

Saturated fat has been unfairly demonised

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While there may be room for legitimate debate about the proportion of coronary heart disease (CHD) that can be attributed to high saturated fat diets, it is time for the sceptics (and vested interests) to abandon denial, stop cherry-picking negative studies and accept the evidence! A causal relationship between saturated fat consumption and CHD is now scientifically convincing in range, quality and consistency.

It has long been universally accepted that increased saturated fat consumption leads to increased total, low-density lipoprotein (LDL), and total/high-density lipoprotein (HDL) cholesterol and that increased total, LDL, and total/HDL cholesterol leads to increased CHD, but sceptics have considered this to be indirect and insufficient evidence that saturated fat and CHD are causally linked. However, the more direct line of evidence, based on randomised controlled trials and cohort studies, demonstrating associations between low-saturated-fat diets and reduced CHD incidence, is now convincing.

It is important to understand that the effect of saturated fat on CHD cannot be examined in isolation from other components of the diet, because when saturated fat is removed from the diet it must be replaced with another macronutrient such as protein, carbohydrate, or other types of fat. Thus, the best science shows the effects of replacing saturated fat with some other component of the diet. In this regard, there is unanimous agreement by expert groups,¹ even the sceptical ones,² that replacing saturated fat with polyunsaturated fat reduces the risk of CHD.

The effect of saturated fat on blood lipids is unequivocal and must surely rank as one of the most extensively researched and well substanti-

ated effects of diet on a risk factor for chronic disease. The results of a meta-analysis of 90 randomised controlled trials of dietary fat modification showed that replacing 5% of energy from saturated fat (about 12 g/d, the amount in 25 g of butter or 45 g of cheese) with polyunsaturated fat reduced LDL cholesterol concentration by 0.26 mmol/L and total/HDL cholesterol by 0.18; the reduction with monounsaturated fat being slightly less, 0.21 mmol/L for LDL cholesterol and 0.15 for the total/HDL ratio.³

There is similarly overwhelming evidence that blood cholesterol is a major cause of CHD. The Prospective Cohort Collaboration,⁴ a meta-analysis of individual participant data from 61 prospective studies with 55 000 vascular deaths, showed that a 1 mmol/L lower total cholesterol was associated with a halving of CHD risk in both sexes at ages 40–49 years, a 33% reduction at ages 50–69, and a 17% reduction at ages 70–89 years. Moreover, multiple meta-analyses of randomised controlled trials show that reducing blood cholesterol with drugs decreases CHD risk in a dose-dependent manner; 19% reduction in coronary mortality per 1 mmol/L reduction in LDL cholesterol.⁵

In contrast, until recently there was some uncertainty about the evidence directly linking saturated fat intake with risk of CHD. The necessary studies are notoriously difficult to do well; a problem not well understood by the sceptics who have misinterpreted negative studies as evidence of no effect. Cohort studies are plagued by measurement error (of dietary intake) and confounding, while randomised trials of sufficient size and duration are extremely expensive to undertake, and maintaining participants on allocated diets is almost impossible. However, several recent

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publications have brought clarity of understanding to the debate.

In an ambitious, well-designed and landmark project, Jakobsen et al. conducted a meta-analysis after obtaining individual participant data from 11 cohort studies.⁶ All studies used validated dietary assessment methods, follow-up ranged from four to 10 years, and included 2155 CHD deaths among 344 696 participants. The results showed, after adjustment for potential confounders, that the risk of CHD death decreased by 26% (95% CI, 11–39%) per 5% lower energy intake from saturated fat and concomitant increase in polyunsaturated fat.

With regard to randomised trials, the best evidence has often been overlooked or examined piecemeal, possibly because most of the trials were published 20 to 40 years ago. Two meta-analyses of randomised controlled trials where the primary intervention was replacement of saturated fat with polyunsaturated fat have been published in the last two years and provide compelling evidence. Skeaff and Miller found that high P/S (polyunsaturated to saturated fat ratio) diets reduced the risk of coronary events by 17% (95% CI, 0–39%, $p=0.05$) compared with lower P/S diets.⁷ Mozaffarian et al. found that high P/S diets reduced risk of myocardial infarction or coronary heart disease death by 19% (95% CI, 5–30%, $p=0.008$).⁸

In conclusion, the evidence indicates that it is quite fair to demonise saturated fat. Fortunately many New Zealanders have been acting on this evidence for years, as reflected in their falling blood cholesterol levels, that began well before the introduction of statins.⁹ Nonetheless, results from the last National Nutrition Survey (1997) showed that New Zealanders consumed, on average, 15% of their energy as saturated fat.¹⁰ Current Australasian nutrition guidelines recommend that, for individuals, saturated fat should not exceed 10% of energy intake; thus, the population mean should be considerably less than 10%. The 2008–09 National Nutrition Survey will report later this year and it is unlikely we will have achieved this. Decreasing saturated fat intake to 5–10% of total energy is warranted and easily achievable for a large proportion of New

Zealand adults by reducing consumption of high fat dairy products, fatty meats, cakes, biscuits, and confectionery products and replacing this with nuts, seeds, plant oils (e.g. soybean, canola, sunflower, or safflower), and doing the margarine table spread for butter swap. Indeed, as the world's largest consumers of butter, estimated to account for almost 20% of our total saturated fat consumption, New Zealanders could improve their cardiovascular health with the stroke of a (butter) knife.

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