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From: K.C.Hayes [kchayes@brandeis.edu]
Sent: Thursday, 6 June 2002 18:29
To: dietandhealth@who.int
Subject: WHO draft on diet and disease

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Dear WHO:

As a nutritionist with expertise concerning the impact of dietary fats and oils on human health, I wish to comment on "the Draft" currently in circulation concerning diet and health from a global perspective. My research for the past 30 years has focused largely on the effect of fats and oils and their interaction with cholesterol on lipoprotein metabolism in several species, including humans. While the overall scope of the report seems fairly well crafted and based on substantial evidence, there are certain aspects that would /should be more carefully constructed, so as not to overstate a point or unfairly bias the consumer against specific products...specifically palm oil in the generic sense. The metabolism of palm oil and related saturated fats have been a major focus of my research efforts for 15yrs.

My concerns are based on several observations...

1. There is not single fat on earth that is perfect, generally because none contains the right amount of or balance in N3 and N6 fatty acids. Many contain too much PUFA to stand alone, ie. high PUFA oils... and others contain too little, ie. those very high in SFA such as coconut oil(1.5% 18:2), palm kernel oil(2%), milk fat(3%), beef fat(4%) and even most cultivars of olive oil (av 18:2 in OO in US market is only about 7%). Palm oil has about 9% and palm olein about 12% 18:2. Thus, the problem with a given SAT FAT is really that it has TOO FEW PUFA relative to its SFA content. It is a better way of viewing the issue, especially if you want to "fix" it on a grand scale.

2. Another point that is oft forgotten or ignored by those responsible for BIG decisions and policy setting, is that even coconut oil is NOT cholesterol raising if sufficient 18:2 is present in the diet (at about 12-15% as 18:2). It only raises cholesterol as the 18:2 is reduced. The point is that any fat can be "corrected" by blending an oil sufficient in 18:2 to counter the SAT fatty acid and/or cholesterol load in the SAT FAT, with the primary SFAs to blame being 12:0+14:0. Fats rich in 16:0+18:0, such as palm oil and especially palm oleins with their 50-60% higher 18:2, are not cholesterol raising compared to the 14:0-rich fats.

3. Several studies from all parts of the globe substantiate my point. For example, one of the first to show that palm oil is not universally cholesterolemic was Mattson-Grundy'83, where 1/3 the population (those with lowest TC) did not respond to palm oil. In general, normolipemics do not have a problem with palm oil, and definitely not with palm olein. The several Malaysian clinical studies and population surveys show this most emphatically, ie. basal TC values on native diets with 25% from fat, largely from palm olein as cooking oil, are less than 200 mg/dl (<5 mmol). Indian data from Ghafoorunissa make that point...as do the original Vergroesen data in Dutch monks comparing olive oil with palm olein, as well as the recent Campos et al. data in Costa Ricans. Palm oil may not be the ideal fat, but from a supply-demand point of view it is so much

more available and cost effective than any competitor that one should not single it out as "defective" simply because we can formulate fancy blends and margarine\$\$\$ in Europe and North America. We cannot afford, from the health standpoint, to make the "rest of the world" feel like second class citizens when their initial choice is really not that bad. The emphasis to consumers and marketers should be to ensure/encourage adequate POLYS intake in the current situation, while explaining in more detail the SAT FAT/POLY issue.

4. The reason I distinguish between these fats is that it is important from a global oils standpoint (and the challenge of feeding the 8 billion on this earth) to understand what is realistically available and affordable for the average household, ie. where are fat calories are going to originate in the future? It is more important that we distinguish between palm kernel, palm stearin, palm oil, and palm oleins for their potential nutritional, health benefits/ risks than simply implying that developing nations' health and agricultural ministers should avoid "palm oil"...That approach is simplistic and naive in my humble opinion because it opens the door to the much worse solution, wherein sat fat replacements will enter in the form of trans fatty acids (from "vegetable oil", implying a safe and acceptable origin), as happened in North America and more recently in Pakistan and India with anaspati. If we can barrow from the Nurses Health Study, each gram or 1%en from trans bears 15x the risk of an equivalent dietary SFA load. Replacing 2%en as TRANS with 2%en from SFAs reduces overall CHD risk by 30%, with probably an equivalent impact on Type 2 diabetes!! Think of it! One does not have to be a rocket scientist to determine that substituting TFAs for SFAs is doing NO population a health favor. And most fat in developing nations is still used for cooking, where one needs a stable (read SFA+MUFA-rich) fat of one sort (read palm olein) or another (read partially hydrogenated VO).

5. Accordingly, I take issue with specific points in "the DRAFT".:

Summary ..page 11. para 2...To focus only on the SFA content of palm oil in the equation is misleading, as indicated above. The reality and preferred educational approach, which would also be less derogatory and even constructive from a health benefit, would state that because the major source of fat in SE Asia is from palm oil, which is already rich in SFA, it would be prudent to ensure an enrichment of the diet (or preferably blended into the original oil source prior to distribution) with a POLY oil like soybean or canola to maintain adequate PUFA intake. (Malaysians consume only 3%en as 18:2 currently, which is still more than populations consuming only 15-18%en as fat). The important concept that is slighted in this statement is an awareness of BALANCE...between SFA and PUFA! Remember SFA are only bad in the relative absence of PUFA, at least between 20-40%en from fat.

page 29...Table 5. With the newer data on TRANS and diabetes risk, I am convinced that trans should be moved up to the "convincing" or at least "probable" category. Our latest data show it behaves like SFA in its relationship with PUFA. If you decrease PUFA intake to 2-3%en, the TRANS effect on TG, and TC, is striking and much worse than equivalent intakes from 16:0 or 18:0. The equivocal data in the literature reflect 18:2 intakes that were inadequately controlled between test diets in certain, highly referenced studies.

page 32. Table 6. One cannot realistically separate lauric+myristic from real food fat sources..of which there are 3..milkfat, coconut oil, and palm kernel oil. Fatty acids are not cholesterolemic..it's the fats containing them. Myristic acid, as such, is much less cholesterol-raising than its equivalent incorporated in coconut oil, 18:2 being equal, implying that the TG molecule is important in certain instances. Lauric studies were conducted with modified TGs. Thus, one cannot justify lauric acid as a "possible" when talking about foods, as we are here , and it should be in the top category with a hyphen to 14:0. The trouble with palmitic is that

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it tags along in generous amount with 12:0+14:0 and is incriminated to a great degree by association (and very well may be a problem in the company of the other two!). On the other hand, super palm olein (rich in 16:0, but no 14:0, and having 14% 18:2 largely in sn2) is essentially comparable to corn oil and better than olive in its cholesterol response in normolipemic systems, providing diet cholesterol is low.

Annex 4...page 25. para 4. This paragraph has a good message in it, in general, but I would again suggest that the emphasis be shifted from the generic term "palm oil" to emphasize relative differences between fractions..eg. after suggesting the genetic enhancement of the stock cultivar by breeding for more PUFA, point out the present possibilities and benefit from preferential consumption of palm olein > palm oil > palmstearins > palm kernel, while re-emphasizing the specific merit in adding a PUFA-rich oil to the more saturated fractions, or even to palm oil itself, prior to market. Most people, including most of the scientific community and certainly not the consumer, haven't got a clue about the broad family of palm oil constituents, let alone their metabolic implications. It seems to me a great opportunity to educate and positively impact millions of lives with the inclusion of a few brief points/words.

Otherwise, I think the job is well done; and authors are to be commended for their hard work and dedication, particularly when one considers the complex issues involved.

KC Hayes