Dear Sir:

I am writing in reference to the recent article by Tholstrup et al (1) entitled "Palm olein increases plasma cholesterol moderately compared with olive oil in healthy individuals." This study does not underscore the fact that when linoleic acid requirements are met, the cholesterol-raising potential of dietary palmitic acid is only minimal in normcholesterolemic healthy individuals. This reminds us of the "linoleic acid threshold" concept first highlighted by Hayes and Khosla (2) in monkeys and the subsequent equality (neutral effects) of palmitic acid (16:0) and oleic acid (18:1) on serum lipid profile reported for human studies that compared palm olein with olive oil (3, 4).

The linoleic acid (18:2) content of the experimental diets in Tholstrup et al's study ranged from 5.17% of energy in the lard diet and 5.36% of energy in the olive oil diet to 5.93% of energy in the palm olein diet. The corresponding SFA contents in the 3 diets were as follows: lard, 13.97% of energy; olive oil, 8.07% of energy; and palm olein, 13.27% of energy. With the dietary linoleic acid amount >5% of energy in all 3 diets, and the fact that SFA raises the "good" HDL cholesterol, the total cholesterol (TC) to HDL-cholesterol ratios (a better indicator than either TC or HDL cholesterol alone) in the 3 dietary groups were comparable (range: 3.37–3.48).

In human feeding trials, it is important that the test fats investigated should provide more than half of total dietary fat calories. In our studies we used test fats amounting to 60–70% of total fat energy in diets containing 30–32% of energy from fat and with dietary periods of ≥5 wk each (3, 5, 6).

It has been established that different fatty acids are incorporated with different efficiencies into serum cholesterol esters and erythrocyte membranes. Katan et al (7) reported that EPA plateaued in serum cholesterol esters after 4 wk, whereas the half-life of erythrocyte membrane fatty acids is 28 d. If these data are indeed accurate, then dietary periods in human feeding trials should each be at least 4 wk. This consideration becomes all the more important if there are no washout periods and if the dietary intervention is not center-based, because dietary compliance by subjects is always an issue.

With due respect to the investigators, the Tholstrup et al study was riddled with experimental shortcomings, namely the following: 1) a 3×3-wk crossover design with no washout period using free-living subjects, 2) subjects gained weight significantly during the study, and 3) subjects' habitual diets differed across different dietary periods. As reported by the investigators themselves, 11 (25%) of the 45 subjects with initial serum TC concentrations >5.2 mmol/L were excluded from the statistical analysis, which violates research protocol and ethics. If TC concentrations slightly more than 5.2 mmol/L are considered "unhealthy," then why was the exclusion criterion for BMI (in kg/m^2) set at ≥30?

Finally, the subjects received experimental diets that provided 207–283 kcal more than their baseline diets, despite the authors' attempt to match the experimental diets to the habitual energy expenditure of the subjects. This caused significant weight gain in subjects. This caused significant weight gain in subjects. This caused significant weight gain in subjects with different efficiencies into serum cholesteryl esters and erythrocyte membranes. Katan et al (7) reported that EPA plateaued in serum cholesterol esters after 4 wk, whereas the half-life of erythrocyte membrane fatty acids is 28 d. If these data are indeed accurate, then dietary periods in human feeding trials should each be at least 4 wk. This consideration becomes all the more important if there are no washout periods and if the dietary intervention is not center-based, because dietary compliance by subjects is always an issue.

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The author had no conflicts of interest.

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Reply to TKW Ng

Dear Sir:

We thank Ng for his interest in our Danish (not Dutch) study, in which we compare the effects of palm oil, olive oil, and lard (1), but we would like to point out that some of the points he raises are based on misconceptions. He writes that the study “underscores the fact that when linoleic acid requirements are met, the cholesterol-raising potential of dietary palm oil is only minimal in normocholesterolemic healthy individuals.” In fact, the aim of our study was to compare effects of refined palm oil, olive oil, and lard on blood lipids in healthy individuals when these fats replaced part of the energy content of the habitual diet. We found that palm oil raised plasma LDL cholesterol compared with olive oil, as one would expect in light of the established knowledge (2, 3). As Ng points out, the cholesterol-raising effects of palm oil can be counterbalanced by a given intake of linoleic acid. If we had added linoleic acid to the palm oil diet, we should have added it equally to the olive oil and lard diets. This would have resulted in lower plasma LDL-cholesterol concentrations after all test diets; however, the overall difference between plasma LDL-cholesterol concentrations after test periods would be equal to that found in our study. However, this consideration was not part of our aim or design, which simply was to investigate the effects of palm oil, olive oil, and lard. Ng emphasizes “that it is important that the test fats investigated should provide more than half of total dietary fat calories.” But why is this important when the majority of people consume less than half of the calories? Surely the effects of any quantity of fat may be investigated as long as the analyses answer the questions raised in the hypothesis? Only if the aim is to show that palm oil does not raise LDL cholesterol do specific conditions have to be met. The last point we would like to address is that Ng considers the study periods to be too short. However, there is general agreement that steady state of blood cholesterol is reached within 14 d (4), and for this reason no one else has questioned the duration of the test periods. This double-blind, crossover study was appropriate to answer the questions posed in the hypothesis, and our finding that palm oil raises cholesterol is in agreement with the majority of results of studies conducted over the past decades.

The author had no conflicts of interest related to this letter.

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Which additional factors may influence the maintenance of vitamin D status?

Dear Sir:

I read with great interest the recent findings of Farrar et al (1) that the recommended amount of summer sunlight exposure did not result in sufficient circulating vitamin D concentrations in adults of South Asian origin living in the United Kingdom. In addition to receiving rigorously standardized UV radiation (UVR) doses, equivalent to brief, regular sun exposure at 53.5°N latitude, subjects also completed food diaries to provide an estimate of vitamin D intake to control for any confounding effect on serum 25-hydroxyvitamin D concentrations. Indeed, although the dietary vitamin D intake in both the white and South Asian groups was low, it was significantly lower in the South Asian group. The authors concluded that “casual sun exposure at UK latitudes plays a much smaller role in maintaining vitamin D status in pigmented groups than in light-skinned groups.” Given that there is no clear evidence that dark-skinned individuals have less ability to produce vitamin D, when exposed to increased UVR doses to compensate for decreased UVR penetration due to pigmentation (2), the extent to which additional factors play a role in contributing to and/or sustaining vitamin D status remains uncertain.

In this context, 2 additional factors, one long recognized and one just emerging, may play important roles. First, an association has been reported between low dietary calcium intake, increased phytate (inositol hexaphosphate) consumption, and vitamin D deficiency in individuals from southern India (3). Increased dietary phytate intake has also been reported in both adults and children of Asian origin living...