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, 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(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(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
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/l), with total fat intakes only about 15% of energy and extremely low intakes of SFA (10). 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). We would consider the evidence for a reduction in risk factors in all these Nordic populations 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.

Disclosures

J. I. P. is member of the scientific advisory board of the food company Mills ASA, Oslo, Norway. I. E. is the current president of the International Union of Nutritional Sciences (IUNS); IUNS has signed a time-limited agreement on scientific cooperation with Unilever. M. R. receives funding for his research group including his own salary from the British Heart Foundation. P. M. K. E. is a member of the Scientific Advisory Board for Unilever, California Walnut Commission, MonaVie, Campbell Soup Company, Abunda and receives research support from The Peanut Institute, General Mills,
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