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Preventing Alzheimer's disease by correcting lifestyle factors?

Alzheimer's disease (AD) is a progressive neurodegenerative disorder that primarily affects cognitive functions, memory, and behavior. It is the most common form of dementia, and causes the loss of connections between nerve cells and ultimately the death of brain cells.

Alzheimer's disease is defined by the presence of amyloid plaques and tangles in the brain that cause progressive memory loss and the inability to perform daily functions. As society continues to age, AD poses a serious challenge to society and the medical community.

The greatest challenge in developing effective drugs for this disease is early diagnosis, before the onset of clinical symptoms. In recent years, research has focused on identifying reliable biomarkers that allow for the diagnosis of this disease.

Fortunately, great advances in the diagnosis and treatment of Alzheimer's disease (AD) have recently been reported. For example, in 2023 two anti-amyloid-β (Aβ) monoclonal antibodies, Lecanemab and Donanemab, received full approval from the U.S. Food and Drug Administration (FDA) for the treatment of early-stage AD. Patients treated with these drugs showed an improvement of approximately 25 % to 35 % in cognitive decline compared to the placebo group [2].

In parallel with these remarkable therapeutic advances, significant progress has been made in the development and validation of diagnostic molecular biomarkers for AD, including cerebrospinal fluid (CSF) assays, positron emission tomography (PET) and, more recently, blood-based biomarkers based on detection of Aβ and phosphorylated Tau (p-Tau), the key constituents of the plaques and tangles that neuropathologically define AD [1].

These blood biomarkers are extremely useful for identifying patients who do not yet present clinical symptoms and who can be treated pharmacologically or included in clinical trials for the development of new therapies.

Blood tests offer a prescreening tool to rule out symptomatic individuals with a low likelihood of Aß pathology. This approach avoids unnecessary CSF amyloid testing, reducing the costs and burden associated with these more invasive and expensive tests.

Indeed, these biomarkers have proven to be extremely useful for monitoring disease progression, and they also allow for the inclusion of patients in early stages of the disease in better-controlled clinical trials than in the past.

In the review published in this issue of JPAD, Hooper C. et al. discuss interesting results on the relationship between these blood biomarkers and modifiable lifestyle factors, such as nutrition, physical activity, sleep, alcohol consumption, smoking and social isolation [3].

Even if the number of publications presented in this review is still small, the idea of using these well-validated plasma biomarkers in AD diagnosis seems intelligent and worthy of further exploration.

In some cases, as the authors note, a dichotomy has been observed between plasma Aβ or Tau levels and clinical status. Therefore, it is crucial to understand their metabolism, since plasma concentrations depend on production, clearance, and brain deposits, among other factors. Furthermore, inflammatory processes associated with certain pathologies can cause variations in these plasma biomarkers.

Despite these limitations, noted and discussed extensively by the authors, the approach remains valid and very interesting.

In the future, the appropriate biomarkers for the diagnosis and monitoring of AD will surely be defined more precisely, their metabolism and the factors involved in their plasma presence will be better understood.

For the time being, as described before, the proposed therapies for AD show only limited effects. For this reason, the biggest challenge to circumvent the tragic consequences of this disease is early diagnosis before the onset of clinical symptoms.

Another strategic alternative, that is well discussed in this review, consists of finding lifestyles compatible with good brain health in order to avoid developing the disease or at least to slow down its onset.

Taking this into account, it will be extremely interesting to evaluate these lifestyle factors (or others) in order to effectively prevent AD.

In fact, having a balanced diet, exercising, sleeping well, not consuming alcohol or tobacco, and having a stimulating social life are common factors for a healthy and long life, well demonstrated in other diseases associated with aging.

Many of these chronic diseases depend, in part, on the existence of modifiable risk factors. A very clear example is obesity and diabetes, or tobacco and cardiovascular disease.

There is now mounting evidence that eating a nutrient-rich, diverse, and balanced diet could not only contribute to overall physical health but also protect against brain decline.

In addition to good nutrition, other factors such as regular physical activity, social and mental engagement, and quality sleep are also crucial for maintaining optimal cognitive health.

Most health systems are designed to "treat and cure disease" and not to "prevent disease and promote health."

This healthcare expenditure is primarily concentrated (up to 75 % of the total) on primary or specialized care for patients with chronic diseases derived from their treatment.

Health promotion and disease prevention, modifying our behaviors and habits, can positively influence the frequency of disease onset or its severity.

Furthermore, let's not forget the old adage, "prevention is better than cure". This statement is an intuitive, acceptable, and politically correct concept.

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In fact, it is no coincidence that our journal is called Journal of Prevention of Alzheimer's Disease.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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