

The Role of Nutrient-Based Epigenetic Changes in Buffering Against Stress, Aging, and Alzheimer's Disease



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KEYWORDS

• Alzheimer dementia • Cognition • Stress • Epigenetics diet • Nutraceuticals

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KEY POINTS

- The marked increase in the incidence of Alzheimer dementia (AD) has become a global health issue.
- Early detection and prevention of AD is feasible with imaging biomarkers.
- Stress-related disorders (metabolic, cardiovascular diseases, posttraumatic stress disorder, late-life depression) are recognized as risk factors for AD.
- Stress and the limbic-hypothalamic-pituitary-adrenal axis intersect with epigenetics in a reciprocal manner, with both positive and negative effects on brain health.
- Chronic stressors affecting hippocampal neurogenesis may set the stage for accelerated aging and cognitive impairment.
- Epigenetics regulation at DNA methylation near stress gene loci may be the missing link in stress responses and AD.
- There is good evidence in support of epigenetic shift regarding epigenetic processes such as DNA methylation, histone modification, and microRNA for aging-related cognitive impairment and AD.
- An epigenetics diet and nutraceuticals targeting epigenomics (omega 3-fatty acids, cocoa, caffeine, spices, red wine, vegetables, fruits, nuts, and dietary supplements) hold promise in epigenetic reprogramming of the aging brain and AD. Preventive trials are needed to examine efficacy in preventing cognitive decline in aging and AD.
- Epigenetics signatures and epigenomics targets represent a new frontier in AD research and geriatric care.
- An integrated service model may be the best approach for translating personalized epigenetics dietary intervention to an integrated care model of psychiatric practice.

INTRODUCTION

Recently, social media has heightened interest toward the public health issue of cognition changes in aging. The estimated global prevalence of Alzheimer disease or Alzheimer dementia (AD) of 24 million in 2005 is expected to increase by 4-fold by the year 2050.^{1,2} Regarding the incidence and prevalence of AD, North America and Western Europe rank highest, followed by Latin America and China. The incidence rate of AD rises exponentially with advancing age, reaching the peak around the seventh and eighth decade. Each year, an estimated 4 to 6 million new cases are identified. Dementia has a projected worldwide incidence of more than 30 million by 2040. In the United States, an estimated 5.2 million Americans are diagnosed with AD: 5 million as late-onset AD (LOAD), and 0.2 million as early-onset dementia (EAD). If the onset of AD can be deferred by as little as 1 year, the prevalence can be reduced by 10%. Early-onset autosomal dominant AD is caused by mutations of genes encoding amyloid precursor protein (APP), presenilin 1 (PSEN1), and presenilin 2 (PSEN2). The $\epsilon 4$ allele of the apolipoprotein E (APOE) gene is now considered as a major genetic risk factor for AD.³

Health economics data conclude that AD has taken a heavy toll on health care costs across the world. A disturbing trend has recently been found in the United States. Between 2000 and 2010, AD-related mortality increased disproportionately in relation to a decrease in deaths from other medical diagnoses. Mortality from AD increased by 68%, compared with reduced mortality rates from cardiovascular disease (16%),

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