Effects of dietary palmitic and oleic acids on lipoprotein cholesterol

Dear Sir:

Recently, Temme et al (1) reported the effects of feeding diets enriched in lauric, palmitic, or oleic acid on plasma lipids in 32 normocholesterolemic men and women. The results showed that lauric acid was more hypercholesterolemic than palmitic acid and both were hypercholesterolemic compared with oleic acid. The subjects consumed solid food diets and the authors managed an 8% of energy exchange between lauric and palmitic acid whereas other fatty acids were held relatively constant. The observed differences between the lauric and palmitic acid–enriched diets could not be explained on the basis of the somewhat higher myristic acid content of the former diet, and accordingly, the observed plasma lipid changes would appear to suggest that lauric acid per se was more hypercholesterolemic than palmitic acid.

However, we believe that another important aspect of the study by Temme et al (1), relating to the effects of palmitic and oleic acids, deserves comment. Among the two test diets rich in palmitic acid or oleic acid, the authors also managed an ≈8% of energy exchange between these fatty acids. The other fatty acids were either unchanged (lauric and myristic) or differed by ≈0.8% of energy (linoleic). It is difficult to assign any significance to such a small difference between the linoleic acid content of the two diets, especially because the dietary fatty acid content was calculated on the basis of food records and used published nutrient databases. Thus, the diets were effectively looking at a bona fide exchange between palmitic and oleic acids. However, this fatty acid exchange resulted in no significant differences in any of the measured variables (namely plasma total cholesterol, low-density-lipoprotein cholesterol, high-density-lipoprotein cholesterol, plasma triacylglycerols or apolipoproteins A-I and B) in their male subjects. This result agrees with recent data from normocholesterolemic men (2–4) and women (2, 4). To our knowledge, this is the first data to report on the “equivalence” of these two fatty acids when subjects consumed diets with a Western-type fat load (≈40% of energy) but not a Western-type cholesterol load (≈25 mg/MJ). The other studies that reported that palmitic and oleic acids exerted equivalent effects on plasma lipids (2–4) used similar cholesterol loads (≈25–30 mg/MJ) but lower dietary fat loads (≈30% of energy).

Palmitic acid, the most abundant saturated fatty acid (SFA) in the diet has generally been regarded as the major contributor to dietary SFA–induced hypercholesterolemia (5). However, beginning with the nonhuman primate studies of Hayes et al (reviewed in reference 6), it was proposed (7) that in situations of “normal” lipoprotein metabolism (ie, the use of normocholesterolemic subjects) with low dietary cholesterol intake (< 300 mg/d) and adequate intakes of linoleic acid (≈4–5% of energy), the dietary fatty acid–induced response to plasma cholesterol is dependent primarily on the intakes of myristic and linoleic acids. In such situations, if myristic and linoleic acids are equalized across diets, the exchange of other fatty acids would not affect plasma lipids. This may partly explain the results of Temme et al. Although the palmitate–oleate exchange did not produce identical lipid responses in their female subjects, a diet-sex effect may have been responsible. It should be pointed out that the regression data on which the palmitate–oleate equivalence scenario was proposed (7), was derived solely from male subjects (cebus monkeys and humans), and its extrapolation to females has yet to be resolved.

We recently reviewed 36 human studies encompassing 148 different diets (8). In 27 of the studies (83 diets), which used normocholesterolemic subjects consuming whole-food diets with < 300 mg dietary cholesterol per day, the observed plasma cholesterol response correlated highly significantly with that predicted on the basis of the dietary myristic and linoleic acid content. Applying this analysis to the male subjects from the Temme et al study, we calculated that the palmitic acid–rich diet would have resulted in a 0.14-mmol/L (5 mg/dL) higher total plasma cholesterol concentration than the oleic acid–rich diet. The observed difference was a nonsignificant 0.19 mmol/L (7 mg/dL). Had the dietary linoleic acid content of both diets been identical, the difference would have been somewhat less. The fact that our analysis closely “predicted” such a response is not important because any regression prediction can match an observed response merely by chance. What is more important is that the data from Temme et al (1), as well as the above-mentioned human studies (2–4), add credence to the underlying metabolic factors (for the palmitate–oleate equivalence), which form the basis for the regression analysis.

Thus, the study by Temme et al (1), in conjunction with recent human studies (2–4), adds further support to the notion that under certain situations (eg, moderate fat load and/or moderate cholesterol load?), palmitic and oleic acids can be “interchanged” without compromising plasma lipids. We would agree with Temme et al (1) that total dietary fat intake may moderate the cholesterol–emeric effect of palmitic acid. This is an important issue that needs to be addressed in future studies.

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REFERENCES


Reply to P Khosla and K Sundram

Dear Sir:

We appreciate the comments made by Khosla and Sundram concerning our recently published paper on the effects of diets enriched in lauric, palmitic, or oleic acids on serum lipids and lipoproteins in healthy women and men (1). We agree that the comparison between palmitic and oleic acids and the importance of dietary intakes of myristic and linoleic acids in regulating plasma cholesterol concentrations, as hypothesized by Hayes et al (2), need further consideration. We, however, do not agree that our study, and especially the data of our male subjects, demonstrated an equal effect of palmitic and oleic acids on serum cholesterol concentrations. The overall results indicated a cholesterol-raising effect of palmitic acid compared with oleic acid (significant increase of 5% compared with oleic acid). However, within our male subjects cholesterol concentrations with the palmitic acid diet did not significantly differ from cholesterol concentrations on either the oleic acid (increase of 3% compared with the oleic acid diet) or the lauric acid diets (decrease of 3% compared with the lauric acid diet). In our view, it is not justified to suggest that in men the effects of palmitic, lauric, and oleic acids on serum cholesterol concentrations are similar: our experiment was designed to detect changes of cholesterol concentrations in the entire group of subjects, and not in women or men separately.

It might be possible that effects of palmitic and oleic acids are sex-dependent. Analysis, however, did not indicate a significant diet-and-sex interaction effect. In addition, the few controlled studies that included both women and men did not find that cholesterolemic effects of palmitic compared with oleic acid diets are different between women and men (3, 4).

From our results we therefore still conclude that under the experimental conditions used, palmitic acid has a cholesterol-raising potency compared with oleic acid in humans. It is possible that certain factors can moderate the cholesterolemic effects of palmitic acid. These factors may include total dietary fat intake, linoleic acid intake, and source of palmitic acid.

We regret that we cannot comment on the calculated predicted cholesterol response in men, because the review mentioned by Khosla and Sundram is, as yet, not available.

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