LETTERS TO THE EDITOR

TABLE 1
Mean plasma homocyst(e)ine concentration and selected characteristics according to categories of usual coffee consumption in a stratified random sample of participants in the baseline examination of the ARIC Study, 1987–1989.

<table>
<thead>
<tr>
<th>Homocyst(e)ine (µmol/L)</th>
<th>0 (n = 159)</th>
<th>&lt; 1 (n = 76)</th>
<th>1 (n = 87)</th>
<th>2–3 (n = 128)</th>
<th>&gt; 3 (n = 87)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total serum cholesterol (mg/dL)</td>
<td>209</td>
<td>217</td>
<td>210</td>
<td>215</td>
<td>223</td>
</tr>
<tr>
<td>(mmol/L)</td>
<td>5.40</td>
<td>5.61</td>
<td>5.43</td>
<td>5.56</td>
<td>5.77</td>
</tr>
<tr>
<td>Dietary folate intake (µg/d)</td>
<td>219</td>
<td>233</td>
<td>220</td>
<td>197</td>
<td>210</td>
</tr>
<tr>
<td>Dietary vitamin B-12 intake (µg/d)</td>
<td>7.15</td>
<td>8.41</td>
<td>8.05</td>
<td>6.95</td>
<td>7.43</td>
</tr>
<tr>
<td>Keys score</td>
<td>42.2</td>
<td>44.0</td>
<td>41.2</td>
<td>41.4</td>
<td>43.8</td>
</tr>
<tr>
<td>Proportion male (%)</td>
<td>10.1</td>
<td>10.9</td>
<td>10.8</td>
<td>9.0</td>
<td>9.7</td>
</tr>
<tr>
<td>Current cigarette smokers (%)</td>
<td>16.1</td>
<td>16.0</td>
<td>10.8</td>
<td>26.3</td>
<td>39.7</td>
</tr>
</tbody>
</table>

Means and proportions were calculated by using analysis of covariance weighted for the stratified sample design. ARIC, Atherosclerosis Risk in Communities.

lesterol-raising effects than filtered coffee (7, 8) is also an unlikely explanation, given that only 2.4% of participants in Nygård et al’s study reported consuming boiled coffee (1).

It is possible that certain lifestyle characteristics (dietary or otherwise) associated with coffee intake in the average Norwegian but not in the average American could explain the discrepant results found in the studies. High consumption of cold breakfast cereal (fortified with folate) in the United States could attenuate the association between coffee intake and homocyst(e)ine. In the ARIC population, participants reporting never consuming breakfast cereal (n = 140) had higher homocyst(e)ine concentrations than the average; restricting the analysis in Table 1 to only these participants, however, did not show any significant trends with coffee consumption, although the numbers might be too small (results not shown). Alternatively, the results of the Norwegian study could stem from residual confounding by smoking or other unmeasured confounders.

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REFERENCES


Reply to FJ Nieto et al

Dear Sir:

We welcome the report of Nieto et al on the relation between plasma total homocysteine (tHcy) and coffee intake in 537 participants of the Atherosclerosis Risk in Communities (ARIC) Study. They were not able to reproduce our finding of an association between coffee consumption and plasma tHcy concentration in 16175 participants of the Hordland Homocysteine Study (1). There are several possible explanations for the discrepant results between the two studies.

We found that the coffee-tHcy relation was particularly pronounced in subjects drinking ≥ 9 cups/d. The population
LETTERS TO THE EDITOR

Truly quantitative dietary studies have stringent requirements

Dear Sir:

I was interested in the discussion by Hayes (1) of the paper by Temme et al (2) and the response to his comments by the authors (3). The paper under discussion reported the serum lipid concentrations induced by three diets, one high in lauric acid (which also contained considerable myristic acid, as emphasized by Hayes), another high in palmitic acid, and the third high in oleic acid. The conclusions of the authors were that "lauric and palmitic acid are hypercholesterolemic compared with oleic acid" and that "lauric acid raises serum total cholesterol concentrations more than does palmitic acid, which is partly because of a greater increase in HDL cholesterol" (2). Although I do not doubt that the saturated fatty acids are hypercholesterolemic compared with oleic acid, I believe this was not a quantitative study and should not be treated as such.

The fat content of the diets was modified by providing margarines, dairy products, and bakery goods. The subjects were given detailed diet instructions and recorded the free-choice items selected and also any deviations from study protocol. They also recorded their food intake for two working days and two weekend days.

There is now abundant evidence that none of the diet-assessment methods available accurately reflect food intake. For example, Sawaya et al (4) compared 24-h recalls, food-frequency questionnaires, and weighed dietary intakes and concluded that "none of the methods studied gave accurate estimates of the usual energy requirements of individual subjects." Many other papers using doubly labeled water to estimate energy expenditure support this finding. In addition, we have the logical conclusion of Mertz (5) that experimental subjects may often be less reliable than those who have not committed themselves in dietary studies. Nevertheless, the dietary data provided by Temme et al (2) are the only information available and they clearly demonstrate the problems with this study.

For example, the mean reported fat intake was approximately 41.5% of energy in the three diets with a SD of 5.5. The individual values are presumably within 2 SD on either side of the mean so fat intake apparently ranged from about 30% to 50%. Similarly, the CVs of palmitic acid intake were 25.4%, 17.4%, and 19.3% for the three diets. This means that the values ranged from 34% to 50% above and below the mean. The CV in linoleic acid intake was about 45% and that of dietary cholesterol was of similar magnitude. Some subjects apparently recorded linoleic acid intakes as high as 8% and, although the mean intake of cholesterol was recorded as ~222 mg/d, some recorded values > 400 mg/d. Because the distributions of intake are undoubtedly skewed—it is unlikely that


 References