

Vitamin E Improves Outcomes in Patients With Alzheimer's Disease

James P. Meschino, DC, MS

In a paper first presented at the 2008 American Academy of Neurology Annual Meeting in Chicago and subsequently published in the journal *Dementia and Geriatric Cognitive Disorders*^{1,2} researchers showed that Alzheimer's patients who supplemented their diet with 2,000 IU per day of vitamin E **had a 26 percent lower mortality rate**. As explained by lead researcher Dr Valory Pavlik, from the Baylor College of Medicine's Alzheimer's Disease and Memory Disorders Center, many previous studies have shown that vitamin E supplementation can slow the progression of Alzheimer's disease.

Vitamin E and Improved Survival Rates

The study by Pavlik, et al., was able to show additional features and benefits of vitamin E supplementation in Alzheimer's patients and answered some common concerns about the safety of using high dosages of vitamin E, and combining vitamin E with conventional drugs used to manage this condition. In their study of 847 Alzheimer's disease patients followed for 4.9 years, results showed the following:

- A 26 percent reduced mortality rate in patients administered 2,000 IU of vitamin E per day compared to patients not taking the vitamin E supplement.
- Combining vitamin E supplementation (2,000 IU per day) with standard drugs used to treat Alzheimer's disease (cholinesterase inhibitors) produced the best overall results with respect to longevity and disease progression. Thus, it appears to be safe to recommend vitamin E supplementation to Alzheimer's patients already taking a cholinesterase inhibitor drug such as Aricept (donepezil), Exelon (rivastigmine), Reminyl (galantamine) and Ebixa (memantine hydrochloride).
- Recent concerns about vitamin E supplementation increasing risk of cancer and heart disease were put to rest, as this study showed that individuals with Alzheimer's disease, who were in a high-risk age group for death from cardiovascular disease and cancer (average age 73.5 ± 8.6 years) showed a 26 percent lower mortality rate than Alzheimer's patients who did not take the 2,000 IU per day of vitamin E.
- Alzheimer's disease patients who used only vitamin E supplementation and did not take any Alzheimer's disease medication still showed a 26 percent lower mortality rate than patients not taking the vitamin E supplement, along with significant disease management outcomes.

The researchers concluded that overall, "[r]egimens that included vitamin E were associated with a 26% lower mortality rate. There was a suggestion that [taking] vitamin E plus a cholinesterase inhibitor was more beneficial than taking either agent alone."

The Alzheimer's Disease Cooperative Study

A landmark study in 2000 known as the [Alzheimer's Disease Cooperative Study \(ADCS\)](#) was one of the first published studies to show that providing Alzheimer's patients with 2,000 IU per day of vitamin E could slow the progression of their disease. The ADCS was formed in 1991 as a cooperative agreement between the National Institute on Aging and the University of California, San Diego. The ADCS is a major initiative for Alzheimer's disease (AD) clinical studies in the federal government, addressing treatments for both cognitive and behavioral symptoms. Reporting in the *American Journal of Clinical Nutrition*, researchers published results showing that vitamin E supplementation (2,000 IU per day) may slow functional deterioration leading to nursing-home placement.

Why Vitamin E Supplementation?

The brains of Alzheimer's disease patients frequently shows generalized atrophy, neuritic plaques (dystrophic axons and dendrites surrounding an amyloid core), and neurofibrillary tangles. Evidence suggests that oxidative stress (free radicals) may lead to permanent cellular damage in the brain that triggers some of the changes seen in the brain of Alzheimer's patients. The presence of excessive ss-amyloid protein formation (an extracellular insoluble protein) is a hallmark feature of the Alzheimer's brain.

A proposed mechanism of ss-amyloid toxicity is that it induces free radicals, which disrupt cellular lipid, protein and DNA. In addition to ss-amyloid, several other processes may also induce oxidative stress in AD. Activated microglial cells found in association with neuritic plaques may release cytokines, pro-oxidants, and free radicals. As well, other etiological factors such as cytoskeletal destabilization, energy deprivation or toxic inflammatory responses may all converge in a common final pathway involving free radicals.

Prior to the Alzheimer's Disease Cooperative Study, earlier clinical trials and epidemiologic studies had suggested that several agents may help prevent the development of AD or slow further deterioration. These agents include not only vitamin E, but also selegiline, estrogen, and anti-inflammatory drugs. One property they all share is the ability to protect against free radical-mediated damage, either directly or indirectly.

The recent study by Pavlik, et al., adds to the body of evidence indicating that supplementing Alzheimer's disease patients with 2,000 IU per day of natural vitamin E, can slow the progression of their disease and lower the mortality rate. Unfortunately, many medical doctors have not been exposed to these studies and thus, vitamin E supplementation is often left out of the management of many Alzheimer's disease cases. In these instances it is up to complementary health care professionals to provide patients and family members with the research and recommendations that have been shown to be meaningful, in regards to vitamin E supplementation (as well as other effective natural interventions). Research suggests that vitamin E supplementation, at a dosage of 2,000 IU per day, is effective, safe, enhances the benefits of conventional Alzheimer's drugs, and does not increase the risk of cancer or heart disease in this older and elderly population.

References

1. Pavlik V, Doody R, Rountree S, Darby E. [Vitamin E use is associated with improved survival in an AD cohort.](#) 2008 American Academy of Neurology Annual Meeting.(Chicago, IL) Poster Sessions III: Aging and Dementia: Clinical III.; # P03.076. Published in 2009 in *Dementia and Geriatric Cognitive Disorders*;28(6):536-40.
2. Grundman M. [Vitamin E and Alzheimer disease: the basis for additional clinical trials.](#) *American Journal of Clinical Nutrition*, 2000;71(2):630S-636s.

