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The evidence base for fat guidelines: a balanced diet

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Harcombe *et al* have conducted a systematic review and meta-analysis of randomised controlled trials (RCTs) that would have been available to the regulatory committees in the USA and the UK when guidelines on dietary fat intake were introduced (in 1977 and 1983, respectively). They found no evidence from RCTs to support the introduction of these guidelines, which leads the authors to question whether they should ever have been introduced in the first place. [1] The negative result of the meta-analysis is unsurprising. The most up-to-date review of cohort and RCT studies draws a similar conclusion that there is very limited evidence to support current guidance.[2] However, whether this means that changes to the health policy should be made, is a more complex question.

There is evidence to say that individual interventions to change behaviour and modify risk do not lead to changes in hard clinical end points. Most of the trials relied on dietary advice to situate participants within their different groups, with some also providing supplements and only one trial (the LA veterans study) actually providing meals. Most made an attempt to measure adherence to the diet prescribed, though self-reported measures of adherence are not convincingly reliable. The latest Cochrane review into multiple risk factor interventions for primary prevention (using education and counselling to modify cardiovascular risk factors) shows no evidence that mortality is reduced in general populations.[3] Since such interventions have generally failed to make a measurable difference, it is unlikely that the advice given in these trials would have done so. This does not mean that the risk factor addressed is not a risk factor.

More broadly, there is a fair argument to say that there are entire fields of science where positive results, even if they were to occur, are unlikely to represent true associations.[4] Much dietary science of the type examined here is likely to fall into this category. However, there is a body of evidence supporting a link between fat consumption and cardiovascular disease that should be considered first.

Some of the results reported in the aforementioned current meta-analysis are controversial. Other reviews of RCTs have shown that the replacement of saturated fat with polyunsaturated fat carries cardiovascular benefits.[5 6] Such disagreements between meta-analysts are not uncommon since small differences in the criteria used to include studies in different parts of the analysis can lead to large differences in reported results.

Epidemiological and ecological evidence suggests a link between fat consumption and heart disease. The seven countries cohort study by Keys[7] referred to by the authors did find that higher serum cholesterol tended to be related to coronary heart disease incidence and that higher saturated fat consumption tended to be related coronary heart disease incidence. These findings were consistent in long-term follow-up.[8] Certainly, a graded relationship between serum cholesterol level and coronary heart disease is a finding in other cohorts[9 10] and lowering serum cholesterol appears to improve clinical outcomes.[11] Serum cholesterol levels therefore remain a cornerstone in the assessment of cardiovascular risk.[12] Occasionally 'natural experiments' have occurred where large population-level declines in coronary heart disease were associated with changes in the supply of dietary fat available as in Eastern Europe in the 1990s.[13]

In summary, there are disagreements in the interpretation of available data from RCTs, but despite this there remain reasons to postulate a causal connection between fat consumption and coronary heart disease. Even with a causal connection, we might expect RCTs to produce negative results because actual human behaviour over long periods of time is unlikely to be altered by individual sessions of dietary advice. Despite this, the existence of nutritional guidelines can be beneficial through altering the content of food available, changing how

food is packaged and through setting normative standards for what is considered healthy. Small reductions in risk factors at a population level might then be expected to have large effects on the rates of disease. Such mechanisms are difficult to replicate in a trial setting. Public policies generally do not require RCT evidence, so to advocate their withdrawal here on the basis of the absence of such evidence seems unusual. How individuals feel about this will depend on their own assessment of the totality of the evidence base and on their personal political values.

There is certainly a strong argument that an overreliance in public health on saturated fat as the main dietary villain for cardiovascular disease has distracted from the risks posed by other nutrients such as carbohydrates.[14 15] Yet replacing one caricature with another does not feel like a solution. It is plausible that both can be harmful or indeed that the relationship between diet and cardiovascular risk is more complex than a series of simple relationships with the proportions of individual macronutrients.

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