

USANA Technical Bulletin

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Vitamin E (d-alpha-Tocopherol)

Technical Background

- Vitamin E (unlike other vitamins) is not a cofactor in the function of specific enzyme systems. Rather, vitamin E is a potent antioxidant that protects cells and tissues from oxidative damage. D-alpha tocopherol is one of the eight natural forms of vitamin E,¹ and the one shown to have the greatest nutritional and biological value.^{2,3}
- Gamma tocopherol, the principal form of vitamin E in the U.S. diet, is also an effective antioxidant, and its activity compliments that of alpha tocopherol.⁴
- The antioxidant activity of vitamin E is far-reaching. Specific activities include suppression of free radical formation, suppression of oxidative chain reactions, repair of damaged cell constituents (particularly cell membranes), and the inhibition of lipid oxidation in the gut, blood stream, tissues, and cells. These actions may help protect against several important degenerative disorders, including Alzheimer's disease,⁵ some cancers,⁶ and heart disease.⁷ An important study involving 2,000 patients with heart disease found that vitamin E supplements reduced heart attacks by 75%.⁸ Two Harvard studies, involving a total of about 135,000 health professionals, found that those who took daily supplements of vitamin E had one-fourth to one-third less coronary risk than those who did not take the supplements.^{9,10}
- In other studies, vitamin E supplementation was shown to boost immune function, particularly in the elderly.^{11,12}

Sources and Recommended Intake

- Tocopherols occur in a limited number of foods. The best dietary sources include almonds, sunflower seeds, walnuts, wheat germ, and soybeans, as well as the oils produced from these materials. However, the amounts found in oils are low because most of the oils we consume have been processed and refined, and vitamin E has been removed. Furthermore, today's diet-conscious population tends to eliminate many foods that provide vitamin E.
- The Recommended Dietary Allowance (RDA) for vitamin E (in d-alpha-tocopherol form) is 15 mg/day for adults, 6-7 mg/day for children, and 11-15 mg/day for adolescents.¹³ Many nutritionists consider these levels to be exceedingly low. The RDA does not take into account factors that may increase a person's individual needs for antioxidant protection (e.g. diet, lifestyle, disease, exposure to pollutants). Sound clinical evidence supports the benefits of vitamin E supplementation at doses of 100 - 400 IU per day (for adults).
- Vitamin E supplements should be taken with meals to promote absorption.
- The succinate and acetate ester forms, which are both suitable for tableting, are more stable than the non-esterified tocopherol and equally bioavailable for humans.¹⁴

Abstracts

Stephens NG, Parsons A, Schofield PM, Kelly F, Cheeseman K, Mitchinson MJ. Randomised controlled trial of vitamin E in patients with coronary disease: Cambridge Heart Antioxidant Study (CHAOS). Lancet 1996 Mar 23;347(9004):781-6. Vitamin E (alpha-tocopherol) is thought to have a role in prevention of atherosclerosis, through inhibition of oxidation of low-density lipoprotein. Some epidemiological studies have shown an association between high dietary intake or high serum concentrations of alpha-tocopherol and lower rates of ischaemic heart disease. We tested the hypothesis that treatment with a high dose of alpha-tocopherol would reduce subsequent risk of myocardial infarction (MI) and cardiovascular death in patients with established ischaemic heart disease. **METHODS:** In this double-blind, placebo-controlled study with stratified randomisation, 2002 patients with angiographically proven coronary atherosclerosis were enrolled and followed up for a median of 510 days (range 3-981). 1035 patients were assigned alpha-tocopherol (capsules containing 800 IU daily for first 546 patients; 400 IU daily for remainder); 967 received identical placebo capsules. The primary endpoints were a combination of cardiovascular death and non-fatal MI as well as non-fatal MI alone. **FINDINGS:** Plasma alpha-tocopherol concentrations (measured in subsets of patients) rose in the actively treated group (from baseline mean 34.2 micromol/L to 51.1 micromol/L with 400 IU daily and 64.5 micromol/L with 800 IU daily) but did not change in the placebo group. Alpha-tocopherol treatment significantly reduced the risk of the primary trial endpoint of cardiovascular death and non-fatal MI (41 vs 64 events; relative risk 0.53 [95% CI 0.34-0.83]; p=0.005). The beneficial effects on this composite endpoint were due to a significant reduction in the risk of non-fatal MI (14 vs 41; 0.23 [0.11-0.47]; p=0.005); however, there was a non-significant excess of cardiovascular deaths in the alpha-tocopherol group (27 vs 23; 1.18 [0.62-2.27]; p=0.61). All-cause mortality was 36 of 1035 alpha-tocopherol-treated patients and 27 of 967 placebo recipients. **INTERPRETATION:** We conclude that in patients with angiographically proven symptomatic coronary atherosclerosis, alpha-tocopherol treatment substantially reduces the rate of non-fatal MI, with beneficial effects apparent after 1 year of treatment. The effect of alpha-tocopherol treatment on cardiovascular deaths requires further study.

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