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Review Article

Nutrition and Neuroprotection in Aging: A Review of Omega-3, Flavonoids, B-Vitamins, MCT Oil, and Ketogenic Protocols for Dementia Prevention

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Abstract

Population aging has led to a global increase in dementia, particularly Alzheimer's disease and vascular dementia. Diet is a key modifiable factor with growing potential for neuroprotection. This review synthesizes mechanistic, epidemiological, and clinical evidence on five nutritional strategies relevant to dementia prevention: omega-3 polyunsaturated fatty acids (PUFAs), flavonoids, B-vitamins, medium-chain triglyceride (MCT) oil, and ketogenic approaches. A narrative review with systematic search elements was conducted using PubMed, Web of Science, and Scopus, prioritizing meta-analyses, large prospective cohorts, and randomized controlled trials involving older adults, mild cognitive impairment (MCI), and dementia. Evidence indicates that higher intake or status of omega-3 PUFAs and flavonoid-rich foods is associated with reduced dementia risk and modest cognitive benefits, particularly in midlife or prodromal stages. B-vitamin supplementation may slow brain atrophy and cognitive decline in individuals with elevated homocysteine, while effects are limited in unselected populations. MCT oil and ketogenic interventions improve cognition by providing alternative brain energy substrates, with greater benefits observed in MCI and APOE ε4 non-carriers. Overall, a multi-target nutritional approach addressing neuroinflammation, vascular health, homocysteine metabolism, and brain energy deficits shows promise for dementia prevention. Future research should emphasize biomarker-guided personalization and integration with broader lifestyle interventions.

Keywords: dementia prevention; omega-3 fatty acids; flavonoids; B-vitamins; ketogenic diet

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1 Introduction

The global prevalence of dementia is projected to exceed 150 million by 2050, with the greatest absolute increases occurring in low- and middle-income countries [1]. Alzheimer's disease accounts for the majority of cases, followed by vascular dementia and mixed phenotypes [1], [2]. Age is the strongest risk factor, but the long prodromal phase and the modest efficacy of disease-modifying pharmacotherapies have directed attention to prevention and risk reduction [2].

Nutritional strategies are particularly relevant to geriatric neuroprotection for several reasons. First, the brain is highly metabolically active and rich in polyunsaturated fatty acids, which are vulnerable to oxidative damage [1], [3]. Second, ageing is accompanied by chronic low-grade inflammation ("inflammaging"), insulin resistance, and vascular dysfunction, all of which can be modified by diet [1], [4]. Third, nutrients can target multiple pathogenic mechanisms amyloid and tau pathology, oxidative stress, mitochondrial dysfunction, synaptic failure, and cerebrovascular compromise simultaneously [1], [5], [6], [7].

Observational studies suggest that adherence to Mediterranean, DASH (Dietary Approaches to Stop Hypertension), and MIND (Mediterranean–DASH Intervention for Neurodegenerative Delay) dietary patterns is associated with reduced incidence of cognitive decline and dementia (Song et al., 2024; Vauzour et al., 2017). These patterns share several common elements: high intake of marine fish (omega-3), plant foods rich in flavonoids and antioxidants, whole grains, and nuts, and low intake of ultra-processed foods, refined sugars, and saturated fats [1], [2], [8].

Beyond whole-diet patterns, there is growing interest in specific nutritional compounds that may exert targeted neuroprotective effects. This review focuses on five such strategies that are mechanistically plausible, backed by human data, and increasingly used in clinical and community settings: (a) marine omega-3 long-chain polyunsaturated fatty acids (LCPUFAs), (b) flavonoids and flavonoid-rich foods, (c) B-vitamins and homocysteine-lowering protocols, (d) MCT oil supplementation, and (e) ketogenic dietary protocols [5], [6], [7], [9], [10], [11], [12].

2 Method

2.1 Narrative Review with Systematic Search Components

A narrative review approach was adopted to integrate mechanistic, epidemiologic, and clinical trial data, supplemented by systematic search components to enhance rigor [1], [5], [6], [7], [9], [10], [13].

Databases included PubMed, Web of Science, and Scopus. The search period extended from database inception to December 11, 2025. Key terms were combinations of nutritional exposures and cognitive outcomes: "omega-3," "DHA," "EPA," "fish oil," "cognitive decline," "dementia," "Alzheimer," "older adults," "elderly" [4], [5]; "flavonoids," "polyphenols," "tea," "cocoa," "berries," "cognition," "dementia risk" [6], [13], [14]; "vitamin B12," "folate," "vitamin B6," "homocysteine," "brain atrophy," "MCI" [7], [9], [15]; and "medium-chain triglyceride," "MCT oil," "ketone," "ketogenic diet," "MCI," "Alzheimer's disease" [10], [11], [12], [16].

We prioritized human studies in adults older than 50 years or with mean age ≥60 years; prospective cohorts evaluating incident dementia, MCI, or trajectories of cognitive decline; RCTs of supplementation or dietary protocols with cognitive or biomarker outcomes; and translational mechanistic studies linking nutritional interventions to brain imaging or biochemical markers [1], [5], [6], [9], [10], [17].

3 Result and Discussion

3.1 Omega-3 Polyunsaturated Fatty Acids

3.1.1 Mechanistic Basis for Neuroprotection

DHA is a major structural component of neuronal membranes and synaptic terminals, influencing membrane fluidity, receptor function, and signal transduction [3], [4]. Experimental models demonstrate that DHA modulates amyloid precursor protein processing, reduces Aβ aggregation, and promotes clearance [4], [18]. DHA-derived lipid mediators, including resolvins and protectins, attenuate neuroinflammation by dampening microglial activation and cytokine release [18]. EPA, while less

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abundant in brain phospholipids, has systemic anti-inflammatory and vascular effects that may indirectly protect the brain [3], [4].

3.1.2 Prospective Cohort Evidence

The comprehensive meta-analysis by Bao-Zhen et al. (2023), combining the Alzheimer's Disease Neuroimaging Initiative cohort with 48 longitudinal studies (N ≈ 103,651), reported that long-term omega-3 supplement users had a 64% lower risk of incident AD in ADNI (hazard ratio [HR] = 0.36, 95% confidence interval [CI] [0.18, 0.72]). Across cohorts, higher dietary DHA and EPA intake was associated with approximately 18–20% lower risk of all-cause dementia or cognitive decline, with a dose–response pattern such that each 0.1 g/day increment of DHA or EPA was associated with an 8–9.9% lower risk [5]. Earlier narrative and integrative reviews converge on similar conclusions: low fish or omega-3 intake is consistently linked to increased risk of dementia and age-related cognitive decline [1], [4]. Some evidence suggests that protective effects may be attenuated in APOE ε4 carriers, although results are heterogeneous across studies [1], [5].

3.1.3 Randomized Controlled Trials

RCTs of omega-3 supplementation show mixed results. In mild-to-moderate AD, several trials of 1–2 g/day DHA/EPA report limited or no benefit on global cognitive measures when therapy is initiated after substantial neurodegeneration has occurred [1], [4]. In contrast, trials in MCI or in older adults with subjective memory complaints tend to demonstrate modest improvements in specific domains such as episodic memory or attention and reduced hippocampal atrophy rates, particularly among individuals with low baseline omega-3 status and elevated homocysteine [1], [5], [7].

Collectively, the RCT literature suggests greater efficacy when omega-3 supplementation is started earlier in the disease continuum, in individuals with low baseline omega-3 index, and possibly in non-APOE ε4 carriers [1], [4], [5].

3.1.4 Practical Implications

For geriatric dementia prevention, practical targets include at least two servings per week of oily fish or 500–1,000 mg/day supplemental EPA+DHA for individuals with low fish intake [1], [5]. Monitoring the erythrocyte omega-3 index could refine dosing, with proposed neuroprotective ranges around 8–12%, though this has yet to be standardized in routine geriatric care [5].

3.2 Flavonoids and Flavonoid-Rich Foods

3.2.1 Mechanism of Action

Flavonoids, including flavonols, flavan-3-ols, anthocyanins, flavanones, flavones, and isoflavones, are abundant in tea, berries, apples, citrus fruits, onions, and cocoa [6], [8]. These compounds exert antioxidant effects, upregulate endogenous defense pathways such as Nrf2, improve endothelial nitric oxide bioavailability, and modulate signaling cascades involved in synaptic plasticity [1], [8]. Through these mechanisms, flavonoids may counteract oxidative stress, neuroinflammation, and vascular dysfunction associated with cognitive aging [1], [6].

3.2.2 Meta-analysis of Flavonoid Interventions

The meta-analysis by Cheng et al. (2022), which synthesized results from 16 RCTs of flavonoid-rich cocoa, berries, tea, and grape products, found small but statistically significant improvements in global cognition, executive function, and processing speed, particularly in older adults and individuals with low baseline cognitive performance. Although effect sizes were modest and trials short (4–24 weeks), this evidence supports a causal contribution of flavonoid intake to cognitive function [6].

3.2.3 Prospective Cohort Evidence

Prospective cohort data strengthen these findings. In two large U.S. cohorts, long-term dietary flavonoid intake was associated with reduced odds of subjective cognitive decline, with the greatest benefit at higher intakes of flavones, flavanones, and anthocyanins [19]. Similarly, in the Framingham Heart Study Offspring cohort, higher consumption of flavonoid-rich fruits such as berries was associated with lower risk of incident dementia [14].

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In UK Biobank (n ≈ 122,000), Jennings et al. (2024) derived a "flavodiet" score based on intake of tea, berries, apples, onions, citrus fruits, and dark chocolate. Participants in the highest quintile of this score had a 28% lower hazard of incident dementia (HR = 0.72, 95% CI [0.57, 0.89]) compared with the lowest quintile, even after adjusting for numerous potential confounders. Importantly, this association persisted in individuals with high genetic risk and in those with hypertension or depression [13].

3.2.4 Dietary Implementation

From a practical standpoint, increasing flavonoid intake can be achieved through culturally adaptable steps: consuming two to three cups of tea per day, eating berries several times per week, and regularly consuming apples, citrus fruits, and moderate amounts of dark chocolate [6], [8], [13]. These choices are compatible with Mediterranean and MIND diet patterns and simultaneously support cardiovascular and metabolic health [1], [2], [8].

3.3 B-Vitamins, Homocysteine, and Brain Atrophy

3.3.1 Homocysteine as a Modifiable Risk Factor

Elevated plasma total homocysteine (tHcy) is an independent risk factor for cognitive decline, brain atrophy, and dementia [7], [9], [15]. B-vitamins—folate, vitamin B12, and vitamin B6—are co-factors in one-carbon metabolism and homocysteine remethylation and transsulfuration pathways [7], [15]. Hyperhomocysteinemia may contribute to cerebrovascular disease, oxidative stress, excitotoxicity, and promotion of amyloid and tau pathology [7].

3.3.2 Observational Evidence

Clarke (2007) highlighted that elevated tHcy is widespread among older adults and is strongly associated with increased risk of dementia. Subsequent analyses by Clarke et al. (2014) demonstrated that even modest elevations in tHcy are associated with faster cognitive decline. Smith et al. (2016) reviewed convergent evidence linking B12 deficiency, low folate, and high homocysteine with increased brain atrophy and cognitive impairment, supporting a causal role for homocysteine in neurodegeneration

3.3.3 Randomized Trials of Homocysteine-Lowering

In the pooled meta-analysis of 11 RCTs (N ≈ 22,000), B-vitamin supplementation effectively lowered homocysteine but produced mixed cognitive results, with most trials in unselected older adults showing no substantial overall cognitive benefit [9]. However, trials specifically targeting older adults with MCI and elevated homocysteine have reported more favorable outcomes. Smith et al. (2016) summarized VITACOG-type trials in which high-dose folic acid, vitamin B12, and vitamin B6 slowed whole-brain atrophy and cognitive decline when baseline tHcy was ≥11 µmol/L. These benefits were particularly evident in participants with adequate omega-3 status, suggesting an interaction between B-vitamins and omega-3 fatty acids [5], [7].

3.3.4 Clinical Implications

In geriatric practice, screening for B12 deficiency and elevated homocysteine is recommended, especially in older adults with MCI, malnutrition, or polypharmacy [7], [15]. Correction of overt deficiency, for example with parenteral B12, is standard of care. High-dose B-vitamin therapy may be considered for individuals with MCI and elevated tHcy, particularly when combined with other neuroprotective nutritional strategies such as omega-3 supplementation [7], [9].

3.4 MCT Oil and Ketogenic Protocols

3.4.1 Cerebral Glucose Hypometabolism and Ketones

Reduced cerebral glucose metabolism, detectable via FDG-PET, is an early hallmark of AD and occurs even at the stage of MCI [10], [12]. In contrast, the brain's ability to utilize ketone bodies—β-hydroxybutyrate and acetoacetate—appears relatively preserved (Fortier et al., 2020; Mentzelou et al., 2023). This "brain energy gap" provides a mechanistic rationale for interventions that elevate circulating ketones, such as MCT supplementation and ketogenic diets [10], [11], [12].

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3.4.2 MCT-Based Interventions

Henderson et al. (2009) conducted one of the earliest RCTs of an MCT-based ketogenic agent (AC-1202) in mild-to-moderate AD. The intervention improved ADAS-Cog scores in APOE ε4 non-carriers compared with placebo, though no significant benefit was observed in ε4 carriers [11]. Fortier et al. (2020) evaluated a multi-ingredient ketogenic drink rich in MCTs in older adults with MCI and found that 6-month supplementation improved several cognitive domains and increased brain ketone uptake on PET imaging. The magnitude of cognitive improvement correlated with circulating ketone levels [10].

3.4.3 Ketogenic Diets and Modified Ketogenic Protocols

Ketogenic diets, which restrict carbohydrate and increase fat to induce endogenous ketogenesis, have also been investigated in AD and MCI. Phillips et al. (2021) conducted a randomized crossover trial of a modified KD in AD patients, reporting improvements in daily functioning and quality of life, with trends towards better cognition. Nagpal et al. (2019) showed that a modified Mediterranean–ketogenic diet modulated the gut microbiome and short-chain fatty acids in MCI, with parallel beneficial changes in AD biomarkers.

However, strict KDs can be challenging, especially in frail older adults, and may pose risks such as weight loss, dyslipidemia, and gastrointestinal side effects [12], [16]. As a result, many clinicians favor moderate MCT supplementation integrated into a generally healthy diet rather than a highly restrictive KD.

3.4.4 Role in Geriatric Neuroprotection

In geriatric neuroprotection, pragmatic approaches include low-to-moderate MCT doses (e.g., 10–30 g/day) titrated according to tolerance and integrated into Mediterranean or MIND-style diets [1], [12]. Monitoring weight, metabolic parameters, and gastrointestinal symptoms is essential. APOE genotype, insulin resistance, and vascular comorbidities may help identify individuals who are most likely to benefit [10], [11], [12].

3.5 Integrative and Translational Perspectives

Nutrition-based neuroprotection is unlikely to depend on single nutrients in isolation. Instead, multiple dietary bioactives operate within interconnected physiological networks [1], [8].

Omega-3 PUFAs, flavonoids, B-vitamins, and ketone-based interventions converge on several shared pathways: reduction of neuroinflammation and oxidative stress, support of mitochondrial function and synaptic plasticity, improvement of endothelial function and cerebral perfusion, and optimization of metabolic and vascular health [1], [5], [6], [7], [9], [10].

Life-course timing is also critical. Adherence to cardioprotective dietary patterns in midlife is associated with lower dementia risk later in life [1], [2]. Yet, nutritional interventions can still yield benefits in late life, especially when they correct specific deficiencies (B12, folate, omega-3) or address brain energy deficits (MCTs, ketogenic strategies) in people with MCI [5], [7], [10].

Precision nutrition approaches—using biomarkers such as omega-3 index, tHcy, insulin resistance markers, and APOE genotype—are increasingly proposed to tailor multi-nutrient interventions to individual risk profiles [5], [7], [14]. Emerging work on gut–brain interactions further suggests that diet-induced changes in the microbiome and its metabolites may modulate neuroinflammation, amyloid processing, and synaptic function [8], [17].

3.6 Geriatric Practice Considerations

Implementing neuroprotective nutrition strategies in older adults requires careful consideration of comorbidities, polypharmacy, frailty, and socio-cultural contexts [1].

Polypharmacy can influence nutrient status and metabolism; for example, metformin and proton pump inhibitors are associated with vitamin B12 deficiency, and anticoagulant therapy may interact with high-dose omega-3 supplementation [7], [15]. Frail or sarcopenic older adults may be vulnerable to weight loss and muscle wasting with restrictive diets; thus, interventions must prioritize adequate protein and caloric intake [1].

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Dietary counselling should align with cultural food practices and economic realities, using affordable, locally available sources of omega-3 (e.g., local fish), flavonoids (e.g., tea, local fruits), and B-vitamins (e.g., leafy greens, fortified cereals, animal products) [13], [14], [19], [20]. In institutional settings, such as nursing homes, modifications to menus and fortified foods may help achieve neuroprotective nutrient intakes [8].

3.7 Future Directions

Future research priorities include large, long-duration RCTs of combined nutritional strategies embedded within multidomain interventions (e.g., diet, physical activity, vascular risk management, cognitive training), with incident dementia as a primary endpoint [1]. Trials should stratify participants by APOE genotype, omega-3 index, tHcy, metabolic status, and baseline diet quality, and explore interactions between these factors [5], [7], [9], [13].

Advanced omics technologies and microbiome analyses can clarify how diet modulates neurodegeneration pathways at the molecular level [8], [17]. Implementation science studies are needed to identify the most effective ways to deliver neuroprotective nutrition in geriatric clinics, primary care, and long-term care facilities.

4. Conclusion

Nutrition is a powerful, multi-target lever for dementia prevention and healthy cognitive aging. Evidence supports a synergistic role for marine omega-3 fatty acids, flavonoid-rich dietary patterns, adequate B-vitamin and homocysteine control, and ketone-based interventions in mitigating the neuropathological and vascular processes that underlie cognitive decline [1], [5], [6], [7], [9], [10], [11], [13].

Integrating these strategies into geriatric practice—tailored to individual biochemistry, genotype, and comorbidity profile—offers a promising route to delay dementia onset and preserve functional independence in older populations [1], [2]. As pharmacologic options remain limited, neuroprotective nutrition will likely become an increasingly central pillar of dementia prevention frameworks worldwide.

5. Declarations

5.1 Author contributions

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5.2 Ethics

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5.3 Conflict of Interest

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5.4 Funding Statement

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