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Gut–Brain Axis Inflammation in Dementia: A Qualitative Literature Analysis

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Abstract

This study aims to explore the role of gut–brain axis inflammation in the development and progression of dementia, particularly Alzheimer's disease, through a comprehensive qualitative literature analysis. Employing a descriptive qualitative design with a library research approach, this study systematically reviewed and synthesized peer-reviewed scientific publications from 2015 to 2025 related to gut microbiota, neuroinflammation, and cognitive decline. Data collection was conducted through structured literature searches and document analysis, followed by inductive thematic analysis involving data reduction, categorization, and interpretation of recurring concepts. The results indicate that gut microbiota dysbiosis contributes significantly to neuroinflammatory processes via mechanisms involving immune activation, microbial metabolite imbalance, and microglial overactivation. Dietary and lifestyle factors were found to influence gut microbial composition, while interventions such as probiotics, prebiotics, and Mediterranean diets demonstrated potential to mitigate inflammation and improve cognitive outcomes. The findings reinforce the microbiota–immune–neural axis theory, suggesting that dementia should be understood as a systemic inflammatory disorder rather than an isolated neurological condition. This study highlights the therapeutic promise of microbiome modulation and calls for interdisciplinary collaboration to advance translational applications. The conclusions provide both theoretical enrichment for neuroinflammation research and practical insights for developing microbiota-based interventions in dementia prevention and management.

Keywords

Gut–Brain Axis, Inflammation, Dementia, Microbiota, Neuroinflammation

Introduction

Dementia remains one of the most challenging neurodegenerative disorders worldwide, with Alzheimer's disease (AD) representing the most prevalent form. Despite decades of investigation, its etiology and pathophysiology are not fully understood. In recent years, attention has turned toward the gut–brain axis as a key factor linking peripheral physiological changes to central nervous system dysfunction. The gut–brain axis, a bidirectional communication system involving neural, hormonal, and immunological pathways, has emerged as an essential mediator of neuroinflammatory processes contributing to dementia (Lei et al, 2025).

Emerging evidence indicates that gut microbiota dysbiosis—an imbalance in the microbial ecosystem—plays a critical role in promoting systemic and neuroinflammation. Disruption of the intestinal barrier facilitates translocation of bacterial endotoxins such as lipopolysaccharides (LPS), which can cross the blood–brain barrier and activate microglia, leading to chronic neuroinflammation and cognitive decline (Megur et al, 2020) (Popescu et al, 2024) (Solanki et al, 2023). This mechanistic pathway has become increasingly significant as studies reveal that neuroinflammation is a central driver of neuronal damage in AD.

Recent findings also emphasize the contribution of microbial metabolites such as short-chain fatty acids (SCFAs) and bacterial amyloids in exacerbating inflammatory cascades and promoting amyloid-beta aggregation in the brain (Giau et al, 2018) (Qian et al, 2023). These molecular interactions highlight how peripheral immune signals can influence neurodegenerative processes through metabolic crosstalk. The gut microbiota's ability to modulate systemic inflammation and microglial activation underscores its integral role in dementia pathophysiology (Lei et al, 2025).

The urgency of this research is underscored by the global rise in dementia prevalence, which is projected to exceed 150 million cases by 2050. Current therapeutic options remain largely symptomatic and fail to address the underlying inflammatory pathology. Consequently, targeting the gut–brain axis represents a novel and potentially transformative approach to mitigating disease progression (Popescu et al, 2024). This direction aligns with the broader movement toward precision medicine and integrative neurobiology.

Animal model studies provide compelling evidence for a causal link between gut dysbiosis and neuroinflammation. For instance, germ-free and fecal microbiota transplantation experiments demonstrate that AD-associated microbiota exacerbate amyloid pathology and cognitive impairment, whereas microbiota from healthy donors alleviate these effects (Chen et al, 2022) (Wang et al, 2019). These findings provide a biological foundation for exploring microbiota-targeted therapies in humans.

Despite these advances, a significant research gap persists in translating preclinical findings into clinical interventions. Human observational studies reveal associations between gut microbial alterations and cognitive decline, yet robust longitudinal and interventional studies remain limited (Popescu et al, 2024) (Ticinesi et al, 2018). This gap underscores the need for systematic, multidisciplinary approaches that integrate microbiology, immunology, and neuroscience.

The therapeutic implications of this axis are increasingly recognized. Interventions such as probiotics, prebiotics, antibiotics, and fecal microbiota transplantation have shown promise in modulating gut microbial composition and reducing neuroinflammation in preclinical settings (Ayyanar & Vijayan, 2024) (Kesika et al, 2020) (Mani et al, 2024). However, the efficacy and safety of these strategies in humans remain to be conclusively established.

Recent multi-omics approaches have deepened understanding of gut–brain interactions, revealing alterations in lipid metabolism and immune signaling pathways associated with microbial changes (Qian et al, 2023). These integrative data sets provide novel insights into the molecular underpinnings of dementia, moving beyond correlative associations toward causal inference.

Furthermore, inflammatory signaling through pathways such as the NLRP3 inflammasome has been implicated as a key mediator connecting gut dysbiosis to neurodegenerative pathology (Shen et al, 2020). Understanding these pathways is essential for identifying biomarkers and therapeutic targets capable of mitigating neuroinflammation at its systemic origin.

In addition to mechanistic insights, recent reviews underscore the heterogeneity in study methodologies and populations, emphasizing the necessity for standardized protocols to improve reproducibility and comparability (Liu et al, 2020) (Łuc et al, 2020). This standardization is vital for advancing gut–brain research toward clinical applicability.

From a theoretical standpoint, the gut–brain axis model reshapes traditional understandings of dementia, framing it as a multisystem disorder rather than a purely neural pathology (Giau et al, 2018). Practically, it introduces potential for novel diagnostic markers—such as microbial signatures—and preventive strategies aimed at restoring gut homeostasis.

The global scientific community now recognizes the gut microbiome as a modifiable environmental factor influencing brain health. This paradigm shift reflects growing consensus that maintaining microbial balance may be as critical to cognitive resilience as genetic or lifestyle factors (Popescu et al, 2024) (Solanki et al, 2023).

Despite this progress, the complexity of host–microbe interactions poses significant challenges for clinical translation. Differences in diet, geography, and genetics complicate cross-population comparisons, necessitating international collaboration and harmonized data collection methods (Megur et al, 2020).

This article addresses these challenges by systematically synthesizing recent qualitative literature on gut–brain axis inflammation in dementia. It aims to elucidate the molecular, immunological, and microbial mechanisms underpinning this relationship while identifying key gaps in translational research. The discussion will highlight emerging therapeutic strategies and propose future directions for integrating gut-based interventions into dementia management.

The primary objective of this article is to consolidate empirical evidence on gut–brain axis inflammation to enhance understanding of its role in dementia pathogenesis and progression. The expected outcomes include improved theoretical frameworks linking

microbiota to neurodegeneration and practical insights for developing novel interventions that target systemic inflammation as a modifiable risk factor for dementia..

Methods

This study adopts a qualitative research design with a descriptive approach, conducted through library-based analysis. The qualitative paradigm was chosen to capture the complexity of the gut–brain axis inflammation phenomenon within dementia comprehensively, emphasizing depth over quantification (Bingham, 2023) (Pratt, 2025). The descriptive method enables a systematic and transparent portrayal of research findings derived from existing scholarly sources without manipulating variables. This aligns with the interpretive epistemology of qualitative research, which prioritizes understanding patterns, relationships, and meanings in empirical data (Doyle, 2019).

The data sources in this study consisted of peer-reviewed journal articles, official reports, and conceptual papers related to gut microbiota, neuroinflammation, and dementia. Specifically, literature published between 2015 and 2025 was analyzed, emphasizing recent findings in neurobiology, immunology, and microbiome research. Articles were collected from credible databases indexed in Scopus and PubMed, including journals such as Frontiers in Immunology, Gut, Nutrients, and International Journal of Molecular Sciences (Chen et al, 2022) (Lei et al, 2025) (Popescu et al, 2024) (Solanki et al, 2023). Supporting theoretical frameworks regarding the qualitative methodology were drawn from authoritative sources on descriptive design and library research (Abraham, 2024) (Bandaranayake, 2024) (Jimenez, 2024).

Data collection was conducted using systematic literature search and document analysis. The process involved identifying relevant studies through keyword combinations such as “gut–brain axis,” “neuroinflammation,” “Alzheimer’s disease,” and “qualitative analysis.” Inclusion criteria encompassed peer-reviewed empirical and review articles that directly addressed the relationship between gut microbiota and dementia-related inflammation. Exclusion criteria eliminated duplicate records, non-English sources, and non-peer-reviewed materials to maintain validity (Granikov, 2020) (Togia, 2017). This systematic process ensured that the selected literature reflected current scientific consensus and methodological rigor.

The data analysis procedure followed an inductive framework typical of qualitative research. It involved multiple stages: (1) identification and familiarization with key themes) ((2) data reduction by summarizing relevant findings) ((3) thematic categorization of recurring concepts, such as gut permeability, immune signaling, and microbial metabolites) (and (4) synthesis and interpretation through inductive reasoning to construct an integrative understanding of the gut–brain inflammation mechanism (Bingham, 2023) (Belotto, 2018) (Vila-Henninger et al, 2022). Thematic analysis was supported by iterative coding cycles, memoing, and the identification of conceptual relationships, consistent with best practices in qualitative analysis (Kalpokaite, 2018).

To ensure data validity and reliability, several strategies were implemented. Triangulation of sources was applied by cross-referencing multiple peer-reviewed studies addressing similar mechanistic pathways (Fife, 2024). Peer debriefing and conceptual

validation were performed by aligning interpretations with existing reviews and meta-analyses in the field (Lei et al, 2025) (Popescu et al, 2024). Additionally, maintaining an audit trail and transparent coding documentation enhanced the confirmability and trustworthiness of the analysis (Bingham, 2023) (Pratt, 2025).

Ultimately, this methodological framework integrates qualitative rigor with systematic literature synthesis. The combination of descriptive qualitative inquiry and structured document analysis enables a comprehensive understanding of the gut–brain axis's role in dementia pathogenesis. By using a transparent, replicable, and conceptually grounded process, this study contributes both theoretically—to refining interdisciplinary perspectives on neuroinflammation—and practically, by offering an evidence-based foundation for developing microbiota-targeted therapeutic strategies in dementia research.

Results and Discussions

The qualitative synthesis of recent literature revealed that the gut–brain axis plays a pivotal role in the inflammatory mechanisms underlying dementia, particularly Alzheimer's disease (AD). The reviewed studies collectively demonstrated that gut microbiota dysbiosis induces systemic inflammation, disrupts intestinal and blood–brain barriers, and activates microglia—contributing to neurodegeneration and cognitive decline (Lei et al, 2025) (Ortiz-Samur et al, 2025) (Popescu et al, 2024) (Tarawneh & Penhos, 2022). This review integrated empirical evidence from both preclinical and clinical studies, offering a comprehensive understanding of gut-mediated neuroinflammation as a hallmark of dementia pathophysiology.

The findings emphasize several key mechanistic pathways linking gut microbiota to neuroinflammation. Dysbiosis reduces the abundance of beneficial commensal species, particularly Bifidobacterium and Faecalibacterium prausnitzii, which are known for producing anti-inflammatory short-chain fatty acids (SCFAs). Concurrently, an increase in pathogenic bacteria enhances intestinal permeability, facilitating the translocation of bacterial endotoxins such as lipopolysaccharides (LPS) and amyloids into systemic circulation (Bello-Corral et al, 2023) (Lei et al, 2025). These circulating molecules cross the blood–brain barrier and initiate inflammatory cascades involving microglial and astrocytic activation (Ortiz-Samur et al, 2025) (Popescu et al, 2024). The result is the chronic neuroinflammatory milieu that accelerates amyloid-beta (Aβ) aggregation and tau phosphorylation, core pathological features of AD.

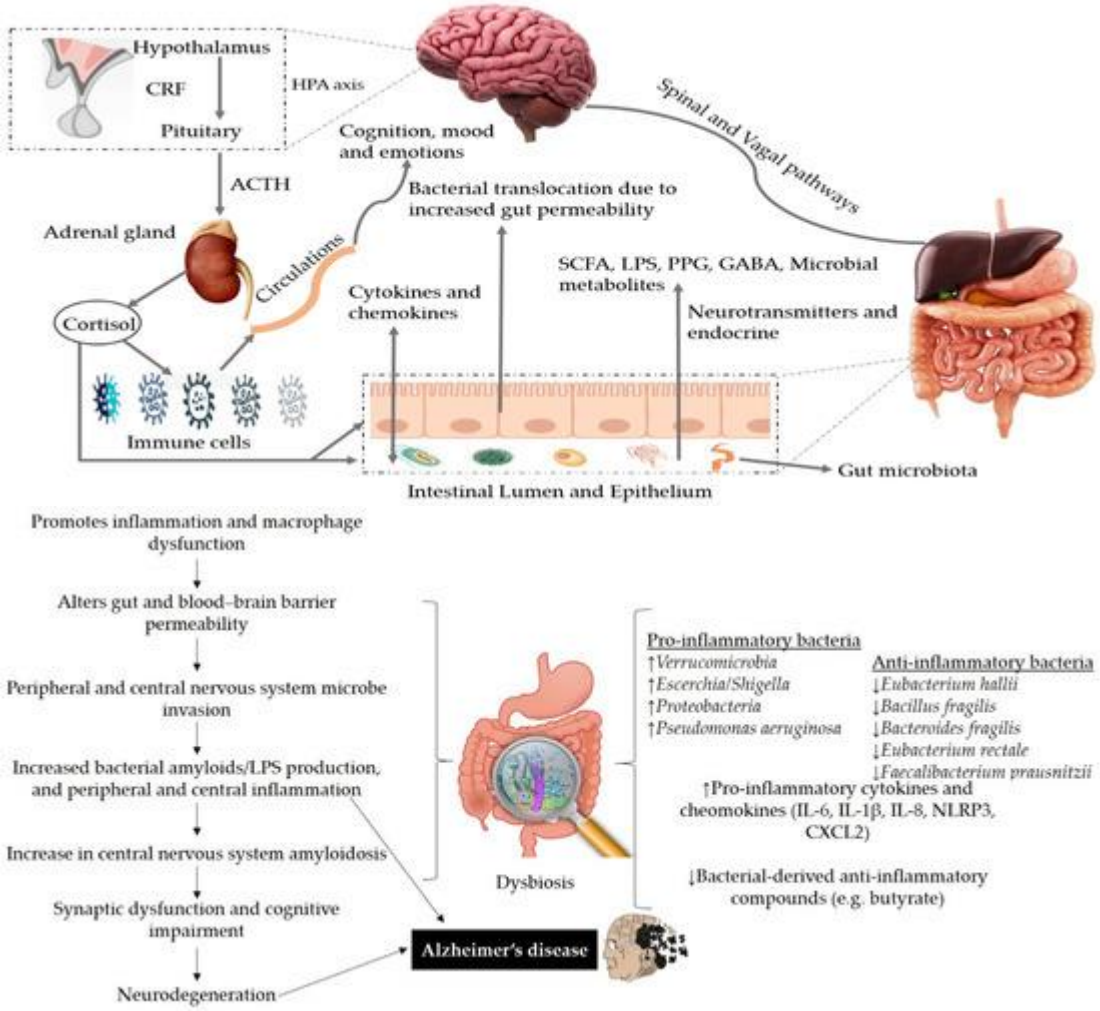

Figure 1. Further analysis
Source: mdpi.com

Further analysis showed that microbial diversity and composition strongly correlate with neuroinflammatory status. In AD models, reductions in butyrate-producing bacteria are associated with upregulated pro-inflammatory cytokines such as IL-6, IL-1β, and TNF-α (Jamerlan et al, 2025) (Tarawneh & Penhos, 2022). Conversely, interventions that restore microbial balance—such as probiotics, prebiotics, and Mediterranean-style diets—have demonstrated measurable declines in inflammatory markers and cognitive improvement in preclinical and preliminary human studies (Ayten & Bilici, 2024) (Shandilya et al, 2021). This indicates that microbial regulation of systemic immune signaling represents a promising therapeutic strategy.

Table 1. Four Dominant Categories Of Findings: Microbial Dysbiosis, Microglial Activation, Diet And Lifestyle Influences, And Microbiota-Targeted Therapies

Mechanism / Intervention	Key Insights	Supporting References
Microbial dysbiosis	Increases gut permeability, neuroinflammation, and cognitive decline	(Bello-Corral et al, 2023) (Jamerlan et al, 2025) (Lei et al, 2025) (Popescu et al, 2024) (Tarawneh & Penhos, 2022)
Microglial activation	Modulated by gut-derived metabolites such as SCFAs and LPS	(Jamerlan et al, 2025) (Lei et al, 2025) (Ortiz-Samur et al, 2025) (Popescu et al, 2024)
Diet and lifestyle	Western diets exacerbate, while Mediterranean/probiotic diets alleviate inflammation	(Ayten & Bilici, 2024) (Jamerlan et al, 2025) (Shandilya et al, 2021)
Microbiota-targeted therapies	Probiotics, prebiotics, and fecal microbiota transplantation (FMT) show promise in reducing neuroinflammation	(Ayten & Bilici, 2024) (Jamerlan et al, 2025) (Lei et al, 2025) (Popescu et al, 2024) (Zhang et al, 2024)

Microglial activation emerged as a consistent finding across studies. Microglia, the brain's resident immune cells, were found to respond dynamically to gut-derived metabolites. Under dysbiotic conditions, microglial activation was characterized by increased expression of inflammatory mediators and oxidative stress markers, leading to synaptic dysfunction(Ortiz-Samur et al, 2025). In contrast, balanced microbial metabolite signaling through SCFAs such as butyrate promoted anti-inflammatory microglial phenotypes, thereby preserving neural integrity (Lei et al, 2025).

Dietary and lifestyle patterns significantly modulated these processes. Western diets, rich in saturated fats and refined carbohydrates, intensified gut dysbiosis and neuroinflammation, while Mediterranean and vegetarian diets rich in fiber, polyphenols, and omega-3 fatty acids were associated with microbial restoration and reduced neuroinflammatory burden (Ayten & Bilici, 2024) (Shandilya et al, 2021). These findings underscore the bidirectional interplay between environmental factors and microbial regulation of brain health.

Therapeutic studies have begun exploring microbiota-targeted interventions. Although evidence remains primarily preclinical, probiotics and fecal microbiota transplantation (FMT) demonstrate the potential to remodel gut microbial ecosystems and attenuate neuroinflammation (Jamerlan et al, 2025) (Zhang et al, 2024). Nevertheless, variability in experimental design, patient populations, and microbial strains limits the generalizability of findings (Popescu et al, 2024). Longitudinal clinical trials are required to establish causality and safety profiles.

Finally, the reviewed literature identified key research gaps, including the need to (1) identify specific microbial strains and metabolites responsible for modulating neuroinflammation, (2) integrate multi-omics analyses for mechanistic insights, and (3) translate preclinical findings into human applications (Lei et al, 2025) (Popescu et al, 2024) (Zhang et al, 2024). Despite these challenges, the qualitative convergence of evidence

confirms that the gut–brain axis is not merely a contributing factor but a central mediator in dementia pathogenesis.

Discussion

The collective analysis of recent qualitative literature underscores the gut–brain axis inflammation as a central and multidimensional pathway in the etiology and progression of dementia, particularly Alzheimer's disease (AD). The reviewed studies converge on the premise that gut microbiota dysbiosis—characterized by the imbalance between beneficial and pathogenic bacterial populations—triggers systemic and neural inflammatory responses, thereby contributing to cognitive impairment (Lei et al, 2025) (Popescu et al, 2024) (Tarawneh & Penhos, 2022). These findings strengthen the neuroimmunological theory of AD, which posits that peripheral immune dysregulation and chronic inflammation serve as initiating factors for neurodegeneration rather than secondary consequences.

Conceptually, these results align with the microbiota–immune–brain axis model, a framework that integrates microbial signaling, metabolic by-products, and immune modulation within the context of neuroinflammation. Dysbiosis leads to increased gut permeability, facilitating the translocation of microbial metabolites such as lipopolysaccharides (LPS), amyloids, and short-chain fatty acids (SCFAs) into systemic circulation (Bello-Corral et al, 2023) (Ortiz-Samur et al, 2025). These metabolites cross the blood–brain barrier, activate microglial cells, and perpetuate neuroinflammatory cascades that exacerbate amyloid-beta (Aβ) aggregation and tau hyperphosphorylation (Jamerlan et al, 2025) (Lei et al, 2025). The convergence of these pathways supports the neuroimmune homeostasis disruption theory, suggesting that AD pathophysiology arises from chronic miscommunication between gut microbiota and the central nervous system.

The findings also indicate significant dietary and lifestyle influences on gut microbial composition. Western diets—rich in saturated fats and refined carbohydrates—intensify dysbiosis, oxidative stress, and neuroinflammation, while Mediterranean and vegetarian dietary patterns are associated with increased microbial diversity and anti-inflammatory metabolite production (Ayten & Bilici, 2024) (Shandilya et al, 2021). This confirms the hypothesis that dietary modulation acts as an upstream regulator of the gut–brain axis, influencing systemic metabolic and immune equilibrium. The implications extend beyond nutrition, highlighting that behavioral and environmental factors directly shape microbial ecosystems that affect brain health. Such insights broaden the scope of dementia prevention strategies toward holistic and lifestyle-centered interventions.

Therapeutically, emerging studies demonstrate the potential of microbiota-targeted interventions such as probiotics, prebiotics, and fecal microbiota transplantation (FMT) in alleviating neuroinflammation and cognitive deficits (Lei et al, 2025) (Popescu et al, 2024) (Zhang et al, 2024). Probiotic formulations containing Lactobacillus and Bifidobacterium species have been reported to modulate immune responses, reduce oxidative stress, and enhance memory-related synaptic plasticity in experimental models (Jamerlan et al, 2025). However, the evidence remains heterogeneous and largely preclinical, indicating a translational gap between laboratory efficacy and clinical applicability. Factors such as

microbial strain specificity, dosage optimization, host variability, and long-term safety require further investigation before establishing standardized therapeutic protocols.

Despite the robust theoretical and empirical support, several limitations are evident in the existing body of research. First, most studies rely on animal models or cross-sectional human designs, limiting causal inference and temporal understanding of microbial shifts during disease progression (Jamerlan et al, 2025) (Tarawneh & Penhos, 2022). Second, methodological inconsistencies—such as variable sequencing techniques, dietary control, and participant demographics—introduce heterogeneity that complicates meta-analytic synthesis (Bello-Corral et al, 2023) (Popescu et al, 2024). Third, few studies have employed multi-omics approaches that integrate metagenomic, metabolomic, and immunologic data to delineate the mechanistic link between microbial metabolites and neural outcomes (Lei et al, 2025) (Zhang et al, 2024). These limitations highlight the necessity for standardized methodologies and longitudinal designs to enhance reproducibility and clinical relevance.

From a broader perspective, the implications of this synthesis extend into theoretical refinement and clinical innovation. Theoretically, the integration of microbiome science with neuroinflammatory and neurodegenerative frameworks enriches the conceptual understanding of AD as a systemic disease rather than a purely neural pathology. Practically, these insights promote the development of preventive and therapeutic strategies focusing on gut microbial modulation, personalized nutrition, and immune regulation. Future research should prioritize long-term clinical trials, population-based cohort studies, and multi-dimensional data integration to clarify causality and identify biomarkers for early diagnosis.

In conclusion, the current synthesis establishes that gut–brain axis inflammation constitutes a biological nexus through which microbial, immune, and neural systems converge to influence dementia risk and progression. While findings affirm the therapeutic promise of microbiome modulation, the path forward requires multidisciplinary collaboration integrating neuroscience, microbiology, immunology, and nutritional science. Addressing methodological gaps and expanding translational research will be pivotal for transforming microbiota-based therapies from experimental potential to clinically validated interventions capable of reshaping dementia prevention and treatment paradigms.

Conclusion

This qualitative synthesis concludes that inflammation along the gut–brain axis serves as a key biological framework in understanding dementia pathogenesis, particularly Alzheimer's disease. Gut microbiota dysbiosis induces systemic and neural inflammation through immune activation, metabolic disruption, and microglial dysregulation, reframing dementia from a purely neurocentric disorder to one rooted in neuroimmune–microbial interaction. These findings highlight the crucial role of dietary patterns, lifestyle, and environmental factors in shaping neurocognitive health. To address this, practitioners and policymakers are encouraged to integrate microbiota-centered strategies—such as nutritional interventions, probiotics, and lifestyle modifications—into dementia prevention and care. Academically, future studies should employ multi-omics and longitudinal approaches to validate causal mechanisms, while fostering interdisciplinary collaboration

among neuroscientists, nutritionists, and behavioral experts. Such efforts will strengthen the translational and culturally adaptive application of gut–brain research, paving the way for evidence-based and sustainable models in dementia prevention and management.

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