From: Dr. Tony NG [tonyng@imr.gov.my]
Sent: Friday, 14 June 2002 04:39
To: dietandhealth@who.int
Subject: Comments on Joint WHO/FAO Expert Consultation's Draft on Diet, Nutrition and the Prevention of Chronic Diseases

14 June 2002

World Health Organization
Nutrition for Health and Development
Diet and Health
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Switzerland

Dear Sir,

Comments on the Joint WHO/FAO Expert Consultation's Draft on "Diet, Nutrition and the Prevention of Chronic Diseases"

Respectfully, I refer to the above Draft by the Joint WHO/FAO Expert Consultation.

In the file attached, I have some comments on the Draft which I hope would get the kind attention and consideration of the Expert Consultation concerned.

Thank you very much.

Yours sincerely,

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COMMENTS ON THE DRAFT- "DIET, NUTRITION AND THE PREVENTION OF CHRONIC DISEASES" BY THE JOINT WHO/FAO EXPERT CONSULTATION ON DIET, NUTRITION AND THE PREVENTION OF CHRONIC DISEASES, GENEVA, SWITZERLAND, 28 JANUARY- 1 FEBRUARY 2002

I must congratulate the Joint WHO/FAO Expert Consultation concerned for the enormous amount of work that they have obviously put into drafting this well-prepared 2002 draft on "Diet, Nutrition and the prevention of chronic diseases".

However, there are two areas in the draft which I feel compelled to comment on, and hope my views would get the consideration of the WHO/FAO Expert Consultation concerned before the Report is finalised.

COMMENT 1:

Page 21, Table 2, Ranges of population nutrient intake goals.

I am happy to note that the Expert Group 2002 has reduced the recommended dietary linoleic acid (LA: 18:2,n-6) intake from the previous high of 10% energy in the 1992 Report to the present 5 to 8 % energy for the prevention of coronary heart disease (CHD). This more realistic LA intake level (5-8% energy) would not "overwhelm" the metabolism of the n-3 fatty acids [α-linolenic acid (ALA), EPA, DHA] when the latter fatty acids constitute at least 1% energy of the diet.

However, I am perplexed by the very strict level, i.e. <7% energy, "imposed" for saturated fatty acids (SFA). It is important to note that we are talking about population nutrient intake goals here and not therapeutic diets for hyperlipidemic patients.

This strict restriction on SFAs (<7% energy) is probably taken from the NCEP Step 2 diet. In the NCEP Step 1 diet model, the 10:10:10 from SFA:MUFA:PUFA percent fat energy is further reduced to <7:10:10. It is questionable, whether the SFA intake can get too low, particularly since SFA may be the primary means of keeping plasma HDL from declining along with the LDL when LA are consumed at very high levels (10% energy).

In the study by Flyn et al. from the Miriam Hospital Nutrition Center at Providence (1999), the NCEP Step 2 diets [isocaloric and hypocaloric to a comparison high-fat high-SFA diet (HFSF)] lowered slightly plasma LDL (-5.1% to –9.0%), but drastically reduced HDL (-14%). Interestingly, the two NCEP Step 2 diets raised plasma LDL/HDL ratios (LDL/HDL= 3.74, 3.91) compared to the HFSF diet (LDL/HDL=3.54). What was particularly disturbing was that the "protective" HDL_2 fraction plunged 38% with the NCEP Step 2 diets. Thus plasma HDL_2 suffers the biggest decline when SFA are removed drastically from the diet.

Also, SFA depress hepatic lipase (HL), slowing down the removal of the leftover triglycerides (TGs) in LDL. This slows down LDL removal (adverse effect) but the good news is that SFA also induce the LDL particle to become larger (more buoyant) and less atherogenic (Zambon et al., 1999). Thus, SFA as a group, have at least two beneficial effects on the plasma lipid and lipoprotein profile namely, i) they raise plasma HDL levels (and thus help maintain healthy LDL/HDL ratios) and ii) they help induce LDL to be become larger (more buoyant) and therefore less atherogenic.

In the final analysis, dietary SFA are necessary for maintaining the best LDL:HDL ratios, depressing HL and inducing large buoyant and less atherogenic LDL. The NCEP Step 2 diet, with the unnecessarily strict restriction (<7% energy) on SFA, is probably not appropriate as the "population nutrient intake goal for the prevention of CHD".

I would like to appeal to the Joint WHO/FAO Expert Consultation concerned to reconsider its stand on the very strict restriction on SFA (<7% energy) under total fat as a population nutrient intake goal. I propose that <10% energy SFA would be a more realistic and appropriate recommendation based on the arguments that I have raised earlier.

In addition, at the very low fat intake level of 15% energy, it would be difficult to meet daily energy and essential fatty acid requirements, as well as prepare tasty, palatable meals. As such, a minimum fat intake of 20% energy would perhaps be more appropriate as a population nutrient intake goal for health in the long term.

COMMENT 2:

Annex 1-4 of Draft, Page 25, Nutrients and CVD, Last para. of Item 3.1

I find that the sentence in the last paragraph of the above section which reads:
"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 cholesterol and the total/HDL ratio" as somewhat of an unfair attack on palm oil, especially when the scientific evidence in the literature shows that this statement by the WHO/FAO Group concerned is not entirely accurate.

As far as the impact of dietary fats on cardiovascular health is concerned, the highest priority in my opinion is to cut down on the intake of hydrogenated fats which contain substantial amounts of trans monoenes (particularly 9-11 position isomers of elaidic acid). The dietary levels of these trans fatty acids (TFAs) have far exceeded 2% energy in some western nations where hydrogenated oils and fats feature prominently in the habitual diet (Hayakawa et al., 2000). Also disturbing is that fact hydrogenated fats are the main component(s) of the popular vegetable ghee, *vanaspati*, consumed in Pakistan and India. So it is reasonable to conclude that TFA intakes are not only high in western developed nations, but also in poor developing countries where hydrogenated oils/fats contribute substantially to the total dietary fat.

Meta-analysis of scientific publications in peer-reviewed journals have indicated that trans monoenes, when consumed at levels >1% energy, raise the LDL/HDL ratio

approximately twice that induced by the cholesterol-raising C12-16 SFA (Ascherio et al., 1999). Thus, TFAs are much worse than the C12-16 SFAs.

Coming back to the issue at hand, palm oil has traditionally been labelled as a saturated fat because of its high palmitic acid content (41%). However the oil has only traces of the cholesterolemic villains, myristic acid (1.0%) and lauric acid (0.5%), but a high content of oleic acid (40%) and 10% of the n-6 PUFA, linoleic acid (LA). The major fraction of palm oil consumed, palm olein, in fact has 12% LA, which is certainly higher than in the much-extolled olive oil which has less than 10% LA.

The impact of palm oil on the CVD risk profile would then depend primarily on the impact of its palmitic acid (16:0) content. There is a large pool of information on this aspect in the literature and it is indeed surprising that the WHO/FAO Expert Group concerned has chosen to ignore this.

Palm oil does not behave like a saturated fat

In his re-examination of the data obtained in early human studies which included palm oil, Hornstra and colleagues from the Limburg University, Maastricht, The Netherlands, had highlighted that when palm oil replaced the habitual fats in typical western diets, the plasma cholesterol levels did not go up but instead went down by 7% to 38%! (Hornstra, 1987). This report and the earlier study on the "arterial loop technique" for the induction of arterial thrombus in rats by different dietary fats by the same research group (Hornstra and Vendelmens-Starrenburg, 1973) have provided early evidence that palm oil, contrary to expectation, does not behave like a saturated fat.

In the study on Malaysian volunteers by Ng et al. (1991) from the Institute for Medical Research in Kuala Lumpur, 83 young normocholesterolemic adults (aged 20-34 years) were randomly allocated to one of three dietary sequences namely, coconut-palm-coconut, coconut-corn-coconut, or coconut oil throughout. Each dietary sequence consisted of 3 consecutive 5-week dietary periods. The test fats formed about 75% of the total fat calories of 30%. The change in serum lipid parameters in the three dietary groups relative to the values "at entry" are shown in Fig. 1.

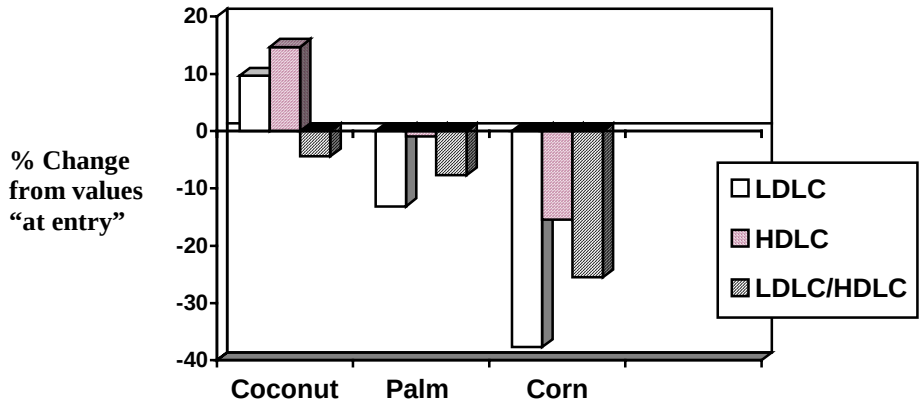


Fig 1: Serum lipid parameters relative to values "at entry" at the end of the three dietary periods [Adapted from Ng et al. (1991)]

It is clear that from the above diagram that palm olein does not behave like a saturated fat and it is very different from coconut oil. More importantly, the palm olein-diet lowered both LDLC by 13.1% and the LDLC/HDLC ratio by 7.7%, compared to values "at entry". In the switch from coconut oil to palm olein, the observed cholesterol reduction (-0.93 mmol/L) was much greater than the values predicted by either the Keys-Anderson equation (-0.67 mmol/L) or the Hegsted equation (-0.86 mmol/L).

In the study on American subjects (males aged 22-43 years, normocholesterolemic) by Heber et al. from the UCLA School of Medicine, the effects on plasma lipids and lipoproteins of 35% energy-fat diets of which half the calories from fat was provided by either palm oil, coconut oil or partially hydrogenated soybean oil were investigated. The test oil was incorporated into muffins or cookies and fed for three 3-week periods to the same individuals with 2 weeks washouts between diets.

Surprise, surprise! Despite raising the level of palm oil in the typical American diet nearly 20-fold (<2% energy to 18% energy), the palm oil period did not have any adverse effect on the serum lipid and lipoprotein profiles. In fact, at the end of the palm oil period, serum levels of the "protective" HDLC increased significantly (+4mg/dL, p=0.04).

Conditional neutrality of palm 16:0

A very provocative suggestion is that the palmitic acid in palm oil may in fact be neutral. The neutrality of palm 16:0 reported in the literature appears to be conditional, namely when the study subjects are normocholesterolemic and consuming a low-cholesterol diet (<300mg/day). Perhaps the earliest and arguably the most impressive was provided by Hayes et al. from Brandeis University (1991), USA in their work in different species of monkeys.

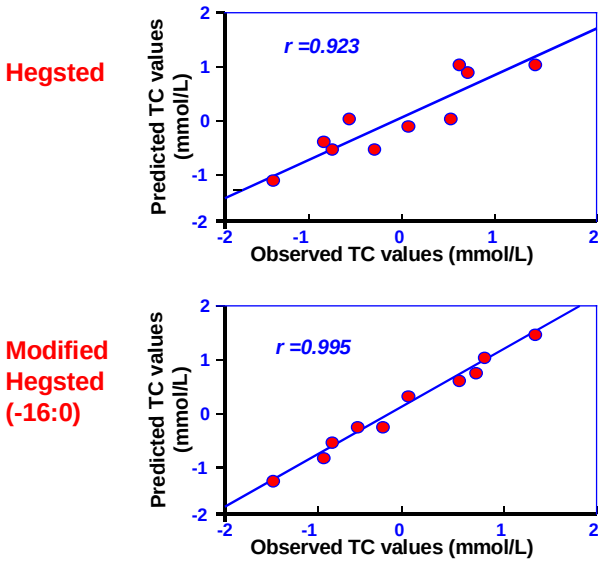
In their study, 24 monkeys from 3 species were rotated through 5 purified diets containing 31% energy as various fat blends for 12-week periods to compare the impact of specific dietary fatty acids on plasma lipids and lipoproteins. Using regression analysis, the investigators found a good fit (r=0.923) of the observed plasma cholesterol levels versus predicted values with the Hegsted equation. However, using a modified Hegsted equation where palm 16:0 was omitted from the equation (considering palm 16:0 as neutral), a near-perfect fit (r=0.995) was obtained for observed versus predicted plasma cholesterol values! (See Fig. 2).

Structure of fat molecules affect the cholesterolemic response

Nature has it that most of the 41% of 16:0 in palm oil are in the 1- and 3-positions of the TG molecule. This triglyceride (TG) structure of the major fat species in palm oil is non-cholesterol raising. When native palm oil is randomized such that the 41% 16:0 is equally distributed among the three "arms" of the TG molecule, the randomized palm oil attains cholesterol-raising properties (Kritchevsky, 2000). Contrast the structure of the TG species in butterfat where the majority of 16:0 is in the 2-(beta-)position which renders

butterfat cholesterol-raising. It is reasonable to propose that the unique structure of the TG species in palm oil are responsible in part for the oil's non-hypercholesterolemic effects reported in many published studies (Ng et al., 1991, Hebet et al. 1992, Ng et al., 1992).

Fig 2: Regression analyses of predicted versus observed plasma TC values in monkeys (Hayes et al. AJCN 1991)



Prothrombotic affects of PUFA, MUFA often overlooked

When we talk about dietary fats and cardiovascular health, one often overlooks the fact that PUFA (LA) and MUFA (principally oleic acid, 18:1) are prothrombotic. Research work by Sanders et al. (1997) from Kings College, London, showed that these unsaturated fatty acids are potent activators of the haemostatic index, factor VIIc, as well as increase plasma fibrinogen levels! This means that after a meal high in LA or oleic acid, the blood is in a hypercoagulable state! By comparison, palmitic acid (16:0) does not have these adverse effects.

Again, I humbly request that the "unfair" attack on palm oil in the Draft of the Report in the Annex Section that I had highlighted earlier be removed. As a parallel, it would also be inappropriate if one is to point a finger at hydrogenated soybean oil in the Report as a major source of trans fatty acids- the "super villains" of CHD risk, and suggests that priority be given to the reduction of TFAs in HSBO in nations where HSBO is a major dietary fat.

Thank you for your attention and consideration.

Comments on the Draft of the WHO/FAO Expert Consultation by:

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The Institute for Medical Research (IMR) is the "research arm" of the Ministry of Health Malaysia. The IMR was established in 1900 and currently the Institute, among other International collaborations, serves as the WHO Regional Centre for Training in Tropical Diseases and Nutrition.

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