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Advice to reduce total and saturated fat, revisited

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The validity of advice to reduce total and saturated fat in order to reduce obesity and coronary heart disease (CHD) has been questioned from time to time.[1,2] International guidelines[3–6] recommend intakes of saturated fatty acids (SFAs) of below 10% of total energy, and polyunsaturated fatty acids (PUFAs) sourced mainly from plant oils, nuts, seeds and fish of 5–11% of total energy.

Implementation of this advice would involve a modest to substantial reduction in saturated fat intake in most Western countries and some increase in polyunsaturated fat. More recently it has been suggested that a wider range of intakes of total fat than had previously been suggested, is acceptable: up to 40% of total energy in the Nordic recommendations.[7] Recommended amounts of monounsaturated fatty acids (MUFAs) are derived by differences (% energy from total fat – [combined % total energy from SAFA + PUFA]), implying a similarly wide range of acceptable intakes.

Such evidence-based recommendations are derived from a consideration of the totality of evidence relating to a wide range of health issues, most importantly cardiovascular disease, and obesity and its wide ranging consequences. It is timely to review the suitability of advice relating to fats in the light of recent publications, and the increasing rates of obesity in New Zealand.

A recent systematic review and meta-analysis examining the association between dietary fatty acids and coronary heart disease (CHD) by Chowdhury et al concluded that "current evidence does not clearly support guidelines that encourage high consumption of polyunsaturated fatty acids and low consumption of total saturated fats, and that nutritional guidelines may need reappraisal to reflect the current evidence".[8]

Fierce criticisms regarding a number of errors and omissions in the paper resulted in its undergoing revisions a day after publication. However the conclusions of the corrected version, now republished, remain unhelpful and a leading group of nutritional epidemiologists from Harvard have continued to call for the article to be retracted for misrepresenting the evidence on dietary fats.[9] Here we consider the limitations of this new review and whether its conclusions are valid.

Chowdhury et al suggest that their review offers substantially new insights into the effects of dietary fats on CHD. They reported finding no significant association between CHD outcomes and intakes of SFA, MUFA, and both omega-3 (n-3 PUFA) and omega-6 polyunsaturated (n-6 PUFA) fats. The review relates principally to observational cohort studies, which have examined dietary intake in relation to subsequent CHD.

As reported in several previous reviews and meta-analyses there was no association between intakes of saturated, monounsaturated and n-6 PUFA and cardiovascular disease when the fatty acid groups were considered in isolation of other dietary

determinants.[10,11] To some extent this could be due to the limitations inherent to dietary assessment methods, which make accurate measurement of the exposure variable difficult.

Chowdhury et al attempted to account for these limitations by including prospective cohort studies which examined associations between CHD and fatty acid intake biomarkers – a theoretically more objective measure of intake.[8] However, the use of biomarkers to estimate fat intake is not well validated; for example the even-chain SFA biomarkers reflect both SFA intakes and endogenous synthesis from carbohydrates and alcohol.

Thus the finding of no link between fat intakes and coronary outcomes by Chowdhury et al. is likely to be confounded by other nutrient intakes, particularly carbohydrate, for which there has been no adjustment.

A rather different result emerges when considering also the nutrients which replace saturated fat (as would be the case in real life) rather than individual nutrients in isolation. The review by Jakobsen et al (2009)[12] did just that in a meta-analysis of studies in which individual participant data was used as distinct from other meta-analyses based on aggregated study results.

This pooling approach is methodologically superior and made it possible to adjust for the same set of confounders across all of the data and to standardize the outcome measures. They found that substitution of 5% of energy from SFA with 5% of energy from PUFA was associated with a 26% reduction in risk of coronary death based on 2155 deaths amongst 344,696 subjects followed-up for 4–10 years. Substitution of SFA with carbohydrate or MUFA was not associated with benefit.

A meta-analysis of eight randomised controlled trials by Mozaffarian et al (2010) provides further evidence that reducing SFA and replacing with PUFA lowers the incidence of CHD events.[13] Results showed that each 5% of total energy replacement of SFA by PUFA was associated with a reduction in CHD risk of 10%, a finding consistent with the expected change in total cholesterol to HDL cholesterol ratio due to alteration in fatty acid intakes.

The apparent discrepancy between the two meta-analyses of the randomised controlled trials[11,13] may be explained by the inclusion in Chowdhury et al of data from the recent re-analysis of the Sydney Diet Heart Study which reported a significant increased risk of coronary heart disease with replacement of SFA with PUFA—a finding at odds with the other studies included in the analysis.[2]

This study involved dietary supplementation with large quantities (15% of total energy intake) of plant oils and margarine containing primarily n-6 PUFA. This is well in excess of current recommendations for PUFA intake. In fact the addition of this trial did little to change the point estimate of relative risk, a benefit of a 14% reduction in CHD events associated with replacement of SFA by PUFA, merely a widening of the confidence intervals.

Additional epidemiological and experimental evidence complements the data from the cohort studies and randomised controlled trials. Reductions in SFA intakes over the past several decades have been accompanied by substantial reductions in mortality from CHD[14] which have occurred in parallel with serum cholesterol concentrations in

much of the Western world.[15–17] This has occurred at all ages, so cannot be entirely attributed to treatment with statin drugs.

In Sweden, an increase in reported intakes of SFAs since 2004 has been associated with an observed increase in serum cholesterol levels.[16] These ecological observations are also supported by findings relating the association between blood cholesterol and CHD in cohort studies. A meta-analysis of prospective cohort studies involving over 900,000 adults shows a linear association between blood cholesterol concentrations and CHD mortality.[18]

Innumerable controlled dietary studies, some dating back to the 1950s, confirm the potential of saturated fat to elevate cholesterol when compared with PUFA and MUFA.[19,20] Thus we submit that the totality of evidence overwhelmingly supports the current guidelines and that the paper by Chowdhury et al adds no convincing new evidence to suggest that these should be revised.

Whether or not to recommend a reduction in total fatty acids is arguably a more contentious issue. Some have argued that there is no need to do so provided fatty acid composition is appropriate.[7] Others claim that reducing total fat encourages an increased intake of carbohydrate, especially sugars.[1]

Of particular relevance to this debate is the finding of Hooper and colleagues who found in a carefully conducted meta-analysis, that reducing total fat intakes has the potential to facilitate weight loss and improve cardiovascular risk factors. With ever increasing rates of obesity this would be an appropriate justification for not further liberalizing recommended intakes of total fat.

Although it is appropriate to regularly review nutrition recommendations in the light of new evidence, given the overall understanding at the present time, we believe that the best quality evidence supports the current advice to reduce the intake of SFA and to replace this with healthier fats from sustainably managed fish, plant oils, nuts and seeds. However saturated fat should not be replaced with refined carbohydrates.

For those who are not overweight there may be no need to appreciably reduce total fat. However for individuals who are overweight and for populations with high rates of overweight and obesity, restricting total fat intakes should remain an important component of dietary advice.

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