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Letter to the Editor

No scientific support for linking dietary saturated fat to CHD

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Pedersen *et al.*[1] express concern that recently published research had downplayed the importance of SFA consumption as a risk factor for CHD[2]. Their main argument is that prospective cohort studies are unreliable. There are of course uncertainties in such studies, but it is difficult to ignore that more than thirty cohort studies have shown that patients with CVD did not eat more SFA than had heart-healthy people; in six of them[3–8], stroke patients had actually eaten less.

To make their case, Pedersen *et al.* presented a small and biased subset of ecological studies apparently linking reduced consumption of SFA to a low incidence of CHD. However, they neglected to mention the many ecological studies that have documented findings from groups with a high consumption of SFA, but with low rates of CHD, including Masai people[9], French[10], Italian-Americans[11] and Polynesians[12]. They also claim that the association between the decline of CHD mortality in Finland and the lowered intake of SFA was causal. However, the decline began in North Karelia 3 years before the start of the cholesterol campaign, and it occurred also in the districts where no advice was given[13].

Pedersen *et al.* asserted that SFA with twelve to sixteen carbon atoms are the most potent LDL- and total cholesterol-raising fatty acids. However, other researchers reported that the serum content of these fatty acids is inversely associated with serum cholesterol[14], and in seven studies, the content of twelve to sixteen carbon fatty acids in the blood or the fat cells was similar or lower in patients with acute CHD than in healthy people[15–21]. The content of certain SFA in the serum reflects the intake of dairy fat[22,23], and such intake is inversely associated with BMI, waist circumference, ratio of LDL:HDL and fasting glucose concentration, and positively associated with HDL and apoA-I[22–25]. In accordance, a meta-analysis of twenty-five cohort studies showed that the lowest total mortality, cardiovascular incidence and mortality, and incidence of diabetes were seen among those with the highest intake of dairy fat[26].

Pedersen *et al.* endorse the many reports emphasising the importance of increasing the intake of PUFA. This advice is not based on randomised, controlled dietary trials, because no such trial has ever succeeded in lowering cardiovascular or total mortality by exchanging SFA with PUFA[27]. Rather, the advice is based on statistical calculations using data from unreliable cohort studies. Pedersen *et al.* refer to a meta-analysis of such trials, the authors of which claimed benefit, but they had excluded two trials, where CHD mortality had increased in the treatment groups[28,29], and included a trial where a decreased risk was seen only in the participants who increased their intake of fish[30], and also the Finnish Mental Hospital Study[31], a trial which does not satisfy the quality criteria for a correctly performed randomised controlled trial. A reduction of SFA was part of the intervention in three multifactorial trials, but these trials were unsuccessful as well[32–34]; in one of these, total mortality was twice as high in the treatment group[33].

Numerous studies on laboratory animals and human subjects have also shown that an increased intake of PUFA, in particular of the *n*-6 type, is associated with many adverse health effects such as allergy, asthma, immunosuppression, decreased fertility, pre-eclampsia, encephalopathy and cancer[35–41]. In accordance with this, Israeli Jews have a high intake of the 'recommended' *n*-6 type of PUFA (from grains and soyabean oil), and they exhibit a high incidence of cancer and CHD mortality compared with other Western countries[42].

In conclusion, Pedersen *et al.* do not provide sufficient evidence to implicate SFA in CHD risk. There is increasingly strong evidence that SFA are not involved[2,28,43–47].

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