MINI REVIEW

Alzheimer's disease, mild cognitive impairment, and the Mediterranean diet are all linked to each other

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ABSTRACT

Alzheimer's Disease (AD) is the leading cause of dementia in Western countries, and its prevalence is increasing as society becomes more long-lived. It's critical to look into elements that may be protective or impact the progression of Alzheimer's disease in order to intervene and lessen the incidence and advancement of the disease. The goal of this study was to conduct a systematic review and meta-analysis to see how a better adherence to the Mediterranean Diet (MD) affects Mild Cognitive Impairment (MCI) and Alzheimer's Disease (AD).

Key Words: *Alzheimer's disease; Mediterranean diet*

INTRODUCTION

Alzheimer's Disease (AD) is the most common form of dementia, accounting for 60% to 70% of all cases worldwide. The global prevalence of Alzheimer's disease is anticipated to triple by 2050 as a result of improved living standards and increased life expectancy, resulting in a slew of societal and economic implications [1]. Dementia is a public health priority according to the World Health Organization. The World Health Assembly adopted the "Global action plan on the public health response to dementia 2017-2025 targets" in May 2017, with the goal of improving the quality of life for dementia patients and carergivers while also reducing the disease's burden on communities and countries [2]. Adoption of healthy food patterns is one of its risk-reduction goals. The plan also emphasizes the importance of implementing multi-sectorial evidence-based interventions that are affordable to the majority of people in order to encourage them to make proactive healthy lifestyle changes and reduce exposure to modifiable risk factors, thereby slowing the disease's rapid spread. There is currently no preventive or curative treatment for Alzheimer's disease. Antipsychotic and antidepressant treatments can help regulate behavioral and psychological symptoms, while cholinesterase inhibitors and N-methyl-D-aspartate receptor antagonists can help lower some cognitive symptoms and are the only farms approved by the Food and Drug Administration as AD meds. Nonetheless, disease-modifying medications are still being researched; therefore providing scientific knowledge on possible risk and protective factors for Alzheimer's disease is quite important. Non-modifiable AD risk variables include age (the strongest known AD risk factor), being female, and having the APO-E4 gene allele, which interact with environmental and biological factors to change AD risk [3-5]. One out of every three new instances of Alzheimer's disease is thought to be caused by modifiable risk factors. Physiological risk factors (mid-life hypertension, diabetes, obesity, or inflammation), concurrent disorders (peripheral arterial disease, reduced cardiac output, or depression), and lifestyle factors are some of the modifiable risk factors with scientific support (education, smoking, physical activity or diet) [6]. The Mediterranean Diet (MD) is characterized by a high intake of vegetables, fruits, legumes, nuts, and whole grains, as well as olive oil as the main source of fat, moderate consumption of fish, low to moderate consumption of dairy products, low intake of poultry, meat, and saturated fatty acids, and moderate consumption of alcohol only during meals [7]. MD has been shown to play a role in the primary and secondary prevention of Cardiovascular Disease (CVD) through enhancing cardio-metabolic health and glycemic management in numerous studies. Furthermore, research evidence shows that MD has cognitive impacts. In non-demented older persons over 60 years of age, a recent meta-analysis found that strict adherence to MD lowered the risk of global cognitive decline. MD adherence is part of a lifestyle pattern shaped by societal, educational, familial, and economic circumstances. As a result, rather than a single mechanism, the protective effect of MD in AD could be explained by a complex network of pathways in which dietary components and other lifestyle factors interact synergistically and additively [8-10]. These interactions may help to reduce AD risk both directly (*via* neuroprotective effects) and indirectly (as protective factors against cardiovascular and metabolic illnesses, which are risk factors for AD). In this vein, previous reviews and meta-analyses have linked MD to enhanced cognitive function, a lower risk of Moderate Cognitive Impairment (MCI), and a lower risk of dementia and Alzheimer's Disease (AD). Changes in AD biomarkers (such as-amyloid (A) deposition, Tau phosphorylation, cortical thickness, or glucose metabolism in the brain) also appear to be 10-20 years ahead of clinical AD symptoms.

As a result, preventative lifestyle interventions should be implemented as early as feasible in life. In fact, implementing preventive interventions that reduce the incidence of Alzheimer's disease or delay its course could cut the number of cases by roughly 9 million over the next 40 years. A meta-analysis looked at the association between diet and the hallmark AD biomarkers (tau and beta-amyloid) and discovered that following the MD reduced the burden of AD biomarkers considerably. According to previous systematic reviews and meta-analyses, increased adherence to MD was inversely related to cognitive deterioration [10]. However, they only looked at a few trials, or they were just interested in MCI and dementia in general, including non-AD dementia. As a result, the goal of this systematic review is to examine the most recent and comprehensive scientific evidence to support MD adherence as a preventive factor for AD and its impact on cognition from a clinical standpoint [11]. We conducted an updated systematic review and dose-response meta-analysis of all studies examining the impact of MD on cognition in people at risk for AD, presenting MCI or AD patients, in order to focus exclusively on the effects of MD adherence on cognition and the incidence or progression to MCI or AD (excluding other types of dementia or diet) [12].

DISCUSSION

The goal of this systematic review and meta-analysis was to determine whether adhering to MD is a favorable factor in reducing the development of MCI and AD based on scientific data. Other systematic reviews and meta-analyses have found that the highest Mediterranean diet score is inversely related to cognitive decline; Wu et al. found a trend of a linear relationship between the Mediterranean diet score and the incident risk of cognitive disorders in their meta-analysis, but the link was not significant. Sigh et al. concluded that increased adherence to the MD was related with a lower chance of acquiring MCI and AD, as well as a reduced risk of advancing from MCI to AD, in addition to MCI and AD. Prospective cohort studies with extended follow-up have been conducted to date. In order to consolidate more information and to focus entirely on AD type dementia and the prior stage of the disease (MCI), we conducted a qualitative analysis of the studies in this field, followed by a detailed and thorough update from a clinical

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standpoint. In a second phase, we investigated the impact of the MD and the risk of acquiring MCI or AD by doing a quantitative study. Seven studies offered strong evidence in regard to MD adherence as a protective factor in relation to AD risk, incidence, or progression from MCI. According to research, preclinical biomarkers for Alzheimer's disease could show 20-30 years before clinical manifestations of the disease. Furthermore, it is thought that changes in cortical thickness occur as a result of a reduction in glucose metabolism and the accumulation of A in the brain. It was found that substantial MD protective effects in MRI volumetry measurements, studies in glucose metabolism in the brain, five studies in A load in the brain, one research in Tau tangles deposition, and one study in A and Tau in the cerebrospinal fluid. It has been proposed that the aetiology of Alzheimer's disease evolves from neurological metabolic failure and pathological deposition of A without cognitive impairment to structural alterations in the brain, culminating in AD cognitive symptoms. There are non-modifiable factors that have been shown to enhance the risk of Alzheimer's disease and have been included as covariates in some studies. APO-E4 genotyping was included in nineteen articles in reference to them.

The consequences of increased cortical thickness in people with high MD adherence were much more pronounced in those who did not inherit the APO-E4 gene. Because the APO-E4 gene has been proposed as a possible modulator of metabolic activities, its existence may impair the protective effects of MD adherence in humans. With the exception of five research that featured CN middle-aged subjects, the majority of the investigations included older persons. The limitations of available data should be considered when interpreting the results of our meta-analysis. Modifiable risk factors which operate synergistically and additively to change AD risk are especially relevant in modulating MD adherence protective effects. Cardiovascular risk factors, exercise, inflammatory indicators, BMI, years of education, smoking, and comorbidities have all been taken into account in certain research when assessing the major findings. Furthermore, due to the difficulties in distinguishing Alzheimer's disease from vascular dementia, some studies excluded people with cardiovascular disorders. Modifiable risk factors which operate synergistically and additively to change AD risk are especially relevant in modulating MD adherence protective effects. Cardiovascular risk factors, exercise, inflammatory indicators, BMI, years of education, smoking, and comorbidities have all been taken into account in certain research when assessing the major findings. Furthermore, due to the difficulties in distinguishing Alzheimer's disease from vascular dementia, some studies excluded people with cardiovascular disorders. MD's significance in the prevention of Alzheimer's disease may be explained by underlying molecular mechanisms. Despite the fact that they are yet unclear, some research propose four distinct mechanisms that may interact to explain how MD protects against Alzheimer's disease: MD may be neuroprotective due to its metabolic benefits, as evidenced by studies that reveal a link between increased MD adherence and improved glucose metabolism in the brain. The role of vascular routes as mediators of MD effects has also been investigated. MD has been shown to minimize cardiovascular risk factors, which are risk factors for Alzheimer's disease. Due to the anti-oxidative qualities of several of its dietary components, MD, on the other hand, has been shown to lower oxidative stress. Finally, MD's anti-inflammatory capabilities may play a key part in its neuroprotective benefits.

Furthermore, other lifestyle habits, such as exercise, that have been hypothesized as independent protective factors for Alzheimer's disease, could interact with MD practices, amplifying their favorable effects. The heterogeneity and variability of the articles included in this review, such as differences in sample size or study designs (the majority of which are cross-sectional), which limit the ability to infer causality; the use of various FFQ and procedures to estimate MD adherence; differences between outcomes measured and techniques used for these purposes; and differences in dietary habits in the regions where studies were conducted, are the main limitations found in this review. Another potential flaw is that the instruments used to determine MD adherence are based on self-reported data, which could result in bias.

CONCLUSION

According to this meta-analysis, higher MD adherence lowers the incidence of MCI and AD. These findings highlight the importance of public health activities and legislation to encourage the adoption of MD practices as a dementia-prevention intervention. These dietary changes, along with other healthy lifestyle and cardiovascular risk factor therapies, should be implemented at a young age as a brain-protective strategy.

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