The Gut–Brain–Microbiota Axis in Psychiatric Disorders: A Review of Mechanisms, Dysfunctions, and Potential Therapies

Julia Chołda

Śląski Uniwersytet Medyczny w Katowicach: Katowice, Silesia, PL

https://orcid.org/0009-0008-0101-6393

Wiktoria Janik

Śląski Uniwersytet Medyczny w Katowicach, Katowice, Silesia, PL

https://orcid.org/0009-0006-8406-3309

Kinga Jamontt

Joannitas Hospital in Pszczyna, dr. W. Antesa 11 Street, 43-200 Pszczyna, Silesia, Poland

https://orcid.org/0009-0002-2755-2975

Magdalena Matlakiewicz

American Heart of Poland S.A. in Katowice, Poland

Warszawska 52, 40-028 Katowice, Poland

https://orcid.org/0000-0003-1305-5070

Aleksander Manasar

Śląski Uniwersytet Medyczny w Katowicach: Katowice, PL

https://orcid.org/0009-0002-1988-3942

Anna Matuszek

Śląski Uniwersytet Medyczny w Katowicach: Katowice, PL

https://orcid.org/0009-0008-3600-9783

Ewa Siedy-Florek

Beskidzkie Centrum Onkologii - Szpital Miejski im. Jana Pawła II, Stanisława Wyspiańskiego 21, 43-300 Bielsko-Biała, Poland

https://orcid.org/0009-0004-6536-024X

Katarzyna Szlachetka

Śląski Uniwersytet Medyczny w Katowicach: Katowice, PL

https://orcid.org/0009-0006-8012-4805

Maja Strzeszyna

Śląski Uniwersytet Medyczny w Katowicach, Katowice, Silesia, PL

https://orcid.org/0009-0000-8599-163X

Zofia Stawowy

Śląski Uniwersytet Medyczny w Katowicach, Katowice, Silesia, PL

https://orcid.org/0009-0004-5864-5343

Abstract

The gut–brain–microbiota axis (GBA) is a bidirectional communication network connecting the gastrointestinal tract, gut microbiota, and the central nervous system. This review explores the GBA's role in the pathophysiology of selected psychiatric disorders—depression, anxiety, and autism spectrum disorders (ASD). The study presents current knowledge of neuroimmune, endocrine, and metabolic pathways involved in microbiota–brain signaling, with special attention to the vagus nerve, HPA axis, gut barrier integrity, and bacterial metabolites such as SCFAs and neurotransmitters. Clinical data indicate altered microbiota composition in patients with psychiatric conditions, showing reduced microbial diversity and increased pro-inflammatory bacteria. Evidence suggests a correlation between dysbiosis, systemic inflammation, and changes in neurotransmission. The paper reviews therapeutic strategies, including probiotics, prebiotics, dietary interventions (e.g., Mediterranean or elimination diets), and fecal microbiota transplantation (FMT). Probiotics such as *Lactobacillus rhamnosus* or *Bifidobacterium longum* show promise in reducing anxiety and depressive symptoms, while FMT appears particularly effective in alleviating both GI and behavioral symptoms in ASD. The findings highlight the therapeutic potential of targeting the microbiota in psychiatric care, although further standardized and long-term studies are needed.

Keywords

gut microbiota, depression, anxiety, autism spectrum disorder, probiotics, fecal microbiota transplantation, gut–brain axis

1. Introduction

The gut–brain–microbiota axis (GBA) describes the bidirectional communication between the gastrointestinal tract (along with its resident microbiota) and the central nervous system (CNS). Although observations of the gut's influence on mood date back to ancient times, systematic scientific research into this phenomenon began only recently (Chaiyasut et al., 2023). Communication occurs through several pathways, including neural (particularly the vagus nerve and the enteric nervous system – ENS), hormonal, and immune signaling.

The enteric nervous system, often referred to as the "second brain," contains millions of neurons located in the gut wall and is capable of independently regulating gastrointestinal functions while simultaneously sending signals to the brain (Loh et al., 2024). A key communication channel is the vagus nerve, which transmits signals in both directions – from the gut to the brain and vice versa.

Clinical observations strongly suggest a link between gut health and mental health. In depression, irritable bowel syndrome (IBS) is often comorbid, and anxiety disorders are frequently associated with gastrointestinal diseases (McGuinness et al., 2022). Furthermore, top-down treatments – such as antidepressants or psychotherapy – are sometimes effective in alleviating IBS symptoms, suggesting deeper systemic interrelations. These findings indicate that psychiatric disorders have not only a neurological but also a somatic dimension, and that microbiota may play a significant role in their pathophysiology.

2. Anatomy and Physiology of the Gut–Brain–Microbiota Axis

The human gastrointestinal tract is home to hundreds to around 2,000 bacterial species. The gut microbiome contains up to 150 times more genes than the human genome, making it a key player in host metabolism (Zhang et al., 2023). Gut bacteria participate in digestion, vitamin synthesis, regulation of intestinal barrier permeability, and modulation of the immune system. Due to its immense metabolic activity, the microbiota is often referred to as the host's "metabolic machinery."

On the other hand, the ENS consists of a network of millions of neurons and demonstrates autonomous regulatory capacities, such as control of gut motility. The vagus nerve serves as the main afferent and efferent connection between the gut and the brain, enabling rapid information exchange. Additionally, enterochromaffin cells in the intestine produce neurotransmitters (e.g., serotonin), and microbial metabolites (such as short-chain fatty acids or bile acids) can enter the bloodstream and affect brain function.

3. Communication Mechanisms Between Gut Microbiota and the Brain

The gut–brain axis functions as a complex communication system, where gut microbiota can influence the central nervous system through multiple interconnected pathways.

One of the most important mechanisms of this interaction involves **neural pathways**, among which the vagus nerve plays a key role. It is the primary route for transmitting afferent (sensory) and efferent (motor) signals between the gut and the brain. It has been demonstrated that vagus nerve stimulation can influence mood and anxiety levels, while its blockade can abolish the effects of certain psychotropic probiotics (Chaiyasut et al., 2023).

A second major category comprises **immune pathways**. The integrity of the intestinal barrier plays a crucial role in preventing pathogens and their components (e.g., lipopolysaccharides – LPS) from entering the bloodstream. When the barrier is compromised, bacterial products can infiltrate the systemic circulation, initiating an inflammatory response. Systemic inflammation can activate microglia in the brain – specialized immune cells of the CNS – which is closely linked to the pathogenesis of mood disorders, depression, and anxiety syndromes (Nikel et al., 2025).

Another regulatory level involves **endocrine pathways**, particularly the **hypothalamic–pituitary–adrenal (HPA) axis**, which governs the body's response to stress. The microbiota influences this axis by modulating cortisol levels and tryptophan metabolism – the amino acid precursor of serotonin. Dysfunctions in the HPA axis are commonly observed in patients with depression and chronic stress. Research indicates that some probiotic strains can restore HPA axis balance, reduce cortisol levels, and improve stress resilience (Zhang et al., 2023).

Still another essential aspect is the **role of bacterial metabolites**. Short-chain fatty acids (SCFAs), such as butyrate, acetate, and propionate, are products of fiber fermentation by gut bacteria and influence many physiological processes, including blood–brain barrier permeability, neuronal activity, and the expression of genes related to neuroplasticity. Other microbial metabolic products – secondary bile acids, neurotransmitters (e.g., GABA, serotonin, dopamine), and their precursors – also participate in regulating emotions, cognitive functions, and behavior (Loh et al., 2024).

All these mechanisms demonstrate that gut–brain communication is not a single-channel system. On the contrary, it represents a multilayered network of interactions between the nervous, immune, and endocrine systems. Understanding these pathways is key to developing effective microbiota-targeted therapies for treating psychiatric disorders.

4. Gut Microbiota and Psychiatric Disorders

4.1 Depression

Depressive disorders are currently among the most common and burdensome mental health conditions worldwide. In recent years, an increasing number of studies have focused on the potential relationship between gut microbiota composition and the development and severity of depressive symptoms. Individuals with depression frequently exhibit **reduced microbial diversity**, meaning a lower number and balance of bacterial species in the gut compared to mentally healthy individuals (McGuinness et al., 2022).

There is often an **increased presence of potentially pathogenic and pro-inflammatory bacteria**, such as *Alistipes* and members of the *Enterobacteriaceae* family, along with a decline in beneficial anti-inflammatory strains that support gut barrier integrity, such as *Faecalibacterium prausnitzii* and *Coprococcus spp.* (Qi et al., 2024). These changes are not only a consequence of metabolic and immune disturbances but may also actively contribute to depression progression by influencing the HPA axis, microglial function, and the production of neuroactive metabolites.

Many studies also demonstrate a **correlation between microbiota alterations and levels of inflammatory markers** in the blood (e.g., interleukin IL-6 and C-reactive protein), confirming the role of inflammation as a potential mediator between gut disturbances and depression (Nikel et al., 2025).

Clinical analyses and meta-analyses have shown that **probiotic supplementation may moderately reduce depressive symptoms**. However, the effectiveness of such interventions depends on several factors. Strains like *Lactobacillus helveticus* and *Bifidobacterium longum* have been identified as potentially the most effective in improving mood and reducing anxiety symptoms (Zhang et al., 2023). Other important factors include dosage (commonly ranging from 10^8 to 10^{11} CFU per day), duration of therapy (usually 4 to 12 weeks), and the presence of comorbid conditions (e.g., IBS, autoimmune diseases) that may influence probiotic response (Chaiyasut et al., 2023).

Some studies also employed combined approaches, such as **synbiotics** (probiotics + prebiotics), which further support the colonization of beneficial bacteria by providing appropriate substrates. However, results remain inconsistent, underscoring the need for further

well-designed clinical trials to identify the most effective strains and therapeutic protocols for patients with depression.

In summary, depression is associated with characteristic alterations in gut microbiota composition that promote chronic inflammation and dysregulation of the HPA axis. These mechanisms represent potential targets for microbiota-based interventions that could complement traditional pharmacological and psychotherapeutic approaches."

4.2 Anxiety Disorders

Anxiety disorders, alongside depression, are among the most commonly diagnosed mental health issues, and their etiology is multifactorial—encompassing genetic, environmental, immunological, and neurochemical factors. In recent years, growing attention has been given to the role of gut microbiota in the pathogenesis of these disorders, particularly in relation to the gut–brain axis.

Patients with anxiety disorders display **distinct alterations in gut microbiome composition**. Typically, there is an overrepresentation of **Proteobacteria**, which are often associated with inflammation and oxidative stress, accompanied by a decrease in **beneficial Firmicutes strains**, known for their anti-inflammatory and immunomodulatory functions (Nikel et al., 2025). This dysbiosis may lead to **increased intestinal permeability ("leaky gut")**, allowing bacterial toxins like lipopolysaccharides (LPS) to enter the bloodstream and trigger inflammatory responses, including those within the central nervous system.

Experimental and clinical studies have demonstrated that **certain probiotic strains can alleviate anxiety symptoms**. *Lactobacillus rhamnosus* and *Lactobacillus plantarum* are among the most thoroughly studied psychobiotic bacteria. Their effectiveness is primarily attributed to their ability to **modulate the GABAergic system** – the brain's main inhibitory system. In animal models, administration of *L. rhamnosus* increased the expression of GABA-A receptors in the prefrontal cortex and amygdala, resulting in reduced anxiety-like behaviors (Chaiyasut et al., 2023).

Additionally, some of these strains influence **HPA axis activity**, lowering cortisol (the stress hormone) levels and decreasing the expression of inflammation-related genes. As a result, subjective symptoms such as tension, restlessness, and irritability may be reduced. Notably, human studies also report **improvements in sleep quality, fewer nocturnal awakenings, and**

better focus and attention stability with probiotic supplementation (Zhang et al., 2023; McGuinness et al., 2022).

Therapeutic effects vary depending on **factors such as dosage** (commonly 10^9–10^{11} CFU/day), **form of administration** (capsules, sachets), **duration of intervention** (typically 2 to 12 weeks), and **baseline patient condition**. In individuals with high stress levels or coexisting somatic disorders, psychobiotic effects may be more pronounced. Despite promising findings, further **randomized placebo-controlled trials** are necessary to standardize treatment protocols and identify the populations most likely to benefit from this form of therapy.

In conclusion anxiety disorders exhibit a dysbiosis profile distinct from that of depression; however, as in depression, impaired gut barrier integrity and activation of inflammatory responses are observed. This suggests the potential for targeted microbiota-modulating strategies to alleviate symptoms.

4.3 Autism Spectrum Disorders (ASD)

Autism spectrum disorders (ASD) are a group of neurodevelopmental conditions characterized by **deficits in social communication** and **repetitive behavior patterns**. A growing body of evidence suggests the possible involvement of **gut microbiota in ASD pathophysiology**, especially considering that **gastrointestinal (GI) symptoms affect 70–90% of children with ASD** (Kraneveld et al., 2024; Zhu et al., 2023).

The **microbial profile of children with ASD differs significantly** from that of neurotypical peers. Many studies report a **reduction in probiotic bacteria**, such as *Bifidobacterium* and *Lactobacillus,* along with an **increase in potentially pathogenic or pro-inflammatory bacteria**, including *Clostridium spp.*, *Desulfovibrio*, *Prevotella,* and *Bacteroides* (Kraneveld et al., 2024). This dysbiosis can result in **increased intestinal epithelial permeability**, allowing microbial metabolites to enter systemic circulation. Compounds such as SCFAs or ammonia may exert **neurotoxic effects on the developing CNS**.

The **gut–brain hypothesis** proposes that microbiota imbalances affect not only GI functioning but also **behavior, emotions, and cognitive functions** in children with ASD. Suggested mechanisms include microbial modulation of **neurotransmitters**, such as serotonin, dopamine, and GABA, which are known to be dysregulated in autistic individuals (Loh et al., 2024). Moreover, microbiota–immune system interactions may lead to **chronic CNS**

inflammation, further complicating the ASD clinical picture. Given the significant role of dysbiosis in the pathophysiology of ASD and its potential impact on central nervous system function, it is justified to explore therapeutic interventions aimed at restoring gut microbiota balance.

In response to these findings, researchers have begun exploring **microbiome-targeted therapies**. Preliminary clinical trials using **probiotics**, especially mixtures containing *Lactobacillus acidophilus*, *Bifidobacterium infantis*, and *L. rhamnosus*, have shown improvements in both **GI symptoms and social behavior**, as well as **reduced stereotypies** (Chaiyasut et al., 2023). **Elimination diets** (e.g., gluten-free, casein-free) have also been used, showing some benefits in psychomotor functioning for certain patients. However, the effectiveness of such approaches remains debated due to methodological limitations and inconsistent outcomes.

The most promising ASD therapy to date appears to be **fecal microbiota transplantation (FMT)**. Randomized controlled trials in children with ASD suggest that FMT can **significantly improve GI symptoms and reduce autism-related behaviors**, as measured both subjectively (by parents) and using standardized scales like CARS or ABC (Zhu et al., 2023). FMT leads to **increased microbial diversity and restored levels of beneficial gut bacteria**, which may directly impact **cognitive and social functioning**.

In summary, **gut microbiota plays a significant role in ASD**, serving as both a potential pathological factor and a therapeutic target. While many results are promising, further well-controlled **placebo-based studies** are needed to determine the long-term efficacy and safety of interventions such as probiotics or FMT.

5. Gut Microbiota-Targeted Therapies

5.1 Probiotics and Prebiotics

An increasing body of evidence points to the potential role of **probiotics and prebiotics** as adjunct therapies in treating mental health disorders, particularly depression and anxiety. A meta-analysis by Zhang et al. (2023), which included randomized clinical trials, found that probiotic supplements produced **a moderate but statistically significant reduction in depressive symptoms**. The therapeutic effect strongly depended on the specific strain used

(most commonly *Lactobacillus acidophilus*, *Lactobacillus helveticus*, *Bifidobacterium bifidum*), dosage, and treatment duration. In most of the analyzed studies, interventions lasted from **4 to 12 weeks**, with doses ranging from **10^8 to 10^{11} CFU per day**.

Mechanisms of probiotic action include **modulation of the HPA axis**, **inflammation reduction**, and **production of neuroactive metabolites** (e.g., SCFAs, tryptophan, gamma-aminobutyric acid – GABA). Chaiyasut et al. (2023) emphasized that probiotics can regulate stress responses by influencing **cortisol levels** and **GABA receptor expression in the CNS**, which is particularly relevant in the context of anxiety treatment.

Prebiotics, such as **inulin** and **fructooligosaccharides**, promote the growth of beneficial bacteria (e.g., *Faecalibacterium prausnitzii*) and enhance SCFA production, which positively affects **intestinal barrier integrity** and **immune system function**. Although fewer clinical studies have been conducted on prebiotics compared to probiotics, their therapeutic potential—especially in chronic inflammation and mood disorders—is promising.

5.2 Diet

Dietary interventions are an important component of strategies aimed at modulating the gut microbiota. The **Mediterranean diet**, rich in fiber, polyphenols, unprocessed plant foods, and omega-3 fatty acids, has drawn particular attention. In a systematic review by McGuinness et al. (2022), this diet was found to **support gut eubiosis** and **reduce pro-inflammatory cytokines** (e.g., IL-6, TNF-α), which are associated with the pathophysiology of depression.

In the context of autism spectrum disorders (ASD), **elimination diets**, including **gluten-free and casein-free diets**, have also been evaluated. Although some studies (Zhu et al., 2023) suggest improvements in social functioning and a reduction in GI symptoms following such interventions, results remain **inconsistent**. Meta-analysis authors stress the importance of individual differences and note a **lack of conclusive evidence** supporting the efficacy of these diets as standard therapeutic approaches.

5.3 Fecal Microbiota Transplantation (FMT)

FMT, or **fecal microbiota transplantation** from a healthy donor, is gaining interest as a method for restoring microbiological balance in the intestines of patients with psychiatric disorders. Zhu et al. (2023) conducted a meta-analysis of randomized controlled trials assessing

the **effectiveness of FMT in children with ASD**. Results indicated **moderate improvements in behavioral symptoms** and **reductions in gastrointestinal complaints**. In these studies, FMT was administered via **oral capsules, rectal enemas**, or **nasoduodenal tubes**, typically in cycles lasting from a few days to a week.

In the case of **depression**, evidence is more limited. However, pilot studies—such as those described by Nikel et al. (2025) suggest **improvements in mood and reductions in anhedonia** following FMT. Still, there is a lack of **large-scale, long-term studies**, so this method remains **outside of standard treatment recommendations**.

Safety concerns—such as **the risk of pathogen transmission** or **immune reactions**—also warrant further investigation and the development of robust **screening protocols**.

Disorder	Microbiota Alterations	Pathophysiological Mechanisms	Most Studied Microbiota-Based Therapies	Clinical Effect	Strength of Evidence
Depression	↓ diversity, ↑ pro-inflammatory bacteria (Alistipes, Enterobacteriaceae), ↓ beneficial bacteria (Faecalibacterium prausnitzii, Coprococcus spp.); ↑ inflammatory markers (IL-6, CRP)	HPA axis dysfunction, inflammation, altered microglial activity, changes in neuroactive metabolites (SCFAs, serotonin)	Probiotics (L. helveticus, B. longum), synbiotics, prebiotics, Mediterranean diet; FMT (pilot studies only)	Reduction in depressive symptoms, mood improvement, preliminary mood improvement (FMT)	Moderate for probiotics/diet; preliminary for FMT
Anxiety Disorders	↑ Proteobacteria; ↓ Firmicutes; increased intestinal permeability ('leaky gut')	HPA axis activation, elevated cortisol, inflammatory gene expression, modulation of the GABAergic system	Probiotics (L. rhamnosus, L. plantarum), potential role for prebiotics	Reduced anxiety and tension, improved sleep quality, better concentration	Moderate for probiotics; limited for prebiotics
ASD	↓ Bifidobacterium, Lactobacillus; ↑ Clostridium, Desulfovibrio, Prevotella, Bacteroides	Dysbiosis → neurotoxic metabolites (SCFAs, ammonia), CNS inflammation, altered serotonin, dopamine, GABA neurotransmission	Probiotics (mixtures: L. acidophilus, B. infantis, L. rhamnosus), elimination diets (gluten-free/casein-free, mixed evidence), FMT	Improvement in GI symptoms, partial improvement in social functioning (probiotics/diets); marked improvement in GI and behavioral symptoms (FMT)	Strongest for FMT; mixed for probiotics and diets

Table 1. Comparative summary of key microbiota-targeted therapeutic findings across psychiatric disorders discussed in this review.

6. Discussion and Future Research Directions

Current research clearly indicates that **gut microbiota plays a significant role** in the gut–brain axis, affecting the functioning of the central nervous system as well as the development and course of psychiatric disorders such as **depression, anxiety disorders, and autism spectrum disorders**. Both observational and interventional clinical studies emphasize that **modulating the microbiota** through **probiotics, prebiotics, diet, or fecal microbiota transplantation (FMT)** can lead to improvements in psychiatric symptoms.

However, despite promising findings, the existing data remain **largely heterogeneous**. The diversity of probiotic strains used, variations in dosages, and differences in therapy duration make it difficult to clearly define **optimal therapeutic protocols**. Furthermore, many studies involve small sample sizes and short follow-up periods, limiting the ability to assess the **long-term efficacy and safety** of these interventions.

Future studies should therefore focus on **large, randomized, and controlled clinical trials**, which will allow for the precise identification of the most effective **strain combinations**, **dosages**, and **treatment durations** tailored to specific psychiatric disorders. **Long-term follow-up** will also be essential to evaluate the **persistence of effects** and identify any potential **side effects**.

A key direction for future research should be an **integrative approach** combining fields such as **genetics, neuroimaging, metabolomics, and microbiota analysis**. Such multidisciplinary studies may lead to a better understanding of the **molecular and neurobiological mechanisms** underlying the gut–brain axis and the specific interactions between microorganisms and the nervous system.

Animal models, particularly germ-free mice and those with modulated microbiota, provide important insights into the mechanisms by which microbiota affect brain function. However, findings from animal studies must be **confirmed in human clinical trials**, due to differences in microbiome composition and the complexity of the human nervous system.

Further research should also explore **synergistic effects** of various therapies (e.g., combining probiotics with diet or psychotherapy) and identify **predictive biomarkers** of response to microbiota-based interventions. The development of **personalized therapies**, tailored to an

individual's microbiota profile and clinical status, could significantly enhance treatment efficacy and reduce adverse effects.

7. Conclusions

The **gut–brain–microbiota axis** is becoming an increasingly recognized and studied element in the **pathophysiology of psychiatric disorders**. It is now evident that **gut microbiota is not a passive bystander**, but an **active participant** in the neurobiological processes that affect **mood, emotions, and cognitive functions**. Continued exploration of this axis may lead to the development of **new, more effective, and personalized methods** for treating conditions such as depression, anxiety disorders, and autism spectrum disorders.

The implementation of **microbiota-based therapies**, including the precise modulation of the gut ecosystem via **probiotics, prebiotics, diet, or FMT**, holds the potential to **complement traditional psychiatric treatments**. However, it is crucial to conduct studies of **high methodological quality** to fully realize this promising direction and to ensure that patients are provided with **safe, effective, and lasting therapeutic solutions**.

Author's contribution

Conceptualization: Julia Chołda, Kinga Jamontt,

Methodology: Magdalena Matlakiewicz, Maja Sterzeszyna

Software: Wiktoria Janik, Aleksander Manasar

Check: Zofia Stawowy, Ewa Siedy-Florek

Formal analysis: Katarzyna Szlachetka, Maja Strzeszyna

Investigation: Julia Chołda, Magdalena Matlakiewicz

Resources: Wiktoria Janik, Aleksander Manasar

Data curation: Anna Matuszek, Ewa Siedy-Florek, Kinga Jamontt

Writing -rough preparation: Julia Chołda, Anna Matuszek, Kinga Jamontt

Writing -review and editing: Zofia Stawowy, Magdalena Matlakiewicz

Visualization: Wiktoria Janik, Aleksander Manasar, Katarzyna Szlachetka

Supervision: Anna Matuszek, Maja Strzeszyna, Katarzyna Szlachetka

Project administration: Julia Chołda, Ewa Siedy-Florek, Zofia Stawowy

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

References

[1] Chaiyasut, C., Prajapati, B. G., & Sivamaruthi, B. S. (2023). Gut microbiota in anxiety and depression: Unveiling the relationships and management options. *Pharmaceuticals, 16*(4), 565. https://doi.org/10.3390/ph16040565

[2] Chaiyasut, C., & Sivamaruthi, B. S. (2018). Influence of probiotic supplementation on brain function: Involvement of gut microbiome, inflammation, and stress pathway. In A. Evrensel & B. O. Unsalver (Eds.), *Gut Microbiota–Brain Axis* doi: 10.5772/intechopen.79511

[3] Foster, J. A., & McVey Neufeld, K. A. (2013). Gut–brain axis: How the microbiome influences anxiety and depression. *Trends in Neurosciences, 36*(5), 305–312. https://doi.org/10.1016/j.tins.2013.01.005

[4] Hsiao, E. Y., et al. (2013). Microbiota modulate behavioral and physiological abnormalities associated with neurodevelopmental disorders. *Cell, 155*(7), 1451–1463. https://doi.org/10.1016/j.cell.2013.11.024

[5] Kang, D.-W., et al. (2013). Reduced incidence of Prevotella and other fermentative bacteria in intestinal microflora of autistic children. *PLoS ONE, 8*(7), e68322. https://doi.org/10.1371/journal.pone.0068322

[6] Kraneveld, A. D., Perez-Pardo, P., & Lopez-Rincon, A. (2024). A robust microbiome signature for autism spectrum disorder across different studies using machine learning. *Scientific Reports, 14*, 814. https://www.nature.com/articles/s41598-023-50601-7

[7] Loh, J. S., et al. (2024). Microbiota–gut–brain axis and its therapeutic applications in neurodegenerative diseases. *Signal Transduction and Targeted Therapy, 9*, 37. doi: 10.1038/s41392-024-01743-1

[8] McGuinness, A. J., et al. (2022). A systematic review of gut microbiota composition in observational studies of major depressive disorder, bipolar disorder and schizophrenia. *Molecular Psychiatry, 27*(4), 1920–1935. doi: 10.1038/s41380-022-01456-3.

[9] Menezes, I. C., et al. (2018). Shotgun metagenomics reveals both taxonomic and tryptophan pathway alterations in major depressive disorder. *Behavioural Brain Research, 357–358*, 29–38. doi: 10.1017/S0033291719003027

[10] Ng, Q. X., et al. (2018). A meta-analysis of the use of probiotics to alleviate depressive symptoms. *Journal of Affective Disorders, 228*, 13–19. https://doi.org/10.1016/j.jad.2017.11.063

[11] Nikel, K., et al. (2025). The impact of gut microbiota on the development of anxiety symptoms—A narrative review. *Nutrients, 17*(6), 933. https://doi.org/10.3390/nu17060933

[12] Qi, D., et al. (2024). Unveiling the gut microbiota blueprint of schizophrenia: A multilevel omics approach. *Frontiers in Psychiatry, 15*, 1452604. https://doi.org/10.3389/fpsyt.2024.1452604

[13] Seo, D.-O., & Holtzman, D. M. (2024). Current understanding of the Alzheimer's disease-associated microbiome and therapeutic strategies. *Experimental & Molecular Medicine, 56*(1), 86–94. doi: 10.1038/s12276-023-01146-2

[14] Severance, E. G., et al. (2016). The gut microbiome in schizophrenia: A systematic review. *Schizophrenia Research, 176*(1), 23–29. https://doi.org/10.1016/j.schres.2016.04.018

[15] Slykerman, R. F., et al. (2017). Effect of probiotics on depressive symptoms: A systematic review and meta-analysis. *Nutrition Reviews, 75*(9), 679–693. doi: 10.1016/j.psychres.2019.112568

[16] Yolken, R. H., & Dickerson, F. B. (2014). Microbiota, infections, and schizophrenia. *Current Opinion in Psychiatry, 27*(3), 185–190.

[17] Zhang, Q., et al. (2023). Effect of prebiotics, probiotics, synbiotics on depression: Results from a meta-analysis. *BMC Psychiatry, 23*, 477. https://doi.org/10.1186/s12888-023-04963-x

[18] Zheng, Y., et al. (2023). The interaction between intestinal bacterial metabolites and phosphatase and tensin homolog in autism spectrum disorder. *Molecular and Cellular Neuroscience, 124*, 103805. https://doi.org/10.1016/j.mcn.2022.103805

[19] Zhu, D., et al. (2023). Efficacy of fecal microbiota transplantation for the treatment of autism in children: A meta-analysis of randomized controlled trials. *Evidence-Based Complementary and Alternative Medicine, 2023*, 5993628. https://doi.org/10.1155/2023/5993628

[20] Zuo, T., & Ng, S. C. (2018). The gut microbiota in the pathogenesis and therapeutic management of autism spectrum disorder. *Gut Microbes, 9*(6), 583–595. doi: 10.3389/fmicb.2018.02247