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1: [Circulation](#). 1989 Sep;80(3 Pt 1):I109-23.

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Mechanisms responsible for inhibition of vein-graft arteriosclerosis by fish oil.

[Sarris GE](#), [Fann JJ](#), [Sokoloff MH](#), [Smith DL](#), [Loveday M](#), [Kosek JC](#), [Stephens RJ](#), [Cooper AD](#), [May K](#), [Willis AL](#), et al.

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Favorable changes in lipoproteins, inhibition of platelet aggregation, reduction of serum thromboxane (TX), altered plasma-membrane fluidity, and reduced production of growth factors (mitogens) have all been implicated as possibly being involved in the inhibition of arteriosclerosis by fish oil (FO), which is rich in omega 3 fatty acids; however, causal relations are mostly lacking. Several putative mechanisms responsible for the salutary effects of FO were investigated in a canine model of accelerated vein-graft arteriosclerosis. Venoarterial autografts (N = 192) were implanted in 48 hypercholesterolemic dogs divided into six groups: group A, control; B, FO (as MaxEPA, 200 mg/kg/day eicosapentaenoic acid); C, aspirin (ASA, 50 mg/kg/day); D, TX synthetase inhibitor (TXSI [CGS-12970], 10 mg/kg/day); E, FO + ASA; and F, FO + TXSI. At sacrifice 3 months later, there was no significant difference in plasma lipoproteins, hepatic low density lipoprotein-receptor concentration, red blood cell fragility, bleeding time, or platelet count compared with controls; the decrease in platelet aggregation (30 +/- 5% [mean +/- SEM]) was similar in all treatment groups. Arterialized vein-graft intimal thickening was significantly inhibited by FO (with or without ASA), while ASA alone was ineffective. Conversely, serum TX was significantly lower only in the ASA and FO + ASA groups. Serum mitogenic activity was higher at 3 months in the control group versus all treatment groups. Compared with baseline values, serum mitogenic activity rose significantly over time in the control and the TXSI groups, and an increase or rising trend was present in all other treatment groups except for the FO-treated animals. Thus, the salutary biologic effect of FO in this hypercholesterolemic model of arterIALIZED vein grafts may have been more related to in vivo inhibition of platelet-mitogen growth factor release than to changes in lipoproteins, low density lipoprotein receptors, platelet function, or eicosanoid metabolism. These observations underscore the need for further studies to clarify the interactions between FO (omega 3 fatty acids) and paracrine cellular mitogenic factors in the context of atherosclerosis prevention.

PMID: 2766520 [PubMed - indexed for MEDLINE]



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