

DHA 'fish oil' supplements do not seem to slow cognitive, functional decline in Alzheimer's disease

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Patients with mild to moderate Alzheimer's disease (AD) who received supplementation with the omega-3 fatty acid docosahexaenoic acid (DHA), believed to possibly reduce the risk of AD, did not experience a reduction in the rate of cognitive and functional decline, compared to patients who received placebo, according to a study in the November 3 issue of *JAMA*, a theme issue on aging.

Joseph F. Quinn, M.D., of Oregon Health and Science University and the Portland VA Medical Center, Portland, Ore., presented the findings of the study at a *JAMA* media briefing at the National Press Club.

"Several studies have found that consumption of fish, the primary dietary source of omega-3 fatty acids, is associated with a reduced risk of <u>cognitive decline</u> or dementia. Some studies have found that consumption of DHA, but not other <u>omega-3 fatty acids</u>, is associated with a reduced risk of Alzheimer disease," the authors write. However, those studies were observational and did not control who received DHA. Animal studies that used DHA showed reductions in Alzheimer-like brain pathology.

Dr. Quinn and colleagues conducted a randomized, controlled trial to examine whether DHA supplementation would slow the rate of cognitive and functional decline in individuals with Alzheimer's disease. The study, which was conducted between November 2007 and May 2009 at



51 U.S. clinical research sites, included 402 individuals with mild to moderate Alzheimer's disease. Participants were randomly assigned to DHA at a dose of 2 grams/day or to identical placebo (60 percent were assigned to DHA and 40 percent were assigned to placebo). Duration of treatment was 18 months. Changes in cognitive and functional abilities were assessed with the Alzheimer's Disease Assessment Scale

(ADAS-cog) and the Clinical Dementia Rating (CDR) sum of boxes. Rate of <u>brain atrophy</u> was also determined by volumetric <u>magnetic</u> <u>resonance imaging</u> (MRI) in a subsample of participants.

A total of 295 participants completed the trial while taking study medication (DHA: 171; placebo: 124). The researchers found that supplementation with DHA had no beneficial effect on rate of change on ADAS-cog score, with the rate of average change in the score over 18 months being 8.27 points for the placebo group and 7.98 points for the DHA group. The rate of points change on CDR sum of boxes over 18 months was 2.93 for the placebo group compared with 2.87 for the DHA group.

Among the individuals participating in the MRI substudy (102 had MRIs at the beginning of the study and at 18 months [DHA group: 53; placebo group: 49]), an analysis showed no effect of DHA treatment on total brain volume change during 18 months.

"In summary, these results indicate that DHA supplementation is not useful for the population of individuals with mild to moderate Alzheimer disease," the authors write.

The researchers add that "because part of the rationale for the trial was epidemiological evidence that DHA use before disease onset modifies the risk of Alzheimer disease, it remains possible that an intervention with DHA might be more effective if initiated earlier in the course of



the disease in patients who do not have overt dementia."

More information: JAMA. 2010;304[17]:1903-1911.

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