

EDITORIALS

Old study sheds new light on the fatty acids and cardiovascular health debate

American Heart Association advice on omega 6 PUFAs cast into doubt

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In a linked research paper (doi:10.1136/bmj.e8707), Ramsden and colleagues report “new” data from an old trial that shed light on the long running debate on whether increasing dietary linoleic acid intake reduces the risk of cardiovascular disease (CVD) or death.¹ Research conducted in the 1960s and 1970s suggested that some of the commonly occurring dietary saturated fatty acids raise total and low density lipoprotein cholesterol concentrations, whereas the omega 6 polyunsaturated fatty acid (PUFA) linoleic acid lowers total and low density lipoprotein cholesterol concentrations.²

Linoleic acid is present in high amounts in vegetable oils such as corn, sunflower, safflower, and soybean oils and in margarines made from these oils. It is the most prevalent PUFA and omega 6 PUFA in most Western diets. As a result of the effects of linoleic acid on cholesterol concentrations, lowering intake of saturated fat and increasing that of PUFAs has been a cornerstone of dietary advice, with the aim of decreasing the risk of cardiovascular disease (CVD).³

The American Heart Association recently repeated advice to maintain, and even to increase, intake of omega 6 PUFAs.⁴ This advice has caused some controversy,^{5,7} because evidence that linoleic acid lowers the risk of CVD is limited—most trials that claimed to investigate the effect of exchanging saturated fat for linoleic acid involved multiple dietary changes or multiple interventions (or both).⁵ In particular, studies lowered trans fatty acid intake or increased omega 3 PUFA intake (or both) at the same time as increasing linoleic acid intake. The impact on CVD risk or mortality of replacing saturated fat with linoleic acid without changes in other fatty acids has rarely been investigated, and no large randomised controlled trial has recently explored this important question.

However, the newly analysed data from the Sydney Diet Heart Study, a randomised controlled trial conducted from 1966 to 1973 and comprising 458 men aged 30–59 years with a recent coronary event (myocardial infarction, acute coronary insufficiency, or angina), fills this gap. Participants were randomised to a diet rich in linoleic acid or continuation of their habitual diet.⁸ Both groups were treated the same in other

respects and received the same advice. Baseline dietary intake data showed an average linoleic acid intake of about 6% of energy and an average saturated fatty acid intake of about 16% of energy. The linoleic acid group was instructed to increase PUFA intake to 15% of energy and to reduce saturated fatty acid intake to less than 10% of energy; participants were provided with liquid safflower oil and a safflower oil based margarine to be used instead of animal fats for cooking, baking, and spreading. Safflower oil is 75% linoleic acid and does not provide other PUFAs. Follow-up was a median of 39 months. Total cholesterol was lowered by an average of 13% in the linoleic acid group. Despite this, higher all cause mortality in the linoleic acid group was reported in 1978,⁸ but death from CVD and coronary heart disease (CHD) were not reported.

In the linked study, Ramsden and colleagues have analysed the original data using modern approaches to create a novel and interesting piece of work. The original data were recorded on a nine track magnetic tape and had to be recovered and converted to a useable format, a not inconsiderable task. The results confirm that the linoleic acid group had a higher risk of all cause mortality (hazard ratio 1.62, 95% confidence interval 1.00 to 2.64), and now show a higher risk of mortality from CVD (1.70, 1.03 to 2.80) and CHD (1.74, 1.04 to 2.92).

The authors then used the new data generated from the Sydney Diet Heart Study to update an earlier meta-analysis.⁵ Two other linoleic acid intervention trials that reported CHD and CVD mortality were included.^{9,10} This updated analysis reported an increased risk of death from CHD (1.33, 0.99 to 1.79) and CVD (1.27, 0.98 to 1.65), although the results were not significant. These findings argue against the “saturated fat bad, omega 6 PUFA good” dogma and suggest that the American Heart Association advisory that includes the statement “higher [than 10% of energy] intakes [of omega-6 PUFAs] appear to be safe and may be even more beneficial”⁴ may be misguided. The more cautious UK dietary recommendations on fat and fatty acids, which include the statement, “There is reason to be cautious about high intakes of omega 6 PUFAs,”¹¹ seem fully justified in the light of the current study’s findings.

The new analysis of these old data provides important information about the impact of high intakes of omega 6 PUFAs, in particular linoleic acid, on cardiovascular mortality at a time when there is considerable debate on this question.^{4-7 11} The findings underscore the need to properly align dietary advice and recommendations with the scientific evidence base. It is important when assessing this evidence base that subtle, and in some cases unsubtle, aspects of study design are properly considered. For example, outcome of studies in which intakes of saturated and trans fatty acids are lowered while intakes of omega 6 fatty acids and omega 3 PUFAs are increased may be most strongly influenced by changes in trans and omega 3 fatty acids. They should not be interpreted as showing an effect of omega 6 PUFAs.

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