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Beneficial effects of a diet with walnuts in Alzheimer's disease**Abha Chauhan and Ved Chauhan**

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Amyloid beta-protein (A β) is the major protein of amyloid deposits in the brain of patients with Alzheimer's Disease (AD). Extensive evidence suggests neurotoxic effects of A β and the role of oxidative stress and inflammation in AD. Walnuts are rich in components that have antioxidant and anti-inflammatory properties. Previous *in vitro* studies have shown that walnut extract inhibits A β fibrillization, solubilizes its fibrils, and has protective effects against A β -induced oxidative stress and cell death in PC12 cells. In the Tg2576 transgenic mouse model of AD (AD-tg), we have reported the beneficial effects of dietary supplementation of 6% (T6) or 9% walnuts (T9) [equivalent to 1 or 1.5 oz of walnuts per day in human] on the memory, learning skills anxiety and motor coordination when compared to AD-tg mice on diet without walnuts (T0). The diets for the experimental and control mice were comparable as regards to total calories and the contents of protein, carbohydrate and fat. To understand the mechanism of beneficial effects of diet with walnuts in AD, we have recently studied the effects of walnuts on A β levels and oxidative stress markers in AD mice. In AD-tg mice on diet with walnuts (T6, T9), the levels of soluble A β were lower in the brain and higher in the blood when compared to T0 mice, suggesting that walnuts in the diet can increase the clearance of A β from brain to the blood. We also observed significant decrease in free radical levels and oxidative damage (lipid peroxidation, protein oxidation) coupled with increased antioxidant status (superoxide dismutase, catalase and glutathione peroxidase) in these T6 and T9 mice on diet with walnuts. In conclusion, these studies suggest that diet with walnuts may have beneficial effects in reducing the risk, delaying the onset, or slowing the progression of AD because walnuts can help to improve memory and learning skills, inhibit A β fibrillization and maintain A β in the soluble form, decrease A β -induced oxidative stress and A β -mediated cytotoxicity and reduce the levels of A β in the brain and increase A β clearance.

Biography

Abha Chauhan is the head of Developmental Neuroscience Laboratory (IBR) at New York. She is also an Adjunct Professor of the Neuroscience doctoral program at the graduate Center of the City University of New York. She has received her MS and PhD from postgraduate Institute of Medical Education and Research, India. From 1983-1984, she has worked as a Research Associate at the Mount Sinai School of Medicine, New York. Then she joined IBR, where she has over 90 publications in the fields of Alzheimer's disease. She has been awarded several research grants as a Principal Investigator and has served as the Editor of the book entitled *Autism: Oxidative stress, Inflammation and Immune Abnormalities*.

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