

## Letters to the Editor

Sir,

A number of the points raised by Erickson (1) require further discussion:

(i) The professional nutrition community should not use qualitative and potentially emotive language such as "highly saturated" to describe a vegetable oil containing 50% unsaturated acids. A proper term would be "partly saturated."

(ii) The development of coronary heart disease is too complex to be described only in terms of blood cholesterol. While the work of Keys (2) and Hegsted (3) provided evidence that stearic acid does not raise blood cholesterol levels, other workers have shown that it is active in its effect on blood platelet aggregation and thrombosis (4,5).

(iii) The report by Laine et al. (6) quoted by Erickson is unsatisfactory in several respects. Twenty-four subjects were selected as having "normal fasting cholesterol and triglyceride values," but later we are informed that "several of our subjects had unusually low cholesterol levels while consuming self-selected diets." In consequence the correction factor advised by Keys is stated to be "most likely invalid," but we are not informed how the authors deal with this problem. Only one initial cholesterol level is quoted. A base line level of 95 mg/dl cholesterol became 151 mg/dl on palm oils, and apparently 115 mg/dl on corn oil and 137 mg/dl on lightly hydrogenated soy oil. The language used to describe the latter two results is somewhat ambiguous, but it seems that in this unusual subject all the oils raised the blood cholesterol level. Furthermore, the palm oil diet contained significantly more cholesterol than the other two.

(iv) What interests the ordinary person most is the likely effect of changing his accustomed diet, and except in the one case quoted above, Laine et al. do not provide this information. Neither do Grande et al. (7) when comparing palm oil, cocoa butter and some synthetic mixtures. All the other studies we have been able to find in which palm oil is used in human dietary trials do enable the direct effect of palm oil consumption to be judged as summarized in Table 1 (Hornstra, G., private communication) (8). On current evidence, therefore, the most that can be claimed for the more unsaturated oils is that they lower blood cholesterol level more than does a predominantly palm oil diet.

(v) Erickson's reference to a "Mediterranean diet" is quite irrelevant. The experiments to which I was drawing attention (13) were carried out with palm oil and high linoleic safflower oil at 40 calories %, i.e., a level

typical of the U.S. diet. The palm oil diet reduced plasma cholesterol by 11% and the safflower oil diet by 27% from the average level on the subjects' normal diet.

(vi) In real life a diet containing only one type of oil is not practical. We should strive to educate the layman that it is the saturated fat content of the diet as a whole which is significant. In the U.S. diet more than half the saturated fat intake comes from various invisible sources of fat, while palm oil consumption has been only 1.2 to 3.0% of the whole in recent years, contributing no more than 0.6 to 1.5% of the saturated fat intake. It would be doing the general public a disservice to focus their attention on such a minor dietary item.

Furthermore, the nutritional behavior of palm oil is favorable, as indicated in references (9-13) and in a recent report by Hornstra et al. (14), contrary to expectations based on its fatty acid composition.

The effect on product composition of a typical food use of palm oil is illustrated in recent analyses by Slover et al. (15). Sixty-three margarines representing major U.S. brands were examined. Three of these were formulated with palm oil. Their saturated acid contents and P/S ratios were close to the average of the other 60.

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## REFERENCES

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6. Laine, D.C., C.M. Snodgrass, E.A. Dawson, M.A. Enen, K. Kuba and I.D. Frantz Jr., *Am. J. Clin. Nutr.* 35:683 (1982).
7. Grande, F., J.T. Anderson and A. Keys, *Am. J. Clin. Nutr.* 23:9, 1194 (1970).
8. *Nutr. Rev.*, 45:205 (1987).
9. Ahrens, E.H., J. Hirsch, W. Insull, T.T. Tsaltas, R. Blomstrand and M.L. Peterson, *Lancet* i:943 (1957).
10. Anderson, J.T., F. Grande and A. Keys, *Am. J. Clin. Nutr.* 29:1184 (1976).

TABLE 1  
 Effect of Palm Oil Diet on Plasma Cholesterol

| Reference            | 9    |      | 10               |                  | 11 <sup>a,b</sup> |     | 12  | 13  |
|----------------------|------|------|------------------|------------------|-------------------|-----|-----|-----|
| No. of subjects      | 1    | 1    | 12               | 12               | 12                | 12  | 20  | 4   |
| Duration, weeks      | >16  | >16  |                  | 2                | 20                |     | 4   | 4   |
| Starting value MG/DL | 502  | 323  | 185              |                  | 188               | 266 | 263 | 256 |
| End value MG/DL      | 401  | 201  | 158 <sup>a</sup> | 167 <sup>a</sup> | 175               | 220 | 224 | 228 |
| Change               | -101 | -122 | -27              | -18              | -13               | -46 | -39 | -28 |
| % Change             | -20  | -38  | -15              | -10              | -7                | -17 | -15 | -11 |

<sup>a</sup>Palm oil: 1], coconut oil: 1].

<sup>b</sup>Apparently a blend of palm oil with other oil was used.

## LETTERS TO THE EDITOR

11. Baudet, M.F., G. Datchet, M. Lasserre and B. Jacotot, *J. Lipid Res.* 25:456 (1984).
12. Mattson, F.H., and S.M. Grundy, *Ibid.* 26:194 (1985).
13. Grundy, S.M., *New England J. Med.* 314:745 (1986).
14. Hornstra, G., A.A.H.M. Hennissen, R. Kalafusz and D.T.S. Tan, *J. Am. Oil Chem. Soc.* 64:636 (1987).
15. Slover, H.T., R.H. Thomson Jr., C.S. Davis and G.V. Merda, *Ibid.* 62:775 (1985).

Sir,

Berger's most recent Letter to the Editor (1) encourages me to reply more or less in kind. Apparently, Berger is still attempting to "prove" that palm oil is not a saturated fat despite its fatty acid composition, and that it somehow doesn't give expected dietary results. In light of the overwhelming body of evidence in the literature to the contrary, his arguments are not very convincing.

In response to the use of the term "highly saturated," I would point out that Berger himself used the term in his first letter (2), and I followed his lead. I've no quarrel with using the term "partly saturated" but have never heard palm oil or other fats called that by anyone else.

I would certainly agree that the development of CHD is complex. Nevertheless, the current educational emphasis is aimed toward reducing serum cholesterol. Dietary advisors are unanimous in advising consumers "to reduce/avoid saturated fats" as one means of reducing their serum cholesterol levels.

The Laine et al. (3) study was designed to compare soybean oils to corn oil, and palm oil was used as a saturated fat control. The results were that the soybean oils and the corn oil reduced serum cholesterol from the levels found with the palm oil diets. This is an indisputable conclusion from the data notwithstanding any other interpretations of minor experimental variations.

Berger's interpretation of the Laine et al. (3) data plus that of Hornstra (4) leads to the not surprising finding that dietary levels of serum cholesterol were lower for all fats and oils dietary regimes than the admission levels, i.e., prior to experimentation. By pointing this out, Berger concludes ". . . the most that can be claimed for the more unsaturated oils is that they lower blood cholesterol more than . . . palm oil . . ." That's all anybody concluded in the first place!

Looking at reductions in serum cholesterol from averages of starting values rather than within those levels on strict dietary regimes, including controls, is a unique way to look at data and may be meaningless. This was pointed out by Mattson and Grundy (5), and I quote ". . . this reduction - (from admission values) - upon starting metabolic ward studies is a well-recognized phenomenon."

My reference to the "Mediterranean Diet" was thought to be relevant due to Berger mentioning that palm oil is "50% unsaturated" and because Mattson and Grundy's names are associated with it and their paper mentions it. In fact, their study is basically on the effect of monounsaturated fatty acids (5). Nevertheless, the key point in Mattson and Grundy's work is their use of palm oil as the saturated fat control. Despite

that, Berger would have us believing that the studies were favorable to palm oil. In fact, Berger says just that "the nutritional behavior of palm oil is favorable - contrary to expectations based on its fatty acid composition." Within this statement he cites references (5-9), almost all of which use palm oil as the source of saturated fat in their experimentation. I fail to see where that could be considered favorable.

In consideration of any reference to the work of Hornstra (4) I would simply submit that neither his techniques nor his research have been, to our knowledge, repeated by any other researcher.

In light of the chemical facts and the overwhelming body of nutritional research results one has to conclude that palm oil is a saturated fat and, as such, it causes an increased level of serum cholesterol when compared to fats and oils with lesser saturation. Such conclusions are irrefutable, and no amount of rhetoric and/or different interpretations of existing data will change them.

Whether or not the current emphasis on reduction of serum cholesterol remains in vogue is a whole new question. It may be that emphasis will switch to such things as platelet aggregation, thrombus formation, etc. in the future. It will then be the time to consider palm oil nutritionally as something other than a saturated fat.

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(The Editor believes that each of these authors now has had adequate opportunity for comment and rebuttal. This current exchange concludes this polemic as far as the Journal is concerned.)