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The MIND Diet and Cognitive Health in Older Adults: Clinical Evidence, Neuroprotective Mechanisms and Research Gaps—A Narrative Review

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Abstract

Background. The MIND diet combines elements of the Mediterranean and DASH dietary patterns and was developed to support cognitive health. Evidence includes observational, randomised and experimental research.

Aim. This narrative review evaluated associations between MIND diet adherence and cognitive outcomes in older adults and compared observational and randomised findings.

Methods. PubMed, MEDLINE and Google Scholar were searched through August 2026. Studies addressing cognitive performance, cognitive decline, mild cognitive impairment or

dementia were included. Experimental studies were used to assess biological plausibility. Findings were synthesised descriptively.

Results. Most observational studies associated greater adherence with better global cognition and memory, slower cognitive decline and lower risks of cognitive impairment and dementia. Experimental evidence supports vascular, metabolic, antioxidant, anti-inflammatory, amyloid-related and gut–brain mechanisms. The principal three-year randomised trial found no significant cognitive or neuroimaging advantage over an active control. Both groups received dietary counselling and caloric restriction and experienced weight loss, while the intervention may have been too short to detect long-term effects. Secondary analyses suggested possible differences according to obesity and achieved adherence, but were exploratory.

Conclusions. The MIND diet is consistent with established cardiometabolic recommendations and can be a part of a broader healthy-ageing strategy. Its specific ability to prevent cognitive decline or dementia remains unconfirmed. Longer randomised trials are required.

Key words: MIND diet; Cognitive Dysfunction; Dementia; Alzheimer Disease.

1. Introduction

Cognitive impairment and dementia are major challenges associated with population ageing. Because opportunities for effective treatment remain limited, increasing attention has been directed towards modifiable lifestyle factors that may support cognitive health. Diet is particularly relevant because it may affect the brain through vascular, metabolic, inflammatory, and oxidative pathways. Dietary patterns may be more informative than individual nutrients because foods are consumed in combination and may produce complementary biological effects.

The Mediterranean-DASH Intervention for Neurodegenerative Delay (MIND) diet was developed specifically to promote healthy brain ageing. It combines selected principles of the Mediterranean and DASH diets while placing additional emphasis on foods considered potentially neuroprotective. The diet encourages the consumption of green leafy vegetables, other vegetables, berries, nuts, legumes, whole grains, fish, poultry, and olive oil. Conversely, it recommends limiting red meat, butter and margarine, cheese, pastries and sweets, and fried

or fast food. Adherence is generally assessed using a 15-component score consisting of 10 recommended and five restricted food groups.

The potential benefits of the MIND diet may result from its high content of unsaturated fatty acids, fibre, vitamins, minerals, polyphenols, and other antioxidant and anti-inflammatory compounds. At the same time, limiting foods rich in saturated fat, added sugar, and refined carbohydrates may support cardiovascular and metabolic health, which are closely related to cognitive ageing. Initial observational research associated greater adherence with slower cognitive decline and a lower incidence of Alzheimer's disease [1, 2]. However, the results of subsequent studies, particularly randomised trials, have not been uniformly consistent, leaving uncertainty regarding the extent to which the MIND diet directly protects against cognitive decline.

This narrative review aimed to critically evaluate whether adherence to the MIND diet is associated with preserved cognitive function and a lower risk of cognitive decline in older adults, and whether findings from observational studies are consistent with randomised evidence.

2. Materials and methods

A narrative literature review was conducted using PubMed, MEDLINE and Google Scholar databases with the final search performed in August 2026. Search terms included combinations of “MIND diet”, “Mediterranean-DASH Intervention for Neurodegenerative Delay”, “cognitive function”, “cognitive decline”, “mild cognitive impairment”, “dementia”, “Alzheimer's disease”, “older adults” and “randomized controlled trial”. Reference lists of relevant articles were also screened to identify additional sources.

English-language human studies evaluating the relationship between MIND diet adherence or intervention and cognitive outcomes in middle-aged or older adults were included. Observational studies, randomised controlled trials and secondary or post hoc analyses of eligible trials were considered. Conference abstracts, editorials, and publications unrelated to cognitive outcomes were excluded. The most up-to-date available evidence identified by the final search was used to present the current state of knowledge regarding the MIND diet and cognitive health.

The included studies were compared according to their design, population, duration, dietary assessment, cognitive outcomes, and principal findings. Because of methodological heterogeneity, the evidence was synthesized descriptively, and no meta-analysis was performed.

2.1. Inclusion criteria

Clinical evidence

1. Human studies involving adults aged ≥ 60 years or populations with a mean age ≥ 60 years.
2. Participants without diagnosed dementia at baseline.
3. Studies evaluating a defined MIND diet score or MIND diet intervention.
4. Cross-sectional, longitudinal, prospective cohort or randomised controlled studies.
5. Studies reporting validated cognitive, mild cognitive impairment or dementia outcomes.
6. Peer-reviewed original articles published in English from January 2015 to the final search date.

Supporting evidence

7. Peer-reviewed original human, animal or cellular studies investigating the MIND diet, its constituent foods, nutrients or bioactive compounds, or closely related Mediterranean or DASH dietary patterns.
8. Studies directly examining mechanisms relevant to neuroprotection, including oxidative stress, neuroinflammation, vascular and blood–brain barrier function, amyloid or tau pathology, synaptic function and the microbiota–gut–brain axis.
9. Mechanistic studies published in English from database inception to the final search date.

Mechanistic studies were used exclusively to describe biological plausibility and were not included in the synthesis of clinical effectiveness or in comparisons between observational studies and randomised controlled trials.

3. The MIND Diet: Origin and Components

The Mediterranean–DASH Intervention for Neurodegenerative Delay (MIND) diet was developed by Martha Clare Morris and colleagues at Rush University Medical Center as a dietary pattern specifically intended to support cognitive health during ageing [1, 2]. It combines selected elements of the Mediterranean diet and the Dietary Approaches to Stop Hypertension (DASH) diet, both of which have demonstrated cardiovascular benefits and have been associated with favourable cognitive outcomes. Unlike its parent dietary patterns, the MIND diet prioritises foods and nutrients supported by evidence concerning neuroprotection and dementia prevention [1, 2].

The MIND diet was initially developed from findings concerning the relationships between dietary components and cognitive decline, Alzheimer’s disease and brain pathology [1, 2]. It places particular emphasis on green leafy vegetables and berries, which are not separately prioritised in the traditional Mediterranean or DASH dietary scores. It also differs from the Mediterranean diet by not requiring high overall fruit consumption, frequent fish consumption or multiple daily servings of dairy products. Consequently, the MIND diet may be less restrictive and potentially easier to follow than some other healthy dietary patterns.

3.1. Components of the MIND Diet

The original MIND diet score consists of 15 dietary components: ten food groups considered favourable for brain health and five food groups considered unfavourable. The brain-healthy components include green leafy vegetables, other vegetables, berries, nuts, whole grains, legumes, fish, poultry, olive oil and wine. The components to be limited include red meat, butter and stick margarine, cheese, pastries and sweets, and fried or fast food [1, 2].

Each component is generally assigned a score of 0, 0.5 or 1, depending on the frequency of consumption. Scores for the 15 components are summed to produce a total ranging from 0 to 15, with higher scores indicating greater adherence. Favourable food groups receive higher scores with increasing consumption, whereas unfavourable groups receive higher scores with lower consumption [1, 2].

The inclusion of wine reflects the structure of the original MIND score rather than a recommendation that non-drinkers should begin consuming alcohol. Contemporary studies sometimes omit alcohol, modify its scoring or replace the original component when adapting

the score to local dietary practices and current health recommendations. Such modifications should be reported clearly because they may reduce comparability between studies.

Table 1. Principal components and intake targets of the original MIND diet

Food group	Dietary target	Selected nutrients and bioactive compounds
Green leafy vegetables	At least 6 servings per week	Lutein, zeaxanthin, folate, phylloquinone, α -tocopherol, nitrate and kaempferol
Other vegetables	At least 1 serving per day	Vitamin C, carotenoids, folate, fibre and diverse polyphenols
Berries	At least 2 servings per week	Anthocyanins, flavanols, ellagic acid and vitamin C
Nuts	At least 5 servings per week	α -Tocopherol, unsaturated fatty acids, α -linolenic acid, magnesium, L-arginine and polyphenols
Legumes	More than 3 servings per week	Folate, fibre, resistant starch, magnesium, plant protein and polyphenols
Whole grains	At least 3 servings per day	Fibre, B vitamins, vitamin E, magnesium, selenium, phenolic acids and phytosterols
Fish (not fried)	At least 1 serving per week	Docosahexaenoic acid (DHA), eicosapentaenoic acid (EPA), vitamin D, selenium and high-quality protein
Poultry	At least 2 servings per week	High-quality protein, niacin, pyridoxine, cobalamin, selenium and zinc

Olive oil	Used as the principal culinary oil	Oleic acid, α -tocopherol, hydroxytyrosol, oleocanthal and oleuropein derivatives
Wine	Approximately 1 glass per day in the original score	Polyphenols such as resveratrol, quercetin and anthocyanins; alcohol should not be recommended for cognitive protection
Red meat and meat products	Fewer than 4 servings per week	Protein, cobalamin, iron and zinc; intake is limited because some products contain substantial saturated fat, sodium and preservatives
Butter and stick margarine	Less than 1 tablespoon per day	Butter contains vitamin A, while some margarines provide vitamin E and phytosterols; intake is limited because of saturated or trans-fat content
Cheese	Fewer than 1 serving per week	Protein, calcium, cobalamin and phosphorus; intake is limited because many cheeses contain substantial saturated fat and sodium
Pastries and sweets	Fewer than 5 servings per week	No consistent neuroprotective constituents; some products may contain cocoa polyphenols or nuts, but are generally rich in refined sugar and saturated fat
Fried or fast food	Fewer than 1 serving per week	No consistent neuroprotective constituents; nutritional content depends on the ingredients, but these foods are often rich in

		sodium, saturated fat and oxidised lipids
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Table notes: Original dietary targets were adapted from the 15-component MIND diet score developed by Morris et al. [1]. Nutrient and fatty-acid profiles were compiled from USDA FoodData Central [3], while representative polyphenols were identified using the Phenol-Explorer database [4, 5]. Compounds listed for green leafy vegetables were additionally based on the analysis by Morris et al. [6]. The listed constituents are representative rather than exhaustive, and their presence should not be interpreted as proof that individual compounds mediate cognitive benefits. Wine is included only because it formed part of the original MIND score; it was omitted from the randomised MIND intervention and should not be considered a clinical recommendation [7].

3.2. Variability in MIND Diet Measurement

Although the original score contains 15 components, its implementation varies across studies. Some food-frequency questionnaires do not include sufficient information about berries, olive oil, wine or fried foods, resulting in modified scores with fewer components or population-specific intake thresholds. Dietary traditions may also affect the interpretation of the original recommendations. For example, olive oil and berries may be consumed more frequently in some populations than in others, while standard portion sizes may differ between countries.

Most observational studies assess adherence using self-reported food-frequency questionnaires. This approach is susceptible to recall error, misclassification and changes in dietary habits over time. Differences in dietary assessment, scoring thresholds and frequency of measurement may therefore contribute to inconsistent findings across studies. These methodological variations should be considered when comparing observational cohorts with each other and with randomised dietary interventions.

4. Potential Neuroprotective Mechanisms

The potential neuroprotective effects of the MIND diet are unlikely to result from a single nutrient or biological pathway. Instead, the dietary pattern may influence cognitive ageing through several interacting vascular, metabolic, inflammatory and neurodegenerative mechanisms. The MIND diet is rich in vegetables, berries, nuts, legumes, whole grains, fish and olive oil, while limiting foods containing high amounts of saturated fat, refined

carbohydrates and sodium. This combination provides fibre, unsaturated fatty acids, vitamins, minerals, carotenoids and polyphenols that may collectively support brain health. Although direct human evidence that the complete MIND diet engages these pathways in the ageing brain remains limited—with most findings derived from cellular and animal models or from studies of individual nutrients, related dietary patterns, and other clinical populations—the underlying molecular mechanisms are well characterised and provide a biologically coherent basis for the pathways discussed below.

4.1. Oxidative stress and antioxidant defence

The brain is particularly vulnerable to oxidative injury because it consumes large quantities of oxygen, contains abundant oxidation-sensitive polyunsaturated fatty acids, and requires continuous mitochondrial adenosine triphosphate production. Reactive oxygen species are generated when electrons escape from mitochondrial respiratory-chain complexes I and III and incompletely reduce molecular oxygen: $O_2 + e^- \rightarrow O_2^{\bullet-}$ [8]. Superoxide is also produced by NOX2 in activated microglia, while inducible nitric oxide synthase generates nitric oxide during inflammation. Superoxide dismutases convert superoxide into hydrogen peroxide [9], which can react with labile ferrous iron through the Fenton reaction:

$Fe^{2+} + H_2O_2 \rightarrow Fe^{3+} + OH^- + \bullet OH$ [10]. The resulting hydroxyl radical initiates peroxidation of polyunsaturated fatty acids in neuronal, mitochondrial, and myelin membranes. Superoxide may also react with nitric oxide to form peroxynitrite, which nitrates tyrosine residues and impairs mitochondrial proteins. Lipid peroxidation produces reactive aldehydes, particularly 4-hydroxynonenal, malondialdehyde, and acrolein. These compounds form covalent adducts with cysteine, histidine, and lysine residues in proteins, disrupting respiratory-chain complexes, ion pumps, membrane transporters, and synaptic proteins. Oxidative injury can also generate 8-oxo-7,8-dihydroguanine, mitochondrial DNA damage, and DNA strand breaks. Several MIND diet components may reduce these processes. α -Tocopherol from nuts, olive oil, seeds, and green leafy vegetables acts as a lipid-soluble chain-breaking antioxidant that limits the propagation of membrane lipid peroxidation. Ascorbic acid from berries and vegetables can regenerate α -tocopherol and contributes to antioxidant protection in aqueous compartments. Lutein and zeaxanthin from spinach, kale, and other leafy vegetables can quench singlet oxygen and reduce lipid-peroxide accumulation [11]. Anthocyanins from berries, including cyanidin-3-O-glucoside, and olive-oil phenols such as hydroxytyrosol and oleuropein derivatives may neutralise reactive radicals, bind redox-active metals, and activate the nuclear factor erythroid 2-related factor 2—antioxidant response element pathway [12, 13]. This pathway increases the

expression of heme oxygenase 1, NAD(P)H quinone dehydrogenase 1, and enzymes required for glutathione synthesis. Selenium from fish, poultry, whole grains, legumes, and nuts supports glutathione peroxidase 4, which removes phospholipid hydroperoxides and limits iron-dependent ferroptosis [14]. Zinc, copper, and manganese from nuts, legumes, whole grains, and vegetables serve as cofactors for cytosolic superoxide dismutase 1 and mitochondrial superoxide dismutase 2 [9]. These mechanisms have been demonstrated mainly in cellular and animal models and therefore provide biological plausibility rather than proof of protection in humans.

4.2. Modulation of Neuroinflammation

Persistent activation of microglia and astrocytes contributes to neuronal and synaptic injury during cognitive ageing and neurodegeneration [15]. Amyloid- β , damaged cellular components, circulating inflammatory signals, and metabolic abnormalities can activate Toll-like receptor 4 and other pattern-recognition receptors on microglia. Signalling through myeloid differentiation primary response 88 promotes nuclear translocation of nuclear factor kappa B, increasing transcription of tumour necrosis factor- α , interleukin-6, pro-interleukin-1 β , cyclooxygenase-2, inducible nitric oxide synthase, and NOX2. A second signal may activate the NLR family pyrin domain-containing 3 inflammasome, leading to caspase-1 activation and cleavage of pro-interleukin-1 β and pro-interleukin-18 into their biologically active forms [16]. Sustained production of these cytokines, prostaglandins, nitric oxide, and reactive oxygen species can impair the blood–brain barrier, alter glutamate homeostasis, reduce synaptic plasticity, and promote neuronal dysfunction [15]. MIND diet components may influence these pathways at several levels. Anthocyanins from berries and hydroxytyrosol and oleuropein derivatives from extra-virgin olive oil have reduced nuclear factor kappa B and NLR family pyrin domain-containing 3 signalling in experimental models [17–20]. Cyanidin-3-O-glucoside has also been shown to promote a less inflammatory microglial phenotype through peroxisome proliferator-activated receptor- γ signalling and to enhance microglial clearance of amyloid- β in experimental systems [21]. Eicosapentaenoic and docosahexaenoic acids from fatty fish are incorporated into neuronal membranes and serve as precursors of specialised pro-resolving mediators, including resolvins, maresins, and neuroprotectin D1 [22]. The MIND diet may support a less inflammatory neural environment through the combined actions of polyphenols, omega-3 fatty acids, antioxidant micronutrients, and fermentable fibre, but direct evidence that these mechanisms mediate cognitive benefits in humans remains limited.

4.3. Vascular and Cardiometabolic Effects

Cerebrovascular disease frequently coexists with neurodegenerative pathology and contributes substantially to cognitive impairment in older adults [23]. Hypertension, diabetes, obesity, dyslipidaemia and stroke are recognised risk factors for cognitive decline and dementia [24]. Because the MIND diet incorporates elements of the Mediterranean and DASH diets, part of its potential cognitive benefit may arise through improved cardiovascular and metabolic health [25].

The diet is low in saturated fat and encourages foods rich in unsaturated fatty acids, fibre, potassium and antioxidant compounds [1]. These characteristics may improve blood pressure, lipid profiles, insulin sensitivity and endothelial function [25, 26]. Better vascular health may preserve cerebral perfusion, reduce small-vessel disease and lower the risk of clinical and subclinical stroke [23, 27].

This pathway is particularly relevant when interpreting observational evidence because cardiovascular health may function both as a mediator and as a confounding factor [28]. Individuals with high MIND diet adherence frequently also engage in more physical activity, smoke less and have better access to preventive healthcare [28, 29]. Statistical adjustment may not fully separate the dietary effect from the influence of these accompanying behaviours [28].

4.4. Amyloid and Tau Pathology

β -Amyloid plaques and phosphorylated tau tangles are defining neuropathological features of Alzheimer's disease. Direct human evidence linking the MIND diet with these abnormalities was provided by Agarwal et al. [30] in a clinicopathological study of 581 deceased participants from the Rush Memory and Aging Project. Participants completed validated food-frequency questionnaires during life and consented to brain donation. Their mean age was 84.2 years at the first dietary assessment and 91.0 years at death, with an average follow-up of 6.8 years.

Following death, the researchers performed standardised brain autopsies and directly quantified cortical β -amyloid load and phosphorylated tau-tangle density. They also calculated a global Alzheimer's disease pathology score incorporating diffuse plaques, neuritic plaques, and neurofibrillary tangles. After adjustment for age at death, sex, education, total energy intake, and genetic risk, greater adherence to the MIND diet was associated with a lower global burden of Alzheimer's disease pathology and a lower β -amyloid load. In contrast, the association with

phosphorylated tau tangles was not statistically significant. These findings indicate that the relationship between MIND diet adherence and overall Alzheimer's disease pathology was driven primarily by differences in β -amyloid deposition. Participants in the highest tertile of green leafy vegetable consumption also had significantly less global Alzheimer's disease pathology than those in the lowest tertile [30].

The prospective assessment of diet followed by direct post-mortem examination represents an important strength of this study. Nevertheless, the results remain observational and cannot demonstrate that the MIND diet prevents β -amyloid accumulation or promotes its clearance. Dietary intake was self-reported, residual confounding remains possible, and participants who underwent brain autopsy may not represent the general older population. The study therefore provides neuropathological support for an association between MIND diet adherence and lower cerebral β -amyloid burden, but not definitive evidence of a causal effect. The lower post-mortem β -amyloid burden [30] should be described as an observational association supported by plausible mechanisms.

Experimental evidence suggests that selected components of the MIND diet may influence β -amyloid production, aggregation, and clearance. Oleocanthal from extra-virgin olive oil has increased P-glycoprotein- and LRP1-associated transport of β -amyloid across the blood–brain barrier in endothelial-cell and mouse models [31]. Oleuropein aglycone, present in olive oil, has activated autophagy through the Ca^{2+} –CaMKK β –AMPK pathway and mTOR inhibition, potentially facilitating the intracellular removal of aggregated proteins [32]. Bilberry anthocyanins have modified β -amyloid fibril formation and toxicity in experimental models, although they have not consistently reduced total cerebral deposits [33]. DHA has promoted non-amyloidogenic Amyloid Precursor Protein (APP) processing in cellular models [34], while DHA and EPA have enhanced A β_{42} phagocytosis by an immortalised human microglial cell line [35]. These findings provide mechanistic plausibility but do not demonstrate that consuming the MIND diet produces equivalent effects or lowers cerebral β -amyloid burden in humans.

4.5. The Gut–Brain Axis

The microbiota–gut–brain axis is a bidirectional communication network through which the intestinal microbiota may influence cerebral function via neural, metabolic, immune and endocrine pathways. Microbial fermentation of dietary fibre produces short-chain fatty acids,

particularly acetate, propionate and butyrate, which can affect intestinal epithelial integrity, systemic inflammation, blood–brain barrier permeability and microglial maturation. In germ-free mice, colonisation with normal intestinal microbiota reduced blood–brain barrier permeability and increased the expression of the tight-junction proteins occludin and claudin-5 [36], while microbial short-chain fatty acids restored microglial maturation and function [37]. Gut microorganisms also metabolise dietary tryptophan into aryl hydrocarbon receptor agonists that can regulate astrocyte-mediated neuroinflammation [38]. Neural communication occurs partly through vagal afferents, as vagotomy abolished the neurochemical and behavioural effects of *Lactobacillus rhamnosus* in mice [39]. Conversely, autonomic and hypothalamic–pituitary–adrenal signalling can modify intestinal motility, permeability and microbial composition. Evidence also links the intestinal microbiota with Alzheimer’s disease pathology. Germ-free APPPS1 mice developed less cerebral β -amyloid pathology than conventionally colonised animals, whereas microbial colonisation increased cerebral β -amyloid levels [40]. In humans with cognitive impairment, brain amyloidosis was associated with a greater abundance of pro-inflammatory intestinal bacteria and higher circulating inflammatory markers [41]. Moreover, transplantation of microbiota from patients with Alzheimer’s disease into microbiota-depleted rats impaired memory-related behaviour and hippocampal neurogenesis [42]. Diet may modify these pathways: a one-year Mediterranean-diet intervention altered the intestinal microbiome of older adults, and preservation of specific microbial taxa was associated with lower frailty and more favourable cognitive outcomes [43]. Foods promoted by the MIND diet, including whole grains, legumes, vegetables, berries and nuts, provide fermentable fibre and polyphenols that may support beneficial microbial metabolism. Nevertheless, it has not been demonstrated that the MIND diet preserves cognition specifically through modification of the gut microbiota. Current findings therefore support the microbiota–gut–brain axis as a biologically plausible contributor to neuroprotection rather than a clinically established causal mechanism.

4.6. Integrated Interpretation

The proposed mechanisms are interconnected. Improved cardiometabolic health may preserve cerebral blood flow and reduce vascular injury, while antioxidant and anti-inflammatory effects may limit neuronal damage. At the same time, bioactive dietary compounds may influence amyloid processing, synaptic function and cognitive resilience. The combined action of these pathways could theoretically produce greater benefits than supplementation with a single nutrient.

Nevertheless, evidence of biological plausibility should not be interpreted as proof of clinical effectiveness. Associations between individual dietary components, biomarkers and cognitive outcomes remain vulnerable to confounding and reverse causation. The proposed mechanisms support the rationale for further investigation but do not, by themselves, establish that the MIND diet prevents cognitive decline.

5. Why Do Observational and Randomised Findings Differ?

5.1. Evidence from Observational Studies

Observational studies have generally associated greater adherence to the MIND diet with better cognitive outcomes, although their findings have not been entirely consistent. In the Memory and Aging Project, participants with the highest MIND diet scores experienced slower cognitive decline over an average follow-up of 4.7 years; the difference between the highest and lowest adherence tertiles was estimated to be equivalent to 7.5 years of younger cognitive age [1]. In a related analysis, high adherence was associated with a 53% lower incidence of Alzheimer's disease compared with low adherence, while moderate adherence was associated with a 35% lower incidence [2]. The PATH Through Life study similarly reported that higher MIND diet adherence was associated with lower odds of developing cognitive impairment over 12 years [44]. Evidence from larger cohorts has also been broadly supportive. An analysis of the Whitehall II Study, Health and Retirement Study and Framingham Offspring Study found that every three-point increase in the MIND diet score was associated with a 17% lower risk of incident dementia [45].

Other studies identified associations with cognitive level rather than subsequent decline. Berendsen et al., analysing data from the Nurses' Health Study, found that long-term MIND diet adherence was associated with better verbal memory, but not with changes in global cognition, verbal memory or Telephone Interview for Cognitive Status scores over six years [46]. Similarly, the Framingham Heart Study found associations with better cognitive performance and larger total brain volume but not with the subsequent rate of cognitive decline [47]. In the Rotterdam Study, higher adherence was associated with a lower risk of dementia during the initial years of follow-up, but the association weakened or disappeared over longer follow-up periods, raising the possibility of reverse causation or residual confounding [28].

More recent findings have not resolved the discrepancy between associations with cognitive level and associations with cognitive change. Using an independent sample of older adults from the Health and Retirement Study and a comparable six-year observation period, Seago et al. reached a conclusion similar to that of Berendsen et al.: greater MIND diet adherence was associated with better cognition at baseline but not with a significantly slower subsequent rate of decline [48]. Consistent with the association with cognitive level, a 2026 cross-sectional analysis of 2,354 older adults from the National Health and Nutrition Examination Survey, with a mean age of 68.9 years, associated higher MIND diet scores with better memory and global cognitive performance [49]. However, the association differed according to race: higher scores were associated with better global cognition among White participants but with worse global cognition among Black participants. These findings require cautious interpretation because of the cross-sectional design and observed socioeconomic and dietary differences between groups [49]. Further longitudinal evidence has examined culturally adapted MIND scores and changes in dietary adherence over time. In a 10-year study of 8,326 participants from the Chinese Longitudinal Healthy Longevity Survey, high adherence to a 12-component Chinese-adapted MIND diet was associated with a lower risk of incident cognitive impairment than low adherence (HR = 0.79, $p < 0.001$) [50]. The use of an adapted score may limit direct comparison with studies employing the original 15-component MIND score. In the REGARDS cohort, changes in dietary intake were evaluated between two assessments among 7,555 participants. Each one-point increase in the MIND diet score was associated with a 10% lower subsequent risk of cognitive impairment (HR = 0.90, 95% CI: 0.85–0.96) [51]. This finding supports the potential relevance of dietary improvement over time rather than adherence measured at a single baseline assessment, although its observational design does not establish causality.

Evidence concerning interactions between diet, brain pathology and cognition remains preliminary. In a cross-sectional study of 66 adults aged 60–82 years, higher MIND diet scores were associated with better memory performance [52]. Greater white-matter hyperintensity volume and lower cortical volume were associated with worse cognition among participants with low MIND adherence, whereas these relationships were attenuated among those with mean or high adherence. No corresponding interaction was identified for hippocampal volume [52]. The small sample and cross-sectional design make these findings hypothesis-generating, but they suggest that MIND diet adherence may modify cognitive resilience to cerebrovascular and cortical pathology.

Overall, observational findings support an association between MIND diet adherence and favourable cognitive outcomes, but the strength and persistence of this relationship vary according to population, follow-up duration, outcome definition and participant characteristics. Recent studies suggest that culturally adapted scores and improvements in adherence over time may also be relevant [50, 51]. However, associations with cognitive performance measured at a single assessment remain more consistent than evidence of slower longitudinal cognitive decline.

5.2. Evidence from Randomised Trials and Secondary Analyses

Randomised evidence is dominated by the three-year MIND Diet Trial conducted by Barnes et al., which enrolled 604 cognitively unimpaired adults aged 65 years or older who were overweight, consumed a suboptimal diet and had a family history of dementia. Participants were assigned to either a structured MIND diet intervention with mild caloric restriction or a control diet with the same caloric restriction. Both groups received dietary counselling and behavioural support, achieving significant weight loss in the final assessment. Global cognition improved by 0.205 standardised units in the MIND group and by 0.170 units in the control group, producing a small, statistically non-significant between-group difference of 0.035 units. Changes in hippocampal volume, total grey- and white-matter volumes and white-matter hyperintensity volume also did not differ significantly between the groups [7]. The trial therefore did not demonstrate that the MIND intervention produced greater cognitive or structural brain benefits than the control intervention over three years.

A subsequent secondary analysis examined whether participant characteristics modified the intervention's effect on cognition [53]. Body mass index was the only statistically significant effect modifier identified. Among participants with a body mass index ≥ 35 kg/m², assignment to the MIND diet was associated with cognitive scores that were 0.040 standardised units higher per year than those in the control group. This subgroup finding does not alter the trial's primary null result and should be considered exploratory until confirmed in prospectively designed studies [53].

A further post hoc analysis of 590 MIND Diet Trial participants examined achieved dietary adherence rather than randomised group assignment [54]. Participants whose MIND diet scores increased substantially—by approximately four points—had a greater annual improvement in global cognition than participants whose scores did not increase ($\beta = 0.035$, 95% CI: 0.006–0.064). Greater adherence was also supported by increased plasma lutein–zeaxanthin and β -

carotene concentrations. However, the association did not differ according to randomised intervention group [54]. Because this analysis compared participants according to achieved adherence rather than randomised assignment, it does not overturn the primary null trial result and remains susceptible to behavioural differences and residual confounding.

5.3. Overall Interpretation

Several methodological differences may explain the apparent discrepancy between observational and randomised findings. Observational cohorts frequently assess habitual dietary patterns maintained over many years or decades, whereas the principal randomised trial evaluated a three-year intervention initiated relatively late in life. If the MIND diet acts by gradually reducing cumulative vascular, metabolic or neurodegenerative injury, three years may be insufficient to produce detectable between-group differences. Conversely, observational associations may overestimate benefit because individuals with high MIND diet adherence may also have higher educational and socioeconomic status, greater physical activity, lower smoking prevalence, better healthcare access and greater participation in cognitively stimulating activities. Statistical adjustment cannot eliminate all residual confounding. Self-reported food-frequency questionnaires introduce additional measurement error, while declining cognition may itself adversely affect food selection and dietary reporting, creating reverse causation.

Randomisation reduces these biases and provides stronger evidence for causality, but its ability to detect an effect depends on intervention duration, adherence, participant characteristics, outcome sensitivity and the behaviour of the control group. Consequently, observational studies suggest that long-term MIND diet adherence may be a marker or contributor to preserved cognitive health, whereas the principal randomised trial indicates that a three-year MIND intervention with mild caloric restriction was not substantially superior to an active control intervention. The observational and randomised findings are therefore not necessarily contradictory: they address different exposure periods and causal questions, and the current evidence remains insufficient to determine whether sustained MIND diet adherence beginning earlier in life directly prevents cognitive decline or dementia.

6. Discussion

6.1. Limitations of the Current Evidence

Beyond the design-related differences discussed above, the current evidence base has important limitations concerning cohort independence, exposure measurement, outcome selection and generalisability. Several influential publications reported separate analyses of partly overlapping participant samples drawn from the same Rush Memory and Aging Project cohort, which contributed data to eight articles included in a recent systematic review [55]. The number of publications may therefore overstate the number of independent populations in which the findings have been replicated.

Dietary assessment is another important limitation. Most studies used self-reported food-frequency questionnaires, which are susceptible to recall error and inaccurate estimation of portion sizes. These problems may be particularly relevant among older adults with subtle cognitive impairment. Some studies assessed diet only once despite follow-up lasting many years, although dietary habits may change with ageing, illness or functional decline. The MIND score has also been modified across studies because information about berries, olive oil, wine or specific food groups was unavailable. Differences in scoring thresholds and omitted components reduce comparability between populations. In addition, the score assigns equal weight to every component and does not fully account for food quality, preparation methods or dietary substitutions.

Substantial heterogeneity also exists in cognitive outcomes. Studies have assessed global cognition, individual cognitive domains, cognitive decline, mild cognitive impairment, all-cause dementia and Alzheimer's disease using different instruments and diagnostic methods. Better performance at a single assessment does not necessarily indicate a slower rate of decline. Repeated cognitive testing may also produce practice effects that obscure small changes. Reverse causation remains possible because preclinical dementia can alter appetite, shopping, cooking and dietary reporting years before diagnosis. Excluding cases diagnosed early during follow-up reduces but does not eliminate this problem.

Generalisability is limited because many studies involved relatively well-educated volunteers from the United States. The minimum effective level of adherence, optimal intervention duration and most appropriate stage of life for initiating the diet remain uncertain. The follow-up period in existing studies such as Barnes et al. [7] may have been too short to detect effects on pathological processes developing over decades. Moreover, mechanistic evidence is derived

largely from individual nutrients, related dietary patterns or preclinical research rather than MIND-specific clinical studies. Publication and selective-reporting bias may further favour positive associations.

Although blinding participants is inherently difficult in dietary interventions and blinded outcome assessment can reduce detection bias, the absence of participant blinding remains an important methodological limitation.

Recent studies have addressed some previous limitations but introduced additional interpretive challenges. The Chinese-adapted MIND score broadened the geographical and cultural scope of the evidence, although modification of the original score reduces direct comparability with other cohorts [50]. Assessment of changes in adherence over time reduced reliance on a single baseline dietary measurement, but dietary intake remained self-reported [51]. The study examining interactions between MIND adherence, brain pathology and cognition included only 66 participants and was cross-sectional [52]. Similarly, the findings concerning obesity and achieved adherence arose from secondary or post hoc analyses of the same MIND Diet Trial and should not be interpreted as independent randomised confirmation [53, 54].

6.2. Clinical Implications

On the basis of the observational and randomised evidence summarised above [1, 2, 7, 45], the MIND diet should not be presented as a treatment for cognitive impairment or a definitive method of preventing dementia. It may instead be recommended as a generally healthful dietary pattern with established cardiovascular and metabolic relevance and possible cognitive benefits.

Recommendations should emphasise realistic and sustainable changes, including greater consumption of vegetables, berries, whole grains, legumes, nuts, fish and olive oil, together with reduced intake of red and processed meat, butter, high-fat cheese, sweets, fried foods and highly processed products. Advice should be adapted to cultural preferences, affordability and food availability. In patients with frailty, sarcopenia, dysphagia, malnutrition or unintentional weight loss, adequate energy and protein intake should take priority over strict adherence. Modification or dietetic supervision may also be necessary in chronic kidney disease, diabetes, gastrointestinal disease or food allergy.

Although the original MIND score included moderate wine consumption, alcohol should not be recommended for cognitive protection. The randomised MIND trial omitted wine from the intervention, and individuals who do not drink alcohol should not be encouraged to begin [7]. Dietary improvement should instead form part of a multidomain strategy that includes

management of hypertension, diabetes, dyslipidaemia, smoking, obesity, physical inactivity, depression, hearing impairment, social isolation and sleep disorders.

The MIND score was developed as a research instrument rather than a validated clinical target [2]. No effective adherence threshold or intervention duration has been established, and routine cognitive testing, neuroimaging or biomarker measurement solely to monitor dietary response is not supported. Patients with suspected cognitive decline should undergo standard clinical assessment regardless of their dietary habits.

6.3. Limitations of This Review

Several limitations of this review should be acknowledged. First, it was conducted as a narrative rather than a systematic review. Although a structured literature search was performed, the search may not have identified all relevant studies, and study selection may have been affected by subjective judgement. Second, the search was limited to publications indexed in the selected databases and written in English, creating a risk of database and language bias. Publication bias is also possible because studies reporting statistically significant associations may be more likely to be published. Third, no formal assessment of the risk of bias or certainty of evidence was performed using validated instruments. A meta-analysis was not conducted because of substantial heterogeneity in study populations, definitions of MIND diet adherence, cognitive assessment methods, outcome measures, and follow-up periods. Consequently, the findings were synthesized descriptively, and the magnitude of the association between adherence to the MIND diet and cognitive outcomes could not be estimated quantitatively. Finally, evidence from randomised research was derived primarily from one trial and its secondary or post hoc analyses, restricting the comparison between observational and experimental evidence.

6.4. Future Research Directions

Further research is required to determine whether the MIND diet has a causal and clinically meaningful effect on cognitive ageing. Future studies should prioritise adequately powered, long-term randomised trials capable of detecting changes in cognitive trajectories and the incidence of mild cognitive impairment or dementia. Follow-up should extend beyond the three-year observation period used in the principal trial and, where feasible, include post-intervention surveillance. Exploratory findings concerning obesity and achieved dietary adherence should be confirmed in prospectively designed studies. Potential effect modification by age, sex, socioeconomic status, baseline diet, cardiovascular risk, obesity, genetic susceptibility and cognitive status should also be examined. Individuals with subjective

cognitive decline or mild cognitive impairment may respond differently from cognitively healthy participants.

Control-group design requires particular attention. Future trials should distinguish the effects of the MIND pattern from those of weight loss, professional support and general dietary improvement. Comparisons with usual care may complement controlled efficacy trials.

Dietary assessment should combine repeated questionnaires with objective measures such as digital food records, meal photographs or nutritional biomarkers. Standardised MIND scoring is needed because modified scores and different component thresholds reduce comparability [46, 47]. Cognitive outcomes should likewise be harmonised and include validated measures of global cognition, memory, executive function, processing speed and attention, together with clinically meaningful outcomes such as incident dementia, independence and quality of life.

Mechanistic studies should investigate vascular, metabolic, inflammatory and neurodegenerative pathways using relevant biomarkers and neuroimaging, while retaining cognitive outcomes as the primary clinical focus. Research should include culturally, geographically and socioeconomically diverse populations and evaluate adapted versions of the dietary pattern. The contributions of individual foods and dietary substitutions also require clarification. The original score's alcohol component should be treated separately in intervention protocols rather than incorporated into dietary recommendations [7]. Finally, pragmatic studies should evaluate long-term adherence, safety, affordability and the independent contribution of diet within multidomain prevention programmes.

7. Conclusions

The MIND diet represents a dietary pattern specifically designed to support cognitive health by combining selected elements of the Mediterranean and DASH diets. It provides a biologically plausible basis for potential neuroprotective effects through vascular, metabolic, anti-inflammatory, and antioxidant pathways.

The MIND diet may be considered a reasonable component of a broader strategy for healthy ageing because it is consistent with established recommendations for cardiovascular and metabolic health. Clinical advice should be individualized and integrated with physical activity, smoking cessation, social and cognitive engagement, and appropriate management of vascular and metabolic risk factors. Further long-term, adequately powered randomised trials using standardized cognitive outcomes, objective adherence measures, and clinically meaningful

endpoints are needed to determine whether the MIND diet provides specific protection against cognitive decline and dementia.

Disclosure

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Declaration of generative AI and AI-assisted technologies in the writing process

During the preparation of this work, the author(s) utilized ChatGPT (OpenAI) with web search enabled / Google Search AI Mode to assist with background literature retrieval and the initial identification of relevant academic sources on The MIND Diet and Cognitive Health in Older Adults. Following the drafting phase, these Large Language Models were further utilized to conduct grammar, editorial, and overall quality checks to refine the manuscript into standard academic English. The author(s) independently reviewed, verified, and synthesized all cited source materials, critically evaluated all language suggestions, and take full responsibility for the accuracy, context, and final content of the manuscript text.

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