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CONNECTION BETWEEN GUT DYSBIOSIS AND GENERALIZED ANXIETY DISORDER: A NARRATIVE REVIEW

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ABSTRACT

Objective: This narrative review aims to summarize current evidence regarding the relationship between gut dysbiosis and Generalized Anxiety Disorder (GAD), with particular emphasis on alterations in gut microbiota composition, proposed biological mechanisms, and potential microbiome-targeted therapeutic strategies.

Methods: A narrative review was conducted using thirteen peer-reviewed publications, including systematic reviews, narrative reviews, mechanistic studies, and clinical investigations examining associations between gut microbiota and anxiety disorders. The selected studies were analyzed to identify recurring microbiome-related findings, proposed mechanisms linking gut dysbiosis and anxiety, and evidence regarding microbiome-based interventions.

Results: The reviewed literature consistently demonstrated significant associations between gut dysbiosis and anxiety disorders. Patients with GAD and related anxiety conditions frequently exhibited reduced microbial diversity, depletion of beneficial short-chain fatty acid-producing bacteria, and increased abundance of potentially pro-inflammatory taxa. Several studies highlighted disturbances in microbial metabolite production, neurotransmitter regulation, immune function, and stress-response pathways. Clinical investigations further revealed alterations in both microbial composition and microbial metabolic activity among individuals with anxiety disorders. Additionally, microbiome-targeted interventions, including probiotics, psychobiotics, and dietary modification, showed potential benefits in reducing anxiety symptoms, although findings remain heterogeneous.

Conclusions: Current evidence supports a meaningful relationship between gut dysbiosis and anxiety disorders. Alterations in gut microbiota may contribute to anxiety pathophysiology through immune, metabolic, endocrine, and neurochemical mechanisms. However, the predominance of cross-sectional studies and methodological heterogeneity limit causal interpretation. Future longitudinal studies and randomized controlled trials are necessary to clarify causal relationships and evaluate the clinical utility of microbiome-based therapeutic interventions in Generalized Anxiety Disorder.

KEYWORDS

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

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Introduction

Generalized Anxiety Disorder (GAD) is a chronic psychiatric condition characterized by excessive and persistent worry accompanied by somatic and psychological symptoms such as restlessness, muscle tension, fatigue, sleep disturbances, impaired concentration, and irritability. The disorder affects millions of individuals worldwide and represents one of the leading causes of psychological distress and functional impairment. Despite significant advances in psychiatric research, the pathophysiological mechanisms underlying GAD remain incompletely understood.

Traditional explanations of anxiety disorders have focused on abnormalities within neurotransmitter systems, including serotonin, gamma-aminobutyric acid (GABA), dopamine, and norepinephrine. Dysregulation of the hypothalamic–pituitary–adrenal (HPA) axis, increased inflammatory activity, genetic predisposition, and exposure to chronic psychosocial stress are also recognized contributors to anxiety pathogenesis. Nevertheless, these mechanisms fail to fully explain the substantial heterogeneity observed among patients, variations in symptom severity, and differences in treatment response.

Over the past decade, increasing attention has been directed toward the role of gut microbiota in mental health. The human gastrointestinal tract contains a highly diverse microbial ecosystem composed of trillions of microorganisms that participate in numerous physiological processes. Beyond their established functions in digestion and nutrient metabolism, gut microorganisms contribute to immune regulation, maintenance of intestinal barrier integrity, production of bioactive metabolites, and modulation of neural signaling pathways. Growing evidence suggests that disturbances in this microbial ecosystem may influence emotional regulation, stress responsiveness, and psychiatric symptomatology.

The concept of the microbiota–gut–brain axis has emerged as a major framework for understanding interactions between gastrointestinal and psychological health. This bidirectional communication network integrates neural, endocrine, immune, and metabolic pathways, allowing signals generated within the gastrointestinal tract to influence brain function and behavior. Conversely, psychological stress and emotional states may alter gut physiology and microbial composition, creating a dynamic relationship between mental health and gastrointestinal function.

Numerous studies included in the present review have demonstrated associations between gut dysbiosis and anxiety disorders. Jiang et al. reported significant alterations in gut microbial composition among patients with Generalized Anxiety Disorder, including reduced microbial diversity and depletion of beneficial bacterial taxa. Similarly, Butler et al. described characteristic microbial signatures associated with anxiety disorders, while Simpson et al. identified consistent relationships between microbial dysbiosis and psychiatric symptomatology across multiple studies.

Several mechanisms have been proposed to explain the relationship between gut dysbiosis and anxiety. Altered microbial communities may affect neurotransmitter production, immune regulation, inflammatory signaling, intestinal permeability, and stress-response systems. Reduced abundance of short-chain fatty acid-producing bacteria has been repeatedly observed among individuals with anxiety disorders, suggesting that microbial metabolites may play a crucial role in emotional regulation. Furthermore, chronic inflammation resulting from dysbiosis may influence brain function through cytokine-mediated pathways and contribute to the development of anxiety symptoms.

The growing interest in microbiome research has also generated considerable enthusiasm regarding microbiome-targeted therapeutic approaches. Probiotics, prebiotics, psychobiotics, dietary interventions, and other strategies designed to modulate gut microbial composition have demonstrated promising results in preliminary studies. Although current evidence remains insufficient to support widespread clinical implementation, these interventions may eventually complement existing treatments for anxiety disorders.

Given the increasing prevalence of anxiety disorders and the limitations of current therapeutic approaches, investigation of the gut microbiota represents an important area of contemporary psychiatric research. Understanding the relationship between gut dysbiosis and anxiety may contribute to the identification of novel biomarkers, improved risk assessment strategies, and development of innovative therapeutic interventions.

Therefore, the aim of this narrative review is to summarize current evidence regarding the association between gut dysbiosis and Generalized Anxiety Disorder, examine potential biological mechanisms linking microbial alterations and anxiety symptoms, evaluate microbiome-based therapeutic strategies, and identify priorities for future research.

Methodology

Study Design

This study was conducted as a narrative review aiming to synthesize current evidence regarding the relationship between gut dysbiosis and Generalized Anxiety Disorder (GAD). Narrative reviews allow integration of evidence derived from multiple research methodologies, including clinical investigations, observational studies, systematic reviews, and mechanistic analyses. The objective of this review was to summarize current knowledge regarding microbiota alterations observed in anxiety disorders, identify proposed biological mechanisms, evaluate microbiome-targeted interventions, and discuss future research directions.

Literature Selection

The review was based exclusively on thirteen peer-reviewed publications provided by the author. The selected studies included:

- systematic reviews,
- narrative reviews,
- observational case-control studies,
- mechanistic reviews,
- clinical microbiome investigations.

No additional sources were included beyond the articles supplied for this review.

Inclusion Criteria

Studies were included if they:

- Investigated gut microbiota and anxiety disorders.
- Examined Generalized Anxiety Disorder or anxiety-related symptoms.
- Evaluated microbial composition, microbial diversity, microbial metabolites, or microbiome-targeted interventions.
- Were published in peer-reviewed journals.
- Were written in English.

Data Extraction

The following information was extracted from each study:

1. study design,
2. study population,
3. microbiota-related findings,
4. anxiety-related outcomes,
5. proposed biological mechanisms,
6. therapeutic implications.

The findings were synthesized thematically and organized into sections focusing on microbial alterations, microbiota-related mechanisms, therapeutic interventions, and future clinical applications.

Table 1. Characteristics of Studies Included in the Narrative Review

Author (Year)	Study Type	Main Topic	Principal Findings
Simpson et al. (2021)	Systematic review	Anxiety and depression	Consistent associations between gut dysbiosis and psychiatric symptoms.
Nikel et al. (2025)	Narrative review	Anxiety symptoms	Reduced microbial diversity and depletion of SCFA-producing bacteria linked to anxiety.
Ataei et al. (2025)	Narrative review	Gut–brain connection	Comprehensive overview of microbial influences on mental health.
Jiang et al. (2018)	Case-control study	Generalized Anxiety Disorder	Significant alterations in microbial composition and reduced diversity in GAD patients.
Butler et al. (2025)	Narrative review	Anxiety disorders	Characteristic microbial signatures identified across anxiety disorders.
Spivak et al. (2023)	Narrative review	Psychiatric disorders	Gut microbiota implicated in multiple psychiatric conditions including anxiety.
MacKay et al. (2024)	Narrative review	Anxiety, PTSD and OCD	Strong evidence supporting microbiota involvement in anxiety-related disorders.
Jiang et al. (2024)	Mechanistic review	Microbiota–gut–brain axis	Detailed description of pathways connecting gut microbiota and anxiety.
Yang et al. (2019)	Systematic review	Microbiota-targeted interventions	More than half of interventions improved anxiety symptoms.
Shaikh et al. (2025)	Systematic review	Mental health and microbiome	Dysbiosis associated with inflammatory and neurochemical disturbances.
Butler et al. (2023)	Clinical study	Social Anxiety Disorder	Altered microbial composition and function observed in affected individuals.
Krupa-Kotara et al. (2023)	Narrative review	Anxiety and stress disorders	Stress-related dysbiosis may contribute to psychiatric symptoms.
Alli et al. (2022)	Systematic review	Depression and microbiome interventions	Psychobiotics and microbiome modulation demonstrate therapeutic potential.

Results

Alterations in Gut Microbiota Composition in Generalized Anxiety Disorder

The most consistent finding across the reviewed literature was the presence of significant alterations in gut microbiota composition among individuals experiencing anxiety symptoms or diagnosed anxiety disorders. Although specific microbial signatures varied between studies, several recurring patterns emerged.

Jiang et al. (2018) conducted one of the first studies directly examining gut microbiota composition in patients with Generalized Anxiety Disorder. The authors reported significantly reduced microbial richness and diversity among GAD patients compared with healthy controls. Furthermore, individuals with GAD demonstrated substantial shifts in microbial community structure, suggesting that gut dysbiosis may represent an important biological feature of the disorder (Jiang et al., 2018).

These findings were subsequently supported by broader reviews examining anxiety disorders more generally. Butler et al. (2025) concluded that anxiety disorders are frequently associated with reduced abundance of beneficial bacterial taxa and disturbances in microbial metabolic activity. Similarly, Nikel et al. (2025) reported that reduced microbial diversity was among the most frequently observed characteristics of individuals experiencing anxiety symptoms.

Microbial diversity is widely recognized as an indicator of ecosystem stability and resilience. Reduced diversity may impair the ability of microbial communities to maintain physiological homeostasis and respond effectively to environmental stressors. Consequently, diminished diversity may increase vulnerability to inflammatory processes, metabolic disturbances, and alterations in neural signaling pathways involved in emotional regulation (Nikel et al., 2025; Butler et al., 2025).

Several studies identified specific bacterial genera that may be relevant to anxiety pathophysiology. Butler et al. (2025) highlighted recurrent reductions in genera including *Faecalibacterium*, *Coprococcus*, and *Eubacterium* among individuals with anxiety disorders. These microorganisms are recognized producers of short-chain fatty acids and contribute to maintenance of intestinal barrier integrity and regulation of immune responses.

Likewise, Jiang et al. (2018) reported increased abundance of potentially pro-inflammatory taxa alongside reductions in beneficial bacterial groups. Such alterations may promote inflammatory signaling and contribute to neurobiological changes associated with anxiety symptom development.

Butler et al. (2023) further expanded these observations by examining the gut microbiome in individuals with social anxiety disorder. The authors identified significant differences not only in microbial composition but also in microbial functional pathways, suggesting that anxiety disorders may involve both taxonomic and metabolic alterations. These findings indicate that microbial activity may be as important as microbial composition in understanding anxiety-related psychopathology (Butler et al., 2023).

The systematic review conducted by Simpson et al. (2021) synthesized evidence from numerous studies investigating anxiety and depression. The authors concluded that although heterogeneity remains substantial, microbial dysbiosis consistently appears to be associated with psychiatric symptomatology. Importantly, reduced abundance of beneficial microorganisms and altered microbial diversity were among the most frequently reported findings across studies (Simpson et al., 2021).

Taken together, these observations suggest that gut dysbiosis represents a recurring biological feature of anxiety disorders and may contribute to disease pathogenesis through multiple interconnected mechanisms.

Specific Bacterial Taxa Associated with Anxiety Disorders

Although no single microbial signature has been universally identified in Generalized Anxiety Disorder, several bacterial genera have repeatedly emerged as potentially important contributors to anxiety-related psychopathology. The reviewed studies consistently demonstrated that anxiety disorders are associated with a shift from beneficial commensal microorganisms toward microbial communities characterized by reduced metabolic efficiency and increased inflammatory potential.

One of the most frequently reported findings concerns reductions in short-chain fatty acid-producing bacteria. Butler et al. (2025) identified recurrent depletion of genera such as *Faecalibacterium*, *Coprococcus*, and *Eubacterium* across anxiety disorders. These bacteria are widely recognized for their role in producing butyrate and maintaining intestinal homeostasis. Reduced abundance of these microorganisms may contribute to impaired epithelial barrier function and increased susceptibility to inflammatory activation.

Nikel et al. (2025) similarly reported that decreased abundance of beneficial SCFA-producing taxa represents one of the most reproducible microbiome findings in anxiety-related research. According to the

authors, depletion of these microorganisms may influence both immune regulation and neural signaling through reductions in microbial metabolite production.

Jiang et al. (2018) reported increased abundance of several potentially pathogenic bacterial groups in individuals with Generalized Anxiety Disorder. Among the most notable findings was the increased representation of *Escherichia-Shigella*, a bacterial group commonly associated with inflammatory processes. Simultaneously, beneficial bacterial populations appeared depleted relative to healthy controls.

The clinical study conducted by Butler et al. (2023) provided further support for these observations. Individuals with social anxiety disorder demonstrated distinct microbiome profiles compared with healthy participants, suggesting that alterations in microbial communities may extend beyond GAD and represent a broader feature of anxiety disorders. Importantly, the authors observed differences not only in microbial taxonomy but also in functional metabolic pathways, indicating that microbiome-associated changes may influence host physiology through multiple mechanisms.

Simpson et al. (2021) noted that while individual studies reported somewhat different microbial signatures, reduced abundance of beneficial taxa and decreased microbial diversity emerged consistently across investigations. The authors emphasized that methodological differences among studies may partially explain discrepancies regarding specific bacterial genera.

Collectively, the reviewed evidence suggests that anxiety disorders are associated with a microbial ecosystem characterized by reduced abundance of beneficial bacteria and increased prevalence of taxa associated with inflammatory activity. Although additional studies are required to identify disorder-specific microbial signatures, these recurring patterns support the hypothesis that gut dysbiosis may contribute to anxiety pathophysiology.

Microbial Metabolites and Their Role in Anxiety

While microbial composition has received considerable attention, growing evidence suggests that microbial metabolites may represent the primary biological mediators linking gut microbiota and anxiety disorders.

Gut microorganisms produce a wide variety of metabolites capable of influencing host physiology. Among the most extensively studied are short-chain fatty acids (SCFAs), including acetate, propionate, and butyrate. These compounds are generated through bacterial fermentation of dietary fiber and exert numerous effects relevant to both gastrointestinal and mental health.

Nikel et al. (2025) highlighted the importance of SCFAs in maintaining intestinal barrier integrity and regulating inflammatory responses. Reduced abundance of SCFA-producing bacteria may therefore result in lower concentrations of beneficial metabolites and contribute to physiological disturbances associated with anxiety disorders.

Similarly, Butler et al. (2025) emphasized that depletion of butyrate-producing bacteria represents one of the most consistent findings across anxiety-related studies. Butyrate serves as an important energy source for intestinal epithelial cells and contributes to maintenance of gut barrier function. Reduced butyrate availability may increase intestinal permeability and facilitate activation of inflammatory pathways.

Ataei et al. (2025) described microbial metabolites as key mediators of communication between the gastrointestinal tract and the central nervous system. According to the authors, microbial products can influence neural signaling directly or indirectly through interactions with immune and endocrine pathways.

The mechanistic review by Jiang et al. (2024) further highlighted the role of microbial metabolites in regulating neurobiological processes. The authors discussed how microbial compounds may affect neurotransmitter synthesis, neuroinflammation, and blood–brain barrier integrity, thereby influencing emotional regulation and stress responsiveness.

An additional mechanism involves microbial modulation of tryptophan metabolism. Tryptophan serves as a precursor for serotonin, one of the neurotransmitters most strongly implicated in anxiety disorders. Alterations in microbial composition may affect tryptophan availability and influence serotonin-related pathways. Several reviewed studies suggested that disturbances in these processes may contribute to anxiety symptom development (Jiang et al., 2024; Ataei et al., 2025).

Together, these findings indicate that microbial metabolites represent a crucial mechanistic link between gut dysbiosis and anxiety disorders. The biological effects of microbial products may extend far beyond the gastrointestinal tract and influence multiple systems involved in emotional regulation.

Inflammation and Immune Dysregulation

Inflammation represents one of the most widely proposed mechanisms connecting gut dysbiosis and psychiatric disorders. Numerous studies included in this review highlighted interactions between altered microbial composition, immune activation, and anxiety symptoms.

According to Shaikh et al. (2025), dysbiosis is frequently associated with increased abundance of pro-inflammatory bacterial taxa and reduced representation of beneficial microorganisms involved in immune regulation. Such alterations may contribute to chronic low-grade inflammation, which has increasingly been recognized as a feature of psychiatric disorders.

One proposed mechanism involves increased intestinal permeability. Reduced abundance of bacteria that support epithelial barrier integrity may allow bacterial products to enter systemic circulation more readily. These microbial components can stimulate immune responses and promote production of inflammatory mediators.

Krupa-Kotara et al. (2023) emphasized that chronic stress itself may influence gut microbial composition and contribute to inflammatory processes. This observation suggests the existence of a bidirectional relationship in which stress promotes dysbiosis while dysbiosis amplifies stress-related physiological responses.

MacKay et al. (2024) similarly described immune pathways as central components of microbiota-related anxiety mechanisms. The authors highlighted evidence suggesting that microbial alterations may influence cytokine production and immune signaling pathways involved in emotional regulation.

The interaction between inflammation and anxiety appears particularly important because immune mediators can influence brain function through multiple routes. Cytokines may alter neurotransmitter metabolism, affect neuroplasticity, and modify stress-response systems. Consequently, chronic inflammatory activation may contribute directly to anxiety symptom development and persistence.

Ataei et al. (2025) argued that interactions among microbiota, immunity, and the nervous system represent one of the most promising frameworks for understanding the biological basis of anxiety disorders. Rather than functioning independently, microbial, immune, endocrine, and neural pathways appear to form an integrated network influencing psychological well-being.

Taken together, the reviewed literature strongly supports a role for immune dysregulation and inflammatory signaling in the relationship between gut dysbiosis and anxiety disorders.

Neurotransmitter Regulation and the Microbiota–Gut–Brain Axis

One of the most frequently discussed mechanisms linking gut dysbiosis and anxiety disorders involves microbial influences on neurotransmitter systems. The reviewed literature suggests that gut microorganisms participate directly and indirectly in the regulation of several neurochemical pathways implicated in anxiety pathophysiology.

According to Jiang et al. (2024), the microbiota–gut–brain axis represents a complex bidirectional communication network integrating neural, endocrine, immune, and metabolic signaling pathways. Through these mechanisms, gut microorganisms can influence central nervous system activity and emotional regulation.

Several bacterial species are capable of producing neurotransmitter-like compounds or influencing host neurotransmitter synthesis. Microbial activity has been linked to regulation of serotonin, dopamine, gamma-aminobutyric acid (GABA), glutamate, and norepinephrine pathways (Jiang et al., 2024; Ataei et al., 2025).

Serotonin has received particular attention because of its central role in anxiety disorders. Although serotonin is traditionally considered a brain neurotransmitter, the majority of serotonin synthesis occurs within the gastrointestinal tract. Microbial communities may influence serotonin availability through modulation of tryptophan metabolism, thereby affecting neural pathways involved in mood and anxiety regulation (Ataei et al., 2025).

Similarly, microbial influences on GABA signaling have attracted considerable interest. GABA represents the primary inhibitory neurotransmitter within the central nervous system and plays a critical role in reducing neuronal excitability. Dysregulation of GABAergic pathways has long been implicated in anxiety disorders. According to several reviews included in this study, alterations in microbial composition may influence GABA-related signaling and contribute to anxiety symptomatology (MacKay et al., 2024; Jiang et al., 2024).

Dopamine-related pathways may also be affected by microbial activity. Although evidence remains less extensive than for serotonin and GABA, reviewed studies suggest that microbial metabolites can influence

dopaminergic signaling through indirect neurochemical mechanisms. Such interactions may affect reward processing, motivation, and stress responsiveness.

Spivak et al. (2023) emphasized that microbial influences on neurotransmitter systems likely represent one component of a broader network of interactions involving immune, endocrine, and neural pathways. Consequently, anxiety disorders should not be understood solely as disorders of neurotransmitter deficiency but rather as conditions arising from complex biological interactions involving multiple physiological systems.

The available evidence therefore suggests that gut dysbiosis may contribute to anxiety symptoms through disturbances in neurochemical signaling, further supporting the biological relevance of the microbiota–gut–brain axis.

The Hypothalamic–Pituitary–Adrenal Axis and Stress Response

The hypothalamic–pituitary–adrenal (HPA) axis constitutes the body's primary neuroendocrine stress-response system and has long been implicated in anxiety disorders. Several studies included in this review highlighted interactions between gut microbiota and HPA-axis regulation.

Chronic activation of the HPA axis is commonly observed in anxiety disorders and results in sustained release of glucocorticoids, particularly cortisol. Although cortisol is essential for adaptation to stress, prolonged activation of stress-response pathways may contribute to psychological distress, immune dysregulation, and neurobiological changes associated with anxiety.

According to Jiang et al. (2024), microbial communities may influence HPA-axis activity through multiple mechanisms. Altered microbial composition can affect immune signaling, inflammatory mediator production, and neural communication pathways, all of which may influence stress responsiveness.

Krupa-Kotara et al. (2023) described chronic stress as both a cause and consequence of microbial dysbiosis. Psychological stress may alter gastrointestinal physiology, intestinal permeability, and microbial composition. In turn, dysbiosis may further amplify stress responses through immune and neuroendocrine mechanisms.

Ataei et al. (2025) similarly emphasized that microbiota-mediated modulation of stress-response systems represents a key component of microbiota–brain communication. The authors proposed that microbial influences on HPA-axis regulation may contribute to the persistence and severity of anxiety symptoms.

These findings support the concept of a bidirectional relationship between psychological stress and gut microbiota. Rather than functioning independently, the HPA axis and gut microbial ecosystem appear to interact continuously, influencing both physiological and psychological health.

Evidence from Clinical Studies

Although many publications included in this review were narrative or systematic reviews, several original clinical investigations provided direct evidence supporting the association between gut dysbiosis and anxiety disorders.

The study conducted by Jiang et al. (2018) remains one of the most important investigations focusing specifically on Generalized Anxiety Disorder. The authors compared gut microbiota profiles of patients with GAD and healthy controls and observed significant reductions in microbial diversity among affected individuals. Furthermore, alterations in specific bacterial taxa suggested a shift toward a dysbiotic microbial ecosystem characterized by increased inflammatory potential (Jiang et al., 2018).

Importantly, the authors reported that some microbiota abnormalities persisted even after symptom improvement. This finding raises the possibility that gut dysbiosis may represent a trait marker associated with anxiety vulnerability rather than merely a consequence of active psychological symptoms.

Additional clinical evidence was provided by Butler et al. (2023), who investigated microbiome composition in individuals with social anxiety disorder. Using advanced sequencing techniques, the authors identified significant differences in both microbial composition and microbial metabolic function compared with healthy controls. These findings demonstrated that anxiety-related microbiome alterations extend beyond GAD and may represent a broader feature of anxiety disorders.

The reviewed clinical studies collectively support the hypothesis that anxiety disorders are associated with measurable biological alterations within the gastrointestinal microbial ecosystem. Although the number of available clinical investigations remains relatively limited, existing findings consistently point toward meaningful microbiota-related differences between affected individuals and healthy populations.

Microbiome-Based Therapeutic Interventions

Growing recognition of microbiota involvement in psychiatric disorders has stimulated interest in microbiome-targeted therapeutic strategies. Several studies included in this review evaluated interventions aimed at modifying gut microbial composition and examined their effects on anxiety symptoms.

The most comprehensive analysis was provided by Yang et al. (2019), who systematically reviewed interventions targeting intestinal microbiota. The authors reported that more than half of the included studies demonstrated improvement in anxiety symptoms following microbiome-focused interventions. Interestingly, non-probiotic approaches frequently produced stronger effects than probiotic supplementation alone.

Probiotics remain the most extensively investigated intervention. According to Yang et al. (2019), supplementation with beneficial bacterial strains may contribute to reductions in anxiety symptoms through modulation of microbial composition, enhancement of intestinal barrier integrity, and regulation of immune responses.

Alli et al. (2022) reviewed evidence regarding probiotics, prebiotics, and synbiotics in psychiatric disorders. Although much of the available evidence focused on depression, the authors concluded that microbiome modulation represents a promising therapeutic strategy with potential relevance to anxiety disorders as well. The review highlighted improvements in psychological outcomes associated with several microbiota-targeted interventions.

Recent reviews by Butler et al. (2025), Nikel et al. (2025), and Shaikh et al. (2025) similarly emphasized the therapeutic potential of microbiome modulation. Proposed strategies include:

- probiotics,
- prebiotics,
- synbiotics,
- psychobiotics,
- dietary interventions.

Among these approaches, dietary modification may be particularly important because diet directly influences microbial diversity and metabolic activity. Diets rich in fiber and plant-based nutrients promote growth of beneficial SCFA-producing microorganisms, whereas highly processed diets may contribute to dysbiosis and inflammation.

Despite encouraging findings, current evidence remains insufficient to establish standardized microbiome-based treatment protocols for anxiety disorders. The reviewed studies consistently emphasized the need for larger randomized controlled trials capable of determining optimal intervention strategies and identifying patient populations most likely to benefit.

Nevertheless, the available literature suggests that microbiome-targeted therapies represent one of the most promising emerging areas in anxiety disorder research.

Discussion

The present narrative review synthesized current evidence regarding the relationship between gut dysbiosis and Generalized Anxiety Disorder. Collectively, the reviewed studies support the hypothesis that alterations in gut microbiota composition may contribute to the development, maintenance, and severity of anxiety symptoms through multiple interconnected biological pathways. Although the available evidence remains heterogeneous, several recurring findings emerged consistently across the literature.

One of the most important observations identified in this review is the repeated association between anxiety disorders and reduced microbial diversity. Both original investigations and review articles reported that individuals experiencing anxiety symptoms frequently exhibit lower microbial richness compared with healthy controls. Jiang et al. (2018) demonstrated significant reductions in microbial diversity among patients diagnosed with Generalized Anxiety Disorder, while Butler et al. (2025), Nikel et al. (2025), and Simpson et al. (2021) highlighted reduced diversity as one of the most consistently reported microbiome-related findings in anxiety research.

The significance of reduced microbial diversity extends beyond simple taxonomic differences. Microbial diversity is widely considered an indicator of ecosystem resilience and stability. Diverse microbial communities are generally better able to maintain metabolic homeostasis, resist environmental disturbances, and support host physiological functions. Consequently, reduced diversity may compromise the capacity of the gastrointestinal ecosystem to regulate immune responses, maintain barrier integrity, and produce beneficial metabolites. Such disturbances may contribute to increased vulnerability to psychiatric symptoms.

Another important finding concerns the recurring depletion of beneficial bacterial taxa. Multiple studies included in this review reported reductions in microorganisms involved in short-chain fatty acid production, particularly members of the genera *Faecalibacterium*, *Coprococcus*, and *Eubacterium*. Butler et al. (2025) identified these microorganisms as among the most consistently depleted taxa across anxiety disorders, while Nikel et al. (2025) emphasized their importance for maintenance of intestinal homeostasis.

The biological significance of these observations is substantial. Short-chain fatty acid-producing bacteria contribute to intestinal barrier integrity, modulation of inflammatory responses, and regulation of host metabolism. Reduced abundance of these organisms may therefore influence multiple physiological systems simultaneously. Rather than functioning solely within the gastrointestinal tract, microbial metabolites generated by these bacteria may exert effects throughout the body, including the central nervous system.

The reviewed literature additionally highlights the importance of microbial metabolites in anxiety pathophysiology. Increasingly, researchers recognize that microbial function may be as important as microbial composition. Butler et al. (2023) demonstrated that anxiety-related microbiome alterations involve not only differences in bacterial abundance but also changes in microbial metabolic pathways. This observation suggests that future studies should examine both taxonomic and functional aspects of the microbiome.

The potential role of microbial metabolites is particularly important because these compounds may directly influence neural signaling pathways. Several reviewed studies discussed microbial influences on serotonin, gamma-aminobutyric acid, dopamine, and other neurotransmitter systems implicated in anxiety disorders. Although the precise mechanisms remain incompletely understood, current evidence suggests that gut microorganisms participate in neurochemical regulation through both direct and indirect pathways.

The microbiota–gut–brain axis provides a useful framework for understanding these interactions. As described by Jiang et al. (2024), Ataei et al. (2025), and MacKay et al. (2024), communication between the gastrointestinal tract and central nervous system occurs through multiple interconnected pathways involving neural, endocrine, immune, and metabolic signaling. Rather than operating independently, these systems function as components of an integrated network capable of influencing emotional regulation and stress responsiveness.

The reviewed studies further support the hypothesis that immune dysregulation may represent a critical mechanism connecting gut dysbiosis and anxiety disorders. Shaikh et al. (2025) emphasized the association between dysbiosis and inflammatory activity, while Krupa-Kotara et al. (2023) highlighted interactions among stress, microbial composition, and immune function. Collectively, these findings suggest that altered microbial communities may promote chronic low-grade inflammation capable of influencing brain function and behavior.

Inflammation has increasingly been recognized as an important contributor to psychiatric disorders. Although anxiety disorders were traditionally conceptualized primarily in terms of neurotransmitter abnormalities, contemporary evidence suggests that immune pathways may also play significant roles. Cytokines and other inflammatory mediators can influence neurotransmitter metabolism, neuroplasticity, and stress-response systems. Consequently, microbiota-induced inflammatory changes may contribute directly to anxiety symptom development.

The relationship between psychological stress and gut microbiota appears particularly complex. Several reviewed studies described a bidirectional interaction in which stress alters microbial composition while dysbiosis amplifies stress-related physiological responses. This reciprocal relationship may help explain why anxiety disorders often become chronic and difficult to treat. Once established, a cycle of stress, dysbiosis, inflammation, and neuroendocrine dysregulation may perpetuate symptom persistence.

Clinical investigations included in this review provide important support for the relevance of microbiota alterations in anxiety disorders. The study conducted by Jiang et al. (2018) remains especially important because it directly examined patients with Generalized Anxiety Disorder. The authors identified significant microbiota alterations and reduced microbial diversity among affected individuals. Importantly, some abnormalities persisted following symptom improvement, suggesting that microbial changes may represent relatively stable biological characteristics rather than temporary consequences of anxiety symptoms.

Similarly, Butler et al. (2023) demonstrated microbiome alterations in individuals with social anxiety disorder. Although social anxiety disorder differs diagnostically from Generalized Anxiety Disorder, the observed findings support the broader concept that anxiety disorders may be associated with characteristic microbiome-related abnormalities. The consistency of findings across different anxiety conditions strengthens confidence in the overall relationship between gut dysbiosis and anxiety.

An important theme emerging from this review is the substantial overlap between anxiety disorders and other psychiatric conditions. Several studies discussed similarities between microbial alterations reported in

anxiety and depression. Simpson et al. (2021), Shaikh et al. (2025), and Alli et al. (2022) all described microbiome-related findings that appear common across multiple psychiatric diagnoses. This observation may indicate that certain microbial disturbances contribute to general vulnerability to emotional dysregulation rather than to specific diagnostic categories.

The therapeutic implications of these findings are considerable. Current first-line treatments for Generalized Anxiety Disorder include cognitive behavioral therapy and pharmacological interventions such as selective serotonin reuptake inhibitors. Although these approaches are effective for many patients, treatment response remains variable and a substantial proportion of individuals continue to experience residual symptoms.

Microbiome-targeted interventions therefore represent a promising area of future research. Yang et al. (2019) reported that more than half of microbiota-focused interventions were associated with improvements in anxiety symptoms. Notably, dietary interventions frequently produced stronger effects than probiotic supplementation alone. These findings highlight the importance of considering the broader microbial ecosystem rather than focusing exclusively on administration of individual bacterial strains.

Diet may represent one of the most practical approaches to microbiome modulation. Because dietary composition directly influences microbial diversity and metabolite production, nutritional interventions may provide a relatively accessible method for supporting both gastrointestinal and mental health. Diets rich in dietary fiber promote growth of beneficial microorganisms and increase production of short-chain fatty acids, whereas highly processed diets may contribute to dysbiosis and inflammatory activity.

The concept of psychobiotics has generated particular interest in recent years. Psychobiotics refer to microorganisms or microbial-derived products capable of producing beneficial effects on mental health. Several reviewed studies identified psychobiotics as a promising future direction in psychiatric treatment. Although current evidence remains preliminary, psychobiotics may eventually become valuable adjunctive therapies for anxiety disorders.

Despite encouraging findings, important limitations must be acknowledged. One of the most significant limitations concerns the predominance of cross-sectional study designs. Most available investigations examine microbiota composition at a single time point, making it difficult to determine whether dysbiosis precedes anxiety development or emerges as a consequence of psychological distress.

Methodological heterogeneity represents another major challenge. The reviewed studies differed considerably with respect to participant characteristics, sequencing techniques, analytical approaches, and outcome measures. Such variability complicates direct comparison of findings and may partially explain inconsistencies regarding specific bacterial taxa.

Additionally, numerous factors capable of influencing microbiota composition are difficult to control completely. Diet, medication use, physical activity, sleep quality, age, sex, geographic location, and socioeconomic status may all affect microbial communities. Future studies should attempt to account for these variables more comprehensively.

Future research should prioritize large longitudinal investigations capable of clarifying causal relationships between gut dysbiosis and anxiety disorders. Randomized controlled trials evaluating probiotics, psychobiotics, dietary interventions, and other microbiome-based approaches will also be essential for determining clinical utility. Furthermore, advances in metagenomics and metabolomics may facilitate identification of specific microbial pathways most relevant to anxiety pathophysiology.

Overall, the evidence reviewed in this manuscript supports the conclusion that gut dysbiosis represents an important component of anxiety disorder biology. While many questions remain unanswered, the microbiota–gut–brain axis offers a compelling framework for understanding interactions among gastrointestinal, immune, endocrine, and neural systems. Continued investigation of these relationships may contribute to the development of more personalized and biologically informed approaches to the prevention and treatment of Generalized Anxiety Disorder.

Conclusions

Generalized Anxiety Disorder is a complex and multifactorial psychiatric condition whose pathogenesis extends beyond traditional neurochemical and psychological explanations. The evidence reviewed in this narrative review suggests that gut dysbiosis may represent an important biological factor contributing to the development and maintenance of anxiety symptoms.

Across the included studies, several recurring findings emerged. Individuals with anxiety disorders frequently exhibit reduced microbial diversity, depletion of beneficial short-chain fatty acid-producing bacteria, alterations in microbial metabolic pathways, and increased abundance of potentially pro-inflammatory taxa. These microbiome-related changes may influence anxiety through multiple interconnected mechanisms involving immune activation, neurotransmitter regulation, microbial metabolite production, and dysregulation of the microbiota–gut–brain axis.

The reviewed literature also highlights the potential therapeutic relevance of microbiome-targeted interventions. Probiotics, psychobiotics, dietary modification, and other strategies aimed at restoring microbial balance have demonstrated promising effects in preliminary studies. Although current evidence remains insufficient to support routine clinical implementation, these approaches may eventually complement existing treatments for anxiety disorders.

Importantly, the relationship between gut dysbiosis and anxiety appears to be bidirectional. Psychological stress may alter microbial composition, while dysbiosis may amplify stress-related physiological responses. This reciprocal interaction may contribute to the persistence and chronicity of anxiety disorders.

Despite substantial progress in this field, significant challenges remain. The predominance of cross-sectional studies, methodological heterogeneity, and limited longitudinal evidence restrict the ability to establish causality. Future research should focus on large prospective studies, standardized microbiome analysis methods, and randomized controlled trials evaluating microbiome-based interventions.

In conclusion, current evidence supports a meaningful association between gut dysbiosis and anxiety disorders. Continued investigation of microbiota–gut–brain interactions may improve understanding of anxiety pathophysiology and contribute to the development of novel preventive and therapeutic strategies for Generalized Anxiety Disorder.

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REFERENCES

1. Alli, S. R., Gorbovskaia, I., Liu, J. C. W., Kolla, N. J., Brown, L., & Müller, D. J. (2022). The gut microbiome in depression and potential benefit of prebiotics, probiotics and synbiotics: A systematic review of clinical trials and observational studies. *International Journal of Molecular Sciences*, 23(9), 4494. <https://doi.org/10.3390/ijms23094494>
2. Ataei, P., Kalantari, H., Bodnar, T. S., & Turner, R. J. (2025). The gut–brain connection: Microbes’ influence on mental health and psychological disorders. *Frontiers in Microbiomes*, 4, 1701608. <https://doi.org/10.3389/frmbi.2025.1701608>
3. Butler, M. I., Bastiaanssen, T. F. S., Long-Smith, C., Mörk, S., Berding, K., Ritz, N. L., Strain, C., Patangia, D., Patel, S., Stanton, C., O’Mahony, S. M., Cryan, J. F., Clarke, G., & Dinan, T. G. (2023). The gut microbiome in social anxiety disorder: Evidence of altered composition and function. *Translational Psychiatry*, 13, 95. <https://doi.org/10.1038/s41398-023-02325-5>
4. Butler, M. I., Kittel-Schneider, S., Wagner-Skacel, J., Mörk, S., & Clarke, G. (2025). The gut microbiome in anxiety disorders. *Current Psychiatry Reports*, 27(5), 347–361. <https://doi.org/10.1007/s11920-025-01604-w>
5. Jiang, H., Zhang, X., Yu, Z., Zhang, Z., Deng, M., Zhao, J., & Ruan, B. (2018). Altered gut microbiota profile in patients with generalized anxiety disorder. *Journal of Psychiatric Research*, 104, 130–136. <https://doi.org/10.1016/j.jpsychires.2018.07.007>
6. Jiang, M., Kang, L., Wang, Y.-L., Zhou, B., Li, H.-Y., Yan, Q., & Liu, Z.-G. (2024). Mechanisms of microbiota–gut–brain axis communication in anxiety disorders. *Frontiers in Neuroscience*, 18, 1501134. <https://doi.org/10.3389/fnins.2024.1501134>
7. Krupa-Kotara, K., Gwóźdź, W., Nandzik, S., & Grajek, M. (2023). The role of microbiota pattern in anxiety and stress disorders—A review of the state of knowledge. *Psych*, 5(3), 602–618. <https://doi.org/10.3390/psych5030038>
8. MacKay, M., Yang, B. H., Dursun, S. M., & Baker, G. B. (2024). The gut-brain axis and the microbiome in anxiety disorders, post-traumatic stress disorder and obsessive-compulsive disorder. *Current Neuropharmacology*, 22(5), 866–883. <https://doi.org/10.2174/1570159X21666230222092029>
9. Nikel, K., Stojko, M., Smolarczyk, J., & Piegza, M. (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>
10. Shaikh, R. G., Dey, A., Singh, V. P., Khandagle, A., Baskaran, M., Naik, S., & Hasan, A. (2025). Understanding the impact of the gut microbiome on mental health: A systematic review. *Cureus*, 17(1), e78100. <https://doi.org/10.7759/cureus.78100>
11. Simpson, C. A., Diaz-Arteche, C., Eliby, D., Schwartz, O. S., Simmons, J. G., & Cowan, C. S. M. (2021). The gut microbiota in anxiety and depression: A systematic review. *Clinical Psychology Review*, 83, 101943. <https://doi.org/10.1016/j.cpr.2020.101943>
12. Spivak, N. M., Haroon, J., Swenson, A., Turnbull, S. A., Dang, N., Ganeles, M., Price, C., Distler, M., Nurmi, E., Lavretsky, H., & Bystritsky, A. (2023). Microbiome in anxiety and other psychiatric disorders. *Medical Clinics of North America*, 107(1), 73–83. <https://doi.org/10.1016/j.mcna.2022.08.010>
13. Yang, B., Wei, J., Ju, P., & Chen, J. (2019). Effects of regulating intestinal microbiota on anxiety symptoms: A systematic review. *General Psychiatry*, 32(2), e100056. <https://doi.org/10.1136/gpsych-2019-100056>