Dear Sir:

In recent letters to the Journal by Horrobin (1) and Zeisel (2), the unexpected failure of β-carotene in clinical trials of lung cancer (3) was explained by the fact that free radicals may be involved both in the elimination of cancer cells and in the generation of mutations that help to initiate cancer. Although the cancer-preventive effects of β-carotene appear to originate mainly in its strong free radical scavenging activity against DNA damage, β-carotene has also been shown to modulate cell proliferation and differentiation through antiproliferative effects on human lung cancer cells (4) and through the enhancement of gap junctional intercellular communication (5). When these effects are taken into account, β-carotene does not appear to promote lung cancer by free radical elimination.

Recently, it was reported that, despite its possible procarcinogenic action as indicated in an editorial by van der Vliet (6), β-carotene acts as an antimtumor promoter rather than as a tumor promoter in a 2-stage model of skin carcinogenesis (7). We speculate that the reason for β-carotene’s failure in the lung cancer trials can be explained by multistage carcinogenesis. Most of the elderly persons with long-term smoking habits who participated in the Alpha-Tocopherol, Beta Carotene Cancer Prevention Study (3) were likely to have mutated genes or premalignant lesions before starting the β-carotene supplementation. Because β-carotene is believed to be most effective in the initiation and promotion stages of carcinogenesis, it may not have shown beneficial effects in the progression stage. Therefore, we believe that β-carotene does not promote cancer.

However, we do not recommend β-carotene as a dietary supplement to prevent cancer, either. A recent study showed an inverse relation between plasma vitamin C and mortality due to cancer (8). Similar results of lower cancer mortality were obtained in another study that examined daily fruit consumption (9). Recently, we reported that most of the antioxidant and antiproliferative activities of apples result not from vitamin C but mainly from the synergistic effects of phytochemicals (nonvitamins) (10). These results suggest that the cancer-preventive effects of vegetables and fruit may result from phytochemicals rather than from β-carotene, vitamin C, and vitamin E. Therefore, we suggest that balanced diets high in phytochemicals and vitamins may be more advantageous than dietary supplements of single vitamins alone.

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REFERENCES

Dear Sir:

In a recent issue of the Journal, Garaulet et al (1) report that adipose tissue from the perivisceral depot (surrounding the gallbladder) has a relatively high saturated fatty acid content and a relatively low monounsaturated fatty acid content compared with fat from other abdominal depots. They also report that there were relatively low monounsaturated fatty acid content compared with fatty acid composition of abdominal adipose tissue samples. However, the interpretation of these findings might be complicated by the authors’ method of selecting their study subjects.

All of the 84 subjects in their study were patients admitted to a hospital for abdominal laparoscopy or laparotomy, but the sites of adipose tissue sampling depended on the patient’s type of surgery and surgical diagnosis (specifically gallbladder, ulcer, or umbilical hernia). Assuming that the subjects providing abdominal adipose tissue samples were probably taken from patient groups with different medical conditions. The perivisceral tissue probably was sampled from patients with gallbladder disease, whereas the omental tissue probably came from patients with ulcer or hernia. If this is true, then any differences observed in the fatty acid compositions could reflect the subject’s medical condition rather than the anatomic site of the adipose tissue specimen. Persons with gallbladder disease tend to have a higher weight and higher glucose and insulin concentrations (2, 3). Because these conditions are also characteristic of the insulin resistance syndrome, it could be important to learn whether the adipose tissue from patients with gallbladder disease (regardless of anatomic site) has a relatively higher concentration of saturated fatty acids.

Fasting serum insulin concentrations are useful in estimating insulin resistance in nondiabetic populations (4) but serve less well in estimating insulin resistance among subjects with impaired glucose tolerance or diabetes (5), ie, persons with diminished insulin production. In Garaulet et al’s study, the potential association between insulin resistance and fatty acid composition in tissue might have been obscured by undiagnosed diabetes among the subjects. Despite the investigators’ intention to exclude patients with diabetes, many of their subjects had impaired fasting glucose or type 2 diabetes as evidenced by their relatively high mean fasting glucose concentrations (x ± SD: men, 6.9 ± 3.8 mmol/L; women, 6.2 ± 2.7 mmol/L). The threshold value for diagnosing impaired fasting glucose is 6.1 mmol/L and for provisionally diagnosing diabetes is 7.0 mmol/L. Thus, many of Garaulet et al’s subjects had some degree of pancreatic insufficiency. Given their heterogeneous study population, perhaps Garaulet et al could use alternative analytic approaches to determine whether the fatty acid composition of adipose tissue is associated with insulin resistance. This would be of interest because previous studies have reported an association between the fatty acid composition of a person’s diet and an elevated risk of type 2 diabetes (6, 7).

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References


