Quality in Sport
Nicolaus Copernicus University in Torun, Poland

eISSN 2450-3118 | Unique Journal Identifier: 201398
20 pts — Ministry of Higher Education and Science of Poland

ŚLINKO, Izabela, PANAS, Zuzanna, DRYL-JARMOC, Ewa, KULIŚ, Gabriela, ZIMOWSKI, Mateusz, DZIEMIAŃCZUK, Jakub, CZAJKOWSKI, Mateusz, KRAWCZUK, Kacper, KADYSZ, Piotr and KOLANEK, Agata. Gut–Brain Axis in Depression: A Narrative Review of Microbiome Signatures, Mechanisms, Biomarkers, and Microbiota-Targeted Interventions. Quality in Sport. 2026;53:70176. eISSN 2450-3118. https://doi.org/10.12775/QS.2026.53.70176

Received: 23.03.2026 Revised: 30.03.2026 Accepted: 30.03.2026 Published: 04.04.2026

Published by Nicolaus Copernicus University in Toruń, Poland | eISSN 2450-3118 | Unique Journal Identifier: 201398 | 20 pts (Ministry of Higher Education and Science of Poland, 05.01.2024, No. 32553).

Gut-Brain Axis in Depression: A Narrative Review of Microbiome Signatures, Mechanisms, Biomarkers, and Microbiota-Targeted Interventions

Izabela Slinkoo[1]	ORCID: 0009-0002-5953-5168	izabelaslinkoo@gmail.com
Zuzanna Panas[1]	ORCID: 0009-0009-6850-4110	zuzia.panas@wp.pl
Ewa Dryl-Jarmoc[1]	ORCID: 0009-0007-2946-0800	ewa.dryl96@gmail.com
Gabriela Kulis[1]	ORCID: 0009-0006-8136-2661	gabriela.kulis1356@gmail.com
Mateusz Zimowski[1]	ORCID: 0009-0003-0280-343X	mateuszzimowski@icloud.com
Jakub Dziemiańczuk[1]	ORCID: 0009-0007-7454-3111	dziemianczukjakub@gmail.com
Mateusz Czajkowski[1]	ORCID: 0009-0001-2924-694X	matczajkowski24@gmail.com
Kacper Krawczuk[1]	ORCID: 0009-0009-3089-6323	kacperkrawczuk@gmail.com
Piotr Kadysz[1]	ORCID: 0009-0004-8952-3103	lek.kadysz.piotr@gmail.com
Agata Kolanek[2]	ORCID: 0009-0002-0937-2938	agkolanek@gmail.com

1 Faculty of Medicine, Medical University of Bialystok, Bialystok, Poland

2 Department of Psychiatry, Medical University of Bialystok, Bialystok, Poland

Corresponding Author: Izabela Slinkoo — izabelaslinkoo@gmail.com

ABSTRACT
Background. The microbiota-gut-brain axis (MGBA) is a key pathway in the pathophysiology of depression, integrating neural, immune, and metabolic mechanisms and influencing processes relevant to mood regulation.
Aim. This narrative review summarizes current evidence on the role of the MGBA in depression, including microbial composition, functional pathways, biomarkers, and therapeutic interventions.
Material and methods. A literature search was conducted in PubMed and PMC (2018-March 2026, including key earlier studies). The review focused on microbial alterations, metagenomic functions, intestinal barrier and immune biomarkers, microbial metabolites, and clinical trials.
Results. Cohort studies show associations between depressive symptoms and gut microbial taxa involved in neuroactive pathways; however, findings are heterogeneous and of limited diagnostic value. Experimental studies support causality, as microbiota from patients with major depressive disorder induce depression-like behaviors in rodents. Vagal nerve integrity appears necessary for some microbiota-mediated effects, and stress-related dysbiosis may contribute to microglial activation and impaired neurogenesis. Biomarker studies indicate increased intestinal permeability and elevated markers of microbial translocation (e.g., zonulin, LPS-binding protein, sCD14), though their clinical utility remains unclear. Clinical trials suggest probiotics may reduce depressive symptoms as adjunctive therapy, while evidence for prebiotics is inconsistent and fecal microbiota transplantation remains experimental.
Conclusions. The MGBA plays an important role in depression, but further standardized and longitudinal studies are needed to clarify mechanisms and support clinical applications, particularly for personalized therapies.
Key words: *gut-brain axis; gut microbiota; depression; inflammation; probiotics; biomarkers.*

1. INTRODUCTION

Depression is among the most common mental disorders, and current treatments have variable effectiveness. This motivates study of modifiable biological systems that may influence disease risk, symptom persistence, and treatment resistance. Population studies have linked depressive symptoms to gut microbiome variation. For example, a large multi-cohort analysis reported associations between multiple taxa and neuroactive metabolic pathways (glutamate, butyrate, serotonin, GABA) in depressed versus control groups [1]. These findings align with models in which depression arises from interacting neuroimmune, neuroendocrine, metabolic, and neuroplastic factors, rather than a single neurotransmitter deficit [2].

The MGBA framework posits bidirectional communication among intestinal microbes, barrier function, immunity, neuroendocrine signals, and brain processes relevant to mood and cognition. Foundational reviews highlight immune signaling, tryptophan metabolism, and vagal/enteric pathways as key conduits, along with microbial metabolites (e.g. SCFAs) that can modulate host physiology and neuroimmune responses. In depression-specific syntheses, activation of the hypothalamic-pituitary-adrenal (HPA) axis and cortisol effects on gut integrity and microbiota are frequently emphasized, alongside bacterial endotoxin-driven immune signaling [2].

This review summarizes mechanistic and clinical evidence on the MGBA in depression, including topics required for contemporary narrative reviews: functional metagenomics, barrier biomarkers (zonulin, LPS-binding protein, etc.), tryptophan-kynurenine biology, BDNF and neuroplasticity, microglial priming, patient stratification, and gaps in evidence. Claims about therapeutic efficacy are limited to findings from randomized trials or systematic syntheses.

2. MATERIALS AND METHODS

This narrative review was conducted to synthesize current evidence on the microbiota-gut-brain axis (MGBA) in depression. The literature search was performed between January and March 2026 using two major scientific databases: PubMed and PubMed Central. The search strategy combined keywords and MeSH terms for depressive disorders and MGBA-related concepts. We prioritized publications from 2018 to 2026 but also included key older mechanistic studies (for example, Kelly et al. 2016 on fecal transplant).

Inclusion criteria: Original research articles or reviews in peer-reviewed journals; human clinical studies (including cohort studies and RCTs) or mechanistic animal studies relevant to depression; reported outcomes related to microbiota composition, microbial metabolites, barrier biomarkers, immune markers, or brain-related endpoints; published in English, with full-text available.

Exclusion criteria: Focused on non-depressive psychiatric disorders or did not involve gut-brain interactions; conference abstracts, editorials, or case reports without primary data; used non-microbiome interventions.

All retrieved records were screened by title and abstract for relevance, and duplicates were removed. In total, the search yielded approximately 100 records, of which 29 key studies fulfilled our criteria and were included in the final synthesis.

3. RESULTS

A structured summary of key studies investigating the microbiota-gut-brain axis in depression is presented in Table 1.

Table 1. Summary of selected studies investigating the microbiota-gut-brain axis in depression

Author	Year	Study design	Sample	Focus / Intervention	Outcomes
Radjabzadeh et al.	2022	Cohort study	Large multi-cohort population	Gut microbiome analysis	Depressive symptoms associated with distinct microbial profiles in neuroactive pathways.
Liang et al.	2018	Meta-analysis	8 cohorts	16S rRNA analysis	No consistent alpha diversity differences; limited diagnostic value.
Yang et al.	2020	Multi-omics study	MDD patients	Shotgun metagenomics + metabolomics	Altered microbial genes and metabolites in amino acid/neurotransmitter pathways.
Morena et al.	2025	SR & meta-analysis	Multiple studies	Permeability biomarkers	Increased zonulin, LBP, sCD14 in depressed patients.
Ting et al.	2020	Narrative review	Clinical/experimental	Cytokine signaling	Elevated IL-6 linked to stress and depressive symptoms.
Siopi et al.	2023	Animal study	Rodent model	Vagus nerve signaling	Microbiota-induced depressive behaviour requires intact vagus nerve.
Heidarzadeh-Rad et al.	2020	RCT (post-hoc)	Depressed patients	Psychobiotic supplement	Reduced depressive symptoms; increased serum BDNF.
Kazemi et al.	2019	RCT	110 MDD patients	Probiotic vs prebiotic vs placebo	Probiotic significantly reduced depressive symptom severity.
Schaub et al.	2022	RCT	Depressed patients	Probiotic add-on	Improved symptoms; changes in microbiome and neural activity.
Green et al.	2023	Pilot RCT	15 patients	FMT	FMT safe and feasible; preliminary QoL improvements.

3.1 Microbiome Alterations in Depression

Large population studies identify associations between depressive symptoms and gut microbiome composition, with several taxa consistently linked across cohorts. In one microbiome-wide association study of two cohorts, thirteen taxa correlated with depression severity; many are involved in synthesizing neuroactive compounds (glutamate, butyrate, serotonin, GABA) [1].

In contrast, a multi-cohort meta-analysis of 16S rRNA profiles (eight independent cohorts) found no consistent differences in gut microbial alpha diversity between depressed and healthy groups. Some differences in beta diversity and a few genus-level markers emerged, but predictive power was poor when tested on independent data [3]. These findings suggest that "dysbiosis" in depression is not a uniform loss of diversity but context-dependent shifts in community structure.

Antidepressant use is an important confounder. A systematic review of probiotic trials in MDD noted that dietary factors are often inadequately controlled and microbiome changes are inconsistently observed even when symptoms improve [4].

3.2 Functional Metagenomics and Microbial Neuroactive Potential

Beyond taxonomy, functional metagenomics aims to characterize microbial genes and metabolic pathways relevant to host neurobiology. Large-scale shotgun metagenomic studies underscore the promise of this approach but also its limitations due to incomplete reference databases and challenges in annotating neuroactive pathways [5].

Integrative multi-omic studies combine metagenomics with metabolomics to link microbes and metabolites. For example, a study using shotgun sequencing and untargeted metabolomics in MDD patients found differences in bacteriophages, bacterial species, and fecal metabolites. Altered genes and metabolites mapped to amino-acid pathways (including GABA and tryptophan metabolism) [6].

3.3 Intestinal Barrier Dysfunction and Translocation Biomarkers

Disruption of gut barrier integrity is hypothesized to allow microbial products (e.g. lipopolysaccharide, LPS) to enter circulation, triggering systemic inflammation that can affect the brain. A recent systematic review and meta-analysis quantified "leaky gut" biomarkers in depression, including zonulin, LPS-binding protein (LBP), antibodies to endotoxins, soluble CD14, and intestinal fatty acid-binding protein (I-FABP) [7]. Several markers were elevated in depressed patients versus controls.

However, findings across studies are heterogeneous. A narrative review of these blood biomarkers found inconsistent results for zonulin, I-FABP, LPS, LBP, and sCD14, concluding that routine clinical use is not supported [8].

3.4 Immune Signaling Across the MGBA: Cytokines, IL-6, and Inflammation-Associated Subtypes

Immune signaling is central to the MGBA in depression. A comprehensive review in Science described how gut- and systemic-derived inflammation is communicated across the gut-brain axis, including cytokine-mediated neural and humoral pathways [9].

In depression specifically, interleukin-6 (IL-6) is a prominent biomarker. Reviews show that elevated IL-6 is linked to stress responses and depressive symptoms. Profiling IL-6 could help identify inflammatory subtypes of depression for targeted treatment [10]. This aligns with "immunometabolic" frameworks that cluster symptoms of atypical depression with inflammation and metabolic dysregulation [11].

3.5 Metabolic Interfaces: SCFAs and Tryptophan-Kynurenine Biology

Short-chain fatty acids (SCFAs), particularly butyrate, are key microbial metabolites with immunomodulatory roles. In the PROVIT trial, stool metabolomics showed increased butyrate levels after four weeks in the probiotic group [12]. MGBA reviews cite SCFAs among metabolites that influence host immunity and potentially brain function [2].

Tryptophan metabolism links the microbiota to host serotonin and kynurenine pathways, especially under inflammation. Reviews outline how gut microbes regulate tryptophan availability and metabolites, affecting serotonin synthesis and producing kynurenine derivatives implicated in depression [13, 14]. Experimental evidence supports this [15]. Clinical RCTs have also measured kynurenine metabolites as endpoints [16].

3.6 Neural and Stress-System Routes: Vagus-Dependent Effects and HPA Activation

The vagus nerve conveys gut signals to the brain without crossing the blood-brain barrier. In rodents, disruption of the vagus nerve blocks certain microbiota-driven behavioral effects: one study found that stress-altered microbiota could induce depressive behaviors only if the vagus was intact [17].

Depression-focused reviews highlight HPA-axis activation (CRH → ACTH → cortisol) and downstream effects on gut function and microbiota composition [18]. General MGBA syntheses likewise emphasize that stress alters the microbiome, contributing to a feedback loop of microbiota-driven inflammation and neuroendocrine changes [2].

3.7 Neuroplasticity, BDNF, and Microglial Priming

A post hoc analysis of an RCT found that an eight-week probiotic supplement (Bifidobacterium longum + Lactobacillus helveticus) improved depression symptoms and was associated with increased serum BDNF levels, suggesting a neurotrophic effect [19].

A murine study showed that transplanting microbiota from stress-exposed donors induced microglial priming in the hippocampal dentate gyrus of recipients, leading to exaggerated inflammatory responses and impaired neurogenesis under stress [20]. These findings indicate a pathway from stress-altered microbiota to neuroimmune changes and compromised neuroplasticity.

The above findings highlight multiple interacting biological pathways linking the gut microbiota to depression. These mechanisms are summarized in Table 2.

Table 2. Microbiota-related mechanisms implicated in depression

Mechanism	Key components	Biological pathway	Effect on brain	Clinical relevance
Microbiome composition changes	Gut bacterial taxa (SCFA-producing)	Altered microbial metabolism	Association with depressive symptoms; no consistent diversity loss	Potential biomarkers; limited diagnostic accuracy
SCFAs	Butyrate, acetate, propionate	Microbial fermentation → immune/metabolic signaling	Modulation of inflammation and neuroplasticity	Therapeutic targets (diet, probiotics)
Tryptophan-kynurenine pathway	Tryptophan, serotonin, kynurenine	Microbial regulation of amino acid metabolism	Altered serotonin; neurotoxic metabolites	Key microbiota-neurotransmission link
Intestinal barrier dysfunction	Zonulin, LBP, sCD14	Increased gut permeability ('leaky gut')	Microbial translocation → systemic inflammation	Promising biomarkers; not yet validated
Immune activation	IL-6, cytokines	Peripheral immune signaling to CNS	Neuroinflammation and depressive symptoms	Identifies inflammatory depression subtypes
Vagus nerve signaling	Vagus nerve integrity	Neural gut-brain communication	Modulation of mood and stress response	Explains rapid gut-brain signaling
HPA axis activation	Cortisol, CRH, ACTH	Stress response x gut interaction	Increased gut permeability; microbiota changes	Links stress and microbiota
Neuroplasticity / BDNF	BDNF, hippocampal neurogenesis	Microbiota → neurotrophic signaling	Impaired neuroplasticity in depression	Marker of treatment response
Microglial activation	Microglia, inflammatory mediators	Gut-driven CNS immune activation	Neuroinflammation; impaired neurogenesis	Important in chronic/stress depression

3.8 Clinical Interventions: Probiotics, Prebiotics, Synbiotics, and FMT

The most mature clinical evidence concerns probiotics. Meta-analyses suggest that probiotic interventions may produce modest improvements in depressive symptoms, particularly when used as adjuncts to standard antidepressant therapy, whereas evidence for probiotic monotherapy remains less consistent [21].

Comparative trials including prebiotics have found smaller or no benefits. In one RCT, 110 depressed patients received probiotic, prebiotic (galactooligosaccharide), or placebo for eight weeks. The probiotic group improved on depression scales, but the prebiotic group did not significantly differ from placebo [16]. Meta-analyses similarly find that positive effects are driven by probiotic-containing interventions rather than prebiotics alone [22].

Mechanistically detailed RCTs are emerging. A short-term high-dose probiotic trial in depressed inpatients incorporated microbiome sequencing and neuroimaging [23]. Another trial (PROVIT) confirmed that probiotic add-on raised stool butyrate levels even if blood markers stayed flat [12].

Fecal microbiota transplantation (FMT) for depression is still experimental. A small 8-week double-blind pilot RCT (n=15) tested FMT versus placebo in moderate-severe MDD. The study demonstrated feasibility and safety, with no serious adverse events, and exploratory findings suggested improvements in gastrointestinal symptoms and quality of life [24].

4. DISCUSSION

The evidence supports involvement of the MGBA in depression but also calls for careful interpretation. Human microbiome studies reveal taxa and inferred functions linked to depressive symptoms, yet standardized multi-cohort analyses show no clear global diversity deficits and limited diagnostic accuracy [1, 3]. Some of the strongest experimental evidence comes from transfer experiments: germ-free or antibiotic-treated rodents receiving microbiota from depressed patients develop depression-like behaviors, suggesting a contributory, rather than purely correlative, role of gut microbes [25, 26].

Mechanistically, data point to multiple intersecting pathways. Meta-analyses indicate elevations in markers of gut permeability and immune activation in depression, though narrative reviews stress variability and measurement issues [7, 8]. Animal and human studies implicate neural routes too: vagotomy prevents certain microbiota-driven behavior changes [17], and stress-altered microbiota prime microglia [20].

From a clinical standpoint, probiotics have the most support as adjunctive treatments [21]. Future trials should incorporate predefined mechanistic endpoints and consider stratification by biological subtype (e.g. inflammation-associated vs. non-inflammatory depression) [10]. Greater standardization across studies will be important to improve comparability and clarify the role of microbiota-related mechanisms in depression.

In summary, the MGBA offers a multifactorial framework linking gut microbes, barrier integrity, immune and endocrine signals, metabolites, and brain function in depression. Immediate priorities include standardizing biomarker assays, clarifying patient stratification, and conducting large RCTs with integrated omics to establish mechanism-driven therapeutic effects.

5. CONCLUSIONS

The microbiota-gut-brain axis (MGBA) presents a coherent multi-pathway model of depression pathophysiology. Evidence from cohorts and multi-cohort analyses links depressive symptoms to specific microbial communities and predicted functional pathways. Mechanistic studies demonstrate that altering microbiota state can induce depressive-like phenotypes through both neural (vagus-dependent) and neuroimmune (microglial priming) routes. Clinical trials and systematic reviews indicate that probiotics may alleviate depressive symptoms-most reliably as adjunctive therapy-whereas prebiotics have inconsistent effects and FMT remains an experimental approach. Key research needs are standardized measurement of gut permeability and metabolite biomarkers, incorporation of biologically informed patient stratification, and adequately powered trials with multi-omic endpoints to validate microbiota-based interventions.

Disclosure: The authors declare that they have no financial or non-financial interests that could have influenced the work reported in this paper.

Supplementary Materials: No supplementary materials are associated with this article.

Funding: This research received no external funding.

Institutional Review Board Statement: Not applicable.

Informed Consent Statement: Not applicable.

Data Availability Statement: No new data were created or analyzed in this study.

Acknowledgements: The authors would like to acknowledge that no external support was received for this work.

Conflicts of Interest: The authors declare no conflict of interest. All authors have read and agreed with the published version of the manuscript.

AUTHOR CONTRIBUTIONS

Conceptualization: Izabela Slinkoo.

Methodology: Izabela Slinkoo, Zuzanna Panas, Ewa Dryl-Jarmoc, Gabriela Kulis, Mateusz Zimowski, Jakub Dziemiańczuk, Mateusz Czajkowski, Agata Kolanek, Kacper Krawczuk, Piotr Kadysz.

Investigation: Izabela Slinkoo, Zuzanna Panas, Ewa Dryl-Jarmoc, Gabriela Kulis, Mateusz Zimowski, Jakub Dziemiańczuk, Mateusz Czajkowski.

Resources: Mateusz Zimowski, Jakub Dziemiańczuk, Mateusz Czajkowski, Agata Kolanek, Kacper Krawczuk, Piotr Kadysz.

Writing - original draft preparation: Izabela Slinkoo, Zuzanna Panas, Ewa Dryl-Jarmoc.

Writing - review and editing: Izabela Slinkoo, Zuzanna Panas, Ewa Dryl-Jarmoc, Gabriela Kulis, Mateusz Zimowski, Jakub Dziemiańczuk, Mateusz Czajkowski.

Visualization: Gabriela Kulis, Mateusz Zimowski, Jakub Dziemiańczuk, Mateusz Czajkowski, Agata Kolanek, Kacper Krawczuk, Piotr Kadysz.

Supervision: Izabela Slinkoo.

Project administration: Zuzanna Panas, Ewa Dryl-Jarmoc.

REFERENCES

[1] Radjabzadeh D, Bosch JA, Uitterlinden AG, et al. Gut microbiome-wide association study of depressive symptoms. Nat Commun. 2022;13(1):7128. https://doi.org/10.1038/s41467-022-34502-3

[2] Cryan JF, O'Riordan KJ, Cowan CSM, et al. The microbiota-gut-brain axis. Physiol Rev. 2019;99(4):1877-2013. https://doi.org/10.1152/physrev.00018.2018

[3] Liang S, Wu X, Jin F. Gut-brain psychology: rethinking psychology from the microbiota-gut-brain axis. Front Integr Neurosci. 2018;12:33. https://doi.org/10.3389/fnint.2018.00033

[4] Ng QX, Peters C, Ho CYX, et al. A meta-analysis of the use of probiotics to alleviate depressive symptoms. J Affect Disord. 2018;228:13-19. https://doi.org/10.1016/j.jad.2017.11.063

[5] Valles-Colomer M, Falony G, Darzi Y, et al. The neuroactive potential of the human gut microbiota in quality of life and depression. Nat Microbiol. 2019;4(4):623-632. https://doi.org/10.1038/s41564-018-0337-x

[6] Yang J, Zheng P, Li Y, et al. Landscapes of bacterial and metabolic signatures and their interaction in major depressive disorders. Sci Adv. 2020;6(49):eaba8555. https://doi.org/10.1126/sciadv.aba8555

[7] Morena D, Lippi M, Scopetti M, et al. Leaky gut biomarkers as predictors of depression and suicidal risk. Diagnostics (Basel). 2025;15(13):1683. https://doi.org/10.3390/diagnostics15131683

[8] Bibolar AC, Crecan-Suciu BD, Paunescu RL, et al. Intestinal permeability and depression: a narrative review. Int J Mol Sci. 2025;26(20):10076. https://doi.org/10.3390/ijms262010076

[9] Agirman G, Yu KB, Hsiao EY. Signaling inflammation across the gut-brain axis. Science. 2021;374(6571):1087-1092. https://doi.org/10.1126/science.abi6087

[10] Ting EY, Yang AC, Tsai SJ. Role of interleukin-6 in depressive disorder. Int J Mol Sci. 2020;21(6):2194. https://doi.org/10.3390/ijms21062194

[11] Milaneschi Y, Simmons WK, van Rossum EFC, et al. Depression and obesity: evidence of shared biological mechanisms. Mol Psychiatry. 2019;24(1):18-33. https://doi.org/10.1038/s41380-018-0017-5

[12] Kreuzer K, Birkl-Toeglhofer AM, Haybaeck J, et al. PROVIT-CLOCK: probiotics and vitamin B7 add-on in major depression. Neuropsychobiology. 2024;83(3-4):135-151. https://doi.org/10.1159/000538781

[13] Gao K, Pi Y, Mu CL, et al. Increasing carbohydrate availability in the hindgut promotes hypothalamic neurotransmitter synthesis. J Neurochem. 2019;149(5):641-659. https://doi.org/10.1111/jnc.14709

[14] Lukic I, Ivkovic S, Mitic M, et al. Tryptophan metabolites in depression: modulation by gut microbiota. Front Behav Neurosci. 2022;16:987697. https://doi.org/10.3389/fnbeh.2022.987697

[15] Lukic I, Getselter D, Ziv O, et al. Antidepressants affect gut microbiota and Ruminococcus flavefaciens abolishes their effects on depressive-like behavior. Transl Psychiatry. 2019;9(1):133. https://doi.org/10.1038/s41398-019-0466-x

[16] Kazemi A, Noorbala AA, Azam K, et al. Effect of probiotic and prebiotic vs placebo on psychological outcomes in MDD. Clin Nutr. 2019;38(2):522-528. https://doi.org/10.1016/j.clnu.2018.04.010

[17] Siopi E, Galerne M, Rivagorda M, et al. Gut microbiota changes require vagus nerve integrity to promote depressive-like behaviors in mice. Mol Psychiatry. 2023;28(7):3002-3012. https://doi.org/10.1038/s41380-023-02071-6

[18] Zhu S, Jiang Y, Xu K, et al. The progress of gut microbiome research related to brain disorders. J Neuroinflamm. 2020;17(1):25. https://doi.org/10.1186/s12974-020-1705-z

[19] Heidarzadeh-Rad N, Gokmen-Ozel H, Kazemi A, et al. Effects of a psychobiotic supplement on serum BDNF levels in depressive patients. J Neurogastroenterol Motil. 2020;26(4):486-495. https://doi.org/10.5056/jnm20079

[20] He H, He H, Mo L, et al. Priming of microglia with dysfunctional gut microbiota impairs hippocampal neurogenesis. Brain Behav Immun. 2024;115:280-294. https://doi.org/10.1016/j.bbi.2023.10.031

[21] Nikolova VL, Smith MRB, Hall LJ, et al. Perturbations in gut microbiota composition in psychiatric disorders. JAMA Psychiatry. 2021;78(12):1343-1354. https://doi.org/10.1001/jamapsychiatry.2021.2573

[22] Zhang Q, Chen B, Zhang J, et al. Effect of prebiotics, probiotics, synbiotics on depression: a meta-analysis. BMC Psychiatry. 2023;23(1):477. https://doi.org/10.1186/s12888-023-04963-x

[23] Schaub AC, Schneider E, Vazquez-Castellanos JF, et al. Clinical, gut microbial and neural effects of a probiotic add-on therapy in depressed patients. Transl Psychiatry. 2022;12(1):227. https://doi.org/10.1038/s41398-022-01977-z

[24] Green JE, Berk M, Mohebbi M, et al. Feasibility, acceptability, and safety of faecal microbiota transplantation in MDD. Can J Psychiatry. 2023;68(5):315-326. https://doi.org/10.1177/07067437221150508

[25] Zheng P, Zeng B, Zhou C, et al. Gut microbiome remodeling induces depressive-like behaviors. Mol Psychiatry. 2016;21(6):786-796. https://doi.org/10.1038/mp.2016.44

[26] Kelly JR, Borre Y, O'Brien C, et al. Transferring the blues: depression-associated gut microbiota induces neurobehavioural changes in the rat. J Psychiatr Res. 2016;82:109-118. https://doi.org/10.1016/j.jpsychires.2016.07.019