THE NEW ZEALAND MEDICAL JOURNAL
Journal of the New Zealand Medical Association

Chewing the saturated fat: should we or shouldn't we?

We respond to an *NZMJ* editorial by Te Morenga and colleagues about the issue of saturated fat.[1] The authors call for quiet, after the link between saturated fat intake and cardiovascular disease (CVD) was questioned.

The editorial authors attack the validity of a recent meta-analysis by Chowdhury of cohort and trial studies which showed no significant association between saturated fat intake, or biomarkers thereof, and CVD.[2] Instead, the editorial authors assert that saturated fat causes CVD, with support from another meta-analysis of cohort studies by Jakobsen which contains individual participant data.[3]

The editorial raises a number of issues. When should statistical evidence that negates a hypothesis be believed, and the conviction overturned? If there are conflicting meta-analyses evaluating the evidence for a hypothesis, which should be selected? Clearly, with saturated fat and its influence on CVD, it is possible to prefer a summary study which supports one's point of view. Less subjectively, the quality of one study over another may be ranked.

Bradford-Hill's causal criteria are useful to apply to the evidence in question. Briefly, these principles suggest that an association is more likely to be causal if there is consistent evidence from different studies, the association is strong, a dose-response association is evident, and experimental randomised trial data supports the hypothesis. Temporality, analogy and biological plausibility are other considerations.

We believe the hypothesis that saturated fat causes cardiovascular disease fails at the first criterion. Summaries of the experimental evidence do not show a consistent association between saturated fat restriction (or substitution) and mortality endpoints.

If saturated fat is the strongest dietary factor that causes CVD, it would be expected that replacement with other types of fat would lead to reduced incidence. The Jakobsen study does not show consistent evidence of benefit from saturated fat avoidance.[3] Rather, only one of the subgroup analyses returns a positive association. Similarly, if saturated fat reduces CVD without adverse effects on other outcomes, we would expect overall mortality to be reduced.

Death is measured with less error than any other disease-specific outcomes. Focus on overall mortality avoids the risk of concluding that an intervention improves one endpoint, but, in reality, is offset by harm to another. For example, a treatment may reduce CVD but increase cancer incidence, so that the effect on overall mortality is neutral. This is possible in the Jakobsen study, since only CVD endpoints are reported.

A number of meta-analyses now support the findings of Chowdhury,[2] showing little backing for the idea that substituting saturated fat with other types reduces CVD.[4–9] A Cochrane review of randomised studies, designed to test the hypothesis that saturated fat influences CVD, showed no association between treatment arm and overall mortality (pooled relative risk 0.98, 95%CI: 0.93–1.04, 71,790 participants, 4292 deaths).[6]

With the high number of participants and deaths reported, a large effect of the intervention is unlikely to be missed. The funnel plot for this analysis showed some evidence of publication bias. That is that small studies which showed harm from saturated fat replacement were unlikely to be published. The reported pooled effect is, therefore, likely to overestimate the benefit of avoiding saturated fat.

So, we conclude, that randomised trial data, which is superior to the observational evidence offered by Jakobsen, does not support either limiting or altering saturated fat intake to improve survival. We also consider that this Cochrane review is less likely to be biased than the surrogate endpoint (low density lipoprotein cholesterol) and ecological studies referred to by the editorial authors. In an editorial that claims to present "the totality of the evidence", we find this omission striking.

The editorial authors argue that the Jakobsen study[3] should be preferred over that by Chowdhury,[2] even though the latter includes randomised studies. Experimental trials are generally considered less biased than those which observe cohorts, due to the randomisation which balances confounders between the treated and control arms.

Other studies support the lack of statistical association between altering saturated fat intake, both from randomised and observational designs.[4,5] One comparative meta-analysis ranks the statistical link between saturated fat and CVD amongst the poorest of a range of dietary factors.[5]

We ask ourselves, "How much more evidence is needed before saturated-fat-based interventions are abandoned?" Popper stated that the hallmark of the scientific method is that a hypothesis is possible to falsify, should it lack supporting evidence.

In the absence of a strong indication of harm, we believe the public should be left to chew the saturated fat, and concern themselves with avoiding dietary factors which consistently cause ill health.[10]

Competing interests and funding: Nil.

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