TABLE 1
Overall prevalence of goiter in Kashmir Valley, by age

<table>
<thead>
<tr>
<th>Age group</th>
<th>Ob</th>
<th>I</th>
<th>II</th>
<th>III</th>
<th>Total number with goiter</th>
</tr>
</thead>
<tbody>
<tr>
<td>5–10 y (n = 5899)</td>
<td>959 (16.26)</td>
<td>1131 (19.17)</td>
<td>347 (5.88)</td>
<td>0</td>
<td>2437 (41.31)</td>
</tr>
<tr>
<td>11–15 y (n = 4297)</td>
<td>681 (15.85)</td>
<td>1077 (25.06)</td>
<td>412 (9.59)</td>
<td>2 (0.02)</td>
<td>2172 (50.55)</td>
</tr>
<tr>
<td>Total (n = 10 196)</td>
<td>1640 (16.08)</td>
<td>2208 (21.66)</td>
<td>759 (7.44)</td>
<td>2 (0.02)</td>
<td>4609 (45.20)</td>
</tr>
</tbody>
</table>

1 Percent of subjects with goiter in parentheses. Overall prevalence of goiter was significantly greater in children aged 11–15 y, P < 0.001 ($\chi^2 df_2 = 85.61$).

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REFERENCES

Palmolein, olive oil, and serum lipids

Dear Sir:

I wish to comment on the report by Choudhury et al (1). These authors attempted to compare the effects of palmolein, which is relatively high in palmitic acid (16:0), and olive oil, which is relatively high in oleic acid (18:1), on serum lipid concentrations. They observed that concentrations of total and low-density-lipoprotein (LDL) cholesterol were very similar and concluded “that 16:0, though saturated, is not always a plasma cholesterol–raising fatty acid”.

The subjects prepared their food at home and were given either palmolein or olive oil in amounts sufficient to provide 50% of their dietary fat along with extensive instructions about how to use them and how to keep the rest of their diet similar. Complete dietary records were kept and translated into food constituents by food composition tables. The authors stated the following: “we believe that our chosen methodology gave us close estimations of the participants’ true intakes” because the volunteers were highly motivated and health conscious. “Compliance was also assessed by measuring fatty acids in different plasma lipid fractions. . .”

Whatever the authors “believe”, there is no way to determine how good or bad the dietary records actually were or how valid the values in the food composition tables were. There are sufficient data to raise doubts about the reliability of all self-reported food intakes (2, 3) and also to suspect that subjects who have committed themselves to dietary studies produce data are biased toward what they have been told to do (4). When you know exactly what you should do and have promised to follow directions, it is difficult to admit that you have failed to follow instructions.

The changes in the fatty acid content of serum lipids only shows that the diet did change. These changes provide no measure of the degree of dietary change. The fall in serum cholesterol after the palmolein and olive oil diets compared with the concentration after the usual diet was $\approx 0.9$ mmol/L (34 mg/dL). According to my calculations, this value was substantially greater than the one predicted by any of the proposed equations (5–8). However, the SDs of the cholesterol concentrations were rather large, as they almost always are: 1.11, 1.26, and 0.99 mmol/L, respectively, for the usual, palmolein, and olive oil diets. What follows are the corresponding mean ($\pm 2$ SEM) total cholesterol concentrations (in mmol/L) and calculated confidence limits for the usual, palmolein, and olive oil diets, respectively: 5.54 ± 0.24 (5.06, 6.02), 4.65 ± 0.27 (4.11, 5.19), and 4.63 ± 0.22 (4.19, 5.07).

The true mean value for the palmolein diet might be as high as 5.19 mmol/L and that for the olive oil diet might be as low as 4.19 mmol/L. This, of course, is a rather crude comparison because the data provided do not allow for an analysis of variance, which was used by the authors to evaluate the results. However, the question is whether or not the lack of statistical significance means that the effects of the two diets were the
same. I believe that the data only show that the test could not distinguish between the effects of the two diets but provides little support for the conclusion that 16:0 is not hypercholesterolemic.

Quantitative nutrition studies require quantitative data on food consumption. The literature contains the results from many rather poorly controlled trials, which do not have the capacity to distinguish between the effects of the diets being studied. Semiquantitative studies should be clearly distinguished from well-controlled trials. The lack of statistically significant differences does not prove that the effects are the same, especially when the assay technique is very imprecise. As the authors note, most of the studies have concluded that palmitic acid is hypercholesterolemic were conducted with institutionalized subjects and all were done with better dietary control than was provided in this study.

DM Hegsted
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REFERENCES

Reply to DM Hegsted

Dear Sir:

We appreciate Hegsted’s comments and would like to respond to them. Our first point is that our result has not changed the public health message, either from our Unit or in Australia. We presume that our result is an exception to the general rule: monounsaturated fatty acids are better for plasma lipids than are saturated fatty acids. We are, however, comforted in our rather unexpected findings that one other group has had the same results in humans, with all food provided (1) and it is difficult to ignore meticulous primate experiments (2).

Although human experiments in a metabolic ward are the gold standard for control of food intake, they provide an artificial environment and are very costly. Most human nutrition trials reported in The American Journal of Clinical Nutrition did not have their subjects closely confined. For example, in one recent volume (859) of the AJCN there were 27 human experiments, but only one was in a ward (actually in an army camp); in all of the others the subjects were free-living. Experiments in free-living subjects vary greatly in probable reliability. There are, however, important techniques for enhancing reliability. In our trial we had intelligent volunteers (incidentally, most were not nutrition students) who were supplied with the two test oils and returned any of the oil not used each week. They were given repeated advice and support by a research dietitian whose sole job it was to do this. We used a set of biomarkers, which showed significant increases of 18:1 n-9 in three different plasma lipids and in platelets during the olive oil period and in 16:0 in four lipid fractions in the palmolin period. The mean increase of oleic acid in these four fractions (as a percentage of fatty acids) during the olive oil period was 11%. Our plasma tocopherol results also corresponded with the different α-tocopherol contents of the two oils. We took fasting blood samples on three mornings at the end of each test-oil period (to minimize the effect of within-individual variation) in a crossover design.

We suggest in our paper that the expected low-density-lipoprotein-raising effect of an estimated exchange of 5% of energy from palmitic for oleic acid was not seen because our subjects were young, lean, and exercised regularly, for the most part. Because the basal diet was low in fat, their plasma and dietary cholesterol concentrations were low. Conditions were similar for the subjects of Ng et al (1). We also see our results as further support for the original finding of Hegsted, that 16:0 is less cholesterol-elevating than is 14:0 (3).

This year, we started a comparison study of another oil high in oleic acid: high-oleic sunflower oil compared with palmolein. However, this time we are using middle-aged subjects, whose low-density-lipoprotein receptors are likely to be more down-regulated.

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