

Letter to the Editor

The importance of reducing SFA intake to limit CHD risk

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In a recently published guest editorial, Pedersen *et al.*⁽¹⁾ discuss the importance of reducing the intake of SFA to limit CHD risk. The authors emphasise the shortcomings of results from prospective cohort studies and discuss other lines of evidence. This evidence leads the authors to conclude that substantial benefits can be found from reducing the intake of SFA. Results from the different lines of evidence in the way presented by Pedersen *et al.*, however, raise several questions.

First, the importance of lowering SFA intakes to lower blood LDL-cholesterol levels is emphasised. However, a meta-analysis of controlled trials examining the effects of replacing carbohydrates with different types of fatty acids showed that SFA not only increase LDL-cholesterol, but also HDL-cholesterol compared to carbohydrates and that the ratio of total:HDL-cholesterol was not changed by this exchange⁽²⁾. The conclusions were that:

Results of prospective observational studies, controlled clinical trials with drugs, mechanistic studies, and genetic 'experiments of nature' all strongly suggest that high concentrations of HDL-cholesterol in the circulation help to prevent coronary artery disease and other CVD. Given these observations, it appears imprudent to ignore the marked effects of diet on HDL-cholesterol.

Results suggest that isoenergetic replacement of SFA with carbohydrates does not improve the serum total:HDL-cholesterol. All natural fats contain both SFA, which do not change this ratio, and unsaturated fatty acids, which lower it. As a result, even the replacement of dairy fat and tropical fats with carbohydrates will increase the ratio of total to HDL-cholesterol.

The effects of dietary fats on total:HDL-cholesterol may differ markedly from their effects on LDL. The effects of fats on these risk markers should not in themselves be considered to reflect changes in risk but should be confirmed by prospective observational studies or clinical trials.

That the effect of diet on HDL-cholesterol should be considered when examining CVD risk, and that all natural fats will improve the ratio of total:HDL-cholesterol compared to carbohydrates, seem to have been disregarded by Pedersen *et al.*⁽¹⁾. Results from a pooled analysis of sixty-one prospective studies supported the need to include HDL-cholesterol in a model relating cholesterol to CHD mortality⁽³⁾. It is not clear why Pedersen *et al.* did not consider the effect of SFA

on HDL-cholesterol or on the ratio of total:HDL-cholesterol when examining the relationship of SFA with CHD.

Second, the substantial reductions in mortality from CVD in North America, Western Europe and Australasia over the last 30 years are said to reflect successful national public health policies to reduce the intake of SFA⁽¹⁾. The authors do not show any evidence that such reductions in SFA intake have actually occurred in these regions. Contrary to this, they state that SFA intakes increased in China over time. A report from the US Department of Agriculture and the US Department of Health and Human Services states that no reductions were found in the intake of SFA in the American diet over the period 1989–1 to 2005–6⁽⁴⁾. Indeed, although the intake of SFA as percentage of total energy (en%) was slightly higher over the first time period (12.3), than over the last three time periods (11.2–11.4), the total amount of SFA in g/d increased slightly over this time (25.7–27.8). More importantly, it is not possible to unequivocally associate changes in SFA intake to changes in CHD mortality over time, since many changes in diet, lifestyle, diagnosis and pharmacological treatments have occurred over the last 30 years.

Third, Pedersen *et al.* state that trials have demonstrated unequivocally that 'replacing SFA, largely from dairy and meat fats, by PUFA reduces serum cholesterol levels and CHD risk', referring to a meta-analysis by Mozaffarian *et al.*⁽⁵⁾. But Mozaffarian *et al.* note that their findings cannot distinguish between the potentially distinct benefits of increasing PUFA *v.* decreasing SFA, and that, given the limitations of each individual trial, the quantitative pooled risk estimate should be interpreted with some caution. Pedersen *et al.* state that the changes in CHD risk occurring in the trials mentioned were attributable to replacing SFA by PUFA, and that in the Leren trial⁽⁶⁾, some *trans*-fatty acids (TFA) were also replaced by PUFA. Ramsden *et al.*⁽⁷⁾ showed that in the Leren trial, the control group consumed 9.6 g/d of TFA, while in the experimental group TFA were restricted. Ramsden *et al.* also noted that a pooled analysis of prospective cohort studies showed that each 2 en% replacement of TFA with PUFA reduces CHD risk by 32%⁽⁷⁾. In addition, they showed that in all seven trials included in the meta-analysis by Mozaffarian *et al.*⁽⁸⁾, non-hydrogenated study oils were substituted for TFA-containing fats, oils and foods. The mean estimated TFA content of the seven control diets was 3.0 en%.

It is not clear why the confounding role of TFA was not considered by Pedersen *et al.*⁽¹⁾.

Fourthly, Pedersen *et al.* refer to a meta-analysis of prospective cohort studies by Siri-Tarino *et al.*⁽⁹⁾ examining the direct relationship between SFA intake and CHD risk. They state that the null results of the studies included in this analysis probably reflect measurement error, residual confounding, over-adjustment by covariates on the causal pathway, and large variations in plasma cholesterol compared to variations in intake of dietary fat. This suggests that within these cohorts, SFA did indeed increase CHD risk, but that the researchers examining the individual cohorts just were not able to capture this effect. But what Pedersen *et al.* do not mention, is that Siri-Tarino *et al.*⁽¹⁰⁾ already responded to these criticisms. In a subset of the data from their meta-analysis, they showed that the effect of SFA on CHD did not differ significantly in cohorts in which the models did not include blood cholesterol concentration as a possible confounder.

In conclusion, Pedersen *et al.* do not consider the effect of SFA on HDL-cholesterol when examining its effect on CHD risk and they do not mention that results from controlled trials replacing SFA by PUFA have been consistently biased by a concomitant decline in TFA intake.

Because prospective cohort studies showed null results from SFA intake on CHD, these results were omitted when judging the evidence for this association. Instead, the authors turned to correlations found in apparently randomly selected ecological studies. No systematic evaluations were made here, and no evidence was presented that the presumed decrease in SFA intakes over the last 30 years contributed to a decline in CVD mortality. While the authors were able to define the possible errors found in prospective cohort studies, no such evaluation was made for the ecological data. Public health recommendations should be based on a transparent evaluation of the lines of evidence included to judge the evidence for an association. In addition, results from these lines of evidence should be judged after systematically reviewing the available literature.

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