Below is the result of your feedback form. It was submitted by () on Friday, June 14, 2002 at 20:59:48

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Scope_of_Activities: Regional, National and International
Mandate_of_the_Organization: Carnegie I research institution with an urban mission for teaching and research
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Comments: Dear Sir,

I am writing to express my objections to some of the “recommendations” from the Joint WHO/FAO Expert consultation on diet, nutrition and the prevention of chronic diseases (Annex 4: The scientific basis for diet, nutrition and the prevention of chronic diseases).

On page 25, the authors note that “…the highest priority should be given to modifying the fatty acid composition of palm oil, because it is becoming the world’s leading source of fat and because palm oil in its present composition raises blood cholesterol and the total/HDL ratio”. This statement is highly objectionable from several points 1) The
authors provide no reference or evidence to support this claim. 2) In the lines immediately preceding this statement the authors note the potential benefits of modification of cooking oils through appropriate admixtures – without mentioning any oil in particular, and then proceed to admonish palm oil. 3) The blanket statement that palm oil in its present form raises cholesterol and the total/LDL ratio – is simply not true. 4) Is the fact that palm oil is becoming the world’s leading source of fat, the reason that the author’s are advocating modification of its fatty acid composition.

The reason the authors are recommending a change in palm oil fatty acid composition is presumably because of its palmitic acid content. Numerous studies over the last decade (in humans) have shown that the effects of palmitic acid on blood lipids are dependent on several factors which include - the concomitant levels of myristic and linoleic acid, the amounts of dietary cholesterol, the total fat load and the LDL receptor status of the subjects - reviewed in (1-3). Thus in hypercholesterolemic subjects consuming diets with high total fat (38-45%en), low levels of linoleic acid, liquid-formula diets or diets with high levels of cholesterol, palmitic acid raises LDL cholesterol (4-7) relative to oleic acid. However when solid-food diets are utilized with more realistic fatty acid exchanges and mildly hypercholesterolemic to normocholesterolemic younger subjects are used, the hypercholesterolemia attributed to palmitic acid is either muted or disappears (8-18). Most of these latter studies have compared practical fatty acid exchanges (~5%en). In “head-on” comparisons palmitic acid has been found to be hypocholesterolemic (19) or similar (20) relative to myristic acid, hypocholesterolemic relative to lauric+myristic acid (10, 12), hypercholesterolemic (5) or similar relative to lauric acid, hypercholesterolemic (4) or similar (17) relative to stearic acid and hypercholesterolemic (4-6, 19) or similar relative to oleic acid (10, 13, 14). The response relative to oleic acid seems to be a function of the choles terolemia of the subjects used, with palmitic acid appearing hypercholesterolemic in hypercholesterolemic (4-6)and mildly hypercholesterolemic subjects, but similar to oleic acid in normocholesterolemic subjects (10, 13, 14). Based on these observations, the assertion that palmitic acid raises cholesterol and TC/HDL is far from clear.

Unfortunately, because palmitic acid is a saturated fatty acid, it is lumped with all other saturated fatty acids. Effects that are attributed to saturated fat consumption in Western populations are the result primarily of meat and dairy intakes. Accordingly extrapolations to palm oil may not be applicable as it is a minor fat in typical Western diets. Furthermore, epidemiological studies indicate that the correlation between saturated fat consumption and CHD is far weaker than was originally thought (21-24), and may simply reflect inadequate fiber intake. In addition most Western diets have cholesterol intakes of 300-600 mg/day and are lacking in fruits and vegetables. To use data from dietary studies in Western populations and make a global recommendation is, I believe, fraught with problems. There is as no epidemiological data relating palm oil consumption to CHD risk in humans. Experiments in animals suggest that palm oil, per se, is not a factor in atherosclerosis as the!
Palmitic acid is located on the sn-1, sn-3 position of the triglyceride whereas only when palmitic acid is in the sn-2 position (as in several animal fats) does it promote atherosclerosis (25).

While 45% of palm oil is palmitic acid, the rest includes a substantial proportion of unsaturated fatty acids ~ 40% oleic acid and ~9-10% linoleic acid. (The oleic acid content of palm oil is higher than that found in cocoa butter, corn oil and soybean oil, while the linoleic acid content can be comparable to that found in olive oil). Simple calculation reveals that based on current US dietary guidelines, (which advocate 7-8%en from SFA, 13-15%en from MUFA and 7-10%en from PUFA), for a diet which provides 30% of total calories from fat, palm oil can represent 55-60% of that fat. This is an extreme situation. When fat in the typical Dutch diet (38%en) was maximally replaced with palm oil (11), blood lipids were not affected. Accordingly, one would not expect any effect if the experiment was repeated with 30%en from fat. In fact studies from several countries (Australia, China, India, Malaysia, Netherlands, United States) which utilized palm oil or palm olein as the major source! of dietary fat have revealed that palm oil does not adversely affect blood lipids (9-11, 14, 26-29). Additionally, the fact that palm oil (especially red palm oil) is a rich source of vitamin A makes it an ideal choice for fighting vitamin A deficiency (30-36).

A full discussion of the rationale behind the studies I have cited as to why palmitic acid has disparate effects on blood cholesterol is beyond the scope of this letter. I merely wanted to bring to the panel’s attention that a blanket statement on palm oil can certainly not be made without providing strong supporting evidence, which the authors have not done. Secondly, if the authors have the necessary evidence it should be made available to the public and be open to discussion prior to making any global policy changes. Thirdly, as nutritionists involved with public health, are the authors forgetting that no single food item or commodity can be judged on the basis of one nutrient (in the case of palm oil – one fatty acid). If that was the case then we would have to advocate eliminating animal foods, dairy products, all partially hydrogenated oils, anything with a high salt content etc. Surely, current recommendations which set limits on total fat intake, as well as the intake! of specific fatty acid classes, inherently provide limitations on how much of a specific fat one can consume.

REFERENCES