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GUT MICROBIOTA AS A KEY REGULATOR OF THE GUT–BRAIN AXIS AND METABOLISM IN THE PATHOGENESIS OF PSYCHIATRIC AND METABOLIC DISORDERS: A NARRATIVE REVIEW

Weronika Teterycz *(Corresponding Author, Email: weronikateterycz9@gmail.com)*
Cardinal Stefan Wyszyński University in Warsaw, Warsaw, Poland
ORCID ID: 0009-0000-7486-458X

Wiktoria Goździejewska
Cardinal Stefan Wyszyński University in Warsaw, Warsaw, Poland
ORCID ID: 0009-0007-5302-1883

Łukasz Jaworek
Cardinal Stefan Wyszyński University in Warsaw, Warsaw, Poland
ORCID ID: 0009-0007-8117-5644

Gabriela Zimka
Cardinal Stefan Wyszyński University in Warsaw, Warsaw, Poland
ORCID ID: 0009-0005-3954-7307

Krystian Bjorgen
Cardinal Stefan Wyszyński University in Warsaw, Warsaw, Poland
ORCID ID: 0009-0008-0225-7439

Lilianna Jasińska
University of Warmia and Mazury in Olsztyn, Olsztyn, Poland
ORCID ID: 0009-0006-0819-8922

Magdalena Roman
Cardinal Stefan Wyszyński University in Warsaw, Warsaw, Poland
ORCID ID: 0009-0004-1261-180X

Martyna Lipiarz
Cardinal Stefan Wyszyński University in Warsaw, Warsaw, Poland
ORCID ID: 0009-0001-3124-9250

Michał Niespodziewański
Cardinal Stefan Wyszyński University in Warsaw, Warsaw, Poland
ORCID ID: 0009-0009-0937-1549

Patrycja Szczygielska
Cardinal Stefan Wyszyński University in Warsaw, Warsaw, Poland
ORCID ID: 0009-0006-2063-3165

Sylwia Hejna
Cardinal Stefan Wyszyński University in Warsaw, Warsaw, Poland
ORCID ID: 0009-0005-6313-3067

ABSTRACT

The gut microbiota has emerged as a central regulator of the gut–brain axis and systemic metabolism, playing a pivotal role in the development of both psychiatric and metabolic disorders. This review synthesizes current evidence from peer-reviewed studies indexed in PubMed to elucidate the mechanisms linking gut microbiota dysbiosis with disease pathogenesis. A structured literature review was conducted, focusing on studies published between 2004 and 2025. Evidence indicates that alterations in microbial composition and function are associated with depression, anxiety, autism spectrum disorders, obesity, type 2 diabetes, and metabolic syndrome. Mechanistically, the gut microbiota influences host physiology through neural, endocrine, immune, and metabolic pathways, including modulation of neurotransmitters, short-chain fatty acids, and inflammatory mediators. The review highlights shared biological pathways between psychiatric and metabolic disorders, emphasizing chronic inflammation and metabolic dysregulation. Emerging therapeutic strategies, including microbiota-targeted interventions such as probiotics, prebiotics, dietary modifications, and fecal microbiota transplantation, demonstrate promising clinical potential. However, heterogeneity in study designs and individual microbiome variability remain significant challenges. This review underscores the importance of integrative, multidisciplinary research and supports the gut microbiota as a novel therapeutic target.

KEYWORDS

Gut Microbiota, Gut–Brain Axis, Dysbiosis, Inflammation, Psychiatric Disorders, Metabolic Syndrome

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Introduction

The human gut microbiota is a dynamic and complex ecosystem comprising trillions of microorganisms that significantly influence host physiology. Advances in sequencing technologies have revealed its critical role in maintaining homeostasis and its involvement in disease pathogenesis (Lynch & Pedersen, 2016).

One of the most significant discoveries in recent years is the concept of the gut–brain axis—a bidirectional communication network connecting the gastrointestinal tract and the central nervous system. This axis integrates neural pathways, including the vagus nerve, endocrine signaling via the hypothalamic–pituitary–adrenal (HPA) axis, and immune-mediated mechanisms (Cryan et al., 2019).

Psychiatric disorders, such as depression and anxiety, are increasingly recognized as systemic conditions influenced by inflammatory and metabolic processes. Similarly, metabolic disorders such as obesity and type 2 diabetes involve chronic low-grade inflammation and altered energy homeostasis. The frequent co-occurrence of these disorders suggests the existence of shared biological mechanisms.

Recent studies indexed in PubMed highlight the gut microbiota as a key mediator linking these conditions. Dysbiosis has been associated with altered neurotransmission, increased intestinal permeability, and systemic inflammation, all of which contribute to disease development (Dinan & Cryan, 2017). Despite growing evidence, the precise mechanisms underlying the interaction between gut microbiota dysbiosis and the development of both psychiatric and metabolic disorders remain insufficiently understood, representing a significant research gap.

Therefore, the aim of this review is to provide a comprehensive synthesis of current evidence regarding the role of gut microbiota in the regulation of the gut–brain axis and metabolism, with a particular focus on its involvement in psychiatric and metabolic disorders, as well as to clarify the underlying biological mechanisms linking these conditions.

This review provides a novel integrative perspective by synthesizing neuroimmune and metabolic mechanisms within a unified microbiota-driven framework linking psychiatric and metabolic disorders.

Methodology

This manuscript was conducted as a structured narrative review aimed at synthesizing current evidence on the role of the gut microbiota in the regulation of the gut–brain axis and systemic metabolism, with particular emphasis on its involvement in the pathogenesis of psychiatric and metabolic disorders.

Although this review does not meet the formal criteria of a systematic review, a transparent and predefined literature search and study selection strategy was applied to enhance reproducibility and methodological rigor.

The approach incorporated key elements of systematic searching, including clearly defined eligibility criteria, structured screening, and thematic synthesis of the available evidence.

Data source and search strategy

A comprehensive literature search was conducted using the PubMed database, which was selected as the primary source of biomedical and life science literature due to its broad coverage of peer-reviewed research relevant to microbiome science, psychiatry, metabolism, and neuroimmunology. The search included studies published between January 2004 and January 2025, in order to capture both foundational investigations and more recent advances in the field.

The search strategy combined Medical Subject Headings (MeSH) and free-text keywords related to the central concepts of the review. Core search terms included: “gut microbiota”, “intestinal microbiome”, “gut–brain axis”, “microbiota–gut–brain axis”, “psychiatric disorders”, “depression”, “anxiety”, “autism spectrum disorder”, “metabolic disorders”, “obesity”, “type 2 diabetes”, “metabolic syndrome”, “dysbiosis”, “intestinal permeability”, and “inflammation.” Boolean operators (AND, OR) were used to combine terms and refine the search. In addition, the reference lists of selected articles were manually screened to identify further relevant publications not captured in the initial search.

Eligibility criteria

Studies were considered eligible if they met the following inclusion criteria:

(1) peer-reviewed articles indexed in PubMed;

(2) publications written in English;

(3) studies published between 2004 and 2025;

(4) human studies, animal studies, or translational research;

(5) original research articles, clinical trials, cohort studies, case–control studies, systematic reviews, and meta-analyses;

and (6) studies directly addressing the relationship between gut microbiota and psychiatric or metabolic outcomes, including mechanistic pathways relevant to the gut–brain axis, inflammation, intestinal permeability, microbial metabolites, or host metabolic regulation.

The exclusion criteria comprised:

(1) non-peer-reviewed publications;

(2) editorials, opinion pieces, and narrative commentaries without substantive analytical content;

(3) case reports with limited generalizability;

(4) studies lacking sufficient methodological clarity;

and (5) articles not directly relevant to the review objective.

Study selection

Study selection was performed in several stages. First, titles and abstracts were screened for thematic relevance. Next, potentially eligible articles underwent full-text assessment. Studies were then selected on the basis of their relevance to the scope of the review, methodological soundness, and contribution to the understanding of microbiota-related neurobiological and metabolic mechanisms. Duplicate records were excluded prior to final inclusion.

Because this study was conducted as a structured narrative review rather than a formal systematic review, no full PRISMA flow diagram was generated. Nevertheless, the review process followed a transparent and stepwise selection strategy to improve reproducibility and minimize arbitrary study inclusion.

Data extraction and synthesis

For each included study, the following information was extracted where available: study design, population characteristics, type of disorder examined, microbiota-related findings, key metabolites or mechanistic pathways involved, major clinical or biological outcomes, and principal conclusions. Particular attention was given to evidence concerning microbial diversity, short-chain fatty acids, intestinal permeability, immune activation, HPA axis regulation, neurotransmitter modulation, and metabolic signaling.

Given the heterogeneity of the available literature in terms of study design, populations, laboratory methods, and reported outcomes, a qualitative narrative synthesis was undertaken rather than a meta-analysis. The evidence was analyzed thematically, with studies grouped according to major biological mechanisms and disease categories. This approach enabled the integration of data from basic science, translational, and clinical research, while also allowing comparison between psychiatric and metabolic conditions within a common conceptual framework.

Appraisal of evidence quality

Although no formal risk-of-bias tool was applied, the methodological quality of the included literature was critically considered during analysis and interpretation. Particular attention was paid to study design, sample size, reproducibility of microbiome assessment methods, control of confounding variables, and the extent to which conclusions were supported by the data. Greater interpretive weight was given to systematic reviews, meta-analyses, well-designed clinical studies, and mechanistic studies with clear biological relevance.

Methodological limitations

Several methodological limitations should be acknowledged. First, the gut microbiome literature is characterized by substantial heterogeneity in sequencing platforms, bioinformatic pipelines, taxonomic resolution, and analytical strategies, which limits direct comparability across studies (Knight et al., 2018). Second, a large proportion of the available evidence remains observational, which constrains causal inference. Third, findings from animal and germ-free models, while mechanistically informative, cannot always be directly extrapolated to human populations. Finally, because only PubMed was searched, relevant studies indexed exclusively in other databases, such as Scopus or Web of Science, may not have been captured.

Despite these limitations, the use of a structured and transparent search strategy, clearly defined eligibility criteria, and thematic synthesis of peer-reviewed evidence supports the reliability of the present review and its relevance as an integrative overview of the field.

Discussion

The findings synthesized in this review provide strong support for the hypothesis that the gut microbiota functions as a central regulator of both neurobiological and metabolic processes, acting as a critical interface between environmental exposures, host physiology, and disease pathogenesis. Importantly, the convergence of evidence across psychiatric and metabolic disorders suggests that these conditions share common mechanistic pathways, with the microbiota–gut–brain axis emerging as a unifying framework.

Integration of Neuroimmune and Metabolic Pathways

One of the most significant insights derived from the reviewed literature is the role of chronic low-grade inflammation as a shared pathophysiological mechanism linking psychiatric and metabolic disorders. Dysbiosis-induced increases in intestinal permeability facilitate the translocation of microbial components, such as lipopolysaccharides (LPS), into systemic circulation. This process triggers immune activation and the release of pro-inflammatory cytokines, including interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF-α), and interleukin-1β (IL-1β) (Cani et al., 2007; Miller & Raison, 2016).

These inflammatory mediators can cross the blood–brain barrier or signal through neural pathways, contributing to neuroinflammation, altered neurotransmission, and behavioral changes. Simultaneously, systemic inflammation promotes insulin resistance and disrupts metabolic homeostasis, thereby linking immune dysregulation to both depression and metabolic syndrome. This dual impact highlights the importance of conceptualizing psychiatric disorders within a broader systemic and immunometabolic framework.

Furthermore, activation of the kynurenine pathway represents a critical molecular link between inflammation and neuropsychiatric outcomes. Under inflammatory conditions, increased conversion of tryptophan to kynurenine reduces serotonin availability while promoting the accumulation of neurotoxic metabolites such as quinolinic acid (O'Mahony et al., 2015). This mechanism provides a biologically plausible explanation for the observed association between dysbiosis, inflammation, and depressive symptomatology.

Table 1. Gut Microbiota Alterations and Mechanisms in Psychiatric and Metabolic Disorders

Disorder	Microbiota Alterations	Key Mechanisms	Biological/Clinical Implications
Depression	Reduced microbial diversity; decreased *Faecalibacterium* and *Coprococcus*; reduced SCFA production	Neuroinflammation; altered neurotransmission; activation of the kynurenine pathway; HPA axis dysregulation	Depressive symptoms; impaired mood regulation; increased inflammatory burden
Anxiety disorders	Dysbiosis; altered *Lactobacillus* and *Bifidobacterium* abundance	HPA axis hyperactivation; vagus nerve signaling; modulation of GABA receptor expression	Increased stress reactivity; anxiety-like behavior; altered emotional processing
Autism spectrum disorders (ASD)	Reduced microbial diversity; increased *Clostridium* species; increased intestinal permeability	Immune activation; gut barrier dysfunction; altered metabolite production	Behavioral abnormalities; gastrointestinal symptoms; neurodevelopmental disturbances
Obesity	Reduced microbial diversity; compositional shifts affecting short-chain fatty acid (SCFA) production, inflammation, and energy harvest; inconsistent findings regarding the Firmicutes/Bacteroidetes ratio	Increased energy harvest; enhanced lipogenesis; low-grade systemic inflammation	Weight gain; adiposity; metabolic imbalance
Type 2 diabetes mellitus	Reduced butyrate-producing bacteria; increased gut permeability	Metabolic endotoxemia (LPS); chronic inflammation; insulin resistance	Impaired glucose metabolism; systemic inflammation; metabolic dysfunction
Metabolic syndrome	Dysbiosis; altered bile acid metabolism; reduced SCFA production	Chronic inflammation; disrupted lipid metabolism; hormonal dysregulation	Cluster of metabolic abnormalities; increased cardiovascular risk

Role of Microbial Metabolites in Host Physiology

Microbial metabolites play a central role in mediating host–microbiota interactions. Short-chain fatty acids (SCFAs), particularly butyrate, acetate, and propionate, have been consistently shown to exert anti-inflammatory effects, regulate intestinal barrier integrity, and influence central nervous system function (Koh et al., 2016).

Butyrate, in particular, acts as a histone deacetylase (HDAC) inhibitor, thereby modulating gene expression and exerting neuroprotective effects. In addition, SCFAs influence microglial activation and blood–brain barrier integrity, suggesting their involvement in neurodevelopmental processes and neuroinflammation (Cryan et al., 2019).

Beyond SCFAs, the gut microbiota is capable of producing and modulating key neurotransmitters and neuromodulators, including gamma-aminobutyric acid (GABA), dopamine, and serotonin. This capacity to influence neurotransmitter systems highlights the potential role of the microbiota in shaping mood, behavior, and cognitive function.

Bidirectional Relationship Between Psychiatric and Metabolic Disorders

A key observation across the reviewed studies is the bidirectional relationship between psychiatric and metabolic disorders. Individuals with obesity are at increased risk of depression, while those with depression frequently exhibit metabolic abnormalities, including insulin resistance and dyslipidemia. This relationship is likely mediated by shared microbiota-driven mechanisms, including chronic inflammation, hormonal dysregulation, and altered neuroendocrine signaling.

The concept of a "metabolic–psychiatric axis" mediated by the gut microbiota represents an emerging paradigm that challenges traditional disease classifications and underscores the need for integrated, interdisciplinary approaches to disease management.

Therapeutic Potential of Microbiota-Targeted Interventions

The growing recognition of the gut microbiota's role in disease pathogenesis has led to increasing interest in microbiota-targeted therapies. Probiotics, prebiotics, and synbiotics have demonstrated potential in modulating microbial composition and improving both metabolic and psychiatric outcomes (Cryan et al., 2019).

Clinical studies suggest that probiotic supplementation may reduce symptoms of depression and anxiety, potentially through modulation of inflammatory pathways and neurotransmitter systems. Similarly, dietary interventions rich in fiber and polyphenols have been shown to enhance microbial diversity and promote the production of beneficial metabolites.

Fecal microbiota transplantation (FMT) represents a more direct strategy for restoring microbial balance. Although it has demonstrated efficacy in certain gastrointestinal conditions, its application in psychiatric and metabolic disorders remains experimental and requires further investigation.

However, despite promising findings, the clinical translation of microbiota-based therapies faces several challenges, including inter-individual variability in microbiome composition, lack of standardized treatment protocols, and limited long-term safety and efficacy data.

Methodological Challenges and Limitations in Microbiome Research

The interpretation of microbiome research is complicated by several methodological challenges. These include heterogeneity in study designs, differences in sequencing techniques (e.g., 16S rRNA vs. metagenomics), and variability in bioinformatic pipelines (Knight et al., 2018).

Moreover, the predominance of observational studies limits the ability to establish causal relationships. While animal models and fecal transplantation studies provide important insights, their direct translation to human populations remains uncertain.

Inter-individual variability in gut microbiota composition, influenced by genetics, diet, environment, and lifestyle factors, further complicates the generalizability of findings and highlights the need for personalized approaches in microbiome research and therapy.

Future Directions and Implications for Clinical Practice

Future research should prioritize longitudinal and interventional study designs to better establish causal relationships and evaluate therapeutic efficacy. The integration of multi-omics approaches, including metagenomics, metabolomics, and transcriptomics, may provide deeper insight into the functional role of the microbiome.

From a clinical perspective, the incorporation of microbiome-based diagnostics and therapeutics into routine practice holds significant promise for the management of complex, multifactorial diseases. Personalized medicine approaches tailored to individual microbiome profiles may represent a transformative direction in both psychiatric and metabolic healthcare.

Collectively, these findings highlight the need to redefine traditional boundaries between psychiatric and metabolic diseases, emphasizing a systems-based approach in which the gut microbiota plays a central role in both pathogenesis and therapeutic strategies.

Conclusions

The present review provides a comprehensive synthesis of current evidence demonstrating that the gut microbiota plays a fundamental and multifaceted role in the regulation of the gut–brain axis and systemic metabolism. The findings consistently indicate that alterations in microbial composition and function—collectively referred to as dysbiosis—are closely associated with the pathogenesis of both psychiatric and metabolic disorders. Importantly, this relationship is bidirectional, reflecting a dynamic interplay between the gut microbiota, the central nervous system, and host metabolic processes.

A key conclusion emerging from this review is the identification of shared pathophysiological mechanisms underlying psychiatric and metabolic conditions. Chronic low-grade inflammation, increased intestinal permeability, immune dysregulation, and altered production of microbial metabolites—particularly short-chain fatty acids and tryptophan-derived compounds—represent central pathways linking these disorders. These mechanisms support the concept of an integrated, microbiota-driven disease model, in which disturbances in gut microbial ecology contribute to both neuropsychiatric symptoms and metabolic dysfunction.

Furthermore, the evidence highlights the critical role of microbial metabolites as key mediators of host–microbiota interactions. Short-chain fatty acids, bile acid derivatives, and kynurenine pathway metabolites exert profound effects on immune signaling, neuroinflammation, and metabolic regulation. These findings emphasize the importance of considering not only the taxonomic composition of the microbiota but also its functional capacity in disease pathogenesis.

From a clinical perspective, the gut microbiota represents a promising target for novel therapeutic strategies. Interventions such as probiotics, prebiotics, synbiotics, dietary modifications, and fecal microbiota transplantation have demonstrated potential in improving both mental and metabolic health outcomes. However, the translation of these approaches into routine clinical practice remains limited by inter-individual variability in microbiome composition, inconsistent treatment responses, and the lack of standardized intervention protocols.

Despite substantial progress, several key challenges remain. Most available studies are observational, limiting causal inference, while methodological heterogeneity—particularly in sequencing techniques, analytical pipelines, and study populations—complicates cross-study comparisons. Additionally, the high degree of inter-individual variability necessitates the development of personalized, precision-based approaches to microbiome-targeted therapies.

Future research should prioritize well-designed longitudinal and interventional studies in human populations to clarify causal relationships and assess long-term efficacy and safety. The integration of multi-omics approaches, including metagenomics, metabolomics, transcriptomics, and proteomics, will be essential for understanding the functional dynamics of the microbiome and its interaction with host systems. Moreover, the identification of disease-specific microbial signatures may enable the development of microbiome-based biomarkers for early diagnosis, risk stratification, and therapeutic monitoring.

Importantly, the translation of microbiome research into clinical practice will require interdisciplinary collaboration across microbiology, neuroscience, endocrinology, psychiatry, and bioinformatics. Such an integrative approach is necessary to fully capture the complexity of the microbiota–gut–brain axis and its role in human health and disease.

Ultimately, advancing our understanding of the gut microbiota may redefine current approaches to the prevention, diagnosis, and treatment of complex psychiatric and metabolic disorders.

The authors declare no conflicts of interest.

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