Nutrition and Cognition in Aging Adults



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KEYWORDS

- Cognitive decline Dementia Nutrients Antioxidants Vitamins
- Polyunsaturated fatty acids

KEY POINTS

- Numerous longitudinal observational studies have suggested that nutrients, such as antioxidants, B vitamins, and ω-3 fatty acids, may prevent cognitive decline or dementia.
- There is very little evidence from well-sized randomized controlled trials (RCTs) that nutritional interventions can benefit cognition in later life.
- Nutritional interventions may be more effective in individuals with poorer nutritional status
 or as part of multidomain interventions simultaneously targeting multiple lifestyle factors.
- Further evidence, notably from RCTs, is required to prove or refute these hypotheses.

INTRODUCTION

The prevalence of dementia, including its most common form, Alzheimer disease (AD), is currently estimated at 36 million people worldwide. Given the expected increase in the number of dementia cases worldwide in the coming years due to demographic aging, finding interventions able to prevent cognitive decline or dementia is a currently a research priority.

Numerous observational studies have suggested a relationship between lifestyle factors, including nutrition and diet, and cognitive function in aging adults (**Table 1**). Some micronutrients, including the B vitamins, antioxidant vitamins, certain essential minerals, and essential fatty acids, as well as macronutrients and dietary patterns, are thought to play a key role in cognitive function, with a large base of evidence from numerous mechanistic animal studies.³

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Table 1 Types of studies used to evaluate the effect of nutrition on cognition					
	Physiopathology	Prospective Observational Studies	Systematic Review/ Meta-Analysis of Prospective Observational Studies	RCTs	Systematic Review/Meta- Analysis of RCTs
Antioxidants	Χ	Х	Χ	Χ	
Polyphenols	Χ	Х	_	Χ	
PUFA	Χ	Х	Χ	Χ	Χ
B Vitamins	Χ	Х	Χ	Χ	Х
Vitamin D	Χ	Х	Χ		
Calorie restriction	Х	_	X	_	_
MeDi	Х	Х	Х	Х	_

The aim of this article is to give an update on the current knowledge regarding the role of nutrition in the prevention of cognitive decline or dementia, based on the results of prospective studies, in particular, randomized controlled trials (RCTs), and to highlight future research perspectives.

SUMMARY OF FINDINGS

The findings from prospective observational studies and RCTs are described below, and the types of studies conducted for each type of nutrient or dietary pattern are summarized in **Table 1**.

Antioxidants

Because the brain is an organ with a high metabolic rate, oxidative stress, which may influence neurodegeneration and neuronal death in AD, 4 is a common phenomenon in its neural tissues. 5 Exogenous antioxidants, including vitamins A (eg, β -carotene), C, and E (tocopherols), and trace minerals, such as manganese, copper, selenium, and zinc, may protect against oxidative stress and could prevent cognitive decline or dementia.

Prospective observational studies

A meta-analysis of 7 cohort studies suggested that higher dietary intakes of vitamins E and C were significantly associated with a decreased risk of AD. 6 The association between β -carotene and risk of dementia was borderline significant. However, a more recent systematic review of 10 population-based cohort studies, which took into account methodological quality, found only limited evidence of a possible protective role of antioxidants on cognitive decline in older adults. 7 This review included biological measures of nutrient status or dietary intakes, and heterogeneous results meant that it was not possible to identify a specific antioxidant nutrient that could be recommended or would merit further study.

Randomized controlled trials

No systematic reviews or meta-analyses of RCTs testing antioxidants in a preventive setting were identified. One trial conducted in individuals with mild cognitive impairment (MCI) found no effect of vitamin E on conversion to AD-dementia after 3 years.⁸ In cognitively healthy populations, the cognitive effects of antioxidants have mainly been tested in add-on studies in trials initially designed for other purposes, and

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