Preventing Amyloid-Beta Plaque Accumulation in Genetically Predisposed Young Adults in the United States

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Introduction

Alzheimer's disease (AD) is advancing at an alarming rate, with a new diagnosis occurring every three seconds. By 2060, it is projected to affect 13.8 million people annually, which is more than twice the number of patients in 2020. Scientists now classify Alzheimer's disease as a silent pandemic, warning that its long-term consequences may surpass those of COVID-19, creating a major public health crisis (Bhuiyan et al., 2023). The primary cause of Alzheimer's disease is the accumulation of beta-amyloid (Aβ) peptides in the brain, generated when the amyloid precursor protein (APP) is cleaved by two enzymes: β-secretase and γ-secretase. When there is an imbalance between the production and clearance of amyloid beta peptides, they aggregate to form insoluble beta-amyloid plaques, which disrupt cell communication, trigger inflammation, and ultimately contribute to neurodegeneration and Alzheimer's Disease (McGirr et al., 2020). AD was the seventh-leading cause of death in the United States, and the World Health Organization recognizes the disease as a global health crisis (Lane et al., 2017). While the significant increase in AD cases is largely linked to the growing global population, other contributing factors include early-life conditions and unhealthy lifestyle choices that promote the accumulation of beta-amyloid plaques, which play a key role in the development of Alzheimer's disease. Therefore, preventative measures targeting these risk factors offer an opportunity to reduce the prevalence of the disease. With cases expected to rise sharply in the coming decades, immediate action is necessary to curb future rates of cognitive decline. Identifying effective early interventions has the potential to transform how society approaches Alzheimer's prevention; rather than waiting until symptoms appear, these strategies could shift healthcare and public policy toward proactive, risk-reducing measures. More specifically, young adults who carry the APOE4 allele, a gene variant that significantly increases the risk of developing Alzheimer's disease (Improta-Caria et al., 2020), could strongly benefit from the findings of this research because it could change the trajectory of their health in later life. The 2024 Alzheimer's Disease Facts and Figures report highlights the growing prevalence of Alzheimer's disease, estimating that 6.9 million Americans aged 65 and older are currently living with the condition, which underscores the urgent need for early intervention and prevention, as no current treatments can stop or reverse the disease's progression (Alzheimer's Association, 2024). This report prompts researchers to explore how early interventions could prevent beta-amyloid plaque buildup, offering a proactive solution to Alzheimer's instead of relying on common reactive methods used to fight against AD. By focusing on preventative strategies during adolescence and early adulthood, individuals could reduce future cognitive decline and mitigate risk before symptoms even begin, helping to shift the approach to treating Alzheimer's disease from late-stage treatment to early prevention. In light of this complexity, the question posed is: “What is the best possible solution to prevent amyloid-beta plaque accumulation in genetically predisposed young adults in the United States?” While there are multiple methods to address this issue, extensive research suggests that the most promising solution for preventing amyloid-beta accumulation in genetically at-risk young adults in the U.S. is a holistic approach that integrates consistent aerobic physical activity, a neuroprotective diet, and high-quality sleep.

Aerobic Exercise

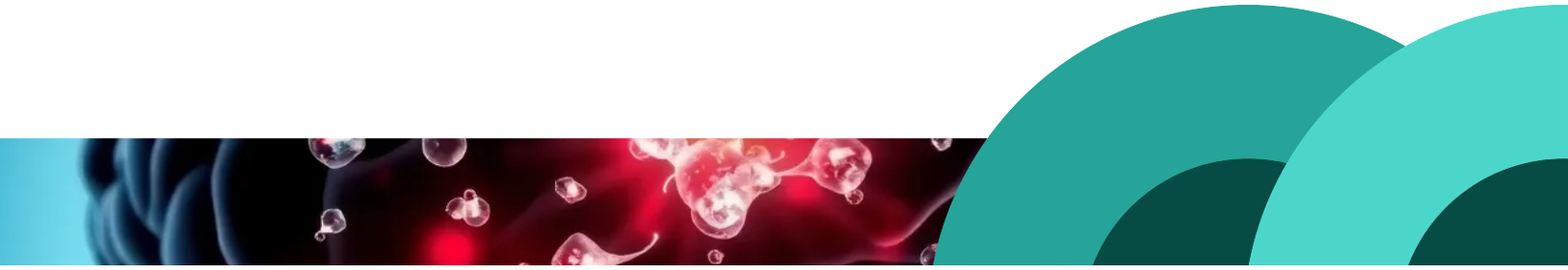
To begin with, engaging in consistent aerobic exercise has been associated with a reduction in beta-amyloid plaque accumulation due to neuroprotective effects at the cellular level, which makes it one viable solution. Wei-Wei Chen, Xia Zhang, and Wen-Juan Huang, researchers affiliated with the Department of Neurology at Xuzhou Central Hospital in Xuzhou, China, found that 40 minutes of physical activities such as the ergocycle, treadmill, and stair-climbing activities over a period of 12 consecutive weeks has been associated with an increase in cerebral blood flow that improves neurogenesis, the creation of new neurons. (Chen et al., 2016). These neuroprotective effects contribute to overall brain health and may counteract beta-amyloid buildup as a result, which reinforces aerobic activity as one method for reducing Alzheimer’s risk. Furthermore, a loss in muscle mass is correlated with an increased risk of developing Alzheimer's disease (AD), and evidence shows that individuals with greater muscle strength tend to have a lower likelihood of experiencing dementia or AD. (Chen et al., 2016). Since greater muscle strength is linked to a lower risk of Alzheimer's disease, consistent aerobic exercise provides neuroprotective benefits contributing to a reduction in amyloid-beta plaque accumulation. Similarly, Alex Cleber Improta-Caria et al., a researcher affiliated with the Post-Graduate Program in Medicine and Health at Federal University of Bahia, revealed that physical exercise has been associated with enhanced cognitive function in Alzheimer's disease patients and a slower rate of cognitive decline in individuals carrying the APOE ε4 allele, who are at higher risk for developing AD (Improta-Caria et al., 2020). Corroborating this finding, the cognitive benefits of exercise lie in its ability to stimulate brain plasticity and release brain-derived neurotrophic factor (BDNF), a protein in the brain that is important for the growth, survival, and function of nerve cells (Improta-Caria et al., 2020), which improves cognitive function and delays or prevents the onset of Alzheimer’s symptoms in at-risk individuals. These neuroprotective effects of physical activity, along with the correlation between exercise and improved brain function, support the idea that exercise helps prevent the accumulation of amyloid plaques, aligning with the broader argument that lifestyle interventions during early adulthood can decrease Alzheimer's risk. While consistent aerobic exercise presents a solid solution to prevent or mitigate the accumulation of amyloid-beta plaques, several limitations must be addressed. Regular physical activities may not be feasible for all individuals, particularly people with physical disabilities, chronic illnesses, or limited access to exercise facilities. Furthermore, sticking to an exercise regimen can be difficult because it requires long-term commitment, which may be challenging for individuals struggling with motivation, time constraints, or mental health. Despite these limitations, the potential of aerobic exercise to reduce beta-amyloid accumulation and slow cognitive decline is significant. Physical activity could be incorporated into public health guidelines, starting in adolescence and continuing throughout adulthood. Communities, schools, and workplaces could promote accessible aerobic exercise programs, ensuring that options are available for individuals of all abilities and backgrounds.

Neuroprotective Nutrition

Another possible solution could be adopting a neuroprotective and nutrient-rich diet, which may slow cognitive decline and neurodegeneration by mitigating inflammation and reducing beta-amyloid toxicity. Samantha McGirr et al., researchers affiliated with the University of Nebraska Medical Center, established that foods containing antioxidants, omega-3 fatty acids, B vitamins, and folate could potentially prevent the progression of Alzheimer's disease. Studies have shown promising results with certain nutrients like Vitamin E and omega-3 fatty acids, particularly in their ability to slow cognitive decline and neurodegeneration, and further trials suggest that B vitamins and omega-3 fatty acids can slow cognitive decline in individuals with mild cognitive impairment. (McGirr et al., 2020). The consistent link between specific nutrients and reduced cognitive decline suggests that a targeted nutritional approach could serve as a viable prevention strategy. By focusing on individuals with mild cognitive impairment, these findings reinforce the importance of early action and suggest that dietary changes could delay or even halt disease progression before irreversible brain damage occurs. This aligns with broader efforts to shift Alzheimer's care from reactive treatment to proactive prevention. For example, Selvaraju Subash et al., researchers affiliated with the Ageing and Dementia Research Group from Sultan Qaboos University, discovered that Tg model mice fed a standard diet showed greater memory loss, anxiety, and motor and learning impairments compared to those supplemented with 2% or 4% date fruit. Date supplementation significantly reduced amyloid-beta (Aβ) protein levels, with the 4% diet providing stronger neuroprotective effects than the 2% diet. (Subash et al., 2015) The greater benefits at 4% highlight the importance of sufficient intake of bioactive compounds in an individual's diet, which reinforces that targeted nutritional changes can prevent or delay AD and underscores the need to integrate functional foods into the lifestyles of those who are genetically predisposed. Notably, Nusrat Zahan Bhuiyan et al., researchers from the Department of Biochemistry and Molecular Biology at the National University of Bangladesh, asserted that a diet rich in fruits, vegetables, fish, olive oil, and nuts protects against cognitive decline. Tomatoes are high in lycopene, which reduces hypertension and chronic disease risk, while broccoli and cabbage offer cancer-preventive compounds. Fatty fish, rich in omega-3 fatty acids like DHA, can reduce Alzheimer's disease (AD) risk by 41% when consumed twice weekly, and olive oil and nuts provide heart-healthy MUFAs and anti-inflammatory benefits. (Bhuiyan et al., 2023). This specific diet, supported by powerful statistics indicating a reduction of AD risk when consumed, highlights the benefits of adopting a diet tailored for cognitive health. Beyond its neuroprotective benefits, this eating pattern could ultimately contribute to increased life expectancy by improving vascular function, reducing systemic inflammation, and supporting cellular resilience, which not only helps prevent cognitive decline in genetically predisposed individuals but also promotes overall longevity and well-being. Correspondingly, following a Mediterranean-style diet rich in these ingredients has been correlated with a reduced risk of Alzheimer's disease, with strict adherence lowering risk by up to 53% and moderate adherence by 35%. (Bhuiyan et al., 2023). The 53% risk reduction with strict adherence suggests that both the quality and consistency of food choices matter. Individuals who not only choose but also integrate this regimen into their lifestyle can reap the benefits of the anti-inflammatory powers of these foods. Mediterranean-style eating is just one example of implementing early prevention strategies to fight against cognitive decline; overall, adopting a neuroprotective diet holds significant promise for reducing Alzheimer's risk. However, accessibility and affordability of key foods like fatty fish, olive oil, and fresh vegetables could be barriers, especially for low-income communities. Additionally, long-term adherence could be challenging for some individuals. To address these challenges, public health campaigns could raise awareness about the importance of diet in preventing cognitive decline, and policymakers could support these efforts by making healthy foods more accessible through subsidies or community programs.

Proposed Solution

In light of these findings and additional research, the most comprehensive and proactive approach to prevent amyloid-beta accumulation in genetically predisposed young adults in the United States is implementing a combination of consistent exercise, a neuroprotective diet, and high-quality sleep. Sleep is an integral part of this combination because it clears harmful brain waste: the findings from a study conducted by Joseph R. Winer and Bryce A. Mander et al., researchers from UC Berkeley and UC Irvine respectively, indicate that poor sleep quality, marked by decreased sleep efficiency and disruptions in non-rapid eye movement, may accelerate amyloid-beta accumulation by hindering the glymphatic system's ability to clear Aβ waste over time (Winer et al., 2020). Contrastingly, uninterrupted sleep supports the brain's glymphatic system, helping prevent harmful plaque buildup and lowering Alzheimer's risk. Moreover, total sleep duration showed a significant inverse association with subsequent amyloid-beta accumulation ($r = -0.36$, $p = 0.04$), while waking



after sleep onset demonstrated a positive trend ($r = 0.31$, $p = 0.08$) (Winer et al., 2020). In other words, shorter overall sleep and more frequent nighttime awakenings were linked to faster rates of Aβ buildup, which establishes lack of sleep as a pertinent risk factor for AD. Therefore, expanding the scope from just exercise and diet to include healthy sleep habits drastically improves the likelihood of reduced amyloid-beta plaque accumulation. The powerful nature of this combined solution is validated by a 2009 study involving a group of 1,880 participants, where adhering to dietary guidelines alone was associated with a 29% reduction in AD risk, while combining diet with regular exercise corresponded to an additional 9% decrease. (Bhuiyan et al., 2023). While it is true that adhering to even one of these strategies could be effective, these insights corroborate the claim that combining them provides the best solution to combat cognitive decline. However, this approach may not be enough for individuals already experiencing rapid Aβ accumulation or who possess a genetic predisposition. Regardless of perfect lifestyle choices, individuals may still develop Alzheimer's if they carry the APOE ε4 allele (Improta-Caria et al., 2020). Additionally, a drastic lifestyle shift such as this could be difficult to maintain throughout life and may not be feasible for everyone. Nevertheless, this solution could pave the way for future discussions about holistic health interventions and even combat Alzheimer's disease on a worldwide scale, ultimately decreasing future healthcare burdens and expenses in the future.

Conclusion

Alzheimer's disease is a growing public health crisis, making early prevention critical. Therefore, combining a neuroprotective diet, consistent exercise, and high-quality sleep is the most comprehensive approach to preventing beta-amyloid plaque accumulation in genetically predisposed young adults. The broader implications of these findings extend beyond Alzheimer's prevention, supporting the role of lifestyle choices in reducing the risk of various age-related diseases and improving long-term public health outcomes. Emphasizing prevention could reshape healthcare policies, alleviate future economic burdens, and shift public understanding toward prioritizing early intervention over late-stage treatment. The fight against Alzheimer's disease does not begin in old age—it starts now. By prioritizing prevention in adolescence and early adulthood, proactive steps can be taken to reduce the risk of beta-amyloid buildup and protect long-term brain health.

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