ARC Journal of Neuroscience
Volume 8, Issue 1 2025, PP 6-16
ISSN 2456-057X
https://doi.org/10.20431/2456-057X.0801002
www.arcjournals.org

Management of Alzheimer's Disease Through Nutrition

Ashok Chakraborty[1*], Smita Guha[2]

[1]*Sacred Heart University, Department of Chemistry, Fairfield, CT, USA*

[2]*St. John's University, Queens, NY, USA*

Received: 04 January 2025 Accepted: 20 January 2025 Published: 13 March 2025

Corresponding Author:** ***Ashok Chakraborty*, Sacred Heart University, Department of Chemistry, Fairfield, CT, USA*

Abstract

Alzheimer's disease and its related health issues are still incurable. It starts generally at age 50 or above and without having any sex bar. Alzheimer's symptoms mainly start from forgetfulness and ultimately end up with dementia, an irreversible complete loss of memory. There is no therapy available though scientists are trying to get some clue for gene and cell therapy, but effective solutions are yet to be obtained. In this scenario, management of the disease through diets, and the pros and cons of the right food choice for the benefit of AD patients have been discussed here. The Mediterranean diet along with DASH diet, in short, known as a MIND diet, are effective for maintaining the better cognition effect and quality of life among AD individuals.

Keywords: *Dementia, Alzheimer's Disease, Neuro-degeneration, Cognition, Ageing, Macro- and Micronutrients*

Abbreviations

AD - Alzheimer's Disease, DASH - Dietary Approaches to Stop Hypertension, MIND - Mediterranean-DASH Diet, Aβ - Amyloid-beta peptide, APOE ε4 - Apolipoprotein E gene, MUFAs - Monounsaturated Fatty Acids, BMI - Body mass index, TFA - Trans-fatty acid, T2DM - Type 2 diabetes mellitus, EGCG - Epigallocatechin-3-gallate

1. Introduction

Alzheimer's disease (AD) is described as an irreversible decline in cognitive and functional abilities, loss of memory and ultimately results dementia, and premature death [1]. This disease generally starts at ages around 50 or above, but early onset of the ailments has also been noted in familial cases indicating some genes are involved along with other sporadic causes [2]. The number of AD cases is gradually growing by number every year, and therefore becomes a major socioeconomic issue [3]. Although the cause of AD is not well-understood, but the deposition of accumulated amyloid-beta peptide (Aβ) and the formation of neurofibrillary *tau* protein tangles in the brain have been documented **[4].** As a cause of AD while nothing is clear at this time, epsilon 4 allele of the apolipoprotein E gene (APOE ε4), diabetes, and smoking are found to be associated with the onset of AD. However, cognitive engagement and physical activities can decrease the risk of AD, as noted [5]. Currently, there is no effective cure of the AD symptoms, however, there is a good correlation between nutrition and the biochemistry of AD as observed during studies with animal models [6-15]. These indicate that may be the appropriate nutritional intervention, a good approach for reducing the risk of AD onset and its progression.

2. AD is Associated with Both Obesity and Malnutrition

Both, obesity and malnutrition are associated with AD [16-18]. Obesity (BMI > 30) in midlife while found to increase the risk of AD, the late-life obesity is associated with the reduced risk of AD [16]. Therefore, manipulation of adiposity could be a guideline to prevent AD [19-23].

3. The Effects of Macro-Nutrients on the Risk of AD

3.1. Fats and Fatty acids

Monounsaturated Fatty Acids (MUFAs) and its derivatives have anti-inflammatory effects *in*

vivo [24]. Higher monounsaturated fatty acid, but not the higher saturated fatty acid, can improve the cognitive function [25]. Total fat, polyunsaturated fatty acid, trans-fat intakes are not found to be associated with cognition changes [26].

Derivatives of MUFA, like phenols, have antioxidant effects, therefore intake of which have suggested to be associated with less cognitive decline [27, 28].

3.1.1. Polyunsaturated Fatty Acids (Omega-3 Polyunsaturated Fatty Acids

These fatty acids have been demonstrated to reduce the Aβ production and reverse the pathological symptoms in AD animal models [28-32]. This omega-3 fatty acids can also delay the cognitive aging and memory decline but only in the mild cases of the disease [33-35].

3.1.2. Saturated Fatty Acids

Moderate intake of saturated fatty acids while exert an increased risk of AD and dementia, especially to APOE epslion4 carriers, a higher intake do not show any such risk. This could be an indication of threshold association of saturated fatty acids with AD [36, 37].

3.1.3. Trans-Fatty Acid (TFA)

TFA might potentially increase the AD risk by producing more Aβ by disturbing the balance of amyloidogenic and non-amyloidogenic processing of amyloid precursor protein [38]. However, in other clinical study no relation was found between greater intake of trans-fat with cognitive decline [38].

3.2. Effects of Carbohydrates

Type 2 diabetes mellitus (T2DM) is a vital risk of getting AD [5]. In the brain of AD patient the reduced level of insulin and its receptor expression have been reported [39-42]. Therefore, a diet high in carbohydrates may cause glycation of multiple neural proteins and develops an AD symptoms [43-45]. However, reliable data from clinical trials with such a diet and AD developments are yet to obtain.

3.3. Effects of Proteins

Meat: Processed meat, especially the red meat is considered as a vital risk factor for developing dementia.

Fish contains long chain omega-3 fatty acids, EPA, and DHA and can reduce the risk of having AD and dementia, especially among them who are the non-carriers of the APOE epslion4 **[46-50].** In a study with more than 800 participants aged from 65 to 94 years, it was found that number AD occurrence is less (~60%) in people who consume fish quite often in a week compared with those who rarely or never ate fish [49].

Eggs: In one study it was found that eggs showed a positive effect on neurocognitive function [51].

4. Effects Micronutrients (Vitamins, and Trace Metals) on AD

- ***Vitamin A and β-carotene*** reduces the deposition of accumulated amyloid β-peptide (Aβ) and fibrils [52-54].
- ***Vitamins B:*** *(Especially the Folic Acid, Vitamin B6, and Vitamin B12):* These vitamins inhibit the oxidative stress and lower the concentrations of homocysteine, and thus helps AD [55-58].
- ***Vitamin C***, as noted both in vitro and in vivo studies, can reduce the Aβ oligomer formation and oxidative stress, and thus expected to help AD symptoms [59, 60].
- ***Vitamin D****:* Vitamin D deficiency (<10 ng/ml) is significantly associated with the development of AD and dementia with time. Consumption of which for at least 12 months can significantly improve the Aβ peptide-related biomarkers and cognitive function [61].
- ***Vitamin E:*** It is a lipid-soluble antioxidant, and can scavenge Aβ-associated free radicals [62-65], and results a slower rate of cognitive decline [66]. The mechanism is due to the activation of Sirt-1 and concomitant inhibition of COX-2, 5-lipoxygenase and NFkβ mediated proinflammatory pathways [67].
- **Metals and Metalloids:** Transition metals are believed to play a role in the pathogenesis of AD by forming reactive species through metal amyloid complexes [68, 69].

Copper: Copper is an essential trace metal needed to maintain our body toxic free. However, high dietary intake of copper in conjunction with saturated and trans-fats can cause a cognitive decline [70, 71]. A meta-analysis of 17 studies with 1425 subjects showed higher levels of serum copper in AD patients than controls [72, 73]. However, a randomized, placebo-controlled trial showed no such progression of AD after oral copper supplementation to 68 subjects [74].

Iron: Iron can cause an oxidative stress, and an imbalance in iron homeostasis is thought to be a

precursor of AD [75, 76]. Diets excessive in Fe together with a high intake of saturated fat acids are not recommended to elderly subjects [70]. However, iron supplementation has been reported to improve attention and concentration among children and adults, as well [77].

Zinc: Zinc deficiency was reported to be associated with cognition loss in AD patients. Supplementation of Zinc was found to reduce both the Aβ and *tau* pathologies in AD Ok model [78, 79].

Selenium is a metalloid and play an important role in the antioxidative defense [80, 81]. AD patients showed a significant lower Selenium level in plasma, erythrocytes, and nails when compared to controls [82]. Supplementations of selenium-containing mixtures can improve cognition [83-85]. However, the relationship between Se supplementation and AD recovery needs further randomized trials.

5. The Effects of other kinds of Foods on AD

Fruits and Vegetables: Fruits and vegetables contain a substantial amount of antioxidants and bioactive compounds like, vitamin E, vitamin C, carotenoids, and flavonoids, and also low in saturated fats, which can decrease the risk of AD and dementia **[86-88].**

Vegetable (only): Vegetables, especially green leafy vegetables, contain more vitamin E than fruits. Therefore, higher vegetable, but not fruit, consumption can slower the rate of cognitive decline as found in a cohort study with 3,718 participants aged 65 years and older [88]. The similar effect was also found with the intake of nuts, cabbage, and root vegetables to diminish age-related cognitive decline in middle-aged individuals [89].

Dairy products: Dairy products are rich in vitamin D, phosphorus, and magnesium, therefore they may reduce the vascular alterations and structural changes in the brain and ultimately reduces the possibilities of having the cognitive decline [90, 91]. However, saturated fat intake from milk products was found to be associated with an increased risk of AD [37, 92]. Unfortunately, no evidences are available yet on the effects of dairy products on AD from a randomized controlled trials.

Drinks:

Caffein: Drinking coffee has been reported to have some protective effect from AD ailments. Coffee being the best source of caffeine it can selectively enhance plasma cytokines and can decrease the risk of AD by 95% as reported **[93,** 94]. However, further prospective studies with proper methodologies are needed to evaluate the benefit of coffee consumption from AD-risk are strongly needed.

Tea: Some observational studies suggest that drinking tea was associated with lower risks of cognitive decline [95, 96]. While black tea was shown to significantly enhance auditory and visual attention compared [97], green tea polyphenols inhibit oxidative stress and protects the individual from having cognitive impairment [98-100]. Further, green tea contains epigallocatechin-3-gallate (EGCG), which has been shown to reduce the β-amyloid deposition and *tau* plaque formation in AD mouse models [101, 102]. The neuro-protective effects of tea consumption could be due to the presence of catechins, L-theanine, polyphenols, and other compounds in tea leaves [103].

Alcohol: Epidemiological studies suggest that light-to-moderate alcohol drinking can reduce the risk of having AD, particularly to them who are the non-carriers of APOE epslion4 **[104-106]**. However, heavy drinking (>2 drinks) along with heavy smoking can cause an early onset of AD especially to those individuals who carry APOE epsilon4 gene **[107]**. However, the mechanisms of the above observations are still unclear. Further, different types of alcohol (wine, beer, and mixed alcohol beverages) may have different effects on AD. Resveratrol and other polyphenols in red wine have been reported to diminish Aβ plaque formation, while beer consumption afford a biofactor, silicon, which can provide benefits to AD **[108-110]**.

6. The Effects of Different Dietary Patterns on AD (either to follow or to restrict)

Dietary pattern, a combination of food components that summarizes an overall diet for a study population, can have various effects on cognitive function and AD (Table 1)

Table 1.

Dietary Patterns	Good or Bad for AD
Western diet	• A Western diet is characterized by higher intake of red and processed meats, refined grains, sweets, and desserts **[111, 112]** • A high-fat Western diet may contribute to the development of AD by impacting Aβ deposition and oxidative stress **[113, 114]**

Japanese Diet.	• Japanese diet mainly contains fish, plant foods (soybean products, seaweeds, vegetables, and fruits), decreased intake of refined carbohydrates and animal fats **[115]** • In a population-based study with a total of >1000 Japanese subjects followed by 15 years, those dietary composition was found to be associated with a reduced risk of AD **[116]**
Healthy Diets	• A healthy diet means consumption of fruits, whole grains, fresh dairy products, vegetables, breakfast cereal, tea, vegetable fat, nuts, and fish but less or none of the followings like, meat, poultry, refined grains, animal fat, and processed meat **[117]** • Participants who follow the healthy diet had a better cognitive performance and a decreased risk of AD **[118, 119]**,
DASH-Style Diets	• The DASH diet (Dietary Approaches to Stop Hypertension) contains plant foods, whole grains, nuts, fruits, vegetables, fish, poultry, and low-fat dairy products **[120]**. • Hypertension is often associated with increased risk for AD **[121]**. A randomized clinical trial with 124 participants who have elevated blood pressure and AD issues became the subjects of the DASH diet and exhibited greater neurocognitive improvements compared to normal subjects along with the controlled blood pressure level **[122]**
Mediterranean Diets (MD)	• The Mediterranean diet is composed of fruits, vegetables, cereals, bread, potatoes, poultry, beans, nuts, olive oil, and fish; a moderate consumption of alcohol; and a lower consumption of red meat and dairy products **[123]** • A meta-analysis with 2,190,627 subjects showed that adherence to the Mediterranean diet can reduce the risk of having AD and also the mortality in AD **[124-126]**
MIND Diet	• The MIND Diet *(Mediterranean-DASH Intervention for Neurodegenerative Delay Diet)* takes the good elements of *Mediterranean Diets (MD)* and DASH diet while minimizing the intake of red meats, butter, margarine, cheese, fast foods, and sweets **[127]** • This diet has been shown to slow down in cognitive decline, and offers a better protection against Alzheimer's disease compared to DASH or MD diets when used separately **[128]**

7. DISCUSSION

Extensive literature studies relating to Alzheimer's diseases and its possible cure when finds no hope, its management through nutrition and choice of correct diet can offer a quality life with better cognition effect. Consumption of refined carbohydrates or a diet with a high glycemic index is associated with increased accumulation of Aβ peptides in the brain. This effect is even worse in APOE-+4 carriers, which is a genetic risk factor associated with AD and dementia, as well as insulin resistance. However, the exact mechanisms underlying this relationship are still unknown **[**129, 130].

In various randomized trials with standardized diagnostic criteria for AD and also epidemiological studies suggest that antioxidants, vitamins, polyphenols, polyunsaturated fatty acids, fish, fruits, vegetables, tea, and light-to moderate consumption of alcohol are beneficial for AD. On the other hand, carbohydrates, fats and whole-fat dairy products are detrimental to AD. However, the effect of all the food items individually is hard to assess as they are correlated with each other. Further, randomized trials are not always feasible, but when undertaken it showed that the dietary supplementation of vitamin E, B, and polyunsaturated fatty acids does not reduce cognitive decline and the risk of AD. This could be due to the fact that those nutrients may protect the disease but once started cannot cure them. Further, inadequate time of the study and complexes from several social and behavioral factors across the life course do not show the expected benefits [131].

A Western diet pattern can increase the inflammation levels hence the risk of getting AD [132], whereas the Mediterranean diet has been shown to improve cognitive outcomes, and decrease memory decline [133-135]. A ketogenic diet can reduce the oxidative stress, inflammation and also reduce the negative effects from glucose metabolism in the brain. Therefore such a diet may also be useful in the management of AD [13]. Since in AD patients a high level of oxidative stress has been noticed, adequate intake

of fruits, vegetables, fish, nuts, legumes, cereal and lower intake of meats, high fat diary, sodium, sweets, and refined grains are recommended. Further, vitamin supplements, like Vitamin D, Vitamin B12, Vitamin E, and Vitamin C can help to combat cognitive and memory decline **[136-144]**. However, further research is needed to improve the quality of evidence relating to the association of many nutrients, foods, and dietary patterns with AD.

8. Conclusion

From the above study it appears that some foods are good for brain health and some are not. Especially, red meat, butter, margarine, cheese, fast foods, sweets, and high-sodium foods are better to avoid, and instead it is good to consume: whole grains, fresh fruits, vegetables, fish, beans, nuts and olive oil for better cognition effect and to avoid any risk of having AD in the life course.

9. Acknowledgment

We acknowledge all our staff members, scientists from Sacred heart University (CT) and St. John's University (NY) for their support during the writing of this review by providing materials and with the editing part.

References

[1] Blennow K, de Leon KM, and Zetterberg H. "Alzheimer's disease," *The Lancet,* 2006; vol. 368 (9533): 387–403.

[2] Jicha GA and Carr SA. Conceptual evolution in Alzheimer's disease: implications for understanding the clinical phenotype of progressive neurodegenerative disease. *Journal of Alzheimer's Disease,* 2010; 19(1): 253–272,

[3] Brookmeyer R, Johnson E, Ziegler-Graham K, and Arrighi HM. Forecasting the global burden of Alzheimer's disease. *Alzheimer's and Dementia,* 2007; 3(3): 186–191.

[4] Jiang T, Yu JT, and Tan L. Novel disease-modifying therapies for Alzheimer's disease. *Journal of Alzheimer's Disease,* 2012; 31(3): 475–492.

[5] Williams JW, Plassman BL, Burke J, and Benjamin S. Preventing Alzheimer's disease and cognitive decline. *Evidence Report/Technology Assessment,* 2010; 193: 1–727.

[6] Dosunmu R, Wu J, Basha MR, and Zawia NH. Environmental and dietary risk factors in Alzheimer's disease. *Expert Review of Neurotherapeutics*, 2007; 7(7): 887–900.

[7] Lau FC, Shukitt-Hale B, and Joseph JA. Nutritional intervention in brain aging: reducing the effects of inflammation and oxidative stress. *Sub-cellular Biochemistry*, 2007; 42: 299–318.

[8] Shah R. The role of nutrition and diet in Alzheimer disease: a systematic review. *Journal of the American Medical Directors Association*, 2013.

[9] Agahi A, Hamidi G, Daneshvar R, Hamdieh M, Soheili M, Alinaghipour A, et al. Does severity of Alzheimer's disease contribute to its responsivenessto modifying gut microbiota? A double blind clinical trial. *Front. Neurol.* 2018; 9: 662. doi:10.3389/fneur.2018.00662

[10] Samadi M, Moradi S, Moradinazar M, Mostafai R, and Pasdar Y. Dietary pattern in relation to the risk of Alzheimer's disease: a systematic review. *Neurol. Sci.* 2019; 40: 2031–2043. doi: 10.1007/s10072-019-03976-3

[11] Goncalves Tosatti J, da Silva Fontes A, Caramelli P, and Gomes K. Effects of resveratrol supplementation on the cognitive function of patients with Alzheimer's disease: a systematic review of randomized controlled trials. *Drugs Aging.* 2022; 39: 285–295. doi: 10.1007/s40266-022-00923-4

[12] Yilmaz R, and Arica Polat B. Vitamin D is associated with cognitive status in patients with Alzheimer's disease. *Turk Geriatr Derg.* 2019; 22: 361–367.

[13] Simsek H, and Uçar A. Is ketogenic diet therapy a remedy for Alzheimer's disease or mild cognitive impairments? a narrative review of randomized controlled trials. *Adv. Gerontol.* 2022; 12: 200–208.

[14] Shrestha L, Shrestha B, Gautam K, Khadka S, and Mahara Rawal N. Plasma vitamin B-12 levels and risk of Alzheimer's disease: a case-control study. *Gerontol. Geriatr Med.* 2022; 8. doi:10.1177/23337214211057715

[15] Miculas D, Negru P, Bungau S, Behl T, Hassan S, and Tit D. Pharmacotherapy evolution in Alzheimer's disease: current framework and relevant directions. *Cells* 2023; 12: 1–26. doi: 10.3390/cells12010131

[16] Fitzpatrick AL, Kuller LH, Lopez OL et al. Midlife and late-life obesity and the risk of dementia: cardiovascular health study. *Archives of Neurology,* 2009; 66(3): 336–342.

[17] Kivipelto M, Ngandu T, Fratiglioni L., et al. Obesity and vascular risk factors at midlife and the risk of dementia and Alzheimer disease. *Archives of Neurology,* 2005; 62(10): 1556–1560.

[18] Luchsinger JA, Cheng D, Tang MX, Schupf N, and Mayeux R. Central obesity in the elderly is related to late-onset Alzheimer disease. *Alzheimer Disease & Associated Disorders,* 2012; 26(2): 101–105.

[19] Luchsinger JA, and Gustafson DR. Adiposity and Alzheimer's disease. *Current Opinion in Clinical Nutrition & Metabolic Care*. 2009; 12(1): 15–21.

[20] Guigoz Y, Lauque S, and Vellas BJ. Identifying the elderly at risk formalnutrition the mini nutritional assessment. *Clinics in Geriatric*

Medicine, 2002; 18(4): 737–757.

[21] Saragat B, Buffa R, Mereu E, et al., Nutritional and psycho-functional status in elderly patients with Alzheimer's disease. *Journal of Nutrition, Health and Aging*, 2012; 16(3): 231–236.

[22] Ousset P-J, Nourhashemi F, Reynish E, and Vellas B. Nutritional status is associated with disease progression in very mild Alzheimer disease. *Alzheimer Disease and Associated Disorders.* 2008; 22(1): 66–71.

[23] Soto ME, Secher M, Gillette-Guyonnet S, et al. Weight loss and rapid cognitive decline in community-dwelling patients with Alzheimer's disease. *Journal of Alzheimer's Disease,* 2012; 28(3): 647–654.

[24] Borniquel S, Jansson EA, Cole MP, Freeman BA, and Lundberg JO. Nitrated oleic acid up-regulates PPARγ and attenuates experimental inflammatory bowel disease. *Free Radical Biology and Medicine*, 2010; 48(4): 499–505.

[25] Vassiliou EK, Gonzalez A, Garcia C, Tadros JH, Chakraborty G, and Toney JH. Oleic acid and peanut oil high in oleic acid reverse the inhibitory effect of insulin production of the inflammatory cytokine TNF-α both in vitro and in vivo systems. *Lipids in Health and Disease*, 2009; 8: article 25.

[26] Okereke OI, Rosner BA, Kim DH, et al., Dietary fat types and 4-year cognitive change in community-dwelling older women. *Annals of Neurology*, 2012; 22(1): 124–134.

[27] Briante R, Febbraio F, and Nucci R. Antioxidant properties of low molecular weight phenols present in the Mediterranean diet. *Journal of Agricultural and Food Chemistry*, 2003; 51(24): 6975–6981.

[28] Naqvi AZ, Harty B, Mukamal KJ, Stoddard AM, Vitolins M, and Dunn JE. Monounsaturated, trans, and saturated fatty acids and cognitive decline in women. *Journal of the American Geriatrics Society*, 2011; 59(5): 837–843.

[29] Calon F, Lim GP, Yang F, et al. Docosahexaenoic acid protects from dendritic pathology in an Alzheimer's disease mouse model. *Neuron*, 2004; 43(5): 633–645.

[30] Hooijmans CR, van der Zee CEEM, Dederen PJ, et al. DHA and cholesterol containing diets influence Alzheimer-like pathology, cognition and cerebral vasculature in APPswe/ PS1dE9 mice. *Neurobiology of Disease*, 2009; 33(3): 482–498.

[31] Lim GP, Calon F, Morihara T, et al. A diet enriched with the omega-3 fatty acid docosahexaenoic acid reduces amyloid burden in an aged Alzheimer mouse model. *Journal of Neuroscience*, 2005; 25(12): 3032–3040.

[32] Perez SE, Berg BM, Moore KA, et al. DHA diet reduces AD pathology in young APPswe/PS1ΔE9 transgenic mice: possible gender effects. *Journal of Neuroscience Research*, 2010; 88(5): 1026–1040.

[33] Canhada S, Castro K, Perry I, and Luft V. Omega-3 fatty acids' supplementation in Alzheimer's disease: a systematic review. *Nutr. Neurosci.* 2018; 21: 529–538.

[34] Samieri C, Morris M, Bennett D, Berr C, Amouyel P, Dartigues J, et al. Fish intake, genetic predisposition to Alzheimer disease, and decline in global cognition and memory in 5 cohorts of older persons. *Am. J. Epidemiol.* 2018; 187: 933–940. doi: 10.1093/aje/kwx330

[35] Moreira S, Jansen A, and Silva, F. Dietary interventions and cognition of Alzheimer's disease patients a systematic review of randomized controlled trial. *Dement. Neuropsychol.* 2020; 14: 258–282.

[36] Solfrizzi V, Frisardi V, Capurso C, et al., Dietary fatty acids in dementia and predementia syndromes: epidemiological evidence and possible underlying mechanisms. *Ageing Research Reviews*, 2010; 9(2):184–199.

[37] Laitinen MH, Ngandu T, Rovio S., et al. Fat intake at midlife and risk of dementia and Alzheimer's disease: a population based study. *Dementia and Geriatric Cognitive Disorders*, 2006; 22(1): 99–107.

[38] Grimm MO, Rothhaar TL, Grosgen S., et al. Trans fatty acids enhance amyloidogenic processing of the Alzheimer amyloid precursor protein (APP). *The Journal of Nutritional Biochemistry*, 2012; 23(10): 1214–1223.

[39] Liu Y, Liu F, Grundke-Iqbal I, Iqbal K, and Gong C.-X. Deficient brain insulin signaling pathway in Alzheimer's disease and diabetes. *Journal of Pathology*, 2011; 225(1): 54–62.

[40] Craft S. Insulin resistance and Alzheimer's disease pathogenesis: potential mechanisms and implications for treatment. *Current Alzheimer Research*, 2007; 4(2): 147–152.

[41] Fr¨olich L, Blum-Degen D, Bernstein H-G. et al. Brain insulin and insulin receptors in aging and sporadic Alzheimer's disease. *Journal of Neural Transmission*. 1998; 105(4-5): 423–438.

[42] Steen E, Terry BM, Rivera EJ et al. Impaired insulin and insulin-like growth factor expression and signaling mechanisms in Alzheimer's disease—is this type 3 diabetes? *Journal of Alzheimer's Disease,* 2005; 7(1): 63–80.

[43] Kikuchi S, Shinpo K, Takeuchi M., et al. Glycation—a sweet tempter for neuronal death. *Brain Research Reviews*, 2003; 41(2-3): 306–323.

[44] Henderson ST. High carbohydrate diets and Alzheimer's disease. *Medical Hypotheses*, 2004; 62(5): 689–700.

[45] Seneff S, Wainwright G, and Mascitelli L. Nutrition and Alzheimer's disease: the detrimental role of a high carbohydrate diet. *European Journal of Internal Medicine*, 2011; 22(2): 134–140.

[46] Barberger-Gateau P, Raffaitin C, Letenneur L., et al. Dietary patterns and risk of dementia: the Three-City cohort study. *Neurology*, 2007: 69(20); 1921–1930;

[47] Dangour AD, Allen E, Elbourne D, Fletcher A, Richards M, and Uauy R. Fish consumption and cognitive function among older people in the UK: baseline data from the OPAL study. *Journal of Nutrition, Health and Aging*, 2009; 13(3): 198–203.

[48] Huang TL, Zandi PP, Tucker KL et al. Benefits of fatty fish on dementia risk are stronger for those without APOE ε4. *Neurology*, 2005; 65(9): 1409–1414.

[49] Morris MC, Evans DA, Bienias JL et al. Consumption of fish and n-3 fatty acids and risk of incident Alzheimer disease. *Archives of Neurology*, 2003; 60(7): 940–946.

[50] Morris MC, Evans DA, Tangney CC, Bienias JL, and Wilson RS. Fish consumption and cognitive decline with age in a large community study. *Archives of Neurology*, 2005; 62(12): 1849–1853.

[51] Kritz-Silverstein D, Bettencourt R. Egg Consumption and 4-Year Change in Cognitive Function in Older Men and Women: The Rancho Bernardo Study. Nutrients. 2024 Aug 19;16(16):2765. doi: 10.3390/nu16162765.

[52] Takasaki J, Ono K, Yoshiike Y, et al. Vitamin A has antioligomerization effects on amyloid-β in vitro. *Journal of Alzheimer's Disease*, 2011; 27(2): 271–280.

[53] Bourdel-Marchasson I, Delmas-Beauviex M.-C, Peuchant E, et al., Antioxidant defences and oxidative stress markers in erythrocytes and plasma from normally nourished elderly Alzheimer patients. *Age and Ageing*, 2001; 30(3): 235–241.

[54] Jimenez-Jimenez FJ, Molina JA, de Bustos F, et al. Serum levels of beta-carotene, alpha-carotene and vitamin A in patients with Alzheimer's disease. *European Journal of Neurology*, 1999; 6(4): 495–497.

[55] Hashim A, Wanga L, Junej K, Yeb Y, Zhao Y, and Ming L.-J. Vitamin b6s inhibit oxidative stress caused by Alzheimer's disease-related Cu(II)-b-amyloid complexes-cooperative action of phospho-moiety. *Bioorganic and Medicinal Chemistry Letters*, 2011: 21(21): 6430–6432.

[56] Nilforooshan R, Broadbent D, Weaving G., et al. Homocysteine in Alzheimer's disease: role of dietary folate, vitamin B6 and B12. *International Journal of Geriatric Psychiatry*, 2011; 26(8): 876–877.

[57] Ho RCM, Cheung MWL, Fu E., et al., Is high homocysteine level a risk factor for cognitive decline in elderly? a systematic review, meta analysis, and meta-regression. *American Journal of Geriatric Psychiatry*, 2011; 19(7): 607–617.

[58] Aisen PS, Schneider LS, Sano M, et al., High-dose B vitamin supplementation and cognitive decline in Alzheimer disease: a randomized controlled trial. *Journal of the American Medical Association*, 2008; 300(15): 1774–1783.

[59] Montilla-L´opez P, Muoz-´Agueda MC, Feij´oo L´opez M, Muoz-Castaeda JR, Bujalance-Arenas I, and T´unez-Fiana I. Comparison of melatonin versus vitamin C on oxidative stress and antioxidant enzyme activity in Alzheimer's disease induced by okadaic acid in neuroblastoma cells. *European Journal of Pharmacology*, 2022; 451(3): 237–243.

[60] Murakami K, Murata N, Ozawa Y, et al., Vitamin C restores behavioral deficits and amyloid-β oligomerization without affecting plaque formation in a mouse model of Alzheimer's disease. *Journal of Alzheimer's Disease*, 2011; 26(1): 7–18.

[61] Jia J, Hu J, Huo X, Miao R, Zhang Y, and Ma F. Effects of vitamin D supplementation on cognitive function and blood Ab-related biomarkers in older adults with Alzheimer's disease: a randomized, double-blind, placebo-controlled trial. *J. Neurol. Neurosurg. Psychiatry* 2019; 90: 1347–1352. doi: 10.1136/jnnp-2018-32 0199.

[62] Guan J.-Z, Guan W.-P, Maeda T, and Makino N. Effect of vitamin E administration on the elevated oxygen stress and the telomeric and sub-telomeric status in Alzheimer's disease. *Gerontology*, 2011; 58(1): 62–69.

[63] Kaneai N, Arai M, Takatsu H, Fukui K, and Urano S. Vitamin E inhibits oxidative stress-induced denaturation of nerve terminal proteins involved in neurotransmission. *Journal of Alzheimer's Disease,* 2012; 28(1): 183–189.

[64] Khanna S, Parinandi NL, Kotha SR, et al. Nanomolar vitamine α-tocotrienol inhibits glutamate-induced activation of phospholipase A2 and causes neuro-protection. *Journal of Neurochemistry*, 2010; 112(5): 1249–1260.

[65] Yatin SM, Varadarajan S, and Butterfield DA. Vitamin E prevents Alzheimer's amyloid β-peptide (1-42)-induced neuronal protein oxidation and reactive oxygen species production. *Journal of Alzheimer's Disease*, 2000; 2(2): 123–131.

[66] Morris MC, Evans DA, Tangney CC, et al., Relation of the tocopherol forms to incident Alzheimer disease and to cognitive change. *American Journal of Clinical Nutrition*, 2005; 81(2): 508–514.

[67] Buglio D, Marton L, Laurindo L, Guiguer E, Araújo A, Buchaim R, et al. The role of resveratrol in mild cognitive impairment and Alzheimer's disease: a systematic review. *J. Med. Food.* 2022; 25: 797–806. doi: 10.1089/jmf.2021.0084.

[68] Craddock TJA, Tuszynski JA, D. Chopra et al.,

The zinc dys-homeostasis hypothesis of Alzheimer's disease. *PLoS ONE*, 2012; 7(3): Article ID e33552, 2012.

[69] Schrag M, Mueller C, Oyoyo U, Smith MA, and Kirsch WM. Iron, zinc and copper in the Alzheimer's disease brain: a quantitative meta-analysis. Some insight on the influence of citation bias on scientific opinion. *Progress in Neurobiology*, 2011; 94(3): 296–306.

[70] Loef M, and Walach H. Copper and iron in Alzheimer's disease: a systematic review and its dietary implications. *British Journal of Nutrition*, 2012; 107(1): 7–19.

[71] Morris MC, Evans DA, Tangney CC. et al., Dietary copper and high saturated and trans fat intakes associated with cognitive decline. *Archives of Neurology*, 2006; 63(8): 1085–1088.

[72] Bucossi S, Ventriglia M, Panetta V et al., Copper in Alzheimer's disease: a meta-analysis of serum plasma, and cerebrospinal fluid studies. *Journal of Alzheimer's Disease*, 2011; 24(1): 175–185.

[73] Squitti R. Copper dysfunction in Alzheimer's disease: from meta-analysis of biochemical studies to new insight into genetics. *J Trace Elem Med Biol.* 2012; 26(2-3): 93-6. doi: 10.1016/j.jtemb.2012.04.012.

[74] Kessler H, Bayer TA, Bach D, et al., Intake of copper has no effect on cognition in patients with mild Alzheimer's disease: a pilot phase 2 clinical trial. *Journal of Neural Transmission*, 2008; 115(8): 1181–1187.

[75] Castellani RJ, Moreira PI, Perry G, and Zhu X. The role of iron as a mediator of oxidative stress in Alzheimer disease. *BioFactors*, 2012; 38(2): 133–138.

[76] Smith MA, Zhu X, Tabaton M, et al. Increased iron and free radical generation in preclinical Alzheimer disease and mild cognitive impairment. *Journal of Alzheimer's Disease,* 2010; 19(1): 353–372.

[77] Falkingham M, Abdelhamid A, Curtis P, Fairweather-Tait S, Dye L, and Hooper L. The effects of oral iron supplementation on cognition in older children and adults: a systematic review and meta-analysis. *Nutrition Journal*, 2010; 9(1): article 4.

[78] Corona C, Masciopinto F, Silvestri E, et al., Dietary zinc supplementation of 3xTg-AD mice increases BDNF levels and prevents cognitive deficits as well as mitochondrial dysfunction. *Cell Death & Disease*, 2010; 1: e91.

[79] Brewer GJ. Copper excess, zinc deficiency, and cognition loss in Alzheimer's disease. *BioFactors*, 2012; 38(2): 107–113.

[80] Ishrat T, Parveen K, Khan MM, et al., Selenium prevents cognitive decline and oxidative damage in rat model of streptozotocin-induced experimental dementia of Alzheimer's type. *Brain Research*, 2009; 1281: 117–127.

[81] Mohamed J, Wei WL, Husin NNA, Alwahaibi NY, and Budin SB. Selenium supplementation reduced oxidative stress in diethyl-nitrosamine-induced hepatocellular carcinoma in rats. *Pakistan Journal of Biological Sciences*, 2011; 14(23): 1055–1060.

[82] Cardoso BR, T. P. Ong TP, Jacob-Filho W, Jaluul O, Freitas MID, and Cozzolino SMF. Nutritional status of selenium in Alzheimer's disease patients. *British Journal of Nutrition*, 2010; 103(6): 803–806.

[83] Chandra RK. Effect of vitamin and trace-element supplementation on cognitive function in elderly subjects. *Nutrition*, 2001: 17(9); 709–712.

[84] Cornelli U. Treatment of Alzheimer's disease with a cholinesterase inhibitor combined with antioxidants. *Neurodegenerative Diseases*, 2010; 7(1–3): 193–202.

[85] Scheltens P, Kamphuis PPGH, Verhey FRJ, et al., Efficacy of a medical food in mild Alzheimer's disease: a randomized, controlled trial. *Alzheimer's and Dementia*, 2010; 6(1): 1.e1–10.e1.

[86] Barberger-Gateau P, Raffaitin C, Letenneur L, et al., Dietary patterns and risk of dementia: the Three-City cohort study. *Neurology*, 2007; 69(20): 1921–1930.

[87] Hughes TF, Andel R, Small BJ., et al., Midlife fruit and vegetable consumption and risk of dementia in later life in Swedish twins. *American Journal of Geriatric Psychiatry*, 2010; 18(5): 413–420.

[88] Morris MC, Evans DA, Tangney CC, Bienias JL, and Wilson RS, Associations of vegetable and fruit consumption with age-related cognitive change. *Neurology*, 2006; 67(8): 1370– 1376.

[89] Nooyens ACJ, Bueno-De-Mesquita HB, van Boxtel MPJ, vanGelder BM, Verhagen H, and Verschuren WMM, Fruit and vegetable intake and cognitive decline in middle-aged men and women: the Doetinchem Cohort Study. *British Journal of Nutrition*, 2011; 106(5): 752–761.

[90] Crichton GE, Bryan J, Murphy KJ, and Buckley J. Review of dairy consumption and cognitive performance in adults: findings and methodological issues. *Dementia and Geriatric Cognitive Disorders*, 2010; 30(4): 352–361.

[91] Lee L, Kang SA, Lee HO, et al., Relationships between dietary intake and cognitive function level in Korean elderly people. *Public Health*, 2001; 115(2): 133–138.

[92] Eskelinen MH, Ngandu T, Helkala E.-L, et al., Fat intake at midlife and cognitive impairment later in life: a population based CAIDE study. *International Journal of Geriatric Psychiatry*, 2008; 23(7): 741–747

[93] Cao C, Wang L, Lin X, et al., Caffeine synergizes with another coffee component to increase plasma GCSF: linkage to cognitive

benefits in Alzheimer's mice. *Journal of Alzheimer's Disease,* 2011; 25(2): 323–335.

[94] Barranco Quintana JL, Allam MF, Del Castillo AS and Navajas RFC, Alzheimer's disease and coffee: a quantitative review. *Neurological Research,* 2007; 29(1): 91–95.

[95] Feng L, Gwee X, Kua E.-H, and Ng T.-P. Cognitive function and tea consumption in community dwelling older Chinese in Singapore. *Journal of Nutrition, Health and Aging,* 2010; 14(6): 433–438.

[96] Ng T.-P, Feng L, Niti M, Kua E.-H, and Yap K.-B. Tea consumption and cognitive impairment and decline in older Chinese adults. *American Journal of Clinical Nutrition.* 2008; 88(1): 224–231.

[97] De Bruin EA, Rowson MJ, van Buren L, Rycroft JA, and Owen GN. Black tea improves attention and self-reported alertness. *Appetite,* 2011; 56(2): 235–240.

[98] Mandel SA, Amit T, Kalfon L, Reznichenko L, and Youdim MBH. Targeting multiple neurodegenerative diseases etiologies with multimodal-acting green tea catechins. *Journal of Nutrition,* 2008; 138(8): 1578S–1583S.

[99] Weinreb O, Mandel S, Amit T, and Youdim MBH. Neurological mechanisms of green tea polyphenols in Alzheimer's and Parkinson's diseases. *Journal of Nutritional Biochemistry,* 2004; 15(9): 506–516.

[100] Xu Y, Zhang J.-J, Xiong L, Zhang L, Sun D, and Liu H. Green tea polyphenols inhibit cognitive impairment induced by chronic cerebral hypo-perfusion via modulating oxidative stress. *Journal of Nutritional Biochemistry,* 2010; 21(8): 741– 748.

[101] Rezai-Zadeh K, Arendash GW, Hou H, et al., Green tea epigallocatechin-3-gallate (EGCG) reduces *β*-amyloid mediated cognitive impairment and modulates tau pathology in Alzheimer transgenic mice. *Brain Research,* 2008; 1214:177–187.

[102] Rezai-Zadeh K, Shytle D, Sun N, et al., Green tea epigallocatechin-3-gallate (EGCG) modulates amyloid precursor protein cleavage and reduces cerebral amyloidosis in Alzheimer transgenic mice. *Journal of Neuroscience,* 2005; 25(38): 8807– 8814.

[103] Song J, Xu H, Liu F, and Feng L. Tea and cognitive health in late life: current evidence and future directions. *Journal of Nutrition, Health and Aging,* 2012; 16(1): 31–34.

[104] Anstey KJ, Mack HA, and Cherbuin N. Alcohol consumption as a risk factor for dementia and cognitive decline: meta-analysis of prospective studies. *American Journal of Geriatric Psychiatry,* 2009; 17(7): 542–555.

[105] Panza F, Capurso C, D'Introno A, et al. Alcohol drinking, cognitive functions in older age, predementia, and dementia syndromes. *Journal of Alzheimer's Disease,* 2009; 17(1): 7–31.

[106] Weyerer S, Sch¨aufele M, Wiese B, et al., Current alcohol consumption and its relationship to incident dementia: results from a 3-year follow-up study among primary care attenders aged 75 years and older. *Age and Ageing,* 2011; 40(4): 456– 463.

[107] Harwood DG, Kalechstein A, Barker WW, et al., The effect of alcohol and tobacco consumption, and apolipoprotein E genotype, on the age of onset in Alzheimer's disease. *International Journal of Geriatric Psychiatry,* 2010; 25(5): 511–518.

[108] Huang T.-C, Lu K.-T, Wo Y.-Y.P, Wu, Y.-J and Yang Y.-L. Resveratrol protects rats from A*β*-induced neurotoxicity by the reduction of iNOS expression and lipid peroxidation. *PLoS ONE,* 2011; 6(12): Article ID e29102.

[109] Karuppagounder SS, Pinto JT, Xu H, Chen H.-L, Beal M.F, and Gibson G.E. Dietary supplementation with resveratrol reduces plaque pathology in a transgenic model of Alzheimer's disease. *Neurochemistry International,* 2009; 54(2): 111–118.

[110] Ho L, Ferruzzi Mario G, Janle E M, et al., Identification of brain-targeted bioactive dietary quercetin-3-O-glucuronide as a novel intervention for Alzheimer's disease. *The FASEB Journal,* 2013; 27(2): 769–781

[111] Gustaw-Rothenberg K. Dietary patterns associated with Alzheimer's disease: population-based study. *International Journal of Environmental Research and Public Health,* 2009; 6(4): 1335–1340.

[112] Fung TT, Stampfer MJ, Manson JE, Rexrode KM, Willett WC, and Hu FB. Prospective study of major dietary patterns and stroke risk in women. *Stroke,* 2004; 35(9): 2014–2019.

[113] Hooijmans CR, Rutters F, Dederen PJ, et al., Changes in cerebral blood volume and amyloid pathology in aged Alzheimer APP/PS1 mice on a docosahexaenoic acid (DHA) diet or cholesterol enriched Typical Western Diet (TWD). *Neurobiology of Disease,* 2007; 28(1): 16–29.

[114] Studzinski CM, Li F, Bruce-Keller AJ, et al., Effects of short-term Western diet on cerebral oxidative stress and diabetes related factors in APP x PS1 knock-in mice. *Journal of Neurochemistry,* 2009; 108(4): 860–866.

[115] Hankey GJ. Nutrition and the risk of stroke. *The Lancet Neurology,* 2012: 11(1): 66–81.

[116] Ozawa M, Ninomiya T, Ohara T, et al., Dietary patterns and risk of dementia in an elderly Japanese population: the Hisayama study. *The American Journal of Clinical Nutrition,* 2013; 97(5): 1076–1082.

[117] Kesse-Guyot E, Andreeva VA, Jeandel C, Ferry M, Hercberg S, and Galan P. A healthy dietary pattern at midlife is associated with subsequent

cognitive performance. *Journal of Nutrition*, 2012; 142(5): 909–915.

[118] Samieri C, Jutand M.-A., F´eart C, Capuron L, Letenneur L, and Barberger-Gateau P. Dietary patterns derived by hybrid clustering method in older people: association with cognition, mood, and self-rated health. *Journal of the American Dietetic Association*, 2008; 108(9): 1461–1471.

[119] Eskelinen MH, Ngandu T, Tuomilehto J, Soininen H, and Kivipelto M. Midlife coffee and tea drinking and the risk of late-life dementia: a population-based CAIDE study. *Journal of Alzheimer's Disease*, 2009; 16(1): 85–91.

[120] Hankey GJ. Nutrition and the risk of stroke. *The Lancet Neurology*, 2012; 11(1): 66–81.

[121] Birkenh¨ager WH, Staessen JA, Casserly IP, et al., Convergence of atherosclerosis and Alzheimer's disease. *The Lancet*, 2004; 363(9426): 2091–2092.

[122] Smith PJ, Blumenthal JA, Babyak MA, et al., Effects of the dietary approaches to stop hypertension diet, exercise, and caloric restriction on neurocognition in overweight adults with high blood pressure. *Hypertension*, 2010; 55(6): 1331–1338.

[123] Scarmeas N, Luchsinger JA, Mayeux R, and Stern Y. Mediterranean diet and Alzheimer disease mortality. *Neurology*, 2007; 69(11): 1084–1093.

[124] Sofi F, Abbate R, Gensini GF, and Casini A. Accruing evidence on benefits of adherence to the Mediterranean diet on health: an updated systematic review and meta-analysis. *American Journal of Clinical Nutrition*, 2010; 92(5): 1189–1196.

[125] Scarmeas N, Stern Y, Mayeux R, and Luchsinger JA. Mediterranean diet, Alzheimer disease, and vascular mediation. *Archives of Neurology*, 2006; 63(12), 1709–1717.

[126] Scarmeas N, Y. Stern, Mayeux R, Manly JJ, Schupf N, and Luchsinger JA. Mediterranean diet and mild cognitive impairment. *Archives of Neurology*, 2009; 66(2); 216–225.

[127] Huang TL, Zandi PP, Tucker KL, et al., Benefits of fatty fish on dementia risk are stronger for those without APOE ε4. *Neurology*, 2005; 65(9): 1409–1414.

[128] Morris MC, Evans DA, Bienias JL, et al., Consumption of fish and n-3 fatty acids and risk of incident Alzheimer disease. *Archives of Neurology*, 2003; 60(7): 940–946.

[129] Gentreau M, Chuy V, Féart C, Samieri C, Ritchie K, Raymond M, et al. Refined carbohydrate-rich diet is associated with long-term risk of dementia and Alzheimer's disease in apolipoprotein E+4 allele carriers. *Alzheimer's Dem.* 2020; 16, 1043–1053. doi: 10.1002/alz.12114

[130] Taylor M, Sullivan D, Morris J, Vidoni E, Honea R, Mahnken J, et al. High glycemic diet is related to brain amyloid accumulation over one year in preclinical Alzheimer's disease. *Front. Nutr.* 2021; 8:741534. doi: 10.3389/fnut.2021.741534

[131] Smith GD. Reflections on the limitations to epidemiology. *Journal of Clinical Epidemiology*, 2001; 54(4): 325–331.

[132] Wieckowska-Gacek A, Mietelska-Porowska A, Wydrych M, and Wojda U. Western diet as a trigger of Alzheimer's disease: from metabolic syndrome and systemic inflammation to neuroinflammation and neurodegeneration. *Ageing Res. Rev.* 2021; 70:101397. doi: 10.1016/j.arr.2021.101397

[133] Nutaitis A, Tharwani S, Serra M, Goldstein F, Zhao L, Sher S, et al. Diet as a risk factor for cognitive decline in African Americans and Caucasians with a parental history of Alzheimer's disease: a cross-sectional pilot study dietary patterns. *J. Prev. Alzheimer's Dis.* 2019; 6: 50–55. doi: 10.14283/jpad.2018.44

[134] Ballarini T, van Lent D, Brunner J, Schroeder A, Wolfsgruber S, Altenstein S, et al. Mediterranean diet, Alzheimer disease biomarkers, and brain atrophy in old age. *Neurology*, 2021; 96: E2920–E2932.

[135] Encarnacion Andreu-Reinon M, Dolores Chirlaque M, Gavrila D, Amiano P, Mar J, Tainta M, et al. Mediterranean diet and risk of dementia and Alzheimer's disease in the EPIC-spain dementia cohort study. *Nutrients*, 2021; 13: 700.

[136] Alam J. Vitamins: a nutritional intervention to modulate the Alzheimer's disease progression. *Nutr. Neurosci.* 2022; 25: 945–962.

[137] Yilmaz R, and Arica Polat B. Vitamin D is associated with cognitive status in patients with Alzheimer's disease. *Turk Geriatr Derg*. 2019; 22: 361–367.

[138] Chai B, Gao F, Wu R, Dong T, Gu C, Lin Q, et al. Vitamin D deficiency as a risk factor for dementia and Alzheimer's disease: an updated meta-analysis. BMC Neurol. 2019; 19: 284. doi: 10.1186/s12883-019-1500-6

[139] Jayedi A, Rashidy-Pour A, and Shab-Bidar, S. Vitamin D status and risk of dementia and Alzheimer's disease: a meta-analysis of dose-response. *Nutr. Neurosci.* 2019; 22: 750–759. doi: 10.1080/1028415X.2018.1436639

[140] Jia J, Hu J, Huo X, Miao R, Zhang Y, and Ma F. Effects of vitamin D supplementation on cognitive function and blood A□-related biomarkers in older adults with Alzheimer's disease: a randomized, double-blind, placebo-controlled trial. *J. Neurol. Neurosurg. Psychiatry* 2019; 90: 1347–1352. doi: 10.1136/jnnp-2018-32 0199

[141] Yang K, Chen J, Li X, and Zhou Y. Vitamin D concentration and risk of Alzheimer disease: a meta-analysis of prospective cohort studies. Medicine 2019; 98: e16804. doi: 10.1097/MD.0000000000016804

[142] Du Y, Liang F, Zhang L, Liu J, and Dou H. Vitamin D supplement for prevention of Alzheimer's disease: a systematic review and meta-analysis. *Am. J. Ther*. 2020; 28: e638–e648.

[143] Shrestha L, Shrestha B, Gautam K, Khadka S, and Mahara Rawal N. Plasma vitamin B-12 levels and risk of Alzheimer's disease: a case-control study. *Gerontol. Geriatr Med.* 2022; 8: 23337214211057715.

[144] Casati M, Boccardi V, Ferri E, Bertagnoli L, Bastiani P, Ciccone S, et al. Vitamin E and Alzheimer's disease: the mediating role of cellular aging. *Aging Clin. Exp. Res.* 2020; 32: 459–464.

Citation: *Ashok Chakraborty, et al. " Management of Alzheimer's Disease Through Nutrition" ARC Journal of Neuroscience, 2025; 8(1): 6-16. DOI:* https://doi.org/*10.20431/2456-057X.0801002.*