ISSN: 3065-4874
Review Article
Journal of Psychiatry and Neurochemistry Research

The Gut-Brain Axis in Psychiatric and Neurodegenerative Disorders: Microbial Influences on Inflammation, Cognition, and Therapeutic Response

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Submitted: 14 July 2025 **Accepted:** 21 July 2025 **Published:** 31 July 2025

https://doi.org/10.63620/MKJPNR.2025.1071

Citation: *Hamza, N., Victorita, B., Vasa, M. C. M., & Sabeur, L. (2025). The Gut-Brain Axis in Psychiatric and Neurodegenerative Disorders: Microbial Influences on Inflammation, Cognition, and Therapeutic Response. J of Psych and Neuroche Res, 3(4), 01-06.*

Abstract

The gut-brain axis is a complex bidirectional communication system between the gastrointestinal tract and the central nervous system, involving neural, hormonal, immune, and microbial pathways. Increasing evidence supports the gut microbiota's critical role in modulating brain function and behavior, with implications for various psychiatric and neurodegenerative disorders. This review examines current literature on the mechanistic interplay between gut dysbiosis and mental health, focusing on conditions such as depression, anxiety, Parkinson's disease (PD), Alzheimer's disease (AD), schizophrenia, bipolar disorder, and autism spectrum disorder (ASD). The vagus nerve serves as a major conduit for microbial metabolites—such as short-chain fatty acids (SCFAs), tryptophan metabolites, and neurotransmitters—affecting mood, cognition, and neuroinflammation. Immune responses involving microglia and cytokines (e.g., IL-6, IL-1β) mediate neuroinflammatory cascades via the microbiota–immune–brain axis. The NLRP3 inflammasome emerges as a central inflammatory mechanism in neurodegeneration. Emerging therapies, including psychobiotics, dietary modifications, prebiotics, probiotics, and fecal microbiota transplantation, show promise but require further validation through robust human trials. Psychotropic medications, particularly SSRIs and tricyclics, exhibit antimicrobial activity, potentially inducing dysbiosis and altering gut barrier functions. While animal and in vitro models offer valuable insights, translation to human clinical outcomes remains limited. Despite these challenges, the gut-brain axis represents a promising frontier for novel diagnostic and therapeutic strategies targeting psychiatric and neurodegenerative diseases. Understanding the strain-specific effects, immune-microbiome crosstalk, and microbiota-derived metabolic pathways is critical for personalized medicine approaches.

Keywords: Gut-brain Axis, Microbiota–Gut–Brain Communication, Neuroinflammation; Psychiatric Disorders, Neurodegenerative Diseases, Short-Chain Fatty Acids (SCFAs)

Introduction

The Gut-Brain axis is a bidirectional and a complex communication pathway which permits the central nervous system (CNS) to exert influence over the gastro intestinal function in response to stress while the gut microbiota regulates the cns via immune, neuroendocrine and vagal pathways. The Gut – Brain axis has significant implications for neurodegenerative, psychiatric, and metabolic disorders. Recent studies have shown the role of the gut microbiome in conditions such as Parkinson's disease with evidence indicating that the gut dysfunction can precede the motor symptoms by decades [1]. The gastrointestinal tract is populated with a diverse community of microbes, the microbiota gut axis represents an important regulator of the glial functions, which makes it an actional target to ameliorate the development

and progression of the neurogenerative diseases. In this review we discuss the mechanisms of the microbiota gut brain axis in the neurodegenerative diseases. Microglial activation and neuroinflammation are pathological hallmarks of neurodegenerative diseases. The microbiota–gut–brain axis represents an important regulator of glial functions, making it an actionable target to ameliorate the development and progression of neurodegenerative diseases. We examine the potential of targeting the intestinal barrier, blood–brain barrier (BBB), meninges, and peripheral immune system to modulate the microbiota–gut–brain axis and counteract glial dysfunction and neurodegeneration [2].

Psych biotics affect the human behavior and the central nervous system process via the gut – brain axis, involving neuronal, immune and metabolic pathways. The vagus nerve serves as a primary conduit for the signals between the gut and the brain, facilitating communications through neuroendocrine and neuroimmune pathways. The afferent fibers from vagus nerve can detect microbial metabolites like short chain fatty acids, neurotransmitters that are produced and modulated by the gut microbes [3, 4]. It can modulate it by detecting the microbial metabolites and relay signals that modulate mood, performance and even neurodegeneration. Its regulatory role spans immune response, digestion and heart rate and emotional states.

In PD the vagus nerve is implicated in the disease development and the progression through its ability to sense the gut- derived signals.

The gut microbiome shapes the immune landscape, influencing the neuroinflammatory process that impact the brain, its composition affects the balance between the proinflammatory and the anti- inflammatory T cell populations, which in turn can modulate the neuropathic pain and other functions [5-8]. Alterations in gut microbiota can shift immune responses from proinflammatory (e.g., Th1 cells) to anti-inflammatory (e.g., Foxp3+ regulatory T cells) profiles. In animal models of neuropathic pain, changes in gut microbiota attenuate pain behaviors by promoting anti-inflammatory immune responses in the CNS. Chronic inflammation mediated by gut microbes is linked to both the metabolic and neurological disorders. Short chain fatty acids module immune response and influence the central appetite regulation. Microbiota- derived neurotransmitters and peptides affect the neuronal signaling and have been implicated in the mood regulation, cognition, and neurodegenerative disease progression. Changes in the microbial metabolites are also associated with both healthy aging and neuropsychiatric conditions like depression and anxiety [9].

Neurotransmitters Produced by Gut Bacteria

Metabolic pathways through which microbiota modulate brain function: Short-chain fatty acids (SCFAs) (e.g., acetate, propionate, butyrate), tryptophan catabolites, and bile acids are important metabolites generated by the gut microbiota [10]. These molecules impact the blood-brain barrier, neurotransmitter synthesis (e.g., serotonin, dopamine), and neuroinflammation—all key components of psychiatric pathophysiology [11]. Immune-Related cells play a role in brain gut communication: major players are microglia (brain resident macrophages), T cells, dendritic cells, and intestinal epithelial cells. Gut-derived signals of inflammation, or the components of microbes, can activate these cells, initiating neuroinflammation associated with disorders such as Alzheimer's and anxiety [12]. Microbiota–Immune–Brain Axis components of the bacterial cell, such as LPS (lipopolysaccharide), activate TLRs (Toll-like receptors) on immune cells, which results in the release of proinflammatory cytokines (e.g., IL-6, TNF-α) [13]. These cytokines can also cross blood–brain barriers, modifying neuronal plasticity and behavior. Similar alterations in the gut microbiota were also witnessed in autistic children and were also associated with the tryptophan metabolic activities and augmented intestinal permeability in autism spectrum disorder (AS). This results in the activation of the immune response and the dysregulation of the CNS signaling pathways important for the social and cognitive dysfunction. Gut permeability and the microbial diversity are modulated by stress hormones (cortisol) [14].

Recent studies highlight the critical role of the NLRP3 inflammasome in the pathogenesis of neurodegenerative diseases such as Alzheimer's and Parkinson's. The NLRP3 inflammasome is a multiprotein complex found in immune cells like microglia, which, upon activation, promotes the maturation and secretion of pro-inflammatory cytokines such as interleukin-1β (IL-1β) and interleukin-18 (IL-18). These molecules are central to the neuroinflammatory response observed in many brain disorders. In Alzheimer's disease, the accumulation of amyloid-beta (Aβ) plaques serves as a danger associated molecular pattern (DAMP) that activates the NLRP3 inflammasome in microglial cells. This activation triggers a chronic inflammatory state, damaging surrounding neurons and accelerating disease progression. A similar mechanism is seen in Parkinson's disease, where misfolded alpha-synuclein aggregates also act as DAMPs, stimulating inflammasome activation and contributing to dopaminergic neuron loss. Moreover, emerging evidence indicates a strong link between gut microbiota and brain health. Dysbiosis—or an imbalance in the gut microbial composition—can promote systemic inflammation by increasing intestinal permeability and allowing bacterial components (e.g., lipopolysaccharides) to enter the bloodstream. These microbial signals can reach the brain, especially if the blood-brain barrier is compromised, and further activate microglial inflammasomes, enhancing neuroinflammatory processes. This gut brain-inflammasome axis suggests that targeting the inflammasome pathway may offer novel therapeutic strategies. Approaches such as direct inhibition of NLRP3 components or restoration of gut microbiota balance through probiotics, dietary interventions, or fecal microbiota transplantation may help mitigate neuroinflammation and potentially slow the progression of neurodegenerative disorders [15].

Depression and Anxiety: Role of Dysbiosis and the Neuro Inflammatory Process

The gut microbiota is dominated by bacteria, with more than 1000 known species of bacteria, 90% of which are Firmicutes and Bacteroidetes. 30 Firmicutes include Clostridium, Lactobacillus, and Ruminococcus and other genera, members of this group ferment dietary fiber into short-chain fatty acids (SCFAs) such as butyrate, which serves as a primary energy source for the cells lining our colon and plays a pivotal role in regulating inflammation. 31 Bacteroidetes, particularly the genera Bacteroides and Prevotella, are adept at breaking down complex carbohydrates, thereby releasing essential nutrients that our bodies would otherwise find inaccessible [16]. Mechanisms of action

"include the inhibition of the growth of pathogenic microorganisms in the gastrointestinal tract (by fostering colonization resistance, improving intestinal transit, producing antimicrobial substances, or helping normalize a perturbed microbiota), production of bioactive metabolites (e.g., short-chain fatty acids), and the reduction of luminal pH in the colon. Species specific mechanisms can include vitamin synthesis, gut barrier reinforcement, bile salt metabolism, enzymatic activity, and toxin neutralization. Other mechanisms, such as cytokine production, specific immunomodulatory activities, and effects on the endocrine and nervous systems, are expressed in a strain-specific manner. Prebiotics are typically non-digestible fibers/complex carbohydrates selectively stimulating the activity of beneficial microorganisms e.g. garlic, bananas. Two important groups are galacto- and fructo-oligosaccharides. Their fermentation (breaking the ingredients down into simpler particles) by gut microbiota produces short-chain fatty acids. These lower the intestinal pH and suppress pathogen growth by supporting immune cell function [17, 18].

Epidemiological research has highlighted the protective effects of dietary modifications, including the intake of fish and omega-3 fatty acids, as well as interventions like probiotics, prebiotics, synbiotics, postbiotics, fecal microbiota transplantation, and 5-hydroxytryptophan regulation in alleviating symptoms of anxiety and depression. Both preclinical and clinical studies suggest that these therapies exert their antidepressant effects through multiple mechanisms, such as the upregulation of neuroactive substances, regulation of monoamine neurotransmitter levels, reduction of oxidative stress and inflammation, and modulation of the hypothalamic–pituitary–adrenal axis and the gut brain axis.

The current body of research, however, has notable limitations. The fundamental mechanisms by which the gut microbiome influences depression and anxiety, as well as the effectiveness of microbial restoration therapies, remain insufficiently understood. Moreover, there is a lack of comprehensive data on the safety and efficacy of interventions such as probiotics, prebiotics, symbiotic, postbiotics, and fecal microbiota transplantation. The optimal dosages for these treatments, as well as the potential role of adjunct therapies like antidepressant medications, remain undetermined.

A study examined the impact of gut microbiome diversity and the composition on depression scores in the Rotterdam Study by controlling the lifestyle factors and the medication use. The analyzed microbiome identified 13 taxa which was significantly associated with depressive symptoms. The studies Rotterdam and HELIUS finalized that the microbiome was predominantly composed of the phyla Firmicutes (77% in Rotterdam and 70% in HELIUS), Bacteroidetes (13% in Rotterdam and 21% in HELIUS), Actinobacteria (0.42% in both cohorts), and Proteobacteria (0.48% in Rotterdam and 0.22% in HELIUS) [19-24]. After reviewing 17 studies researchers observed that the three is increased levels of of Eggerthella, Atopobium, and Bifidobacterium, and a decrease in Faecalibacterium among MDD patients compared to controls.

A study also analyzed the fecal samples of the study participants who were previously clinical diagnosied with social anxiety disorder into plastic containers containing an Anaerogen sachet. The gut microbiota of patients with SAD differed from those of healthy controls in terms of overall composition as well as in relation to specific genus- and species-level differentially abundant features. No differences were found in functional diversity between the two groups. This study demonstrates, for the first time, that the gut microbiome is compositionally and functionally altered in people with social anxiety disorder (SAD) compared with healthy controls. It was found that Anaeromassilibacillus sp An250 to be present in almost half of our SAD group but in only one healthy control [25].

Schizophrenia and Dysbiosis

A study revealed the cognitive impairment connection between the gut microbiota and the individuals with schizophrenia. It was shown that there is a higher relative abundance of Actinomycetota, Bacteroidota, Euryarchaeota, Fusobacteria, and Pseudomonadota, and lower abundance of Bacillota, Tenericutes, and Verrucomicrobia compared to the control group ($P < 0.05$) [26]. Comparison of the gut microbiome of 25 individuals with chronic schizophrenia to 25 matched controls. The findings revealed significant differences in microbial composition, with increased Anaerococcus and decreased Haemophilus, Sutterella, and Clostridium genera in schizophrenia patients [27]. A similar change was also observed in the patients with bipolar disorders with increased Actinobacteria, particularly the Coriobacteriaceae and the Coriobacteria family, and reduced microbial diversity related to longer illness duration in BD patients [28].

Role of Probiotics, Prebiotics, and Diet in Managing Psychiatric Symptoms

Probiotics, prebiotics and diet proved to have an important role in managing psychiatric symptoms. The main issues discussed were anxiety and depression amongst other psychiatric disorders. Psychobiotics alone showed a signicant amelioration in anxiety by improving body composition, however when combined with a healthy diet, the effect increased. Although probiotic administration didn't show any significant effect on anxiety, depression or cortisol levels, it effectively alleviated sympotms associated with physical stress in students undergoing exams. Good results were also seen in pregnancy, as well as in post-partum, where they also lowered the odds ratio for the mentioned above conditions. Aditionally, the usage of these supplements impacted the amygdala, which is considered a major center of emotions A combination of complex food matrices along with diverse dietary fibers and probiotics stimulates the growth of gut microbes, leading to the production of neuroactive compounds and health-promoting metabolites [29, 30]. Another important role would have the short chain fatty acids that release appetite-regulating hormones by stimulating enteroendocrine L cells. Several small studies have analyzed the relation between autism spectrum disorder and dietary changes that involve gluten- and casein-free diets. They showed that 81% of the participants improved after 3 months (31). A study that included 14 epileptic and 30 healthy infants concluded a 50% decrease in seizure frequency after following a ketogenic diet, leading to both clinical and microbiota improvements [31]. Probiotics were helpful in controlling stress-related symptoms like nausea and vomiting, but no significant change in STAI anxiety ratings was seen between probiotic users and those using other stress-management methods. In elderly patients, the combination of a healthy

diet and probiotics/monoterpenes/argan oil reduced depressive symptoms [32]. When analyzing the studies, multiple beneficial effects were observed. However, there were limitations—due to the variability in strains of probiotics and prebiotics, it was difficult to determine the specific effect of each strain.

Fecal transplantation is an innovative treatment proposed for the treatment for mental health disorders [33]. A study also explains the effects of the fecal transplantation on the alcohol – induced anxiety reducing the symptoms if the intervention is started before the onset of the symptoms. On the other hand, the animal studies show that the transferring of the microbiota from the affected patients to healthy mice can induce anxiety- like symptoms supporting the role of the gut brain axis in the psychiatric illness [34].

Another study explained that the Fecal transplant has advantages over the traditional antidepressants which includes faster onset of action, benefits lasting over 6 months and fewer side effects and also better compliance since it's not administered daily. Despite its potential it still has notable limitations due to the lack of standard definition of the healthy microbiome. Difficulty in identifying the optimal donor profiles and also the small sample sizes and limited diversity in studies.

Impact of Psychotropic Medications on Gut Microbiota

In vitro screening studies have shown that approximately 25% of human-targeted drugs, including many psychotropic medications, exhibit antimicrobial effects on gut bacteria, altering microbiota composition without necessarily affecting human intestinal permeability [35]. Animal studies notably involving the SSRIs and SNRIs have demonstrated the increased ileal permeability in rats, alongside the microbiome alterations even while the intestinal permeability remains unaffected in humans. The use of antidepressants has been associated with elevated risk of Clostridioides difficile infection, presumably mediated by drug induced changes in gut microbial ecology [36]. Several tricyclic antidepressants (e.g., desipramine) show strong antimicrobial effects in vitro across multiple strains including Salmonella typhimurium, Bacillus spp., Vibrio cholerae, and MRSA-like organisms; SSRIs including fluoxetine and escitalopram inhibit growth of E. coli, E. faecalis, S. aureus, Lactobacillus rhamnosus, and Bifidobacterium bifidum, and can alter microbial carbohydrate metabolism, membrane transport and signaling pathways [37].

Psychotropic medications—including SSRIs, tricyclics, SGAs, and even non psychiatric agents like propranolol—demonstrate antimicrobial properties that alter gut microbiota composition and metabolism, sometimes without affecting intestinal barrier function in humans. Such drug induced dysbiosis may contribute to metabolic disturbances, increased infection risk (e.g. C. difficile), and possibly secondary drug microbiome resistance development.

However, most evidence remains preclinical or in vitro, and human clinical data are limited. Particularly, precise quantification of antidepressants at gut luminal concentrations, strain specific microbial effects, long-term consequences, and mitigation strategies (e.g. probiotics) require further controlled human trials.

Discussion

Recent advancements highlight the gut microbiome's central role in regulating neuroinflammation and behavioral changes across psychiatric conditions. The vagus nerve functions as a crucial communication pathway, sensing microbial metabolites such as SCFAs and neurotransmitters (3,4). These signals affect brain functions, including mood, cognition, and neurodegeneration. In PD, vagus nerve signaling is implicated in disease initiation (3), and gut dysbiosis promotes chronic inflammation, contributing to glial dysfunction and dopaminergic neuron death.

Microbial metabolites like SCFAs, tryptophan catabolites, and bile acids affect immune balance, neurotransmitter synthesis, and blood-brain barrier integrity (10,11). Immune responses involving microglia, T cells, and dendritic cells are modulated by microbial components such as LPS, activating TLRs and promoting cytokine release (12,13). These cytokines can traverse the BBB and alter synaptic plasticity, contributing to psychiatric disorders like depression, anxiety, and ASD.

In depression, dysbiosis alters SCFA production and reduces beneficial taxa such as Faecalibacterium, while increasing pro-inflammatory genera such as Eggerthella and Atopobium (19–24). Similarly, schizophrenia and bipolar disorder are associated with distinct microbial alterations, including increased Actinomycetota and decreased microbial diversity (26–28).

Therapeutically, psychobiotics, prebiotics, and dietary interventions demonstrate mood-regulating properties, especially when combined with healthy nutrition (29–32). Fecal microbiota transplantation shows promise for rapid symptom relief and long-term effects, yet challenges such as donor variability and standardization persist (33,34). Psychotropic medications also alter gut microbial ecosystems, potentially contributing to adverse metabolic or infectious outcomes (35–37).

Despite promising preclinical data, human clinical trials remain limited in scope, and strain-specific effects are poorly understood. Integrative approaches are required to harness microbiome-targeted therapies for psychiatric and neurological disorders.

Conclusion

The gut-brain axis plays a pivotal role in the pathogenesis and progression of psychiatric and neurodegenerative disorders through its regulation of immune, metabolic, and neuronal pathways. Alterations in gut microbiota composition and function influence neurotransmitter synthesis, immune cell activation, and brain inflammation, contributing to symptoms in disorders such as depression, anxiety, Parkinson's disease, and schizophrenia. Interventions targeting the microbiota—such as probiotics, psychobiotics, and fecal microbiota transplantation—hold therapeutic potential, but further rigorous, large-scale human studies are needed. The antimicrobial properties of psychotropic drugs may further complicate gut microbiota balance, warranting personalized strategies to mitigate adverse effects. Overall, a deeper understanding of the gut-brain-microbiota axis offers promising

avenues for diagnostics and therapeutics in mental health care.

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