Vitamin B supplementation did not slow cognitive decline in patients with Alzheimer disease

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High-dose vitamin B supplementation for patients with mild to moderate Alzheimer disease did not slow the rate of cognitive decline, according to a study in the October 15 issue of *JAMA*.

Evidence of homocysteine (an amino acid produced by the body) elevation in Alzheimer disease (AD) and the involvement of homocysteine in neuropathological mechanisms suggest that reduction of homocysteine may offer an approach to altering the disease. B vitamins that influence homocysteine metabolism have been considered as a therapeutic option to reduce risk of AD or slow its progression, according to background information in the article. According to the authors, prior studies of B vitamins to reduce homocysteine in AD have not had sufficient size or duration to assess their effect on cognitive decline.

Paul S. Aisen, M.D., of the University of California, San Diego, and colleagues conducted a clinical trial to determine if reduction of homocysteine levels with high-dose supplementation with folic acid and vitamins B6 and B12 for 18 months would slow the rate of cognitive decline in 409 individuals with mild to moderate AD. Participants were randomly assigned to two groups of unequal size to increase enrollment (60 percent treated with high-dose supplements [5 mg/d of folate, 25 mg/d of vitamin B6, 1 mg/d of vitamin B12] and 40 percent treated with identical placebo). A total of 340 participants (202 in active treatment

group and 138 in placebo group) completed the trial while taking study medication. Cognitive abilities were measured via testing with the Alzheimer Disease Assessment Scale (ADAS-cog).

The researchers found that even though the vitamin supplement regimen was effective in reducing homocysteine levels, it had no beneficial effect on the primary cognitive measure: the rate of change in ADAS-cog score did not differ significantly between treatment groups. The authors did find that symptoms of depression were more common in the high-dose supplement group.

"Many studies suggest that relative elevation of homocysteine is characteristic of AD, and laboratory research implicates homocysteine in neurodegenerative mechanisms. High-dose B vitamin supplementation in individuals with normal levels of B vitamins was effective in reducing homocysteine levels. However, our study does not support the treatment of individuals with mild to moderate AD and normal vitamin levels with B vitamin supplements," the authors conclude.

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