

Role of Omega-3 Fatty Acids in Coronary Disease Prevention Strategies

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Introduction

Coronary Heart Disease (CHD) remains the leading cause of death worldwide, driven primarily by atherosclerosis, lifestyle factors and metabolic disorders. Among various nutritional strategies proposed for cardiovascular prevention, omega-3 fatty acids have gained substantial attention due to their biological properties and epidemiological associations with reduced cardiovascular morbidity and mortality. Omega-3 fatty acids, especially Eicosapentaenoic Acid (EPA) and Docosahexaenoic Acid (DHA), found predominantly in fatty fish, possess a unique profile of lipid-modifying, anti-inflammatory, anti-thrombotic and cardioprotective effects. As dietary supplements or functional food components, omega-3s are now considered valuable tools in the prevention and management of coronary artery disease, both as primary prevention and as part of secondary prevention strategies post-myocardial infarction. This paper explores the scientific basis, clinical evidence and therapeutic implications of omega-3 fatty acids in coronary disease prevention [1].

Description

Omega-3 fatty acids play a vital role in modulating key physiological pathways that directly influence cardiovascular health. EPA and DHA help reduce plasma triglyceride levels by decreasing hepatic production of Very-Low-Density Lipoprotein (VLDL) and enhancing the clearance of chylomicron remnants. This lipid-lowering effect is dose-dependent and has been observed consistently in both healthy individuals and those with hyperlipidemia. Additionally, omega-3s exert a mild effect on raising High-Density Lipoprotein (HDL) cholesterol and may modestly influence Low-Density Lipoprotein (LDL) particle size, improving overall lipid profiles and reducing atherogenicity. Beyond lipid modulation, omega-3s have significant anti-inflammatory properties. Chronic low-grade inflammation contributes to the development and progression of atherosclerosis and omega-3s counteract this by suppressing the production of pro-inflammatory cytokines like TNF- α , IL-1 β and IL-6. They also enhance the production of anti-inflammatory resolvins and protectins, derived from EPA and DHA. These biochemical effects contribute to the stabilization of atherosclerotic plaques, reducing the likelihood of rupture and thrombosis and thus lowering the risk of acute coronary events such as myocardial infarction. Omega-3s also improve vascular endothelial function, which is critical in maintaining arterial health. Endothelial dysfunction is a key early marker of atherosclerosis and is linked with impaired nitric oxide bioavailability, oxidative stress and vascular inflammation. Clinical studies have shown that EPA and DHA enhance endothelial-dependent vasodilation and reduce arterial stiffness, leading to better blood pressure control and vascular reactivity. These benefits are particularly relevant in hypertensive patients or those with metabolic syndrome, where endothelial dysfunction is prominent.

Furthermore, omega-3s exert anti-arrhythmic effects, making them crucial in the

prevention of sudden cardiac death. EPA and DHA alter cardiac cell membrane composition, modulating ion channel function and stabilizing myocardial electrical activity. By reducing heart rate variability and the likelihood of ventricular arrhythmias, omega-3s contribute to a decreased risk of fatal cardiac events. Studies such as the GISSI-Prevenzione and the JELIS trial have confirmed these protective effects, especially in post-myocardial infarction populations. Recent clinical trials have added nuance to our understanding of omega-3 supplementation. The REDUCE-IT trial demonstrated that high-dose purified EPA significantly reduced the risk of cardiovascular events in patients with elevated triglycerides despite statin therapy. In contrast, the STRENGTH trial, which used a mixed EPA/DHA formulation, did not show similar benefits, suggesting a possible superiority of EPA-only supplements. These discrepancies highlight the importance of formulation, dose and patient selection in determining omega-3 efficacy and underline the need for personalized approaches in their clinical use [2].

Conclusion

Omega-3 fatty acids represent a well-tolerated and scientifically validated component of cardiovascular disease prevention strategies, particularly in the context of coronary heart disease. Through a combination of lipid-lowering, anti-inflammatory, endothelial-enhancing and anti-arrhythmic mechanisms, EPA and DHA help mitigate several modifiable risk factors associated with atherosclerosis and coronary events. While early observational studies and clinical trials laid the foundation for their use, recent high-quality trials like REDUCE-IT have further strengthened the evidence base, especially for purified EPA formulations in high-risk populations. However, results have varied based on study design, patient characteristics and formulation types, pointing to the necessity of tailoring omega-3 supplementation to individual patient profiles. Given the global burden of CHD and the relative safety profile of omega-3s, their integration into preventive cardiology whether through dietary means or pharmacological supplementation remains a compelling and proactive strategy. Ongoing research will continue to refine the therapeutic parameters and broaden their application in cardiovascular medicine.

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