

## Role of Mind Diet in Preventing Dementia and Alzheimers Disease

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**Abstract:** There is sufficient epidemiological evidence to suggest a strong correlation exists between diet, lifestyle, psychosocial factors, education, cardiovascular risk factors and the onset and consolidation of dementia and Alzheimer's disease (AD). Researchers have shown AD, diabetes, obesity, insulin resistance, and cardiovascular disease, hypercholesterolemia are strongly interrelated pathologies. Dementia is a global concern, placing a significant financial burden on patients, carers, and health care systems. Since its discovery almost a century ago, AD is still considered as an eminent cause of irreversible form of dementia in aging population and developed countries. Considering the absence of available disease-modifying therapies for dementia and AD treatment, there is a great need in preventing and delaying the onset of cognitive impairment in healthy older subjects. Strategies to delay metabolic, vascular risk burden and introducing nutritional interventions seem to be promising approaches to delay neurocognitive decline and reduce the risk of AD and other non-psychiatric co-morbidities. However the role of diet in cognitive health, mental well being and prevention of dementia in older adults is being extensively studied. Here we review the role of MIND (Mediterranean-DASH diet intervention for neurodegenerative delay) diet which has recently gained much attention due to its beneficial effects associated with overall diet composition, for the delay of dementia and AD.

**Keywords:** Diet; MIND; Cognitive decline; Cognitive impairment; Cognition; Healthy food, Alzheimer's disease.

### 1. INTRODUCTION

Alzheimer's disease (AD) is a neurodegenerative disorder clinically characterized by cognitive and behavioral impairment. It is progressive and currently incurable with a long presymptomatic period and is expected to increase in the coming decades due to progressive ageing of the world population [1]. Each year there are 9.9 million new cases of dementia worldwide, equal to about one new case diagnosed every 3.2s [2]. In 2015, approximately 47 million people worldwide were diagnosed with dementia, which is the seventh leading cause of death worldwide [3]. AD is a disease of later life, affecting one in four people 85 years of age or over, and the incidence is expected to rise in the coming years, with 131.5 million estimated cases by 2050 [4]. Apart from the burden directly linked with this progressive neurodegenerative condition, health care costs are increasing exponentially over time [5]. By delaying the disease onset by 5 years is enough to reduce the health expenditure and prevalence by half [6].

Increasing evidence indicates that the pathological hallmark of the disease [e.g., amyloid beta (A $\beta$ ) plaques and neurofibrillary tangle (NFT) formation] start appearing several decades before the onset of clinical symptoms [7,8] which is also known as pre clinical phase of AD where we can try preventive strategies to delay the process of cognitive decline. The apolipoprotein E  $\epsilon$ 4 (ApoE  $\epsilon$ 4) allele has been shown to be a major genetic risk factor for more rapid cognitive decline and earlier onset of AD [9, 10]. Metabolic syndrome (MetS) which includes diabetes, hypertension, obesity, high cholesterol, insulin resistance and cardiovascular disease are deeply interconnected with AD pathology [11]. Insulin resistance and high levels of plasma homocysteine, which represent MetS-related risk factors, have a major impact on cerebral physiology and morphology, A $\beta$  deposition and build-up of neuritic plaques [12]. Impairment of mitochondrial metabolism pathways and decrease in ATP production causes neuronal injury and represents an early

event in the progression of AD [13, 14]. Nonglycosylated full length and C-terminal truncated A $\beta$  precursor protein (APP) have been found to accumulate in neuronal mitochondria in AD affected subjects [15]. A $\beta$  overproduction causes mitochondrial damage, dysfunction of mitochondrial complexes I and IV, increase of reactive oxygen species, and depletion of adenosine triphosphate (ATP), which in neurons may lead to neurotransmission and axonal transport impairment and degeneration [16]. In summary the neuropathology and etiology of AD indicate that a complex series of molecular mechanisms is involved, including the amyloid hypothesis, abnormal mitochondrial function, oxidative stress, formation of reactive oxygen species, and neuroinflammation as the important mainstream molecular pathways. In turn, the neuroinflammatory cascade may be synergistically associated with immunosenescence and gut dysbiosis, intercepting the trajectories of the aging brain and the progression to dementia.

Whole brain atrophy rates range from 0.3 to 0.7% per year in healthy older adults [17]. Hippocampal volume decreases at a rate of 2–4% per annum in healthy aged individuals [18]. On contrary MRI in AD, reveals widespread cortical atrophy, particularly within medial temporal lobe structures including the amygdala, hippocampus, and parahippocampal gyrus [19], with whole brain atrophy rates ranging from 1 to 4% per year [17]. FDG-PET studies have shown that between 20 and 70 years of age, global cerebral metabolic rate decreases by 12–13% [20]. Pittsburgh Compound B (PiB)-PET is used to visualise and quantify deposition of the protein beta-amyloid (A $\beta$ ) within the living brain. Elevated levels of cerebral A $\beta$  in apparently healthy persons are found in 12% of those in their 60s, 30% of those in their 70s, and at least 50% of those over 80 years of age. Moreover, PiB-PET determined cerebral A $\beta$  levels correlate with rate of memory decline in healthy older adults consistent with the notion that A $\beta$  accumulation promotes neuronal death, which ultimately results in cognitive impairment [21].

Most healthy adults experience age related decline in a variety of cognitive domains including spatial orientation, episodic memory, speed of processing, language, decision-making abilities, judgment, and abstract thinking [22]. Subjective memory complaints (SMC) represent self-identified changes in memory and are strongly associated with ageing [23, 24]. They

may be considered as an early marker of subsequent cognitive decline in multiple domains, and are a possible precursor of mild cognitive impairment (MCI) and neurodegenerative disease such as Alzheimer disease (AD) and dementia [24–27]. Elevated levels of cortisol and lower levels of estrogen and testosterone have all been associated with the development of dementia [28–30]. Assessment of thyroid stimulating hormone levels is considered a standard screening test for the diagnosis of dementia, as both hyper and hypothyroidism can interact with brain metabolism and if found can be reversible [31].

Altogether, all these studies indicate that complex systemic, metabolic, and endocrine mechanisms are responsible in the etiopathogenesis of dementia and AD. Adding nonpsychiatric comorbidities to a wide spectrum of MetS-associated illness plus cardiovascular ailments, it is possible that nutritional patterns and habits may play a great role in the onset of cognitive decline and AD in different geographical location and population. With lot of epidemiological evidence in previous studies, researchers in this field suggest that modifiable lifestyle factors, including diet, could play an important role in maintaining brain and cognitive health. A healthy diet characterized by high consumption of plant foods, whole grains, olive oil and fish, and ensuring an adequate intake in various vitamins (vit A,B,C,D,E) and minerals could help to strengthen cognitive abilities by stimulating memory [32,33].

Plant-based, healthy dietary interventions have shown promising results also for the amelioration of gut dysbiosis [34], which has been associated with the etiopathogenesis of AD and amyloid formation [35,36]. In particular, the Mediterranean Diet (MD) has been associated with a better cognitive function and a lower risk of cognitive impairment, AD [37, 38,] and subjective cognitive function [39]. The Dietary Approaches to Stop Hypertension (DASH), initially designed to reduce elevated blood pressure, has also been associated with a better cognitive function in a number of studies [40–42].

Recently, a new concept of diet aimed at protecting the brain, namely the MIND diet, was developed [43]. The MIND diet includes many dietary components from the MD and DASH, as well as specific components such as green leafy vegetables and berries known for their neuroprotective role [44]. The MIND diet score was found to be a better predictor of cognitive

decline than either the MD or DASH diet scores in the Rush Memory and Aging Project [43]. Studies has showed that adherence to the MIND diet was associated with a better cognitive health [43, 45, 46] and a lower risk of AD [47].

MIND diet is a mix of components of the MD and the DASH diet, with some modifications to optimize brain health. It includes ten “brain healthy” food groups (green leafy vegetables, other vegetables, nuts, berries, and beans, whole grains, fish, poultry, olive oil, and wine) and five unhealthy food groups (red meats, butter and margarine, cheese, pastries and sweets, and fast fried foods) [43]. Adjibade et al., reported beneficial effect of the MIND diet on subjective memory complaint(SMC)and cognitive functions or dementia could be attributed to healthy components such as vegetables (especially green leafy vegetables), nuts, berries, beans, whole grains, fish, poultry, olive oil, and wine. Indeed, healthy foods accounted in the MIND diet provide high amounts of several nutrients, including carotenoids, vitamins B, C, D, E, and polyphenols [43, 44, 45]. Most of these nutrients have been shown to have anti-inflammatory properties and are required for normal physiological functioning of the brain [46,48,49]. Dietary fibers also play an important role in cognition, in particular via their influence on gut microbiota composition [50, 51].

Previous studies, including the milestone Rotterdam study, have pointed out the relevance of nutrition in counteracting brain neuro-degeneration [52]. Although several nutritional approaches have been considered as possible alternatives like low GI diet, Prudent healthy diet, Anti inflammatory diet to the currently existing drugs for AD, this line of research has only been partially explored and has not resulted in any solid evidence [53,54,56]. It is possible that most of the studies are done in USA till date with little evidence from other parts of the world like south East Asia, Africa etc. Recent advances has indicated that epigenetics can throw some light on our current understanding based on imaging , CSF markers, neuropathology on such a complex neuro-degenerative pattern. DNA methylation, histone modifications, and micro RNAs are the principal epigenetic mechanisms involved in AD pathophysiology [55,57,58]. Nutrition is now believed by many workers to be a modifiable factor that seems to strongly impact on AD pathology by modulating its phenotypic expression [57, 58].

## 2. DISCUSSION

Etiopathogenesis of dementia and AD is very complex. Currently available pharmacological treatments for AD and dementia can only temporarily stabilize symptoms in a minority of patients, without effects on long-term prognosis or survival. The clinical evidence that has been collected in recent years for DASH, MD, and more recently MIND able to modify the natural history of neurodegenerative pathologies, in particular AD, thereby preventing their development or slowing down their progression. The strength of these dietary approaches lies in their multifactorial composition. In fact, nutrient-dense foods can interact with each other with potentially synergistic effects on different metabolic and cellular signaling pathways, leading to neuroprotection and maintaining health of individual neurons .But there are considerable methodological difficulties in conducting in vitro and in vivo studies to document the effects of such dietary patterns on the brain, due to the intrinsic complexity of a whole diet, the micro- and macronutrient composition, the importance of food cooking methods, and unavailability in different geographical areas.

Animal studies suggest possibility that these dietary patterns might influence A $\beta$  or Tau metabolism, and require further assessment and confirmation [59, 60]. At a systemic level, they can also indirectly improve cognition by reducing cardiovascular risk factors such as lipid levels, blood glucose, and blood pressure [61, 62]. The core mechanisms studied extensively till date are neurovascular dysfunctions [63-67], oxidative stress [68-74], neuroinflammation [75-79], gut microbiota dysbiosis [80-84], adult hippocampal neurogenesis [85-87]. A few recent studies, however, have suggested that the prevalence of dementia might be subject to change over time and that in higher income countries, both its prevalence and incidence might have decreased over the past two decades [88-90], owing to improved prevention of vascular morbidity and higher levels of education [88].

With regards to nutrition, attention to overall diet composition rather than single nutrient supplementations seems to be a more sound and effective approach for the prevention and management of AD risk, in light of the fact that nutrients present in foods synergistically interact with each other may it be macro or micro nutrient. High adherence to healthful dietary patterns MIND diet in particular, has shown to

be associated with lower rates of cognitive decline and reduced risk of developing dementia and AD [46,91-97], Hosking et al recently reported cognitively protective effects of the MIND diet generalize to a population outside the United States also and greater MIND diet adherence was associated with 19% reduced odds of developing clinically diagnosed MCI/dementia after 12-years of follow-up and with 53% reduction in the odds of impairment.

### 3. CONCLUSION

As we have discussed in our review dementia and particularly AD has many interlinked risk factors, it is hardly possible to control all of them together at a point of time so preventive strategy is must. There is strong epidemiological evidence for a protective role of the neuroprotective dietary patterns on cognitive decline and dementia in older persons. Cross-country variation in food supply, dietary behaviors, and other lifestyle factors determine the generalizability of dietary effects on risk for cognitive impairment and dementia. Enthusiasm of MIND diet has led researchers to test it in post a stroke patient who slows cognitive decline (98) and Parkinsons disease (PD) where it reduced and delayed progression of parkinsonism in old age [99]. Dietary patterns in early onset dementia remains unknown. Clearly, more studies are required in future in other populations and geographic locations to further evaluate the protective effects.

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