

PERSPECTIVE ARTICLE

Alzheimer's disease as a biosocial disorder:
Toward integrative and personalized
interventionKhuraman Miryusifova^{1,2*} , İrana Galandarlı¹ , and Jale Alasgarova²¹Research Institute of Living System, Ministry of Science and Education, Baku, Azerbaijan²Research, Development, and Innovation Center of Excellence, Baku State University, Baku, Azerbaijan

Abstract

Alzheimer's disease (AD) is conventionally defined by amyloid- β deposition and tau-related pathology. However, the inherent clinical heterogeneity of the disease suggests that these molecular markers alone are insufficient to fully characterize its progression. This article introduces a comprehensive "biosocial framework" for understanding AD, which posits that social determinants of health—such as social isolation, socioeconomic status, and chronic stress—are not merely passive risk factors; rather, their effects may become biologically embedded and contribute to the pathogenesis of the disease. We examine the neurobiological pathways linking psychosocial adversity to neurodegeneration, specifically through the chronic activation of the hypothalamic–pituitary–adrenal axis, elevated glucocorticoid levels, and systemic inflammation. This physiological cascade compromises blood–brain barrier integrity, fuels microglial dysfunction, and accelerates the accumulation of pathological protein aggregates. By distinguishing this model from the traditional biopsychosocial approach, we propose testable hypotheses concerning epigenetic modifications and gene–environment interactions. Ultimately, we argue that the future of dementia prevention lies in integrating early blood-based biomarker detection with tailored behavioral and psychological interventions. This paradigm shift toward a personalized, integrative care model is essential for addressing the multifactorial nature of AD, moving beyond purely molecular treatments to incorporate modifications to lifestyle and social environments as fundamental components of clinical management and long-term care.

***Corresponding author:**Khuraman Miryusifova
(miryusifovaxuraman@mail.ru)

Citation: Miryusifova K, Galandarlı İ, Alasgarova J. Alzheimer's disease as a biosocial disorder: Toward integrative and personalized intervention. *Health Psychol Res.* 2026;14(3):026220112.
doi: 10.36922/HPR026220112

Received: May 29, 2026**Revised:** July 1, 2026**Accepted:** July 1, 2026**Published online:** July 14, 2026

Copyright: © 2026 Author(s). This is an Open-Access article distributed under the terms of the Creative Commons AttributionNonCommercial 4.0 International (CC BY-NC 4.0), which permits all non-commercial use, distribution, and reproduction in any medium, provided the original work is properly cited.

Publisher's Note: AccScience Publishing remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.

Keywords: Alzheimer's disease; Biosocial framework; Integrative interventions; Personalized interventions

1. Introduction

Alzheimer's disease (AD) is the most common cause of dementia worldwide and remains one of the most significant challenges associated with population aging. Traditionally, AD has been viewed as a neurodegenerative disorder driven by amyloid- β deposition and tau-related pathology. AD exhibits clinical heterogeneity that these core biological mechanisms alone fail to explain.¹

Over the past decade, research has increasingly demonstrated that cognitive decline is influenced by a complex interaction of biological, behavioral, psychological, and social

factors throughout the lifespan.¹ In this perspective article, we argue that adopting a biosocial framework may provide a more comprehensive understanding of AD and facilitate more effective prevention and intervention strategies.

2. Why the traditional biomedical model is insufficient

Although the biomedical model has clarified key pathological processes and the *APOE* $\epsilon 4$ allele has highlighted the role of genetic susceptibility, genetic predisposition alone fails to explain individual variations in clinical outcomes. Instead, the “cognitive reserve” concept suggests that education and intellectual engagement enhance the brain's capacity to compensate for pathology, explaining why individuals with similar neuropathological burdens exhibit markedly different cognitive manifestations. Similarly, accumulating evidence indicates that metabolic, behavioral, and psychosocial factors significantly modify disease risk and progression.¹ Therefore, understanding AD requires moving beyond a purely biological framework toward a more integrative perspective that recognizes the interaction between biological and social determinants of health.

3. Biosocial impact of cardiometabolic and behavioral risk factors

A biosocial framework highlights the lifelong interaction between biological and social determinants of cognitive health. Cardiometabolic and vascular conditions, such as hypertension and diabetes, impair neuronal function through vascular dysfunction and oxidative stress—conditions deeply influenced by lifestyle and socioeconomic status. Behavioral factors, particularly physical activity, enhance neuroplasticity and reduce dementia risk, whereas sedentary habits accelerate decline. Furthermore, psychological adversity and social isolation activate neuroendocrine pathways, promoting neuroinflammation and hippocampal dysfunction.¹ Consequently, AD should be understood not merely as molecular pathology, but as the cumulative result of genetic susceptibility, metabolic health, lifestyle behaviors, and social environments.

4. Neurobiological mechanisms linking social determinants to brain pathology

A growing body of evidence suggests that the effects of social determinants on AD are mediated through a series of interconnected biological pathways. Understanding how psychosocial adversity becomes biologically embedded is therefore critical for explaining the mechanisms through which social environments influence neurodegeneration.

Long-term exposure to social isolation, insufficient social support, and socioeconomic disadvantage has been associated with persistent activation of the hypothalamic–pituitary–adrenal axis. Sustained stimulation of this stress-response system results in prolonged glucocorticoid secretion, particularly elevated cortisol levels, which can promote systemic inflammatory activity and enhance the release of pro-inflammatory cytokines.²

Chronic peripheral inflammation impairs blood–brain barrier integrity by disrupting endothelial tight junction proteins, allowing systemic immune mediators to enter the central nervous system early in AD progression. This increased blood–brain barrier permeability facilitates inflammatory signaling, which triggers sustained microglial activation.³ These dysfunctional microglia exhibit impaired β -amyloid clearance, accelerating plaque formation—a hallmark of AD.⁴ Concurrently, chronic stress elevates glucocorticoids, stimulating kinases that promote tau hyperphosphorylation. Furthermore, persistent neuroinflammation synergistically exacerbates tau pathology and neurofibrillary tangle development. Collectively, these findings demonstrate that psychosocial stressors may contribute to AD pathology through interacting neuroendocrine, vascular, and immune pathways to fuel amyloid accumulation, tau abnormalities, and neuroinflammation.

5. Testable predictions and practical implications of the biosocial model

It is essential to distinguish the proposed biosocial approach from the conventional biopsychosocial model. While the traditional biopsychosocial framework treats biological, psychological, and social factors as interacting but distinct components, the biosocial approach posits that social factors are biologically embedded. Rather than merely influencing health outcomes indirectly, social determinants—such as isolation and chronic stress—actively alter neuroendocrine and immune pathways, directly driving neuroinflammation and accelerating neurodegenerative progression.

The biosocial model proposes three key testable hypotheses for future research:

- (i) Epigenetic mechanisms: Social isolation may induce epigenetic modifications in microglial genes, thereby accelerating tau pathology and disease progression.
- (ii) Gene–environment interaction: Supportive social environments may mitigate the clinical expression of genetic vulnerability in *APOE* $\epsilon 4$ carriers, suggesting that lifestyle modulates genetic risk.

(iii) Biomarker dynamics: Social interventions designed to reduce chronic stress may induce measurable decreases in neuroinflammation, specifically reflected in blood levels of glial fibrillary acidic protein.

By framing social factors as active drivers—rather than passive risks—of AD's biological pathogenesis, this model provides a robust foundation for developing more comprehensive and integrated dementia prevention strategies.

6. Implications for prevention, early detection, and integrated care in Alzheimer's disease

Health psychology plays a central role in dementia prevention and management. By promoting behavior change, improving treatment adherence, and reducing chronic stress, psychological interventions target modifiable risk factors and enhance the effectiveness of biomedical treatments.

Blood-based biomarkers enable the early detection of AD pathology before the onset of significant cognitive symptoms.⁵ However, their clinical value depends on integrating biomarker assessment with behavioral and psychosocial interventions rather than using them as diagnostic tools alone.

A practical biosocial framework begins with the assessment of cognitive, psychosocial, and metabolic risk factors in primary care. Individuals identified as being at high risk can then undergo biomarker testing, which supports both diagnostic accuracy and motivation for lifestyle modification. Within this framework, health psychologists play an essential role in risk communication, facilitating behavior change, and promoting long-term adherence to preventive recommendations. Digital health platforms and remote monitoring systems can further improve accessibility, support continuous follow-up, and reduce healthcare burden. Integrating biological assessment with psychosocial interventions therefore provides a more personalized and effective approach to dementia prevention and early disease management.

7. Conclusion

Alzheimer's disease should no longer be viewed solely as a neurodegenerative disorder, but rather as a complex condition shaped by the combined influence of biological and social factors. Although the accumulation of amyloid-beta and tau proteins plays a central role in disease pathogenesis, recent studies indicate that genetic predisposition interacts with metabolic health, lifestyle factors, psychological factors, and the social environment.

These factors may not completely prevent the disease, but they may influence the timing and rate of cognitive decline. Adopting this perspective may improve risk-factor management, the development of patient-centered healthcare services, and the reduction of the growing global burden of dementia. Therefore, future research and healthcare policies should be based on an integrative model that combines biological and psychosocial approaches.

Acknowledgments

None.

Funding

None.

Conflict of interest

The authors declare that they have no competing interests.

Author contributions

Conceptualization: Khuraman Miryusifova

Writing—original draft: Khuraman Miryusifova

Writing—review & editing: İrana Galandarlı, Jale Alasgarova

Ethics approval and consent to participate

Not applicable.

Consent for publication

Not applicable.

Availability of data

Not applicable.

References

1. Livingston G, Huntley J, Sommerlad A, *et al.* Dementia prevention, intervention, and care: 2020 report of the Lancet Commission. *Lancet*. 2020;396(10248):413–446.
doi: 10.1016/S0140-6736(20)30367-6
2. Burke MR, Sotiropoulos I, Waites CL. The multiple roles of chronic stress and glucocorticoids in Alzheimer's disease pathogenesis. *Trends Neurosci*. 2024;47(11):933–948.
doi: 10.1016/j.tins.2024.08.015
3. Zhang Q, Yang G, Luo Y, *et al.* Neuroinflammation in Alzheimer's disease: insights from peripheral immune cells. *Immun Ageing*. 2024;21:38.
doi: 10.1186/s12979-024-00445-0
4. Fruhwürth S, Zetterberg H, Paludan SR. Microglia and amyloid plaque formation in Alzheimer's disease: evidence, possible mechanisms, and future challenges. *J Neuroimmunol*. 2024;390:578342.

doi: 10.1016/j.jneuroim.2024.578342

5. Grande G, Valletta M, Rizzuto D, *et al.* Blood-based

biomarkers of Alzheimer's disease and incident dementia in the community. *Nat Med.* 2025;31(6):2027–2035.

doi: 10.1038/s41591-025-03605-x