

Eicosapentaenoic acid for prevention of major coronary events

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Mitsuhiko Yokohama and colleagues ¹ noted that non-fatal coronary events, but not cardiac deaths, were lower in hypercholesterolaemic Japanese patients who were given 1800 mg per day of the omega 3 fatty acid eicosapentaenoic acid (EPA) than in controls. At first, these results seem to contrast with other published data which indicate that omega 3 fatty acid preparations, including the end product of this metabolic series, docosahexaenoic acid (DHA), also reduce cardiac death. ² However, these findings are expected, given the chemistry and metabolism of EPA versus DHA.

EPA, by contrast with DHA, is not incorporated very efficiently into cell phospholipids (including cardiac myocytes). Since the antiarrhythmic effects, and the reduction of cardiac mortality, of omega 3 are related to the incorporation of these fatty acids into heart phospholipids, the effects of EPA would be lower than those of DHA. ^{3,4}

No data on basal concentrations of omega 3 fatty acids were presented. They should be high compared with those in Western populations. After treatment, EPA concentrations in plasma lipids should increase, while those of DHA should not. In fact, they should be high already owing to fish intake, and the conversion of EPA to DHA, rather inefficient under conventional dietary conditions, should be even lower in the presence of high levels of DHA. ⁵ Data on fatty acid profiles before and after treatment would facilitate interpretations.

EPA inhibits the production of proinflammatory eicosanoids more efficiently than DHA, since EPA competes with the omega 6 fatty acid arachidonic acid for oxygenases. Non-fatal coronary events, possibly related to inflammatory processes, should thus be best counteracted by EPA.

We declare that we have no conflict of interest.

References

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