The Gut–Brain Axis and Psychosis: A Systematic Review of Microbial Metabolites and Their Impact on Dopaminergic Pathways in Schizophrenia

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Abstract

Schizophrenia is a psychiatric disorder that has psychotic, cognitive, and negative symptoms, with dopaminergic dysfunction. The gut–brain axis is identified as an important modulator for neurochemical function. Following PRISMA 2020 guidelines, this systematic review is conducted through searches (PubMed, Scopus, SpringerLink, Taylor & Francis, and Google Scholar). After removing duplicates and screening, six studies included three clinical studies, two translational studies involving fecal microbiota transplantation (FMT), and one rodent model. The findings highlighted three main themes:(1) the dysregulation of the tryptophan–kynurenine pathway, with elevated kynurenine and reduced kynurenic acid linked to disrupted dopaminergic and glutamatergic signaling; (2) changes of microbiota composition and metabolite imbalances, which included fewer butyrate producers and more succinate generators, which were associated with neurotransmitter dysregulations; and (3) relationships within clinical symptoms, in which metabolites correlated with comprehension of negative/cognitive deficits. Collectively, microbial dysbiosis and metabolite imbalance influence dopamine biosynthesis, receptor activity, immune, metabolic, and neurochemical signaling.

Keywords: Cognitive and negative symptoms, Dopaminergic dysfunction, Gut–brain axis (GBA), Microbiota dysbiosis, Schizophrenia, Tryptophan–kynurenine pathway

Introduction

Schizophrenia is a complex psychiatric chronic syndrome that includes psychotic and negative symptoms like hallucination, delusions, disorganized speech, cognitive deficits, and decreased motivation (Marder & Cannon, 2019). That involves impairing mental processing as well as executive functions (Marder & Cannon, 2019). Schizophrenia is among the top 10 global causes of disability. Moreover, it affects approximately 1% of the world's population (Marder & Cannon, 2019). The disorder develops in early adulthood, especially in males rather than females, and it is rare before the age of 16 years (Nafe et al., 2025). In spite of the extensive research done, the pathophysiology mechanisms of schizophrenia remain incompletely understood (Nafe et al., 2025). Various factors can lead to schizophrenia as some theories emphasize spiritual beliefs, physical problems, traumatic events, stress in social life, substance abuse, and heredity (Marutani et al., 2022). All those have a relationship with neurochemical imbalance, particularly the dysregulation of dopaminergic signaling as is the main neurochemical in the onset and progression of the disorder (Crotchett et al., 2025). Through recent years, it has become an acceptable idea that schizophrenia may not arise only from

abnormalities within the brain, but rather through an integration of factors including the entire body (Mousavinejad et al., 2025). This new outlook has provided researchers with new direction to investigate how peripheral systems, especially the gastrointestinal tract and the diverse microbial communities it contains, might influence brain development, neural function, and behavior (Cipriani & Juster, 2024), (Mousavinejad et al., 2025).

The gut–brain axis (GBA) means the bidirectional communication network that connects the central nervous system (CNS) and the gastrointestinal system, encompassing neural, hormonal, metabolic, and immunological pathways (Zhang et al., 2025). The gut-brain axis creates a complex bidirectional communication system that permits a constant interaction and singling between gut microbiome and the brain (Petrut et al., 2025), that enables the microbial metabolites, which are substances produced by gut bacteria, to affect various brain and neurophysiological functions (Randeni & Xu, 2025). Recent scientific advancements in metagenomics (studying microbial genes), neuroimaging, and neuroimmunology have strongly revealed the link between gut microbes and various neuropsychiatric disorders like schizophrenia, depression, autism, anxiety, and more (Hadrich et al., 2025). Especially interesting is the ability of microbial metabolites (short-chain fatty acids (SCFAs), tryptophan catabolites, and other neuroactive factors) to influence the functioning of the brain by their action on the neuroinflammation, blood–brain barrier integrity, neurotransmitter production and synaptic plasticity (Kurhaluk et al., 2025).

Among these mechanisms, the influence of microbial metabolites on dopaminergic pathways holds particular significance in the context of schizophrenia (Vascellari et al., 2020). The dopamine hypothesis, the most neurochemical contributor of schizophrenia disorder (Vellucci et al., 2025), posits that hyperactivity in subcortical dopaminergic pathways contributes to the positive symptoms like hallucinations and delusions (Marder & Cannon, 2019), while hypoactivity in cortical dopamine circuits may underlie negative and cognitive symptoms (Marder & Cannon, 2019).

Several studies in both animal models and clinical populations have demonstrated that perturbations in gut microbiota can alter dopamine biosynthesis (Jamerlan et al., 2025), affect dopaminergic receptor expression, and modulate reward and motivational circuits through systemic immune signaling and vagus nerve activation (Cichon et al., 2025). Additionally, microbial dysbiosis has been associated with increased intestinal permeability (leaky gut) and elevated systemic inflammation (Chen et al., 2025), which are known to impact brain development and dopaminergic transmission (Chen et al., 2025).

Despite these great findings, the field is still in its early days and the specific direction of relationships between microbial metabolites and dopaminergic dysfunction in schizophrenia is still not fully understood. The existing literature is fragmented between pre-clinical and clinical studies using different methods; therefore, results are often inconclusive. Hence, an integrative

synthesis of the literature is needed, to assess the strength, consistency and boundaries of the current evidence.

This systematic review mainly aims to provide a critical analysis of the existing literature on the contributions of microbiota-derived metabolic products from the gut, to the regulation of the dopaminergic system in schizophrenia. This review attempts to synthesize the findings from experimental models and clinical studies to provide clarity around the mechanisms by which specific compounds of microbial metabolites can influence dopaminergic signaling pathways and contribute to the pathophysiology of psychosis. Such a gut–microbiota–dopamine axis could inform future targeted microbiome-based interventions and promote a more comprehensive approach for diagnosing and treating schizophrenia.

Methods

1. *Protocol and registration*

This systematic review is conducted according to Preferred Reporting Items for Systematic Reviews and Meta-Analysis (PRISMA 2020) guidelines. The review protocol has been developed.

2. *Eligibility Criteria*

The Eligibility Criteria is defined based on the PICO (Population, Intervention, Comparison, Outcome) framework. The eligible studies include those that examine schizophrenic individuals diagnosed by ICD-10 (Population), investigate changes in gut microbial metabolites (Intervention), compared with healthy controls or baseline conditions (Comparisons), and assess dopaminergic pathway dysfunction (Outcome).

Table 1. Summary of eligibility criteria structured according to the PICO framework for inclusion in this systematic review.

PICO Element	Description
Population (P)	Individuals with schizophrenia or psychosis; animal models mimicking these conditions diagnosed by ICD-10

Intervention (I)	Alterations in gut microbial metabolites (e.g., SCFAs, tryptophan metabolites, GABA, etc.)
Comparison (C)	Healthy controls or baseline/sham conditions
Outcome (O)	Dopaminergic pathway dysfunction (e.g., dopamine levels, receptor changes, behavioral or imaging biomarkers)

Inclusion Criteria

This systematic Review included peer-reviewed scholarly studies, studies clearly measuring microbial metabolites and dopaminergic outcomes, studies in English, and studies recently published since 2000 or earlier.

Exclusion Criteria

This Systematic Review excluded review articles, meta-analysis, editorials, conference abstracts or non peer-review research papers, Studies lacking outcome relevance like no dopaminergic or metabolite data, and studies that focus on unrelated mental disorders or neurotransmitters.

3. Information Sources

A Systematic Review was done on Google Scholar, PubMed, Scopus, Taylor & Francis Online, and SpringerLink (Nature) databases, selecting studies published between January 2000 and July 2025, using Boolean with combinations of main keywords related to gut microbiota, microbial metabolites, schizophrenia, and dopamine. This boolean is used during searching ("gut microbiota" OR "gut microbes" OR "intestinal microbiome" OR "microbial metabolites" OR "short-chain fatty acids" OR "tryptophan metabolism") AND ("schizophrenia" OR "psychosis" OR "psychotic disorders") AND ("dopamine" OR "dopaminergic pathway" OR "dopamine receptor" OR "dopaminergic dysfunction").

All retrieved citations were exported from PubMed (.nbib), Scopus (.ris), SpringerLink (.ris), and Taylor & Francis (.ris), and imported into Rayyan. Rayyan has shown that it is a highly helpful website that has the ability to significantly reduce the workload of authors of systematic reviews by reducing the time-consuming step of choosing and screening which research to include in the review (Ouzzani et al., 2016).

Duplicates were automatically removed. Studies were independently screened based on titles and abstracts, with full texts assessed for final inclusion. A total of approximately 465 articles were retrieved across databases. After duplicate removal around 117. 348 records remained for screening. Of these, 310 were excluded for reasons including wrong population (n = 136), wrong outcome (n = 96), wrong study design (n = 94), foreign language (n = 3), wrong publication type (n = 99), and outdated publication date (n = 17). After screening around 27 full-text articles were assessed for eligibility, a total of 6 studies met all inclusion criteria and were included in the final systematic review.

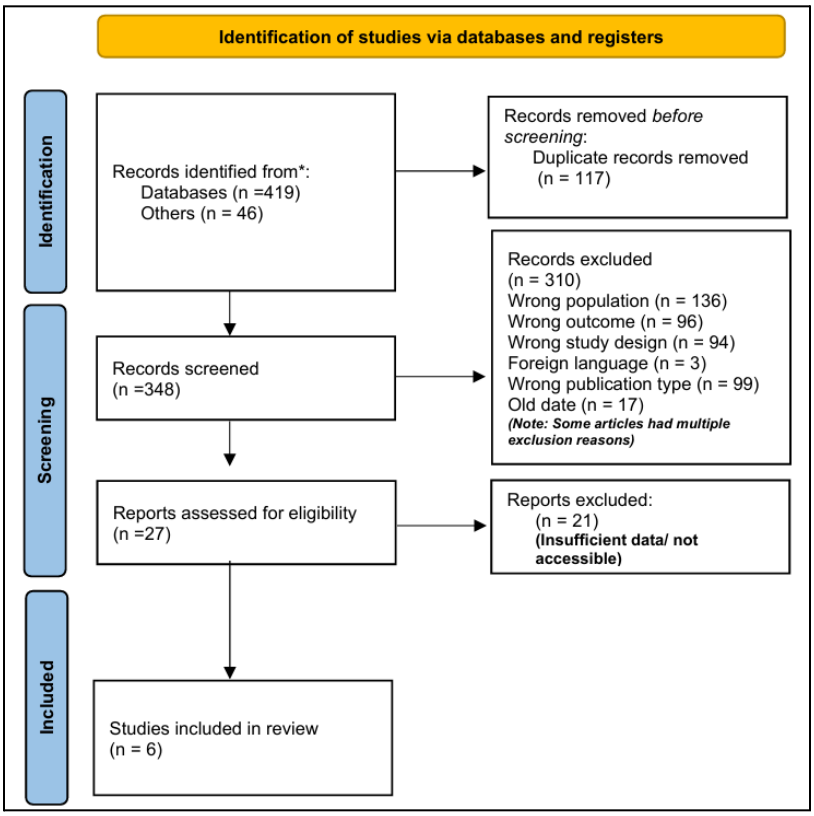

Figure 1. The flow diagram illustrates the PRISMA flow diagram

Results

A total of six studies met the inclusion criteria mentioned and were used in this systematic review. The studies vary in design, including three clinical investigations, two translational human-to-mouse fecal microbiota transplantation (FMT) studies, and one rodent experimental model. Collectively, they investigate the role of gut microbiota–derived metabolites, such as tryptophan derivatives, short-chain fatty acids, succinate, in modulating dopaminergic and related neurochemical pathways. The key findings of the six studies are mentioned in Table 2.

Table 2. Comparative overview of included studies on gut microbiota, metabolites, and dopaminergic outcomes in schizophrenia and related disorders

Author/ Year	Study type	Population/ Model	Microbial / metabolic focus	Dopaminergic / serotonergic outcomes	Key findings
Liu et al., 2023 (Medicina)	Case–control (clinical)	MDD (n=24), SCZ (n=22), HC (n=23)	Tryptophan metabolites (5-HIAA, indole, kynurenine); fatty acids (PUFA, MUFA, SFA)	Indirect modulation via Trp–serotonin–kynurenine pathway; FA alterations	SCZ & MDD had ↓Trp metabolites and ↓omega-3 PUFAs; SCZ also showed ↓SFA/MUFA. Metabolite changes correlated with symptom severity.
Sekine et al., 2016 (Neurochem Res)	Experimental (in vitro + in vivo rodent)	Rat cortical slices; BALB/c mice	Kynurenine uptake & KYNA production via LAT transporters	KYNA ↓dopamine & glutamate via NMDA/α7nACh inhibition	LAT inhibitor (BCH) reduces KYN uptake and KYNA production; supports the role of KYNA in dopaminergic/glutamatergic imbalance in SCZ.
Zhu et al.,	FMT into mice	FMT from SCZ	Altered kynurenine	SCZ-FMT mice: ↑KYN,	SCZ microbiota induced

2020 (Mol Psychiatry)		patients vs healthy controls	pathway; ↓SCFA-producing bacteria	↓KYNA → dopaminergic dysregulation	SCZ-like behaviors and metabolic disturbances in mice.
Wei et al., 2024 (Schizophrenia)	FMT human–mouse translational study	20 SCZ patients (on antipsychotics), 15 HC → SPF mice	Dysbiosis: ↓Faecalibacterium, Coprococcus, Bacteroides; ↑Bifidobacterium, Shigella	Dysregulation of GABAergic synapse genes; inflammatory pathways activated	SCZ-FMT mice showed hyperactivity, anxiety, and social deficits. Microbiota are linked to neurotransmission and immune regulation.
Ghorbani et al., 2024 (Egypt J Neurol Psychiatry Neurosurg)	Pilot human case–control	10 SCZ vs 10 HC	↑Succinate-producers (Phascolarctobacterium); ↓Butyrate-producers (Roseburia, Dorea)	SCZ patients: ↑dopamine & serotonin; ↓BDNF (ns); negative microbial–neurochemical correlations	Dysbiosis correlated with neurotransmitter imbalance; suggests therapeutic potential of microbiota modulation.
Van der Walt et al., 2025 (SA J	Pilot longitudinal clinical	15 SCZ patients, baseline vs 6 weeks post-antipsychotics	Tryptophan metabolites (KYNA, QUIN), 5-HT, 5-HIAA, BDNF, GABA	↓KYNA, ↓5-HIAA, ↓BDNF, ↓GABA; no change in dopamine	Antipsychotics improved positive/general but not negative symptoms; persistent

Psychiatry)					deficits linked to Trp metabolism & GABA-glutamate dysfunction.

Across the six studies, microbial metabolites, particularly short-chain fatty acids (SCFAs) and tryptophan–kynurenine derivatives, have effect in the dysregulation of dopaminergic and related neurotransmitter pathways in schizophrenia. Clinical studies revealed associations between changes in metabolite profiles and neurotransmitter levels, while FMT and animal models demonstrated causal effects of microbiota manipulation on dopaminergic activity, serotonin turnover, and behavior. Three primary mechanistic themes were identified:

1. *Tryptophan–kynurenine pathway dysregulation*

Liu (2023) found schizophrenia patients had lower tryptophan metabolites and fatty acids, whose concentrations were associated with severity of symptoms. Zhu (2020) showed that fecal microbiota transplantation (FMT) from schizophrenic subjects increased kynurenine and decreased KYNA in mice, resulting in schizophrenia-like behavior. Sekine (2016) confirmed that KYNA suppresses dopamine and glutamate transmission through NMDA and α7nACh, correlating elevated levels of KYNA with dopaminergic dysfunction. Van der Walt (2025) showed that antipsychotic medication decreased KYNA and 5-HIAA, correlating symptom changes with first-serotonin metabolites. This shows a stable imbalance of tryptophan, serotonin, and kynurenine.

2. *Microbiota composition and metabolite imbalances*

Ghorbani (2024) reported an increase in succinate-producing bacteria and a decrease in butyrate-producing bacteria in schizophrenia patients. This was linked to higher dopamine and serotonin levels and lower BDNF. Wei (2024) found that FMT from schizophrenia patients to mice caused hyperactivity, social deficits, and an increase in GABAergic and inflammatory pathways. These findings indicate that specific microbial taxa and their metabolites exert a measurable impact on dopaminergic, serotonergic, and neurotrophic signaling.

3. *Clinical symptom correlations*

In Van der Walt (2025), improvement in general and positive symptoms was associated with serotonin metabolites (5-HT, 5-HIAA). Negative symptoms were still with no change and were associated with alterations of GABA and QUIN. Liu (2023) linked metabolic disturbances with schizophrenia and major depressive disorder symptom severity, indicating the importance of

these biomarkers. Ghorbani (2024) and Wei (2024) also support the suggestion that compositional variation in microbes is associated with behavioral or symptomatic impacts. Clinical (Liu 2023; Van der Walt 2025) and experimental (Sekine 2016; Zhu 2020) evidence suggests dopaminergic imbalance results from changes in tryptophan metabolism.

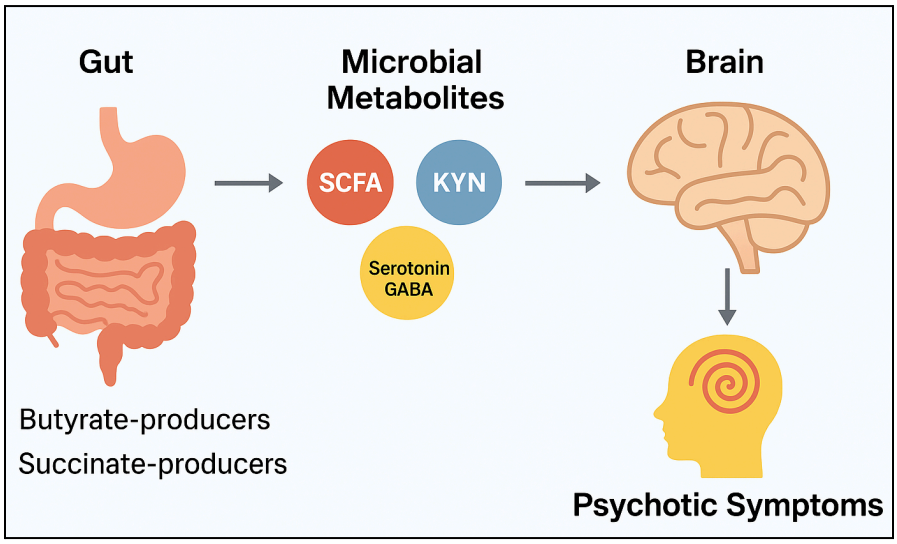

Figure 2. Summarize the results and research objective: Gut–brain axis involvement in psychosis.

Discussion

The present review consolidates evidence implicating gut microbiota–derived metabolites in dopaminergic dysregulation of schizophrenia. In both preclinical and human research, tryptophan–kynurenine metabolism and SCFA profiles were systematically manipulated across the collection of included studies with measurable influences on dopamine signaling, synaptic plasticity, and behavioral phenotypes. These findings indicate that microbial metabolites are not epiphenomena but rather functional regulators of host neurobiology.

Quantitative Evidence

Quantitative evidence from the included studies confirms correlational as well as causal relationships. Liu (2023) discovered significant reductions in the metabolites of tryptophan and fatty acids in schizophrenic patients. Zhu (2020) and Wei (2024) demonstrated that fecal microbiota transplantation from patients replicated central behavioral phenotypes in mice, with concomitant elevations in kynurenine and decreases in kynurenic acid. Clinical cohorts also documented biochemical alterations: Ghorbani (2024) reported increased dopamine and serotonin but decreased BDNF with respect to dysbiosis, and Van der Walt (2025) showed that treatment reduced kynurenic acid, 5-HIAA, BDNF, and GABA, in accord with improvements in

positive and general symptoms. Mechanistic specificity was provided by Sekine (2016), who reported that LAT inhibition reduced production of kynurenic acid, thereby directly implicating peripheral metabolism in central dopamine–glutamate imbalance.

Mechanistic Considerations

Dysregulation of the tryptophan-kynurenine pathway is a principal mechanism identified in these investigations. Increased kynurenine levels and decreased kynurenic acid shift the equilibrium toward excitotoxicity and NMDA receptor hypofunction, a process long hypothesized to be the basis of negative and cognitive symptoms in schizophrenia. The available evidence implies that a deficiency of SCFA, especially a reduction in butyrate, is implicated in impaired dopamine biosynthesis and synaptic modulation. SCFAs modulate microglial activation, regulate tyrosine hydroxylase expression, and induce epigenetic effects by inhibiting histone deacetylase. The combined results indicate that microbial dysbiosis can converge on dopamine dysfunction through several biochemical routes.

Translational Implications

Microbial metabolites have the potential to act as biomarkers for treatment response and disease stratification, according to translational research. It is possible to integrate ratios like kynurenine/tryptophan and SCFA signatures into biomarker panels for precision psychiatry. Targeted interventions on the microbiota, including probiotics, dietary changes, SCFA supplementation, and pharmacological manipulation of the kynurenine pathway, could be viable additions to antipsychotic treatment. These approaches may be of particular interest to negative and cognitive symptoms, which are not sufficiently addressed by dopamine receptor antagonists.

Limitations

This review has several limitations. First, there are not many studies that focus on microbial metabolites, dopaminergic pathways, and schizophrenia. Most of the available evidence is still developing, with only a few published studies directly exploring this connection. Second, access to some full-text articles was limited, which may have resulted in excluding potentially relevant studies. As a high school student researcher, having limited access to resources decreased my capacity to identify all qualifying publications, which created the potential for selection bias. Third, considerable heterogeneity existed among the studies included in design (animal versus human), sample size, diagnostic instruments, techniques used to measure microbial metabolites, and assessment of dopaminergic outcomes. This heterogeneity rendered it challenging to conduct a meta-analysis. Fourth, potential confounders such as antipsychotic medication use, dietary habits, and comorbid conditions were inconsistently controlled across

studies. Finally, the bidirectional and dynamic nature of the gut–brain axis suggests longitudinal and mechanistic studies are required to establish causality, but such studies remain absent

Future Directions

Future investigations should focus on long-term multi-omics methods that combine metagenomic sequencing, targeted metabolomes, and functional neuroimaging. These designs would help map changes in microbes to dopaminergic activity over time. Randomized controlled trials of interventions that modify microbiota are also needed to test therapeutic effects. Understanding these pathways will improve models of schizophrenia and might lead to new interventions guided by biomarkers.

Conclusion

This systematic review included just Six studies exploring the microbial metabolites within the gut–brain axis and their possible effects on dopaminergic mechanisms in psychosis. Although the available literature remains limited and heterogeneous, the findings support the gut-brain axis as a promising avenue of research, while it highlights the growing evidence that microbial metabolites can influence dopaminergic pathways and engage with schizophrenia and related psychoses. The present data are insufficient to guide clinical practice; however, it furnishes a valuable basis for subsequent research and the development of novel therapies. Currently available evidence remains fragmented, but by drawing attention to this gap, the review provides a starting point for future research. In conclusion, a more profound comprehension of the microbial factors related to psychosis could generate novel approaches for early diagnosis, prevention, and therapeutic interventions.

Abbreviations

Abbreviation	Definition
BDNF	Brain-Derived Neurotrophic Factor
CNS	Central Nervous System
FA	Fatty Acids
FMT	Fecal Microbiota Transplantation
GABA	Gamma-Aminobutyric Acid
GBA	Gut–Brain Axis

HC	Healthy Control(s)
ICD-10	International Classification of Diseases, 10th Revision
KYN	Kynurenine
KYNA	Kynurenic Acid
LAT	Large Neutral Amino Acid Transporter
MDD	Major Depressive Disorder
MUFA	Monounsaturated Fatty Acids
NMDA	N-Methyl-D-Aspartate (Receptor)
PRISMA	Preferred Reporting Items for Systematic Reviews and Meta-Analyses
PROSPERO	International Prospective Register of Systematic Reviews
PUFA	Polyunsaturated Fatty Acids
QUIN	Quinolinic Acid
SCFA	Short-Chain Fatty Acids
SCZ	Schizophrenia
SFA	Saturated Fatty Acids
SPF	Specific Pathogen-Free (mice)
Trp	Tryptophan
5-HT	Serotonin (5-hydroxytryptamine)
5-HIAA	5-Hydroxyindoleacetic Acid

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