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The role of gut microbiota in the pathogenesis of depression

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ABSTRACT

Depression is a severe, common mental health disorder with complex contributing factors. It is a severe problem in modern medicine, because standard therapies (such as pharmacological and psychotherapy) often are not enough to achieve complete symptom relief. Encouragingly, the role of gut microbiota in the development and progression of mental disorders may open new therapeutic pathways. In this study, we aimed to summarize the role of the gut microbiota in the pathogenesis of depression. The potential therapeutic benefits of modulating this system are also important. This study demonstrates how a personalized approach to each patient can improve treatment outcomes.

Keywords: gut microbiota; gut-brain axis; depression; psychobiotics; inflammation

1. INTRODUCTION

As discussed above, depression is still a major public health issue worldwide. Interest in gut microbiota, the microorganisms in the digestive tract and their link to mental health has grown in recent years (Forssten et al., 2022). Research indicates that the gut microbiota affects mental health through several mechanisms. These include vagus nerve signaling, modulation of the immune system, regulation of tryptophan (a precursor of serotonin) metabolism, hormone signaling and the production of neuroactive compounds (Godos et al., 2023). An imbalance in the gut microbiota, called dysbiosis, can disrupt these mechanisms. Focusing on microbial homeostasis could be a promising target for preventing or treating mental health disorders, such as depression.

Unfortunately, traditional treatments such as pharmacotherapy and psychotherapy do not always provide sufficient improvement. In some cases, side effects make it difficult for patients to continue therapy (Asher et al., 2017). What is interesting is that recent studies have suggested that supporting gut health may help to reduce depressive symptoms. For example, a UK Biobank study found that people who ate more fruits, vegetables and fiber reported better sleep and overall mental well-being (Hepsomali and Groeger, 2021). In this review, we summarize what we currently know about the link between the gut microbiota and depression. We also describe how daily habits such as diet, physical activity and sleep can affect

gut health. We also focused on the importance of an individualized approach to patient care can support treatment through the use of different clinical studies.

2. REVIEW METHODS

The review search was conducted between July and August 2025 using major scientific databases, including PubMed and Google Scholar. The selected articles were analyzed and summarized in this review. We used keywords such as depression, gut microbiota, gut-brain axis and psychobiotics during the search process. The search covered studies from January 2010 to August 2025 with some earlier works included when relevant.

We included only full-text, peer-reviewed articles in English or Polish. We included both original research and review articles. In total, we initially identified 274 records. First, we removed duplicates. We then looked through the titles and abstracts. This process left 68 full-text articles for detailed assessment. We selected thirty studies for final inclusion. The main limitation of this review is the diversity in study designs and methodologies, which can make direct comparison between studies difficult. Figure 1 shows the selection process.

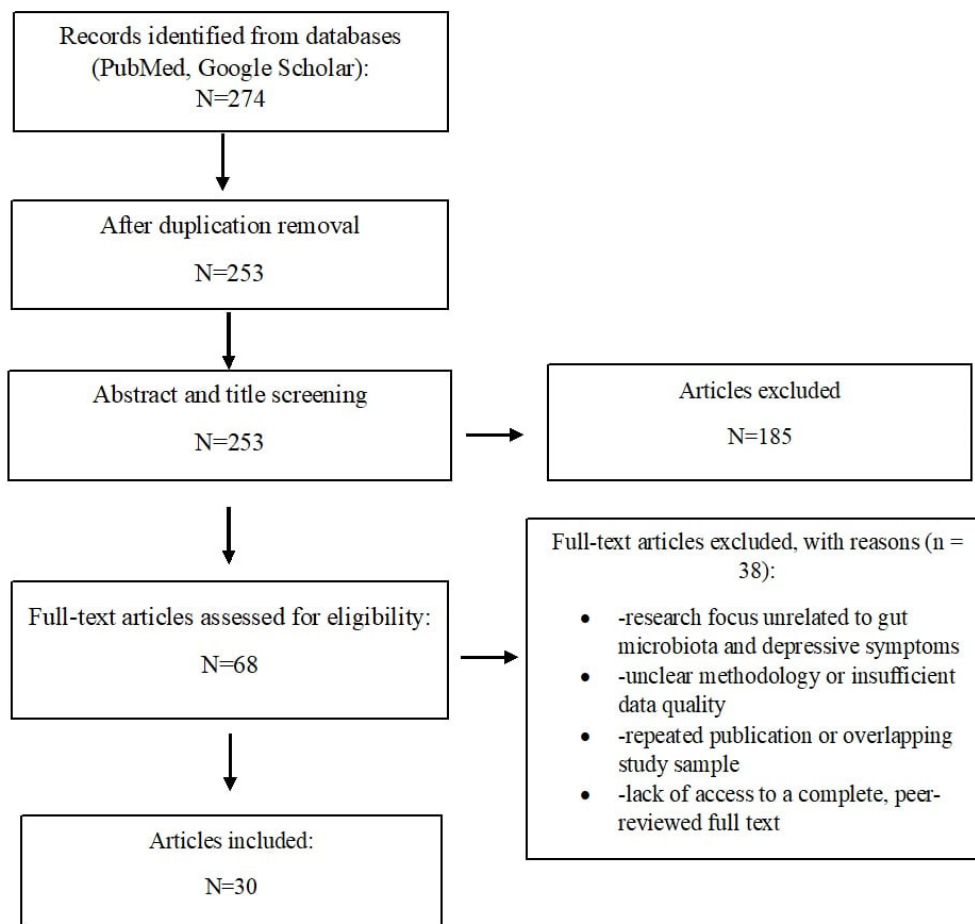


Figure 1. The PRISMA diagram illustrates the steps of identifying, screening and selecting studies for the review.

3. RESULTS & DISCUSSION

Gut-Brain Axis – Functional Basis

Recent research highlights the connection between the gut and the brain, called the gut-brain axis. The connection is two-directional and involves both the organs and the microorganisms in the gut. In this interaction bacteria from the Bacteroides and Firmicutes groups play a key role (Rinninella et al., 2019). The gut-brain axis communicates through several pathways, including neural, hormonal, immune and metabolic routes (Dinan and Cryan, 2017). A key part of this system is the autonomic nervous system. In

particular, the vagus nerve carries signals between the gut and the brain (Góralczyk-Bińkowska et al., 2022). The system helps maintain internal balance and influences emotional regulation, behavior and cognitive functions. The gut and the brain communicate in both directions. Changes in the gut can affect brain activity and psychological stress or brain activity can also influence gut function. The gut–brain axis consists of three interconnected systems: neural, immune and endocrine–metabolic. Disturbances in one of these systems may trigger dysfunction in the others. It may result in persistent symptoms such as reduced motivation, fatigue or impaired concentration.

A key part of this system is the intestinal barrier, which includes cellular tight junctions and protective mucus layers. The integrity of the barrier determines whether antigens reach immune cells and influence inflammation throughout the body. In this case it can cause inflammation and the patient may feel unwell. Diet, stress and medications (such as antibiotics or NSAIDs) can affect the barrier. Individual differences in the gut microbiota may explain why people respond differently to stress and experience variations in mood.

Influence of Gut Microbiota on Nervous System Function

The gut microbiota influences the nervous system by modulating neurotransmitters such as serotonin, norepinephrine, dopamine, glutamate and GABA (Winter et al., 2018). Some gut bacteria can influence the synthesis and metabolism of these neurotransmitters. Others can produce them directly. For example, *Escherichia*, *Enterococcus* and *Streptococcus* can synthesize serotonin (Dinan et al., 2015). *Bifidobacterium* and *Lactobacillus* are known to produce GABA (Reardon, 2014). *Bacillus* and *Serratia* are associated with dopamine production. *Lactobacillus* can influence acetylcholine levels, while *Escherichia* affects norepinephrine levels (Lyte, 2011). In this case, when these neurotransmitters become unbalanced, they can contribute to the development of disorders such as depression. For example, serotonin (5-hydroxytryptamine, 5-HT) plays a key role in regulating sleep, appetite, pain perception and overall mood. Because depressive symptoms are often associated with reduced serotonergic activity, selective serotonin reuptake inhibitors (SSRIs) are commonly used in treatment (Cowen and Browning, 2015). Dopamine is important for motivation and the reward system. People with depression often cannot feel pleasure in activities they once enjoyed. This is linked to lower dopamine activity (Dunlop and Nemeroff, 2007). In addition, norepinephrine is essential for energy, concentration and stress response. Low levels may lead to fatigue, impaired attention and psychomotor slowing. GABA is the primary inhibitory neurotransmitter in the central nervous system (CNS), maintaining the balance between excitation and inhibition. Reduced GABA levels relate to depression and symptoms such as anxiety and tension (Luscher et al., 2011). Overall, the gut microbiota may influence neurotransmitter levels, making it an essential factor in the gut-brain axis. From a practical perspective, these neural effects may present as changes in sleep patterns, appetite or stress tolerance. Clinicians routinely monitor patients during follow-up appointments.

Influence of Gut Microbiota on Immune System and Inflammation in Depression

Beyond its role in neurotransmission, the gut microbiota plays a crucial part in regulating immune function. It produces a wide range of fermentation products, including short-chain fatty acids (SCFAs) derived from dietary fiber and mucin glycans, as well as indoles and hydroxy acids. Interestingly, the gut microbiota is often referred to as a „hidden organ” because of its significant influence on the host’s body function (Kim et al., 2016).

Evidence suggests that dysbiosis may lead to intestinal barrier dysfunction, commonly referred to as a „leaky gut” (Incefi et al., 2022). Reduced SCFA levels, particularly butyrate, weaken tight junctions that maintain epithelial integrity (Kelly et al., 2015). Reduced SCFAs weaken tight junctions. This allows bacterial molecules, such as lipopolysaccharide (LPS), to enter the bloodstream and activate the immune system. The process causes overactivation of immune cells and increased production of pro-inflammatory cytokines, including interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF- α) and interleukin-1 beta (IL-1 β). These cytokines can cross the blood-brain barrier. There, they may impair CNS function by reducing hippocampal neurogenesis, activating microglia and affecting serotonin metabolism (Felger and Lotrich, 2013). These changes can contribute to depression symptoms, including low mood, anxiety, cognitive problems and anhedonia, which is the inability to feel pleasure in normally enjoyable activities.

Chronic activation of the HPA axis leads to a prolonged increase in cortisol levels. Elevated cortisol can cause neurodegeneration, neurotransmitter imbalance and impaired emotional regulation, which are characteristic features of depression (Pariante and Lightman, 2008). Clinically, patients with elevated inflammatory activity often report somatic symptoms such as disturbed sleep, psychomotor slowing and fatigue. Also, they usually do not respond well to typical antidepressants. In such cases, anti-inflammatory lifestyle changes (such as a Mediterranean-style diet, physical activity and maintaining a healthy weight) may help restore microbial diversity and increase SCFA production.

Another point to consider is that astrocytes and endothelial cells play a crucial role in shaping the neuroimmune environment. Inflammation can increase blood-brain barrier permeability, allowing more cytokines to reach the brain and prolong depressive symptoms. This highlights the need for therapies that strengthen the gut barrier and modulate immune responses.

Probiotics, Prebiotics and Psychobiotics in the Context of Depression Treatment

Psychobiotics are specific microbial strains. When given in the right amounts, they may help treat mental health conditions such as depression (Dinan et al., 2013). These microorganisms can influence neurotransmitter metabolism (as discussed above), reduce inflammation and modulate the HPA axis. *Lactobacillus* and *Bifidobacterium* are two of the most studied psychobiotics. Clinical evidence shows that supplementation with a probiotic containing *Lactobacillus helveticus* R0052 and *Bifidobacterium longum* R0175 can reduce depressive symptoms in people under chronic stress. Strains of *L. helveticus* also improve sleep quality in elderly patients (Messaoudi et al., 2011). Researchers observed a similar effect in patients with clinically diagnosed depression, where probiotic use was associated with reduced inflammation and improved overall mood (Gawlik-Kotelnicka, et al., 2021).

Prebiotics are dietary compounds that help beneficial gut bacteria grow. Fructo oligosaccharides and galacto oligosaccharides, for example, increase *Bifidobacterium* and may improve gut-brain communication (Schmidt et al., 2015). Postbiotics, which are the bioactive metabolites beneficial to the host produced by the gut microbiota, are another interesting area of study. In the context of depression, SCFAs have shown anti-inflammatory and neuroprotective effects and may support neurotransmitter production (Silva et al., 2020).

Importantly, selecting a microbiota-targeted therapeutic strategy should consider the patient's habitual diet, including fiber intake and the diversity of plant-based foods. Other factors to consider are the patient's health conditions (such as irritable bowel syndrome or metabolic syndrome) and individual preferences. Adherence to recommended changes is typically higher when individuals incorporate interventions into everyday habits, such as increasing consumption of fermented foods, whole grains and legumes, rather than relying only on supplementation.

Future of Microbiota-Targeted Therapy – Challenges and Research Directions

Ongoing research on gut microbiota in depression has opened new ways to develop personalized treatments. Although the research on psychobiotics, probiotics and prebiotics is promising, their clinical application remains limited to specific patient groups. Patient responses to treatment differ because of diet, lifestyle, genetics and, most importantly, gut microbiota composition. To handle this variability, researchers examine the gut microbiota of each patient. This helps select the most suitable probiotic (Johnson and Foster, 2018).

One promising area of research is the development of next-generation psychobiotics. These are genetically modified bacterial strains that can produce neurotransmitters like GABA or serotonin at levels useful for therapy (Sharma et al., 2021). Dietary modulation also represents a key component of microbiota-targeted therapy. Future clinical studies should include diverse patient populations and consider lifestyle and behavioral factors. They should also assess the long-term safety and effectiveness of these treatments. Advancing this field will require study designs that integrate multi-omics data (such as genomics, metabolomics and proteomics).

Ethical aspects will also play a critical role in the future implementation of microbiota-based therapies. For example, everyone should have a fair chance to receive these treatments. Patients must agree to have their gut bacteria studied. Clear rules are also needed for using specially engineered bacteria in treatment.

Another important consideration is determining the optimal timing for using gut-focused treatments, either at the beginning of depression care or later on. Another question is how to combine these treatments with therapy, better sleep, or exercise. Such combinations may improve resilience, speed up recovery and lower the risk of relapse.

In this review, we tried to collect current evidence on the relationship between the gut microbiota and depression. We focused on its role in the gut-brain axis, neurotransmitter regulation, immune response and inflammatory processes. The reviewed data support the hypothesis that dysbiosis in gut microbiota may contribute to the onset and persistence of depressive symptoms through several connected mechanisms. These include dysregulation of neurotransmitter metabolism, increased inflammation and alteration in the HPA axis activity. However, besides the increasing evidence connecting microbiota imbalance to depression, the clinical applicability remains not fully understood yet.

Several studies suggest that probiotics can reduce inflammation and support neurotransmitter production through the gut microbiota. However, the therapeutic effects vary depending on strain selection, dosage and individual patient factors, including diet, stress and comorbidities. Therefore, a personalized approach to microbiota-targeted therapies appears necessary.

Current evidence shows that dietary and lifestyle strategies can increase microbial diversity. They may also support conventional treatments for depression. Despite promising results, many studies remain insufficient. Ethical issues are important. Patients must give consent for microbiome testing. Rules are needed for using engineered bacteria. These points are crucial as these therapies reach clinical use. The main effects and functions of gut microbiota in the pathogenesis of depression in Table 1 for clarity.

Table 1. Summary of role of gut microbiota in the pathogenesis of depression

| System/Area | Key mechanisms | Research conclusions |
|--------------------------------|---|--|
| Gut–brain axis | Bidirectional neural, hormonal, immune and metabolic communication between the gut and the brain, with a key role of the vagus nerve. | Disturbances within the gut–brain axis may impair emotional regulation, cognitive function and behavior, contributing to depressive symptoms (Rinninella et al., 2019; Dinan and Cryan, 2017; Góralczyk-Bińkowska et al., 2022). |
| Neurotransmitter modulation | Gut microbiota participates in the synthesis and regulation of serotonin, dopamine, GABA, norepinephrine and acetylcholine. | Alterations in microbial composition may lead to neurotransmitter imbalance, resulting in low mood, anhedonia, anxiety and sleep disturbances (Dinan et al., 2015; Lyte, 2011; Reardon, 2014; Cowen and Browning, 2015). |
| Immune system and inflammation | Dysbiosis increases intestinal permeability and promotes the release of pro-inflammatory cytokines such as IL-6, TNF- α and IL-1 β . | Chronic inflammation may impair neurogenesis, activate microglia and exacerbate depressive symptoms while reducing response to standard antidepressant therapy (Kelly et al., 2015; Felger and Lotrich, 2013; Inczeffi et al., 2022). |
| HPA axis dysregulation | Pro-inflammatory cytokines stimulate the hypothalamic–pituitary–adrenal axis, leading to prolonged cortisol secretion. | Sustained HPA axis activation may cause neurodegenerative changes and emotional dysregulation characteristic of major depression (Pariante and Lightman, 2008). |
| Psychobiotics and probiotics | Specific probiotic strains modulate neurotransmitter metabolism, reduce inflammation and influence HPA axis activity. | Supplementation with selected psychobiotics has been associated with reduced depressive symptoms and improved sleep quality, particularly in stressed or depressed individuals (Dinan et al., 2013; Messaoudi et al., 2011; Gawlik-Kotelnicka et al., 2021). |
| Prebiotics and postbiotics | Prebiotics promote the growth of beneficial bacteria, while postbiotics such as SCFAs support gut barrier integrity. | Increased SCFA production exerts anti-inflammatory and neuroprotective effects that may support mood regulation and complement conventional depression treatment (Schmidt et al., 2015; Silva et al., 2020). |

4. CONCLUSION

Current evidence suggests that gut microbiota plays a key role in the development and progression of mental disorders, such as depression. Microbiota-directed strategies include probiotics, prebiotics and diet or lifestyle changes. They may add benefits to conventional drug treatments. More research is needed to confirm their effectiveness. Future research should focus on an individualized approach to the patient. Clinical studies should focus on identifying bacterial strains, determining the correct dose and monitoring patients to ensure both safety and effectiveness. Research into microbiota-related pathways is still in its early stages. Further studies may reshape how we diagnose and treat depression in the future.

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Data and materials availability

All data associated with this study will be available based on reasonable request to the Corresponding Author.

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