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GUT MICROBIOTA AND THE GUT–BRAIN AXIS IN ANXIETY DISORDERS: CURRENT EVIDENCE AND THERAPEUTIC PERSPECTIVES

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ABSTRACT

Anxiety disorders are among the most prevalent mental health conditions worldwide and constitute a significant public health challenge. Their pathogenesis is multifactorial and involves complex interactions between genetic, environmental, immunological, and neurobiological factors. In recent years, increasing attention has been directed toward the potential role of the gut microbiota and the gut–brain axis in the development and progression of anxiety disorders.

The aim of this narrative review was to summarize current knowledge regarding the relationship between gut microbiota and anxiety disorders, with particular emphasis on the biological mechanisms underlying gut–brain communication and potential therapeutic implications. The review was based on scientific publications indexed in PubMed, Scopus, and Google Scholar databases, including preclinical studies, clinical trials, systematic reviews, and meta-analyses published mainly between 2010 and 2025.

Available evidence indicates that gut microorganisms moderate central nervous system functioning through neural, immunological, endocrine, and metabolic pathways. Dysbiosis has been associated with altered stress responses, neuroinflammation, disturbances in neurotransmitter regulation, and hypothalamic–pituitary–adrenal axis dysfunction. Both animal and human studies suggest that alterations in gut microbiota composition may contribute to anxiety-related symptoms.

Although microbiota-targeted interventions such as probiotics, prebiotics, dietary modifications, and fecal microbiota transplantation show promising results, current evidence remains insufficient for routine clinical implementation. Further well-designed clinical studies are necessary to clarify the role of gut microbiota in anxiety disorders and to evaluate the effectiveness of microbiota-based therapeutic strategies.

KEYWORDS

Gut Microbiota, Gut–Brain Axis, Anxiety Disorders, Dysbiosis, Psychobiotics

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1. Introduction

Anxiety and depressive disorders are among the most prevalent mental health conditions worldwide and may lead to significant psychological distress as well as substantial impairment in social and occupational functioning. It is estimated that together they affect approximately 10% of the global population (Lubecka et al., 2022). Anxiety disorders comprise a group of psychiatric conditions characterized by excessive and persistent fear, anxiety, and tension that are difficult to control and often disproportionate to the actual threat (Simpson et al., 2021). The most commonly diagnosed anxiety disorders include generalized anxiety disorder (GAD), social anxiety disorder (SAD), panic disorder (PD), and agoraphobia (Simpson et al., 2021). Although these disorders differ in their clinical presentation, they share the common feature of excessive anxiety leading to considerable impairment in daily functioning (Simpson et al., 2021). The pathogenesis of anxiety disorders is complex and involves the interaction of genetic, neurobiological, and environmental factors (Liśkiewicz, 2019).

Anxiety disorders frequently coexist with other psychiatric and somatic diseases. In particular, they commonly occur together with depressive disorders, which may further worsen disease course and quality of life (Simpson et al., 2021). Moreover, numerous studies indicate a relationship between anxiety disorders and gastrointestinal diseases, especially irritable bowel syndrome (IBS) (Liśkiewicz, 2019). The coexistence of these conditions suggests the presence of shared biological mechanisms, potentially involving disturbances in gut–brain axis communication (Kelly et al., 2016).

The gut microbiota is a complex ecosystem of microorganisms inhabiting the human gastrointestinal tract and is involved in maintaining physiological homeostasis (Jiang et al., 2015). It consists primarily of

bacteria, but also includes archaea, fungi, viruses, including bacteriophages, and protozoa (Jiang et al., 2015). It is estimated that the number of microorganisms colonizing the human gastrointestinal tract reaches approximately 10^{13}–10^{14} cells, while the total number of microbial genes greatly exceeds the number of genes in the human genome (MacKay et al., 2019). Gut microorganisms participate in numerous physiological processes, including nutrient metabolism, regulation of immune responses, and maintenance of intestinal barrier integrity (MacKay et al., 2019). Recent studies indicate that the gut microbiota participates in the regulation of central nervous system function through dynamic bidirectional communication between the gut and the brain, referred to as the gut–brain signaling (Sarkar et al., 2018). The gut–brain axis is a multifactorial communication network involving neural, immunological, hormonal, and metabolic pathways (Sarkar et al., 2018).

Despite the growing number of studies investigating the relationship between the gut microbiota and mental health, the exact mechanisms linking gut microbial alterations with anxiety disorders remain incompletely understood. In particular, there is still limited clarity regarding the role of dysbiosis, neuroinflammatory processes, microbial metabolites, and gut–brain signaling pathways in the development and progression of anxiety-related symptoms.

The aim of this paper is to review the current state of knowledge regarding the role of the gut microbiota and the microbiota-mediated communication in the pathogenesis of anxiety disorders, with particular emphasis on the biological mechanisms underlying this relationship.

The literature review was based on recent scientific publications concerning the gut microbiota and the gut–brain axis in the context of anxiety disorders, including both preclinical and clinical studies. Studies relevant to the understanding of biological mechanisms and potential therapeutic implications were included in the analysis.

2. Methodology

This study was conducted as a narrative review of the current scientific literature concerning the role of the gut microbiota and the gut–brain axis in the pathogenesis of anxiety disorders. The review aimed to summarize and critically discuss the available evidence regarding the biological mechanisms linking gut microbial composition with anxiety-related symptoms, as well as the potential therapeutic implications of microbiota-targeted interventions.

A literature search was performed using the PubMed, Scopus, and Google Scholar databases. Publications published between January 2010 and March 2025 were considered eligible for inclusion, with particular emphasis placed on studies published within the last decade in order to reflect the most recent advances in microbiome research.

The search strategy was based on combinations of the following keywords and Medical Subject Headings (MeSH) terms: “gut microbiota”, “gut microbiome”, “gut–brain axis”, “microbiota–gut–brain axis”, “anxiety disorders”, “anxiety”, “stress response”, “dysbiosis”, “psychobiotics”, “probiotics”, “prebiotics”, “neuroinflammation”, and “fecal microbiota transplantation”. Boolean operators (“AND”, “OR”) were applied to optimize the search process and identify publications relevant to the topic.

The review included original experimental studies, observational clinical studies, randomized controlled trials, systematic reviews, meta-analyses, and narrative reviews investigating the relationship between gut microbiota and anxiety disorders. Both preclinical and clinical studies evaluating gut microbiota composition, gut–brain axis mechanisms, neurobiological pathways, and microbiota-targeted therapeutic interventions were considered eligible for inclusion.

The inclusion criteria comprised:

- studies concerning gut microbiota and anxiety disorders or anxiety-related symptoms,
- studies investigating gut–brain axis mechanisms,
- publications evaluating microbiota-targeted interventions, including probiotics, prebiotics, dietary interventions, synbiotics, and fecal microbiota transplantation,
- peer-reviewed articles published in English or Polish,
- studies providing relevant biological, clinical, or mechanistic data related to the discussed topic.

The exclusion criteria included:

- publications unrelated to anxiety disorders,
- studies lacking relevance to gut microbiota or gut–brain axis mechanisms,
- duplicate publications,
- conference abstracts without full-text availability,
- articles with insufficient methodological quality or incomplete data.

The collected literature was analyzed qualitatively, with particular focus on biological mechanisms involved in gut–brain communication, including neural, immunological, endocrine, and metabolic pathways. Special attention was paid to studies investigating dysbiosis, neuroinflammation, hypothalamic–pituitary–adrenal (HPA) axis dysregulation, neurotransmitter modulation, and microbial metabolites such as short-chain fatty acids and tryptophan-derived compounds.

Due to the heterogeneity of available studies in terms of methodology, analyzed populations, microbiota assessment techniques, and therapeutic interventions, a quantitative synthesis or meta-analysis was not performed. Instead, the available evidence was narratively synthesized to provide a comprehensive overview of current knowledge regarding the role of the gut microbiota in anxiety disorders.

3. Gut Microbiota

3.1. Composition and Functions of the Gut Microbiota

The composition of the gut microbiota is highly diverse and includes numerous groups of microorganisms inhabiting the human gastrointestinal tract, among which bacteria constitute the dominant component (Martin et al., 2018). The most abundant phyla include Firmicutes, Bacteroidetes, Actinobacteria, and Proteobacteria (Martin et al., 2018). These microorganisms remain in dynamic equilibrium with the host and contribute to physiological homeostasis (Martin et al., 2018). The microbial community inhabiting the gastrointestinal tract is characterized by considerable interindividual and intraindividual variability, meaning that its composition may change depending on multiple environmental and physiological factors (Góralczyk-Bińkowska et al., 2022). It is estimated that the number of microorganisms colonizing the human gastrointestinal tract reaches approximately 10^{13}–10^{14} cells, while their collective genetic material significantly exceeds the size of the human genome (Sender et al., 2016; Qin et al., 2010). Therefore, the gut microbiota is sometimes described as an additional "metabolic organ" due to its numerous functions essential for proper host functioning (Stephens et al., 2018).

As presented in Table 1 (tab. 1), the gut microbiota performs multiple metabolic, immunological, protective, and neuroactive functions.

Table 1. Key functions of the gut microbiota

Function of the microbiota	Mechanism of action	Significance for the host
Metabolic	Fermentation of indigestible dietary components and production of short-chain fatty acids (SCFAs), such as acetate, propionate, and butyrate	Energy supply for intestinal epithelial cells; regulation of glucose and lipid metabolism
Immunological	Stimulation and modulation of the immune response and influence on the maturation of immune cells	Maintenance of immune homeostasis and protection against pathogens
Protective (intestinal barrier)	Support of intestinal barrier integrity and competition with pathogenic microorganisms	Prevention of pathogen colonization in the gut
Neuroactive	Production and modulation of neurotransmitters and metabolites affecting nervous system function	Influence on gut–brain axis communication and regulation of nervous system function
Developmental	Influence on the development of the immune system and host metabolism	Proper development and maintenance of organismal homeostasis

Source: authors' own elaboration based on Jiang et al. (2015), MacKay et al. (2019), and Sarkar et al. (2018).

Gut microorganisms are involved in the breakdown of dietary components and the production of biologically active metabolites, including short-chain fatty acids (SCFAs) (Sarkar et al., 2018). These metabolites constitute an important source of energy for intestinal epithelial cells and contribute to the regulation of metabolic processes within the host organism (Koh et al., 2016). The gut microbiota also plays a significant role in the regulation of immune responses and the maturation of immune cells (Cao et al., 2021). Furthermore, it supports the integrity of the intestinal barrier and limits colonization by pathogenic microorganisms (Cao et al., 2021).

An increasing body of evidence suggests that the gut microbiota may also influence the functioning of the central nervous system (Martin et al., 2018). This communication occurs through complex neural, immunological, and hormonal mechanisms collectively referred to as the gut–brain axis (Carabotti et al., 2015).

Gut microorganisms affect the host organism not only through metabolic and immunological processes but also through the production of neuroactive compounds (Martin et al., 2018). It has been demonstrated that certain intestinal bacteria are capable of synthesizing neurotransmitters and other signaling molecules that may modulate nervous system function (Martin et al., 2018; Góralczyk-Bińkowska et al., 2022).

These compounds include gamma-aminobutyric acid (GABA), serotonin, dopamine, and noradrenaline. The ability to produce such neurotransmitters has been identified in bacterial genera including *Lactobacillus*, *Bifidobacterium*, *Escherichia*, and *Bacillus* (Góralczyk-Bińkowska et al., 2022).

Selected examples of gut microorganisms capable of neurotransmitter production and their potential biological significance are presented in Table 2 (tab. 2). The ability of the gut microbiota to synthesize neuroactive compounds constitutes one of the key mechanisms of communication between the gastrointestinal tract and the central nervous system within this signaling network (Martin et al., 2018). Among the most important substances produced by gut microorganisms are GABA, serotonin, dopamine, and noradrenaline, all of which play a substantial role in the regulation of mood, behavior, and stress responses (Góralczyk-Bińkowska et al., 2022). It has been shown that bacteria belonging to the genera *Lactobacillus* and *Bifidobacterium* contribute to GABA metabolism, whereas other microorganisms participate in serotonin synthesis pathways through modulation of tryptophan availability (Góralczyk-Bińkowska et al., 2022; Cao et al., 2021).

Table 2. Selected gut microorganisms involved in neurotransmitter production

Microorganism	Produced neurotransmitter / metabolite	Potential biological significance
Lactobacillus	GABA (gamma-aminobutyric acid)	Modulation of neuronal activity and potential reduction of anxiety symptoms
Bifidobacterium	GABA	Regulation of gut–brain axis function and influence on stress response
Escherichia	Serotonin	Influence on gastrointestinal motility and nervous system function
Bacillus	Dopamine	Involvement in the regulation of cognitive and emotional processes

Source: authors' own elaboration based on MacKay et al. (2019), Martin et al. (2018), and Góralczyk-Bińkowska et al. (2022).

It should be emphasized, however, that most of these neurotransmitters do not directly cross the blood–brain barrier, and their effects on the central nervous system occur indirectly (Cao et al., 2021; Mayer, 2011). These mechanisms include vagus nerve activation, modulation of the immune system, and effects on the hypothalamic–pituitary–adrenal (HPA) axis (Cao et al., 2021; Mayer, 2011). In particular, the vagus nerve plays a crucial role in transmitting signals from the gastrointestinal tract to the brain, as confirmed by experimental studies in which the effects of certain bacterial strains disappeared following vagotomy (Carabotti et al., 2015). Additionally, metabolites produced by the gut microbiota, such as short-chain fatty acids, alter neuronal function and neuroinflammatory processes, thereby indirectly affecting central nervous system activity (Pelczarska et al., 2024). The influence of gut microorganisms on the nervous system also

involves regulation of neurotransmitter receptor expression and modulation of synaptic plasticity, which may be relevant to the pathogenesis of anxiety disorders (Cao et al., 2021).

Therefore, the ability of the gut microbiota to produce and modulate neuroactive compounds represents an important component of the complex interaction network within the gut–brain axis, integrating metabolic, immunological, and neuronal signaling pathways (Martin et al., 2018; Cao et al., 2021).

3.2. Factors Influencing Gut Microbiota Composition

The composition of the gut microbiota is not static and may change under the influence of numerous environmental and lifestyle-related factors (Sarkar et al., 2018). One of the most important modulators of the intestinal microbial community is diet, which significantly affects both the diversity and abundance of microorganisms inhabiting the gastrointestinal tract. Long-term dietary habits have been shown to induce persistent alterations in gut microbiota composition, thereby shaping its functional profile (Merlo et al., 2024).

A diet rich in dietary fiber promotes the growth of carbohydrate-fermenting bacteria and enhances the production of short-chain fatty acids (SCFAs), whereas a diet low in fiber and high in fat may contribute to unfavorable alterations in the composition of gut microorganisms (Patil et al., 2025). SCFAs, particularly butyrate, play a crucial role in maintaining intestinal barrier integrity, regulating inflammatory processes, and influencing host metabolism (Mayer, 2011). In contrast, highly processed diets may reduce gut microbiota diversity and increase the abundance of bacteria with pro-inflammatory potential (Patil et al., 2025; Spiller et al., 2025).

Pharmacological agents also exert a substantial influence on the gut microbial community, especially antibiotics. Their use may lead to a reduction in microbial diversity and disruption of the balance between individual bacterial species (Spiller et al., 2025; Jernberg et al., 2010). Such disturbances may persist long after the completion of therapy and may promote colonization of the gastrointestinal tract by pathogenic microorganisms (Spiller et al., 2025; Jernberg et al., 2010). Additionally, other classes of medications, including proton pump inhibitors and nonsteroidal anti-inflammatory drugs, have also been associated with alterations in gut microbiota composition (Skonieczna-Żydecka et al., 2023).

Other important factors influencing the gut microbial ecosystem include age, mode of delivery, lifestyle, physical activity, and exposure to stress (Sarkar et al., 2018; Robertson et al., 2019). Colonization of the gastrointestinal tract begins during the neonatal period, and both the mode of delivery (vaginal birth versus cesarean section) and feeding method (breastfeeding versus formula feeding) substantially affect the initial composition of the gut microbiota (Robertson et al., 2019). Later in life, factors such as chronic stress and physical inactivity may further contribute to structural and functional alterations within the microbial community (Foster et al., 2017).

The combined effects of these factors may lead to disturbances in gut microbiota composition, which in some cases result in a disruption of microbial balance referred to as dysbiosis (Simpson et al., 2021; Skonieczna-Żydecka et al., 2023). Dysbiosis is characterized by reduced microbial diversity and an imbalance between beneficial and potentially pathogenic bacterial species (Morais et al., 2022).

An increasing number of studies suggest that dysbiosis may play a significant role in the pathogenesis of various diseases, including metabolic disorders, inflammatory diseases, and psychiatric conditions (Fan & Pedersen, 2021). Proposed mechanisms include enhanced inflammatory responses, impaired intestinal barrier function, and alterations in the production of bacterial metabolites affecting the host organism (Morais et al., 2022; Fan & Pedersen, 2021).

3.3. Dysbiosis and Its Significance for Health

Gut microbiota dysbiosis has been described in the course of numerous diseases, including obesity, type 2 diabetes mellitus, inflammatory bowel diseases, and autoimmune disorders (Fan & Pedersen, 2021). In the pathogenesis of these conditions, important mechanisms involve increased intestinal permeability, enhanced inflammatory responses, and alterations in the production of bacterial metabolites (Bischoff, 2021). Disruption of intestinal barrier integrity may facilitate the translocation of bacterial components, such as lipopolysaccharide (LPS), into systemic circulation, leading to activation of the immune system and the development of chronic low-grade inflammation (Bischoff, 2021; Cani et al., 2007).

In recent years, increasing attention has also been directed toward the potential role of dysbiosis in the pathogenesis of psychiatric disorders. Alterations in gut microbiota composition modulate gut–brain axis functioning through modulation of immune responses, production of bacterial metabolites, and interactions with the host nervous system (Morais et al., 2022; Dalile et al., 2019). Particular significance has been

attributed to disturbances in short-chain fatty acid (SCFA) production and alterations in tryptophan metabolism, both of which may affect serotonin synthesis and mood regulation (Dalile et al., 2019; Agus et al., 2018).

Within the context of mental health disorders, special attention has been paid to the potential association between dysbiosis and the occurrence of anxiety and depressive disorders (Simpson et al., 2021; Morais et al., 2022). It has been suggested that changes in gut microbiota composition moderate stress response regulation through modulation of the hypothalamic–pituitary–adrenal (HPA) axis and may also contribute to neuroinflammatory processes within the central nervous system (Skonieczna-Żydecka et al., 2023; Foster et al., 2017). Chronic inflammation and disturbances in neurotransmitter balance may consequently affect the functioning of brain structures involved in emotional regulation, such as the amygdala and hippocampus (Cryan et al., 2020). The relationship between dysbiosis and psychiatric disorders appears bidirectional (Cryan et al., 2020; Dinan & Cryan, 2023).

4. The Gut–Brain Axis

4.1. Definition of the Gut–Brain Axis

The gut–brain axis is a bidirectional communication system between the gastrointestinal tract and the central nervous system, involving complex interactions among the gut microbiota, nervous, immune, and endocrine systems (Martin et al., 2018; Mayer, 2011; Cryan et al., 2019). It functions as a dynamic signaling network integrating intestinal physiology with central nervous system activity (Cryan et al., 2019).

Gut microorganisms exert both local effects within the gastrointestinal tract and systemic effects influencing nervous system function and neurobiological processes (Cryan et al., 2019).

Disturbances in microbiota–brain communication functioning may affect stress response regulation, emotional processes, and behavior (Dinan & Cryan, 2023; Cryan et al., 2019; Farzi et al., 2023; Liu et al., 2021). In particular, impaired communication between the gut and the brain may lead to alterations in the activity of the hypothalamic–pituitary–adrenal (HPA) axis, resulting in dysregulated stress responses (Morais et al., 2022).

A schematic representation of the major communication mechanisms within the gut–brain signaling is presented in Figure 1 (Fig. 1).

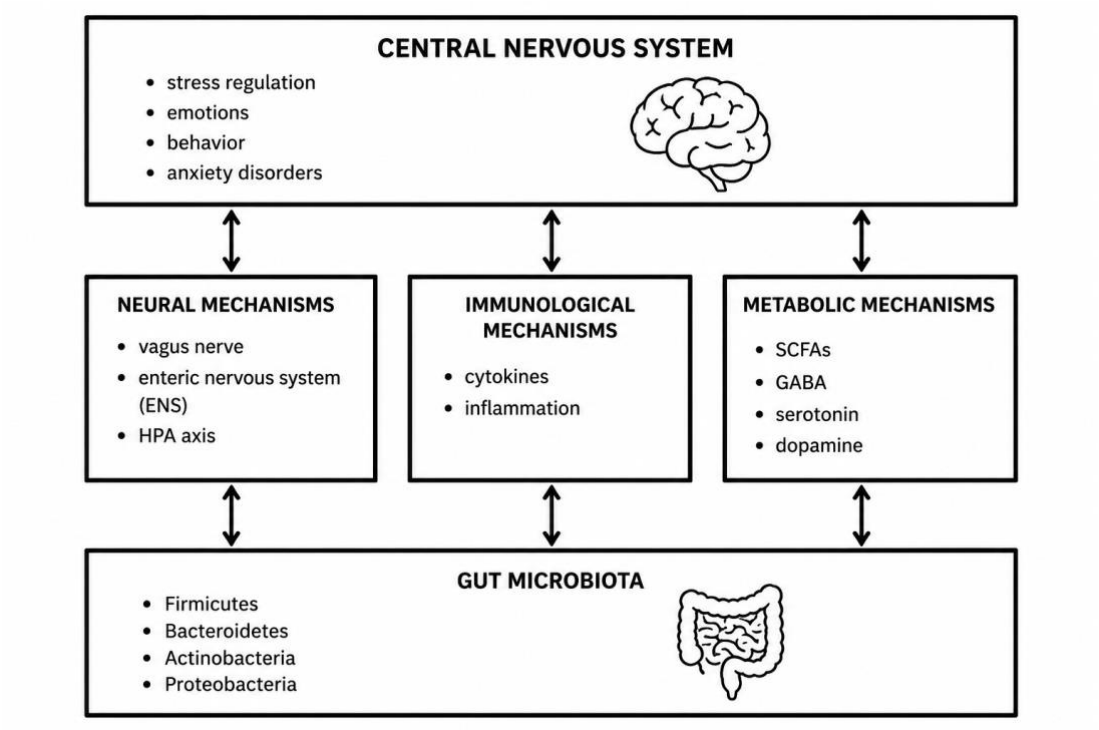

Fig. 1. *Major communication pathways within the gut–brain axis*

Source: authors' own elaboration based on Martin et al. (2018), Mayer (2011), and Cryan et al. (2019).

Communication between the gut microbiota and the central nervous system involves neural, immunological, and metabolic pathways (Cryan et al., 2019). Key components involved in this communication include the vagus nerve, the enteric nervous system (ENS), inflammatory mediators, and bacterial metabolites such as short-chain fatty acids (SCFAs) and neuroactive compounds (Dalile et al., 2019; Cryan et al., 2019).

The neural pathway primarily involves direct communication via the vagus nerve, which transmits signals from the gastrointestinal tract to brain regions responsible for the regulation of emotions and behavior (Cryan et al., 2019). The gastrointestinal tract also possesses its own intrinsic nervous system, referred to as the enteric nervous system (ENS), which interacts closely with the central nervous system and constitutes an important component of the gut–brain axis (Breit et al., 2018; Margolis et al., 2021).

Immunological mechanisms involve the influence of gut microorganisms on immune cell activation and cytokine production, which may affect brain function and neuroinflammatory processes (Margolis et al., 2021). In turn, metabolic mechanisms are associated with the production of bacterial metabolites, including SCFAs, which may modulate neuronal activity, blood–brain barrier integrity, and inflammatory responses (Dalile et al., 2019).

These neural, immunological, and metabolic mechanisms do not function independently but instead form a complex network of interconnected biological processes enabling bidirectional communication between the gut microbiota and the central nervous system (Cryan et al., 2020; Margolis et al., 2021). The integration of these signals plays a key role in maintaining physiological homeostasis and supporting normal cognitive and emotional functioning (Margolis et al., 2021).

4.2. Neural Mechanisms

One of the most important communication mechanisms within the gut–brain axis involves neural signal transmission between the gastrointestinal tract and the central nervous system. A key role in this process is played by the vagus nerve, which constitutes the principal pathway for bidirectional communication between the gut and the brain (Morais et al., 2022). The vagus nerve transmits both afferent signals from the gastrointestinal tract to the brain and efferent signals modulating intestinal function and the organism's response to stress-related stimuli (Morais et al., 2022).

Experimental studies support the importance of this pathway. In a study conducted by Bravo et al. (2011), administration of *Lactobacillus rhamnosus* in mice altered GABA receptor expression in the brain and reduced anxiety-like behaviors, with these effects being dependent on the integrity of the vagus nerve. These findings indicate that microbial signals can modulate vagal nerve activity and thereby influence brain structures involved in emotional regulation and stress responses (Bravo et al., 2011; Strandwitz, 2018).

The gut microbiota may affect vagal activity through the production of neuroactive compounds such as gamma-aminobutyric acid (GABA), serotonin, and dopamine, as well as through interactions with enteroendocrine cells within the gastrointestinal tract (Strandwitz, 2018; Bellono et al., 2017). Through the secretion of hormones and neuropeptides, these cells may activate vagal afferent endings and initiate signal transmission to the central nervous system (Morais et al., 2022; Bellono et al., 2017).

Another important component of communication within the microbiota–brain communication is the enteric nervous system (ENS), often referred to as the "second brain," which functions partially independently of the central nervous system while remaining in continuous interaction with it (Morais et al., 2022; Margolis et al., 2021). The ENS integrates local signals derived from the gut microbiota and the intestinal environment and subsequently transmits this information to the brain via the vagus nerve (Morais et al., 2022; Margolis et al., 2021).

An additional key element involved in gut–brain communication is the hypothalamic–pituitary–adrenal (HPA) axis, which plays a central role in the regulation of the stress response (Sudo et al., 2004). Animal studies have demonstrated that alterations in gut microbial composition modulate HPA axis activity and the levels of stress hormones, including cortisol (Sudo et al., 2004). In particular, germ-free animals have been shown to exhibit exaggerated stress responses, which were normalized following colonization with specific bacterial strains (Sudo et al., 2004).

Dysregulation of the HPA axis is frequently observed in patients with anxiety and depressive disorders, suggesting a possible role of gut microorganisms in the modulation of stress responses (Sah et al., 2024). Chronic HPA axis activation has been associated with functional and structural alterations in brain regions such as the hippocampus and amygdala, which are critically involved in emotional regulation (Sah et al., 2024; Rosas-Sánchez et al., 2025).

These neural mechanisms do not operate in isolation but remain closely interconnected with immunological and metabolic processes, forming a complex regulatory network within the microbiota-mediated communication (Morais et al., 2022; Rosas-Sánchez et al., 2025).

4.3. Immunological Mechanisms

Immunological pathways represent one of the major communication mechanisms within the gut–brain axis. (Hooper et al., 2012; Zheng et al., 2020; Fung et al., 2017; Yoo & Mazmanian, 2017). Gut microorganisms interact with the immune system through modulation of immune cell activity as well as through the production of cytokines and other inflammatory mediators (Hooper et al., 2012; Zheng et al., 2020). The gut microbiota influences the function of T lymphocytes, dendritic cells, and macrophages, thereby regulating the balance between pro-inflammatory and anti-inflammatory immune responses (Zheng et al., 2020; Fung et al., 2017; Yoo & Mazmanian, 2017).

Cytokines produced in response to alterations in gut microbiota composition may affect central nervous system functioning by influencing neuroinflammatory processes and neuronal activity (Morais et al., 2022; Erny et al., 2015). Chronic low-grade inflammation is considered an important factor in the pathogenesis of anxiety disorders through modulation of stress responses and the functioning of brain structures responsible for emotional regulation (Hodes et al., 2015; Dantzer, 2022; Rea et al., 2016; Thion et al., 2018). Particular importance has been attributed to pro-inflammatory cytokines such as interleukin-6 (IL-6) and tumor necrosis factor alpha (TNF-α), both of which may significantly influence central nervous system activity (Hodes et al., 2015; Dantzer, 2022).

These cytokines influence the central nervous system both directly, by crossing the blood–brain barrier, and indirectly, through activation of glial cells, including microglia (Morais et al., 2022; Dantzer, 2022). Microglial activation contributes to the enhancement of neuroinflammatory processes and alterations in neuronal functioning, potentially affecting synaptic plasticity as well as the regulation of mood and behavior (Thion et al., 2018; Camilleri, 2019).

The gut microbiota also plays a crucial role in maintaining intestinal barrier integrity. Disruption of this barrier may result in increased intestinal permeability and translocation of bacterial components into systemic circulation (Camilleri, 2019). This phenomenon, commonly referred to as “leaky gut,” facilitates the passage of lipopolysaccharide (LPS) and other bacterial products into the bloodstream, leading to activation of the immune system and amplification of systemic inflammatory responses (Camilleri, 2019; Kelly et al., 2017).

Chronic activation of the immune system impact tryptophan metabolism through activation of the kynurenine pathway, resulting in reduced serotonin availability and the formation of neuroactive metabolites capable of affecting brain function (Gao et al., 2020). These disturbances may play a significant role in the pathogenesis of mood and anxiety disorders (Gao et al., 2020).

Gut microorganisms also contribute to the regulation of anti-inflammatory responses through their effects on regulatory T cells (Treg), which are responsible for maintaining immune tolerance and limiting excessive inflammatory reactions (Morais et al., 2022).

4.4. Metabolic Mechanisms

An important component of communication within the gut–brain axis involves metabolites produced by gut microorganisms, which shape the functioning of the central nervous system (Morais et al., 2022; Dalile et al., 2019). Short-chain fatty acids (SCFAs) are among the most important microbial metabolites, including acetate, propionate, and butyrate, which are generated during the fermentation of indigestible dietary components (Koh et al., 2016; Cryan et al., 2019).

SCFAs contribute to the maintenance of intestinal barrier integrity and exhibit anti-inflammatory properties that influence immune system function (Dalile et al., 2019). They also participate in gut–brain communication through neuroimmunological and metabolic mechanisms (Morais et al., 2022; Dalile et al., 2019). SCFAs may affect the nervous system by modulating microglial activity and regulating the expression of genes associated with neuroplasticity (Braniste et al., 2014). Furthermore, studies have demonstrated that SCFAs moderate blood–brain barrier permeability, thereby modulating the transport of substances between systemic circulation and the central nervous system (Braniste et al., 2014).

Among SCFAs, butyrate has attracted particular attention due to its pleiotropic biological effects and its potential influence on central nervous system functioning (Dalile et al., 2019; Braniste et al., 2014).

Butyrate is one of the most important short-chain fatty acids produced by intestinal bacteria such as *Faecalibacterium prausnitzii* and *Roseburia* spp. (Koh et al., 2016). It constitutes the primary energy source

for colonocytes and plays a crucial role in maintaining intestinal barrier integrity (Koh et al., 2016; Louis & Flint, 2017).

In addition, butyrate may influence the central nervous system through its effects on glial cells and neurons, as well as through regulation of gene expression associated with neuroplasticity, partly via inhibition of histone deacetylases (HDACs) (Stilling et al., 2016; Davie, 2003). Consequently, it may affect epigenetic processes involved in the regulation of brain function and stress responses (Stilling et al., 2016; Davie, 2003).

The gut microbiota is also capable of producing neuroactive compounds such as gamma-aminobutyric acid (GABA), serotonin, dopamine, and noradrenaline (Silva et al., 2020). These substances impact neuronal function as well as the regulation of emotional processes and stress responses (Morais et al., 2022; Dalile et al., 2019). Although most of these neurotransmitters do not directly cross the blood–brain barrier, they may affect the nervous system indirectly through activation of intestinal receptors, modulation of immune system activity, and interactions with the vagus nerve (Bonaz et al., 2018).

An important role is also played by metabolites generated during tryptophan metabolism, which regulate serotonergic signaling and the kynurenine pathway (Gao et al., 2020; Xu et al., 2025; Zhao et al., 2025). Alterations in tryptophan metabolism may lead to disturbances in neurotransmitter balance and the formation of neurotoxic or neuroprotective compounds, potentially contributing to the pathogenesis of anxiety and depressive disorders (Liu et al., 2021; Gao et al., 2020; Zhao et al., 2025).

The gut microbiota may additionally affect the endocrine system through modulation of intestinal hormone secretion, including glucagon-like peptide-1 (GLP-1) and peptide YY (PYY), both of which are involved in the regulation of appetite and metabolism and may also influence central nervous system functioning (Grasset et al., 2017; Abildinova et al., 2024).

Metabolites produced by gut microorganisms may exert effects on the nervous system both directly and indirectly through modulation of immune responses and activation of neural pathways, including the vagus nerve (Grasset et al., 2017; Masi et al., 2024).

4.5. Gut Microbiota and Anxiety Disorders

An increasing body of evidence suggests that the gut microbiota may play an important role in the pathogenesis of anxiety disorders through complex interactions involving neurobiological, immunological, and endocrine mechanisms (Zeng et al., 2023; Bautista et al., 2025; Jiang et al., 2024; Butler et al., 2019). Alterations in gut microbial composition may lead to disturbances in gut–brain axis functioning, thereby affecting emotional regulation, stress responses, and behavior (Zeng et al., 2023; Nikel et al., 2025).

One of the key mechanisms linking the gut microbiota with anxiety disorders involves modulation of the hypothalamic–pituitary–adrenal (HPA) axis. Dysbiosis may contribute to excessive activation of the HPA axis, resulting in increased secretion of stress hormones, including cortisol, which may promote heightened stress reactivity and the persistence of anxiety-related responses (Bautista et al., 2025). Dysregulation of the HPA axis is frequently observed in patients with anxiety disorders and constitutes an important element of their pathophysiology (Jiang et al., 2024; Eskandar, 2025).

Neurochemical mechanisms associated with the production and regulation of neurotransmitters also play a substantial role in this relationship. Alterations in gut microbial composition may affect the availability of gamma-aminobutyric acid (GABA), serotonin, and dopamine, all of which participate in mood regulation and emotional processing (Liu et al., 2021). Reduced GABA signaling and disturbances within the serotonergic system may contribute to increased neuronal excitability and the exacerbation of anxiety symptoms (Liu et al., 2021; Bautista et al., 2025).

Another important aspect is the influence of the gut microbiota on immunological and neuroinflammatory processes. Dysbiosis may promote chronic low-grade inflammation associated with increased production of pro-inflammatory cytokines such as interleukin-6 (IL-6) and tumor necrosis factor alpha (TNF-α) (Jiang et al., 2024). These cytokines may affect the central nervous system by influencing the functioning of brain structures involved in emotional regulation, including the amygdala and hippocampus (Jiang et al., 2024; Nikolova et al., 2024).

Bacterial metabolites, particularly short-chain fatty acids (SCFAs), are also of considerable importance, as they participate in the regulation of neuroimmunological processes and influence synaptic plasticity (Ullah et al., 2023). Disturbances in SCFA production may lead to alterations in central nervous system functioning and increased susceptibility to the development of anxiety disorders (Jiang et al., 2024).

Another important mechanism linking the gut microbiota with brain function involves tryptophan metabolism (Jiang et al., 2024; Miyamoto et al., 2024). Microbial imbalance may shift tryptophan metabolism

toward the kynurenine pathway, resulting in reduced serotonin availability and the production of neuroactive metabolites (Miyamoto et al., 2024; Hou et al., 2023). These processes participate in mood regulation and increase the risk of anxiety-related symptoms (Hou et al., 2023).

Together, these findings indicate that gut microbiota disturbances are likely involved in the pathogenesis of anxiety disorders, although the precise mechanisms underlying these interactions remain the subject of intensive ongoing research (Liu et al., 2021; Rea et al., 2016). Better understanding of these mechanisms may support future therapeutic development (Jiang et al., 2024).

5. Studies on Gut Microbiota in Anxiety Disorders

In recent years, there has been a rapid increase in research investigating the relationship between the gut microbiota and anxiety disorders. These studies include both preclinical investigations using animal models and clinical studies conducted in human populations (Bautista et al., 2025; Jiang et al., 2024). Preclinical studies primarily focus on the effects of gut microbiota manipulation on behavior, stress responses, and nervous system functioning, whereas clinical studies investigate differences in microbiota composition among patients and evaluate the effects of interventions such as probiotics, dietary modifications, prebiotics, and fecal microbiota transplantation (FMT) (Simpson et al., 2021; Liu et al., 2021).

Increasing evidence also indicates that alterations in gut microbiota composition may be associated not only with the occurrence but also with the severity of anxiety symptoms (Simpson et al., 2021; Jiang et al., 2024; Eskandar, 2025).

5.1. Preclinical Studies

Preclinical studies provided the first evidence supporting the relationship between gut microorganisms and central nervous system functioning. Animal models demonstrated that germ-free animals exhibit altered stress responses and anxiety-like behavioral disturbances (Sah et al., 2024; Clarke et al., 2013). These animals were found to display exaggerated hypothalamic–pituitary–adrenal (HPA) axis activity, altered cortisol responses, and changes in neurotransmitter levels, indicating a critical role of the gut microbiota in stress regulation (Cryan et al., 2019; Clarke et al., 2013).

Experimental studies have also shown that alterations in gut microbiota composition may affect the development and functioning of brain structures involved in emotional regulation, particularly the limbic system (Clarke et al., 2013). Germ-free animals exhibited changes in the expression of genes associated with neuroplasticity and serotonergic signaling, especially within the hippocampus and amygdala (Margolis et al., 2021; Clarke et al., 2013). Some studies additionally demonstrated reduced expression of brain-derived neurotrophic factor (BDNF), a molecule essential for neuronal survival and synaptic plasticity (Clarke et al., 2013).

One of the strongest arguments supporting the role of gut microbiota in behavioral regulation originates from studies involving fecal microbiota transplantation (FMT). Transfer of microbiota from animals displaying anxiety-like or depressive behaviors induced similar behavioral disturbances in recipient animals, suggesting the possibility of transferring behavioral phenotypes through gut microbial communities (Morais et al., 2022; Zheng et al., 2020; Kelly et al., 2017). Among currently available preclinical findings, germ-free animal models and fecal microbiota transplantation experiments provide some of the strongest evidence supporting a functional role of gut microbiota in behavioral regulation. The transfer of anxiety-like phenotypes through microbiota transplantation strongly suggests that gut microorganisms may actively contribute to neurobehavioral processes rather than merely accompany them.

Experimental studies have further demonstrated that administration of specific bacterial strains may significantly influence nervous system functioning. A notable example is *Lactobacillus rhamnosus*, which was shown to modulate GABA receptor expression and reduce anxiety-like behavior in a vagus nerve-dependent manner (Bravo et al., 2011). Similar anxiolytic effects were observed for other probiotic strains, including *Bifidobacterium longum*, which was associated with reduced stress responses and modulation of HPA axis activity (Liu et al., 2021; Bercik et al., 2011).

Additional studies revealed that manipulation of gut microbiota composition through antibiotics, dietary interventions, or probiotic supplementation may lead to substantial behavioral and neurochemical changes in animal models (Bercik et al., 2011; Bailey et al., 2004). Depending on the type of intervention and the resulting microbial composition, these alterations were associated with either increased or decreased anxiety-like behaviors (Rea et al., 2016; Bercik et al., 2011). Antibiotic-induced dysbiosis has been linked to impaired

social behavior, altered neurotransmitter signaling, and increased inflammatory responses within the central nervous system (Bailey et al., 2004).

Recent experimental studies have also suggested that gut microbiota regulate myelination processes, microglial maturation, and synaptic remodeling, further emphasizing its role in brain development and emotional regulation (Zeng et al., 2023; Butler et al., 2019). Moreover, chronic stress itself has been shown to alter gut microbial composition in animal models, supporting the concept of a bidirectional relationship between stress-related disorders and gut microbiota disturbances (Jiang et al., 2024).

Mechanistic evidence linking gut microbiota with anxiety-related behavior remains substantially stronger in animal models than in human studies. While preclinical findings consistently demonstrate microbiota-mediated effects on stress responses and neurobiology, translation of these observations into clinically meaningful human outcomes remains limited (Morais et al., 2022; Rea et al., 2016).

5.2. Clinical Studies

Clinical studies increasingly support a relationship between gut microbiota composition and anxiety disorders. Research has demonstrated that individuals with anxiety disorders may exhibit a distinct microbial profile compared with healthy controls (Simpson et al., 2021; Morais et al., 2022). These differences involve not only alterations in microbial composition but also changes in the metabolic activity of gut microorganisms (Morais et al., 2022).

The majority of available clinical evidence derives from observational cohort studies, case–control studies, randomized controlled trials, and systematic reviews published between 2010 and 2025. Most studies included adult populations diagnosed with generalized anxiety disorder (GAD), social anxiety disorder (SAD), panic disorder (PD), mixed anxiety-depressive disorders, or stress-related symptoms. Gut microbiota composition was most commonly assessed using 16S rRNA gene sequencing, metagenomic analysis, or stool microbiome profiling techniques.

Several analyses demonstrated reduced microbial diversity and alterations in the abundance of selected bacterial taxa among patients with anxiety disorders (Jiang et al., 2015). In particular, reduced levels of bacteria with potential anti-inflammatory properties, such as *Faecalibacterium* and *Bifidobacterium*, together with increased abundance of microorganisms associated with inflammatory processes, have been observed (Jiang et al., 2015; Morais et al., 2022). Importantly, no consistent microbiota profile specific to anxiety disorders has yet been identified. Different studies report variable alterations in bacterial taxa, which may result from differences in study populations, sequencing methods, dietary patterns, psychiatric comorbidities, and medication exposure.

Some studies additionally reported alterations in bacterial metabolites, including short-chain fatty acids (SCFAs), as well as disturbances in tryptophan metabolism among individuals with anxiety symptoms (Dalile et al., 2019; Gao et al., 2020).

However, it remains unclear whether observed alterations in gut microbiota composition may represent either causal mechanisms or secondary consequences of psychiatric disorders, making interpretation of clinical findings particularly challenging (Simpson et al., 2021; Jiang et al., 2015). One of the major unresolved issues is whether gut microbiota alterations constitute a causal factor in anxiety disorders or merely reflect secondary consequences of chronic stress, altered dietary habits, medication use, and lifestyle-related factors. Although associations between dysbiosis and anxiety symptoms are frequently reported, establishing direct causality in human populations remains particularly challenging. Factors such as diet, lifestyle, pharmacotherapy, sleep quality, substance use, and chronic stress may additionally influence gut microbial composition and therefore constitute important confounding variables in clinical analyses (Morais et al., 2022). Dietary habits remain one of the most important confounding variables in microbiome research. Since diet profoundly influences gut microbial composition, it is often difficult to determine whether observed microbiota alterations are associated with psychiatric pathology itself or reflect nutritional and lifestyle differences among patients.

An increasing number of studies have also investigated the effects of microbiota-targeted interventions. Randomized controlled trials have demonstrated that probiotic supplementation may contribute to improvements in psychological functioning, reductions in stress levels, and attenuation of anxiety symptoms (Mayer, 2011; Liu et al., 2021; Messaoudi et al., 2011). In several studies, probiotic administration was associated with reduced perceived stress, improved mood, and decreased cortisol concentrations, suggesting potential modulation of gut–brain signaling mechanisms (Liu et al., 2021).

The observed effects, however, were heterogeneous and depended on factors such as the bacterial strain used, duration of intervention, dosage, and characteristics of the study population (Liu et al., 2021; Szczygieł

& Samochowiec, 2019). Beneficial effects were most frequently reported for strains belonging to the genera *Lactobacillus* and *Bifidobacterium*, which are often referred to as "psychobiotics" due to their potential influence on mental health (Mayer, 2011).

Certain studies additionally demonstrated that probiotic supplementation participate in biological parameters associated with stress and inflammation, including cortisol levels, C-reactive protein (CRP), and pro-inflammatory cytokines (Liu et al., 2021; Jiang et al., 2024). These findings indicate that microbiota-targeted therapies could influence through modulation of neuroendocrine and immunological pathways involved in anxiety disorders.

At the same time, available findings remain inconsistent, and some clinical trials failed to demonstrate significant therapeutic effects (Liu et al., 2021). Methodological limitations represent an important challenge in this field of research. Many studies involve relatively small sample sizes, short follow-up periods, heterogeneous diagnostic criteria, and variability in probiotic formulations and microbiota assessment methods (Jiang et al., 2024; Szczygieł & Samochowiec, 2019). Moreover, differences in dietary habits, medication use, and baseline microbiota composition may substantially affect study outcomes.

Recent systematic reviews and meta-analyses indicate that although microbiota-targeted interventions appear promising, current evidence remains insufficient to establish definitive clinical recommendations (Zeng et al., 2023; Jiang et al., 2024).

Overall, current evidence supports the existence of biologically plausible links between gut microbiota and anxiety disorders; however, the field remains in an early stage of clinical translation. Preclinical studies provide robust mechanistic evidence, particularly regarding HPA axis modulation, neuroinflammation, and microbial metabolite signaling. In contrast, human studies remain heterogeneous and frequently limited by methodological variability, small sample sizes, and insufficient control of confounding factors. Consequently, although microbiota-targeted interventions appear promising, their clinical utility in psychiatry has not yet been definitively established (Simpson et al., 2021; Liu et al., 2021).

6. Limitations of Available Evidence

Despite the growing interest in the role of gut microbiota in the pathogenesis of anxiety disorders, the currently available scientific evidence still has numerous methodological and interpretative limitations. Many available studies are observational, which makes it difficult to establish clear causal relationships between alterations in gut microbiota composition and the occurrence of anxiety disorders (Simpson et al., 2021; Liu et al., 2021). It remains unclear whether the observed dysbiosis is a cause of psychiatric disorders, a consequence of them, or the result of coexisting environmental and metabolic factors (Morais et al., 2022; Jiang et al., 2024).

Another important limitation is the high heterogeneity of the available studies. Individual analyses differ in terms of sample size, diagnostic criteria, participant characteristics, methods used to assess gut microbiota, and the type of analyzed interventions (Liu et al., 2021; Zeng et al., 2023). Many clinical studies included relatively small patient groups, limiting the generalizability of the findings to the broader population (Liu et al., 2021).

An additional issue is the lack of standardization in microbiota assessment methods. Differences concern, among others, biological sample collection, sequencing techniques, and bioinformatic data analysis (Bautista et al., 2025). This makes direct comparison between studies difficult and complicates the identification of microbiological patterns specifically associated with anxiety disorders (Jiang et al., 2024).

Moreover, gut microbiota composition is influenced by numerous confounding factors, including diet, medication use, physical activity, comorbidities, stress, and lifestyle (Morais et al., 2022). These factors are often insufficiently controlled in clinical studies, which may affect the obtained results and their interpretation (Zeng et al., 2023). Antibiotics, probiotics, and psychotropic medications are of particular importance, as they may substantially alter the composition of gut microbiota (Jernberg et al., 2010; Foster et al., 2017).

Limitations also apply to preclinical studies. Although animal models provide valuable insights into the mechanisms of the gut–brain axis, their direct translation to humans remains limited due to species-specific differences in microbiota composition, nervous system organization, and immune responses (Morais et al., 2022; Rea et al., 2016).

In interventional studies involving probiotics and psychobiotics, the results are frequently inconsistent. Variations in bacterial strains, dosages, duration of treatment, and patient characteristics make it difficult to assess the effectiveness of individual interventions (Mayer, 2011; Liu et al., 2021). Furthermore, most currently available studies involve relatively short follow-up periods, meaning that the long-term effects of gut

microbiota modulation remain insufficiently understood (Liu et al., 2021). Further standardized longitudinal studies are needed to clarify the role of gut microbiota in anxiety disorders.

7. Therapeutic Implications

In recent years, growing interest has been directed toward the potential use of gut microbiota modulation in the treatment of anxiety disorders. Current evidence indicates that modulation of gut microbial composition could become a promising complementary therapeutic strategy for the management of these conditions (Fan & Pedersen, 2021; Winter et al., 2023).

One of the most extensively investigated approaches involves the use of probiotics, commonly referred to as psychobiotics. Psychobiotics are defined as live microorganisms that, when administered in adequate amounts, may exert beneficial effects on mental health through modulation of the gut microbiota and gut–brain signaling (Mayer, 2011). Experimental and clinical studies have demonstrated that selected bacterial strains may contribute to the reduction of anxiety symptoms, improvement of psychological well-being, and attenuation of stress-related responses (Mayer, 2011; Hou et al., 2022).

The most frequently studied psychobiotic strains belong to the genera *Lactobacillus* and *Bifidobacterium*. Some studies reported that supplementation with *Lactobacillus rhamnosus*, *Bifidobacterium longum*, or multi-strain probiotic formulations was associated with reductions in anxiety scores, perceived stress levels, and cortisol concentrations (Liu et al., 2021; Cryan et al., 2019). Potential mechanisms include modulation of neurotransmitter production, reduction of systemic inflammation, improvement of intestinal barrier integrity, and regulation of hypothalamic–pituitary–adrenal (HPA) axis activity (Cryan et al., 2019; Jiang et al., 2024). Although psychobiotics represent a promising therapeutic concept, currently available clinical effects appear relatively modest and inconsistent across studies. Positive findings are frequently derived from small-scale trials, whereas larger randomized studies often fail to demonstrate clinically significant improvements.

Nevertheless, available findings remain heterogeneous. Differences in probiotic strains, dosages, treatment duration, study populations, and assessment methods contribute to variability in clinical outcomes (Liu et al., 2021; Messaoudi et al., 2011). Additionally, many studies involve relatively small sample sizes and short follow-up periods, limiting the generalizability of current evidence.

Diet also plays a major role in shaping gut microbial composition and may therefore influence mental health outcomes. Diets rich in dietary fiber, polyphenols, and fermented foods promote the growth of beneficial microorganisms and enhance the production of short-chain fatty acids (SCFAs), which may positively affect microbiota–brain communication functioning (Morais et al., 2022). In contrast, highly processed diets rich in saturated fats and refined sugars may contribute to dysbiosis, increased intestinal permeability, and enhanced inflammatory responses (Morais et al., 2022).

Particular attention has recently been given to dietary patterns such as the Mediterranean diet, which has been associated with reduced inflammation, greater microbial diversity, and improved mental health outcomes in several observational studies (Jiang et al., 2024; Butler et al., 2019).

Potential therapeutic strategies also include the use of prebiotics, synbiotics, and fecal microbiota transplantation (FMT). Prebiotics are non-digestible compounds that selectively stimulate the growth of beneficial microorganisms, whereas synbiotics combine probiotics with prebiotic substrates (Liu et al., 2021). Preliminary studies suggest that these interventions may positively influence stress responses and emotional functioning, although evidence regarding their effectiveness in anxiety disorders remains limited (Liu et al., 2021).

Fecal microbiota transplantation has emerged as another promising but still experimental therapeutic approach. Although FMT has demonstrated efficacy in gastrointestinal disorders such as recurrent *Clostridioides difficile* infection, its application in psychiatric disorders remains under investigation (Kelly et al., 2017). Experimental studies suggest that transplantation of healthy microbiota regulate behavior and stress responses; however, clinical evidence in anxiety disorders is currently insufficient to support routine therapeutic use (Kelly et al., 2017).

In the context of future research directions, increasing attention is being devoted to personalized medicine approaches that consider the individual gut microbiota profile of each patient (Bischoff, 2021). Such strategies may enable the selection of optimal therapeutic interventions, including specific probiotic strains, dietary modifications, or combined microbiota-targeted therapies tailored to individual biological characteristics (Cani et al., 2007).

Advances in microbiome sequencing technologies, metabolomics, and systems biology may facilitate the identification of microbial biomarkers associated with anxiety disorders and treatment response.

Despite promising findings, microbiota-targeted interventions in anxiety disorders remain largely experimental. Before routine clinical implementation, further high-quality randomized controlled trials are needed involving larger patient populations, standardized therapeutic protocols, and long-term follow-up (Foster et al., 2017; Fan & Pedersen, 2021). Future studies should also focus on identifying the specific mechanisms underlying microbiota–brain interactions and determining which patient populations may benefit most from these therapeutic strategies.

8. Conclusions

Anxiety disorders represent a major public health concern, and their pathogenesis is multifactorial, involving complex interactions among genetic, environmental, and neurobiological factors.

Available evidence suggests that gut microorganisms alter central nervous system activity through neural, immunological, and metabolic mechanisms, thereby affecting stress response regulation, emotional processes, and behavior. Disturbances in microbial balance may contribute to the development of anxiety disorders, supporting the hypothesis that dysbiosis may play a role in their pathogenesis.

Preclinical studies provide substantial evidence supporting the relationship between gut microbiota and anxiety-related behavior, whereas clinical studies indicate possible differences in gut microbial composition among patients with anxiety disorders. At the same time, it should be emphasized that currently available data remain limited, and study findings are not always consistent.

Growing interest has also emerged regarding the use of microbiota-targeted interventions, such as probiotics, prebiotics, dietary modifications, and other gut microbiota modulation strategies, as supportive approaches in the treatment of anxiety disorders. Although preliminary findings appear promising, these methods require further well-designed clinical studies to confirm their efficacy, safety, and long-term therapeutic value.

In summary, the gut microbiota represents an important, although still incompletely understood, component of the pathogenesis of anxiety disorders. Improved understanding of the mechanisms underlying microbiota–brain communication interactions may contribute to the development of novel diagnostic and therapeutic strategies in psychiatry in the future.

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