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Dietary Supplementation with Macronutrients and Vitamins to Prevent and Slow the Progression of Alzheimer's Disease: A Comprehensive Review

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ABSTRACT

Alois Alzheimer, in 1901, first described Alzheimer's Disease, a progressive neurological disorder that stands as the leading cause of dementia. Its prevalence is alarmingly escalating globally, correlating with increased life expectancies. Current treatments for Alzheimer's Disease are limited, with available medications being expensive and often accompanied by adverse health effects. This necessitates the exploration of natural, readily accessible, and practical therapeutic approaches to both prevent and decelerate the disease's progression. Dietary awareness emerges as a significant natural intervention. Recent research has increasingly focused on the link between nutrition and Alzheimer's Disease, demonstrating a strong correlation between dietary patterns and the disease's development. A well-balanced, sustainable diet, rich in essential macronutrients (proteins, carbohydrates, and fats) and supplemented with vital vitamins, holds potential in preventing Alzheimer's onset in healthy individuals and significantly slowing its advancement in diagnosed patients. This review aims to analyze current research on the impact of macronutrients and vitamins in mitigating Alzheimer's Disease.

Keywords: Alzheimer's Disease, nutrition, macronutrients, prevention, vitamins

INTRODUCTION

In 1901, the German psychiatrist and neuropathologist, Alois Alzheimer, encountered Auguste Deter, a 51-year-old patient exhibiting behavioral anomalies impacting daily life, persistent paranoia, severe memory impairment, and dementia symptoms. Intrigued, Alzheimer closely monitored the patient's symptom progression until her death in 1906. Hypothesizing that these symptoms stemmed from physiological and biochemical brain alterations, Alzheimer sought to examine Deter's brain post-mortem. Obtaining approval, he conducted detailed microscopic analyses, revealing significant brain changes, including amyloid plaques and neurofibrillary tangles, indicative of pathological signs. In 1906, he published his findings on these observations. Dr. Emil Kraepelin, advocating for pathological bases of mental disorders, embraced Alzheimer's findings, naming the condition 'Alzheimer's' (1-4).

Alzheimer's Disease (AD), the most prevalent cause of dementia, is a neurological condition primarily resulting from the accumulation of abnormally folded amyloid beta protein in the brain, classifying it as a proteopathy (5, 6). The disorder manifests through various histopathological, molecular, and biochemical abnormalities, such as amyloid beta protein deposits, neuronal loss, neurofibrillary tangles, dystrophic neurites, increased pro-death gene activation, impaired energy metabolism, mitochondrial dysfunction, chronic oxidative stress, and DNA damage (7). These abnormalities also reflect brain insulin resistance and deficiency, with biochemical consequences overlapping with type 1 and type 2 diabetes. Hence, recent studies emphasize insulin resistance and glucose metabolism impairment in AD pathogenesis, suggesting its reclassification as “type 3 diabetes” (8, 9).

AD, a progressive neurodegenerative condition marked by cognitive decline, lacks a definitive cure, and its medications often induce side effects (10). Consequently, a tailored daily diet and supplementary intake become crucial for AD patients. Dietary adjustments and conscious nutrition programs can modify the disease’s trajectory, slow its progression, and enhance patient quality of life (11, 12).

This review explores the relationship between AD and diet, focusing on vitamin and macronutrient programming, based on current literature.

PROTEIN, CARBOHYDRATES, AND FATS IN ALZHEIMER'S DISEASE

The impact of diet and lifestyle on cognitive function and neurodegenerative diseases has increasingly been examined, particularly in the last three decades. Studies on macronutrient (carbohydrate, fat, and protein) effects on AD development and progression have intensified (13). Cross-sectional and epidemiological studies highlight nutritional parameters' influence on human cognition (14). Chronic conditions like type 2 diabetes, insulin resistance, obesity, cardiovascular disease, and hypertension are dementia risk factors (15). Similarly, dietary patterns contribute to cognitive impairment and AD risk. Optimal macronutrient balance through diet enhances cognitive function, while poor dietary habits can lead to metabolic disorders and chronic diseases (16).

Recent studies indicates that excessive carbohydrate intake, especially fructose, may contribute to AD development. Elevated plasma glucose and monosaccharides can cause glycation of fat, cholesterol, and oxygen transport proteins, altering their structure. This leads to neuronal cholesterol deficiency, impairing nerve cell function. Over time, this results in disrupted glutamate signaling, increased oxidative damage, mitochondrial and lysosomal dysfunction, and higher microbial infection risk, leading to apoptosis. These metabolic disorders can significantly contribute to neurodegenerative diseases, including Alzheimer's (17).

A study at the University of Kansas Medical Center observed brain amyloid plaque changes in cognitively normal older adults with high-glycemic diets. Results showed increased amyloid deposition in temporoparietal cortex regions in adults with high-carbohydrate diets, especially those with high amyloid status, over a year (18). Animal studies have shown that chronic high-calorie diets, rich in sugar and saturated fatty acids, can induce brain damage, triggering inflammation in areas like the hypothalamus, hippocampus, and frontal cortex, increasing oxidative stress, and causing metabolic disturbances, reduced dendritic spines, and memory loss (19).

The ketogenic diet, high in fat, moderate in protein, and low in carbohydrates, has been used since 500 BC, notably for epilepsy (20). It remains a natural treatment for drug-resistant epilepsy (21-23). Clinical studies show long-term therapeutic benefits for epilepsy patients on ketogenic diets (24-26). Recent research suggests it can reduce anti-seizure medication use (27). A pilot study on AD patients showed slight cognitive improvements after 12 weeks on a ketogenic diet (28). Animal studies indicate potential symptomatic and disease-modifying effects in neurodegenerative disorders, including AD and Parkinson's, and protective effects in brain injury and stroke (29-32).

The Mediterranean Diet, introduced by Ancel Keys, emphasizes unrefined grains, fruits, vegetables, legumes, olive oil, moderate fish and poultry consumption, and limited dairy and red meat (33-36). A study of 512 patients found that a Mediterranean-like diet correlated with greater medial-temporal gray matter volume, better memory, and reduced amyloid and p-tau pathology (37). A large cohort study showed high adherence to the Mediterranean diet was associated with better cognitive performance and reduced memory decline (38). Overall, a diet low in high-glycemic carbohydrates, rich in healthy fats, and balanced in animal and vegetable proteins may be beneficial for brain health.

VITAMINS AND ALZHEIMER'S DISEASE

Vitamin A derivatives, crucial in brain cellular processes, show decreased serum/plasma concentrations in AD (39-41). Retinoid deficiency may predispose to AD by increasing cerebral amyloid β accumulation (42). Studies on transgenic mice showed that all-trans retinoic acid, a vitamin A metabolite, reduced brain amyloid beta accumulation and tau phosphorylation, improved spatial learning, and decreased neuronal degeneration (43).

A study of elderly men showed that B vitamin supplements reduced Aβ40 protein increase, suggesting a potential role in AD prevention (44). Meta-analyses indicate that vitamin B supplementation slows cognitive decline, especially with early and long-term intervention, and that folate intake is associated with reduced dementia incidence (45). Folic acid and vitamin B12 supplements improved cognitive performance and reduced inflammation in elderly patients with mild cognitive impairment (46). These supplements also prevented AD-like memory deficits (47). Low vitamin B12 intake was associated with accelerated cognitive decline and affected DNA methylation of redox-related genes (48).

Vitamin C deficiency can disrupt neurotransmitter synthesis, increase oxidative stress, and promote amyloid beta plaque accumulation, contributing to AD neurodegeneration. Meta-analyses suggest vitamin C deficiency plays a role in AD progression, and supplementation may be beneficial (49). Vitamin

C attenuates neuroinflammation and inhibits amyloid beta peptide accumulation (50). It may also have preventive and therapeutic effects on mental illnesses like depression, schizophrenia, and anxiety (51).

Vitamin D, a steroid hormone, influences calcium-phosphorus metabolism, cardiovascular health, immune responses, and brain function (52-55). Low vitamin D levels are associated with neurodegeneration and cognitive decline (56). Vitamin D affects neurotransmitter levels, regulates neurotrophic factors, and aids in amyloid beta peptide clearance (57-59). It may also help clear amyloid plaques through innate immune cells and alleviate cognitive deficits (60). Vitamin E, an antioxidant, may protect cognitive health by scavenging free radicals (61, 62). Low vitamin E levels increase AD risk, and a vitamin E-rich diet may slow AD progression (63).

Vitamin K, crucial for sphingolipid metabolism, affects brain cell proliferation, differentiation, and survival (64). It also impacts Gas-6 protein and protein S molecules, vital for cognitive function (65). Vitamin K deficiency is associated with cognitive impairment (66). Vitamin K2 may prevent apoptosis, oxidative stress, and microglial activation in neuronal cells, showing promise against AD (66).

CONCLUSION

AD is a progressive neurodegenerative disorder that affects millions of individuals globally and continues to increase in prevalence due to the aging population. Despite extensive research, no definitive cure for AD currently exists. Moreover, the pharmacological treatments available are associated with numerous health complications, primarily due to their adverse side effects and high cost, which significantly limit their long-term applicability and effectiveness. In light of these limitations, there is a growing interest in natural and non-pharmacological approaches aimed at preventing or decelerating the progression of the disease. Among these, conscious and evidence-based nutritional strategies have garnered particular attention. Emerging research increasingly supports a strong correlation between dietary habits and the onset and progression of AD. Numerous findings suggest that this condition is intimately linked to long-term nutritional behaviors. A well-structured and sustainable diet—balanced in macronutrients (proteins, carbohydrates, and fats) and adequately supplemented with essential vitamins and micronutrients—has the potential not only to delay the onset of AD in healthy individuals but also to significantly slow its progression in those already affected. Nevertheless, it must be acknowledged that further scientific investigation is required to elucidate the precise mechanisms by which diet influences AD pathophysiology. As the etiopathogenesis of Alzheimer's Disease remains partially understood, more studies are essential to validate existing data and to explore new dietary intervention strategies.

In conclusion, while pharmacological treatments remain integral to the current clinical management of AD, nutrition-based approaches offer a promising complementary avenue that is both accessible and low-risk. Continued interdisciplinary research is imperative to better define the role of diet in the prevention and management of Alzheimer's Disease and to develop standardized nutritional guidelines that can be implemented in clinical and public health contexts.

DECLARATIONS

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Contributions: This study was conducted by a single author.

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