for these disorders. These fatty acids, which are derived from the diet, are a major component of neuronal membranes and are of particular importance in brain development and function. Low levels of n-3 PUFAs in the brain affect the brain dopamine systems and, when combined with appropriate genetic and other factors, increase the risk of developing these disorders and/or the severity of the disease.

Keywords: Brain; PUFA; carbons

Introductio

PUFAs in the n-3 and n-6 (omega-3 and omega-6) families play a number of important physiological roles as components of cell membranes, as signaling mediators, and as precursors of signaling mediators. The role of PUFAs, particularly n-3 PUFAs, in brain development is well established [1]. There is also increasing evidence that suboptimal levels of n-3 PUFAs, as a result of inadequate diet or metabolic deficiencies, appear to interact with genetic and environmental factors in the etiologies of a variety of neuropsychiatric disorders.

PUFAs are important dietary fats containing more than one double bond, and are named according to the number of carbons they contain, the number of double bonds, and the position of the first double bond from the methyl end. Long- chain PUFAs, which are at least 20 carbons in length, have important functional roles as components of membrane phospholipids and as signaling molecules in all tissues including the brain [2]. Biologically important long-chain PUFAs include docosahexaenoic acid (DHA or 22:6n-3), a 22-carbon PUFA with six double bonds and the first double bond at the third carbon from the methyl end, is part of the n-3 class of PUFAs. DHA constitutes approximately 12–15% by weight of the total fatty acids in the human brain. The major species of n-6 PUFAs in brain is arachidonic acid (AA or 20:4n-6), which is 20 carbons in length, with four double bonds beginning with the sixth carbon from the methyl end, and which makes up 8–11% of the total fatty acids in the brain.

As elements of the cell membrane, long-chain PUFAs contribute to the membrane's physicochemical properties, and thus impact the function of lipid rafts and membrane-bound proteins, such as receptors, transporters, and ion channels.

The human body can only form carbon–carbon double bonds after the 9th carbon from the methyl end of a fatty acid [3]. Therefore, ALA and linoleic acid are considered essential fatty acids, meaning that they must be obtained from the diet. ALA can be converted into EPA and then to DHA, but the conversion (which occurs primarily in the liver) is very limited, with reported rates of less than 15% [4]. Therefore, consuming EPA and DHA directly from foods and/or dietary supplements is the only practical way to increase levels of these fatty acids in the body.

ALA is present in plant oils, such as flaxseed, soybean, and canola oils. DHA and EPA are present in fish, fish oils, and krill oils, but they are originally synthesized by microalgae, not by the fish. When fish consume phytoplankton that consumed microalgae, they accumulate the omega-3s in their tissues.

A deficiency of essential fatty acids—either omega-3s or omega-6s—can cause rough, scaly skin and dermatitis .Plasma and tissue concentrations of DHA decrease when an omega-3 fatty acid deficiency is present. However, there are no known cut-off concentrations of DHA or EPA below which functional endpoints, such as those for visual or neural function or for immune response, are impaired.

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