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THE GUT–BRAIN AXIS IN NEURODEGENERATIVE DISEASES: LINKING MICROBIOTA DYSBIOSIS, BARRIER DYSFUNCTION, AND NEUROINFLAMMATION

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Abstract

Neurodegenerative diseases (NDDs), including Alzheimer's disease, Parkinson's disease, multiple sclerosis, and amyotrophic lateral sclerosis, are characterized by progressive neuronal dysfunction and loss. Neuroinflammation, immune dysregulation, oxidative stress, and mitochondrial dysfunction are increasingly recognized as interconnected mechanisms in disease progression. The gut–brain axis provides a bidirectional link between the gastrointestinal tract and central nervous system through neural, immune, endocrine, and metabolic pathways. Gut microbiota dysbiosis may contribute to neurodegeneration through impaired intestinal barrier integrity, altered microbial metabolites, systemic inflammation, and blood–brain barrier dysfunction. Changes in short-chain fatty acids, tryptophan-derived metabolites, bile acids, and microbial products such as lipopolysaccharide may modulate immune and glial signaling, including TLR4/NF-κB and NLRP3 pathways. Evidence is particularly prominent in Alzheimer's and Parkinson's diseases, although microbial alterations remain heterogeneous across studies. The gut–brain axis represents a potentially modifiable interface linking microbial dysbiosis and neuroinflammation. Microbiome-targeted interventions show promising but inconsistent results. Further integration of microbiomics, metabolomics, immunophenotyping, and longitudinal clinical data is needed to establish causality and advance precision strategies for neurodegenerative diseases.

Keywords: *gut–brain axis; gut microbiota; dysbiosis; neuroinflammation; neurodegenerative diseases; blood–brain barrier; microbiome; microglia.*

INTRODUCTION

Neurodegenerative diseases (NDDs) are heterogeneous disorders characterized by progressive neuronal dysfunction and loss, resulting in cognitive, motor, behavioral, and functional impairment. Alzheimer's disease (AD), Parkinson's disease (PD), multiple sclerosis (MS), and amyotrophic lateral sclerosis (ALS) involve complex interactions among neuronal, glial, metabolic, and immune mechanisms. Increasing evidence indicates that neuroinflammation is not merely a secondary response to neuronal injury but may actively contribute to disease initiation, progression, and propagation. In AD, microglial and astrocytic activation is associated with amyloid-β and tau pathology, whereas in PD, neuroinflammatory responses interact with α-synuclein pathology and dopaminergic neuronal degeneration. These processes are accompanied by oxidative stress, mitochondrial dysfunction, impaired synaptic homeostasis, and blood–brain barrier (BBB) disruption, supporting an integrated model of neurodegeneration involving peripheral immunity and systemic metabolic signals.

The gut–brain axis has emerged as a potential interface connecting intestinal microbial ecology with brain physiology and disease. It encompasses bidirectional communication between the gastrointestinal tract and central nervous system through neural, immune, endocrine, and metabolic pathways. The vagus

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nerve and enteric nervous system provide major neural routes, while circulating cytokines, microbial metabolites, and hypothalamic–pituitary–adrenal signaling contribute additional pathways. The intestinal barrier and BBB further regulate the movement of microbial products and host-derived signals. The gut microbiota is a metabolically active ecosystem capable of producing or modifying short-chain fatty acids (SCFAs), bile acid derivatives, tryptophan metabolites, neurotransmitter-related molecules, and microbial components such as lipopolysaccharide (LPS). Recent work emphasizes that microbial metabolites may represent functional mediators of gut–brain communication because several metabolites can enter the circulation and interact with pathways relevant to neurodegenerative pathology. (Loh et al., 2024; Fu et al., 2024; Gut microbial metabolism in Alzheimer's disease and related dementias, 2024). (Abdullah et al., 2021)

Gut dysbiosis may contribute to neurological disease through disruption of intestinal barrier integrity, increased intestinal permeability, altered microbial metabolites, and systemic immune activation. LPS and other microbial-associated molecular patterns can activate innate immune signaling, including TLR4/NF-κB pathways, promoting pro-inflammatory mediator production. However, the relevance of these pathways to human neurodegenerative disease should be interpreted cautiously because much of the mechanistic evidence derives from experimental models, whereas human studies predominantly demonstrate associations between microbial alterations, metabolites, and disease phenotypes.

The intestinal barrier and BBB represent critical interfaces in this process, creating a potential continuum of gut dysbiosis → intestinal barrier dysfunction → systemic inflammation → BBB dysfunction → CNS immune activation. Experimental evidence indicates that microbial signals and metabolites can influence barrier function, while human studies have demonstrated that BBB dysfunction is associated with AD pathology, cognitive impairment, and neuroinflammatory processes. A 2024 study using DELCODE and DESCRIBE cohorts found that BBB dysfunction was associated with AD-related pathology and cognitive impairment, supporting the importance of barrier integrity in neurodegeneration

Beyond microbial composition, SCFAs, tryptophan metabolites, and bile acid derivatives represent important molecular mediators of gut–brain communication. These metabolites can influence epithelial integrity, immune regulation, cellular metabolism, microglial function, and host signaling pathways. Human cohort studies have begun to link SCFA profiles with AD pathology. In a 2024 cohort study involving 124 participants, fecal SCFAs were examined in relation to amyloid positivity and CSF biomarkers, providing human evidence for investigating microbial metabolites as potential intermediates between gut microbiota and AD-related pathology.

Evidence from AD and PD provides increasing support for an association between gut microbial alterations and neurodegenerative phenotypes. In AD, human studies have identified differences in microbial composition and function associated with cognitive impairment and AD-related biomarkers. Recent metagenomic research further demonstrated that gut microbiome composition and functional characteristics differed according to AD status and were associated with CSF biomarkers.

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In PD, human studies similarly demonstrate alterations in gut microbial composition and functional pathways. A 2024 multi-country meta-analysis of shotgun sequencing datasets identified reproducible differences in microbial diversity and several microbial taxa and metabolic pathways, although the specific microbial signatures varied across populations. (Nishiwaki et al., 2024). Diet also appears to be an important modifier, as a 2024 study of 85 individuals with PD found associations between higher diet quality, fiber intake, and putative butyrate-producing bacteria.

METHODS

This narrative literature review was conducted to synthesize current evidence on the role of the gut–brain axis in neurodegenerative diseases, with particular emphasis on gut microbiota dysbiosis, intestinal and blood–brain barrier dysfunction, microbial metabolites, systemic inflammation, microglial activation, and neuroinflammation. Literature searches were performed using PubMed/MEDLINE, Scopus, Web of Science, and Google Scholar. Publications from January 2020 to August 2026 were prioritized to capture recent evidence, while earlier seminal studies were considered when necessary to provide essential mechanistic context.

The search strategy combined controlled vocabulary and free-text terms, including “gut–brain axis,” “gut microbiota,” “dysbiosis,” “neuroinflammation,” “neurodegenerative diseases,” “Alzheimer’s disease,” “Parkinson’s disease,” “microbial metabolites,” “blood–brain barrier,” “microglia,” and “intestinal barrier.” Peer-reviewed original studies, systematic reviews, meta-analyses, and relevant narrative reviews published in English were considered. Studies focusing on human populations, translational research, and mechanistic experimental evidence were prioritized.

Relevant literature was critically synthesized according to disease mechanisms, microbial alterations, microbial metabolites, immune signaling pathways, barrier dysfunction, and therapeutic implications. Particular attention was given to consistency of findings, methodological limitations, potential confounding factors, and the distinction between association and causality.

RESULTS

Gut microbiota alterations in neurodegenerative diseases

The reviewed literature consistently indicates an association between gut microbiota alterations and neurodegenerative diseases, particularly Alzheimer’s disease (AD) and Parkinson’s disease (PD). However, specific microbial signatures vary considerably across studies, suggesting that disease-associated dysbiosis is influenced by geographical, dietary, methodological, and clinical factors. In PD, a 2024 meta-analysis of shotgun metagenomic datasets from six countries identified increased microbial α-diversity and alterations in several bacterial taxa, including increased Akkermansia muciniphila and decreased Roseburia intestinalis and Faecalibacterium prausnitzii. Functional analyses also demonstrated reductions in microbial pathways related to riboflavin and biotin biosynthesis, as well as decreased fecal short-chain fatty acids (SCFAs) and polyamines.

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Recent human studies further support the association between gut microbial alterations and neurodegenerative phenotypes. In AD, shallow-shotgun metagenomic analysis demonstrated differences in microbial composition and function between individuals with and without AD dementia, with microbial features associated with CSF biomarkers of AD pathology. Similarly, a 2025 study examining different stages of AD evaluated gut microbiota and SCFA profiles across cognitively unimpaired and cognitively impaired groups, further supporting investigation of microbial metabolites during disease progression.

In AD, human studies similarly report differences in gut microbial composition and associations with cognitive impairment, although findings are not sufficiently consistent to define a universal microbiome signature. These observations support the concept that functional dysbiosis, rather than changes in individual bacterial taxa alone, may be more relevant to neurodegenerative processes.

Gut dysbiosis, barrier dysfunction, and systemic inflammation

The literature indicates that gut dysbiosis may impair intestinal barrier integrity and increase exposure to microbial-derived molecules. Altered microbial products can promote peripheral immune activation and inflammatory signaling, providing a potential pathway between intestinal dysfunction and CNS inflammation. This relationship is particularly relevant because systemic inflammatory mediators may influence blood–brain barrier integrity and subsequently affect the neuroimmune environment.

Microbial metabolites and neuroinflammation

Microbial metabolites represent an important molecular link between intestinal microbiota and brain function. SCFAs, bile acid derivatives, and tryptophan metabolites can influence epithelial integrity, immune regulation, cellular metabolism, and microglial activity. Human cohort evidence is beginning to connect these metabolites with neurodegenerative biomarkers. In a cohort of 124 participants, fecal SCFAs were evaluated in relation to AD diagnosis, amyloid positivity, and CSF biomarkers, providing clinical evidence for further investigation of SCFAs as potential mediators of gut–brain interactions.

In PD, a multi-country shotgun metagenomic meta-analysis identified reductions in fecal SCFAs and polyamines alongside alterations in microbial functional pathways, although the direction and magnitude of these alterations varied across populations (Nishiwaki et al., 2024).

These mechanisms provide a plausible connection between gut dysbiosis and activation of innate immune pathways, including TLR4/NF-κB and NLRP3-associated signaling. Persistent neuroimmune activation may subsequently contribute to oxidative stress, synaptic dysfunction, and neuronal injury.

Diet and other host-related modifiers

The reviewed evidence indicates that gut microbiota composition is strongly influenced by host and environmental factors. In PD, a 2024 human study involving 85 patients demonstrated associations between diet quality and fiber intake with microbial diversity and the abundance of putative butyrate-producing bacteria. These findings highlight diet as an important potential confounder when interpreting associations between microbiota composition and neurodegenerative disease. Consequently, differences in diet, medication exposure, geography, age,

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and disease stage should be considered when comparing microbiome profiles across studies

Evidence from Alzheimer's and Parkinson's diseases

AD and PD currently provide the strongest clinical evidence for gut–brain interactions. In PD, gastrointestinal manifestations may precede motor symptoms, supporting investigation of gut-associated mechanisms in disease development. However, current evidence does not establish that PD consistently originates in the gut.

In AD, microbiome alterations have been associated with cognitive impairment and pathological processes involving amyloid-β, tau, and neuroinflammation. Nevertheless, the direction of causality remains uncertain, as disease progression, medication, diet, age, and lifestyle may independently alter the gut microbiome.

Microbiome-targeted interventions

Clinical investigations have evaluated dietary interventions, probiotics, prebiotics, synbiotics, and fecal microbiota transplantation as potential strategies for modifying neurodegenerative disease-related outcomes. A 2024 systematic review and meta-analysis demonstrated growing clinical interest in these approaches but also highlighted substantial heterogeneity among interventions and outcomes. Consequently, microbiome modulation remains promising but cannot yet be considered an established disease-modifying treatment for neurodegenerative diseases.

DISCUSSIONS

Gut dysbiosis as a potential contributor to neurodegeneration

The current evidence supports an association between gut microbiota alterations and neurodegenerative diseases, particularly Alzheimer's disease (AD) and Parkinson's disease (PD). Importantly, emerging human evidence supports an association rather than a uniform causal relationship. A 2024 multivariable Mendelian randomization analysis investigated potential causal relationships between gut microbiota, microbiota-derived metabolites, and AD, illustrating the growing use of genetic epidemiological approaches to address causality. However, such approaches remain dependent on the validity of genetic instruments and cannot substitute for longitudinal experimental or clinical evidence.However, the observed heterogeneity across studies suggests that neurodegeneration cannot be explained by a single disease-specific microbial signature. This interpretation is supported by a 2024 meta-analysis of shotgun metagenomic studies in PD, which demonstrated differences in microbial composition across countries and identified increased α-diversity in patients with PD. Importantly, microbial taxa associated with specific functional pathways differed between geographic populations, emphasizing the influence of environmental and host-related factors.

These findings suggest that the biological relevance of dysbiosis may depend less on the presence or absence of individual bacterial taxa and more on microbial functional capacity. Changes in microbial metabolic pathways may alter the availability of SCFAs, polyamines, vitamins, bile acid derivatives, and other bioactive metabolites. Therefore, future interpretation of microbiome studies

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should move beyond taxonomic descriptions toward integrated analyses of microbial genes, metabolites, host immune responses, and clinical phenotypes.

From intestinal dysfunction to neuroinflammation

A plausible mechanistic pathway connecting gut dysbiosis with neurodegeneration involves disruption of intestinal barrier integrity and subsequent systemic immune activation. Altered microbial communities may modify epithelial function and increase exposure to microbial-associated molecular patterns such as LPS. Activation of innate immune pathways, including TLR4/NF-κB signaling, may promote systemic inflammatory responses that subsequently influence BBB integrity and CNS immune activity.

Nevertheless, this pathway should be interpreted as a biologically plausible mechanism rather than an established causal sequence. Human studies remain predominantly associative, and factors such as age, diet, medication, disease severity, and lifestyle can simultaneously influence both microbiota composition and neurodegenerative outcomes. Thus, gut dysbiosis may function as a contributor, consequence, or amplifier of neurodegeneration rather than a single initiating factor.

The BBB may represent an important intermediate interface in this process. Human cohort data indicate that BBB dysfunction is associated with AD pathology and cognitive impairment, while experimental studies provide additional evidence that microbial metabolites can influence barrier integrity. However, direct evidence demonstrating that gut dysbiosis causes BBB disruption in humans remains limited.

Microbial metabolites as functional mediators

The emphasis on microbial metabolites provides a more mechanistically informative framework than taxonomic composition alone. SCFAs, particularly acetate, propionate, and butyrate, can influence epithelial integrity and immune regulation, while microbial tryptophan metabolites and bile acid derivatives can modulate host signaling pathways. Changes in these metabolites may consequently influence microglial activity, inflammatory signaling, and neuronal homeostasis.

The PD metagenomic meta-analysis provides an important example: microbial functional alterations were accompanied by reductions in fecal SCFAs and polyamines. However, these observations should not be interpreted as proof that reduced metabolites directly cause PD. Rather, they support the hypothesis that microbial metabolic dysfunction may represent an intermediate phenotype linking intestinal dysbiosis with neurological disease.

Microglia as a potential convergence point

Microglia may represent a critical convergence point between peripheral microbial signals and CNS pathology. Experimental evidence indicates that microbial-associated inflammatory signals can influence innate immune pathways, including TLR4/NF-κB and NLRP3-associated signaling, while human studies provide indirect support through associations between gut microbial alterations, circulating metabolites, inflammatory phenotypes, and neurodegenerative biomarkers. Therefore, these pathways should currently be regarded as mechanistic hypotheses supported by complementary experimental and clinical evidence rather than established causal mechanisms in humans.

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Evidence from Alzheimer's and Parkinson's disease

AD and PD currently provide the strongest clinical evidence supporting the gut–brain axis hypothesis. In PD, gastrointestinal abnormalities may occur before classical motor manifestations, making the gut particularly relevant to disease pathogenesis. Nevertheless, the hypothesis that PD consistently originates in the gut remains unresolved and should not be generalized to all patients.

In AD, alterations in gut microbiota have been associated with cognitive impairment and pathological processes involving amyloid-β, tau, and neuroinflammation. However, similar to PD, current evidence does not establish whether microbiome alterations precede disease development or emerge during disease progression.

Thus, the most defensible interpretation is that gut dysbiosis may modify disease susceptibility or progression rather than independently determine disease onset.

Therapeutic implications

The therapeutic potential of manipulating the gut microbiome is increasingly investigated through dietary interventions, probiotics, prebiotics, synbiotics, and fecal microbiota transplantation. A 2024 systematic review and meta-analysis demonstrated growing clinical evidence for microbiome-based interventions in neurodegenerative diseases but also identified substantial heterogeneity in intervention types and clinical outcomes.

Clinical trials of probiotics, prebiotics, synbiotics, and fecal microbiota transplantation remain heterogeneous, and microbiome-targeted interventions should therefore be considered investigational rather than established disease-modifying therapies (Chui et al., 2024).

This heterogeneity is important because "microbiome modulation" should not be considered a single therapeutic strategy. Different interventions can produce different microbial and metabolic effects, and the same intervention may have different outcomes depending on baseline microbiota, diet, medication exposure, disease stage, and host characteristics. Consequently, future interventions may need to shift from generalized probiotic approaches toward precision microbiome modulation, potentially guided by microbial function and metabolomic profiles.

Translational challenges and future perspectives

Several barriers currently limit translation into clinical practice. First, microbiome composition varies substantially between individuals and populations. Second, medication and dietary factors can act as major confounders. Third, many mechanistic findings originate from experimental models that cannot fully reproduce human disease. Fourth, observational associations cannot establish temporal or causal relationships.

Future research should therefore integrate metagenomics, metabolomics, immune profiling, and longitudinal clinical phenotyping. Such approaches may help determine whether particular microbial functions or metabolites represent biomarkers, therapeutic targets, or merely consequences of neurodegeneration.

CONCLUSIONS

Current evidence indicates that the gut–brain axis represents an important immunometabolic interface linking intestinal microbial ecology with

neuroinflammatory processes in neurodegenerative diseases. Gut microbiota dysbiosis may influence disease progression through disruption of intestinal barrier integrity, altered microbial metabolites, systemic immune activation, blood–brain barrier dysfunction, and subsequent modulation of microglial and astrocytic responses. Microbial-derived metabolites, including short-chain fatty acids, tryptophan metabolites, and bile acid derivatives, may serve as important molecular mediators of these interactions.

However, the available evidence does not yet establish gut dysbiosis as a primary cause of neurodegeneration. Microbiome alterations may represent a contributor, consequence, or amplifier of disease, while age, diet, medication, geography, lifestyle, and disease severity may substantially influence microbiome profiles. Clinical evidence for microbiome-targeted interventions is promising but remains heterogeneous. A 2024 systematic review and meta-analysis found improvements in overall disease burden following microbiome-modulating interventions, but emphasized small sample sizes and substantial methodological heterogeneity.

Thus, the gut–brain axis should currently be regarded as a potentially modifiable pathway rather than an established therapeutic target for disease modification.

RECOMMENDATIONS

Future research should prioritize longitudinal and well-controlled human studies capable of distinguishing causality from association. Integration of metagenomics, metabolomics, immunophenotyping, and clinical phenotyping is recommended to identify functional microbial signatures rather than relying solely on bacterial abundance.

Clinical trials of probiotics, prebiotics, synbiotics, postbiotics, dietary interventions, and fecal microbiota transplantation should employ standardized microbiome assessment, adequate sample sizes, longer follow-up, and clinically meaningful neurological outcomes. Current evidence supports continued investigation but does not yet justify routine microbiome-based therapy as a replacement for established neurodegenerative disease management.

Ultimately, future research should move toward precision microbiome medicine, in which individual microbial composition, metabolic function, host immune phenotype, disease stage, diet, and medication exposure are integrated to identify patients most likely to benefit from gut–brain axis modulation.

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