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The Gut-Brain Axis: Role of Gut Microbiota in Depression - Systematic Review

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ABSTRACT

The gut-brain axis (GBA) is a bidirectional biochemical communication system that connects the digestive tract and the central nervous system. Emerging studies show that the transmitters released by the gut microbiome can even influence brain development at birth, contributing to many disorders such as anxiety, depression, autism spectrum disorders, and others. With evidence showing the ability of probiotics and dietary interventions to restore the normal gut microbiota balance, their potential therapeutic use in some GBA-caused disorders emerges. This review synthesizes current knowledge and links between GBA dysbiosis and selected mental disorders, showing a potential for GBA-targeted, non-pharmaceutical interventions in their treatment and prevention. Understanding the gut-brain axis dysbiosis presents new ways for more efficient, targeted therapies for some of the mental disorders.

Keywords: gut-brain axis, microbiota, depression, major depressive disorder

1. INTRODUCTION

Mental disorders are emerging as a big social health concern affecting people worldwide. Major depressive disorder (MDD) manifests itself through depressed mood, anhedonia, loss of motivation, weight gain or loss, sleep disorder, energy loss, and suicide. It is brought about by a complex interplay between hereditary factors and environmental factors. As reported in the World Health Organization 2023 fact sheets, approximately 5 % of the global adult population is affected by depression. Depressive disorders made up 56 million disability adjusted life years (DALYs) in 2021, which represents a doubling of the 1990 DALY total (Rong et al., 2025).

The development of depression uses social, psychological, and biological backgrounds, which affect physical health conditions. The severe social and economic impacts of these disorders require a deeper understanding of their disease mechanisms and the development of significant therapeutic approaches. A review

of the existing evidence regarding microbial markers that stimulate the growth of depression and treatment progression, as well as an assessment of how conventional therapies can be improved through gut microbiota-based interventions, such as prebiotics, probiotics, special diets, and fecal microbiota transplantation (FMT), is provided.

2. REVIEW METHODS

For this narrative review, we searched PubMed and Google Scholar using the terms "MDD," "depression," and "microbiota," "gut-brain axis," "microbiome," "short-chain fatty acids," or "probiotics." We included randomized controlled trials, meta-analyses, systematic reviews, observational studies, preclinical and clinical studies, as well as clinical recommendations. The review focuses on research that clarifies microbiota–brain interactions and potential treatments based on this connection. The researchers followed the PRISMA guidelines during the screening of the collected articles. In total, 29 studies were incorporated into the review (Figure 1).

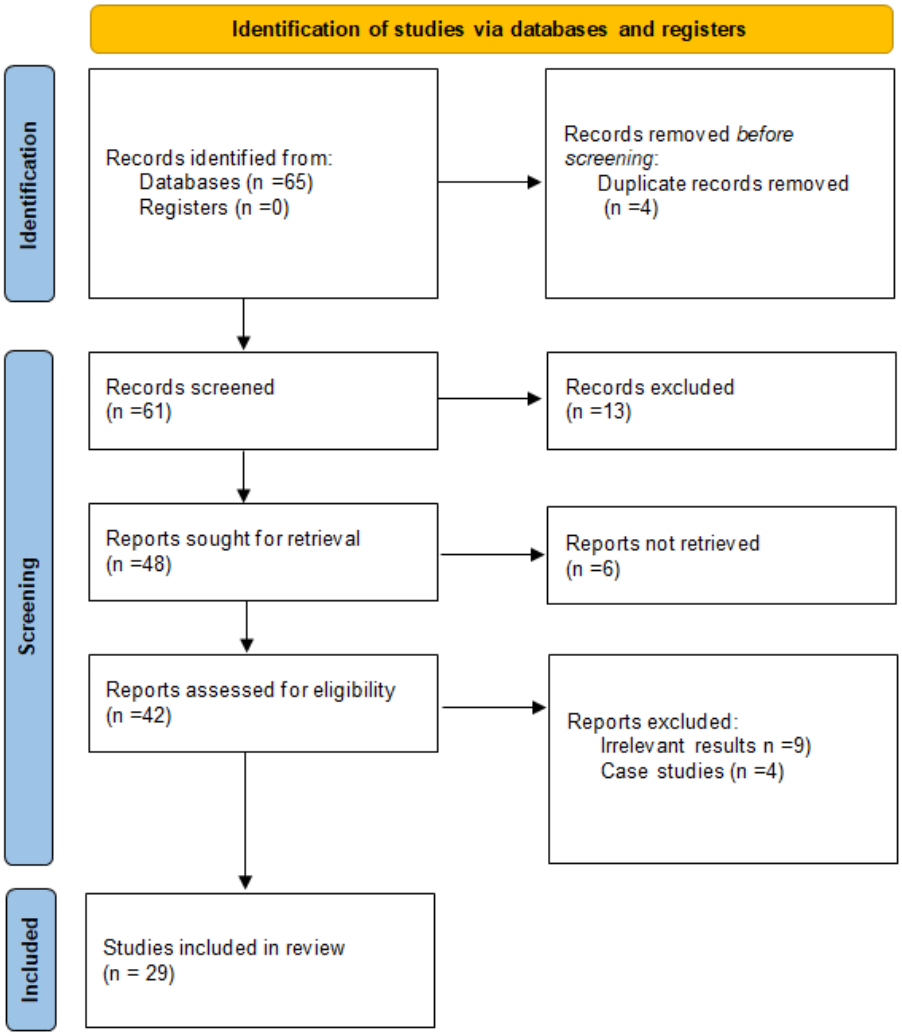

Figure 1. Prisma flow chart

3. RESULTS & DISCUSSION

Depression

The exact molecular and cellular processes that cause depression are complex. Therefore, despite multiple decades of investigation, scientists have not yet fully understood its mode of functioning. The current understanding of depression points to two main categories

of brain problems, which include structural and functional issues that affect monoamine neurotransmitter systems and produce neuroinflammatory responses (Zou et al., 2024). Research indicates that depression relates to three main factors, including decreased cortical thickness and elevated cortisol levels from HPA axis hyperactivation and impaired neuroplasticity through BDNF reduction and blood–brain barrier permeability increase. Modern research shows depression creates a widespread body system disorder that impacts all major physiological networks in addition to the central nervous system. Given depression's multifactorial etiology, it remains a disorder that is challenging to treat effectively.

Many patients struggle because first-line treatment (SSRIs and SNRIs) often takes weeks to work, frequently requires medication adjustments, and remains ineffective for some. Even when supplemented with nonpharmacological interventions (e.g., psychotherapy and noninvasive brain stimulation), 10 % of patients still fail to achieve adequate improvement (Pilon et al., 2019). Since we are still piecing together what drives depression at a molecular level, shifting focus to the GBA could offer fresh insights and point the way to entirely new, personalized treatments (Campaniello et al., 2022).

Microbiota

The trillions of microorganisms that inhabit the human body are collectively referred to as the human microbiota (Pantazi et al., 2023). Antibiotics, stress, and diet are just a few of the environmental variables that can affect the gut microbiota ecosystem. Humans have a unique gut profile that is influenced by their environment, as the gut microbiota is formed during fetal development, and they interact with it from a young age. Because it controls immune system activity, nutrient absorption, and general physiological processes, the microbiota is a key factor in determining our current state of health (Ursell et al., 2012). The microbial community is provided with an environment by the host, which enhances digestion and makes nutrients more readily available. When the balance of intestinal microbes is disrupted, a condition known as dysbiosis occurs, making individuals more susceptible to illness (Eckburg et al., 2005). The Bacteroides enterotype is associated with a diet high in fats and proteins, while the Prevotella enterotype corresponds to diets rich in carbohydrates (Wu and Hui, 2011).

Neurotransmitters, Gut-brain axis, and Bacterial Metabolites

Recent advances also emphasize the key contribution of gut microbiota and the microbiota–gut–brain axis (MGBA) to the determinacy of neural function, as well as cognitive and susceptibility to depression, in both health and disease (Nazir et al., 2025). The MGBA is a complex, bidirectional system that integrates neural, immunologic, and endocrine-metabolic responses simultaneously to regulate CNS activity (Ursell et al., 2012; Colella et al., 2023). At the neural level, microbial products have also been shown to affect neurotransmitter release and signaling. Metabolites derived from tryptophan, including indoles and short-chain fatty acids (SCFA)-butyrate and propionate, have been demonstrated to be integral to governing the gut-interactions with the brain associated with neuroinflammation, synaptic plasticity, and regulation of mood (Spichak et al., 2021; Gheorghe et al., 2019; Dalile et al., 2019).

Integral to communication in the body, the vagus nerve is a critical pathway for communication between the enteric nervous system and limbic circuits that govern stress and emotional responses (Sudo et al., 2004). Interruptions in these signaling chains might be involved in the development of depression and anxiety disorders (Jiang et al., 2015), rather consistently. Metabolites play a crucial role in immune regulation, influencing the balance between anti-inflammatory Treg cells and pro-inflammatory Th17 cells (Erny et al., 2015; Colella et al., 2023). Furthermore, these immunologic alterations are also closely associated with the maintenance of the intestinal epithelia: an altered gut microbiota enables translocation of microbial-related substances that trigger HPA axis activation and are responsible for the systemic chronic inflammatory state (Guida et al., 2018; Herselman et al., 2022). Additionally, the gut microbiota can regulate neurotransmitter biosynthesis, potentially producing analogues that directly bind to the host's receptors (Caspani et al., 2019).

These findings demonstrate that interactions between fluctuations in neurotransmitters and microbial metabolites underpin the biochemical and signaling basis of the microbiome-gut-brain axis (MGBA). Aberrations of these processes—due to microbial dysbiosis, inflammation, or dysfunctional metabolite signaling—feature in the pathophysiology of depression, highlighting the potential of the microbiome to serve as both a marker of and target for therapeutic intervention in mood disorders (Rong et al., 2025; Nazir et al., 2025).

Depression and Gut Microbiota

Researchers have used various methods to determine the composition of the gut microbiota, including stool cultures, endoscopic aspiration of intestinal fluid, and surgical samples. Nevertheless, bacterial sampling does not fully reflect the composition of their metabolites, which are often affected by other regulatory mechanisms, such as post-transcriptional regulation and pathway

interactions. Metabolic profiling and metabolomic technologies have thus been incorporated into more gut microbial analysis (Tang et al., 2020).

Disrupted barrier integrity during dysbiosis allows bacterial products, such as lipopolysaccharide, to enter the circulation. That can trigger systemic cytokine release and microglial activation in the brain (Erny et al., 2015). Germ-free animals demonstrate abnormal stress reactivity and behavioral alterations that emphasize the gut microbiota's critical role in regulating CNS functions (Dalile et al., 2019). In animal models, microbiota depletion with antibiotics attenuates neuroinflammation and improves outcomes in multiple sclerosis and Alzheimer's disease (Minter et al., 2017). In contrast, targeted probiotics reduce the pro-inflammatory cytokine response and normalize microglial activity (Wang and Kasper, 2014). Human studies likewise associate specific microbial signatures with inflammatory biomarkers in major depressive disorder and autism spectrum disorder. The exact microbial metabolites and host pathways involved, however, remain incompletely defined (Jiang et al., 2015). The gathered data show that the gut microbiome appears to be a promising therapeutic target for future precision-based treatments of depression. Bifidobacterium and Lactobacillus commensal strains produce anxiolytic and antidepressant effects through vagal afferent signaling, which modulates serotonergic and GABAergic neurotransmission (Sudo et al., 2004).

Depression and gut–brain axis

Compositional modifications in the gut microbiota of patients suffering from major depressive disorder (MDD) compared to healthy controls have been demonstrated in research done by Naseribafrouei et al., (2014). Psychological stress has the capacity to modify the composition of the gut microbiota, and dysbiosis of this microbial group, in turn, can affect emotional behaviors. Evidence is the increased prevalence of anxiety, psychosis, and depression, which has been observed even up to a distance of 5-10 years after antibiotic use. One recent analysis reported a correlation of antibiotic exposure with subsequent depression, likely as a result of the reduction in gut microbial diversity by antibiotics. Research has shown that the fecal microbiota of patients suffering from depressive disorder is altered (Guida et al., 2018). The reported differences include Bacteroidetes, Proteobacteria, Actinobacteria, and Firmicutes, as well as the genera Enterobacteriaceae, Alistipes, Faecalibacterium, Bifidobacterium, and Blautia. An unhealthy diet and environmental exposures that modify the structure of gut microbiota apparently leads to the new rise of depression (Herselman et al., 2022).

Accumulating evidence has linked the gut microbiota to the pathophysiology of MDD. Consequently, the impact of the gut microbiota on the efficacy of antidepressants should receive equal attention. The effects of probiotics, prebiotics, and faecal microbiota transplantation on depression, including MDD, were investigated in a few randomised controlled trials (Zhang et al., 2023). Scientists evaluated these interventions using standardized depression rating scales. Most of them involved probiotics supplementation using strains of Lactobacillus and Bifidobacterium. Many RCTs reported that probiotic supplementation- often as an adjunct to conventional pharmacotherapy led to significant alleviation of depressive symptoms compared to placebo. A meta-analysis of 19 trials (1,405 participants) showed a substantial reduction in mean depression scale scores in patients receiving probiotics or symbiotics versus the control group. An observation revealed a minimal impact on mood in healthy patients (Desbonnet et al., 2008).

Prebiotics, in contrast to probiotics, are substrates that promote the growth of select, beneficial bacteria. The few RCTs conducted to date have not provided convincing evidence for alleviating depression symptoms. The most recent meta-analysis showed that prebiotic supplements did not show any better results than a placebo in treating depression in patients. The effectiveness of synbiotics as a treatment for depression appears to stem from their probiotic content, as they produce results that match those of probiotics alone. Fecal Microbiota Transplantation (FMT) is a relatively new and understudied approach in the treatment of depressive disorders. Until 2023, only one human trial on FMT in depression had been conducted. The first reports appeared only recently, and are mostly preliminary in nature (Green et al. 2023)

Experimental studies in rat models have demonstrated that administering Bifidobacterium infants can reverse stress-induced hyperactivation of the HPA axis and reduce depression-like behavior. A 14-day supplementation increased plasma tryptophan and kynurenic acid levels, and reduced cortical 5-HIAA and amygdaloid OPAC, alongside the suppression of pro-inflammatory cytokines IFN-gamma, TNF-alpha, and IL-6 (Desbonnet et al., 2008). The results, considering the most significant trials, are presented in Table 1.

Table 1. Results of interventions related to gut microbiota in the treatment of depression

Intervention	Reference Study	Outcome	Notes
Probiotics (e.g., *Lactobacillus*, *Bifidobacterium*)	Zhang et al., 2023; Desbonnet et al., 2008	Significant reduction in depressive symptoms, especially when combined with antidepressants	Improved depression scores in patients; minimal effect on healthy individuals
Probiotic *Bifidobacterium infants* (animal studies)	Desbonnet et al., 2008	Reduced depressive symptoms	Reversed stress-induced HPA hyperactivation; Increased plasma tryptophan, Suppressed pro-inflammatory cytokines;
Synbiotics (probiotics + prebiotics)	Zhang et al., 2023	Comparable to probiotics alone in efficacy	Prebiotic role remains unclear
Prebiotics	Zhang et al., 2023	No significant improvement vs. placebo	Inconclusive evidence for clinical effectiveness
Fecal Microbiota Transplantation (FMT)	Zhang et al., 2023; Guida et al., 2018	Potential antidepressant value	Mostly early-stage and exploratory research
Antibiotic-induced microbiota depletion (animal studies)	Minter et al., 2017	Improved outcomes in neurodegenerative models	Reduced neuroinflammation; only demonstrated in multiple sclerosis and Alzheimer's disease models
Antibiotic	Liu et al., 2022	Increased depression incidence up to 5–10 years later	Likely due to long-term disruption of gut microbial diversity

HPA: Hypothalamic-Pituitary-Adrenal Axis

4. CONCLUSION

The connection between gastrointestinal microbiota and depression is modifying our understanding of disease development and progression, leading to new opportunities for biomarker development and therapeutic interventions that target the modification of microbial communities. Multiple RCTs and animal studies demonstrate that probiotics could positively influence patients with depression. The clinical value of gut microbiota-based treatments for depression remains unclear because different studies have produced conflicting results. Probiotics and dietary interventions provide noninvasive treatment options that can elevate the effectiveness of antidepressants as well as minimize side effects. Future research should focus on studying drug-microbiota interactions in patients who receive multiple medications, rather than relying on single-agent studies. The development of strong microbiota-based diagnostic and prognostic biomarkers requires the creation of predictive and prognostic tools from blood, feces, and urine samples. The development of optimal probiotic formulations requires defining the most suitable strains, dosages, and timing of administration, as well as the administration of antidepressants.

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Informed consent

Not applicable.

Ethical approval

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Conflict of interest

The authors declare that there is no conflict of interest.

Data and materials availability

All data associated with this work are present in the paper.

REFERENCES

1. Campaniello D, Corbo MR, Sinigaglia M, Speranza B, Racioppo A, Altieri C, Bevilacqua A. How diet and physical activity modulate gut microbiota: evidence, and perspectives. Nutrients 2022;14:2456. doi:10.3390/nu14122456.
2. Caspani G, Kennedy S, Foster JA, Swann J. Gut microbial metabolites in depression: understanding the biochemical mechanisms. Microb Cell 2019;6(10):454-81. doi: 10.15698/mic2019.10.693.
3. Colella M, Charitos IA, Ballini A, Cafiero C, Topi S, Palmirotta R, Santacroce L. Microbiota revolution: how gut microbes regulate our lives. World J Gastroenterol 2023;29(28):4368-83. doi: 10.3748/wjg.v29.i28.4368.
4. Dalile B, Van Oudenhove L, Vervliet B, Verbeke K. The role of short-chain fatty acids in microbiota-gut-brain communication. Nat Rev Gastroenterol Hepatol 2019;16(8):461-478. doi:10.1038/s41575-019-0157-3.
5. Desbonnet L, Garrett L, Clarke G, Bienenstock J, Dinan TG. The probiotic Bifidobacteria infantis: an assessment of potential antidepressant properties in the rat. J Psychiatr Res 2008;43(2):e34-9. doi:10.1016/j.jpsychires.2008.03.009.
6. Eckburg PB, Bik EM, Bernstein CN, Purdom E, Dethlefsen L, Sargent M, Gill SR, Nelson KE, Relman DA. Diversity of the human intestinal microbial flora. Science 2005;308(5728):1635-8. doi: 10.1126/science.1110591.
7. Erny D, Hrabě de Angelis AL, Jaitin D, Wieghofer P, Staszewski O, David E, Keren-Shaul H, Mahlakoiv T, Jakobshagen K, Buch T, Schwierzeck V, Utermöhlen O, Chun E, Garrett WS, McCoy KD, Diefenbach A, Staeheli P, Stecher B, Amit I, Prinz M. Host microbiota constantly controls maturation and function of microglia in the CNS. Nat Neurosci 2015;18(7):965-77. doi:10.1038/nn.4030.
8. Gheorghe CE, Martin JA, Manriquez FV, Dinan TG, Cryan JF, Clarke G. Focus on the essentials: tryptophan metabolism and the microbiome-gut-brain axis. Curr Opin Pharmacol 2019;48:137-145. doi: 10.1016/j.coph.2019.08.004.

9. Green JE, Berk M, Mohebbi M, Loughman A, McGuinness AJ, Castle D, Chatterton ML, Perez J, Strandwitz P, Athan E, Hair C, Nierenberg AA, Cryan JF, Jacka F. Feasibility, acceptability, and safety of faecal microbiota transplantation in the treatment of major depressive disorder: a pilot randomized controlled trial. Can J Psychiatry 2023;68(5):315-26. doi:10.1177/07067437221150508.
10. Guida F, Turco F, Iannotta M, De Gregorio D, Palumbo I, Sarnelli G, Furiano A, Napolitano F, Boccella S, Luongo L, Mazzitelli M, Usiello A, De Filippis F, Iannotti FA, Piscitelli F, Ercolini D, de Novellis V, Di Marzo V, Cuomo R, Maione S. Antibiotic-induced microbiota perturbation causes gut endocannabinoidome changes, hippocampal neuroglial reorganization and depression in mice. Brain Behav Immun 2018;67:230-45. doi:10.1016/j.bbi.2017.09.001.
11. Herselman MF, Bailey S, Bobrovskaya L. The effects of stress and diet on the "brain-gut" and "gut-brain" pathways in animal models of stress and depression. Int J Mol Sci 2022;23(4):2013. doi:10.3390/ijms23042013.
12. Jiang H, Ling Z, Zhang Y, Mao H, Ma Z, Yin Y, Wang W, Tang W, Tan Z, Shi J, Li L, Ruan B. Altered fecal microbiota in patients with major depressive disorder. Brain Behav Immun 2015;48:186-94. doi:10.1016/j.bbi.2015.03.016.
13. Liu L, Wang H, Zhang H, Chen X, Zhang Y, Wu J, Zhao L, Wang D, Pu J, Ji P, Xie P. Toward a deeper understanding of gut microbiome in depression: the promise of clinical applicability. Adv Sci (Weinh) 2022;9(22):2203707. doi:10.1002/advs.202203707.
14. Minter MR, Hinterleitner R, Meisel M, Zhang C, Leone V, Zhang X, Oyler-Castrillo P, Zhang X, Musch MW, Shen X, Jabri B, Chang EB, Tanzi RE, Sisodia SS. Antibiotic-induced perturbations in microbial diversity during post-natal development alters amyloid pathology in an aged APP/PS1 murine model of Alzheimer's disease. Sci Rep 2017;7(1):10411. doi:10.1038/s41598-017-11047-w.
15. Naseribafrouei A, Hestad K, Avershina E, Sekelja M, Linløkken A, Wilson R, Rudi K. Correlation between human fecal microbiota and depression. Neurogastroenterol Motil 2014;26(8):1155-62. doi:10.1111/nmo.12378.
16. Nazir MM, Ghaffar W, Mustafa G, Saeed S, Ijaz MU, Ashraf A. Modulating depression through the gut-brain axis: the role of gut microbiota in therapeutic interventions. Naunyn Schmiedebergs Arch Pharmacol 2025. doi: 10.1007/s00210-025-04464-6.
17. Pantazi AC, Balasa AL, Mihai CM, Chisnoiu T, Lupu VV, Kassim MAK, Mihai L, Frecus CE, Chirila SI, Lupu A, Andrusca A, Ionescu C, Cuzic V, Cambrea SC. Development of gut microbiota in the first 1000 days after birth and potential interventions. Nutrients 2023;15(16):3647. doi: 10.3390/nu15163647.
18. Pilon D, Joshi K, Sheehan JJ, Zichlin ML, Zuckerman P, Lefebvre P, Greenberg PE. Burden of treatment-resistant depression in Medicare: a retrospective claims database analysis. PLOS One 2019;14(10):e0223255.
19. Rong J, Wang X, Cheng P, Li D, Zhao D. Global, regional and national burden of depressive disorders and attributable risk factors, from 1990 to 2021: results from the 2021 Global Burden of Disease Study. Br J Psychiatry 2025;1-10. doi: 10.1192/bjp.2024.266.
20. Spichak S, Bastiaanssen TFS, Berding K, Vlckova K, Clarke G, Dinan TG, Cryan JF. Mining microbes for mental health: determining the role of microbial metabolic pathways in human brain health and disease. Neurosci Biobehav Rev 2021;125:698-761. doi: 10.1016/j.neubiorev.2021.02.044.
21. Sudo N, Chida Y, Aiba Y, Sonoda J, Oyama N, Yu XN, Kubo C, Koga Y. Postnatal microbial colonization programs the hypothalamic-pituitary-adrenal system for stress response in mice. J Physiol 2004;558(Pt1):263-75. doi:10.1113/jphysiol.2004.063388.
22. Tang Q, Jin G, Wang G, Liu T, Liu X, Wang B, Cao H. Current sampling methods for gut microbiota: a call for more precise devices. Front Cell Infect Microbiol 2020;10:151. doi:10.3389/fcimb.2020.00151.
23. Ursell LK, Metcalf JL, Parfrey LW, Knight R. Defining the human microbiome. Nutr Rev 2012;70 Suppl 1(Suppl 1): S38-44.
24. Wang Y, Kasper LH. The role of microbiome in central nervous system disorders. Brain Behav Immun 2014;38:1-12. doi:10.1016/j.bbi.2013.12.015.
25. Wu SV, Hui H. Treat your bug right. Front Physiol 2011;2:9. doi: 10.3389/fphys.2011.00009.
26. Zhang Q, Chen B, Zhang J, Dong J, Ma J, Zhang Y, Jin K, Lu J. Effect of prebiotics, probiotics, synbiotics on depression: results from a meta-analysis. BMC Psychiatry 2023;23(1):477. doi:10.1186/s12888-023-04963-x.
27. Zou Y, Zhang Y, Tu M, Ye Y, Li M, Ran R, Zou Z. Brain-derived neurotrophic factor levels across psychiatric disorders: a systematic review and network meta-analysis. Prog Neuropsycho pharmacol Biol Psychiatry 2024;131:110954. doi: 10.1016/j.pnpbp.2024.110954.