Dementia Conference 2018: Beneficial effects of a diet with walnuts in Alzheimers disease

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Amyloid beta-protein (AB) is that the major protein of amyloid deposits within the brain of patients with Alzheimer's disease (AD). Extensive evidence suggests neurotoxic effects of AB and also 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 Aß fibrillization, solubilizes its fibrils, and has protective effects against Aβ-induced oxidative stress and necrobiosis in PC12 cells. within the Tg2576 transgenic mouse model of AD (AD-tg), we've 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 put next 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 therefore the contents of protein, carbohydrate and fat. To understand the mechanism of beneficial effects of diet with walnuts in AD, we've recently studied the consequences of walnuts on Aβ levels and oxidative stress markers in AD mice. In AD-tg mice on diet with walnuts (T6, T9), the degree of soluble Aβ were lower within the brain and better within the blood in comparison to T0 mice, suggesting that walnuts within the diet can increase the clearance of $A\beta$ from brain to the blood. We also observed significant decrease in atom levels and oxidative damage (lipid peroxidation, protein oxidation) as well as increased antioxidant status (superoxide dismutase, catalase and glutathione peroxidase) in these T6 and T9 mice on diet with walnuts. last, these studies suggest that diet with walnuts may have beneficial effects in reducing the chance, delaying the onset, or slowing the progression of AD because walnuts can help to boost memory and learning skills, inhibit AB fibrillization and maintain ABB within the soluble form, decrease ABinduced oxidative stress and Aß-mediated cytotoxicity and reduce the degree of $A\beta$ within the brain and increase $A\beta$ clearance.

Dementia is defined by age-related progressive impairment of cognitive function in several domains (memory, learning, judgment, orientation, language, and comprehension), thereby affecting the daily activities of life and social event in elderly people. The prevalence of dementia has been increasing over the years thanks to a rise within the aging population. In 2015, 47 million people were tormented by dementia worldwide, and 131 million people are estimated to own dementia by 2050. Dementia occurs mainly in people older than 65 years old when comorbidity is additionally a standard occurrence. it's generally preceded by mild cognitive impairment (MCI). this estimate is that 33% of elderly people die with dementia. The annual cost of dementia care globally is roughly \$818 billion.

Alzheimer's disease (AD) may be a devastating neurodegenerative disorder that gently ends up in state of mind and decline of cognitive functions over a fundamental quantity of 5–20 years. Although there are many sorts of dementia, AD ranks because the commonest reason behind dementia among elderly people, and it accounts for over 60% of dementia cases. Vascular dementia is that the second most typical dementia,

followed by dementia with Lewy bodies. AD affects about 25 million people worldwide, including 5.5 million people within the u. s.. a complete of 10% of the population over 65 years old, and nearly 50% of individuals over 85 years old, suffer from AD. The neuropathological hallmarks of AD include neuronal loss and progressive accumulation of fibrillar amyloid beta-protein (A β) as amyloid plaques, and of paired helical filaments as neurofibrillary tangles within the brain. In AD, the key amyloid protein is amyloid beta-protein (A β) of 40 or 42 amino acids, which exists as soluble or fibrillar A β . Neuro-pathological changes in AD occur slowly over decades before the clinical symptoms of dementia are diagnosed.

MCI is taken into account an intermediate state between healthy aging and early dementia. The prevalence of MCI is 10%–20% of individuals 65 years old or older. Although these people can perform daily activities, they're considered at higher risk of developing dementia within 3–10 years. Therefore, now period provides a possible targeted intervention window to cut back the chance, prevent, or delay the onset and progression of cognitive impairment and dementia. MCI is classed as either amnestic MCI (with impaired memory) or non-amnestic MCI (no effect on memory). About 50% of individuals with amnestic MCI develop dementia in three years.

About 35% of dementia cases are attributed to modifiable risk factors, which include vascular (cardiovascular disease, hypertension, stroke), metabolic (diabetes, midlife obesity), head trauma, depression, and lifestyle factors (diet quality, alcoholism abuse, sleep deprivation). it's estimated that 33% of dementia cases may be delayed or prevented through better management and targeted intervention of those risk factors, particularly hypertension, depression, diabetes, and obesity. The association between diet and health is becoming increasingly clear, with extensive evidence that plant foods rich in flavonoids and phenols are efficient as defensive antioxidants, thus reducing oxidative stress, which is understood to contribute to the pathophysiology of the many diseases, including neurological disorders, disorder (CVD), hypertension, and diabetes. In the following sections, we review evidence that early intervention with a walnut-enriched diet can reduce the danger and/or delay the onset or slow the progression of cognitive decline and dementia due to (a) the elevated oxidative stress and inflammation involved within the aging process and dementia and (b) the antioxidant and antiinflammatory components of walnuts.

Foot Note: This work is partly presented at 13th Annual Conference on Dementia and Alzheimers Disease December 13-15, 2018 at Abu Dhabi, UAE

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