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*Abstract*

# Gut Microbiota and Neuroinflammation in the Pathophysiology of Alzheimer's Disease: A Narrative Review

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**Introduction:** Alzheimer's disease (AD) is a progressive, multifactorial neurodegenerative condition characterized by the accumulation of amyloid-beta plaques, alterations in tau protein, neuronal loss, and chronic neuroinflammation. Recent evidence suggests that changes in the gut microbiota may influence these processes through the microbiota–gut–brain axis, which involves neural, immune, and metabolic pathways. In this context, microbiota modulation has been investigated as a potential complementary strategy for preventing cognitive decline and managing AD. **Objective:** To analyze recent evidence on the involvement of the gut microbiota in the pathophysiology and progression of Alzheimer's disease, highlighting potential therapeutic strategies targeting the microbiota–gut–brain axis. **Methods:** This narrative literature review was conducted using the PubMed/MEDLINE and SciELO databases, covering publications from 2025 and 2026. The search used terms related to Alzheimer's disease, gut microbiota, the gut–brain axis, and neuroinflammation. Priority was given to systematic reviews and studies addressing associations among gut dysbiosis, intestinal barrier permeability, systemic inflammation, neuroinflammation, and cognitive changes. **Results and Discussion:** Aging may alter the composition and reduce the diversity of the gut microbiota, promoting dysbiosis and impairing metabolic and immune functions. These changes may increase intestinal permeability and the translocation of microbial products, contributing to systemic inflammation and changes in communication between the gut and brain. In AD, amyloid-beta accumulation promotes microglial and astrocyte activation. Although initially involved in aggregate clearance and protection of the central nervous system, persistent glial activation promotes the release of proinflammatory mediators, exacerbating synaptic dysfunction and neuronal degeneration. Studies also indicate that metabolites produced or modulated by the microbiota, such as short-chain fatty acids, may influence the integrity of the intestinal and blood–brain barriers and the regulation of immune responses. Interventions involving probiotics, prebiotics, specific dietary patterns, bioactive compounds, and fecal microbiota transplantation have shown promising results for inflammatory markers and cognitive parameters, primarily in experimental models. However, methodological heterogeneity and the limited availability of clinical trials preclude definitive conclusions regarding their efficacy. **Conclusion:** The gut microbiota may contribute to the neuroinflammatory mechanisms associated with Alzheimer's disease through immune and metabolic changes and alterations in biological barrier integrity. Modulation of the microbiota–gut–brain axis has potential as a complementary strategy but still requires validation through controlled clinical trials and longitudinal studies. Therefore, its therapeutic benefits are not sufficiently established to support a specific clinical recommendation.

**Keywords:** Alzheimer Disease; Gut–Brain Axis; Gut Microbiota; Neuroinflammation.

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