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Food and nutrition in the prevention of dementia–A mini-review

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Abstract

Dementia has become a widespread public health issue. Preventing Alzheimer's disease and other forms of dementia is a significant challenge. Observational studies suggest that certain nutritional factors may provide cognitive benefits. However, nutritional epidemiology studies have only inconsistently found an association between food or nutrition and cognitive decline, dementia, or Alzheimer's disease. Clinical trials of dietary and nutritional interventions have generally been unable to demonstrate a reduction in cognitive impairment. The available intervention results were usually small, heterogeneous, and statistically insignificant. Long-term interventions may be necessary to demonstrate an effect. Nevertheless, our understanding of risk factors for dementia and potential preventive measures is improving. There is high potential for preventing dementia; nearly half of cases could theoretically be prevented by eliminating 14 modifiable risk factors. Four of these risk factors—including hypertension, type 2 diabetes, high cholesterol, and obesity—are nutrition-related. Lifestyle changes related to diet and nutrition that aim to decrease the risk of dementia should include maintaining a systolic blood pressure of 130 mmHg or less starting at age 40, detecting and treating high LDL cholesterol from midlife, and maintaining a healthy weight and treating obesity, which also helps prevent diabetes.

Keywords: Alzheimer's disease; Dementia; Food; Nutrition; Prevention.

1. Introduction

Dementia is defined as an acquired impairment in multiple cognitive functions. The cognitive impairments experienced by people with dementia are often severe enough to affect their social and professional lives, leading to challenges in daily living and reducing their quality of life. In 2021, over 55 million people worldwide were living with dementia. This figure is expected to rise significantly, reaching approximately 80 million by 2030 and 140 million by 2050. Dementia has therefore become a widespread public health issue that places a significant individual, societal, and financial burden on those affected and their communities (Alzheimer's Disease International, 2015).

Dementia is not a disease, rather it is a syndrome, a set of behavioral and cognitive symptoms caused by various diseases or disorders that affect the brain. The most common form of dementia is Alzheimer's disease (AD), which is caused by neurodegeneration and accounts for about 60% of diagnoses. However, many other conditions can also lead to dementia. Vascular dementia, for example, is caused by restricted blood flow due to strokes, which reduce the delivery of oxygen and glucose to the brain, resulting in global cognitive impairment. Other diseases that include dementia are Lewy body dementia, frontotemporal dementia, Creutzfeldt-Jakob disease, and Parkinson's disease dementia. Dementia is often associated with more than one neuropathological condition; typically, Alzheimer's disease occurs alongside vascular brain pathology (mixed dementia).

Alzheimer's disease is a progressive neurodegenerative disorder involving the gradual deterioration of cognitive abilities, including memory, learning, perception, attention, executive func-

tions, and language (Lange et al., 1995; Weintraub et al., 2012; Scheltens et al., 2016). It is associated with a significant decline in daily activities, and symptoms typically appear late in life after a prolonged preclinical phase of progressive neuropathology spanning many years, or even decades.

Alzheimer's disease is a proteinopathy, meaning it is associated with the aggregation and accumulation of misfolded proteins (Kosik and Shimura, 2005; DeTure and Dickson, 2019). The neuropathological hallmarks of progressive neurodegeneration in Alzheimer's disease are the presence of extracellular amyloid deposits (senile plaques), which contain misfolded amyloid-β peptides, and the formation of intracellular aggregates of hyperphosphorylated tau proteins (neurofibrillary tangles) (Butterfield and Lauderback, 2002; Crews and Masliah, 2010; DeTure and Dickson, 2019; Price et al., 1991). Prior to the onset of Alzheimer's disease, numerous brain regions, primarily in the frontal and temporal lobes, show significant atrophy. The neurodegenerative process leading to severe brain atrophy can take many years. Oxidative stress and chronic neuroinflammation are two major mechanisms involved in the pathophysiology of Alzheimer's disease. The aggregation of amyloid-β produces large quantities of free radicals, which cause oxidative stress and accelerate neuronal dysfunction. Ultimately, this leads to widespread brain atrophy (Butterfield and Lauderback, 2002; Querfurth and LaFerla, 2010).

The administration of N-methyl-D-aspartate (NMDA) receptor antagonists and acetylcholinesterase (AChE) inhibitors has shown no significant efficacy in treating AD (Scheltens et al., 2016). The advent of disease-modifying therapies for dementia is a long-awaited breakthrough. However, the results of available trials vary from modestly positive to neutral, and their clinical implications are unclear. Preventing AD is a major public health challenge (Lange, 2018a, 2021). New interventions that can slow the progression of the disease and delay its onset are needed.

2. Nutrition in dementia

Studies have suggested that food and nutrition may play a role in Alzheimer's disease and other types of dementia (Gustafson et al., 2015; Huhn et al., 2015). Approaches to addressing Alzheimer's disease through nutrition include the Mediterranean diet (Lange, 2019), ketogenic diets (Lange et al., 2017), and medical foods (Lange et al., 2019a; Lange et al., 2019b). Various food bioactives, such as polyunsaturated fatty acids (Lange, 2020b, 2020a) and natural antioxidants (Lange and Li, 2018; Lange et al., 2020; Lange, 2018b), have been proposed as potential methods for reducing the risk of dementia.

Dementia has long been considered an inevitable consequence of aging. However, there is a growing recognition that modifiable lifestyle behaviors can influence one's risk of developing dementia (Lange, 2018a; Livingston et al., 2020). Recent evidence indicates that up to 45% of dementia cases could be prevented or delayed by addressing 14 risk factors throughout one's life. These risk factors include lower levels of education, hypertension, hearing impairment, smoking, obesity, depression, physical inactivity, diabetes, limited social interaction, excessive alcohol consumption, traumatic brain injury, exposure to air pollution, untreated vision loss, and high LDL cholesterol (Livingston et al., 2024). Hypertension, diabetes, high cholesterol, and obesity are among the risk factors for dementia related to nutrition.

Interestingly, the incidence of dementia is decreasing in high-income countries, likely due to improvements in education, nutrition, healthcare, and lifestyle. This decrease in cognitive decline and dementia may be an unintended consequence of improved vascular health and healthier lifestyles. For example, reducing vascular damage by treating high blood pressure and reducing smoking has likely contributed to the decline in age-related dementia cases.

2.1. Hypertension

Studies have found that consuming large amounts of salt increases blood pressure (Feng et al., 2017; Garfinkle, 2017) and raises the risk of cardiovascular disease (Takachi et al., 2010). However, modest reductions in salt intake have been shown to lower blood pressure in people with hypertension (He et al., 2005). Persistent high blood pressure in midlife is associated with an increased risk of late-life dementia.

One study examined 1,440 individuals and found that elevated systolic blood pressure (≥140 mmHg) in midlife (mean age 55 years) was associated with an increased risk of developing dementia over an 18-year follow-up period (McGrath et al., 2017). Another study of 8,639 individuals found that a systolic blood pressure reading of 130 mmHg or higher at age 50, but not at ages 60 or 70, was associated with an increased risk of dementia (Abell et al., 2018). Among individuals aged 45 to 61 years with persistent systolic blood pressure readings of 130 mmHg or higher, the risk of dementia was elevated compared to individuals without hypertension, even among those free of cardiovascular disease (Abell et al., 2018).

The mechanisms underlying the link between midlife hypertension and dementia may include reduced brain volume and increased white matter hyperintensity, though not amyloid deposition (Lane et al., 2019).

Two meta-analyses of randomized controlled trials have found that antihypertensive medication protects against cognitive impairment and dementia (Hughes et al., 2020; Peters et al., 2022). However, a meta-analysis with a short follow-up period showed no such protective effect (Cunningham et al., 2021). A meta-analysis of individual participant data from 17 studies (mean age: 72.5 years; mean follow-up period: 4.3 years) revealed that individuals with untreated hypertension had a higher risk of developing dementia than healthy controls. However, this risk was reduced or eliminated with treatment (Lennon et al., 2023). A meta-analysis of 31,090 adults without dementia at baseline and with a follow-up period of at least five years found that individuals with hypertension who took antihypertensive medication were at a lower risk of dementia than those who did not (Ding et al., 2020).

In summary, high blood pressure in middle age increases the risk of developing dementia. This effect appears to be particularly pronounced if hypertension remains untreated for a long time.

2.2. Type 2 diabetes

Diabetes is one of the most well-documented risk factors for dementia. Individuals with type 2 diabetes in middle age (though not necessarily in older age) are at an increased risk of developing dementia compared to those without diabetes. A prospective cohort study of 10,095 participants found that the risk of dementia increased with each five-year decrease in age at type 2 diabetes diagnosis up to age 70. Poorly controlled diabetes increases the risk of dementia (Barbiellini Amidei et al., 2021).

The mechanisms involved in the association between diabetes and increased dementia risk likely include a strong vascular component, as microvascular and macrovascular complications (e.g., stroke) are prevalent in individuals with diabetes (Xue et

al., 2019). Furthermore, insulin resistance is a common molecular mechanism linking diabetes and AD. Insulin resistance has been found to lead to increased amyloid β toxicity, tau hyperphosphorylation, oxidative stress, and neuroinflammation (Michailidis et al., 2022).

In summary, the overall improvement of diabetes control, but not the achievement of very low blood sugar or weight loss without also improving diabetes control, may result in a decrease in the risk of dementia (Livingston et al., 2024).

2.3. LDL cholesterol

High-quality evidence indicates that having high LDL cholesterol levels in midlife increases the risk of developing dementia. One study examined 1,189,090 participants and found a link between high LDL cholesterol levels (over 3 mmol/L) and an increased risk of dementia (Mukadam et al., 2023). Another large cohort study of over 1.8 million people followed for an average of 7.4 years found that higher baseline LDL cholesterol levels were associated with an increased risk of all-cause dementia (Iwagami et al., 2021). A meta-analysis of three cohort studies involving 1,138,488 participants revealed that a 1 mmol/L increase in LDL cholesterol corresponded to an 8% higher incidence of all-cause dementia among adults under 65 years old who were monitored for over 12 months (Wee et al., 2023). Another cohort study of 94,184 individuals, followed from an average age of 58, found that those who did not adhere to dietary guidelines (consuming fewer than three weekly servings of fruits and vegetables and frequently consuming sugar-sweetened beverages and processed meats) were more likely to have high LDL cholesterol (Kjeldsen et al., 2022). After a median follow-up period of nine years, individuals with low adherence to these guidelines were more likely to develop types of dementia other than Alzheimer's disease than those with high adherence. Those taking lipid-lowering drugs were not at an increased risk of dementia. A meta-analysis of 36 cohort studies found that people taking statins to lower their LDL cholesterol were less likely to develop all-cause dementia or Alzheimer's disease than people with untreated high cholesterol (Olmastroni et al., 2022).

One possible mechanism linking LDL cholesterol to dementia is the link between high brain cholesterol levels and an increased risk of stroke, as well as the accumulation of amyloid β and tau proteins in the brain (Wee et al., 2023).

In summary, a high concentration of LDL cholesterol in midlife is associated with an increased risk of dementia. However, the quality of the available evidence supporting the efficacy of managing dyslipidemia in midlife to reduce the risk of cognitive decline and dementia is low (Livingston et al., 2024).

2.4. Obesity

Midlife obesity has been identified as a risk factor for dementia. A review of 19 longitudinal studies including 589,649 people aged 35–65 with follow-up periods of up to 42 years found that obesity (BMI ≥ 30) was associated with late-life dementia, while being overweight (BMI 25–30) was not (Albanese et al., 2017). Another meta-analysis of individual-level data from 1.3 million adults found that a higher BMI measured prior to probable preclinical dementia onset was associated with an increased dementia risk (Kivimäki et al., 2018). A systematic review and meta-analysis of 14 prospective studies involving 77,890 participants reported that midlife obesity was associated with subsequent all-cause dementia (Qu et al., 2020). A study of central obesity involving 5,060,687 participants from 16 cohort studies showed that a larger waist circumference was associated with a higher risk of cognitive impairment and dementia (Tang et al., 2021).

As obesity is associated with hypertension and diabetes and is prevalent among individuals who exercise infrequently, the correlation between obesity and dementia may be influenced by other dementia risk factors. However, most studies adjusted for health conditions such as hypertension, stroke, blood lipid levels, and diabetes, which should minimize the effect of these intermediaries.

A meta-analysis of weight-loss intervention studies including 13 longitudinal studies (n = 551) and seven randomized controlled trials (n = 468) of overweight (BMI 25–30) or obese (BMI ≥30) participants found that even a modest 2-kg intentional weight loss among trial participants was associated with improvements in cognition at a median follow-up period of six months (Veronese et al., 2017). These results suggest that healthy behaviors may have beneficial effects even if weight loss is insufficient to alter obesity status.

The mechanisms through which obesity contributes to the risk of developing dementia remain unclear. Potential pathophysiological links between obesity and dementia include chronic, low-grade systemic inflammation, vascular dysfunction, and metabolic disruption (insulin resistance) that damages brain structure and function (Wong Zhang et al., 2023)

In summary, obesity, especially in middle age, increases the risk of developing dementia later in life. However, moderate, permanent weight loss can significantly reduce this risk.

3. Other potential dietary factors

Investigating the role of individual dietary components in preventing dementia is challenging, and existing studies have produced inconsistent results (Livingston et al., 2020). An unhealthy diet may be a modifiable risk factor for dementia. However, people often consume a mixture of healthy and unhealthy foods and beverages as part of their lifestyles. Thus, the observed effects of nutrition on the brain may be independent of or correlate with other healthy behaviors (Yassine et al., 2022).

Studies in nutritional epidemiology have found inconsistent associations between diet and cognitive decline, dementia, or Alzheimer's disease. Clinical trials of dietary and nutritional interventions have generally been unable to demonstrate a reduction in cognitive impairment. The available intervention results were usually small, heterogeneous, and statistically insignificant (Lange et al., 2022).

Recently, the Mediterranean diet and consumption of ultra-processed foods have received particular attention for their potential effects on cognitive decline and dementia. These topics are discussed in more detail below.

3.1. Mediterranean diet

The effects of the Mediterranean diet on cognitive decline and dementia remain unclear. Because the World Health Organization (WHO) was uncertain about the effects of this diet on dementia risk, they issued a conditional recommendation in 2019 (World Health Organization, 2019). More recently, a systematic review and meta-analysis of 16 cohort studies with follow-up durations ranging from 2.2 to 41 years found that a high-quality diet was associated with a lower risk of dementia than a low-quality diet (Liu et al., 2020). Another meta-analysis of three cohort studies

with 224,049 participants found that a higher MIND diet score (Mediterranean-DASH Intervention for Neurodegenerative Delay; a combination of the Mediterranean diet and specific healthy foods) was associated with a lower risk of dementia (Chen et al., 2023). A systematic review reported that the Mediterranean diet has a protective effect against global cognitive decline (in 10 of 21 studies), incident dementia (in 3 of 8 studies), and Alzheimer's disease (in 2 of 4 studies) (Townsend et al., 2023). However, a prospective cohort study of 28,025 midlife individuals (median follow-up: 19.8 years) found that adherence to dietary recommendations or a modified Mediterranean diet adjusted for cultural variations was not associated with a reduced risk of dementia, Alzheimer's disease, or vascular dementia (Glans et al., 2023). Another study of 60,298 people (mean age at baseline: 63.8 years; mean follow-up: 9.1 years) showed that higher adherence to the Mediterranean diet was associated with a lower risk of dementia than lower adherence, regardless of genetic predisposition (Shannon et al., 2023). Additionally, a cohort study of 581 older adults (mean age at initial evaluation: 84.2 years) revealed an inverse correlation between Mediterranean and MIND dietary patterns and amyloid β load, phosphorylated tau tangles, and overall Alzheimer's disease pathology at autopsy (Agarwal et al., 2023).

3.2. Ultra-processed food

Ultra-processed foods are industrial creations derived from natural foods or synthesized from other organic compounds. They often contain preservatives, flavorings, and colorings. Examples include fast food, instant soups, and ready-to-eat or microwaveable meals. These foods tend to be high in sugar, salt, saturated fat, and trans fat while being low in fiber and protein. The definition of ultra-processed foods varies across studies and is vague.

A systematic review and meta-analysis of observational studies reported that high consumption of ultra-processed foods is associated with an increased risk of dementia (Henney et al., 2024). This suggests that ultra-processed foods could contribute to cognitive decline. However, since no statistically significant relationship was found between moderate intake of these foods and dementia prevalence, a robust dose-response relationship between quantity consumed and dementia could not be demonstrated.

A cross-sectional study of 3,632 people aged 60 years or older found no association between overall cognitive performance or memory and the percentage of daily dietary energy intake from ultra-processed foods (Cardoso et al., 2022). However, a longitudinal study of 10,775 participants revealed that individuals in the top three quartiles for ultra-processed food consumption exhibited faster rates of global cognitive and executive function decline compared to those in the lowest quartile (Gomes Gonçalves et al., 2023). However, the short follow-up period of these studies means that reverse causation bias cannot be ruled out.

There may be a direct relationship between ultra-processed food consumption and dementia due to the high energy density and poor macronutrient and micronutrient profiles of these foods (high in saturated fat, trans fat, and refined carbohydrates, and low in fiber and sodium). Foods high in saturated fat and salt and low in fiber can alter gut microbial diversity, potentially causing pathogenic changes in the brain via the gut-brain axis. These foods may also contain artificial flavors and other chemical additives that could damage nerve cells.

Additionally, there may be an indirect relationship between ultra-processed foods and dementia through the development of cardiovascular risk factors such as hypertension, hyperlipidemia, obesity, and type 2 diabetes.

In conclusion, a diet high in fruits and vegetables (e.g., the Mediterranean diet) and low in ultra-processed foods has many health benefits and affects risk factors for dementia, such as hypertension, type 2 diabetes, and obesity. However, there is insufficient evidence to demonstrate the beneficial effects of this diet on dementia prevention.

4. Conclusion

Our understanding of the risk factors for dementia and potential preventive measures is improving. There is great potential to prevent dementia; nearly half of cases could theoretically be prevented by eliminating 14 modifiable risk factors. Four of these risk factors, namely, high blood pressure, diabetes, high cholesterol, and obesity are nutrition-related.

Clinical trials of nutritional and dietary interventions have generally been unable to demonstrate a reduction in cognitive impairment. The available intervention results were usually small, heterogeneous, and statistically insignificant. Long-term interventions may be necessary to demonstrate an effect.

Lifestyle changes related to diet and nutrition that aim to decrease the risk of dementia should include maintaining a systolic blood pressure of 130 mmHg or less beginning at age 40, detecting and treating high LDL cholesterol from midlife, and maintaining a healthy weight and treating obesity as early as possible to also help preventing diabetes.

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