REVIEW ARTICLE

CONTENTS

# The role of the MIND diet in prevention and treatment of Alzheimer's disease: A literature review

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## ABSTRACT

**Aim:** Recent research increasingly point to modifiable risk factors, especially dietary patterns, as potential tools to prevent or delay neurodegeneration. This review evaluates the impact of the MIND diet on the prevention and progression of AD and compares it with other dietary interventions.

**Materials and Methods:** A literature search was conducted using the PubMed and Google Scholar databases for articles published from January 2015 to January 2025, focusing on the influence of the MIND diet, as well as other dietary patterns, on AD progression and cognitive performance.

**Conclusions:** While the MIND diet shows promise as a feasible non-pharmacological strategy, current evidence is largely observational and limited by population heterogeneity and inconsistent adherence definitions. Short-term randomized controlled trials are less conclusive. Long-term clinical trials are needed to establish causality. Despite these limitations, the MIND diet remains a practical and potentially effective approach to reducing cognitive decline and delaying the onset of AD.

**KEY WORDS:** Alzheimer disease, neuroprotection, dietary patterns, neurodegenerative diseases, diet therapy

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## INTRODUCTION

Alzheimer's Disease (AD) represents one of the most serious health problems facing aging societies, affecting millions of people worldwide. The number of patients affected by AD in the United States alone is estimated to increase to 13.8 million by 2060, compared to approximately 6.9 million cases in 2024, indicating a significant increase from the current number [1]. Pathological changes in AD, such as the accumulation of beta-amyloid (Aβ) plaques, the formation of neurofibrillary tangles and chronic inflammation within the central nervous system , disrupt neuronal function, resulting in synaptic dysfunction and impaired neurotransmission [2]. Disease progression increases susceptibility of neurons to degeneration, leading to widespread neuronal loss, especially in areas critical for memory processing and cognitive function, such as the hippocampus and cerebral cortex. As a consequence, the quality of life of patients deteriorates and the burden on healthcare system increases [3]. The increase in life expectancy and changing demographic structures make it essential to search for effective preventive and therapeutic strategies [4].

Anti-inflammatory dietary patterns, such as the Mediterranean diet (MedDiet) and the Dietary Approaches to Stop Hypertension (DASH) diet, characterized by a high intake of foods that help reduce inflammation and oxidative stress, may exert neuroprotective effects by inhibiting neuroinflammation associated with AD [5]. The Mediterranean-DASH Diet Intervention for Neurodegenerative Delay (MIND) diet incorporates key elements from both diets, making it one of the most promising dietary approaches [6].

Since the introduction of the MIND diet pattern in 2015, it has been consistently linked to reduced cognitive decline and favorable brain outcomes. Studies suggests it may play a significant role in reducing oxidative stress and modulating inflammatory processes within the brain [7]. Appropriate nutritional strategies may support cognitive function and brain health [8], which may be particularly important in genetically predisposed populations.

## AIM

The primary aim of this review is to evaluate the potential of the MIND diet in the prevention and progression of AD, including an assessment of its impact on genetically predisposed populations. The review also explores the proposed neuroprotective mechanisms, compares the MIND diet with other dietary strategies, and identifies areas requiring further research.

## MATERIALS AND METHODS

We conducted a literature search for relevant papers in PubMed and Google Scholar databases. The search strategy incorporated combinations of the following keywords: "MIND diet", "Alzheimer's disease", "cognitive decline", "cognitive impairment", "nutritional intervention", "diet", "Mediterranean diet", "Ketogenic diet" and "APOE 4". Inclusion criteria were restricted to peer-reviewed publications published between January 2015 and January 2025. Exceptions were made for earlier foundational studies where necessary to define core concepts. Studies were considered eligible if they focused on human subjects, were written in English, and were available in full text. After title and abstract screening, 67 studies meeting the initial criteria were retained for review. Full-text articles were then assessed for eligibility. In total, 35 studies met all inclusion criteria and were selected for detailed analysis.

## REVIEW

### MIND DIET OVERVIEW

The MIND diet represents a dietary pattern that combines elements of the Mediterranean and DASH diets, with modifications based on the most significant findings in nutrition and dementia, to improve brain health and mitigate age-related cognitive decline. The basic principle of the MIND diet is to encourage the consumption of ten food groups, each with proven cognitive benefits, while limiting the consumption of five food groups that may promote neurodegenerative processes (Table 1) [6].

**Table 1.** Core principles of the MIND diet [6]

| Food group | Recommendation |
|---|---|
| Green leafy vegetables | ≥6 servings per week |
| Other vegetables | at least once daily |
| Berries | ≥2 times per week |
| Nuts | ≥5 times per week |
| Olive oil | main source of fat |
| Whole grains | ≥3 servings per day |
| Fish | at least once per week |
| Beans | ≥4 times per week |
| Poultry (not fried) | ≥2 times per week |
| Wine | 1 glass per day |
| Butter, margarine | <1 tablespoon/day |
| Cheese | <1 serving/week |
| Red Meat and products | <4 times/week |
| Fast fried foods | <1 time/week |
| Pastries and sweets | <5 times/week |

The MIND diet recommends regularly including leafy green vegetables, such as spinach, kale, and lettuce, which are abundant in folate, vitamin E, carotenoids, and flavonoids-nutrients associated with reduced risk of dementia and cognitive decline. Another key component of the MIND diet is berries eaten at least twice a week, which has been linked to improved cognitive function and better performance on cognitive tests [6]. Similarly, research suggests that the frequent consumption of nuts, another component of the diet, may support cognitive function and slow its age-related decline. The bioactive compounds found in nuts, including omega-3 fatty acids and polyphenols, play a role in reducing oxidative stress and enhancing neuroprotection [9].

The MIND diet promotes the regular consumption of whole grain products, which are a source of polyphenols, that protect the brain against oxidative stress and neurodegeneration [10]. They also contain various phytochemicals that may influence cognitive outcomes, with brown rice specifically containing higher levels of γ-amino butyric acid, which, in combination with ferulic acid, has been suggested to enhance spatial learning in some model of AD [11]. Legumes, such as beans, peas, lentils, pulses, peanuts, and chickpeas, are an important component of the diet [8] due to their high content of plant-based protein [12], as well as polyphenols and folates [10]. These nutrients may help reduce oxidative stress and lower homocysteine levels [10], which, when elevated, can compromise the blood-brain barrier, cause neuronal damage, alter Aβ production, and further promote oxidative stress. The MIND diet also includes the consumption of fish rich in omega-3 fatty acids, recommended at least once a week [6]. Consuming fatty fish more than twice a week is linked to a 41% lower risk of AD compared with eating fish less than once a month [8]. Olive oil is another key dietary component. Biophenols present in olive oil act as potent scavengers, capturing free radicals by combining with peroxide and alkoxyl radicals and chelating trace metals [13]. Oleuropein and oleocanthal, two biophenols found in olive oil, may have neuroprotective potential,

**Table 2.** Bioactive components associated with foods in the MIND diet and their impact on AD risk reduction

| Bioactive component | Food group | Mechanisms of action |
|---|---|---|
| Folate | Leafy green vegetables, legumes | Reduce homocysteine level [12] |
| Vitamin E | Leafy green vegetables, whole grain products | Inhibition of Aβ plaque deposition [7] |
| Carotenoids | leafy green vegetables, other vegetables whole grain, legumes | Reduce oxidative stress [12]; strong antioxidants |
| Omega-3 Fatty Acids | Fish Nuts | Anti-inflammator effects; reduce oxidative stress, enhance neuroprotection |
| Polyphenols (general) | Legumes, nuts | Reduce oxidative stress, enhance neuroprotection [11]; brain protection against oxidative stress and neurodegeneration [12] |
| Flavonoids | Berries, leafy green vegetables, other vegetables, whole grain products | Suppression of microglia-induced inflammation, oxidative stress [11] |
| Oleuropein | Olive oil | Significant antioxidant properties, protecting nerve cells from neurotoxin-induced apoptosis; potential to lower Aβ levels; prevent its aggregation, and decrease the expression of glutaminyl cyclase, an enzyme associated with Aβ synthesis [12]; |
| Oleocanthal | Olive oil | Anti-inflammatory activity through cyclooxygenase inhibition; may possess neuroprotective potential against AD by reducing tau protein polymerization, inhibiting Aβ aggregation, and enhancing Aβ clearance from the brain [18]. |
| Plant-based protein | Legumes | A source of protein in the diet that allows to limit the consumption of animal products containing saturated fatty acids [14] |

particularly in protecting against neuroinflammation and Aβ aggregation [14]. Eating a diet rich in vegetables, fruits, legumes, nuts, and seeds, while using olive oil as the primary culinary fat, is an effective strategy for reducing the risk of developing AD, particularly as a preventative approach rather than a treatment after the disease has occurred [15]. The bioactive compounds and their mechanisms of action responsible for the neuroprotective effect as a key element of the MIND diet are summarized in Table 2.

At the same time, the MIND diet emphasizes the elimination of products high in saturated and trans fats, which negatively impact brain health. This includes limiting red meat and meat products, butter and stick margarine, full-fat cheese, pastries, sweets, and fried or fast foods. Diets rich in saturated and trans fats, and low in polyunsaturated and monounsaturated fats can impair blood-brain barrier function [6], promote the accumulation of Aβ plaques, and contribute to cognitive decline [9]. Excessive meat consumption, particularly red meat, common in developed countries has been linked to dementia and AD [16]. The typical Western diet, characterized by low dietary fiber and high levels of animal protein and saturated fat, is associated with gut dysbiosis. This gut imbalance can trigger both local and systemic immune-mediated inflammation, leading to neuroinflammation, a well-documented contributor to neurodegenerative diseases [17]. Chronic inflammation, driven by pro-inflammatory cytokines and bacterial components, facilitates the entry of neurotoxic substances into the brain, exacerbating neurodegenerative processes [18]. Additionally, the consumption of added sugars, particularly in ultra-processed foods (UPFs) and sugar-sweetened beverages, has been associated with cognitive decline. Studies suggest that diets high in glucose, fructose, high-fructose corn syrup, high-glycemic-index carbohydrates, and salt contribute to brain atrophy, neuronal loss, and may ultimately increase the risk of developing AD [19].

Moreover, this dietary pattern has been shown to provide protection against several cardiovascular risk factors, such as hypertension and elevated LDL cholesterol, which also contribute to the development of dementia and AD. These protective effects are achieved, at least in part, by modulating key pathological processes underlying AD, including oxidative stress, inflammation, and insulin resistance [17].

## ASSOCIATIONS BETWEEN THE MIND DIET AND ALZHEIMER'S DISEASE RISK

Empirical evidence supporting the effectiveness of the MIND diet comes mainly from large, multicenter cohort studies. In the Rush Memory and Aging Project (MAP),

involving 923 individuals aged 58 to 98 years and followed for an average of 4.5 years, those in the highest tertile of MIND diet adherence had a 53% lower risk of developing AD [20]. Notably, even moderate adherence conferred significant benefits, distinguishing the MIND diet from other dietary patterns such as DASH or the MedDiet. However, it is important to note that participants with the highest MIND scores generally had more favorable demographic and health profiles, including higher levels of education, lower BMI, a lower incidence of diabetes, and higher levels of physical and cognitive activity. Nevertheless, even after adjusting for these variables, the protective effect of the MIND diet remained statistically significant after multivariate adjustment, although somewhat attenuated [20]. Similar findings have been reported in other populations. An analysis combining three large cohorts (Whitehall II in the UK, and the Health and Retirement Study and Framingham Offspring Study in the US) comprising a total of 18,136 participants, showed that every 3-point increase in the MIND score was associated with a 17% relative reduction in dementia risk. In this analysis, the protective effect of the MIND diet was particularly evident among non-smokers, while no significant effect was observed in smokers [21].

In the European Three-City Bordeaux cohort, which included 1,412 participants followed for almost 10 years, each one-point increase in MIND adherence was associated with a 12% lower risk of developing AD [22]. This effect was independent of demographic and health factors. Furthermore, neuroimaging data from a subgroup of 175 participants revealed an association between higher MIND scores and greater microstructural integrity of the brain's white matter. Notably, no significant differences were observed in grey matter volume (based on VBM analysis), suggesting that the MIND diet may help preserve the integrity of neural connections rather than increase brain volume [22].

Data from the Chinese CHNS study (n = 4,066) indicated that a 3-point increase in the MIND score corresponded to a 0.110 z-score improvement in cognitive function, roughly equivalent to one year of younger cognitive age [7]. Benefits were also evident in long-term lifestyle analyses. In the CHAP study (Chicago Health and Aging Project), individuals aged 65 who adhered to 4–5 healthy lifestyle habits lived an average of 4.5 years longer (women) and 6.4 years longer (men) without dementia compared to those with one or no healthy habits [23].

In a systematic review of 40 studies (32 cohort studies), van Soest et al. reported that 70% confirmed a significant inverse association between MIND diet adherence and dementia risk, particularly in North American populations [24]. Observational analyses, including both cross-sectional and longitudinal studies, support a strong relationship between higher MIND diet adherence and better cognitive performance. Among 960 American adults, slower cognitive decline was observed across five cognitive domains: episodic memory, semantic memory, spatial memory, processing speed, and working memory [6].

However, not all studies are fully consistent with each other. One larger study (n = 16,058) did not confirm a significant association between the MIND diet and cognitive function [25], highlighting the need for further research.

## CLINICAL APPLICATIONS OF THE MIND DIET

Despite promising findings from cohort studies, data from randomized controlled trials remain more inconclusive. The largest intervention study to date was a three-year randomized trial conducted at two centers in the US (Chicago and Boston), involving 604 individuals at risk of AD (ages 65–84, with family history of AD, BMI > 25), but without clinically significant cognitive impairment. It compared the effects of a calorie-restricted MIND diet to a standard calorie-restricted control diet. Although both groups showed cognitive improvement, the difference between them was not statistically significant (0.035 units; p = 0.23), and MRI outcomes (hippocampal, white matter, and grey matter volumes) did not differ between the groups [26]. The authors suggest that improvement in both groups may have been partially attributable to practice effects resulting from repeated cognitive testing, as well as to the similar levels of support provided to both groups. They also note that the full potential effects of the MIND diet may require a longer intervention period to fully manifest.

In a review by Devranis et al., a small randomized trial (n = 37) was included, in which a three-month MIND diet intervention in obese women (BMI = 32) led to statistically significant improvements in working memory, verbal memory, short-term memory, attention, visual scanning, and, to some extent, executive function [27].

## BENEFITS FOR GENETICALLY PREDISPOSED INDIVIDUALS

In the context of AD prevention, attention is being paid to the effectiveness of diets among individuals with the APOE ε4 allele - the main genetic risk factor for sporadic AD [28]. Studies indicate that carriers of this allele not only have an increased risk of developing AD but may also respond differently to dietary interventions [29]. In a sample of 389 older adults (52% women, mean

**Table 3.** Comparison of the MIND, DASH, mediterranean, and ketogenic diets in the context of Alzheimer's disease

| Feature | MIND | DASH | Mediterranean | Ketogenic |
|---|---|---|---|---|
| General dietary recommendations | Emphasis on 10 groups of food, limits red meat, butter, sweets, fried food, cheese [6] | Low sodium, reduced saturated fats and refined sugars, high intake of vegetables and fruits [13] | High intake of vegetables, fruits, legumes, olive oil, nuts; moderate alcohol; limited red meat [20] | Very low carbohydrate (<30–50 g/day), high fat intake (up to 90% of energy) [31] |
| Mechanism of action | Antioxidant, anti-inflammatory, vascular protective effects [8, 10] | Blood pressure reduction; improved vascular health [13] | Antioxidant, anti-inflammatory; improved lipid profile and endothelial function [27, 32] | Ketosis; ketone bodies as alternative energy substrate [31] |
| Effects in AD | Associated with slower cognitive decline and reduced AD risk [20, 22, 23] | Some protective effects, mainly at very high adherence to the diet [27] | 18% lower risk of cognitive decline, 30% lower risk of AD; reduced amyloid burden [32] | Potential cognitive benefits; limited data; reduced ROS and neuroinflammation [27, 31]; ambiguous results in APOE4 carriers [13, 27] |

age 69 years), including individuals with mild cognitive impairment (MCI) and siblings of patients with AD, a significant association was found between MIND diet adherence and improved memory performance [30]. After adjustment for covariates such as age, education, physical activity, BMI, and APOE ε4 status, regression models confirmed that this effect was independent of genetic risk. Although the analysis did not test for interaction effects between diet and genotype, the inclusion of APOE ε4 as a covariate contributes valuable insights into the potential efficacy of the MIND diet in populations at elevated risk of AD.

## COMPARISON OF THE MIND DIET WITH OTHER DIETARY INTERVENTIONS

Other dietary interventions, including the MedDiet, the DASH diet, and the ketogenic diet, may affect inflammatory pathways involved in the development of AD, although their underlying principles and mechanisms of action differ (Table 3).

### *MIND VS. KETOGENIC DIET*

The ketogenic diet (KD), characterized by a low-carbohydrate, high-fat diet, induces ketosis. In this state ketone bodies become the brain's primary energy source in the absence of glucose [31]. Reduced production of reactive oxygen species (ROS) in the brain has been observed in this state, potentially beneficially impacting brain function by reducing neuroinflammation associated with neurodegenerative diseases. In AD, where brain glucose metabolism is impaired, ketone bodies - particularly β-hydroxybutyrate (βHB) - may help compensate for energy deficits [31]. βHB has been shown to reduce ROS production by modulating mitochondrial complex I, which may reduce neurotoxicity in AD. The KD also increases uncoupling proteins (UCPs), enhancing oxidative phosphorylation - often impaired in AD - and elevates the NAD+/NADH ratio, thereby protecting neurons from damage [31].

Both clinical and preclinical studies indicate that the KD may improve cognitive function in patients with MCI. In one study, administration of medium-chain triglycerides (MCTs) to individuals with AD resulted in better performance on the ADAS-cog scale and higher blood ketone levels [13]. A six-week low-carbohydrate diet (20 g per day) also significantly improved verbal memory in older adults with MCI. However, some findings suggest that the effectiveness of such interventions may be limited in carriers of the ApoE4 allele [13].

While the KD may benefit those with impaired glucose regulation, it has some practical limitations. Strict carbohydrate restriction can potentially lead to the so-called "ketogenic flu" (symptoms such as nausea, constipation, dizziness) [31], as well as the risk of high intake of saturated fat if not well-balanced. This can potentially contribute to inflammation and adversely affect cardiovascular health. Given these limitations, the MIND diet appears to offer a safer and more practical option for long-term, daily use, especially in older adults.

### *MIND VS. DASH DIET*

Originally developed to reduce hypertension, the DASH diet has also been shown to demonstrate beneficial effects on cognitive functions [27]. Its core principles—reducing sodium, simple sugars, and saturated fats while promoting high intake of vegetables and fruits- partially align with MIND diet recommendations. Evidence from clinical trials indicates that adherence to

the DASH diet significantly improves executive functions and psychomotor speed compared to control groups. Observational data further suggest that it may slow overall cognitive decline [13].

While the MIND diet incorporates components of both the DASH diet and MedDiet, it introduces more targeted recommendations with a greater emphasis on specific brain-healthy foods not present in other dietary patterns [20]. Both the DASH and MIND diets show improvements in executive function and psychomotor speed; however, the MIND diet appears more promising in terms of dementia prevention. Existing findings indicate that the protective effect of the DASH diet is primarily observed with very high adherence level to the diet, whereas the MIND diet shows a linear relationship between the degree of adherence and reduced risk of dementia [27].

### *MIND VS. MEDITERRANEAN DIET*

The MedDiet forms the conceptual basis of the MIND diet model, promoting a high intake of vegetables, fruits, legumes, and unsaturated fats - primarily from olive oil [27]. Both diets provide numerous antioxidants - including flavonoids and phenolic substances - which have been shown to play a role in reducing neuroinflammatory processes associated with the pathogenesis of AD [27, 32]. Differences between the two diets include the extent of restriction of foods potentially harmful to brain health and the inclusion of specific ingredients, such as berries and green leafy vegetables, as key components of the MIND diet.

A meta-analysis of multiple studies demonstrated that adherence to the MedDiet is significantly associated with lower risk of cognitive decline (by 18%), dementia (by 11%), and AD (by 30%) [32]. Studies examining the relationship between the MedDiet and cerebral Aβ accumulation, using positron emission tomography (PET) imaging in older adults, have demonstrated that higher adherence to the diet is associated with reduced Aβ plaque formation, lower cerebral Aβ accumulation, and an improved neuroimaging biomarker profile [27]. Additionally, MedDiet has been shown to positively impact lipid parameters, blood pressure, endothelial function, and reduce inflammatory markers such as C-reactive protein (CRP) and interleukin-6 (IL-6) [33]. Furthermore, MedDiet adherence has been linked to specific alterations in gut microbiota composition. The NU-AGE one-year dietary intervention revealed that MedDiet adherence was associated with favorable changes in gut flora, reduced frailty, enhanced cognitive function, and a decrease in inflammatory markers, including CRP and interleukin-17 (IL-17) [27]. Evidence from over 250 studies suggests that the MedDiet may reduce the risk of cognitive decline among APOE ε4 carriers [29]. In some analyses, this effect was even more pronounced among APOE ε4 carriers. However, results were not consistent across all studies.

Observational studies also reveal that individuals with AD are less likely to adhere to MedDiet recommendations - only 1.4% of AD patients demonstrated high adherence, according to the MEDAS scale, compared to 5.5% in the control group [34]. This may suggest a potential relationship between cognitive decline and deviation from a healthy dietary pattern, though further confirmation is needed.

## DISCUSSION

Although the scientific evidence supporting the effectiveness of the MIND diet is promising, it requires critical interpretation to assess its consistency, limitations, and validity. Its effectiveness in practice depends on a number of factors related to daily use, which can significantly influence adherence and health outcomes. Analysis of existing research reveals both barriers and the potential for adaptation, suggesting directions for further research and clinical interventions.

One such barrier is factors related to nutrition education - low levels of nutritional knowledge in at-risk populations may limit the effectiveness of interventions in the absence of adequate educational support. In addition, practical barriers such as food cost and availability may reduce adherence. Notably, it has been observed that the MIND diet can be difficult to maintain due to the high price of key ingredients such as olive oil, nuts, berries, and fish—especially during off-season and in low- and middle-income countries, where the lack of local production limits availability and reduces adherence to the diet [27]. However, there are ways to overcome this limitation. The MIND diet can be culturally adapted, as exemplified by the MIND-NL version developed for the Dutch population, which incorporates local products and a modified adherence scale [35]. Such adaptations may improve the generalizability and effectiveness of the diet across different cultural groups.

Beyond issues of adherence, the timing of dietary intervention may also influence its effectiveness. From a preventive medicine perspective, implementing the MIND diet already in midlife - before the onset of clinically manifest cognitive decline - appears particularly promising. In the MAP study, which involved participants with an average age 81, significant reductions in cognitive decline rates were observed. Participants with the highest adherence exhibited cognitive aging

trajectories comparable to those of individuals approximately 7.5 years younger [6]. Similarly, positive effects were observed in the CHNS cohort (mean age: 62), where the MIND diet was associated with better cognitive performance [7]. These findings emphasize that the MIND diet may be effective not only for secondary prevention in older adults but also for primary prevention in middle-aged individuals.

However, while potential of the diet is encouraging, it must be interpreted in light of important limitations within the current body of evidence. Although observational data continue to accumulate, high-quality confirmation from randomized controlled trials (RCTs) remains sparse. Existing RCTs - though methodologically sound - have failed to demonstrate clear differences between intervention and control groups. This may be due to factors such as the relatively short duration of interventions, test–retest bias, or unexpectedly high adherence in the control groups. Nevertheless, current study provides valuable insights into the feasibility of dietary interventions and their potential impact in populations at elevated risk of cognitive decline. These findings collectively provide a foundation for future trials that integrate longer follow-up, biomarker-based endpoints, and genetic stratification - ultimately confirming that diet is a central component of dementia prevention strategies.

## CONCLUSIONS

The accumulated evidence - from both cohort and clinical studies - demonstrates the considerable potential of the MIND diet as a promising preventive approach to AD. Although large-scale interventional trials remain limited, the consistency of observational findings and their alignment with known neurodegenerative mechanisms support the recognition of the MIND diet as one of the most promising dietary interventions. Its design combines targeted neuroprotective components with practical feasibility in clinical settings. Future research should incorporate long-term follow-up, diverse populations, and the evaluation of neurodegeneration biomarkers. Particularly important is the adaptation of the MIND model to local contexts, along with the integration of dietary interventions into broader health education initiatives. Such an approach may represent a tangible step toward effective population-level dementia prevention.

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## CONFLICT OF INTEREST

The Authors declare no conflict of interest

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