comparable with the rest of the cohort because they did not respond to the initial invitation. Thus, we do not have data from the summer months. A look at the data from the rest of the year showed that blood pressure values were the same during the autumn, winter, and spring.

Unfortunately, we do not have data on outdoor compared with indoor activity. Accordingly, we are not able to reassess the blood pressure data as suggested by Boucher. In retrospect, I regret that the above information was not included in the original article (1).

Dear Sir:

Dairy consumption and bone health

In a recent review article that examined the evidence of a link between dairy foods and bone health, Weinsier and Krumdieck (1) conclude that “the body of scientific evidence appears inadequate to support a recommendation for daily intake of dairy foods to promote bone health in the general US population.” They base their conclusions on a division of studies into those that showed a favorable effect of dairy food intake on bone health and those that did not. They prioritized studies according to the strength of the evidence. Category A studies (the strongest) were randomized controlled trials or longitudinal cohorts with ≥3000 participants who were followed for >5 y. However, these 2 types of studies are not equivalent. Observational studies can accurately assess the outcome measures of bone mineral density or fracture, but their ability to assess dietary intakes is weak. Large size does not overcome that weakness; it merely adds a spurious sense of accuracy. The number of epidemiologic studies that did not show a significant relation is not surprising considering the weak ability to determine the independent variable.

Observational studies of the effect of folate on neural tube defects showed a pattern nearly identical to that found for calcium. It was the randomized controlled trials that confirmed the importance of increasing folate intake during the reproductive years.

If only randomized controlled trials were assigned to category A, then studies showing a favorable relation between dairy food intake and bone health would number 5 and span age groups ranging from 11 y through menopause. Studies showing an unfavorable effect of dairy food intake on bone health would have no category A studies.

Thus, using the same evidence as that used by the authors, we conclude that adequate dairy consumption is supportive of good bone health. One of us recently reviewed this same literature and came to just that conclusion (2).

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REFERENCES

Reply to CM Weaver and RP Heaney

Dear Sir:

We appreciate the comments of Weaver and Heaney, whom we respect as being internationally recognized experts in the field of calcium metabolism and bone disease. They raise a valid concern that randomized controlled trials (RCTs), especially double-blind trials, are needed to establish causal relations in clinical investigations and that even large longitudinal cohort studies may not be equivalent for this purpose. As they indicate, there are both strengths and weaknesses in longitudinal observational studies, notably, the ability of such studies to detect hard endpoint outcomes such as bone fractures and their limited ability to accurately assess dietary intake. However, there are also shortcomings in the few RCTs on the effect of dairy foods on bone status.

In our review (1) we classified 2 RCTs (2, 3) in the favorable-effect category because each study showed less bone loss in the dairy-supplemented group. On the other hand, at the end of the intervention neither trial resulted in greater bone mass in the dairy-supplemented than in the nonsupplemented group. Four of the 5 RCTs classified in the favorable-effect category were not blinded and did not have a placebo control. Hence, confounding variables were not always removed. This is evident in one RCT of adolescent girls in which the dairy-supplemented group had a 50% greater energy intake than did the control group and in which energy intake correlated significantly with greater bone mineral content (3). A further potential shortcoming of the available RCTs was summarized in Heaney’s (4) recent review article in which he states that “all controlled manipulations of calcium intake produce a bone remodeling transient, generally expressing itself during the first year of treatment.” The average length of the 5 RCTs assigned to the favorable-effect category was 1.5 y, with a range of 14 wk to 3 y; none included a baseline period of
adapation to the intervention. Thus, there is the risk that the positive effects seen in these RCTs may be, as Heaney (4) pointed out, inflated by being a compound of the remodeling transient plus an improvement in bone balance. By contrast, observational studies do not alter customary calcium intake, thereby avoiding the confounding problems of the remodeling transient.

Nevertheless, assuming that observational studies should not be given level A strength-of-evidence status equal to that of RCTs, downgrading the one observational study in the unfavorable-effect category (5) to level B status does not change the results. That is, the reported ratio of favorable to unfavorable effects for the stronger-evidence categories A and B remains low and unchanged at 2:1 (6 favorable and 3 unfavorable outcomes). We believe that the more important point is that there are too few carefully designed studies of the effects of dairy foods on bone health.

Weaver and Heaney conclude that inadequate dairy consumption supports good bone health. We agree with their conclusion, if qualifying terms are added to clarify that adequate consumption of certain types of dairy foods appears to be supportive of good bone health among select age, race, and sex groups. Two important caveats are as follows. J) A clear distinction must be made between milk and its derived dairy products in discussions of dairy foods and bone status. Many dairy foods, particularly processed cheese products and cottage cheese, have markedly different ratios of calcium to potassium, sodium contents, and renal acid loads than does milk, and may have markedly different effects on the way calcium is metabolized. 2) There are too few studies in males and ethnic minorities for conclusions to be drawn about the effect of any dairy food on bone health in these groups, which together represent more than one-half of the US population. Hence, the body of scientific evidence is inadequate to support a recommendation for daily intake of dairy foods to promote bone health in the general US population.

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REFERENCES

Abnormal fatty acid status in patients with Crohn disease

Dear Sir:

We read with interest the article by Jeppesen et al (1) that assessed the influence of administration of enteral or parenteral nutrition on plasma phospholipid essential fatty acid (EFA) concentrations in patients with malabsorption. This was a well-designed study that included 4 groups of patients; 2 groups received parenteral nutrition (groups C and D), group A had fat malabsorption of <50% of fat intake, and group B had fat malabsorption of >50% of fat intake. Group C received parenteral nutrition containing lipids and EFA and group D received fat-free parenteral nutrition. EFA absorption was negligible in groups C and D and EFA supplementation in group C was not enough to completely reverse biochemically EFA deficiency. The authors concluded that EFA requirements in patients receiving parenteral nutrition are higher than the amounts recommended to patients with preserved intestinal absorption.

However, as McCowen et al (2) pointed out in a letter to the Journal, a major concern with the study is that most of the patients had Crohn disease. In fact, an abnormal fatty acid profile has been described in patients with active (3) and inactive (4) Crohn disease and in both plasma (3, 4) and intestinal mucosa (5). Plasma long-chain n–3 polyunsaturated fatty acids (PUFAs) were significantly elevated in patients with Crohn disease (3) and a similar but nonsignificant trend was observed for n–6 long-chain PUFAs in patients with inactive disease (4). This finding is consistent with results of the study by Jeppesen et al that showed higher values of both 20:3n–6 and 20:4n–6 in all the groups studied (including most patients with Crohn disease) than in the control group (1).

In addition, elevated concentrations of long-chain n–6 PUFAs were observed in the intestinal mucosa of patients with Crohn disease (5). Similar findings were observed in patients with ulcerative colitis and in experimental colitis, suggesting that there is a common biochemical response to intestinal inflammation (5).

Enteral nutrition has a primary effect on the course of Crohn disease, inducing remission in 60% of patients (6). It is not known which component or components of the diet are responsible for the control of inflammation, but there are some data suggesting that the lipid composition of the diet may play a crucial role (6, 7).

Thus, the authors should be cautious before recommending high doses of parenteral EFAs in patients with Crohn disease because these patients have increased amounts of products of these fatty acids, especially in intestinal mucosa (5). This maneuver might exacerbate the activity of the disease (8). This nutritional recommendation would probably be more useful in patients with short-bowel syndrome and EFA deficiency due to another non-immune–mediated disease.

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