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GUT MICROBIOTA AND DEPRESSION: MECHANISTIC LINKS WITHIN THE GUT–BRAIN AXIS — A NARRATIVE REVIEW

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ABSTRACT

INTRODUCTION

Depression affects over 280 million people worldwide and remains insufficiently responsive to standard pharmacological treatment in approximately 30 to 40 percent of patients. Increasing evidence links the microbiota gut brain axis with the pathophysiology of depressive disorders, however current findings remain heterogeneous and predominantly correlational.

OBJECTIVES

To critically synthesize current evidence on the role of gut microbiota in depression, with a focus on the reproducibility of microbiota alterations, the validity of proposed biological mechanisms within the gut brain axis, and the clinical relevance of microbiota targeted interventions.

METHODS

A narrative review was conducted based on a PubMed literature search performed between October and November 2025, with updates in January and March 2026. The search included publications from the last decade using predefined terms related to gut microbiota and depression, with filters applied for human adult populations. Studies were selected through screening of titles, abstracts and full texts according to predefined inclusion and exclusion criteria. A total of 50 publications were included in the final synthesis.

RESULTS

The most consistent finding is a qualitative shift in gut microbiota towards a pro inflammatory profile rather than consistent changes in overall diversity. This includes depletion of short chain fatty acid producing genera such as Faecalibacterium, Coprococcus and Roseburia, and enrichment of taxa associated with increased lipopolysaccharide related inflammatory potential such as Eggerthella and Alistipes, although findings remain heterogeneous across studies. These alterations are linked to interconnected mechanisms involving hypothalamic pituitary adrenal axis hyperactivation, increased intestinal permeability, neuroinflammation and disrupted tryptophan metabolism. Available human data are predominantly correlational and do not allow determination of whether microbiota changes are causal or secondary. Among therapeutic approaches, dietary interventions show the most consistent evidence, probiotics demonstrate moderate effects when used as adjunctive therapy, and evidence on fecal microbiota transplantation remains limited.

CONCLUSIONS

Gut dysbiosis in depression appears to function as a non specific amplifier of neuropsychiatric vulnerability rather than a direct cause of the disorder. Microbiota targeted interventions may be considered as adjunctive strategies, while clarification of causal relationships requires longitudinal studies and standardized methodological approaches.

Keywords: gut microbiota, depression, gut brain axis, dysbiosis, probiotics, diet

1. INTRODUCTION

Depression is a complex disorder involving a combination of genetic, neurobiological, immunological and environmental factors [1, 2]. According to the World Health Organization, it affects over 280 million people globally, making it a leading cause of disability. Its prevalence increased significantly during the COVID-19 pandemic [1, 3, 4]. Despite the availability of traditional pharmacological and psychological therapies, their efficacy remains limited. Current data suggests that 30–40% of patients do not respond sufficiently to standard antidepressants, and treatment is often hindered by high relapse rates, adverse side effects and withdrawal symptoms [5]. This profound clinical challenge highlights the need for new pathophysiological models that extend beyond traditional neurochemical frameworks and incorporate systemic mechanisms involved in depression.

Consequently, there has been an increasing growth of interest in the microbiota–gut–brain (MGB) axis. The gut microbiota appears to be an important, potentially modifiable element affecting the functioning of the central nervous system. This bidirectional communication network links the gut and the brain through neural (e.g., the vagus nerve), immune, and neuroendocrine (e.g., the hypothalamic-pituitary-adrenal axis) pathways, mediated by microbial metabolites such as short-chain fatty acids (SCFAs) and neurotransmitter precursors [4, 6, 7]. Emerging evidence indicates that gut dysbiosis — an imbalance in the microbial community — contributes to depressive symptoms by increasing intestinal permeability, triggering systemic neuroinflammation, and disrupting tryptophan and serotonin metabolism [6, 8].

Although research in this area has expanded rapidly, the findings remain scattered across numerous original and review sources, and the field is marked by considerable complexity and ongoing debate. The majority of current human studies are cross-sectional and correlational, making it difficult to definitively establish whether gut dysbiosis is a causal factor in depression, or merely a consequence of disease-related physiological and lifestyle changes [4, 9]. Furthermore, there is considerable inconsistency regarding specific microbial alterations across studies, largely attributed to methodological heterogeneity and the profound influence of confounding factors, including diet, age, BMI, and the use of psychotropic medications, which inherently alter the microbiota [9, 10]. Given this complexity, there is value in presenting a structured narrative synthesis of current knowledge that can guide both researchers and clinicians navigating this evolving field.

The novelty of this review lies in addressing a key limitation of the existing literature, namely the insufficient reproducibility of findings and the predominance of correlational evidence in studies of gut microbiota in depression. The available data are heterogeneous and do not allow definitive conclusions as to whether the observed microbiota alterations represent stable features of depression or secondary phenomena. Accordingly, this review provides a critical evaluation of the reproducibility of reported microbiota changes [10, 12], the validity of proposed biological mechanisms within the gut brain axis [22, 30], and the extent to which current evidence supports causal rather than associative relationships [4, 9]. In addition, the review analyzes clinical evidence on microbiota targeted interventions and identifies methodological limitations that complicate the interpretation of results [7, 48, 10, 12]. The aim of this review is to critically synthesize current evidence on the role of gut microbiota in depression, with a focus on the reproducibility of microbiota alterations, the validity of proposed biological mechanisms within the gut brain axis, and the clinical relevance of microbiota targeted interventions.

OBJECTIVES

- To identify the most reproducible changes in gut microbiota composition and diversity in depression.
- To evaluate the validity of biological mechanisms linking gut dysbiosis with depressive symptoms.
- To determine whether current evidence supports a causal relationship between gut microbiota and depression.
- To analyze clinical evidence on microbiota targeted interventions, including diet, probiotics, and fecal microbiota transplantation.
- To identify key methodological limitations and define priorities for future research.

2. METHODS

A narrative review format was chosen given the substantial methodological heterogeneity across primary studies and the need for an integrative synthesis of mechanistic, compositional, and therapeutic evidence that extends beyond the scope of a single meta-analytic question.

A literature search was initially conducted between October and November 2025 using the PubMed database. The search covered publications from the last decade that addressed the relationship between gut microbiota and depression. The following search terms were used: "gut microbiota", "intestinal microbiota", "microbiome composition", and "dysbiosis". These terms were combined with ("depression" OR "depressive disorder") using the Boolean operator AND. Filters such as "humans" and "adult" were applied during the initial search to prioritise studies involving adult populations.

The literature search was updated in January and March 2026 to include recently published studies relevant to the topic. Additional relevant publications were identified through targeted searches in PubMed as well as through manual screening of reference lists and related articles.

The analysis encompassed various types of publications, including meta-analyses, clinical and experimental studies, clinical trials, and systematic or narrative reviews. Although priority was given to high-level evidence, such as meta-analyses and systematic reviews when available, other relevant studies were also included to provide a comprehensive overview of the topic.

Relevant publications were identified through screening of titles and abstracts, and their full texts were reviewed when necessary to assess their relevance to the scope of this review. Following the screening of titles, abstracts, and full texts, a total of 50 publications were included in the final narrative synthesis. To guide the literature review, the following research questions were formulated:

- What alterations in the composition and diversity of the gut microbiota are most consistently observed in individuals with depression.
- What biological mechanisms link gut microbiota dysbiosis with the development of depressive symptoms within the microbiota gut brain axis.
- Which microbial metabolites and signaling pathways play a key role in gut brain communication in the context of depression.
- What microbiota targeted therapeutic interventions have been investigated for their potential role in the treatment of depression.

Inclusion criteria: publications from the last 10 years, articles written in English and available in full text, studies investigating the composition, diversity, or function of the gut microbiota in the context of depression or major depressive disorder, publications addressing biological mechanisms of the microbiota gut brain axis, including inflammatory, neuroendocrine, or metabolic pathways associated with depressive disorders.

Exclusion criteria: studies addressing postpartum depression, works focused exclusively on a single bacterial strain, publications discussing diet without analysing its relationship with the gut microbiota, studies conducted exclusively in healthy populations, articles not directly related to the scope of the review.

3. RESULTS

3.1 COMPOSITION OF GUT MICROBIOTA IN DEPRESSION

The analysis of the composition of the gut microbiota is of significant importance in understanding its impact on the pathophysiology of depressive disorders, as well as in the development of research on more effective methods of diagnosis, treatment, and monitoring of the course of the disease [1, 2, 11]. The research focuses on two aspects:

the overall composition of the gut flora, measured by diversity indices, and the identification of changes in the abundance of individual bacterial taxa between healthy individuals and those with depression [12, 13].

In research, diversity is measured in two distinct ways. Alpha diversity is a measure of biodiversity that assesses the number and proportion of species in a sample from a single individual [12]. Meta-analyses and systematic reviews indicate that, although it was originally assumed that species impoverishment could be a characteristic feature of depression, most studies do not observe statistically significant deviations in alpha diversity [10, 12, 14]. However, the data on this subject are inconsistent, and individual studies indicate a decrease or, less frequently, an increase. For instance, large population studies occasionally report a negative association between alpha diversity and depressive symptoms [15]. Conversely, studies focusing on late-life depression occasionally report a paradoxical increase in alpha diversity indices among depressed individuals not taking medication [16].

Beta diversity is defined as the comparison of changes between individuals or groups, such as a study group and a control group [12]. The findings of these studies provide more conclusive results, indicating that although the number of species may be similar, their quality is different [10, 14]. However, no distinctive patterns that are characteristic of any specific disease have been identified yet. Instead, the observed microbial shifts appear to be shared across multiple psychiatric conditions [14]. To overcome these transdiagnostic overlaps and discover precise signatures, recent studies frequently utilize metagenomic profiling and machine learning algorithms to identify specific bacterial biomarkers for depression [1, 11, 17].

Despite the limitations of the existing literature, it is repeatedly indicated that there are changes in the microbiota profile towards a more pro-inflammatory state in individuals diagnosed with depression [2, 10, 14]. The most frequently documented alterations include an increase in the number of bacteria producing lipopolysaccharides (LPS) and a decrease in the number of taxa responsible for the production of short-chain fatty acids (SCFA) [1, 2]. Specifically, there is a consistent depletion of beneficial genera such as Faecalibacterium, Coprococcus, and Roseburia [10–12, 15]. Simultaneously, patients with depression often show an enrichment of potentially pathogenic taxa, including Eggerthella, Alistipes, Sellimonas, and Enterobacteriaceae [14, 15, 17].

Changes in the composition of the microbiome provide the biological basis for the mechanisms by which the microbiome can modulate hormone secretion, immune response, and neurotransmitter production [1, 2]. The key findings from representative studies and meta-analyses investigating these microbial shifts are synthesized in Table 1.

Table 1. Summary of studies on gut microbiota composition in depression

Author, year	Study design	Population and sample size	Depression assessment	Microbiota assessment method	Key microbial changes	Main findings
Averina et al., 2024	Review	Number of included studies n = 252	Not applicable	16S rRNA sequencing; metagenomics	↓ SCFA-producing taxa,↑ LPS-producing taxa	Pro-inflammatory shifts underlie microbiome-driven modulation of immune response, hormone secretion, and neurotransmitter production
Varesi et al., 2023	Review	Number of included studies n = 347	Not applicable	16S rRNA sequencing; metagenomics	↓ SCFA-producing taxa,↑ LPS-producing taxa	Gut microbiota acts as key mediator of brain-gut interactions; consistent pro-inflammatory shifts suggest potential targets for innovative

						therapeutic approaches
Gao et al., 2023	Meta-analysis / systematic review	Number of included studies n=44	DSM, ICD-10, HDRS, CES-D, PHQ-9	Pooled analysis ;16S rRNA and metagenomics	No significant alpha diversity differences	Genus-level shifts are more informative than phylum-level or alpha diversity metrics
McGuinness et al., 2022	Systematic review	Number of included studies n=44	Clinical diagnosis	Pooled analysis	↓ Faecalibacterium, Coprococcus, Roseburia; altered beta diversity	Beta diversity more informative than alpha diversity; depletion of beneficial genera consistent across studies despite similar species richness
Nikolova et al., 2021	Meta-analysis / systematic review	Number of included studies n=59	Clinical diagnosis / validated scales	Pooled analysis	↑ Eggerthella, Alistipes, Sellimonas, Enterobacteriaceae; altered beta diversity	Microbial shifts are transdiagnostic; beta diversity reveals qualitative differences despite similar species richness
Angelova et al., 2023	Case-control	Patients with depression n=36; controls n=38; age range 18-54	Clinical diagnosis	Metagenomics	↓ Faecalibacterium prausnitzii	Metagenomic profiling with machine learning appears promising for advancing microbiota-based diagnosis and disease monitoring
Radjabzadeh et al., 2022	Cohort	n=2,593; age range 19–87	CES-D, PHQ-9	16S rRNA sequencing	↓ Faecalibacterium, Coprococcus, Roseburia; ↑ Eggerthella, Alistipes, Sellimonas, Enterobacteriaceae	Reduced alpha diversity and depletion of beneficial genera linked to depressive symptoms in large population-based cohort
Wang et al., 2025	Case-control	Patients with depression n=52; controls n=56	HAMD-17	Metagenomics	↓Faecalibacterium, Roseburia; ↑ Alistipes	Machine learning may help identify depression-specific bacterial biomarkers beyond transdiagnostic microbial patterns

Abbreviations: SCFA, short-chain fatty acids; LPS, lipopolysaccharides; 16S rRNA, 16S ribosomal ribonucleic acid; DSM, Diagnostic and Statistical Manual of Mental Disorders; ICD-10, International Classification of Diseases, 10th Revision; HDRS / HAMD-17, Hamilton Depression Rating Scale; CES-D, Center for Epidemiological Studies-Depression scale; PHQ-9, Patient Health Questionnaire-9.

3.2 MECHANISMS LINKING GUT MICROBIOTA TO DEPRESSIVE DISORDERS

The correlation between gut dysbiosis and depression is not merely a statistical one; rather, it is based on specific, multidirectional biological mechanisms [8, 18]. The microbiota communicates with the brain through a complex network of neuroendocrine, immune, and neuronal pathways [19, 20]. This bidirectional communication is collectively referred to as the microbiota-gut-brain axis, and its disruption is currently a central focus in neuropsychiatric research [1, 21]. A comprehensive understanding of these mechanisms is crucial for the development of effective and targeted therapeutic strategies [4, 22, 23].

Hypothalamic-pituitary-adrenal axis

The gut microbiome has been demonstrated to play a pivotal role in the regulation of the HPA axis, affecting its development and reactivity [1, 21]. This axis is the body's primary stress response system, and its hyperactivity can result in increased cortisol secretion by the adrenal glands [8, 22]. Prolonged exposure to excess cortisol can induce structural damage, reduced neurogenesis, and atrophy in brain regions critical for mood and cognition, such as the hippocampus and prefrontal cortex [8]. Studies utilizing germ-free animal models confirm that the absence of gut microbes leads to exaggerated HPA axis responses, evidenced by elevated circulating levels of adrenocorticotropic hormone (ACTH) and corticosterone following stress exposure [18, 24]. This, in turn, has been shown to result in the onset of more severe depressive symptoms, particularly anxiety reactions [20, 21, 25].

It is important to note that this relationship is bidirectional [8]. The composition of the microbiome exerts a regulatory influence on the activity of the HPA axis, while the level of cortisol in the blood, in turn, affects the composition of the microbiome, thereby establishing a vicious pathophysiological cycle [3, 8, 20].

Inflammatory and immune mechanisms

Inflammatory theories of depression propose that intestinal dysbiosis leads to intestinal barrier dysfunction and increased intestinal permeability [6, 9]. This process facilitates the translocation of bacterial components and metabolites from the gut lumen to the mesenteric lymph nodes and peripheral circulation [9]. Among these components, lipopolysaccharide (LPS), a pro-inflammatory constituent of Gram-negative bacteria, plays a particularly important role [9, 26]. Consequently, patients with major depressive disorder often exhibit increased IgA- and IgM-mediated immune responses directed against LPS [1, 9].

The presence of circulating LPS activates systemic immune responses, leading to elevated concentrations of pro-inflammatory cytokines, including interleukin-6 (IL-6) and tumor necrosis factor-α (TNF-α). In addition, increased levels of non-specific inflammatory markers such as C-reactive protein (CRP), haptoglobin, and fibrinogen are frequently observed in these patients [1, 6, 9].

Pro-inflammatory cytokines can cross the blood–brain barrier through specific transport mechanisms [1, 20, 27]. Recent studies have identified molecular transport systems that actively facilitate the passage of immune-modulating cytokines across this selectively permeable barrier [28, 29]. The resulting increase in central inflammatory signaling contributes to the inhibition of neurogenesis and activation of microglia, the primary immune effector cells of the central nervous system [2, 6]. Activated microglia release reactive oxygen species and additional cytokines, creating a pro-inflammatory environment that disrupts synaptic plasticity and promotes neuronal damage. This process ultimately leads to neuroinflammation within the brain [6, 21, 29]. Such neuroinflammatory signaling has been implicated in the development of depressive symptoms, including anhedonia and fatigue [22, 25, 27, 28].

These cytokine-driven behavioral changes, collectively referred to as sickness behavior, are thought to have evolved as adaptive responses to infection but may also contribute to vulnerability to depressive disorders in modern environments [21, 28].

The impact of bacterial metabolites

It is hypothesized that bacterial metabolites may function as signaling molecules in communication with the brain [1, 18, 23, 30]. Short-chain fatty acids (SCFAs) are produced in the large intestine as a result of the fermentation of substances that are not digested in the small intestine, primarily dietary fiber and resistant starch. The most extensively studied SCFAs are butyrate, propionate, and acetate [3, 18, 27, 30, 31]. These molecules serve as natural ligands for free fatty acid receptors, including FFAR2 and FFAR3, which are widely expressed on

enteroendocrine, immune, and neural cells [30]. Their key functions are as follows: firstly, they strengthen the intestinal barrier by providing an energy source for colonocytes; secondly, they exert anti-inflammatory effects by inhibiting histone deacetylases; and thirdly, they stimulate the production of brain-derived neurotrophic factor (BDNF), which is crucial for neurogenesis and neuronal plasticity [1, 27, 30–32]. Conversely, reduced levels of these essential metabolites are frequently observed in individuals with depression and are directly correlated with the exacerbation of depressive symptoms. Some SCFAs, such as acetate, have been observed to be able to cross the blood–brain barrier and act directly by promoting the maturation of microglial cells [18, 23]. Beyond SCFAs, other microbially derived compounds, such as secondary bile acids, lactate, and glycerophospholipids, are increasingly recognized as critical modulators of this gut–brain crosstalk [21, 33].

Tryptophan

Tryptophan is an essential amino acid that is a precursor to serotonin and kynurenine [3, 27]. Alterations in serotonergic neurotransmission have long been implicated in the pathophysiology of depression [3, 22, 34]. Notably, over 90% of the body's serotonin is synthesized within the gastrointestinal tract by enterochromaffin cells, a process heavily dependent on microbial stimulation of the tryptophan hydroxylase 1 (TPH1) enzyme [22, 29, 34].

Inflammation of the intestines, driven by dysbiosis, activates the IDO enzyme, which shifts tryptophan metabolism towards the kynurenine pathway [3, 29]. This activation is predominantly triggered by pro-inflammatory cytokines, particularly interferon-gamma (IFN-γ), linking peripheral immune responses to altered neurotransmitter availability [29, 34]. This results in a deficiency of tryptophan, leading to a reduction in serotonin synthesis [3]. Additionally, this process is accompanied by the synthesis of neurotoxic metabolites, including quinolinic acid [3, 23, 29, 34]. Quinolinic acid acts as an N-methyl-D-aspartate (NMDA) receptor agonist promoting excitotoxicity, while the production of neuroprotective kynurenic acid is simultaneously suppressed [29, 34].

Direct production of neurotransmitters

It is important to note that certain species of bacteria inhabiting the intestines possess the capacity to directly synthesise neurotransmitters [20, 26, 35–37]. For instance, Lactobacillus and Bifidobacterium strains can produce GABA, while other genera like Escherichia, Enterococcus, and Bacillus are involved in the synthesis of serotonin and dopamine [3, 37, 38]. Specific strains such as Lactobacillus plantarum and Bacillus subtilis are capable of producing acetylcholine precursors, further expanding the neurochemical repertoire of the gut microbiome [31, 37]. Although peripherally produced neurotransmitters typically do not cross the blood-brain barrier directly, they can act locally through the ENS (enteric nervous system) and modulate central nervous system functions and stress responses via vagal afferent pathways [1, 6, 38, 39].

The function of the vagus nerve

The vagus nerve, the longest cranial nerve, represents the most direct connection between the intestines and the brain [6, 18, 23]. It has been determined that approximately 80% of its fibres are afferent, which means that they transmit information from the intestines to the brain [27]. These fibers, located in the intestinal wall, have been shown to respond to bacterial metabolites, neurotransmitters, and immune system cells [18, 23, 39]. Recent findings emphasize that enteroendocrine cells, also referred to as neuropod cells, act as crucial chemosensors that rapidly transduce luminal microbial signals to vagal afferents through synaptic connections utilizing glutamate and serotonin [40]. These signals are transmitted through the vagus nerve to the nucleus of the solitary tract in the brain, and from there to higher brain structures such as the amygdala, hippocampus, and prefrontal cortex [18, 19, 38, 39]. Animal studies have demonstrated that vagotomy prevents the anxiolytic effects of certain probiotics and the emergence of anxiety-like behaviours, thereby providing evidence for its role in the communication between the gut and the brain [23, 32].

The observed mechanisms demonstrate the potential for modifications in the gut microbiome to initiate a sequence of biological processes, which, in turn, can contribute to the manifestation of depressive symptoms [18, 20, 41].

3.3 MODULATION OF THE GUT MICROBIOTA

An understanding of the mechanisms by which alterations in the composition of the intestinal microbiota lead to depressive disorders has facilitated progress in the area of therapeutic intervention. The effectiveness of interventions such as dietary changes, probiotic and prebiotic use, or fecal microbiota transplantation (FMT) is currently being intensively studied [2, 4, 36, 42]. These microbiota-targeted strategies primarily aim to restore microbial eubiosis, reinforce intestinal barrier integrity, and attenuate central neuroinflammation [17, 43, 44]. By reversing these underlying pathophysiological processes, such interventions offer a promising adjunctive approach to alleviating depressive symptoms and improving patients' overall mental well-being [45, 46].

The main microbiota-targeted interventions currently investigated in the context of depression are summarized in

Table 2.

Table 2. Overview of microbiota-targeted interventions in depression

	Mechanism of Action	Level of Evidence	Clinical Potential	Key Limitations	References
Probiotics (Psychobiotics)	Modulation of neurotransmission; HPA axis regulation and cortisol reduction; reduction of pro-inflammatory cytokines (IL-6, TNF-alpha); increased hippocampal BDNF expression.	Animal models; randomized controlled trials (RCTs); meta-analyses; systematic reviews.	Reduction of depressive and anxiety scores; adjunct therapy for MDD; improvement in sleep quality, stress resilience, and cognitive function.	Strain-specific effects; heterogeneous dosages; small clinical sample sizes; limited long-term longitudinal data.	[2, 7, 18, 22, 38, 44]
Prebiotics	Selective stimulation of Bifidobacterium and Lactobacillus; increased SCFA production; enhancement of gut barrier integrity; modulation of hippocampal BDNF.	Animal models; small-scale RCTs; observational studies; meta-analyses.	Improvement in emotional processing; potential anxiolytic and antidepressant-like effects.	Often ineffective as standalone therapy for clinical MDD; inconsistent results in human trials; limited high-quality clinical studies.	[7, 21–23, 30, 38]
Synbiotics	Synergistic restoration of gut eubiosis; reduction of systemic inflammation (LPS, TNF-alpha); improved epithelial barrier protection; modulation of amino acid metabolism.	Animal models; small RCTs; systematic reviews; multicenter trials.	Effective adjunctive intervention for psychiatric symptoms; potential for cognitive restoration in comorbid populations.	Limited data specifically for MDD; complexity in defining optimal strain-prebiotic combinations; lack of standardized formulations.	[2, 31]
Mediterranean Diet	Enrichment of fiber-consuming, anti-inflammatory bacteria (e.g., Faecalibacterium); increased SCFA production; reduction of oxidative stress and pro-inflammatory markers.	Large-scale observational cohorts; longitudinal studies; RCTs (e.g., SMILES trial); meta-analyses.	Significant reduction in depression risk and severity; improvement in quality of life; protective role against neurodegenerative disorders.	Reliance on self-reported dietary adherence; confounding lifestyle and socioeconomic factors; difficulty isolating microbiota-independent mechanisms.	[2, 21, 23, 36, 47]
Fecal Microbiota Transplantation (FMT)	Rapid restoration of microbial diversity; re-establishment of serotonergic/	Preclinical transfer studies (human-to-rodent);	High therapeutic potential for treatment-resistant depression (TRD);	Safety and standardized screening concerns; invasive	[1, 2, 22, 46, 48]

	glutamatergic balance; modulation of neuroinflammation.	case reports; small open-label clinical trials; pilot RCTs.	rapid symptom relief; correction of severe dysbiosis-linked behaviors.	delivery modes; high "non-responder" rates; unknown long-term durability and donor-recipient compatibility.	

Abbreviations: FMT, fecal microbiota transplantation; SCFA, short-chain fatty acids; HPA, hypothalamic-pituitary-adrenal axis; RCT, randomized controlled trial; BDNF, brain-derived neurotrophic factor.

Probiotics, Prebiotics and Synbiotics

Probiotics are defined as preparations containing live microorganisms [2]. Probiotics that have a beneficial effect on mental health are sometimes referred to as "psychobiotics" [20, 31, 32]. Prebiotics contain substances that provide nourishment for beneficial intestinal bacteria, such as fibre [1, 23, 31]. Synbiotics are probiotics and prebiotics in one preparation [7, 20].

A number of clinical studies have indicated that supplementation with probiotics and synbiotics is associated with a modest but statistically significant reduction in depressive symptoms when compared to placebo [7, 49]. Meta-analyses suggest that these clinical benefits are most pronounced when multi-strain probiotics are administered as an adjunctive treatment to standard antidepressant medications, rather than as a standalone therapy [14]. Preparations containing Lactobacillus and Bifidobacterium strains appear to be of particular importance [4, 20]. In contrast, the evidence regarding prebiotics is considerably less clear. Despite the evidence from animal studies demonstrating a modulation of anxiety behaviours and increased BDNF concentrations in the hippocampus, there remains a lack of conclusive evidence to support such an effect in humans [7, 23]. In order to determine the most effective strains, doses, and durations of therapy, larger-scale studies are required [7, 49].

Dietary modifications

Nutritional intake has been demonstrated to be significantly associated with the structure of the gut microbiota, often exerting a more profound influence on its composition than host genetics [32, 43]. A diet high in saturated fats, simple sugars, and low in fibre does not provide the necessary substrates for SCFA synthesis [43]. Instead, it promotes an increase in the population of bacteria that produce pro-inflammatory substances, ultimately lowering microbial diversity [22, 43]. On the other hand, a diet with a high fibre content, as well as polyphenols (found in fruits, vegetables, and olive oil), and omega-3 fatty acids, has been shown to help restore the balance of the intestinal ecosystem [1, 2, 22]. Furthermore, specific omega-3 polyunsaturated fatty acids like EPA and DHA play a pivotal role in regulating dopaminergic and serotonergic neurotransmission and can restore the microbiota ecosystem following stress or dysbiosis [31, 50]. Recent advanced mediation analyses have confirmed that the gut microbiota acts as a significant mediator in the relationship between adherence to a Mediterranean dietary pattern and the reduction of depressive symptoms [47]. While establishing a fundamentally stable architecture of the intestinal ecosystem is typically associated with a prolonged duration, detectable shifts in some bacterial species can occur within 24 hours of dietary modification [18, 43]. Thus, placing a greater emphasis on nutritional intake and implementing personalized dietary interventions aligned with these principles are vital for their role in both long-term prevention and the rapid enhancement of treatment outcomes [47, 50].

Fecal microbiota transplantation (FMT)

Fecal microbiota transplantation (FMT) is the most invasive intervention of those presented, although the degree of invasiveness varies depending on the delivery route, which ranges from oral capsules to direct gastrointestinal administration via colonoscopy or enema [1, 2, 46, 48]. The procedure involves the transfer of the entire ecosystem of microorganisms from a healthy donor to the recipient's gastrointestinal tract in order to restore microbial homeostasis and correct dysbiosis [1, 2, 7, 22, 48].

Recent meta-analyses of randomized controlled trials confirm that FMT may alleviate depressive symptoms, particularly in the short- to mid-term. Direct gastrointestinal delivery routes appear to yield a larger therapeutic effect size than oral capsules [48]. The development of this method could be a crucial step forward for patients suffering from treatment-resistant depression [46, 48]. This is evidenced by recent case reports and pilot studies showing rapid symptom improvement within just four weeks of utilizing FMT as an adjunctive therapy [46]. However, current

evidence also suggests that the long-term efficacy of FMT may wane over time, highlighting the potential need for repeated administration protocols rather than a single intervention [46, 48].

Nevertheless, further research is required on the long-term safety of the procedure, the identification of potential donors, and the matching of donors and recipients. The optimization of FMT will likely rely on artificial intelligence-driven algorithms for precise donor-recipient matching to ensure successful microbial engraftment, minimize adverse effects, and sustain a long-term clinical response [22, 46].

4. DISCUSSION

One of the most significant findings to emerge from this review is that the absence of a distinct microbiota 'signature' specific to depression is not merely a methodological artefact; it may itself be a biologically meaningful observation. The transdiagnostic nature of the observed microbial alterations, which are shared by depression, bipolar disorder and schizophrenia, suggests that gut dysbiosis acts as a non-specific factor that amplifies neuropsychiatric risk rather than being a direct cause of any single disorder [12, 14, 23]. This reframing shifts the question from 'Which bacteria cause depression?' to 'Under what conditions does dysbiosis lower the threshold for depressive symptoms to emerge in a predisposed individual?'. This is a model that is more consistent with the multifactorial aetiology of depression, and it opens up more research opportunities than searching for a single diagnostic biomarker. Notably, the microbiota–gut–brain axis is a highly complex and dynamic system in which microbial metabolites, immune signalling and neuroendocrine pathways interact simultaneously, making it improbable that a single microbial factor explains the observed associations alone [22, 32].

A second key finding of this review is that the MGB axis pathways have a highly interconnected, self-reinforcing structure. Although the individual mechanisms — HPA axis dysregulation, neuroinflammation, tryptophan metabolism diversion and reduced SCFA production — are often discussed in isolation, their true pathophysiological significance lies in their interaction with each other. Gut dysbiosis reduces the number of taxa that produce SCFA, weakening the intestinal barrier and allowing LPS to move from the gut into the blood. LPS in the blood then activates pro-inflammatory cytokines, which divert tryptophan towards the kynurenine pathway. This results in the production of quinolinic acid, which promotes NMDA receptor-mediated excitotoxicity and suppresses BDNF-dependent neurogenesis. Cortisol, released via HPA hyperactivation, further reshapes the microbial community, perpetuating the cycle [6, 8, 21, 34]. Recognising this cascade as a network rather than a series of parallel processes has direct therapeutic implications: interventions targeting a single node may be ineffective not because the gut-brain hypothesis is incorrect, but because the pathology is distributed across the network. Therefore, multi-targeted strategies combining dietary fibre, probiotics and anti-inflammatory components may be more effective at disrupting this cycle.

The three therapeutic categories reviewed differ substantially in their evidence base. Dietary interventions are supported by the most robust observational evidence, including recent mediation analyses confirming that gut microbiota mediate the relationship between Mediterranean dietary patterns and depressive symptoms. Probiotic trials have produced modest but statistically significant effect sizes, with the most significant results observed when multi-strain preparations are used along with antidepressants rather than as a standalone treatment [21, 31, 47]. FMT is mechanistically compelling and supported by early clinical evidence, particularly in treatment-resistant populations [46, 48].

One of the most challenging issues in this field is the confounding role of psychotropic medication. In most studies, either medicated patients are excluded or the effects of medication are not adequately controlled, despite microbiota composition being altered by antidepressants, antipsychotics and mood stabilisers independently. This creates a bidirectional relationship: dysbiosis may contribute to depression, but the medications used to treat it may also alter the microbiota, resulting in changes that are difficult to distinguish from those associated with the disease. Future research designs should treat medication status as a primary area of interest, investigating whether modulation of the microbiota can enhance the response to antidepressants or whether specific microbial profiles predict treatment resistance.

Interpreting the evidence reviewed here also requires acknowledging broader methodological constraints. A considerable amount of variety in sample collection, sequencing techniques, and population selection limits cross-study comparability, while the predominantly correlational nature of the human data that is available leaves unresolved whether microbiota alterations come before depression or follow from it. Addressing this will require large-scale, longitudinal cohort studies with standardised protocols, alongside multi-omics approaches and machine learning–based biomarker discovery [4, 10–12].

5. CONCLUSIONS

The findings of this review indicate that the most reproducible feature of gut microbiota in depression is not a

quantitative change in diversity, but a qualitative shift towards a pro inflammatory profile, characterized by a reduction in short chain fatty acid producing bacteria and an increase in taxa associated with lipopolysaccharides. This directly addresses the objective of identifying consistent microbiota alterations and confirms the absence of a specific microbial signature of depression.

The analysis of biological mechanisms shows that the association between dysbiosis and depressive symptoms is mediated through interconnected immune, neuroendocrine and metabolic pathways, including hyperactivation of the hypothalamic pituitary adrenal axis, neuroinflammation and alterations in tryptophan metabolism. These mechanisms are supported by experimental and clinical data, however their significance is expressed as a complex network of interactions rather than a single causal pathway, which corresponds to the objective of evaluating mechanistic validity.

With regard to causality, the available evidence is predominantly correlational and does not allow determination of whether microbiota alterations precede the development of depression or arise as its consequence, including the effects of treatment. This corresponds to the objective of assessing causality and highlights the need for longitudinal studies and standardized protocols.

The analysis of therapeutic approaches shows that the most consistent evidence is observed for dietary interventions, whereas probiotics demonstrate a moderate effect when used as an adjunct to antidepressants. Evidence on fecal microbiota transplantation remains limited to early stage and small scale studies. This corresponds to the objective of evaluating clinical interventions and indicates their possible role only as adjunctive strategies.

Finally, the review identifies key methodological limitations, including heterogeneity of study designs, variability in microbiota assessment methods, and the impact of psychotropic medications as a significant confounding factor. Addressing these limitations is a priority for future research and is necessary to improve reproducibility and clarify causal relationships.

DISCLOSURE

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USE OF AI

The translation from the native language of the authors into English was assisted by an AI-powered translator. AI tools were also used for stylistic and linguistic editing of the manuscript. No AI assistance was used for the generation or interpretation of scientific content. The authors have verified the entire text and take full responsibility for its accuracy and content.

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Authors declare no conflicts of interest.

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