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Role of Omega Fatty Acids in Atherosclerosis and Coronary Artery Disease

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Abstract

IOmega-3 long chain polyunsaturated fatty acids (PUFAs) have been popularized in recent years as beneficial nutrients with cardioprotective effects. Omega-3 PUFAs are so named because of a double bond between the 3rd and 4th carbon of the polycarbon chain.

Keywords: Fatty acids; Cardioprotective; Triglyceride therapy

Introduction

Omega-3 long chain Polyunsaturated Fatty Acids (PUFAs) have been popularized in recent years as beneficial nutrients with cardioprotective effects. Omega-3 PUFAs are so named because of a double bond between the 3rd and 4th carbon of the polycarbon chain. They are "poly-unsaturated" with hydrogen atoms, as their carbon chains contain multiple double bonds. Three omega-3 long chain PUFAs are typically discussed in the context of medical therapy, the first being Alpha-Linolenic Acid (ALA). ALA is an essential precursor omega-3 that is converted by the body into Eicosapentaenoic Acid (EPA) and docosahexaenoic acid (DHA) [1]. However, this conversion is not very efficient in humans. Omega-3s are best obtained through the diet, but they are available as supplements as well. Omega-3s are common in poultry and most famously found in fish such as salmon, herring, trout, and sardines [2].

Role in Atherosclerotic Disease

The role of omega-3 PUFAs in the treatment of atherosclerosis is not as clear as its role in triglyceride therapy, but there is strong evidence to suggest clinical efficacy. As stated earlier, EPA and DHA have the greatest therapeutic effect and were the omega-3s most often studied. Omega-3 PUFAs influence gene transcription [3]. It is thought that when omega-3s incorporate into the cellular membrane, they disrupt cholesterol rafts, changing the fluidity of cell membranes. This releases endothelial relaxing factors, like nitric oxide, decreasing vascular tone [4]. It has been shown that after 3 months of omega-3 supplementation in obese adolescents with demonstrated vascular inflammation, vasoconstrictive responses and endothelial function improved [5].

In addition to changing the endothelial response, omega-3 PUFAs seem to modulate the inflammatory response through inhibition of cyclooxygenase-2 (COX-2). While this mechanism is unclear, omega-3 PUFAs incorporate directly into the plaque. Decreased COX-2 activity is associated with decreased release of matrix metalloproteinases (MMPs), which have been implicated in the thinning of the atherosclerotic plaque cap that makes the plaque more prone to rupture [5]. It has been demonstrated that when patients were treated with omega-3s prior to surgery, carotid artery plaques had decreased levels of RNA for MMPs 7,9, and 12 [6]. The same study found that in the 3-week treatment period the plaques showed a decreased number of foam cells and T-cells and had less inflammation and increased stability. However, over the short, 3-week presurgical treatment period, there was no change in primary outcome [7]. In another placebo-controlled study, obese adolescents treated with EPA for one year had improved vascular function; reduced inflammation; and decreased levels of lymphocytes, monocytes, TNF-alpha, interleulin-1, and interleukin-6 [8].

COX-2 inhibition affects platelet aggregation as well. EPA has been shown to reduce platelet aggregation and may have a beneficial effect on certain cardiovascular thrombotic disorders [9]. These effects may be enhanced by reduction in serum lipid levels. Elevated postprandial triglycerides were shown to be associated with increased plasminogen activator 1 and factor 7, increasing thrombosis risk and CHD events. By altering lipid levels and regulating inflammatory mediators, endothelial function, inflammation, plaque stability, and platelet aggregation, omega-3 PUFAs have demonstrated a multifaceted, protective effect against atherosclerosis.

Conclusion

Omega-3 fatty acids can significantly reduce the occurrence of CVD events in patients with coronary artery disease. A food-based approach to increasing omega-3 fatty acids is preferable, although supplements are a suitable alternative. The potential mechanisms by which omega-3 PUFAs benefit atherosclerosis include lowering triglyceride levels, improving the effects of statin therapy, improving endothelial function, blocking pro-inflammatory pathways, and impairing platelet aggregation though COX-2 inhibition.

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