

Letters to the Editor

SUPPLEMENTATION VERSUS DIET FOR ANTIOXIDANT BENEFITS

To the Editor:

Thank you for Dr. Klausner's excellent article, "Nutrition, Lipid Oxidation, and Coronary Artery Disease" (Klausner VB: *Hospital Physician* 1999;35[7]:27-38). Future supplement research could show more accurate results if study protocols were more closely aligned with the actual in vivo biochemical mechanisms of electron transport by antioxidants.

For example, the vitamin E usually used in studies is α -tocopherol. However, the body uses many more of the E subtypes during free radical neutralization, therefore mixed tocopherols should be used. Also, various antioxidants work in concert, not one at a time. Supplementation with just one may actually increase oxidation in certain pathways. Likewise, a certain percentage of homocystinurics require supplemented methyl groups in addition to extra B₁₂, B₆, and folate because of dietary or genetic decreases in the methyl group supply.

Given the reality of what people actually eat as opposed to the ideal diet, supplementation with a variety of antioxidants is the safe and wise choice for prevention and treatment of coronary artery disease. Well designed studies will then doubtless show what has proved to be true in practice.

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In reply:

Dr. Schumacher raises several good points in his letter. As I mentioned in the article and as Dr. Schumacher alludes, acquiring vitamins and minerals from natural sources is always more beneficial. When a person eats a healthy diet including a wide variety of fruits, vegetables, and healthy oils, they not only ingest the

specific recommended nutrients, but also other nutrients that provide an increased or synergistic effect. For instance, bioflavonoids are an example of nutrients that act synergistically with vitamin C to increase vitamin C absorption and to preserve the structure and function of capillary endothelium. Other examples include proanthocyanidins from grapes and catechins from green tea, which are both potent antioxidants.

The added points that Dr. Schumacher makes are valid. First, the research on these other compounds is scarce, and second, many antioxidants act "in concert" with one another. For this reason, when supplementation is necessary, various combinations of supplements may be used to enhance the antioxidant effect (ie, vitamin E with mixed tocopherols). I am in full agreement that future research should be designed with a wider combination of nutrients to represent the in vivo mechanisms of antioxidation.

The last point that Dr. Schumacher makes is that certain people with elevated homocysteine caused by an error in the tetrahydrofolate cycle may require a methyl donor to increase the capacity of methylation. Specifically, in context of this biochemical error, homocysteine is unable to be converted to methionine, which is a precursor to an important methyl donor S-adenosylmethionine. Hence, homocysteine accumulates and S-adenosylmethionine is unavailable for methylation pathways. Methylation is an essential step for DNA transcription and gene expression. Clinically, this unavailability causes cytotoxicity resulting in vascular damage and neurodegeneration. The treatment for this metabolic disorder is supplementation with folic acid, vitamin B₆, vitamin B₁₂, and a methyl donor such as trimethylglycine.

I thank Dr. Schumacher very much for his insightful comments.

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