In conclusion, the majority of studies support the concept that palmitic acid in palm oil raises LDL cholesterol. The state of the palm oil literature is very similar to that of the plant sterol literature. Although meta-analyses convincingly show a 10% decrease in LDL cholesterol with plant sterols and stanols, there is a lower response than the Tholstrup et al study? Is there any valid reason why palmitic acid from palm oil should differ in its effects from palmitic acid from dairy, beef, and lard? In palm oil, palmitic acid is placed in the sn-1 and sn-3 position of the glycerol backbone, whereas it is positioned in the sn-2 position in animal fats. However, Zock et al (9) showed convincingly that this had little effect on LDL-cholesterol concentrations, which parallel Tholstrup et al’s (2) findings in this current article and Forsythe et al (10) in 2007.

In this issue of the Journal there are 2 studies that tested the cholesterol-raising effects of palmitic acid–rich palmolein and that reach apparently different conclusions. Tholstrup et al (1) found in 32 participants that palm oil elevated LDL cholesterol significantly by 0.22 mmol/L (\(P < 0.001\)) compared with olive oil, whereas Voon et al (2) found the difference between palm oil and olive oil to be nonsignificant at 0.14 mmol/L \((P > 0.05\) after Bonferroni correction). However, statistically, it can be shown that these 2 values are not different from one another \((P = 0.3)\), and that in considering both articles together the weighted mean change is \(-0.17\) mmol/L \((P < 0.01)\) or \(-6.5\%\). Other than experimental variation, was there any reason for the Voon et al article to show a lower response than the Tholstrup et al study?

In the Voon et al study, food that was cooked in the test fat by a professional cook was provided to the participants for 5 d, whereas during weekends the participants used the test fat themselves as a cooking oil. Cooked products did not appear to be tested for their fat content. Total fat intake was 31% of energy with 9.7% palmitic acid. Only one fasting blood test was taken at the end of 5 wk, and most participants were women. In the Tholstrup et al study, fat intake was higher (36%), although palmitic acid intake was the same (10%). The chief difference between dietary strategies was that the test fat was baked into buns and cakes so that there was more control over fat intake. The statistical model was more complicated with many more covariates, particularly energy intake and fat intake, and it is not clear what effect this had on the final means. Two blood tests were taken at the end of each period to improve precision. The exclusion of 11 volunteers with a total cholesterol of >5.2 mmol/L, although unusual, is not likely to have increased the effect size. The study consisted only of men, which may have been a factor in the difference because men appear to have a greater LDL response to fat changes than do women (3).

The results of the Tholstrup et al study are very similar to those of Temme et al (4), who, in a study in which all food products were provided, showed that a palmitic acid–rich diet elevated LDL cholesterol by 0.22 mmol/L compared with oleic acid in 32 healthy subjects (men and women) over 6 wk. Palmitic acid was provided from both dairy fat and palm stearin (approximately one-third) and contributed 8.5% of energy in a 40% fat diet.

Vega-Lopez et al (5) showed that palm and trans fat–containing margarines led to 16–18% higher LDL concentrations than did canola-based margarines. This study also referred to another 6 positive palm oil studies in a variety of countries, which used liquid diets, margarines, oils added to food products, and cooking fats, and referred to 4 negative studies, 2 of which are noted here. The elevation in LDL cholesterol with palm oil varied from 0.16 to 0.6 mmol/L.

The 2 earlier negative studies provided the test fats as cooking oils (6, 7). The problem with this type of dietary intervention is that it is difficult to be sure of the amount of fat consumed unless the cooking fat is weighed before and after cooking (or the cooked products, which would also need to be analyzed for fat content as well), which was not done in either study (both relied on food records). In addition, the amount of palmitic acid consumed was lower than in the Voon et al (2) and Tholstrup et al (1) studies.

In 1992 Ng et al (6) compared 7% of energy as palmitic acid from palmolein with 7% of energy as oleic acid from olive oil in a 33% fat diet with two-thirds of the fat from the test cooking fat over a 6 wk period in 33 young healthy subjects. LDL cholesterol was identical for both diets. A similar result was found by Choudhury et al (7) in a study in which palmolein and olive oil were provided as cooking fats for 30 d each in 21 young healthy subjects. There was an increase of 5% of energy as palmitic acid and 7% of energy as oleic acid in the diets. A large, parallel-design Chinese study (8) in 120 participants showed a decrease in LDL cholesterol with palm fat–based fried products (cooked by a local chef), which was similar to that seen with soybean oil, whereas lard elevated LDL cholesterol. It is not clear why this study differed from those described above.

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First published online November 9, 2011; doi: 10.3945/ajcn.111.027060.
is a wide response to the same dose and a not inconsiderable number of negative studies (22 of 59) (11).

The author did not declare any conflicts of interest.

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