The role of nutrition in the prevention of cognitive decline

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Purpose of review
Dementia is a growing concern and underscores the urgent need for effective preventive measures targeting modifiable risk factors. Nutrition is a key player in the onset and progression of inflammation and cognitive decline. This review provides a comprehensive overview of the effects of different dietary patterns, vitamins and nutrients for preventing cognitive decline, mainly among healthy individuals and those with mild cognitive impairment.

Recent findings
The Mediterranean diet, omega-3 long-chain polyunsaturated fatty acids and B vitamins are the most investigated, with evidence supporting protection against cognitive decline among older adults varying across studies. More recent interventions examined in this review, such as MIND Diet, are promising with positive results, but further research is needed to conclusively establish their efficacy. It is also crucial to consider complete lifestyle as physical activity for preventing cognitive decline.

Summary
Definitive conclusions are difficult to draw. Future studies should adopt a comprehensive approach and focus on multinutrient strategies and whole diets.

Keywords
anti-inflammatory, dementia, diet pattern, mild cognitive impairment, nutrients

INTRODUCTION

The number of people living with dementia is expected to rapidly increase in our ageing society, around 152 million in the world by 2050. This constitutes a significant health concern with vast medical, social, and economic implications [1]. Aside from the recent promising trial of lecanemab, which observed a modest reduction in cognitive decline compared with placebo in the early stages of Alzheimer's disease [2], there is currently no effective strategy to delay or reverse cognitive impairment. The most promising strategy to prevent cognitive decline is to target modifiable risk factors.

One of the key players in the mechanism of cognitive dysfunction is inflammation. Chronic low-grade inflammatory stress leads to an oxidative environment via cell-mediated immunity. These neuroinflammatory processes include amyloidosis, neuronal death, and neurodegeneration. Numerous studies emphasize the critical influence of diet on inflammation onset and progression. A Western diet with an excess of saturated fatty acids and simple sugars is now a known environmental risk factor for Alzheimer's disease (promote insulin resistance, neuroinflammation, deposition of insoluble proteins in the brain and induce microbiota dysbiosis) [3].

What role does nutrition play in preventing the emergence and progression of inflammation and cognitive decline? Current evidence supports certain dietary patterns (such as Mediterranean diet, DASH diet, the ketogenic diet and so on) and nutrient supplementation [omega-3 long-chain polyunsaturated fatty acids (omega-3 LC-PUFA), B-vitamins and antioxydants] in preventing cognitive decline, but with high heterogeneity between trials. This review summarizes our present

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KEY POINTS

- The Mediterranean DASH diet Intervention for Neurological Delay is a combination of the Mediterranean and DASH diets. This dietary pattern is particularly promising for reducing the risk of developing dementia.
- Ketogenic diet and dietary restriction require further investigation concerning their possible adverse effects in the elderly and long-term adherence.
- Given their good general tolerance and some positive results on global cognition, a diet rich in omega-3 long-chain polyunsaturated fatty acids and B-vitamin supplementation may be recommended.
- Research using an integrative approach, including physical activity, appears to have a more beneficial impact on cognitive function and warrants further exploration.

understanding of various nutritional interventions and their effects.

DIET PATTERNS

Mediterranean diet

The Mediterranean diet (MediDiet) has long been traditionally practiced by people in Mediterranean countries. It is based on high consumption of plant-based foods, low consumption of dairy products and red meat, and moderate consumption of wine. Fish and olive oil are the main source of fats. Although the underlying mechanisms are not understood, they include lowering cerebrovascular risk factors, reducing systemic inflammation and slowing rates of cerebral Aβ accumulation in older adults. Additionally, MediDiet also decreases the level of proinflammatory gut bacteria [3].

Meta-analyses show conflicting results (Table 1), high adherence to MediDiet appears to be associated with a lower risk of mild cognitive impairment (MCI) and Alzheimer's disease [4,5▪▪], and improvement in both working and episodic memory [5▪▪]. However, some recent meta-analyses have found no effect on global cognition and attention [5▪▪] and no significant association with MCI or Alzheimer's disease [6].

The evidence of the protective effects of MediDiet is inconsistent across different studies, and these conflicting results may be due to considerable variation in food quality within MediDiet among studies. The observed benefits could be attributed to the high intake of antioxidants, unsaturated fatty acids and vitamins, either individually or synergistically.

Dietary approaches to stop hypertension

The Dietary Approaches to Stop Hypertension (DASH) diet is recognized as an effective dietary pattern for lowering blood pressure. As with the MedDiet, the DASH diet recommends a high intake of plant-based foods. In contrast, the DASH eating plan places greater emphasis on low sodium intake and does not recommend alcohol consumption [3].

Most of the studies on DASH diet are observational studies (longitunal or cross-sectional) and concern primary prevention (no individuals with cognitive impairment) [7,8]. A limited number of studies have found that adherence to the DASH diet is associated with better cognitive function, a slower rate of global cognitive decline and a lower risk of Alzheimer's disease. In general, research has not identified a link between adherence and the DASH diet and cognitive performance or decline [7,8]. These studies have several limitations due to their design with self-reported food consumption and limited data from the food frequency questionnaire.

The DASH diet alone appears to offer no significant benefits for adults, indicating that it may need to be combined with other dietary patterns (Mediterranean diet) or nondietary interventions to produce a significant neuroprotective effect against cognitive decline.

Mediterranean-dash diet intervention for neurological delay

The Mediterranean-DASH diet Intervention for Neurological Delay (MIND) has been designed to protect the brain and prevent dementia. This diet is a combination of the MediDiet and the DASH diets, focusing on foods rich in components known for their neuroprotective properties (antioxidants, B vitamins, polyphenols, omega-3 LC-PUFA). A unique aspect of the MIND diet is its specific recommendations for consuming berries and green leafy vegetables [3].

A recent meta-analysis suggested that higher adherence to the MIND diet is associated with better cognitive function and potentially slower cognitive decline (Table 1) [9]. This is further supported by another meta-analysis, which demonstrates that adherence to the MIND diet is linked to a decreased risk of developing dementia in middle-aged and older individuals [10].

Although these results are promising, they are observational. Further research, with interventional studies, is needed to conclusively establish the efficacy of the MIND diet.

Ketogenic diet

The ketogenic diet is characterized by high fat and very low carbohydrate intake, which produces a

Table 1. Recent meta-analyses (2020 and later) examining the effects/associations of various dietary patterns, vitamins and nutrients on cognition and risk of mild cognitive impairment/Alzheimer's disease in healthy and mild cognitive impairment individuals

Healthy/MCI individuals	Cognition		Risk of MCI		Risk of AD/dementia	
MediDiet	*Obs* ± Coelho-Junior: 2021, lower decline in global cognition: RR = 0.26 (95% CI = 0.23–0.29)	*Obs* -	*Obs* ± Garcia-Casares: 2021, RR = 0.91 (95%CI = 0.85-0.97) Fu: 2022, RR = 0.75 (95% CI = 0.66–0.86)	*Obs* - Coelho-Junior: 2021, no significant association with MCI	*Obs* ± Garcia-Casares: 2021, RR = 0.89 (95% CI = 0.84-0.93) Fu: 2022, RR = 0.71 (95% CI = 0.56–0.89)	*Obs* - Coelho-Junior: 2021, no significant association with AD
	Int ± Fu[a]: 2022, improve episodic [SMD = 0.20 (95% CI: 0.09–0.30)] and working memories [SMD = 0.17 (95% CI: 0.01–0.32)]	*Int* - Fu[a]: 2022, no signficant improvement on global cognition and adverse effect on attention [SMD = -0.41 (95% CI: -0.67 to 0.16)]	*Int* ±	*Int* -	*Int* ±	*Int* -
MIND diet	*Obs* ± Huang: 2023, prospective study + meta analysis, 1 SD increment of MIND score = + 0.042 units (95% CI = 0.020-0.065) in global cognitive function z-score	*Obs* -	*Obs* ±	*Obs* -	*Obs* ± Chen: 2023, pooled HR = 0.83 (95% CI = 0.72–0.95)	Obs -
	Int ±	*Int* -	*Int* ±	*Int* -	*Int* ±	*Int* -
Caloric restriction/ Intermittent fasting	*Obs* ±	*Obs* -	*Obs* ±	*Obs* -	*Obs* ±	*Obs* -
	Int ± Lu[a]: 2023, dietary restriction = improve cognitive function (global + memory) (95% CI = 0.09–0.34, $P < 0.01$)	*Int* -	*Int* ±	*Int* -	*Int* ±	*Int* -
Omega-3 LC-PUFA	*Obs* ±	*Obs* -	*Obs* ± Zhu: 2020, RR = 0.86 (95% CI = 0.75–0.98) for omega-3 LC-PUFA intake	*Obs* -	*Obs* ± Wei: 2023, RR = 0.82 ($P = 0.001$) for DHA intake Kosti: 2022, 30% reduction (95% CI = 0.54–0.89) in AD risk for fish intake (up to 250 g per week)	*Obs* -
	Int ± Zhang: 2020, omega-3 LC-PUFA = improve global cognition (WMD = 0.85, 95% CI = 0.04–1.67) Alex: 2020, omega-3 LC-PUFA = benefits on memory function (Hedge's g = 0.31, $P = 0.003$) Brainard: 2020, effect of omega-3 LC-PUFA on MMSE (MD 0.10, 95% CI 0.03–0.16) Yang[a]: 2023, DHA or/and EPA = benefits on global cognition (SMD = 0.51, 95% CI = 0.12–0.91)	*Int* - Alex: 2020, omega-3 LC-PUFA = no effect on global cognitive function (Hedge's g = 0.02, 95% CI = -0.12–0.154) Kosti[a]: 2022, EPA + DHA = no significant improvement on general cognition	*Int* ±	*Int* -	*Int* ±	*Int* -
B vitamins	*Obs* ±	*Obs* - Zhang: 2021, no benefit in cognition for high B vitamins intake	*Obs* ±	*Obs* -	*Obs* ± Zhang: 2021, RR = 0.44 (95% CI = 0.18–0.71) for high folate intake Wang: 2022, HR = 0.61 (95% CI = 0.47–0.78) for high folate intake	*Obs* -

Table 1 (*Continued*)

Healthy/MCI individuals	Cognition		Risk of MCI		Risk of AD/dementia	
	Int ± Suh: 2020, improve global cognition (SMD = -0.18, 95% CI = -0.30 to -0.06) Li: 2021, improve global cognitive (SMD = 0.36, 95% CI = 0.18–0.54, $P < 0.01$) Wang[a]: 2022, improve cognitive function (MMSE) (MD = 0.14, 95%CI = 0.04–0.23)	*Int* – Behrens : 2020, no significant effect on cognitive function ($P = 0.39$) Markun : 2021, no significant effect on cognitive function of B12 alone Chang[a]: 2023, no significant effect on MMSE	*Int* ±	*Int* -	*Int* ±	*Int* -
Antioxidants	*Obs* ±	*Obs* -	*Obs* ±	*Obs* -	*Obs* ±	*Obs* – Wang : 2021, no significant association for vitamine E intake: RR = 0.81 (95% CI = 0.50–1.33)
	Int ± Tsai: 2021, curcumin = improve working memory ($P = 0.015$) De Vries : 2021, polyphenols = improve working and episodic memory ($P < 0.001$) Davinelli: 2021, carotenoids = improve cognitive functions (95% CI = 0.08–0.20, $P < 0.0001$) Hepsomali: 2021, polyphenols = improve rapid visual information processing speed ($P = 0.02$) Cheng[a]: 2022, dietary flavonoids = improve cognitive performance (g = 0.148, $P < 0.001$)	*Int* - Ammar: 2020, no significant effect of polyphenol rich diet on executive functions Khorshidi: 2021, no significant effect of RSV on cognitive and memory performance Morton: 2021, no significant effect of fruit derived polyphenol on executive function Pereira[a]: 2022, no significant effect of Se on cognitive function (MMSE & ADAS-Cog tests) Farag[a]: 2022, no significant effect of polyphenols on executive function	*Int* ±	*Int* -	*Int* ±	*Int* -
Vitamin D	*Obs* ±	*Obs* -	*Obs* ±	*Obs* -	*Obs* ±	*Obs* -
	Int ±	*Int* – Du: 2020, no significant difference in MMSE, verbal fluency, verbal memory	*Int* ±	*Int* -	*Int* ±	*Int* -

AD, Alzheimer's disease; ADAS-Cog, Assessment Scale-Cognitive subscale; ALA, Alpha-linolenic acid; CI, confidence interval; DHA, docosahexaenoic acid; EPA, eicosa pentaenoic acid; HR, hazard ratio; Int +/-, meta analysis on inteventionnal studies with positive/negative results; MCI, mild cognitive impairement; MediDiet, Mediterranean Diet; MIND, Mediterranean-Dietary Approaches to Stop Hypertension (DASH) Intervention for Neurodegenerative Delay; MMSE, Mini–Mental State Examination; n-3 LC-PUFAs, N-3 long-chain polyunsaturated fatty acids; Obs +/-, meta analysis on observationnal studies with positive/negative results; PUFA, Polyunsaturated fatty acids; RR, relative risk; RSV, Resveratrol; SD, standard deviation; Se, Selenium; SMD, standardized mean difference.

[a]Studies published in the last 18 months and with outstanding interest (large meta-analysis of RCTs).

metabolic switch and increases ketone body production. The brain uses ketone body as an alternate energy source, which offers neuroprotective benefits. This fact has been established since the 1920 s, and ketogenic diet is the first nutritional intervention successfully used for treating children with drug-resistant epilepsy. More recently, ketogenic diet seems promised for cognition. In animal studies, various mechanisms have been demonstrated: protection against Aβ neurotoxicity, decreased neuroinflammation, enrichment in beneficial gut microbiota and so on. Different types of KDs exist: the classical KD, the modified Atkins diet (MAD) and the MCT diet in which fats are provided with medium-chain triglyceride (MCT) intake [11].

Most clinical trials involved Alzheimer's disease patients and showed short-term cognitive improvements in the intervention groups. They mostly use ketogenic diet with MCT intake. Some studies focusing on patients with MCI have shown positive outcomes on cognitive evaluations. However, some studies involving Alzheimer's disease patients suggest that neurological effects depend on the apolipoprotein E4 (ApoE4) genotype. Although most studies are randomized controlled trials (RCTs), they are heterogeneous and limited due to the small sample size and short follow-up times. In addition, some studies report poor adherence because of gastrointestinal side effects [3,11].

Ketogenic diet is a promising way to improve cognitive function. Due to the difficulty in inducing ketogenesis through a sufficiently strict diet, MCT supplementation is recommended. Nevertheless, this could lead to a higher consumption of saturated fatty acids, potentially increasing the risk of inflammation and cardiovascular issues [11]. Furthermore, in elderly patients, possible concerns due to significant weight loss induced by ketogenic diet. Further large sample RCTs are neededto examine the adverse effects and adherence to the diet.

Other diet patterns

Dietary restrictions: caloric restriction and intermittent fasting

Caloric restriction diet aims at reducing daily caloric intake while ensuring sufficient intake of essential nutrients. Caloric restriction appears to have many positive properties notably anti-ageing effects. In particular, caloric restriction has shown neuroprotective effects in animal models (decreased neuroinflammation and accumulation of Aβ plaques and so on). In humans, some interventionnal studies in healthy adults found improvement in working memory, verbal memory, and spatial processing. However, one study found decreased cognitive flexibility and another found that a daily caloric deficit was not significantly associated with changes in cognitive performance [3].

These results seem interesting but are very heterogeneous. Adherence to long-term low-calorie diets seems inadequate, especially in older adults. As such, intermittent fasting, a diet pattern consisting of alternate periodic fasting and re-feeding, serves as a more natural and potentially more acceptable approach. Although numerous animal models for intermittent fasting exist, human studies are still limited. Interventional studies found that intermittent fasting upregulated proteins protective against Alzheimer's disease in healthy individuals and increases cognitive performance (both intermittent fasting and caloric restriction groups). One large longitudinal study found a better global cognitive scores for older patients with MCI who regularly practice intermittent fasting [3].

A meta-analysis of an RCT assessing the cognitive effect of dietary restriction (caloric restriction and intermittent fasting) found a significant positive effect on cognitive function in individuals with MCI, with no difference between caloric restriction and intermittent fasting (Table 1) [12▪▪]. Given these findings, caution is warranted, particularly among the older people who may be at risk of malnutrition. More robust research is essential to conclusively determine the cognitive efficacy of dietary restriction strategies.

Nordic diet

The conventional Nordic diet is based on ingredients from Scandinavia. It features a high intake of vegetables, fruits, berries, fish and whole grains, with limited to moderate consumption of meat and alcohol. The nordic diet is associated with reduced cardiovascular risk factors and a lower risk of ischemic stroke. A few observational studies have assessed the beneficial effects on cognition: high adherence to the Nordic Diet is associated with a lower risk of MMSE decline in nondemented adults. Additionally, an active lifestyle seems to amplify this diet's positive impact on cognitive function. However, these findings require further validation through large interventional studies [13].

VITAMINS AND NUTRIENTS

Omega-3 long-chain polyunsaturated fatty acids

Long-chain polyunsaturated fatty acids (LC-PUFA) are essential fatty acids with two main families:

(1) Omega-3 LC-PUFA: these include eicosapentaenoic acid (EPA) and docosahexaenoic acid

(DHA), commonly found in fish and fish oils, and alpha-linolenic acid (ALA), found in seeds and oils, green leafy vegetables, nuts, and beans.
(2) Omega-6 LC-PUFA: including linoleic acid, found in grains, meats and seeds of most plants.

The human brain is composed largely of fatty acids (60%, of which DHA accounts for 20–30%). Therefore, LC-PUFA are vital elements of neuronal cell membranes and help protect against neuronal injury. They play an important role in various aspects of the brain (synaptic transmission, neuroplasticity and so on). Notably, extensive research supports the hypothesis that omega-3 LC-PUFA limit neuroinflammation.

In an observational meta-analysis (Table 1), an increase in dietary fish intake has been associated with lower risks of dementia, particularly Alzheimer's disease [14▪▪]. A more recent meta-analysis suggested that higher levels of omega-3 LC-PUFA in the blood correlate with a reduced risk of cognitive decline. Moreover, consuming omega-3 LC-PUFA, particularly DHA, could reduce the overall risk of dementia or cognitive impairment by approximately 20% [15,16].

Some meta-analyses of interventional studies have reported significant benefits for individuals with Alzheimer's disease [17] and MCI [18,19▪▪] (Table 1). However, there is no supporting evidence that omega-3 LC-PUFA supplementation provides cognitive benefits for either older or younger nondemented individuals [14▪▪,20,21].

Despite mixed results and given their good general tolerance, a diet rich in omega-3 LC-PUFA combined with low saturated fat intake is recommended to lower the risk of cognitive decline (GRADE 1B), according to Canadian experts [22].

B-vitamins

B vitamins have been studied for their potential effect on cognitive function because of their role in homocysteine metabolism. Hyperhomocysteinemia is now considered a well established independent risk factor for cognitive impairment and dementia. Homocysteine is generated through the methylation of methionine and is removed from the body via two different routes. One pathway involves folate and vitamin B12, whereas the other depends on vitamin B6. In terms of diet, vitamin B6 can be found in foods such as grains, legumes, and nuts. Folate is abundant in leafy green vegetables, and vitamin B12 is primarily sourced from dairy, meat and other animal products. Several studies have examined the targeted administration of folic acid (vitamin B9), vitamin B6 and vitamin B12 for their ability to lower homocysteine levels and their effect on antioxidative stress, DNA methylation and expression of both β-secretase and γ-secretase. Nevertheless, the direct impact of B vitamins on cognitive function remains inconclusive.

Folate deficiency increases the risk of Alzheimer's disease, whereas sufficient intake of folate is a protective factor against Alzheimer's disease according to observational meta-analyses (Table 1) [23▪▪,24].

Numerous interventional studies have investigated the influence of B vitamin supplement intake on cognitive function, cognitive decline and Alzheimer's disease. Some meta-analyses have indicated a significant beneficial effect of B-vitamins on global cognitive function [23▪▪,25], particularly with supplementation periods exceeding 3 months [26]. However, other meta-analyses have reported no positive effects (Table 1) [27,28▪▪,29]. There could be a more favourable effect of folates compared with vitamins B12 and B6.

In 2018, an international consensus emphasized that the public health impact of elevated homocysteine levels in older adults should consider B vitamin supplementation as a well tolerated, simple and inexpensive way to lower homocysteine levels [30].

Antioxidants

Numerous nutrient such as vitamin C, vitamin E and carotenoids, along with nonnutrients such as polyphenols, have antioxidant capabilities. In addition, essential minerals such as selenium, zinc and copper act as cofactors in antioxidative enzymes or proteins. These antioxidants prevent the propagation and formation of free radicals by different mechanisms. The brain is particularly vulnerable to oxidative damage caused by free radicals. The risk of such oxidative stress increases with age and is a significant risk factor for age-associated cognitive decline. This relationship has been investigated especially in the case of Alzheimer's disease. Many studies have explored the association between cognitive function and antioxidant supplementation [31].

Some meta-analyses have been recently published, particularly concerning polyphenols, but with negative [32▪▪,33] or moderately positive results [34–36], mainly in healthy and MCI individuals (Table 1). Resevratrol (RSV), a natural polyphenol present in a variety of plants and plant extracts (including grapes, red wine and blueberries), has shown promising results in animal models but not yet confirmed in human [37]. A meta-analysis reported no significant effect of RSV on cognitive outcomes in human clinical trials [37]. Flavonoids are also a subtype of polyphenols found in foods or supplements (berries, cacoa, tea, ginkgo biloba and so on), whose beneficial effects on cognitive

performance have been demonstrated by a recent, in-depth meta-analysis [38▪▪]. Curcumin, another type of polyphenol, also appears to be associated with improvements in working memory in a meta-analysis [39].

Concerning other antioxidants: no definitive conclusions can be made at this time on α-tocopherol (vitamin E) because of the lack of association between vitamin E supplementation and Alzheimer's disease [40]. Concerning carotenoids, a recent meta-analysis showed a statistically significant improvement in cognitive functions after the administration of carotenoids in healthy individuals [41]. Regarding selenium supplementation (alone or with other nutrients) in Alzheimer's disease and MCI individuals, some cognitive improvements were observed but were not statistically significant according to the meta-analysis findings (Table 1) [42▪▪].

The significance of the interventions is limited, and these contrasting results may be partly due to the heterogeneity of the studies (various types and dosages of antioxidants used). Some authors suggest that the beneficial effects of polyphenols depend on the ingested dose and bioavailability. Large RCTs are essential for definitive conclusions.

Vitamin D

Vitamin D appears to have a significant role in normal brain physiology through its receptor, which is present in the neurons and glial cells of key brain areas such as the hippocampus. Emerging data suggest that low concentrations of Vitamin D are associated with dementia (Vitamin D reducing Alzheimer's disease's hallmarks such as amyloid beta and phosphorylated tau). Sun exposure is the primary source of Vitamin D, which can also be obtained through dietary sources such as fish products, eggs and certain fats and oils [43].

While observational studies indicate a correlation between low levels of vitamin D and decreased cognitive abilities, interventional studies have shown no evidence for a beneficial effect of vitamin D supplementation on cognitive performance, even among those with vitamin D deficiencies (Table 1) [44].

A recent Cochrane meta-analysis did not find evidence that Vitamin D supplementation prevents cognitive decline or dementia in cognitively healthy adults [45].

CONCLUSION

Caloric intake and diet composition appear to be essential factors for cognitive health. Increasing evidence points to the beneficial effects of specific dietary patterns (like the Mediterranean Diet and the promising MIND diet) and certain nutrients (like fish rich in omega-3 LC-PUFA and B-vitamins: particularly folates) in reducing the risk of age-related cognitive decline and Alzheimer's disease.

Nonetheless, the outcomes of observational and interventional studies are not always consistent, and there is significant heterogeneity between studies. Additionally, single nutrients might not be powerful enough to produce clinically meaningful changes, suggesting that future studies should focus on multinutrient strategies and whole diets. It is crucial to view eating habits not only as a means of nutrient acquisition but also as a central part of a lifestyle aimed at sustaining both physical and mental well being. In this context, physical activity and sleep patterns should also be integrated into a complete lifestyle in a holistic approach.

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Conflicts of interest

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Papers of particular interest, published within the annual period of review, have been highlighted as:

- ▪ of special interest
- ▪▪ of outstanding interest

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