CORRESPONDENCE

VITAMIN E AND THE RISK OF CORONARY DISEASE

To the Editor: The two studies suggesting a substantial reduction in the risk of coronary heart disease associated with the use of vitamin E supplements (May 20 issue)¹ ² provide further compelling evidence about the importance of oxidation in the process of atherogenesis. We take issue, however, with the conclusion that it is premature to recommend vitamin E supplementation for the prevention of coronary heart disease.³

Until recently, physicians have generally maintained a healthy skepticism about vitamin supplementation, viewing it as unnecessary but probably harmless (when given in reasonable dose ranges). Vitamin E appears to provide substantial protection against coronary heart disease at minimal cost. Numerous studies have used vitamin E in the dose range of 200 to 800 IU per day, with virtually no reported toxicity.⁴ The arguments for withholding this treatment from patients who have coronary heart disease or are at high risk for it while investigation continues are unconvincing, especially in the context of current practices in clinical cardiology. For example, the use of coronary angioplasty (which is expensive and is associated with considerable morbidity as compared with vitamin E) has grown exponentially since its introduction in 1977. Yet randomized trials documenting the effectiveness of elective coronary angioplasty in prolonging life or reducing cardiac events are still not available. At the very least, physicians should sanction the use of vitamin E in patients who have coronary heart disease or are at high risk, especially the growing number of patients who ask permission to use it. A substantial number of physicians in the United States are sufficiently convinced of the potential benefits and nontoxic nature of vitamin E to supplement their own diets with it. If vitamin E is good enough for physicians, should it not be good enough for our patients?

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To the Editor: Reduction in the oxidation of low-density lipoproteins by the antioxidant action of vitamin E is a possible explanation of the findings of Stampfer et al. and Rimm et al. However, the platelet is the principal protagonist in the development of the cardiovascular complications (e.g., myocardial infarction and ischemic stroke) that constituted the end points of the study. My colleagues and I have shown that dietary vitamin E supplementation is very effective in inhibiting platelet aggregation ex vivo.¹ This effect of vitamin E is probably mediated by an action not directly related to its antioxidant activity.² In distinct contrast, vitamin E was a poor inhibitor of platelet aggregation (release) when tested ex vivo,³ even though it showed good inhibition in vitro.⁴ This may be due to the inherently very high antioxidant capacity of plasma, which obscures the effect of the vitamin E supplement. Reduced platelet adhesiveness may have played an important part in preventing cardiovascular complications in the two studies.

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