mmol/L, an effect they suggest is due to terpenoids in coffee. However, I cannot find any comments in the paper about whether the subjects in the study took milk or cream in their coffee. I recently quit drinking coffee at work and as a result reduced my intake of milk by ~500 mL (2 cups)/d. It seems to me that the reduction in cholesterol could have been due, at least in part, to a reduction in saturated fat intake resulting from not drinking the milk or cream otherwise used in coffee. The subjects in this study stopped drinking an average of nearly 5 cups (~875 mL) coffee/d. If they were using 30 mL whole milk/cup, they reduced their milk intake by 150 mL/d, an amount that contains 5 g fat, of which ~60% is saturated. Assuming an energy intake of 8.4 kJ/d, reducing milk intake by 150 mL/d and replacing it with water or juice would reduce saturated fat by nearly 1.5% of energy. According to the Key’s equation (2), this would be expected to reduce serum cholesterol by ~0.1 mmol/L, or 35% of the observed effect of coffee. If the subjects used cream in their coffee, then the reduction in saturated fat intake could be 2–3 times more than that for milk and might account for the entire effect Christensen et al observed. Of course, if Norwegians like their coffee black, then these musings are nothing more than that!

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REFERENCES

Reply to TMS Wolever

Dear Sir:

Our study, recently published in the Journal, concluded that abstention from filtered coffee reduces the concentrations of plasma homocysteine and serum cholesterol (1). We conducted a randomized prospective intervention study organized as an unblinded controlled trial with the participants randomly assigned to 3 different treatment groups that were to consume for 6 consecutive weeks no coffee, 1–3 cups (~175–525 mL) coffee/d, or ≥4 cups (~700 mL) coffee/d. Inclusion criteria were age 24–69 y, a history of daily consumption of coffee for ≥5 y, and no daily tobacco smoking for the past 6 mo. To assess the coffee–total homocysteine as well as the coffee–total cholesterol association, we controlled for both dietary habits and brewing methods. All participants were asked to follow their usual diet during the trial: the coffee-consuming groups were permitted to drink the type of coffee to which they were accustomed. Before the randomization step, data were recorded on the participants’ usual diets (including whether they used milk in their coffee) in the year before entering the trial. After finishing the trial, the participants were asked to report any dietary changes that had taken place during the trial (2). Wolever asks whether the reduction in plasma homocysteine and serum cholesterol concentrations we observed could have been caused by a reduction in milk intake by the group that abstained from coffee.

Before random assignment, 91% of the participants reported that they consumed their coffee black, which agrees with our general impression that most Norwegians prefer black coffee. After finishing the trial, the vast majority of the participants in all groups reported that they had not changed their total intake of milk during the study. Of the 69 participants in the coffee-abstaining group who filled in the questionnaire, 4 (5.8%) reported that they had reduced their intake of milk or dairy products during the trial. Eight of the 69 (11.6%) reported that they had increased their milk intake while abstaining from coffee. In the group that consumed the highest amount of coffee, the corresponding numbers were 4 of 70 (5.7%) reporting a reduction in milk intake and 5 reporting an increase (7.1%). One participant in each of the above groups did not answer this question. Chi-square statistical tests did not show any significant differences between the reported differences in milk intake in the 3 groups.

On the basis of these data, we conclude that reduced milk intake is not likely to explain the observed reduction in plasma homocysteine or serum cholesterol. The observed association is in line with the results of other intervention studies (3, 4).

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REFERENCES
High-calcium diets and fracture prevention

Dear Sir:

Contrary to Hegsted’s comment that there is little evidence that high calcium intakes effectively prevent fractures (1), there is ample evidence that such is the case (2–4). In a similar vein, he refers to increasing evidence that diets high in fruit and vegetables are beneficial in preventing fractures, yet the references cited are far from convincing (5–7). Most puzzling is the question: Why do populations who consume low-calcium diets have fewer fractures than do Western societies who consume high-calcium diets?

To begin with, in Western societies with high calcium intakes, the consumers of high-calcium diets are not the women who most need the calcium (8, 9). If, as one suspects, the low-calcium consumers referred to are Asian, it must be taken into account that quantification of fractures in many Asian countries has been sporadic at best. Moreover, Asians have some protective factors against hip fractures that whites lack, such as shorter hip axis lengths and smaller frames with lower centers of gravity (10, 11). People in Asia tend to lead a more active lifestyle, which helps build strong bones, and they do not have to walk in snowy, icy conditions, which increase the risk of slipping and falling. In addition, Westerners have a higher life expectancy than do people in many Asian countries, allowing greater opportunity to develop osteoporosis (12).

Hegsted’s statement that recommended calcium intakes are now so high that it is difficult, if not impossible, to devise practical diets that meet these recommendations is also puzzling. How about the Food Guide Pyramid (13) or Canada’s Food Guide (14)? Three daily servings of milk products—for example, an 8-oz glass of skim milk with breakfast, lunch, and dinner—with a balanced diet yields about 1200 mg Ca.

The puzzlement continues with the reference to the unreliability of dietary intakes: the references cited all refer to an underestimation of energy intake, not to intakes of specific nutrients. Surely calcium, coming as it does from one main food group, is much less likely to be inaccurately estimated.

The fact that long-standing recommendations to increase calcium intakes appear to have had little or no effect on the prevalence of osteoporosis or fractures in the United States in no way proves that the recommendations are invalid, anymore than increasing levels of obesity in the United States prove that the long-standing recommendations to reduce fat intake are invalid.

To cite Kanis’s assertion (15) that there are no adequately controlled studies to show whether increased calcium intake has an effect on skeletal Consolidation or subsequent fracture risk before or after longitudinal growth has ceased is to dismiss the work of many respected investigators in the field, including that of Heaney (3).

Perhaps most puzzling of all is the reference to the Harvard Nurses Health Study (16). First, the results of this study were not statistically significant, but more to the point, why not refer to more recent, contradictory data by the same authors (17)? This 1998 study examined vitamin D receptor genotype and the risk of bone fractures, also using data from the Nurses Health Study. It observed a greater risk of bone fractures for women who were older, leaner, or less physically active or who had low calcium intakes.

In fact, a recent analysis of 139 articles on the role of calcium in skeletal health published over the past 25 y provides convincing evidence for calcium’s benefits (3). In all but 2 of 52 investigator-controlled calcium-intervention studies, an increase in calcium intake improved bone balance, increased bone gains during growth, reduced bone loss in later years, or lowered fracture risk. Similar beneficial effects of calcium were found in ≈75% of 86 observational studies.

Although many factors, nutritional and nonnutritional, contribute to bone health, the beneficial effect of calcium is clearly major. More evidence confirms these results, as noted by Heaney in his recent study (3): “Since submission of this manuscript, 13 additional reports have been published, one metabolic study, 4 randomized controlled trials, and 8 observational studies. All 13 found a benefit from extra calcium.”

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