

Dietary changes appear to affect levels of biomarkers associated with Alzheimer's disease

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Following a low–saturated fat and low–glycemic index diet appears to modulate the risk of developing dementia that proceeds to Alzheimer's disease (AD), although making a switch to this dietary pattern may not protect those already experiencing cognitive difficulty, according to a report in the June issue of *Archives of Neurology*.

Previous research has suggested multiple links between diet and cognitive ability, the authors note as background information. Health conditions in which insulin resistance (the body's inability to use insulin effectively) is a factor—obesity, type 2 diabetes, cardiovascular disease and high cholesterol levels—have also been associated with "pathological brain aging." However, studies of specific foods have not found conclusive evidence of an influence on Alzheimer's risk. "Thus," the authors write, "a more promising approach to the study of dietary factors in AD might entail the use of whole-diet interventions, which have greater ecologic validity and preserve the nutritional milieu in which fat and carbohydrate consumption occurs."

Jennifer L. Bayer-Carter, M.S., from Veterans Affairs Puget Sound Health Care System, Seattle, and colleagues sought to compare a high–saturated fat/high–simple carbohydrate diet (a macronutrient pattern associated with type 2 diabetes and insulin resistance) with a low–saturated fat/low–simple carbohydrate diet; the interventions were named HIGH and LOW, respectively. The authors evaluated the effects



of these diets in 20 older adults who were healthy and 29 older adults who had amnestic mild cognitive impairment (aMCI), meaning they experienced some memory problems; the latter condition is often considered a precursor to AD. In a four-week randomized, controlled trial, 24 participants followed the HIGH diet and 25 followed the LOW diet. The researchers studied participants' performance on memory tests as well as their levels of biomarkers (biological substances indicative of AD), such as insulin, cholesterol, blood glucose levels, blood lipid levels and components of cerebrospinal fluid (CSF).

Results of the study were different for the group that had aMCI and the group of healthy participants. In the latter group, the LOW diet decreased some CSF biomarkers of AD as well as total cholesterol levels. However, among individuals with aMCI, the LOW diet increased levels of these biomarkers. Some changes to biomarkers, such as CSF insulin levels, were observed in both groups. Additionally, the LOW diet improved performance on delayed visual recall tests for both healthy and memory-impaired participants, but did not affect scores on other cognitive measures.

The findings indicate that "for healthy adults, the HIGH diet moved CSF biomarkers in a direction that may characterize a presymptomatic stage of AD," explain the authors. They believe that the different results in participants with aMCI may show that dietary interventions are not as effective in later stages of cognitive impairment. "The therapeutic effects of longer-term dietary intervention may be a promising avenue of exploration," the authors conclude. "In addition, identification of the pathophysiologic changes underlying dietary effects may reveal important therapeutic targets that can be modulated through targeted dietary or pharmacologic intervention."

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