Chewing the saturated fat: how many more negative studies do we need?

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We respond to the recent editorial and letters written by Swinburn,[1] Jackson and Ni Mhurchu[2] which address the effect of saturated fatty acids (SFA) in the diet on human health. Jackson and Ni Mhurchu[2] question the evidence that several meta-analyses of dietary trials that have failed to demonstrate a clear effect of modifying SFA intake. Instead, they argue that the trials suffer from low statistical power, are contaminated, have diluted treatment effects, and that the negative statistical evidence presented in summary meta-analyses is less important than other biomarker and ecological studies.

To restate our argument, we believe that if reducing saturated fat were truly to reduce cardiovascular disease (CVD) incidence, without adverse effects on other causes of death, it would also improve overall mortality. The evidence that overall mortality is not reduced cannot be easily dismissed. Unlike disease specific outcomes, such as CVD, there are no competing risks associated with overall mortality, and measurement is less error prone and more objective than for specific disease outcomes. Overall mortality is also clinically relevant: a reduction means that following this dietary advice leads to people living for longer, not swapping one cause of death for another. This is implied by a meta-analysis which reports a reduction for a disease-specific outcome, but not for overall mortality, if statistical power is sufficient.

Jackson and Ni Mhurchu argue that overall mortality reduction is not observed because there are few deaths in the meta-analyses. We have taken a closer look at this claim by examining the latest Cochrane meta-analysis of trials which addresses the effect of SFA intake (versus usual care). It shows no association between SFA intake and overall mortality.[3] In this meta-analysis,[3] there were many participants (55,858) and many deaths (3,276) and essentially no difference in mortality in the two groups (1,377/22,819; 6.3% in reduced SFA and 1,899/33,039; 5.8% in the usual diet group). This study is large enough and would have had sufficient power to detect a reduction, at least one that is clinically meaningful, if there were a SFA effect.

Swinburn's editorial[1] argues that the disappointing trials with hard endpoints may be dismissed in favour of the evidence that links saturated fat intake with markers of risk:

> *"The rock-solid, central planks of the saturated fat intake to heart disease relationship are that diets high in saturated fat increase LDL [low density lipoprotein]-cholesterol and that high LDL-cholesterol is a major risk factor for coronary heart disease".*

However, this evidence is sinking sand, as The American Academy of Nutrition and Dietetics has recently concluded, stating:

> *"The evidence is clear that changes in LDL and HDL [high density lipoprotein] induced by diet cannot be assumed to correspond to the expected changes in actual cardiovascular disease risk, and thus this body of evidence that uses lipoproteins as surrogate endpoints for cardiovascular disease must be excluded from considerations of the impact of diet on cardiovascular health."*[4]

No evidence of benefit to survival, or cardiovascular disease, has been reported from several other meta-analyses on the subject.[5-7] In short, the enormous resource

spent on reducing saturated fat has led to disappointing results. While the negative evidence continues to accumulate, and continues to be dismissed, we renew our advice to the public to chew the saturated fat, and focus on restricting components of the diet that are consistently associated with poor health: sugar,[8] and starch.[9,10]

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