Dear Madam/Sir,

As a health scientist involved in dietary fat and fatty acid research for more than 3 decades, I very much welcome your efforts to summarize the current ‘state of the art’ concerning the potential role of diet and nutrition in the prevention of chronic diseases. I wholeheartedly agree with most of your conclusions and recommendations as well as with the selection of published literature these conclusions and recommendations are based on. However, I have some reservations with respect to the way in which you deal with palmitic acid as a dietary constituent and palm oil as an edible oil.

As summarized below and documented in Enclosure 1, I think that there is insufficient scientific evidence to support the opinion that there are specific health concerns attached to the dietary use of palm oil, as is suggested by your statement on page 11 of the draft.
It is true that palm oil is rich in palmitic acid and that dietary palmitate has been shown to affect the plasma lipoprotein profile in a negative way as compared to stearic, oleic and linoleic acids. However, this effect is considerably smaller than for lauric and myristic acids (properly dealt with in Annex 4), and results of published intervention studies are by no means consistent (not mentioned in Annex 4). Thus, at the (recommended) fat level of 30% of energy, palmitic acid rich diets have repeatedly been shown not to increase plasma total- and LDL-cholesterol levels (1,2). In addition, evidence is increasing that the potential cholesterol-raising effect of dietary palmitic acid largely depends on the dietary cholesterol level, and can efficiently be counteracted by linoleic acid (3-5). I, therefore, think that the evidence is ‘insufficient’ rather than ‘convincing’ for listing palmitate among the dietary fatty acids associated with increased cardiovascular risk (Table !).

The same holds for the consumption of palm oil. We convincingly demonstrated that replacement of the habitual dietary fat in The Netherlands by palm oil did not adversely affect the plasma cholesterol profile. On the contrary, it significantly increased the plasma HDL-cholesterol content and significantly decreased the LDL:HDL(2+3) cholesterol ratio and the plasma content of triacylglycerols (6), as well as the plasma Lp[a] concentration (7). At least part of these cardiovascular risk lowering effects of dietary palm oil likely results from the fact that the presence of ‘hard stock’ (palmitic acid) in palm oil reduces the need for the dietary incorporation of (partly) hydrogenated edible oils, and thereby lowers the consumption of trans unsaturated fatty acids. These fatty acids are known to increase plasma LDL-cholesterol and Lp[a] concentrations and to reduce the plasma HDL-cholesterol content, and I fully agree with the recommendation (item (b) on page 33) to minimize the consumption of trans unsaturated fatty acids. Since for organoleptic and food technology reasons the presence in dietary fats of a certain amount of ‘hard stock’ is required, replacement of (partly) hydrogenated edible oil by relatively saturated oils like palm oil seems desirable to me. I, therefore, feel that there is no reason for ‘singling-out’ palm oil when discussing the health concerns attached to fat consumption (page 11). In addition, I think that there is no evidence to support the statement on page 25 of Annex 4 that "palm oil in its present composition raises cholesterol and the total/HDL ratio". As summarized above, this statement is far too general, if not incorrect, and should at least be extended to include the edible oils palm oil has been compared with in this respect.

One issue that seems to have escaped the attention of your Annex-4 Working Group entirely is the potentially beneficial role of the tocopherols and tocotrienols present in palm oil (8). These compounds have strong antioxidant effects (9) and some tocotrienols have been claimed to reduce cholesterol synthesis in a way comparable to HMG-CoA reductase inhibitors (10-12). It is true that –as properly dealt with in Annex 4– intervention trials with a-tocopherol and other antioxidants do not support their cardioprotective efficacy suggested from observational studies, but comparable studies with tocotrienols have not been reported and should be promoted. Although I also realize
that the cholesterol synthesis inhibiting effect of tocotrienols is debatable (13,14), I feel that the tocotrienols should be given proper attention in a document so important as your report. This also holds for the potential role of tocotrienols in cancer prevention (15-18), which in my opinion needs to be discussed in Annex 5.

Finally, since atherosclerosis is now generally acknowledged to have an important inflammatory component (19,20), our observation that palm oil consumption causes the production of Tumor Necrosis Factor in lipopolysaccharide-stimulated whole blood in vitro to decrease (21), bears some relevance in the context of dietary lipids and cardiovascular risk.

Summarizing, I feel that there is a role for palm oil in meeting the world’s demand for useful and versatile edible oils, which should not be jeopardized by incomplete information. If required, I will be happy to discuss these issues in greater detail with your Working Group.

Yours sincerely,

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For NUTRIM’s scope of activities, mission, interest in diet, nutrition and the prevention of chronic diseases, and funding, please see Enclosure 2.

Professor Hornstra is also the Scientific Director of NutriScience B.V., an independent contract research and consultancy company under the Maastricht University Holding and specialized in novel and functional foods. The mission of NutriScience is:
7. to support the nutritional ingredient and food industry with advise and research in the area of novel and functional foods and ingredients
8. to act as an interface between industry and Maastricht University for the integration of applied and fundamental nutritional research.
For further information on NutriScience, please see Enclosure 3.
Enclosure 1: References (see below)
Enclosure 2: NUTRIM Annual Report 2000 (not included here; see www.nutrim.unimaas.nl)
Enclosure 3: NutriScience brochure (not included here; see www.NutriScience.nl)

Enclosure 1: References


End.