

New Evidence That Vitamin E Supplementation Improves Outcomes In Alzheimer's Patients:

A 2008 study adds to the body of evidence

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In a paper presented at 2008 American Academy of Neurology Annual Meeting.(Chicago, IL, Poster Sessions III: Aging) researchers showed that Alzheimer's disease patients who supplemented their diet with 2000 IU per day of Vitamin E had a 26% lower mortality rate. As explained by the lead researcher, Dr Valory Pavlik from the Baylor College of Medicine's (Alzheimer's Disease and Memory Disorders Center, Houston, TX), many previous studies have shown that Vitamin E supplementation can slow the progression of Alzheimer's disease. The study by Pavlik et, was able to show additional features and benefits of Vitamin E supplementation in Alzheimer's disease 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, who were followed for 4.9 years, their results showed the following:

- A. A 26% reduced mortality rate in the patients administered 2000 IU of Vitamin E per day compared to Alzheimer's disease patients not taking the Vitamin E supplement.
- B. Combining Vitamin E supplementation (2000 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)
- C. 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' 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% lower mortality rate than Alzheimer's patients who did not take the 2000 IU per day of Vitamin E.
- D. In the Alzheimer's disease patients who used only Vitamin E supplementation (2000 IU per day) and did not take any Alzheimer's disease medication, these patients still showed a 26% lower mortality rate than patients not taking the Vitamin E supplement, along with significant disease management outcomes.

Overall the researchers concluded, "Regimens that included vitamin E were associated with a 26% lower mortality rate. There was a suggestion that of vitamin E plus a cholinesterase inhibitor was more beneficial than taking either agent alone".

The Alzheimer's Disease Cooperative Study

A landmark study in the year 2000 known as the Alzheimer's Disease Cooperative Study was one of the first published studies to show that providing Alzheimer's patients with 2000 IU per day of Vitamin E could slow the progression of their disease. The Alzheimer's Disease Cooperative Study (ADCS) was formed in 1991 as a cooperative agreement between the National Institute on Aging (NIA) 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 (2000 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. 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 β -amyloid protein formation (an extracellular insoluble protein) is a hallmark feature of the Alzheimer's brain. A proposed mechanism of β -amyloid toxicity is that it induces free radicals, which disrupt cellular lipid, protein, and DNA. In addition to β -amyloid, several other processes may also induce oxidative stress in AD. Activated microglial cells found in association with neuritic plaques may release cytokines, prooxidants, and free radicals.

As well, other etiological factor such as cytoskeletal destabilization, energy deprivation, or toxic inflammatory responses may all converge in a common final pathway involving free radicals, as well.

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 vitamin E, selegiline, estrogen, and anti-inflammatory drugs. One property that they all share is the ability to protect against free radical-mediated damage, either directly or indirectly.

Conclusion

The recent study (2008) by Pavlik et al adds to the body of evidence indicating that supplementing Alzheimer's disease patients with 2000 IU per day of natural Vitamin E, can slow the progression of their disease and lower the mortality rate by 26% (over a five-year period). 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). The study by Pavlik et al suggests that Vitamin E supplementation, at a dosage of 2000 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:

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2. Grundman M. Vitamin E and Alzheimer disease: the basis for additional clinical trials. American Journal of Clinical Nutrition, Vol. 71, No. 2, 630S-636s. 2000.