

Neurodegenerative Diseases: The Effect of Omega-3 Fatty Acids on Neuroinflammation

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ABSTRACT

Omega-3 fatty acids' anti-inflammatory qualities and impact on the integrity of neuronal membranes make them promising for the treatment of neurodegenerative diseases including Parkinson's and Alzheimer's. Omega-3 and omega-6 Polyunsaturated Fatty Acids (PUFAs) influence neuroinflammation and cognitive function, making them essential for brain health. Neurodegeneration and mental disorders are associated with deficiency in omega-3 polyunsaturated fats. According to clinical research, taking omega-3 supplements-especially those containing DHA and EPA-may help with cognitive performance and lower neuroinflammation in people with Parkinson's and Alzheimer's illnesses. Mechanistically, omega-3 PUFAs control inflammatory pathways and synaptic function, while DHA suppresses tau protein phosphorylation and A β buildup. Omega-3 PUFAs can be obtained through diet, such as fatty fish, although supplementation may be required, particularly in populations with limited consumption or conversion capacity. One useful application of omega-3 fatty acid fortification is providing easy methods to integrate it in everyday diets. Overall, study is needed to determine whether omega-3 fatty acids can prevent or treat neurodegenerative diseases.

Keywords: Omega-3 Fatty Acids, Neuro Degenerative Disorder, Docosahexaenoic Acid, Elcosapentaenoic Acid.

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Received: 01-12-2024;

Revised: 14-02-2025;

Accepted: 29-04-2025.

INTRODUCTION

Omega-3 fatty acids belong to the antilipemic class and are important in the treatment of hyperlipidemia. They may also be beneficial in the treatment of neurodegenerative illnesses. Omega-3 fatty acids are noted for their many therapeutic benefits and are mostly utilized to treat and maintain hyperlipidemia. They have promise in neurodegenerative disorders such as Alzheimer's Disease (AD) because of their capacity to maintain cognitive function, lower inflammation and improve brain health. One essential component that affects glial cell activity and cognition, especially in the early stages of AD, is Docosahexaenoic Acid (DHA). Prospective studies indicate a link between higher omega-3 consumption and reduced AD risk, despite conflicting information on the effectiveness of omega-3 in preventing AD or cognitive decline being presented by observational research and clinical trials. Interestingly, people with AD frequently have lower omega-3 levels than cognitively normal people counterparts in good health. The way it works is by maintaining

synaptic connections and neuronal integrity, especially in areas of the brain that are important for memory, like the hippocampus. While studies are still being conducted to determine the exact dosage, pharmacodynamics and pharmacokinetics, omega-3 fatty acids show promise as an adjuvant treatment for the treatment of hyperlipidemia and the prevention of neurodegenerative diseases.¹

Symptoms of Impulsivity and Aggression

In both mental and non-psychiatric groups, a lack of omega-3 fatty acids is linked to aggressive behaviors, impulsivity and hostility. Studies on rats have also examined the connection between omega-3 PUFAs and aggression and the results indicate that both an omega-3 PUFA shortage and high omega-6 PUFA consumption are associated with an increase in aggressive behaviors. The general population appears to respond better to omega-3 delivery when it comes to decreased hostility. According to a study done on a sample of Australian prisoners, the degree of hostility and aggressive behavior was inversely correlated with the levels of n-3 PUFAs. The meta-analysis of multiple trials suggests that omega-3 fatty acid supplementation can be beneficial in preventing and reducing aggressive behaviors in adults and children. The connection between aberrant PUFAs.²



DOI: 10.5530/ijopp.20250251

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The Role of Omega-3 Fatty Acids in Brain Health

Particular vitamins have drawn attention for their potent antioxidant properties and Polyunsaturated Fatty Acids (PUFAs) for their potential to improve health. Polyunsaturated Fatty Acids (PUFAs) include omega-3 and omega-6 fatty acids, which have 2 or more double bonds in their carbon chain backbone. Among the omega-6 fatty acids are Arachidonic Acid (AA), γ -Linolenic Acid (GLA) and Linoleic Acid (LA). 2 types of omega-3 fatty acids are Docosahexaenoic Acid (DHA) and Eicosapentaenoic Acid (EPA). However since humans can only synthesize so much of these fatty acids, they should be obtained through food or supplementation. Impact on cognitive function, aging and development. Omega-3 fatty acid insufficiency is. This highlights the significance of omega-3 fatty acids. In contrast, deficiencies in Eicosapentaenoic Acid (EA) and Docosahexaenoic Acid (DA) lead to mental illnesses and regulate inflammatory processes and mental health. Their effects also extend directly to receptor function and neural membrane fluidity. Long recognized for their critical role in maintaining brain homeostasis, omega-3 supplements and enriched meals have limited application in psychiatry due to the lack of clear outcomes from randomized clinical trials examining their therapeutic potential. To assess whether omega-3 fatty acids are useful in preventing and treating NDs, high-quality clinical trials are desperately needed.³

By inhibiting tau protein phosphorylation, DHA stops the disassembly of intraneuronal microtubules and the development of neurofibrillary tangles. DHA can also lessen the amount of A β that accumulates in neurons and prevent it from forming, which lessens the toxicity that A β and neuronal death c. An investigation conducted recently indicates that DHA, but not AA, can inhibit the synthesis of inducible nitric oxide synthase, the generation of reactive oxygen species, the activation of tumor necrosis factor α in microglia cells and the formation of oligomeric A β (oA β) and the oA β -induced increase of phosphorylated cytosolic phospholipase A2. In oA β -stimulated microglia, DHA can regulate AA metabolism by downregulating oxidative and inflammatory pathways and increasing the Nrf2/heme oxygenase-1 antioxidative pathway. Kotania S. *et al.* (2006) reported that the supplementation of 240 mg/day of AA and DHA produced a significant improvement in immediate memory and attention score in a group of patients with mild cognitive dysfunction and organic brain lesions but with no diagnosis of AD (no-AD) However, no significant differences were observed when compared to the AD group. The OmegAD study (2015) assessed how supplementing with EPA (0.6 g/day) and DHA (1.7 g/day) affected the patients' ability to produce resolvins over six months. Supplementing with EPA and DHA preserved the generation of resolvins, preventing detrimental alterations in cognitive function, according to an analysis of mononuclear cells from the peripheral blood of patients.⁴

Mechanisms of Action: Omega-3s Combat Neurodegeneration

The effects of supplementing patients with Parkinson's or Alzheimer's disease with omega-3 polyunsaturated fatty acids on their cognitive function are reviewed in detail in this study. Omega-3 and omega-6 PUFA classes include α -linolenic acid, eicosapentaenoic acid and docosahexaenoic acid, respectively and linoleic acid and arachidonic acid, respectively, are the main groups of PUFAs. When it comes to PUFAs, DHA and ARA are the most significant. To be exact, DHA makes up 10-20% of all lipids in the brain and more than 90% of ω -3 PUFAs. Synaptic terminals, mitochondria and endoplasmic reticula are the primary locations where it is integrated into phosphatidylcholine, phosphatidylethanolamine and phosphatidylserine, with lesser quantities. DHA can, in fact, alter physiological and cellular processes such neurotransmitter release, gene expression, myelination, membrane fluidity and the release of neuroinflammation and proliferation of neurons While ARA is produced from LA by desaturation and carbon chain elongation, DHA is produced from ALA. Since humans lack the conversion enzyme 3-desaturase, they are able to produce both saturated and Monounsaturated Fatty Acids (MUFAs), which are needed to produce ALA and LA. Due to the identical conversion enzyme requests made by LA and ALA, there is competitive inhibition between the two substrates. The transformation of omega-3 fatty acids into omega-6 fatty acids is aided by delta-6-desaturase. But an increase in LA consumption might tip the scales in favor of converting omega-6 PUFA, which would prevent ALA from converting to DHA. After intestinal and hepatic metabolism, esterified DHA in food is released by intestinal lipases in the small intestine as free, unesterified form (DHA-FFA). It can also be found esterified in phosphatidylcholine and triglycerides, or as free DHA linked to low-density lipoprotein or albumin. Endothelial lipases, Fatty Acid-Binding Proteins (FABP) and Apolipoprotein E (ApoE) dissociate the various forms in the Blood-Brain Barrier (BBB) using both active and passive mechanisms. The DHA is carried throughout the central nervous system by astrocyte-produced FABP and ApoE. Through the action of coenzyme A, DHA is primarily integrated into membrane phospholipids in the stereo specifically numbered-2 position. Yet DHA can be liberated from membrane phospholipids via hydrolysis processes aided by phospholipase. In response to dynamic cellular events and obstacles during development and aging, both synthesis and hydrolysis represent mechanisms. Additionally crucial to eicosanoids' participation in inflammation is their synthesis from DHA, EPA and ARA (prostaglandins, thromboxanes and leukotrienes). The phospholipid is hydrolyzed by the Phospholipase A2 enzymes (PLA2), which liberate fatty acids. It follows that there is an increase in free fatty acid levels due to an inflammatory stimulation involving particular cell activation. Bioactive lipid release is mostly associated with 3 forms of PLA2: the PLA2 that is cytosolic calcium-dependent through

the action of coenzyme A, DHA is primarily integrated into membrane phospholipids in the stereospecifically numbered-2 position. Yet DHA can be liberated from membrane phospholipids via hydrolysis processes aided by phospholipase. In response to dynamic cellular events and obstacles during development and aging, both synthesis and hydrolysis represent mechanisms. Additionally crucial to eicosanoids' participation in inflammation is their synthesis from DHA, EPA and ARA (prostaglandins, thromboxanes and leukotrienes). The phospholipid is hydrolyzed by the Phospholipase A2 enzymes (PLA2), which liberate fatty acids. It follows that there is an increase in free fatty acid levels due to an inflammatory stimulation involving particular cell activation. Bioactive lipid release is mostly associated with three forms of PLA2: the PLA2 that is cytosolic calcium-dependent, cPLA2 is one of them that exhibit substrate selectivity for phospholipids that include Arachidonic Acid (AA). Though cPLA2 may also hydrolyze phospholipids containing EPA, under some circumstances, the low amount of this fatty acid permits cPLA2 to release AA. Cyclooxygenase (COX) and 5-Lipoxygenase (5-LOX) metabolize prostaglandins, leukotrienes and thromboxanes, which in turn control the regulation of inflammation (Figure 1). ARA is a precursor of the 5-series leukotrienes, thromboxanes and the 2-series prostaglandins. As a result, EPA acts to reduce inflammation while ARA promotes it. Additionally, 5-LOX produces anti-inflammatory eicosanoids like the E-series resolvins from EPA and the D-series resolvins from DHA. These eicosanoids include maresins, protectins and resolvins.

In Human, Mature males generally convert less than 5% of ALA to DHA, whereas females convert more effectively. The nutrition of the fetus throughout pregnancy is affected by this variation. Generally speaking, women consume less omega-3 than men do and as they age, their capacity to convert particularly delta-6 desaturase activity to decreases. For older people in particular, this means that preformed omega-3 dietary supplements are necessary for a sufficient intake of EPA and DHA. Modern diets heavy in omega-6 and deficient in omega-3 have a detrimental effect on cognitive function and aging when combined with a decrease in physical activity. Maintaining an appropriate intake of EPA and DHA is stressed in current recommendations.⁵

Evidence from Research Studies

Neurological disorders (e.g., depression, ADHD, autism, Alzheimer's, Parkinson's) and macular degeneration have been shown to respond effectively to treatment with omega 3 fatty Acids (DHA and EPA).⁶ Early-stage neurodegenerative disorders can benefit from the use of omega-3 fatty acid treatment, which is a safe and well-tolerated biological intervention.⁷

The activities of O3FA as FFAR agonists have been studied by molecular docking studies, which ended up resulting in the development of receptor-specific targeted agonists for neurodegenerative disease treatment.⁸ n-3 PUFAs can prevent neuroinflammation and microglial activity, safeguard astrocyte function to produce neurotrophins and improve neurodegeneration by synthesizing n-6 PUFAs, the precursors of inflammatory mediators.⁹ Cyclooxygenase-2/Lipoxygenase

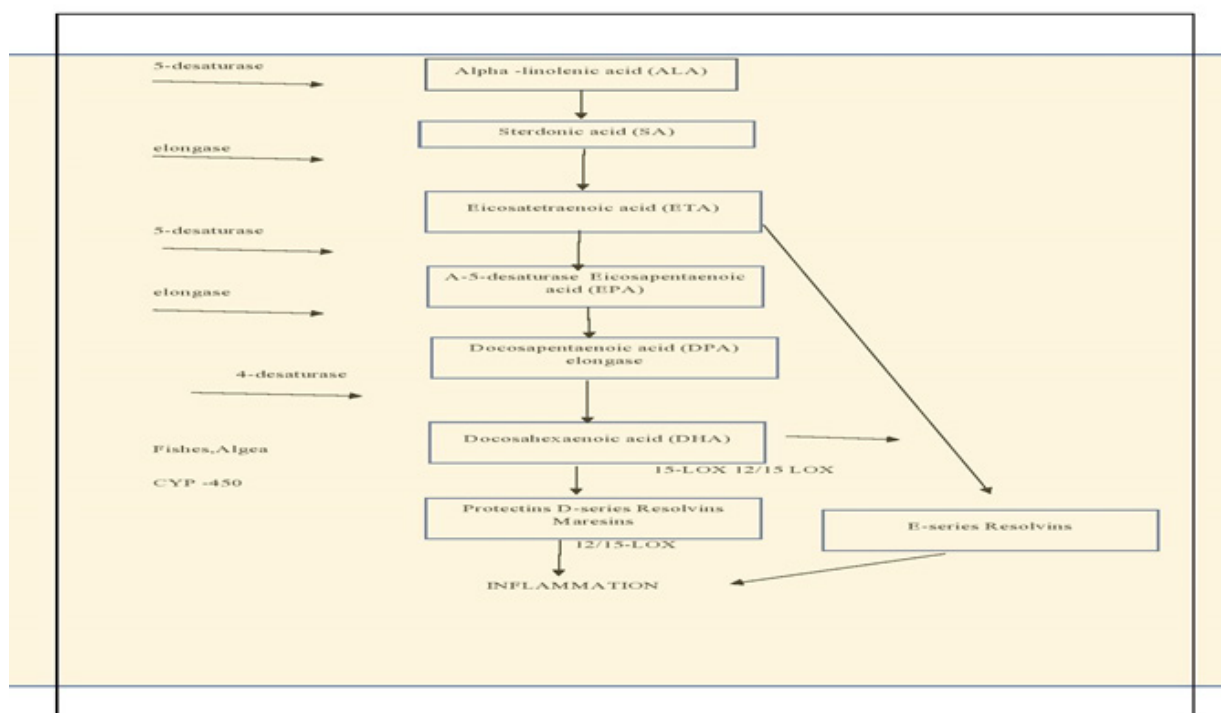


Figure 1: It represent the “mechanism of action”: Omega-3s Combat Neurodegeneration.

(COX-2/LOX) pathways biosynthesize resolvins and protectins from omega-3 fatty acids, which contribute to controlling inflammation in neural tissues.¹⁰ Omega(n)-3 Polyunsaturated Fatty Acids (PUFAs), which are primarily found in seafood, may mitigate the effects of Alzheimer's Disease (AD) by glial activity and membrane function. It may also compete with inflammatory n-6 metabolites to clear tau phosphorylation and A β plaques, hold down the release of proinflammatory cytokines and block the nuclear factor-kappa-B pathway, which decreases neurodegeneration and improves memory.¹¹

PRACTICAL APPLICATIONS

Presumably, an essential component of the Mediterranean diet, Alpha-Lipoic Acid (ALA) is the most prevalent ω -3 fatty acid in the western diet. Moreover, ALA is well-known for its potential benefits against depression, inflammation and neuroprotection. Presumably, an essential component of the Mediterranean diet, Alpha-Lipoic Acid (ALA) is the most prevalent ω -3 fatty acid in the western diet. Moreover, ALA is well-known for its potential benefits against depression, inflammation and neuroprotection. Although it can be obtained from meals including green leafy vegetables, whole-grain cereals, dairy products and meat, ALA is present in large amounts in plant oils, seeds and walnuts.¹²

Plants are the primary provider of ω -3 PUFAs in maritime environments. In many plant-based diets (such as linseed/flaxseed, soybean and rapeseed oils), ALA is the most prevalent ω -3 PUFA.¹³

As the precursor of EPA and DHA, it too has an animal origin. They are further concentrated in the food chain and are mostly produced by marine algae.¹⁴ For many years, especially in industrialised nations, food fortification-fortifying food with vitamins A, B and D, iodine and iron-has been used as a successful technique to combat nutritional deficiencies. A term used to describe common foods that, in addition to having the usual nutritional content, also contain some substance that has extra health benefits is "designer foods." In traditional medicine, they have been used for a long time in East Asia to lower the risk of many ailments. They are also known as "functional" or "fortified" foods. Designer foods improve diet and prevent shortages by avoiding the need to change eating habits and routinely delivering sufficient levels of nutrients.¹⁵ Foods enhanced with long-chain ω -3 polyunsaturated fatty acids are gaining popularity because of their affordability, easy availability and growing knowledge of the health advantages they provide. It is necessary to consume enough ω -3 fatty acids to counteract the effects of ω -6 fatty acids, especially considering how little ALA is converted into EPA and DHA.¹⁶

Meat

Although meat and animal products are an important part of the diet, they are low in DHA, EPA and ALA. There are meat

products made from adding ω -3 PUFAs to animal feed, but too much of these fatty acids in diet might negatively impact rumen activity and milk output.¹⁷ Linseed oil is often added to the diets of lamb and beef cattle in order to improve the level of ω -3.¹⁸ To effectively incorporate EPA and DHA into animal feed, a combination of fish oil and marine algae containing long-chain ω -3 PUFAs has been employed. This has been demonstrated with lambs.¹⁹ Dairy products: Consumers find dairy products enhanced with omega-3 fatty acids appealing. As a result, several designer milks have been created and studies have been done on how these affect human health.²⁰ A typical approach of raising the amount of ω -3 PUFA in milk involves feeding ruminants' diets with linseeds and/or linseed oil, marine oils and/or algae, or a mix of the aforementioned.^{21,22} Sunflower seeds are added to the cow's feed to create designer milk that has a higher CLA concentration. Nonetheless, rumen biohydrogenation limits the rise in ω -3 fatty acids in milk.²³ Nutrient enrichment is another method of acquiring ω -3 supplemented milk products. Consuming milk supplemented with minerals, vitamins, EPA, DHA, oleic acid, fish oil and other nutrients has been linked to advantages in health.²⁴

Bakery products

Bread is a practical option for ω -3 fatty acid fortification since lipids are added to the dough during the production process. Eggs and dairy products containing ω -3 PUFAs have been used to make breads, cakes and cookies.²⁵ Eggs: The blood pressure and serum lipid profile of some patient groups were observed to improve while consuming such ω -3 enhanced eggs. A high-CLA diet has been used to produce other designer eggs, which significantly reduced inflammation in mice when tested on them.^{26,27} An economical, nutrient-dense food source that is commonly consumed, eggs are easily enhanced with extra nutrients. By altering the fatty acid makeup of egg yolks, the concept of eggs as designer foods emerged in the 1930s.²⁸ Altering the chickens' food is a typical method to increase the amount of omega-3 fatty acids in their eggs. If hens are fed sunflower, flaxseed, or marine algal oils, the saturated fatty acids in their yolks can be substituted with omega-3 fatty acids.^{29,30}

CONCLUSION

Nootropics, especially omega-3 fatty acids, are essential for reducing inflammation, promoting brain tissue regeneration and maintaining cognitive performance. DHA derivatives improve cognition in the early stages of Alzheimer's disease, while supplementation experiments have mixed results. Decreased omega-3 levels have been associated with Alzheimer's disease in observational studies. Addiction and impulsivity are correlated with omega-3 insufficiency, which can be treated with supplements. Mechanistically, DHA decreases A β deposits by preventing neuroinflammation and tau protein phosphorylation. Including omega-3-rich foods in meals, such as dairy and baked products, is one practical strategy. Despite the encouraging effects,

more high-quality trials are necessary to maximize omega-3's effectiveness in neurodegenerative disorders.

CONFLICT OF INTEREST

The authors declare that there is no conflict of interest.

AUTHOR CONTRIBUTIONS

MILAGRIN XAVIER has revised and PRIYADHARSHINI R conceptualized the review, conducted the literature search and wrote the manuscript. SREELAKSHMI S and TANUVARTHINI S B contributed to data analysis, writing and revising the manuscript. AISHWARYA S assisted with the methodology and provided critical revisions. HEMA V supervised the project, provided mentorship and approved the final manuscript. All authors have read and approved the final manuscript, which was subsequently read and approved by the whole author.

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Cite this article: Priyadharshini R, Sreelakshmi S, Tanuvarthini S.B, Aishwarya S, Hema V, Xavier M. Neurodegenerative Diseases: The Effect of Omega-3 Fatty Acids on Neuroinflammation. *Indian J Pharmacy Practice*. 2025;18(4):362-6.