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THE GUT-BRAIN AXIS: EXPLORING THE IMPACT OF GUT MICROBIOTA ON MENTAL HEALTH

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ABSTRACT

The microbiota-gut-brain axis represents a complex bidirectional communication network linking the intestinal microbiota with the central nervous system through neural, endocrine, immune, and metabolic pathways. Growing evidence suggests that alterations in gut microbiota composition may contribute to the pathophysiology of several mental disorders, including depression, anxiety disorders, bipolar disorder, and attention-deficit/hyperactivity disorder. This review aims to summarize current knowledge on the mechanisms by which gut microbiota may influence mental health and to discuss microbiota-targeted therapeutic approaches. Dysbiosis may affect brain function through vagus nerve signaling, hypothalamic-pituitary-adrenal axis activation, increased intestinal permeability, systemic inflammation, cytokine release, altered neurotransmitter metabolism, and reduced production of short-chain fatty acids. Studies indicate that individuals with psychiatric disorders often show reduced microbial diversity, depletion of beneficial anti-inflammatory and butyrate-producing bacteria, and enrichment of pro-inflammatory microbial taxa. These changes may promote neuroinflammation, oxidative stress, impaired stress regulation, and disturbances in serotonin, dopamine, gamma-aminobutyric acid, and tryptophan-related pathways. The review also examines the potential role of probiotics, prebiotics, synbiotics and fecal microbiota transplantation in modulating the gut microbiome and improving psychiatric symptoms. Although some clinical and experimental findings suggest beneficial effects, especially in depression and anxiety, the available evidence remains heterogeneous. Further well-designed, long-term clinical studies, are required to clarify causal mechanisms, identify effective microbial interventions, and determine their place in mental health prevention and treatment.

KEYWORDS

Gut-Microbiota, Microbiota-Gut-Brain-Axis, Mental Health, Dysbiosis, Probiotics, Fecal Microbiota Transplantation

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Introduction

The gut microbiota, a complex community of microorganisms inhabiting the gastrointestinal tract, influences many physiological processes in the human body, including immune system function, metabolism, and brain activity. The gut-brain axis (GBA) is a bidirectional communication network connecting the gut microbiota with the central nervous system through neural, hormonal, immune, and metabolic pathways [1]. Key mechanisms involved in this communication include vagus nerve signaling, hypothalamic-pituitary-adrenal (HPA) axis regulation, microbiota-derived metabolites, neurotransmitter production, and inflammatory responses. Recent studies in both humans and animals suggest that gut microbiota plays an important role in the development and modulation of mental disorders, particularly depression, anxiety disorders, bipolar disorder and attention-deficit/hyperactivity disorder [2]. Certain strains of *Lactobacillus* and *Bifidobacterium* may influence stress responses, depressive symptoms, and the perception of stressful situations, whereas dysbiosis, defined as an imbalance in microbial diversity and composition, may promote inflammation, impair neurogenesis, and increase susceptibility to psychiatric disorders [3]. The development of the human gut microbiota begins early in life and may be affected by factors such as mode of delivery, antibiotic therapy, diet, and illness [4]. Therefore understanding the relationship between gut microbiota and mental health may help identify new supportive strategies, including probiotics, prebiotics, and fecal microbiota transplantation.

Methodology

The basis of this article is a literature review of the last 10 years, conducted using the PubMed and Google Scholar databases with the following keywords: “gut microbiota”, “gut-brain axis”, “mental health”, “dysbiosis”, “depression”, “anxiety disorders”, “bipolar disorder”, “ADHD”, “probiotics”, “prebiotics” and “fecal microbiota transplantation”. The analysis included systematic reviews, meta-analyses, observational studies, clinical studies, experimental animal studies and expert reviews. The selected papers were evaluated in the context of the role of gut microbiota in the development and modulation of mental disorders, with particular attention to mechanisms involving the vagus nerve, hypothalamic-pituitary-adrenal axis, immune and cytokine signaling, neurotransmitter metabolism and short-chain fatty acids. The review also considered the potential therapeutic opportunities and future challenges associated with microbiota-targeted interventions, including probiotics, prebiotics, synbiotics and fecal microbiota transplantation, in supporting mental health prevention and treatment.

Results

Mechanistic Pathways Linking Gut Microbiota with Mental Health

The relationship between gut microbiota and mental health is mediated by several interconnected biological pathways that form the microbiota-gut-brain axis. These mechanisms include neural signaling through the vagus nerve, neuroendocrine regulation via the hypothalamic-pituitary-adrenal axis, immune and cytokine-mediated responses, microbial influence on neurotransmitter metabolism, and the production of short-chain fatty acids. Together, these pathways may explain how alterations in gut microbiota composition can contribute to changes in brain function, stress reactivity, inflammation and development or progression of psychiatric symptoms.

Neural Signaling Through the Vagus Nerve

The gut-brain axis enables bidirectional communication between the gastrointestinal tract and the central nervous system through neural, endocrine, immune and metabolic pathways [5]. Microbiota-derived metabolites can enter systemic circulation and reach extraintestinal organs, which means that microbial signaling may influence brain function not only locally in the gut but also at a systemic level [6]. The gut microbiome is considered a key regulator of host health because microbial molecules can affect metabolic and inflammatory pathways that also interact with distant organs, including the brain [7]. The gut microbiota has an extensive metabolic repertoire that complements human enzymes and shapes and shapes the biochemical profile of diet-derived compounds, creating a biological basis for gut-to-brain metabolic signaling [8].

Hypothalamus-Pituitary-Adrenal Axis (Stress Hormones)

Gut microbiota and its metabolites participate in neuroendocrine signaling mediated by the hypothalamic-pituitary-adrenal axis, which is essential for regulating the stress response [5]. Microbially produced short-chain fatty acids, especially acetate, propionate and butyrate, may influence central regulatory mechanisms through hypothalamic neuronal activation and changes in neuropeptide expression [8]. Alterations in gut microbial composition have been linked with changes in gut-to-brain signaling pathways, including mechanisms involving hypothalamic neurons and host bioactive lipids [7]. Because gut microbiota regulates both innate and adaptive immunity, dysbiosis may create a pro-inflammatory physiological environment that can contribute to stress-related mental health disturbances [9].

Immune/Cytokines

Dynamic interactions between gut microbiota and the host immune system are necessary for intestinal homeostasis and for limiting excessive inflammatory responses [10]. The gut microbiota-immune axis is important because innate immune cells release cytokines and chemokines that can shape both local intestinal responses and broader systemic inflammation [9]. Microbiota-derived metabolites such as short-chain fatty acids (SCFAs), secondary bile acids, and tryptophan catabolites support regulatory immune responses and help maintain epithelial barrier integrity [11]. When intestinal barrier function is impaired, bacteria, endotoxins, and inflammatory mediators may translocate into circulation, contributing to systemic inflammation that can affect brain-related processes [12].

Neurotransmitters and Metabolites

Gut-microbiota-derived metabolites act as a key effector molecules because they can be produced from dietary precursors, enter the bloodstream, and influence host physiological responses [6]. Tryptophan-derived microbial metabolites can regulate immune responses through aryl hydrocarbon receptor signaling, linking microbial metabolism with inflammation-sensitive pathways relevant to mental health [13]. Short-chain fatty acids can influence enteroendocrine cell activity and promote the production of gut hormones such as glucagon-like-peptide-1, peptide YY and serotonin, which are involved in gut-brain communication [14]. Microbial metabolites produced near the intestinal barrier can regulate host responses, including immune activity, epithelial permeability, and processes connected with neurotransmission [12].

Short-Chain Fatty Acids (SCFAs)

SCFAs are produced mainly by bacterial fermentation of dietary fibers and include acetate, propionate, and butyrate [14]. SCFAs reduce local inflammation and maintain intestinal barrier integrity by binding to G-protein-coupled receptors and by influencing gene expression through histone and deacetylase inhibition [10]. Butyrate and other SCFAs support immune homeostasis by strengthening epithelial barrier function and promoting regulatory immune responses [11]. Dietary fiber can increase the production of microbial metabolites such as SCFAs, which then interact with epithelial and mucosal immune cells to regulate inflammation [13].

Depression and Gut Microbiome

The gut–brain axis is mediated by microbiota-derived molecules such as short-chain fatty acids, gamma-aminobutyric acid (GABA), and tryptophan metabolites. Dysbiosis has been associated with disruptions in gut–brain signaling pathways, potentially contributing to neuroinflammation, increased blood–brain barrier permeability, activation of the hypothalamic–pituitary–adrenal (HPA) axis, and elevated inflammatory markers, all of which may exacerbate depressive symptoms [15]. Excessive activation of the HPA axis may promote the production of reactive oxygen and nitrogen species, leading to oxidative stress, damage to cellular macromolecules (including proteins, lipids, and DNA), and impairment of antioxidant defense systems. Additionally, increased translocation of bacterial lipopolysaccharide (LPS) derived from Gram-negative bacteria may activate the inflammatory response system (IRS), resulting in elevated levels of pro-inflammatory cytokines [16]. In individuals with chronic depression, increased levels of immunoglobulins IgA and IgM directed against LPS have also been reported [17]. Furthermore, the gut microbiome of patients with depression appears to be characterized by an imbalance favoring pro-inflammatory bacteria over anti-inflammatory species. In particular, patients with major depressive disorder (MDD) have been shown to exhibit an increased relative abundance of bacteria from the phyla Bacteroidetes and Proteobacteria, along with a decreased abundance of Firmicutes [18]. Therefore, modulation of the gut microbiota and improvement of microbiota–gut–brain axis function may represent a promising complementary strategy in the prevention and treatment of depression [19].

Anxiety Disorders and Gut Microbiota Dysbiosis

Studies indicate that anxiety disorders are associated with major alterations in gut microbiota composition, including reduced microbial abundance and diversity. Patients with anxiety and generalized anxiety disorder (GAD) were shown to have lower levels of Firmicutes and SCFA-producing bacteria, alongside increased amounts of Bacteroidetes and Fusobacteria [20]. Anxiety-related gut dysbiosis has also been linked to reduced abundance of anti-inflammatory bacteria, including *Faecalibacterium* and *Lachnospira*, together with increased levels of pro-inflammatory microbial taxa [21]. Moreover, certain bacterial groups such as *Blautia* and *Butyricicoccus* appear to exert protective effects against anxiety disorders, whereas *Eubacterium brachy* and *Coprococcus* were positively associated with anxiety severity and risk [22]. Similar observations were reported in individuals with social anxiety disorder (SAD), who exhibited distinct gut microbiome profiles compared with healthy controls. Fecal microbiota transplantation from SAD patients to mice induced increased social fear behaviors and changes in oxytocin-related signaling, while microbiome-based interventions, including probiotics and other psychobiotic approaches, have demonstrated potential in reducing anxiety symptoms by modulating inflammatory, immune, and neuroendocrine pathways [23].

Bipolar Disorder and Gut Microbiota Dysbiosis

Bipolar disorder (BD) is a chronic psychiatric disorder characterized by alternating episodes of depression and mania that worsen daily functioning and quality of life. Increasing evidence suggests that genetic predisposition, neurotransmitter imbalance, and inflammatory processes, including elevated levels of IL-6, TNF- α and C-reactive protein (CRP), may contribute to the pathophysiology and progression of bipolar disorder [24]. Studies investigating BD have also shown considerable alterations in gut microbiota composition, including reduced microbial heterogeneity and reduced abundance of beneficial butyrate-producing bacteria such as *Faecalibacterium prausnitzii*. Altered levels of this bacterium were associated with poorer self-reported health and greater illness intensity [25, 26]. Enhanced levels of pro-inflammatory gut microbial populations particularly *Eggerthella*, observed in patients with BD were associated with gastrointestinal and systemic inflammatory processes. Increased presence of these microorganisms may contribute to neuroinflammation and gut-brain axis dysregulation through a reduction of butyrate-producing bacteria and increased production of pro-inflammatory cytokines [27]. Disruptions within the microbiota-gut-brain axis in BD may also impact hypothalamic-pituitary-adrenal (HPA) axis activity, intestinal barrier dysfunction and neurotransmitter regulation leading to abnormal stress responses and increased inflammatory activity. Microbiota-targeted interventions, including probiotics and dietary modulation, may therefore represent promising supportive strategies for improving psychiatric symptoms and clinical outcomes in patients with BD [28]. Probiotic supplementation involving *Lactobacillus* spp. and *Bifidobacterium lactis* was associated with reduced psychiatric rehospitalization rates, improvement of gastrointestinal symptoms, and alleviation of manic symptoms among patients with BD [29].

Attention-deficit/hyperactivity disorder and Gut Microbiota Dysbiosis

Attention-deficit/hyperactivity disorder (ADHD) is a common neurodevelopmental disorder characterized by persistent patterns of inattention, hyperactivity, and impulsivity that significantly impair daily functioning and quality of life [30]. Increasing evidence suggests that disturbances within the microbiota-gut-brain axis may contribute to ADHD pathophysiology through immune dysregulation and altered neuroinflammatory signaling. Children with ADHD exhibited increased levels of bacterial genera such as *Agathobacter*, *Anaerostipes*, and *Lachnospiraceae* UCG-010, while enrichment of nervous system and immune-related microbial pathways was also observed. Lower plasma levels of TNF- α were additionally associated with greater symptom severity and reduced gut microbiota diversity, suggesting a potential interaction between gut dysbiosis, inflammatory imbalance, and ADHD progression [31]. Higher *Bifidobacterium* levels were associated with increased activity of microbial pathways involved in phenylalanine synthesis, an important precursor of dopamine. These findings suggest that altered bacterial metabolism may influence dopaminergic neurotransmission and reward-processing mechanisms potentially involved in attentional deficits and impulsive behaviors in ADHD [32]. Reduced abundance of anti-inflammatory *Faecalibacterium* were further linked to excessive production of pro-inflammatory cytokines and neuroinflammatory processes potentially associated with cognitive dysfunction and behavioral abnormalities related to hyperactivity [33]. Differences in short-chain fatty acid-producing microbiota were also observed, including lower levels of the *Ruminococcaceae* family together with elevated presence of *Bacteroides uniformis* and *Bacteroides ovatus*, suggesting potential disturbances in microbial metabolic activity [34]. Dietary patterns were also closely linked to variations in gut microbiota composition among children with ADHD, particularly regarding fat and fiber intake. Higher fat consumption correlated with microbial profiles involved in behavioral abnormalities, whereas increased dietary fiber intake was connected to a more beneficial bacterial profile and potentially protective microbiota-gut-brain axis effects [35].

Interventions

Influence of Probiotics and Prebiotics on the Gut Microbiome and Mental Health

Probiotics and prebiotics are considered potential microbiota-targeted interventions because the gut microbiome can communicate with the central nervous system through gut-brain axis. Probiotics are live microorganisms which, when administered in adequate amounts, may provide health benefits to the host [36]. Prebiotics are non-digestible food components that can support host health by modulating the composition and activity of the gut microbiota [37]. Together with synbiotics and postbiotics, probiotics and prebiotics are described as potential psychobiotics because they may influence microbiota-brain interactions and mental health outcomes. The gut-brain axis involves neural pathways such as vagus nerve, endocrine pathways such as cortisol and the HPA axis, and immune pathways involving cytokines. Through these mechanisms, modulation of the gut microbiome may influence stress reactivity, neuroinflammation, neurotransmitter signaling and mood regulation. Probiotics are thought to contribute to a more balanced gut environment by suppressing pathogens and interacting with the host microbiota. Common probiotic preparations used in studies often include *Lactobacillus* and *Bifidobacterium* species, although the effects may depend on the exact strain, dose, and duration of supplementation [36]. Probiotics may also influence gut barrier function and immune activity, which is relevant because inflammation and increased cytokine activity are discussed as possible mechanisms in major depressive disorder. Some probiotic strains may affect neurotransmitter-related pathways, including serotonin, dopamine, norepinephrine, and GABA signaling, which could partly explain their possible antidepressant effects [38]. Prebiotics may support the growth of beneficial bacteria and increase microbial metabolites such as short-chain fatty acids, which are involved in immune regulation and gut-brain communication [5]. Prebiotics such as inulin, fructo-oligosaccharides, and galacto-oligosaccharides can selectively increase populations of beneficial bacteria, especially *Bifidobacterium* and *Lactobacillus*. Both probiotics and prebiotics may increase SCFA levels and SCFAs can benefit host immunity through mechanisms such as inhibition of pro-inflammatory NF- κ B pathways and induction of regulatory T cells [9]. Clinical evidence suggests that probiotics may reduce depressive and anxiety symptoms, but the results are heterogeneous and should be interpreted cautiously. In a meta-analysis of randomized controlled trials in clinically diagnosed samples, probiotics significantly reduced depressive symptoms and moderately reduced anxiety symptoms compared with control conditions. In the same analysis prebiotics did not show a statistically significant effect on depressive symptoms, although a non-significant trend toward improvement was observed [37]. Another systematic review and meta-analysis found that probiotics slightly improved depression and psychiatric distress at the quantitative level, but most probiotics did not clearly affect mood, stress, anxiety, or depression in qualitative comparisons with placebo [39]. Evidence from patients with major depressive disorder remains limited because probiotic supplementation showed only modest effects on depressive symptoms and did not consistently change gut microbiota diversity or composition after four to eight weeks. This suggests that short-term probiotic use may not be sufficient to produce stable microbiome changes, and longer interventions may be needed to detect clinically meaningful effects.

Fecal Microbiota Transplantation

Fecal Microbiota Transplantation (FMT) may improve depressive-like behavior by restoring gut microbiota balance, protecting the intestinal barrier, and reducing intestinal and neuroinflammatory changes, as shown in a stress-induced animal model [40]. Clinical evidence from randomized trials suggests that FMT can significantly reduce depressive symptoms, particularly in patients with disorders of gut-brain interaction such as irritable bowel syndrome, although the strength of the effect may depend on the route of administration and follow-up duration [41]. However, another meta-analysis of randomized controlled trials found insufficient evidence for a significant overall antidepressant effect of FMT and emphasized that changes in gut microbiota composition do not always clearly correspond with improvement in depressive symptoms [42].

Discussion

The findings of this review support the concept that the microbiota-gut-brain axis is a multidirectional system rather than a single pathway, involving neural, endocrine, immune, and metabolic communication between the gastrointestinal tract and the central nervous system [1,5]. Microbiota-derived metabolites may enter systemic circulation and influence distant organs, which strengthens the biological plausibility that intestinal microorganisms can affect brain-related processes beyond local gastrointestinal effects [6,7]. In this context, short-chain fatty acids appear especially important because they are produced through bacterial fermentation of dietary fibers and contribute to intestinal barrier integrity, immune regulation, and gut hormone signaling [11,13,14]. The interaction between gut microbiota and the hypothalamic-pituitary-adrenal axis suggests a mechanism whereby dysbiosis may contribute to maladaptive stress responses and stress-related psychiatric symptoms [5,7,9]. Similarly, impaired intestinal barrier function may facilitate the translocation of bacterial products and inflammatory mediators, promoting systemic inflammation that can influence neuroimmune pathways involved in mental health [10,11,12].

In depression, reported alterations include disruption of gut-brain signaling, oxidative stress, inflammatory activation, and an imbalance favoring pro-inflammatory microbial taxa over anti-inflammatory species [15,16,18]. Increased immunoglobulin responses against lipopolysaccharide in chronic depression further support the hypothesis that bacterial translocation and immune activation may participate in depressive pathophysiology [17]. However, because much of the available evidence includes observational studies and review data, these findings should be interpreted mainly as associations rather than definitive proof that dysbiosis directly causes depressive disorders [15,18,19]. In anxiety disorders, reduced microbial diversity and decreased abundance of short-chain fatty acid-producing or anti-inflammatory bacteria indicate that microbiota composition may be related to inflammatory and metabolite pathways involved in anxiety symptoms [20,21,22]. The observation that fecal microbiota transplantation from patients with social anxiety disorder increased social fear behavior in mice provides relevant experimental support for a possible causal contribution of microbiota to anxiety-like behavior, although translation from animal models to human clinical practice remains limited [23]. The evidence reviewed for bipolar disorder suggests that gut microbiota alterations may overlap with immune dysregulation and systemic inflammation, which are already considered relevant to the pathophysiology and course of this condition [24, 27,28]. Decreased microbial heterogeneity and changes in butyrate-producing bacteria such as *Faecalibacterium prausnitzii* may be clinically important because butyrate-related pathways support intestinal barrier function and immune homeostasis [11,25,26]. Probiotic supplementation in bipolar disorder appears promising as a supportive approach, but current findings are not sufficient to define standardized strains, doses, or treatment duration [28,29, 36]. In attention-deficit/hyperactivity disorder, associations between microbiota composition, cytokine levels, phenylalanine metabolism, and short-chain fatty acid producing bacteria suggest that microbial mechanisms may contribute to neuroinflammatory and dopaminergic pathways relevant to attention and impulsivity [31,32,33,34]. Dietary factors may also be important in attention-deficit/hyperactivity disorder because fiber intake was associated with more favorable microbial profiles, whereas higher fat intake correlated with profiles linked to behavioral abnormalities [35].

Microbiota-targeted interventions represent an important but still developing field in mental health care [36,37]. Probiotics and prebiotics may influence mental health through modulation of gut barrier function, immune signaling, neurotransmitter-related pathways, and short-chain fatty acid production [5,9,36,38]. Nevertheless, clinical evidence remains inconsistent, as meta-analyses have reported modest or heterogeneous effects of probiotics on depression, anxiety, mood, stress, and psychiatric distress [37,39]. The evidence for fecal microbiota transplantation is also inconclusive, since one meta-analysis suggested reduction in depressive symptoms, whereas another found insufficient evidence for a significant overall antidepressant effect [41,42]. These inconsistencies may reflect differences in patient populations, diagnostic criteria, intervention protocols, route of administration, donor microbiota composition and follow-up duration [37,39, 41,42]. Future research should prioritize well-designed longitudinal and randomized clinical trials to clarify whether microbiota changes are a cause, consequence, or modifier of psychiatric disorders and to determine which microbiota-based interventions can be safely integrated into prevention and treatment strategies [15,21,28,37,42].

Conclusions

The available evidence indicates that gut microbiota may play an important role in mental health through the microbiota-gut-brain axis, which involves neural, endocrine, immune, and metabolic pathways. Dysbiosis may contribute to psychiatric symptoms by promoting inflammation, impairing intestinal barrier integrity, altering hypothalamic-pituitary-adrenal axis activity, disturbing neurotransmitter metabolism, and reducing the production of beneficial short-chain fatty acids. Changes in gut microbiota composition have been observed in depression, anxiety disorders, bipolar disorder, and attention-deficit/hyperactivity disorder. Suggesting the microbial imbalance may be associated with both emotional and neurodevelopmental disturbances. Microbiota-targeted interventions, including probiotics, prebiotics, synbiotics, dietary modulation, and fecal microbiota transplantation, may offer promising supportive strategies for mental health prevention and treatment. However, current findings remain heterogeneous, and the causal relationship between gut microbiota alterations and psychiatric disorders has not yet been fully established. Future research should focus on large, well-designed, long-term clinical studies to identify specific microbial signatures, clarify underlying mechanisms, determine optimal probiotic strains and doses, and assess the long-term safety and efficacy of microbiota-based therapies. Further studies are also needed to evaluate whether personalized microbiome modulation could become an effective component of psychiatric care.

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