

Guest Editorial

The importance of reducing SFA to limit CHD

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Uncertainty has recently been expressed as to the role of SFA for the development of atherosclerosis and CHD⁽¹⁾. This confusion, created primarily by interpretation of results from prospective cohort studies⁽²⁾, was recently thoroughly discussed with strong emphasis on the shortcomings of such studies⁽³⁾. The seriousness of the problem makes it desirable to broaden the scope of this discussion with a main focus on the public health implications.

Confusion in the scientific literature on these issues may easily be misused by the food industry to promote their interests. More serious, however, is the potential damage this uncertainty may cause to public health strategies and the priority given to saturated fat and serum LDL-cholesterol reduction in the prevention of CHD. Thus, an important report on non-communicable diseases (NCD) recently published as an input to the forthcoming UN High-Level Meeting (UN HLM) in September 2011 listed tobacco control and salt reduction as its top priorities among the well-known risk factors for NCD⁽⁴⁾. Furthermore, at the recent conference of Global Health Ministers in Moscow, the WHO listed smoking cessation, alcohol controls and dietary salt reduction as particularly cost-effective interventions to reduce the risk of NCD while also giving special attention to the promotion

of healthy diets (low in SFA, *trans*-fatty acids (TFA), salt and sugar, and high in fruit and vegetables)⁽⁵⁾. In the draft outcome document of the HLM on the prevention and control of NCD, cost-effective measures to reduce risk factors mentioned are: tobacco and alcohol control; reducing salt and sugar intake; replacing *trans*-fats in foods with polyunsaturated fats; promoting public awareness about diet; and physical activity⁽⁶⁾. Thus, among risk factors, the role of SFA reduction and serum LDL-cholesterol reduction are listed but not specifically mentioned as priority tasks.

We agree with these reports in their emphasis on the multiple risk factors and urgency for action. Our main concern, however, is to emphasise the importance of lowering SFA intakes to reduce blood LDL-cholesterol levels at a time when there are tendencies to downplay the importance of SFA^(1,7,8). There have been substantial reductions in mortality from CVD in North America, Western Europe and Australasia over the last 30 years that reflect successful national public health policies to reduce the intakes of SFA, in addition to promoting smoking cessation and controlling blood pressure. Nevertheless, excessive intakes of SFA remain a major public health problem not only in affluent countries but are also

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now a major and increasing problem in low- and middle-income countries where 80% of cardiovascular deaths occur.

Recent critics of the role of SFA have questioned the rigour of the early dietary trials of CVD prevention and questioned current public health policy on limiting the intake of SFA⁽⁸⁾; they suggest that more attention should be paid to increased intake of PUFA⁽⁹⁾. The trials demonstrate unequivocally that replacing SFA, largely from dairy and meat fats (but in the Leren trial also with some TFA), by PUFA reduces serum cholesterol levels and CHD risk⁽⁹⁾. That replacement of SFA by a variety of carbohydrate-containing foods also reduces CHD risk may be inferred from ecological studies, e.g. in Finland. CHD was also almost non-existent in rural China when mean cholesterol levels were approximately 3.5 mmol/l (1350 mg/dl), with total fat intakes only about 15% of energy and extremely low intakes of SFA^(10,11). These observations, replicated in many other countries, should not be ignored even if meta-analyses of prospective cohort studies suggest no independent associations of SFA intake with CHD risk⁽²⁾. The null results of the latter studies⁽²⁾ 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^(3,12–15). The role of SFA risks may also be overlooked, given the strong emphasis on TFA⁽¹⁶⁾ and the incorrect proposition that the CHD epidemic in affluent societies has been primarily linked to a high consumption of TFA⁽¹⁷⁾.

Atherosclerosis is the pathological basis of CHD and other CVD. Extensive research over the last century has established that atherosclerosis is a complex process in which disturbed lipoprotein metabolism plays a critical role. Elevated levels of apo B containing cholesterol-rich lipoprotein particles drive the development of atherosclerosis in humans and in experimental animals, even in the absence of other known risk factors⁽¹⁸⁾. Accumulation of these LDL particles in the intima and binding of apo B to the extracellular proteoglycans appear to be the initial step in atherosclerosis⁽¹⁹⁾ with further triggering of inflammatory reactions⁽²⁰⁾. The amount and type of dietary fat to a large extent determines the number of circulating LDL particles and blood levels of total cholesterol. Moreover, SFA with 12–16 carbon atoms are the most potent LDL- or total-cholesterol-raising fatty acids⁽²¹⁾.

Recently, several studies have quantified the proportion of the decline in CHD mortality that is explained by changes in risk factors and treatment. Ford & Capewell estimated that almost half to three-quarters of the lower CHD mortality in the USA may be explained by risk factor reduction, with the remainder being attributed to more effective treatment for dyslipidaemia and hypertension⁽²²⁾. Further studies in European populations also suggest that reduction in CVD risk factors is more important than improvements in treatment. The relative importance of the different risk factors in different populations will of course depend on the magnitude of change. In the USA, 24% of the decline in CHD mortality has been ascribed to a reduction in total cholesterol⁽²²⁾. However, in the Nordic countries where national preventive programmes have been conducted since the early 1960s, dietary changes with a

reduction in population mean total cholesterol explain a far greater proportion of the decline in CHD mortality.

Finland has experienced an over-80% fall in CHD mortality due to a large extent to the reduction in total cholesterol⁽²³⁾; lower blood pressure and less smoking in men amplify this cholesterol-lowering benefit^(23,24). In Finland, a reduced SFA intake was the main reason for the fall in blood cholesterol caused by a massive decline in dairy fat consumption⁽²⁵⁾. The intake of TFA from partially hydrogenated oils has always been low in Finland; so its removal cannot explain either the reduction in blood cholesterol or the impressive fall in CHD mortality⁽²⁵⁾. By contrast, in Norway, the consumption of TFA was high and its removal may explain part of the decline in national cholesterol levels. However, the major part of the reduction was explained by reduced SFA intake⁽²⁶⁾. Similar analyses have recently been reported for Iceland where lowering of cholesterol accounted for 32% of the CHD mortality reduction⁽²⁷⁾. In all these Nordic populations, a modest initial increase in PUFA intake (which has, however, been stable for the last 25 years at 5–6% of energy) may have contributed to the early but not the later fall in blood cholesterol.

These examples illustrate the substantial benefits of reducing intake of SFA. While we would strongly endorse the high priority given to smoking and salt reduction made elsewhere⁽⁴⁾, we would consider the evidence for a reduction in intake of SFA by reducing intake of animal- and high-SFA-containing vegetable fat sources to be even stronger than when this policy was introduced by the WHO in the early 1980s. It should therefore also be included in the highest priority category. We approve of the relatively simple and cost-effective measures for the food industry, highlighted by the reports proposing removing salt and most TFA from the food supply. However, TFA removal must not sidetrack us from the very substantial quantitative importance of reducing SFA from the food supply.

SFA reduction has proven exceptionally cost-effective; so governments should not be distracted by industrial pressure or problematic new analyses of prospective studies to change dietary policies which in affluent societies have been remarkably successful in limiting CVD before the role of TFA became clear. The WHO should continue to support the member states based on its earlier successful policy about saturated fats in order to combat the burden of CVD now arising in poorer countries as saturated fat intakes escalate.

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