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## The Role of Gut Microbiota in the Pathophysiology of Depression: A Narrative Review

Karolina Wojciechowska<sup>1</sup> [KW], ORCID <https://orcid.org/0009-0001-7048-1335>

E-mail: [karolina8wojciechowska@gmail.com](mailto:karolina8wojciechowska@gmail.com)

Izabela Zajkowska<sup>1</sup> [IZ], ORCID <https://orcid.org/0009-0002-8526-7339>

E-mail: [zajkowska.izabela@wp.pl](mailto:zajkowska.izabela@wp.pl)

Patrycja Zabrocka<sup>2</sup> [PZ], ORCID <https://orcid.org/0009-0001-5834-5277>

E-mail: [39916@student.umb.edu.pl](mailto:39916@student.umb.edu.pl)

Julia Martowska<sup>1</sup> [JM], ORCID <https://orcid.org/0009-0006-2804-5368>

E-mail: [julia.wiaterek@gmail.com](mailto:julia.wiaterek@gmail.com)

Ewelina Choroszewska<sup>1</sup> [EC], ORCID <https://orcid.org/0009-0000-7609-7265>

E-mail: [choroszewska2000@gmail.com](mailto:choroszewska2000@gmail.com)

Julia Baran<sup>2</sup> [JB], ORCID <https://orcid.org/0009-0005-9569-3149>

E-mail: [barjul99@gmail.com](mailto:barjul99@gmail.com)

Wiktor Warych<sup>1</sup> [WW], ORCID <https://orcid.org/0009-0003-2569-6833>

E-mail: [w.warych26@gmail.com](mailto:w.warych26@gmail.com)

<sup>1</sup> Śniadeckiego Voivodeship Hospital in Białystok, ul. M. C. Skłodowskiej 26, 15-278

Białystok, Poland

<sup>2</sup> University Clinical Hospital in Białystok, ul. M. C. Skłodowskiej 24a, 15-276 Białystok, Poland

**Corresponding Author:** Karolina Wojciechowska, [karolina8wojciechowska@gmail.com](mailto:karolina8wojciechowska@gmail.com)

**Abstract:** Depression is a complex psychiatric disorder with incompletely understood pathophysiology. Increasing evidence implicates gut microbiota dysbiosis in depression through the microbiota-gut-brain axis. This narrative review summarizes experimental and clinical studies published between 2013 and 2025 examining associations between gut microbiota and depressive disorders. Findings indicate that depression is linked to altered microbial diversity, immune activation, intestinal barrier dysfunction, dysregulation of the hypothalamic-pituitary-adrenal axis, and disturbances in tryptophan metabolism. While preclinical studies suggest a potential causal role of gut microbiota in depressive-like behavior, human evidence remains largely associative. Modulation of gut microbiota represents a promising but still exploratory direction for future depression research and therapy.

**Background:** Depression is a leading cause of disability worldwide and remains a major public health challenge due to its complex and multifactorial pathophysiology. Increasing evidence suggests that alterations in the gut microbiota may contribute to the development and progression of depressive disorders through the microbiota-gut-brain axis.

**Aim:** The aim of this review was to summarize and critically evaluate current evidence regarding the association between gut microbiota and depression, with particular emphasis on biological mechanisms and potential clinical implications.

**Material and Methods:** A narrative review of the literature was conducted based on experimental, clinical, and translational studies investigating the relationship between gut microbiota and depressive disorders. Peer-reviewed articles published between 2013 and 2025 were analysed, including animal studies, human observational studies, and systematic reviews.

**Results:** Patients with depression frequently exhibit alterations in gut microbiota composition, including reduced microbial diversity and changes in the abundance of taxa involved in short-chain fatty acid production, immune regulation, and neurotransmitter metabolism. Experimental studies demonstrate that microbiota manipulation, including faecal microbiota transplantation, can induce or alleviate depression-like behaviours. Proposed mechanisms include immune activation, increased intestinal permeability, dysregulation of the hypothalamic-pituitary-adrenal axis, and altered tryptophan metabolism.

**Conclusions:** Current evidence supports a significant association between gut microbiota and depression; however, causality has not been definitively established. The gut microbiota

represents a promising target for novel diagnostic and therapeutic strategies, although further well-designed longitudinal and interventional studies are required.

**Key words:** Depression; Gut microbiota; Microbiota-gut-brain axis; Dysbiosis.

## 1. Introduction

Depression is a heterogeneous psychiatric disorder characterized by persistent low mood, anhedonia, cognitive impairment, and a range of somatic symptoms. Despite advances in pharmacological and psychotherapeutic approaches, a significant proportion of patients fail to achieve full remission, highlighting the need for a more comprehensive understanding of its underlying biological mechanisms. Traditional hypotheses of depression have focused on monoaminergic dysfunction, hypothalamic-pituitary-adrenal (HPA) axis dysregulation, neuroinflammation, and genetic vulnerability. However, none of these models alone sufficiently explain the complexity and variability of depressive disorders.

In recent years, increasing attention has been directed toward the role of the gut microbiota in mental health. The human gastrointestinal tract harbours trillions of microorganisms that collectively influence host metabolism, immune function, and neurodevelopment. Through the microbiota-gut-brain axis, intestinal microorganisms communicate bidirectionally with the central nervous system via neural pathways, immune signalling, endocrine mechanisms, and microbial metabolites. Disruptions in this system, commonly referred to as dysbiosis, have been implicated in a variety of neuropsychiatric conditions, including major depressive disorder.

Clinical studies have demonstrated that individuals with depression often present with altered gut microbiota composition compared with healthy controls, although results regarding specific taxa vary considerably. Importantly, experimental evidence from animal models has provided support for a potential causal role of the microbiota in depressive behaviour. Germ-free animals and those receiving faecal microbiota transplantation from depressed patients exhibit behavioural, neuroendocrine, and immunological changes consistent with depression-like

phenotypes. These findings suggest that the gut microbiota may not merely be a consequence of depressive disorders but an active participant in their pathophysiology.

Understanding the interaction between gut microbiota and depression may provide novel insights into disease mechanisms and open new avenues for personalized diagnostics and therapeutics. Therefore, a comprehensive synthesis of current evidence is warranted to clarify the biological relevance and clinical implications of microbiota alterations in depression.

## **2. Research materials and methods**

This review was conducted as a narrative synthesis of experimental and clinical studies examining the relationship between gut microbiota and depression. The literature analyzed in this review consisted of peer-reviewed original research articles, systematic reviews, and meta-analyses published between 2013 and 2025.

The included studies encompassed a wide range of methodologies, including animal models of depression, human observational and case-control studies, fecal microbiota transplantation experiments, and investigations of microbiota-targeted interventions. Both preclinical and clinical studies were considered to provide a translational perspective on the microbiota-gut-brain axis in depression.

Articles were selected based on their relevance to the topic, scientific rigor, and contribution to understanding the mechanistic links between gut microbiota and depressive disorders. Attention was given to studies addressing immune activation, intestinal barrier function, neuroendocrine regulation, neurotransmitter metabolism, and microbial-derived metabolites such as short-chain fatty acids. Only full-text articles published in English were included.

Given the heterogeneity of study designs, populations, and analytical techniques, a quantitative meta-analysis was not performed. Instead, findings were synthesized qualitatively to identify consistent patterns, proposed mechanisms, and existing gaps in the current body of evidence.

## **3. Research results**

### **3.1. Alterations in Gut Microbiota Composition in Depression**

Numerous clinical studies have demonstrated that individuals with depression exhibit alterations in gut microbiota composition compared with healthy controls. Although the specific taxa associated with depression vary across studies, a general pattern of dysbiosis has been consistently observed. This dysbiosis is often characterized by reduced microbial diversity

and altered relative abundance of bacterial groups involved in metabolic and immunomodulatory processes.

Several studies have reported a decreased abundance of short-chain fatty acid (SCFA)-producing bacteria, including members of the genera *Faecalibacterium*, *Coprococcus*, and *Roseburia*, in patients with depressive disorders. SCFAs, particularly butyrate, play a critical role in maintaining intestinal barrier integrity and regulating inflammatory responses. A reduction in these bacteria may therefore contribute to increased intestinal permeability and systemic inflammation, both of which have been implicated in the pathophysiology of depression.

Conversely, an increased abundance of potentially pro-inflammatory taxa, such as *Alistipes* and certain members of the family *Enterobacteriaceae*, has been observed in some cohorts of depressed patients. However, findings at the taxonomic level remain inconsistent across studies, likely due to differences in study populations, dietary habits, medication use, sequencing methodologies, and geographic factors. These inconsistencies have led to the suggestion that functional characteristics of the microbiota may be more relevant to depression than specific bacterial taxa.

### **3.2. Evidence from Animal Models and Fecal Microbiota Transplantation**

Preclinical studies provide stronger support for a causal relationship between gut microbiota and depression-like behaviour. Germ-free animals display altered stress responses, changes in neurotransmitter systems, and abnormal behaviour, underscoring the importance of microbial colonization for normal brain development and function.

Faecal microbiota transplantation (FMT) experiments have been particularly informative. Transfer of gut microbiota from patients with depression into germ-free or antibiotic-treated rodents induces behavioural phenotypes resembling depression, including anhedonia, increased anxiety-like behaviour, and impaired stress coping. These behavioural changes are accompanied by neuroendocrine alterations, such as hyperactivation of the hypothalamic-pituitary-adrenal axis, and increased expression of pro-inflammatory cytokines in both peripheral tissues and the central nervous system.

Importantly, microbiota manipulation in animal models has also demonstrated therapeutic potential. Administration of specific probiotic strains or restoration of microbial diversity has been shown to reverse depression-like behaviours, normalize stress hormone levels, and reduce neuroinflammation. These findings suggest that gut microbiota is not only associated with depressive phenotypes but may actively modulate emotional behaviour.

### **3.3. Neuroimmune and Neuroendocrine Mechanisms**

One of the most widely studied mechanisms linking gut microbiota to depression involves immune system activation. Dysbiosis can disrupt the integrity of the intestinal barrier, leading to increased permeability and translocation of microbial components such as lipopolysaccharides into systemic circulation. This process may trigger chronic low-grade inflammation, which is a well-recognized feature of depression in a subset of patients.

Elevated levels of pro-inflammatory cytokines, including interleukin-6 and tumour necrosis factor- $\alpha$ , have been reported in individuals with depression and are associated with symptom severity and treatment resistance. These cytokines can influence brain function directly by crossing the blood-brain barrier or indirectly by activating neural and humoral signalling pathways. Chronic inflammation may also impair neurogenesis and synaptic plasticity, contributing to the development and persistence of depressive symptoms.

In addition to immune mechanisms, gut microbiota plays a crucial role in regulating the hypothalamic-pituitary-adrenal axis. Dysregulated microbial signalling has been shown to enhance stress-induced cortisol release, thereby exacerbating stress sensitivity and emotional dysregulation. Animal studies indicate that normalization of gut microbiota can attenuate exaggerated HPA axis responses to stress.

### **3.4. Microbial Metabolites and Neurotransmitter Pathways**

Gut microbiota contributes to the synthesis and metabolism of several neuroactive compounds, including serotonin, gamma-aminobutyric acid, dopamine, and their precursors. Approximately 90% of the body's serotonin is produced in the gastrointestinal tract, and its availability is influenced by microbial regulation of tryptophan metabolism.

Alterations in the kynurenine pathway of tryptophan metabolism have been linked to depression, with increased diversion of tryptophan toward neurotoxic metabolites under inflammatory conditions. Dysbiosis may therefore reduce serotonin availability while simultaneously promoting neuroinflammation through kynurenine pathway activation.

Short-chain fatty acids represent another important class of microbial metabolites with neuromodulator properties. SCFAs can influence microglial activation, blood-brain barrier integrity, and gene expression related to synaptic plasticity. Reduced SCFA production observed in depressed individuals may thus contribute to both peripheral and central mechanisms of depression.

### 3.5. Microbiota-Targeted Interventions

Emerging evidence suggests that modulation of gut microbiota may have therapeutic potential in depression. Probiotic and prebiotic interventions, often referred to as psychobiotics, have been shown to improve depressive symptoms in some clinical trials, although results remain variable. Dietary interventions aimed at increasing fiber intake and promoting microbial diversity have also been associated with improved mental health outcomes.

In addition to nutritional approaches, interactions between antidepressant medications and gut microbiota have gained increasing attention. Certain antidepressants can alter microbial composition, while gut bacteria may influence drug metabolism and bioavailability. This bidirectional interaction highlights the importance of considering gut microbiota in personalized treatment strategies for depression.

## 4. Discussion

The findings summarized in this review indicate a significant association between gut microbiota dysbiosis and depression; however, the nature of this relationship remains complex and multifaceted. The main biological mechanisms linking gut microbiota dysbiosis to depression and the corresponding levels of evidence are summarized in Table 1.

Mechanism	Microbiota-related changes	Key biological effects	Type of evidence	Clinical relevance
Altered microbiota composition	Reduced microbial diversity; decreased SCFA-producing taxa (e.g., <i>Faecalibacterium</i> , <i>Coprococcus</i> spp.); increased pro-inflammatory taxa	Impaired intestinal barrier function; reduced anti-inflammatory signaling	Human observational studies; animal models	Potential biomarkers; dietary and probiotic targets
Increased intestinal permeability and inflammation	Dysbiosis-associated disruption of gut barrier	Translocation of microbial components; chronic low-grade systemic inflammation	Human clinical studies; animal models	Relevance for inflammation-associated and treatment-resistant depression

Immune system activation	Altered microbial signaling	Elevated pro-inflammatory cytokines (e.g., IL-6, TNF- $\alpha$ ); neuroinflammation	Human and preclinical studies	Target for anti-inflammatory and microbiota-modulating therapies
HPA axis dysregulation	Impaired microbiota-stress signaling	Exaggerated cortisol response; animal increased stress sensitivity	Mainly animal studies	Stress-related vulnerability to depression
Altered tryptophan metabolism	Dysbiosis affecting serotonin and kynureneine pathways	Reduced serotonin availability; increased neurotoxic metabolites	Animal and human studies	Mechanistic link to mood regulation and antidepressant response
Reduced microbial metabolites	Decreased SCFA and indole derivative production	Impaired microglial regulation; altered synaptic plasticity	Preclinical and translational studies	Emerging targets for psychobiotic interventions
Effects of microbiota modulation	Probiotics, prebiotics, dietary interventions, FMT	Partial normalization of behavior, inflammation, and stress responses	Mostly animal studies; clinical trials	Experimental therapeutic strategies

While preclinical studies provide compelling evidence for a causal role of the gut microbiota in modulating depressive-like behaviour, human studies are largely observational and characterized by substantial heterogeneity. Differences in study design, population characteristics, dietary patterns, medication use, and analytical methods likely contribute to the lack of consensus regarding specific microbial taxa associated with depression.

A recurring theme across studies is the functional relevance of microbial communities rather than taxonomic composition alone. Alterations in microbial metabolic capacity—particularly those affecting short-chain fatty acid production, immune modulation, and neurotransmitter

metabolism-appear to be more consistently associated with depressive phenotypes than changes in individual bacterial genera. This functional perspective may help reconcile conflicting findings at the taxonomic level and supports a shift toward metabolomic and functional analyses in future research.

Neuroinflammation emerges as a key mechanistic link between gut microbiota and depression. Dysbiosis-associated increases in intestinal permeability and systemic inflammation may activate immune pathways that negatively affect brain function, neurogenesis, and synaptic plasticity. Importantly, inflammatory mechanisms are not uniformly present in all patients with depression, suggesting that microbiota-related pathways may be particularly relevant in specific subtypes of the disorder, such as inflammation-associated or treatment-resistant depression.

The hypothalamic-pituitary-adrenal axis represents another critical interface between gut microbiota and emotional regulation. Experimental studies indicate that microbial signals influence stress reactivity and cortisol secretion, potentially exacerbating vulnerability to depression under chronic stress conditions. Restoration of microbial balance has been shown to normalize stress responses in animal models, highlighting the therapeutic relevance of microbiota modulation.

From a clinical perspective, microbiota-targeted interventions offer promising but still preliminary therapeutic opportunities. Probiotics, prebiotics, dietary modification, and faecal microbiota transplantation have demonstrated antidepressant-like effects in preclinical models and modest benefits in some clinical trials. However, variability in intervention protocols, strain specificity, treatment duration, and outcome measures limits the generalizability of current findings. Moreover, emerging evidence that gut microbiota influences the metabolism and efficacy of antidepressant medications underscores the importance of considering host-microbiota interactions in personalized treatment approaches.

Finally, recent studies exploring microbiota-based risk prediction models suggest that gut microbial profiles may serve as potential biomarkers for depression. Although these approaches are still in early stages of development, they represent a promising step toward non-invasive diagnostic and prognostic tools.

## 5. Conclusions

The available evidence supports a meaningful association between gut microbiota and depression, mediated through immune, neuroendocrine, and metabolic pathways within the microbiota-gut-brain axis. Preclinical studies strongly suggest a causal role of microbial

dysbiosis in the development of depressive-like behaviors, whereas human studies remain predominantly associative.

Gut microbiota represents a promising target for novel diagnostic and therapeutic strategies in depression; however, further longitudinal, mechanistic, and interventional studies are required to establish causality and clinical efficacy. Future research should prioritize standardized methodologies, functional microbiome analyses, and stratification of depressive subtypes to better define the role of gut microbiota in mental health.

## **Disclosure**

### **Author's contribution**

Conceptualization: [KW], [JM]

Methodology: [EC], [PZ], [WW]

Check: [JB], [JM], [IZ]

Investigation: [WW], [KW], [JB]

Data curation: [JM], [PZ], [EC], [IZ]

Writing - rough preparation: [IZ], [JB], [PZ]

Writing - review and editing: [KW], [WW]

Visualization: [PZ], [EC], [IZ]

Project administration: [KW], [WW], [JM]

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## Conflict Of Interest

The authors declare no conflict of interest.

**All authors have read and agreed with the published version of the manuscript.**

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