Moderate alcohol consumption and the risk of cardiovascular disease

Dear Sir:

We read with interest the results of the fifth examination cycle of the Framingham Offspring Study concerning plasma concentrations of the cardiovascular disease risk factor total homocysteine (tHcy), as reported by Jacques et al (1) in the Journal. The authors concluded that different dietary and lifestyle factors—such as intakes of vitamin B-6, riboflavin, alcohol, and caffeine; smoking; and hypertension—influence circulating tHcy concentrations. We wish to emphasize the influence of alcohol consumption on concentrations of plasma homocysteine.

Pathologically raised concentrations of plasma homocysteine have been reported in patients with chronic alcoholism (2, 3) and during alcohol withdrawal (4), whereas normal concentrations are found in alcohol-intoxicated patients who are not alcohol dependent (5). It has been proposed that ethanol-induced hyperhomocysteinemia may be a significant factor in the increased incidence of coronary artery disease and stroke related to high alcohol consumption (6, 7). However, the results of various epidemiologic studies suggest that moderate alcohol intakes of the equivalent of 2 drinks/d (20–40 g alcohol/d) of any kind of alcohol, especially red wine, are associated with a reduced incidence of coronary artery disease, a phenomenon referred to as the French paradox (8).

In a previous study (9), we examined whether mild-to-moderate alcohol consumption—referred to as social drinking—changes plasma tHcy concentrations. We compared the plasma concentrations of abstinent individuals with those of non-alcohol-dependent social drinkers who consumed 30 g alcohol/d over a period of 6 wk. The social drinkers were further divided into 3 groups according to the source of alcohol consumed (beer, red wine, or spirits). We found abstinent individuals to have significantly lower concentrations of endogenous tHcy than did consumers of beer, red wine, or spirits. Additionally, consumers of red wine and spirits had pathologically raised plasma tHcy concentrations at the end of the observation period, whereas the concentrations in beer consumers were significantly raised but still within the normal range (9).

These results support Jacques et al's observation that lifestyle habits, especially the consumption of alcohol, significantly influence concentrations of plasma tHcy. In contradiction to the cardioprotection of alcohol suggested by the French paradox, we postulate that the elevated concentrations of tHcy in subjects with a social drinking pattern of regular, moderate alcohol intake are a risk for cardiovascular disease. Nevertheless, further investigations and controlled studies are needed to clarify the possible association between social drinkers' alcohol consumption, homocysteine concentrations, and the risk of cardiovascular diseases.

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Abstention from filtered coffee reduces the concentrations of plasma homocysteine and serum cholesterol

Dear Sir:

Christensen at al (1) describe a randomized trial showing that abstaining from coffee reduced serum cholesterol by 0.28

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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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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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 ≈236-mL (8-oz) glass of skim milk with breakfast, lunch, and dinner—with a balanced diet yields ≈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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Reply to HB MacDonald

Dear Sir:

I expected a number of adverse comments about my recent article in the Journal (1). After all, high-calcium diets have been promoted by the dairy industry, the nutrition establishment, and much of the medical profession for 80 y or more. The dairy interests have a large stake in this and cannot be expected to be unbiased.

It is not useful to reiterate all of the arguments presented in my original paper (1). Whatever the strengths and weaknesses of the epidemiologic studies within the United States may be, they provide little or no support for high-calcium diets (2, 3). Increases in calcium retention from balance trials and increases in bone mineral, however measured, are usually interpreted as beneficial (4). Yet the calcium retentions reported in many trials are obviously unreal, and most balance trials are interpreted to yield false retentions (5). It remains to be shown that modest increases in bone mineral actually reduce the fracture rate. Wilkin (6) argues that bone density is not a good predictor of hip fracture and that "some 85% of the contribution to the rise in fracture rate with age is unrelated to bone density." He also states that the data on the antiresorptive drug risedronate show that the risk of fracture had fallen well before bone density peaked and that such trials suggest that "antiresorptive drugs can halve the risk of fracture . . . without restoring significant bone density." He adds that "high turnover of bone seems to be intrinsically unstable, whereas low bone density need be weak only if its low mineral content results from chronically high bone turnover."

Many factors do or have been suggested to modify the risk of osteoporosis and fractures, including sex, genetics, stature, exercise, obesity, and intakes of vitamin D, vitamin A, calcium, fluoride, sodium, potassium, protein, fruit, and vegetables. Ironically, if high calcium intakes are beneficial, supplements may be more helpful than dairy products because high animal protein intakes increase urinary calcium excretion. Most epidemiologic studies in the United States have failed to identify any of the above dietary factors as serious risk factors, although the data on vitamin D appear rather convincing in some situations, and, given the limitations of such studies, I find the data on fruit and vegetables most interesting.

Although more quantitative data on fracture rates in various parts of the world are welcome, we know that populations around the world that use few dairy products and have relatively low calcium intakes develop reasonably well and are obviously not falling apart from fractures. On the other hand, fracture rates are obviously high in the countries that consume the Western-type diet. The evidence that the administration of the hydroxymethylglutaryl-CoA reductase inhibitors (the statins, widely used to reduce serum cholesterol concentrations) also reduce fracture rates is substantial, although the results of any controlled trials are not yet available (7). The statins block the mevalonate pathway, which clearly suggests that dietary practices that promote high cholesterol concentrations also induce a mechanism that makes bone fragile in the elderly. Perhaps this unknown mechanism stimulates bone turnover, as suggested by Wilkin (6), also via the mevalonate pathway, and is also blocked by the statins. Research devoted to this possibility, rather than to the oft-repeated studies of the effects of calcium, should be rewarding.

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Is dietary carbohydrate essential for human nutrition?

Dear Sir:

I read with interest the article by Dewailly et al (1) regarding diet and cardiovascular disease in the Inuit of Nunavik, but I was disappointed that no information regarding macronutrient intake was presented or considered in the estimation of cardiovascular

risk. The traditional Inuit diet consists primarily of protein and fat, somewhat similar to the low-carbohydrate diets promoted in popular weight-reducing diets (2). These diets have caused concern among nutritionists because of the metabolic changes and health risks associated with limited carbohydrate consumption (3). However, in exploring the risks and benefits of carbohydrate restriction, I was surprised to find little evidence that exogenous carbohydrate is needed for human function.

The currently established human essential nutrients are water, energy, amino acids (histidine, isoleucine, leucine, lysine, methionine, phenylalanine, threonine, tryptophan, and valine), essential fatty acids (linoleic and α-linolenic acids), vitamins (ascorbic acid, vitamin A, vitamin D, vitamin E, vitamin K, thiamine, riboflavin, niacin, vitamin B-6, pantothenic acid, folic acid, biotin, and vitamin B-12), minerals (calcium, phosphorus, magnesium, and iron), trace minerals (zinc, copper, manganese, iodine, selenium, molybdenum, and chromium), electrolytes (sodium, potassium, and chloride), and ultratrace minerals (4). (Note the absence of specific carbohydrates from this list.)

Although one current recommended dietary carbohydrate intake for adults is 150 g/d, it is interesting to examine how this recommendation was determined at a recent international conference (5):

"The theoretical minimal level of carbohydrate (CHO) intake is zero, but CHO is a universal fuel for all cells, the cheapest source of dietary energy, and also the source of plant fiber. In addition, the complete absence of dietary CHO entails the breakdown of fat to supply energy [glycerol as a gluconeogenic substrate, and ketone bodies as an alternative fuel for the central nervous system (CNS)], resulting in symptomatic ketosis. Data in childhood are unavailable, but ketosis in adults can be prevented by a daily CHO intake of about 50 g. This value appears to approximate the quantity of glucose required to satisfy minimal glucose needs of the CNS and during starvation. The Group therefore concluded that the theoretical minimum intake of zero should not be recommended as a practical minimum.…about 100 g of glucose/d are irreversibly oxidized by the brain from the age of 3–4 y onward. However, this excludes recycled carbon, gluconeogenic carbon, for example from glycerol, and it does not account for glucose used by other non-CNS tissues. For example, in the adult, muscle and other non-CNS account for an additional 20–30 g of glucose daily. For this reason a safety margin of 50 g/d is arbitrarily added to the value of 100 g/d and the practical minimal CHO intake set at 150 g/d beyond the ages of 3–4 y."

Thus, although carbohydrate could theoretically be eliminated from the diet, the recommended intake of 150 g/d ensures an adequate supply of glucose for the CNS. However, it appears that during starvation (a condition in which the intakes of carbohydrate, protein, and fat are eliminated), an adequate amount of substrate for the CNS is provided through gluconeogenesis and ketogenesis (6). The elimination of dietary carbohydrate did not diminish the energy supply to the CNS under the conditions of these experiments. Second, carbohydrate is recommended to avert symptomatic ketosis. In the largest published series on carbohydrate-restricted diets, ketosis was not typically symptomatic (7).

The most direct way to determine whether carbohydrate is an essential nutrient is to eliminate it from the diet in controlled laboratory studies. In studies involving rats and chicks, the elimination of dietary carbohydrate caused no obvious problems (8–12). It was only when carbohydrate restriction was combined with glycerol restriction (by substituting fatty acids for triacylglycerol) that chicks did not develop normally (13). Thus, it appears that some minimum amount of a gluconeogenic precursor is essential—for example, glycerol obtained from fat (triacylglycerol) consumption. More subtle abnormalities from carbohydrate elimination might not have been observed in these studies. In addition, the essentiality of some nutrients is species-specific; therefore, these studies do not provide convincing evidence that elimination of dietary carbohydrate is safe in humans (4).

The usual way to discover the essentiality of nutrients is through the identification of specific deficiency syndromes (4). I found no evidence of a carbohydrate deficiency syndrome in humans. Protein deprivation leads to kwashiorkor, and energy deprivation leads to marasmus; however, there is no specific carbohydrate deficiency syndrome. Few contemporary human cultures eat low-carbohydrate diets, but the traditional Eskimo diet is very low (≈50 g/d) in carbohydrate (2). It is possible that if more humans consumed diets severely restricted in carbohydrate, a carbohydrate deficiency syndrome might become apparent.

When carbohydrates are eliminated from the diet, there is a risk that intakes of vitamins, minerals, and perhaps yet unidentified beneficial nutrients provided by carbohydrate-rich foodstuffs (eg, fiber) will be inadequate. There are case reports of extreme dieters who probably developed deficiencies. One dieter who only ate cheese, meat, and eggs (no vegetables) was reported to have developed thiamine-deficient optic neuropathy (14). Another dieter may have developed a relapse of acute variegate porphyria (15). However, most of the current low-carbohydrate, weight-reducing diets advocate the consumption of low-carbohydrate vegetables and vitamin supplements.

Although there is certainly no evidence from which to conclude that extreme restriction of dietary carbohydrate is harmless, I was surprised to find that there is similarly little evidence to conclude that extreme restriction of carbohydrate is harmful. In fact, the consequential breakdown of fat as a result of carbohydrate restriction may be beneficial in the treatment of obesity (7). Perhaps it is time to carefully examine the issue of whether carbohydrate is an essential component of human nutrition.

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Reply to EC Westman

Dear Sir:

In response to Westman, we acknowledge that no information regarding macronutrient intake was presented or considered in our estimation of cardiovascular risk among the Inuit of Nunavik. We are aware that factors other than those considered in our study may be partially responsible for the observed differences in the incidence of cardiovascular disease between the Inuit and Western populations. However, on the basis of current knowledge and data from the Santé Québec Health Survey (1, 2), we believe that the associations among variables in our study were well quantified and met the goal of the study (3), which was to verify the relation between plasma phospholipid concentrations of the n−3 fatty acids eicosapentaenoic acid and docosahexaenoic acid and various cardiovascular disease risk factors among the Inuit of Nunavik.

As mentioned in the introduction section of our article, important changes occurred in the traditional Inuit diet, primarily between 1950 and 1970 when the Inuit population settled into permanent communities. Previous to this time period, the Inuit lived off the land, rivers, lakes, and the sea and appear to have avoided nutritional deficiencies by eating all animal parts (4). Their diet was traditionally high in protein and fat and low in carbohydrate. However, even though the Inuit diet is still rich in meat and fish today (including game), the Santé Québec Health Survey conducted among the Inuit of Nunavik in 1992 showed no evidence of a very low carbohydrate intake among this population. On the basis of a 24-h dietary recall, the average carbohydrate intake in this population was estimated to be 202 g [or, 24.38 g MJ (102 g/1000 kcal)] on the day before the survey (2).

The mean contribution of carbohydrate (42%) to the total energy intake of the Inuit population appeared to be somewhat lower than that reported (47%) in the survey conducted among the Quebec population in 1990, and tended to be higher in the younger than in the older Inuit (2). On the other hand, the percentage contribution of energy from protein (20%) and lipids (37%) was, on average, higher in the Inuit population than in the Quebec population (16% and 34%, respectively). However, the contribution of saturated fatty acids tended to be slightly lower in the Inuit than in the Quebecers, and the n−3 fatty acid intake of the Inuit was substantially higher than that of the Quebecers.

As described in Subjects and Methods, a 24-h dietary recall was used to assess the amounts of marine foods consumed by men and women in the Inuit community on the day before the survey. The value of the 24-h dietary recall in assessing the intake of groups is well established (5). It provides a fairly accurate estimate of a population's average intake. As reported by Willett (5), we believe that the investigation of relations between cardiovascular disease risk factors and macronutrient intakes requires the use of an estimate of individual intakes over >1 d. For this reason, we did not want to present correlations between individual macronutrient intakes (including carbohydrates) on a single day and cardiovascular disease risk factors. On the other hand, plasma concentrations of n−3 fatty acids are good biomarkers of fish intake. Indeed, it is generally recognized that the measurement of eicosapentaenoic acid and docosahexaenoic acid in plasma phospholipids discriminates long-term fish eaters from nonfish eaters quite well (6–8).

In conclusion, even though the carbohydrate intake of the Inuit population is slightly lower than that of the Quebec population, we do not consider this intake to be very low. Moreover, we believe that the traditional diet of the Inuit must be maintained because it provides a high intake of n−3 fatty acids, which contributes to the low prevalence of cardiovascular diseases in this population.

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