

Prebiotics and Mental Health: Current Evidence and Emerging Perspectives

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Abstract

Neuropsychiatric disorders such as schizophrenia, attention deficit hyperactivity disorder (ADHD), and autism spectrum disorder (ASD) pose complex challenges to cognition, behaviour, and emotional regulation, with rising rates of treatment resistance further complicating management. Emerging evidence implicates the gut microbiome, acting through the gut–brain axis, in the pathophysiology of these conditions. Prebiotics, non-digestible compounds such as inulin and fructo-oligosaccharides, promote the growth of beneficial gut bacteria and enhance the production of short-chain fatty acids (SCFAs), neurotransmitters including serotonin, dopamine, and other metabolites that support neural function and emotional health. Preclinical studies, including maternal immune activation models in mice, demonstrate that prebiotic supplementation can mitigate cognitive deficits and behavioural abnormalities associated with microbial dysbiosis. Although human clinical trials remain limited, early findings suggest that dietary prebiotic interventions may improve symptoms in selected neuropsychiatric populations. Key challenges persist, however, including inter-individual variability in microbiome composition, optimal dosing strategies, and long-term efficacy. This review synthesises current evidence on the role of prebiotics in neuropsychiatric disorders, highlighting underlying mechanisms, therapeutic potential, and the need for personalised treatment approaches. It emphasises the importance of longitudinal, well-powered studies to translate preclinical findings into effective, microbiome-targeted adjunctive therapies for neuropsychiatric conditions.

Keywords: Anxiety, Depression, Gut–brain axis, Microbiome, Neuroinflammation, Prebiotics

1. Introduction

Neuropsychiatric disorders encompass a wide spectrum of neurological and psychiatric conditions that affect emotions, cognition, behaviour, and overall well-being in humans [1,2]. They are distinguished from purely psychiatric disorders by the presence of identifiable pathological changes in the nervous system [1]. Common neuropsychiatric conditions include schizophrenia, bipolar disorder, autism spectrum disorder, and attention-deficit/hyperactivity disorder (ADHD) [2]. Despite their substantial public health burden, these disorders are often underdiagnosed, underreported, and

undertreated in low- and middle-income countries. This under-recognition poses significant threats to national growth and development, as neuropsychiatric disorders have been described as a “silent pandemic” with profound effects on productivity, social cohesion, and overall human capital [3, 4].

The aetiology of neuropsychiatric disorders is multifactorial, involving genetic predisposition, neurochemical imbalances, environmental influences, and structural and functional alterations in different brain regions [5, 6]. Furthermore, there is considerable overlap in the clinical manifestations of neurological and psychiatric conditions, particularly in cognitive, emotional, and behavioural symptoms [2, 7]. For

instance, Alzheimer's disease and multiple sclerosis, though primarily neurological, often produce significant cognitive deficits. Conversely, psychiatric disorders such as schizophrenia, depression, and bipolar disorder are also associated with cognitive decline [8]. Similarly, emotional and behavioural disturbances including personality change, impulsivity, and mood instability are evident in neurological conditions like Parkinson's disease, traumatic brain injury, and epilepsy, as well as in psychiatric conditions such as anxiety and personality disorders [9,10].

Given this complexity, there is growing interest in systemic factors that bridge neurology and psychiatry. One such factor is the gut-brain axis, where the gut microbiome has emerged as a key modulator of neurodevelopment, cognition, and behaviour. This recognition has spurred investigation into microbiome-targeted interventions such as prebiotics, which may hold promise as novel adjunctive strategies in the prevention and management of neuropsychiatric disorders.

2. Prevalence of neuropsychiatric disorders

The Global Burden of Disease (GBD) Study reported that neuropsychiatric disorders account for approximately 13% of total disability-adjusted life years (DALYs) and 32.4 years lived with disability globally [11, 12]. Depression and anxiety disorders alone affect more than 500 million people worldwide [13], while schizophrenia affects between 0.3–0.7% of the global population [13]. The COVID-19 pandemic has further exacerbated this burden, with an estimated 25% global increase in depression and anxiety cases reported during the early phase of the pandemic [11].

Neurodegenerative conditions such as Alzheimer's disease and dementia affect over 50 million people globally, with projections indicating that this number will rise to nearly 140 million by 2050 due to population ageing in Asia, Europe, and the Americas [14,15]. In contrast, low- and middle-income countries, particularly in Africa, face unique challenges, including inadequate numbers of mental health professionals, poor surveillance systems, and underfunded or outdated mental health institutions [16, 17]. Stigma and cultural misinterpretations further contribute to under-reporting and treatment gaps [18]. According to the WHO, one in four Africans is affected by a neuropsychiatric disorder, yet the majority receive no formal treatment [13]. As of 2007, the prevalence of reported depression cases in sub-Saharan Africa was estimated at 5.4 million [19, 20].

In Nigeria, where the population exceeds 200 million, studies suggest that 20–30% of individuals are affected by neuropsychiatric conditions [19, 21]. Lifetime prevalence of depression has been reported at 26.2%, while anxiety disorders

affect approximately 10–15% of the population [24, 25]. Substance use disorders particularly involving cannabis and codeine are increasingly common among Nigerian youth, further predisposing the nation to a rising burden of neuropsychiatric illness [22, 23]. Schizophrenia and other psychotic disorders have an estimated prevalence of 1–2% [26, 27].

These global and regional epidemiological trends underscore not only the vast burden of neuropsychiatric disorders but also the inadequacy of existing diagnostic, therapeutic, and policy responses. Pharmacological and psychotherapeutic approaches to therapy, though beneficial, remain limited by issues such as treatment resistance, side effects, poor accessibility, and cultural barriers. These limitations have prompted a growing search for innovative, low-cost, and scalable strategies to complement existing treatments. In this context, the gut-brain axis and microbiome-targeted interventions including prebiotics have gained increasing attention as potential avenues for improving cognitive, emotional, and behavioural outcomes in neuropsychiatric disorders.

3. Neuroanatomical Disruptions in Neuropsychiatric Disorders

Neuropsychiatric disorders, representing a confluence of cognitive and emotional dysregulation, are known to arise from neuroanatomical and neurochemical changes that manifest as complex behavioural outcomes [28, 29]. The prefrontal cortex (PFC), hippocampus, basal ganglia, and amygdala undergo significant alterations in connectivity (Figure 1), plasticity, and neurotransmission in these disorders [30, 31].

The prefrontal cortex plays a crucial role in executive function, emotional regulation, attention, and decision-making [32]. Advances in neuroimaging have made it possible to localize disruptions within these circuits. For instance, functional magnetic resonance imaging (fMRI) studies reveal hypoactivation in the dorsolateral PFC and hyperactivation in the ventromedial PFC, contributing to social and affective deficits [32-34]. In schizophrenia, reduced PFC activity, diminished grey matter volume, and disrupted hippocampal-thalamic connectivity has been documented [35, 36]. In ADHD, delayed maturation and reduced right inferior frontal lobe volume, which mediates inhibitory control, are implicated in hyperactivity and impulsivity [37, 38].

Similarly, reduced hippocampal volume, disrupted circuitry, and hyperactivity are associated with bipolar disorder, depression, and schizophrenia [39, 40]. Amygdala hyperactivity, especially in anxiety and depression, amplifies

threat perception [41]. In the basal ganglia, particularly the substantia nigra, altered dopaminergic signalling impairs reward processing and motor regulation, features prominent in ADHD and schizophrenia [42, 43].

Evidence from animal studies further reinforces these links. Chronic stress or exposure to neurotoxins induces hippocampal neurodegeneration and PFC dysfunction, leading to working memory impairments and anxiety- or depression-like behaviours [44–47].

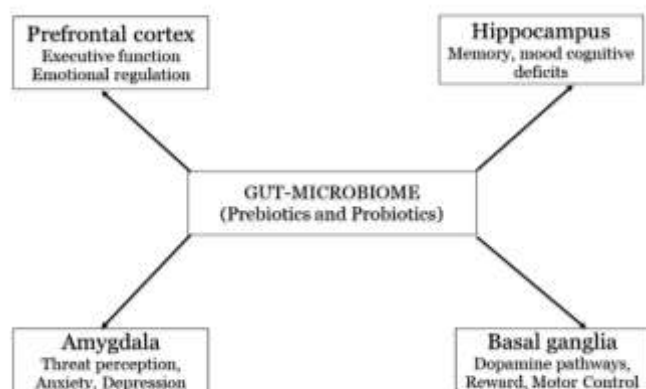


Figure 1: Neuroanatomical disruptions in neuropsychiatric diseases and their putative links to the gut-brain axis. The schematic highlights major brain regions implicated in neuropsychiatric disorders, PFC, hippocampus, amygdala, and basal ganglia showing how structural and functional abnormalities contribute to cognitive, emotional, and behavioural symptoms. Arrows from the gut microbiome illustrate emerging evidence that prebiotics and their metabolites (e.g., SCFAs, serotonin, dopamine precursors) may modulate these circuits through the gut–brain axis.

4. Neurochemical Disruptions in Neuropsychiatric Disorders

In the central nervous system, neurotransmitters act as key messengers, transmitting signals between neurons and regulating a wide range of physiological and psychological functions [48]. Both neurological and psychiatric disorders are characterised by alterations in specific neurotransmitter (Figure 2) systems [48, 49].

Dysregulation of the monoaminergic (serotonergic and noradrenergic) pathways has been strongly implicated in mood and attentional disorders. Altered serotonergic signalling within the dorsal raphe nucleus and prefrontal cortex, coupled with elevated norepinephrine levels, has been associated with anxiety- and depressive-like symptoms as well as attentional deficits in ADHD [37, 50]. Serotonin dysfunction contributes to neurological conditions such as migraine, and psychiatric conditions such as depression and anxiety [51, 52]. Similarly, norepinephrine imbalance impairs

arousal and vigilance, exacerbating symptoms across anxiety and attentional disorders.

The principal inhibitory neurotransmitter, γ -aminobutyric acid (GABA), plays a central role in psychiatric conditions such as anxiety disorders, and neurological disorders like epilepsy [53, 54]. Reduced GABAergic function leads to impaired inhibitory control, increasing susceptibility to hyperexcitability and dysregulated emotional responses. Dopamine functions both as a neurotransmitter and hormone, central to reward processing, mood regulation, and motor activity [55]. Hyperdopaminergic tone has been linked to cognitive deficits, impulsivity, and psychotic symptoms in schizophrenia; hyperactivity and inattention in ADHD; and blunted reward sensitivity in depression [28, 42, 56]. Dysfunction of the dopaminergic system also underpins motor disorders such as Parkinson’s disease and is consistently associated with schizophrenia [57].

Dysregulation of the hypothalamic–pituitary–adrenal (HPA) axis leads to elevated cortisol levels, impairing hippocampal neurogenesis and prefrontal cortical plasticity [58]. This contributes to stress-related vulnerability and the progression of mood and cognitive disorders. N-methyl-D-aspartate (NMDA) receptor hypofunction disrupts excitatory glutamatergic transmission, impairing synaptic plasticity and causing cortical hyperexcitability. Such dysfunctions produce neuronal injury and have been implicated in schizophrenia and depressive disorders [59, 60]. These findings underscore the centrality of the disruption of neurotransmitter systems in the pathophysiology of neuropsychiatric disorders, highlighting the interplay between excitatory, inhibitory, monoaminergic, and neuroendocrine systems in shaping cognitive, emotional, and behavioural outcomes.

5. Behavioural Signatures of Neuropsychiatric Disorders in Humans and Translational Animal Models

Neuropsychiatric disorders are often characterised by distinct behavioural patterns (Table 1) that can be studied both in humans and replicated in animal models, particularly rodents, to provide insights into underlying mechanisms and therapeutic targets.

In anxiety disorders, humans typically present with avoidance behaviours, excessive worry, hypervigilance, and somatic tension [61]. Translationally, these features are modelled in rodents using paradigms such as the open field test, elevated plus maze, and social interaction test [62], where animals

demonstrate hypervigilance and avoidance-like behaviours [63].

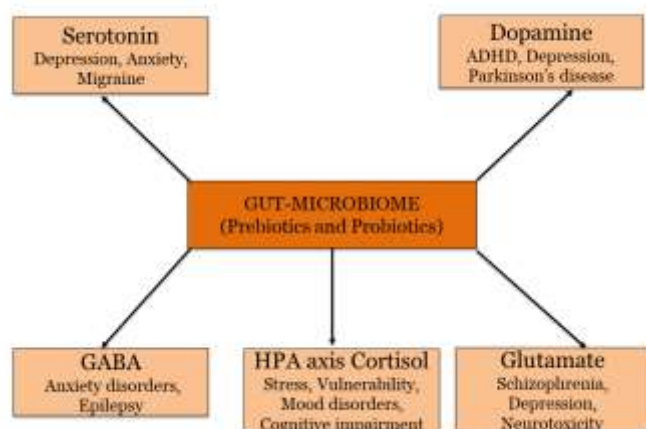


Figure 2. Neurotransmitter disruptions and associated neuropsychiatric disorders. The diagram illustrates how dysregulation of major neurotransmitter systems (serotonin, dopamine, GABA, glutamate/NMDA, and the HPA axis) are linked to distinct neuropsychiatric outcomes such as depression, anxiety, schizophrenia, ADHD, Parkinson's disease, epilepsy, and stress-related disorders. Arrows from the gut microbiome illustrate emerging evidence that prebiotics and their metabolites (e.g., SCFAs, serotonin, dopamine precursors) may modulate these circuits through the gut–brain axis.

Schizophrenia in humans is marked by a triad of positive symptoms (hallucinations and delusions), cognitive deficits (disorganised thoughts), and negative symptoms (social withdrawal) [64, 65]. These manifestations are mimicked in animal models through pharmacological manipulations (e.g., ketamine or phencyclidine exposure) or neurodevelopmental lesions such as the neonatal ventral hippocampal lesion model [66]. Rodents under these conditions exhibit behavioural analogues including sensorimotor gating deficits, cognitive impairments, and hyperambulation [66].

In bipolar disorder, the human clinical picture encompasses mood swings, manic and depressive episodes, cognitive hypofunction, social withdrawal, and disturbances of circadian rhythm [67]. Animal models, often employing open field paradigms [68], capture aspects of the disorder, with rodents demonstrating hyperlocomotion during manic-like states, alongside anhedonia and heightened stress reactivity during depressive-like phases [68].

Autism spectrum disorder (ASD) is typified in humans by flattened or labile affect, rigid adherence to routines, reduced eye contact, and abnormal sleep patterns [69]. Translational models include open field and social interaction tests [70], with rodents displaying reduced preference for social novelty and repetitive self-directed behaviours such as excessive grooming [70].

In major depressive disorder, individuals may experience suicidal ideation, anhedonia, fatigue, and social withdrawal [71]. Rodent models utilise paradigms like chronic mild stress and the forced swim test [72, 73], which elicit behavioural correlates such as behavioural despair and loss of interest in rewarding stimuli (anhedonia) [72, 73].

Finally, attention-deficit/hyperactivity disorder (ADHD) manifests in humans through hyperactivity, impulsivity, inattention, and executive dysfunction [74]. Rodent models include the spontaneous hypertensive rat (SHR) and dopamine transporter (DAT) knockout mice [75,76]. These animals present translationally relevant behaviours such as inattention, hypersensitivity, and hyperactivity [75,76]. Together, these behavioural signatures observed in humans are mirrored in rodent models through carefully designed tests and genetic or pharmacological manipulations. This translational alignment underscores the value of animal studies in dissecting the neurobiological basis of neuropsychiatric disorders and advancing therapeutic discoveries.

6. Nutrition and Neuropsychiatric Disorders

The interaction between nutrition and neuropsychiatric disorders has become an area of significant scientific inquiry, bridging neuroscience, psychiatry, and nutritional science [77, 78]. Accumulating evidence suggests that dietary patterns, specific nutrients, and gut–brain interactions contribute to the onset, progression, and management of these conditions [79, 80].

Balanced nutrition is fundamental for maintaining gut microbiome integrity, which in turn influences the gut–brain axis, neurotransmitter synthesis (for example, serotonin production from tryptophan), brain plasticity, cognitive processes, and the regulation of oxidative stress and inflammatory responses [81, 82]. Deficiencies in key nutrients can disrupt these mechanisms, thereby predisposing individuals to, or exacerbating, neuropsychiatric disorders [78, 82].

Notably, the SMILES trial, a randomised controlled study, provided compelling evidence that dietary improvement could significantly reduce depressive episodes [83]. Similarly, adherence to Mediterranean-style diets, rich in fruits, vegetables, whole grains, and fish, has been consistently associated with a reduced risk of depression [84].

Within this context, prebiotics dietary components that selectively promote the growth of beneficial gut bacteria have emerged as a promising adjunct in modulating neuropsychiatric outcomes. By enhancing gut microbial composition and function, prebiotics may indirectly influence

neurochemical pathways and brain function, thereby mitigating behavioural and emotional disturbances.

Accordingly, this review seeks to explore the potential role of prebiotics as dietary supplements in the prevention and management of neuropsychiatric disorders, synthesising evidence from preclinical and clinical studies to clarify their therapeutic promise.

Table 1: Behavioural phenotypes of neuropsychiatric disorders

Disorders	Human behavioural features	Animal models	Rodent behavioural features
Anxiety	Avoidance, excessive worry, hypervigilance, and somatic tension [61]	Open field test, Elevated plus maze, social interaction test [62]	Hypervigilance, avoidance [63]
Schizophrenia	Positive symptoms – hallucinations, delusions Cognitive deficits – disorganised thoughts Negative symptoms – social withdrawal [64,65]	Ketamine/PCP models, neonatal ventral hippocampal lesion [66]	Sensorimotor gating deficits, cognitive impairment, hyperambulation [66]
Bipolar disorder	Mood swings, manic and depressive episodes, cognitive hypofunction, social withdrawal, altered circadian rhythm [67]	Open field test [68]	Hyperlocomotion, anhedonia, increased stress reactivity [68]
Autism spectrum disorder	Flattened/labile affect, routine rigidity, reduced eye contact, abnormal sleep pattern [69]	Open field test, social interaction test [70]	Reduced social novelty preference and increased repetitive grooming behaviour [70]
Depression	Suicidal ideation, anhedonia, fatigue, and social withdrawal [71]	Chronic mild stress and forced swim test [72,73]	Behavioural despair, anhedonia [72,73]
ADHD	Hyperactivity (physical/mental), impulsivity, inattention, and executive dysfunction [74]	Spontaneous hypertensive rats (SHR), dopamine transporter (DAT)-knockout mice [75,76]	Inattention, hypersensitivity [75,76]

6.1 Prebiotics

Prebiotics (Table 2) are non-digestible food components, typically derived from high-fibre meals, that selectively stimulate the growth and activity of beneficial microbes within the human gut microbiota [85]. By fostering microbial balance, prebiotics indirectly influence host physiology, including gut health, metabolism, immune regulation, and brain function. Common dietary sources include whole grains,

bananas, leafy greens, onions, garlic, soybeans, and artichokes [85].

The original definition of prebiotics, which remained largely unchanged for nearly 15 years, was limited to a small group of carbohydrates such as lactulose, galactooligosaccharides (GOS), and short- and long-chain fructans, including fructooligosaccharides (FOS) and inulin [86,87]. More recently, additional classes such as pectin, resistant starch, glucomannan, beta-glucan, and arabinoxylan have also been recognised for their prebiotic potential [85, 86].

6.2 Lactulose

Lactulose is a synthetic disaccharide composed of fructose and galactose that does not occur naturally in foods. Within the colon, it is fermented by microbes such as *Bifidobacterium* and *Lactobacillus*, producing short-chain fatty acids (SCFAs) like acetate, propionate, and butyrate [88,89]. These metabolites provide energy to colonocytes, lower colonic pH, enhance mineral absorption (e.g., calcium, magnesium), strengthen intestinal barrier function, and modulate inflammatory pathways [88].

Clinically, lactulose is prescribed in the management of hepatic encephalopathy a neuropsychiatric complication of liver disease driven by hyperammonaemia where it reduces plasma ammonia levels by improving gut barrier function and microbial balance [90, 91, 92]. A randomised controlled trial (2 g/day) in healthy Japanese women also demonstrated improved bowel function and increased bifidobacteria counts following lactulose supplementation [93].

Animal studies further support its neuroprotective potential. In rodent models, lactulose supplementation increased microbial diversity and reduced pathogenic bacteria [94, 95]. In pregnant mice, treatment enhanced SCFA production and downregulated factors linked to adverse neurodevelopmental outcomes [94, 96]. In hyperammonaemia rat models, lactulose reduced plasma ammonia, improved motor coordination, and attenuated neuronal hyperactivity, suggesting protective effects on brain function [97, 98].

6.3 Galactooligosaccharides (GOS)

Galactooligosaccharides consist of galactose units synthesised via enzymatic transglycosylation of lactose, producing compounds resistant to digestion in the small intestine [99]. They are selectively fermented by gut microbes, particularly *Bifidobacterium* and *Lactobacillus* [100]. Naturally, GOS occur in human breast milk, where they play a role in shaping the infant gut microbiota; however, most dietary GOS are

produced commercially and incorporated into supplements and functional foods [99].

Beyond gut modulation, GOS also exhibit immunomodulatory effects, suppressing pro-inflammatory cytokines and promoting anti-inflammatory responses [101, 102]. A double-blind, placebo-controlled study in 59 healthy adults demonstrated significant bifidogenic effects of a novel GOS mixture [103]. Similarly, GOS supplementation (2.8 g/day) in patients with ulcerative colitis improved stool consistency and microbial diversity, although inflammatory markers were unchanged [104].

Preclinical studies highlight the neuroprotective potential of GOS. In a mouse model of amyotrophic lateral sclerosis (ALS), supplementation delayed disease onset, reduced motor neuron death, and increased folate and vitamin B12 levels, while modulating inflammatory and apoptotic pathways [105, 106]. In aged mice, GOS improved gut barrier integrity by enhancing mucin production, reducing intestinal permeability, and enriching beneficial bacteria such as *Bacteroides* and *Lactobacillus*, thereby attenuating age-related systemic inflammation [107].

6.4 Straight-Chain Fructans (FOS and Inulin)

Straight-chain fructans, including fructooligosaccharides (FOS) and inulin-type fructans, are polysaccharides composed of linear fructose chains [108]. They occur naturally in chicory root, onions, garlic, artichokes, bananas, asparagus, leeks, and whole wheat, with chicory root being the primary source of industrial inulin production [85,108]. FOS, consisting of 2–10 fructose units with a terminal glucose [109], are strongly bifidogenic. A systematic review of 8 human and 20 animal studies reported that FOS supplementation increased *Bifidobacterium* abundance, enhanced SCFA levels, stimulated colonic IgA production, and suppressed pro-inflammatory cytokines [110].

An in vitro fermentation study demonstrated that FOS promoted bifidogenesis and SCFA production in an age-dependent manner [111]. In a high-fat diet-induced obesity mouse model, combined FOS and GOS supplementation restored synaptic plasticity markers (p-CREB, BDNF, NeuN), improved spatial memory, and reduced neuroinflammation [112]. Inulin exists in both short-chain (oligofructose) and long-chain (true inulin) forms, with a degree of polymerisation ranging from 2 to 60 [113]. A placebo-controlled study in 30 adults showed that daily supplementation with 5–8 g of inulin increased bifidobacteria counts in faeces, with effects inversely related to baseline microbiota levels [114]. Similarly, in type 2 diabetes patients, 16 g/day of inulin–

oligofructose for 6 weeks increased SCFA production and bifidobacterial populations compared to controls [115].

Table 2. Summary of Prebiotics: Sources, Gut Effects, and Neuropsychiatric Relevance

Prebiotic	Main Sources	Gut Microbiota Effects	Neuropsychiatric/Clinical Relevance
Lactulose	Synthetic disaccharide (not naturally occurring in food)	↑ <i>Bifidobacteria</i> , ↑ <i>Lactobacilli</i> , ↑ SCFAs (acetate, propionate, butyrate); ↓ colonic pH; ↑ mineral absorption; improved gut barrier integrity [88,89]	Clinically used for hepatic encephalopathy (reduces ammonia); in rodents, improves motor function, ↓ neurotoxicity, enhances neurodevelopmental outcomes [90–98]
Galactooligosaccharides (GOS)	Human breast milk (natural); commercial supplements (enzymatic conversion of lactose) [99]	↑ <i>Bifidobacteria</i> and <i>Lactobacilli</i> ; ↓ pro-inflammatory cytokines; improved stool consistency [100–104]	In ALS mouse models, delayed disease onset and protected neurons; in aged mice, improved gut barrier, reduced systemic inflammation, and enhanced cognition [105–107]
Fructooligosaccharides (FOS)	Chicory root, onions, garlic, bananas, asparagus, leeks, wheat [108,109]	↑ <i>Bifidobacteria</i> , ↑ SCFAs, ↑ IgA; ↓ pro-inflammatory cytokines; age-dependent bifidogenic effects [110–111]	In obese mice, restored synaptic plasticity markers, improved spatial memory, and ↓ neuroinflammation [112]
Inulin (short- and long-chain)	Chicory root (primary source), bananas, leeks, asparagus, wheat, garlic [85,108,113]	↑ <i>Bifidobacteria</i> (dose-dependent), ↑ SCFAs, improved gut metabolic balance [114–115]	In type 2 diabetes, improved gut microbiota composition; potential indirect benefits for brain function via metabolic and inflammatory regulation [115]

7. Gut Microbiota

The gut microbiota refers to the complex community of microorganisms residing in the gastrointestinal tract (GIT), playing a central role in maintaining homeostasis by regulating metabolic and physiological processes of the host [116]. Each individual harbours a unique and abundant microbial composition, with the phyla *Firmicutes* and *Bacteroidetes* together accounting for approximately one-quarter of the total gut microbiome [116, 117].

The human gut contains between 300 and 500 distinct bacterial species, with considerable inter-individual variability in composition [120]. Because the upper digestive tract is

highly acidic and enriched with bile and pancreatic secretions toxic to many microbes, the majority of bacteria colonise the large intestine, where conditions are more favourable [120]. Importantly, while some bacterial species may be pathogenic, the continuous host–microbe interaction typically provides mutual benefits, contributing to nutrient metabolism, immune function, and energy balance [118, 121, 122].

Beyond digestion and absorption, the gut microbiota exerts systemic effects on host health. Early clinical evidence of a gut–brain link was observed when a patient with hepatic encephalopathy achieved full neurological recovery following antibiotic therapy [123]. Since then, mounting evidence has implicated the gut microbiome in modulating brain function and behaviour, with studies showing that alterations in microbial communities can influence the risk and severity of neurological and psychiatric conditions [66, 124]. Thus, the gut microbiota is now recognised not only as a regulator of gastrointestinal and metabolic processes but also as a critical player in the gut–brain axis, with profound implications for neuropsychiatric health.

7.1 Gut Microbiome Classes

The gut microbiome of a healthy host is dominated by bacteria, primarily belonging to the phyla *Firmicutes* (e.g., *Lactobacillus*, *Faecalibacterium*, *Ruminococcus*, *Clostridium*, and *Enterococcus*), *Bacteroidetes* (e.g., *Bacteroides*, *Prevotella*, and *Parabacteroides*), *Actinobacteria*, and *Proteobacteria* [125, 126]. The diversity and abundance of these microbes vary depending on host-related factors such as age, diet, lifestyle, and health status [126, 127]. Functionally, gut microbes ferment non-digestible dietary fibres in the colon, producing short-chain fatty acids (SCFAs)—notably acetate, propionate, and butyrate—that serve as energy sources for colonocytes, strengthen intestinal barrier integrity, and maintain epithelial health. They also synthesise key neurotransmitters (e.g., γ -aminobutyric acid [GABA], serotonin, and dopamine), facilitating gut–brain communication, cognition, and mood regulation. In addition, the microbiome produces bile acid metabolites that influence glucose uptake, antimicrobial activity, and lipid digestion [128,129]. Other important contributions include the biosynthesis of vitamins and cofactors (vitamin K, vitamin B12, folate, and biotin) and the metabolism of dietary polyphenols, thereby supporting host metabolic and systemic health [130,131].

7.2 Gut Microbiota and Brain Health

Communication between the gut and the brain occurs through neural, hormonal, and immune pathways—collectively termed the gut–brain axis. Microbial activity influences mood, cognition, stress resilience, and neurodevelopment by modulating SCFA production, neurotransmitter activity, and inflammatory signaling. The first clinical evidence of a gut–brain connection dates back decades: a patient with hepatic encephalopathy experienced full neurological recovery following antibiotic treatment, which altered gut microbiota composition [123].

Recent research has revealed key roles of the microbiome in neurodevelopmental and neuropsychiatric disorders [124]. In murine models, maternal immune activation (MIA) during pregnancy induced cognitive deficits and altered serotonin (5-HT_{2A}) receptor expression in offspring. These impairments were prevented by antibiotic treatment in prepubertal mice, suggesting microbiota-dependent modulation of neurodevelopment. Transfer of gut microbes from MIA-affected mothers into healthy mice reproduced similar cognitive deficits, implicating dysbiosis in the pathophysiology of disorders such as schizophrenia and autism spectrum disorder (ASD) [124,132]. Increased abundance of *Clostridia* and *Bacteroides* in MIA models has been directly linked to behavioural abnormalities.

In humans, microbial dysbiosis has also been linked to structural and functional brain changes. A cohort study of >1,000 participants with cognitive impairment reported reduced SCFA-producing microbes such as *Odoribacter*, which correlated with smaller hippocampal and white matter volumes and increased cerebrospinal fluid (CSF), suggesting microbe-driven neuroinflammation [133]. Another population-based study found that higher microbial diversity was associated with better cognitive performance, with *Verrucomicrobia* linked to enhanced processing speed and *Akkermansia* (a mucin degrader) associated with both improved cognition and intestinal health [134]. Additional studies report that SCFA-producing microbes correlate with stronger brain connectivity and reduced anxiety and depression in young adults [91,135]. A therapeutic approach, faecal microbiota transplant (FMT), further underscores the influence of gut microbes on the brain. By transferring stool from healthy donors into recipients with dysbiosis, FMT has been shown across multiple human and animal studies to restore microbial balance and improve behavioural and cognitive outcomes [136, 137].

Conclusion

Neuropsychiatric disorders such as schizophrenia and autism spectrum disorder (ASD) are characterised by complex neurological and behavioural impairments that significantly affect cognition, emotion, and quality of life. While current therapeutic strategies including pharmacological and behavioural interventions offer some benefit, the growing prevalence of treatment resistance remains a critical challenge.

This review highlights the emerging role of gut microbiota modulation as a promising adjunctive or alternative strategy for managing neuropsychiatric disorders. Approaches such as dietary enrichment with prebiotic fibres and faecal microbiota transplantation (FMT) have been shown to restore microbial balance, enhance the production of beneficial metabolites (e.g., SCFAs, neurotransmitters), and support gut–brain communication. These effects may contribute to improved cognitive performance, emotional regulation, and overall mental well-being. Taken together, the evidence positions the gut microbiota as a viable therapeutic target for both the treatment and long-term management of neuropsychiatric conditions, opening pathways for the development of personalised, microbiome-based interventions.

Recommendations and Future Directions

Targeting microbial dysbiosis offers a novel opportunity to complement existing therapies for schizophrenia, ASD, and related disorders, particularly in treatment-resistant cases. To advance this field, several directions should be prioritised: Future interventions should integrate microbiome profiling with genetic and clinical data, enabling tailored therapeutic strategies that account for individual variability in microbial composition and host response. Bridging the gap between rodent model findings and human clinical trials is essential. Research should validate mechanisms such as SCFA production, neurotransmitter modulation, and immune regulation in human populations. Incorporating microbiome health into mental health policies will be key. This may include the development of dietary guidelines promoting fibre-rich, microbiota-supporting foods, as well as early microbiome-based screening for individuals at risk of neuropsychiatric disorders. Future studies should adopt long-term designs to assess how microbiota-targeted interventions influence disease trajectory, treatment resistance, and relapse risk over time. By combining advances in microbiome science with precision psychiatry, the field is poised to develop innovative, sustainable, and patient-centered approaches to neuropsychiatric care.

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