Non-Pharmaceutical Interventions for Alzheimer's Disease: The Role of Diet, Exercise, and Sleep in Slowing Disease Progression

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Received December 05, 2024
Accepted March 16, 2025
Electronic access April 15, 2025

Alzheimer's disease (AD) is a prevalent health problem and requires much more proactive engagement regarding the level of prevention and treatment. In lives, diet, physical activity, and sleeping are the main factors that count in the reduction of risk. These three diets include the Mediterranean, DASH, and MIND diets, which are more nutritionally dense, leading to enhanced neurological activity due to their ability to reduce neuroinflammation in the brain. To date, the most beneficial exercise forms for the brain also activate neurogenesis and lower inflammation. Another critical factor is sleep because sleep disturbances may enhance AD progression; poor sleep may facilitate amyloid accumulation, leading to quicker cognitive decline. This paper reviews current research into the effects of diet, exercise, and sleep on AD and emphasizes their importance in the non-pharmacological management of the disease.

Keywords and Common Abbreviations: Alzheimer's Disease (AD), Cognitive Behavioural Therapy (CBT), Mild Cognitive Impairment (MCI), Physical Activity (PA), Brain-Derived Neurotrophic Factor (BDNF), Amyloid beta (Aβ), streptozotocin (STZ), randomized controlled trials (RCTs), Mini-Mental State Examination (MMSE)

Introduction

Alzheimer's Disease (AD) is a leading cause of dementia, impacting millions globally. The disease is predicted to increase significantly because of a dramatic surge in the aged population, thus it represents a critical area of concern for both researchers and health care providers. Most current therapeutic approaches to AD have targeted symptoms, such as memory loss and cognitive decline, with extremely limited efficacy in stopping the disease's process. Such medications include cholinesterase inhibitors and MDA receptor antagonists, which give temporary relief but do nothing to address the root causes of AD. Although there is an advance in understanding the disease, the clear mechanisms that lead to the development of AD are not ascertained with clarity yet. These knowledge gaps emphasize the need for further research in understanding AD and developing more effective therapies.

Cognitive Behavioral Therapy (CBT) is a well-established intervention that is widely used to treat various disorders. It focuses on modifying negative thought patterns, helping individuals develop healthier behaviors. It can also manage AD, especially in its early stages. Indeed, several studies have illustrated the use of CBT in the pursuit of an improved quality of life for AD patients. While the early results are promising, much remains to be learned about its long-term efficacy and how it will be integrated into broader treatment schemes.

CBT is being increasingly explored as an approach to the management of AD, offering a lifestyle-based strategy to the pharmaceutics-based treatments currently in use. While AD is partly characterized by emotional and psychological challenges, its impact on memory and cognitive function is most striking. CBT is promising in addressing these symptoms and thus may improve the overall quality of life in AD patients. It may help alleviate distress due to AD by facilitating diet, PA, and sleep. This review will go over recent findings regarding the effect of diet, exercise, and sleep on the course of AD in the context of CBT and its major role in the development of non-pharmacological interventions against this disease as they do not involve any medications or drugs.

The Crucial Role of a Healthy Diet in Preventing AD

A form of CBT that can help prevent the progression of AD in patients with early to mid-stage AD focuses on a healthy diet. For example, eating a diet high in antioxidants and vitamins, as well as omega-3 fatty acids from fish, may help prevent AD. Studies have shown that fish consumption aids with dietary intake of omega-3 fatty acids as it averts brain damage due to their anti-inflammatory characteristics. Roman et al. found that higher omega-3 fatty acids in the blood led to a reduced chance of cognitive decline, while higher omega-6 fatty acids increased risk. Recent research highlights astaxanthin, a strong antioxidant found in seafood, for its potential neuroprotective effects[1]. Additionally, studies suggested that a walnut-enriched diet may delay the development of AD and mild cognitive impairment (MCI). Long-term walnut supplementation in animal studies improved memory, learning, motor skills, and anxiety in transgenic mouse models for AD. In the PREDIMED trials, adults on a Mediterranean diet with nuts, including walnuts, had better cognitive function than those on a low-fat diet[2]. Including nutrient-rich foods in your diet that have high levels of omega-3 fatty acids and antioxidants, such as fish and walnuts, play a key role in slowing cognitive decline and improving cognitive function in patients with early to mid-stage AD. Major dietary factors in AD development include deficiencies in antioxidants (vitamins E and C), vitamins B6 and B12, and excess saturated fatty acids. Antioxidants help reduce oxidative stress and inflammation, while B vitamins are crucial for reducing homocysteine metabolism[3]. High stages of homocysteine metabolism are linked to a higher risk of AD by contributing to neurotoxicity[4]. Proper nutrition is vital for AD prevention and management, as malnutrition is common in AD patients due to changes in appetite regulation and eating habits. Consequently, ensuring adequate energy intake is essential, as both deficits and nutritional surpluses negatively affect health. Current dietary guidelines suggest 1500–2000 kcal daily for the elderly, with relatively higher protein needs to combat age-related muscle loss and chronic diseases. Fat intake should comprise no more than 30% of daily energy consumption, with an emphasis on prioritizing unsaturated fats over saturated fats. Additionally, carbohydrate intake should be balanced, avoiding high-glycemic diets that may increase Amyloid beta (Aβ) aggregation and AD risk[5]. Reduced calcium metabolism in neurons can also contribute to AD through synaptic loss, tau protein accumulation, dendrite pruning, and more. Ultimately, improving calcium activity in neurons is a reassuring approach to lowering the effect of AD[6]. Magnesium, as a natural calcium channel blocker, has also been studied for its effects on cognition. A study from the Personalized Prevention of Colorectal Cancer Trial found that decreasing the calcium-to-magnesium ratio through the use of magnesium supplements for 12 weeks improved scores in adults over age 65 on the Montreal Cognitive Assessment test[7]. Emphasizing adequate intake of essential nutrients and properly managing fat carbohydrate consumption, is crucial for preventing and managing AD.

Consumption of polyunsaturated fats (PUFAs), especially omega-3 fatty acids DHA and EPA, is directly linked to the regulation of inflammation, a key factor of AD. Higher intake of PUFAs is associated with a lower risk of AD[4]. More specifically, PUFAs offer neuroprotective benefits by enhancing neuroplasticity, stimulating synaptogenesis, and regulating neuronal signal pathways[8]. Scientific findings reveal that mixed results regarding the benefits of omega-3 and α-lipoic acid supplements in slowing functional decline in AD patients, suggesting a need for further research to clarify the efficacy of PUFA supplementation in treating cognitive symptoms of AD, especially in individuals with nutrient deficiencies[9]. Polyphenolic compounds such as flavonoids, phenolic acids, and tannins, found in blueberries, olive oil, walnuts, cocoa, and tea, may slow down cognitive decline associated with aging. Studies have also demonstrated the benefits of grape juice in improving memory and brain health, quercetin in reversing AD symptoms in mice, and flavanols in protecting against AD. Epigallocatechin-3-Gallate (EGCG) from green tea has shown potential in reducing Aβ accumulation and improving cognitive function in AD models[10]. Methylxanthines, such as caffeine, found in coffee and energy drinks, also have anti-inflammatory, antioxidant, and neuroprotective properties. Some studies have shown caffeine may reduce Aβ production and memory impairment[1]. These findings show the potential of specific nutrients and dietary patterns in mitigating AD, stressing the need for more studies to fully understand their impact on cognitive health.

In following specific nutrient guidelines, consumers often consult dietary patterns through renowned diets such as the Mediterranean diet, the DASH diet, and the MIND diet, which are known for their potential benefits in promoting cognitive health. The Mediterranean diet consists of vegetables, fruits, nuts, legumes, whole grains, fish, olive oil, and moderate wine intake which are linked with a lowered likelihood of MCI. Studies have linked higher adherence to the Mediterranean diet with a mitigated risk of developing AD[?] . The DASH diet, another dietary regime to reduce intake of fats and sodium, originally designed to prevent high blood pressure, includes reduced-fat meats, whole grains, fruits, vegetables, and low-fat dairy. Hence, a positive association exists between this diet, improved cognitive function, and PA, which leads to a lower risk of AD[11]. Finally, the MIND diet combines aspects of the Mediterranean and DASH diets, consisting of berries, leafy greens, nuts, legumes, whole grains, and fish. Research suggested the MIND diet may slow cognitive decline more effectively than the individual Mediterranean or DASH diets[12]. These diets emphasized the importance of specific food choices in stimulating cognitive health and potentially lowering the probability of developing AD (see Figure 1).

Fitness Strategies to Help Counteract AD

PA helps prevent and mitigate AD, highlighting the growing prevalence of AD despite extensive research. Additional evidence also supports the beneficial effects of neurodegenerative diseases, including AD[13]. PA encompasses any bodily movement requiring energy expenditure, which differs from exercise, a structured subset aimed to improve physical fitness. Epidemiological studies have underscored that regular PA and exercise positively impact hippocampal volume; as a result, preventing age-related neurological decline due to the hippocampus' role in neuroplasticity[14]. More specifically, regular PA, character-

Table 1 Dietary Components, Cognitive Benefits, and Supporting Studies

Dietary Component	Cognitive Benefits	Supporting Studies
Omega-3 Fatty Acids (Fish, Walnuts)	Reduces brain inflammation, lowers cognitive decline risk	Roman et al.; PREDIMED trials
Antioxidants (Astaxanthin, Vitamin E & C)	Neuroprotective effects, reduces oxidative stress	Various antioxidant studies
B Vitamins (B6, B12)	Lowers homocysteine levels, reduces neurotoxicity risk	Homocysteine metabolism studies
Polyunsaturated Fats (PUFAs)	Regulates inflammation, enhances neuroplasticity	PUFA supplementation trials
Polyphenols (Flavonoids, EGCG, Methylxanthines)	Improves memory, slows cognitive decline	Grape juice, green tea, and caffeine studies

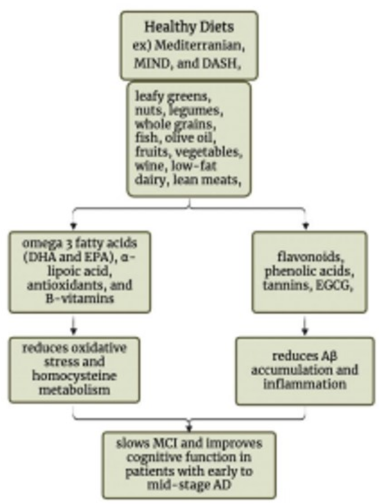

Fig. 1 Flow chart illustrating the key components of various diets and their contributions to AD prevention. This visual guide breaks down how specific nutrients and food groups within diets like the Mediterranean, DASH, and MIND diets assist in reducing susceptibility to AD.13 The chart highlights that omega-3 fatty acids are found in fish, antioxidants are abundant in fruits and vegetables, and phenolic acids are in nuts and olive oil. These aspects of each diet function to combat oxidative stress, reduce inflammation, and support overall brain health [11]. MIND - Mediterranean-DASH Intervention for Neurodegenerative Delay; DASH - Dietary Approaches to Stop Hypertension; DHA - Docosahexaenoic acid; EPA - Eicosapentaenoic acid; EGCG - epigallocatechin-3-gallate; Aβ - Amyloid Beta; MCI - Mild cognitive impairment; AD - Alzheimer's Disease

ized by 150 minutes of PA per week, reduces cognitive decline risk and AD-associated biomarkers by 35-38% [15]. PA increases cerebral blood volume in humans, enhancing brain function and supporting the development of nervous tissue, which slows down mental deterioration associated with aging and brain disorders. Metabolites in the brain play a crucial role in modulating various processes. Exercise induces the overexpression of PGC1α, converting kynurenine, a neuroinflammatory metabolite, into kynurenic acid, which cannot cross the blood-brain barrier (BBB) [16]. Additionally, lactate produced during exercise promotes angiogenesis and hippocampal neurogenesis, potentially modulating neuroinflammation [17]. These processes help maintain brain health and delay the onset of Alzheimer's Disease by reducing neuroinflammation and supporting the brain's structural integrity and function.

High-intensity interval training may reduce AD risk more effectively than moderate-intensity continuous training through strengthening Brain-Derived Neurotrophic Factor (BDNF) and cardiorespiratory fitness while decreasing AD risk factors like obesity and high blood pressure. Nevertheless, it's crucial to recognize that that continuous high-intensity exercise may also promote proinflammatory pathways [18]. In addition, resistance training has demonstrated the ability to enhance mental abilities, protect against hippocampal degeneration, and increase irisin levels, beneficial for brain health [19]. Exercise also influences AD-related neuroimaging biomarkers, with aerobic exercise potentially increasing hippocampal volume and functional connectivity [20]. These findings highlight the numerous benefits of physical exercise, suggesting it can downscale the likelihood of AD through various pathways, including risk factor prevention and brain health promotion [21]. Despite promising preclinical studies that have suggested many biological factors driving the protective effects of exercise against AD development, more clinical research is needed to confirm these benefits and to pinpoint the most effective exercise interventions for preventing or slowing the progression of AD [22]. This underscores the empowering role of exercise in reducing AD risk and the importance of further research in developing effective exercise-based strategies against AD.

In their study examining physical exercise, including both aerobic and resistance training, Rosa et al. found that this dynamic can potentially delay the onset of AD [23]. Combining exercise with other multimodal interventions like nutrition plans and mental stimulation has also shown promise in reducing cognitive decline[10]. Rosa et al. further expressed that exercise induces beneficial changes at the brain's anatomical and molecular levels, promoting synaptogenesis and the production of neurotrophic factors like BDNF, which enhance brain plasticity. Moreover, exercise reduces pro-inflammatory cytokines and boosts anti-inflammatory responses, demonstrating its anti-inflammatory effects. These findings highlight exercise regimens as a beneficial tactic for managing AD, as it supports contributing to brain health through various protective mechanisms.

Panza, G. A. et al. (2018) conducted a meta-analysis that demonstrated fitness enhances mental acuity, with endurance training providing a beneficial outcome. This neural improvement is attributed to increased enhanced attention, executive function, processing speed, and memory[24]. Animal studies further support this, showing that exercise, such as wheel running, can lower the probability of AD by enhancing neuroplasticity. These benefits are associated with mechanisms such as increased production of BDNF in the hippocampus, angiogenesis in the motor cortex, and improved blood flow to the brain. In a study using a streptozotocin (STZ)-induced AD rat model, consistent activity significantly suppressed tau phosphorylation in the brain using E3 ubiquitin ligase (see Figure 2). The analysis revealed a transition of activated microglia from promoting inflammation to reducing it, increasing anti-inflammatory cytokine activity in the hippocampus. This led to a notable decline in STZ-induced oxidative mitochondrial damage in response to exercise[25]. In humans, exercise increases brain activity in the left dorsolateral frontal lobe, posterior parietal zone, and anterior cingulate region. Aerobic exercise enlarges the anterior hippocampus, resulting in better memory due to elevated BDNF levels, which play a key role in neurogenesis in the hippocampal dentate subdivision[10]. These findings emphasize the importance of the function of exercise in enhancing cognitive ability and mitigating the progression of AD through a variety of physiological mechanisms.

Research indicates that exercise can significantly enhance daily living activities and delay mental deterioration in seniors, with aerobic exercise showing significant benefits in Focus, reaction time, decision-making skills, and memory retention. However, earlier analyses have failed to thoroughly examine forms of PA[26]. To address this gap, a review and meta-analysis of randomized controlled trials (RCTs) was conducted to study the effects of cardiovascular workouts on intellectual ability, using the Mini-Mental State Examination (MMSE) as a primary outcome measure in AD patients. The meta analysis demonstrated that aerobic activity greatly improved neurological performance in those with AD, as indicated by enhanced MMSE outcomes. Subgroup review found that aerobic sessions lasting 30 minutes, totaling less than 150 minutes per week, and conducted up to three times per week were the most effective[27]. Greater improvements were observed in patients with worse initial cognitive status. The potential mechanisms for these enhancements include increased levels of BDNF, better circadian rhythm regulation, and improved vagal tone[28]. The study concluded that specific patterns of aerobic exercise were particularly beneficial for cognitive function in AD patients, especially those with more severe impairments initially, providing reassurance for the potential of exercise in promoting better brain health in individuals living with AD.

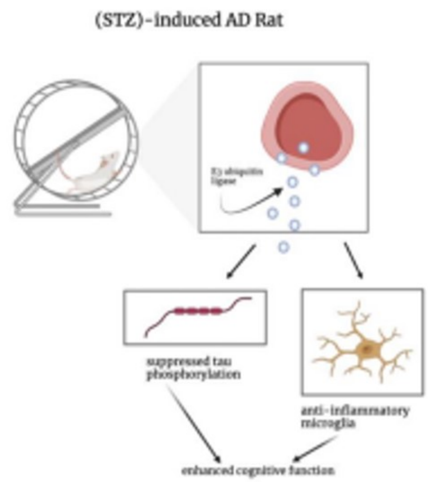

Fig. 2 Slowing the progression of AD and MCI on a (STZ)-induced AD rat through exercise. This image highlights the significant impact of exercise on health. Fitness promotes the production of E3 ubiquitin ligase in the brain, which suppresses tau phosphorylation and reduces pro-inflammatory microglia. These effects contribute to improved cognitive function, helping to slow the development of AD and MCI[25]. STZ - Streptozotocin

The Importance of Sleep Health in Reducing the Risk of AD

Emerging research highlights the critical role of sleep health in the development and progression of AD, with growing evidence suggesting that sleep disturbances may not only be a symptom but also a potential risk factor. While genetic factors play a significant role in an individual's risk of developing dementia, particularly AD, lifestyle factors such as sleep disturbances are increasingly recognized as contributing to this risk[29]. Sleep is essential for memory consolidation, yet nearly half of adults over 60 struggle with both initiating and maintaining sleep[30]. Evidence indicates that sleep disturbances contribute to cognitive decline and may increase the risk of AD by boosting Aβ levels in the brain (see Figure 3)[31]. Traditionally viewed as a consequence of AD, sleep disturbances may also play a causal role, creating a mutual relationship between sleep and AD biomarkers. Beyond amyloid accumulation, sleep disturbances also trigger systemic inflammation, further exacerbating the pathological cycle of AD. Sleep disturbances trigger systemic inflammation, which is considered an early event in AD progression, as immune responses contribute to Aβ burden and disease pathogenesis[32]. Sleep disturbances can elevate systemic inflammation markers, such as proinflammatory cytokines and C-reactive protein, which may lead to the transition of microglial cells into a primed, hyperactivated and dysfunctional state. These chronically activated microglial cells exhibit reduced amyloid clearance and increased production of inflammatory cytokines, contributing to local central nervous system inflammation and AD progression. This creates a feedback loop where inflammation aggravates the accumulation of AD pathology, further activating and priming more microglial cells[31]. Additionally, amyloid accumulation triggers the expression of IL-12 and IL-23, leading to further inflammation and neuronal damage, thus accelerating cognitive decline[33]. This links systemic inflammation and cognitive decline, emphasizing the role of inflammation in the progression of AD and highlighting sleep disturbances as a modifiable risk factor that could potentially mitigate inflammatory processes associated with AD.

Sleep dysfunction affects many elderly individuals, with over half frequently reporting sleep complaints. This widespread issue, along with available interventions, makes sleep an appealing target for AD prevention[34]. The relationship between sleep dysfunction and dementia remains unclear due to the lack of RCTs. Several mechanisms suggest that sleep dysfunction might lead to AD, including increased systemic inflammation and decreased clearance of neurotoxic metabolites like amyloid[35]. Studies show that sleep deprivation can reduce amyloid clearance and alter its metabolism, potentially contributing to amyloid accumulation. Evidence also links self-reported sleep dysfunction with increased brain amyloid burden and disrupted memory consolidation[36]. Sleep apnea syndromes are another factor, causing intermittent brain hypoxia and potentially leading to cognitive decline[37]. Decreased REM sleep has been tied to a greater risk of developing AD[38]. The overall inconsistency in study methods complicates drawing definitive conclusions. However, evidence suggests that addressing sleep issues may enhance cognition in patients with MCI, though it is still uncertain whether early sleep interventions can lower the risk of developing AD.

The bidirectional relationship between sleep patterns and AD stand as a sign of cognitive decline[39]. Sleep is crucial for cognitive processing, memory consolidation, and removing Aβ and tau proteins, all associated with AD. Sleep disruptions frequently occur prior to the development of cognitive decline, suggesting they might be early biomarkers for dementia risk[35,38]. According to a comprehensive review, various sleep disturbances like insomnia and obstructive sleep apnea are linked to a higher likelihood of cognitive impairment[40]. Medications for sleep disturbances in dementia are commonly used, though evidence for their effectiveness is limited. Melatonin and trazodone may help, particularly when combined with non-pharmacological interventions, which should be the first-line therapy due to the risks of adverse effects from hypnotics[41]. Non-pharmacological treatments have shown promise, such as light therapy, acupressure/acupuncture, and mindfulness[42]. However, additional studies on these interventions to evaluate their effectiveness are currently limited and varied, preventing definitive conclusions.

AD is seen as causing sleep problems that correlate with its severity. A recent theory suggests a dual relationship where not only does AD cause sleep disturbances, but poor sleep can also contribute to the accumulation of Aβ proteins (see Figure 4)[31]. Studies show that sleep is crucial for clearing these proteins, and sleep deprivation can accelerate their build-up, leading to further neurodegeneration. Therefore, improving sleep would be an effective way to slow the progression of AD[39]. AD pathology can begin 1-2 decades before a dementia diagnosis, providing a window for early intervention, particularly in the MCI or early AD stages. Early diagnosis through molecular biomarkers is increasingly common, offering opportunities for targeted interventions[40]. There are various AD sleep enhancement targets, including pharmacological and non-pharmacological approaches. The focus on non-pharmacological methods is growing due to potential side effects and interactions in older adults. These methods include lifestyle interventions and novel technologies like sound or electrical brain stimulation and bright light therapy[41]. Addressing sleep disturbances in AD patients is critical, as improving sleep quality not only holds the potential for slowing disease progression by mitigating Aβ accumulation but also offers broad health benefits through various pharmacological and non-pharmacological approaches. Thus, enhancing sleep health emerges as a promising strategy not only for improving overall well-being but also for potentially mitigating the progression of AD.

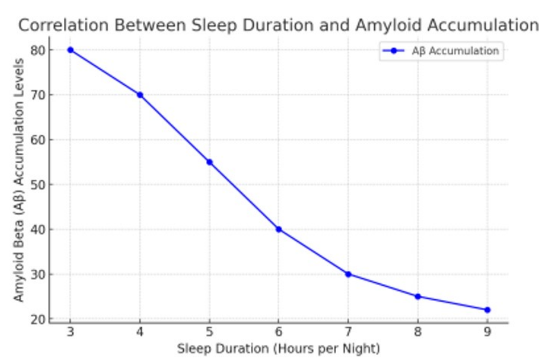

Fig. 3 Chart illustrating the inverse relationship between sleep duration and amyloid accumulation. The function that sleep plays in removing neurotoxic waste is further supported by the fact that people who have had little sleep—that is, less than six hours—have increased amyloid accumulation, whereas those who have had seven to nine hours show reduced accumulation.

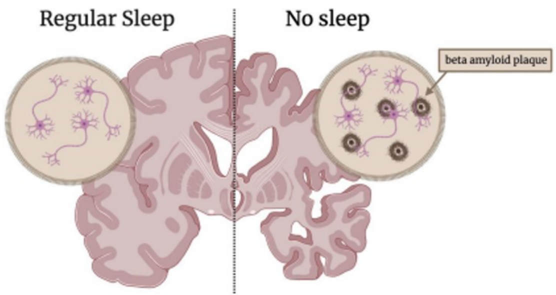

Fig. 4 The effects of irregular sleep patterns on neurons and the accumulation of beta-amyloid plaques. The left side of the image illustrates a healthy brain where neurons are free from beta-amyloid deposits, symbolizing clean synaptic connections. Without any accumulation of beta-amyloid plaques, the brain can withstand smooth communication between neurons, which is crucial for memory, learning, and problem-solving[29]. This clear synaptic network represents what unhealthy sleep patterns do to the brain[30] The image on the right depicts neurons surrounded by beta-amyloid plaques, which accumulate in the spaces between them. These plaques disrupt synaptic connections, leading to impaired communication between neurons[42].

Discussion

Diet, exercise, and sleep are very influential in the prevention and management of AD. A diet with lots of omega-3 fatty acids, antioxidants, and vitamins is, therefore, very rewarding to one's protection from cognitive decline, alongside regular PA and good sleep. This will reduce the rate of inflammation and promote good brain health to possibly slow down the course of this disease. Moreover, sleep health should be emphasized, as these disturbances not only promote AD pathology but can also serve as early markers for the disease. Overall, modifications in diet, exercise programs, and strategies for sleep optimization are promising interventions that bring improvement in cognitive abilities to people at risk. Going forward, these risk-reducing lifestyle modifications may be enlisted in public health policy as a formidable intervention against the worldwide burden of AD.

While AD continues to be a major challenge, focusing on lifestyle changes-a healthy diet, proper exercise, and better sleeping-may form a new and hopeful direction of public health policy. These CBT interventions have demonstrated promise in the general enhancement of reducing the advancement rate of neuroinflammation and oxidative stress. Unlike drugs, which are usually expensive and of limited benefits, lifestyle modifications provide an easier and more effective way of slowing down the disease process. They cannot reverse the cognitive decline at all or sometimes have expensive financial costs. Considering inadequacy, medications to deliver a potential benefit researching alternative techniques such as CBTs could provide a lot more feasible future management of symptoms.

There is a growing interest in how cognitive activities, such as reading, puzzles, and mind games, could slow the progression of AD and MCI. While these activities have been suggested in some studies to build cognitive reserve, further research should be done to explain the impact of cognitive stimulation on the brain. Determining the optimal types, frequencies, and length of these activities may help identify how they improve quality of life for persons living with AD. These activities stimulate cognitive functions such as memory, problem-solving, and critical thinking-activities that keep the brain working. As the brain is constantly being stimulated, such activities develop neuronal connections that can be resilient to neurodegenerative assault. This could, therefore, provide an insight into how cognitive activities function and maybe even further lead to new therapeutic strategies; hence, the need for more research in developing effective interventions not only in the management of AD but also in MCI.

Research Limitations and Future Directions

This study explores the role of behavioural, lifestyle, and cognitive therapies for non-pharmacological interventions in the management of Alzheimer's disease, with most presenting favourable outcomes. It is, however, important to highlight a few limitations influencing the scope of the research. One important barrier is that the absence of long, large-scale clinical trials prevents the ability to assess the possibility of efficacy that is enduring over time. Most of the studies that were published had small sample sizes and short periods of follow-up, which could be contributing factors to heterogeneity in results and limit generalizability. Also, due to the serious disparity regarding study techniques and participant demographics, including socioeconomic conditions, access to care, and baseline cognitive performance, results cannot be applicable.

It is not to say that all treatments work, although this research did not find any studies with negative outcomes within the set boundaries. Instead, the absence of conflicting findings within the scope of this review underlines the need for more diverse research into the future, in order to understand better the limitations and inconsistencies of any. Larger, more diverse sample sizes are needed for future research, and techniques need to be standardized in order to improve reliability. Moreover, broad clinical trials will be necessary to test long-term effects with the aim of finding out how non-pharmacological techniques can be sustainable and scalable. The lacuna thus can be filled up by future research, providing a more sophisticated understanding in the management of AD and helping to create evidence-based, long term care strategies.

Methods

This study did a systematic review of the literature to assess the effectiveness of various CBTs on early to mid-stage AD patients, whether dietary, fitness, or sleep interventions. References were taken mostly from PubMed; supplementary searches on Google Scholar and ScienceDirect have been conducted in order to increase coverage. The search strategy used Boolean operators and specific keywords like "Alzheimer's Disease" OR "Mild Cognitive Impairment" AND "diet", "Alzheimer's Disease" AND "fitness", and "Alzheimer's Disease" AND "sleep". Filters applied prioritized studies that were peer reviewed and within the last seven years, allowing for older studies if they were foundational or directly referenced in more recent literature. I focused on studies that explored non-drug approaches to improving cognitive function in Alzheimer's patients, specifically diet, exercise, and sleep. I excluded research that only looked at medications, lacked human clinical data, or did not measure cognitive outcomes clearly.

Acknowledgments

I would like to thank Yoo Jin Jung for her valuable mentorship and guidance throughout the research and writing process. I am also grateful to Timothy Belton for his mentorship and attentive editing, as well as to Anish Kumar for his insightful feedback and considerate suggestions. A special thank you to BioRender for providing the tools that enabled the creation of my figures.

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