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REVIEW ARTICLE

The Role of Gut-Microbiota in Neuropharmacology

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ABSTRACT
The gut microbiota, a complex community of microorganisms residing in the gastrointestinal tract, plays a pivotal role in maintaining human health through its interactions with the gut–brain axis (GBA). This bidirectional communication network links the enteric nervous system, central nervous system, endocrine signaling, immune pathways, and microbial metabolites. Disruptions in gut microbiota composition, termed dysbiosis, have been increasingly associated with neurological and psychiatric disorders such as Alzheimer's disease, Parkinson's disease, multiple sclerosis, autism, anxiety, and depression. Mechanistically, gut microbes influence neuropharmacology by modulating neurotransmitter synthesis (including serotonin, dopamine, GABA, and acetylcholine), immune responses, and inflammation. They also affect drug metabolism, efficacy, and toxicity through microbial enzymatic activity, highlighting their relevance in pharmacomicrobiomics. Therapeutic strategies such as probiotics, psychobiotics, prebiotics, synbiotics, and fecal microbiota transplantation (FMT) have demonstrated potential in restoring microbial balance and alleviating neurological symptoms. Furthermore, emerging research emphasizes microbiome-based interventions as promising tools in personalized medicine for neurodegenerative and neuropsychiatric diseases. However, methodological inconsistencies in microbiome studies and the complexity of host–microbial interactions continue to challenge reproducibility and clinical translation. Future directions include integrating high-throughput sequencing, bioinformatics, and systems biology to unravel causal mechanisms, develop standardized research protocols, and optimize microbiome-targeted therapies. Overall, understanding the gut–brain–microbiota axis provides valuable insights into disease pathophysiology and offers innovative approaches for neuropharmacological interventions, underscoring the gut microbiota as both a diagnostic biomarker and a therapeutic target in modern neuroscience
Keywords: *Gut microbiota, Gut-brain axis , Neuropharmacology, Dysbiosis, Neurotransmitters*

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INTRODUCTION

The gut microbiota (GM) is the collective term for the trillions of microorganisms that live in the human body, the majority of which are found in the gastrointestinal tract. There are four major phyla—Bacteroidetes, Firmicutes, Proteobacteria, and Actinobacteria—as well as two minor taxa, Verrucomicrobia and Fusobacteria, make up the majority of the GM, albeit its composition fluctuates depending on host-related conditions. In addition to interacting with the host's gut epithelium, these commensal bacteria also work together to improve immunity and preserve homeostasis [1]. The gut microbiota's ability to influence brain-related processes implies that it initiates the synthesis of immune factors, such as cytokines and inflammatory mediators, that target the central nervous system (CNS) and the enteric nervous system (ENS). A part of the peripheral nervous system, the autonomic nervous system controls physiological functions that are not under conscious control. Through the coordination of complementary reactions between the sympathetic and parasympathetic neural systems, it regulates essential visceral functions. The discovery of the autonomic nervous system branch known as the ENS significantly increased our understanding of the bidirectional communication between the central nervous system (CNS) and the digestive tract [2]. Through the GBA, GM can affect neurological processes that shape mood, cognition, and brain development, including axonal growth, apoptosis, synaptogenesis, myelination, neurogenesis, and even the control of circulating growth factors like insulin-like growth

factor-1 (IGF-1). As a highly complex "second brain" inside the GIT, the ENS itself, which includes the myenteric (Auerbach's) and submucosal (Meissner's) plexuses, communicates directly with the central nervous system (CNS) via vagal and autonomic routes. Neurological and psychiatric conditions such as Alzheimer's disease, Parkinson's disease, multiple sclerosis, autism spectrum disorder, epilepsy, amyotrophic lateral sclerosis, Huntington's disease, stroke, and depression have all been linked to the development and progression of dysregulation of GM composition. On the therapeutic front, strategies such as probiotics, prebiotics, postbiotics, symbiotics, antibiotics, and fecal microbiota transplantation (FMT) are being actively explored to restore microbial balance and alleviate neurological symptoms [3]. The microbiome, which was previously disregarded, is now understood to be a "drugable target" that can be altered purposefully (probiotics, for example) or accidentally (antibiotics, for example), impacting the safety and effectiveness of medications. The gut microbiota is becoming more and more recognized as being essential to risk assessment, personalized medicine, and drug safety due to its involvement in metabolic variability and host interactions [4].

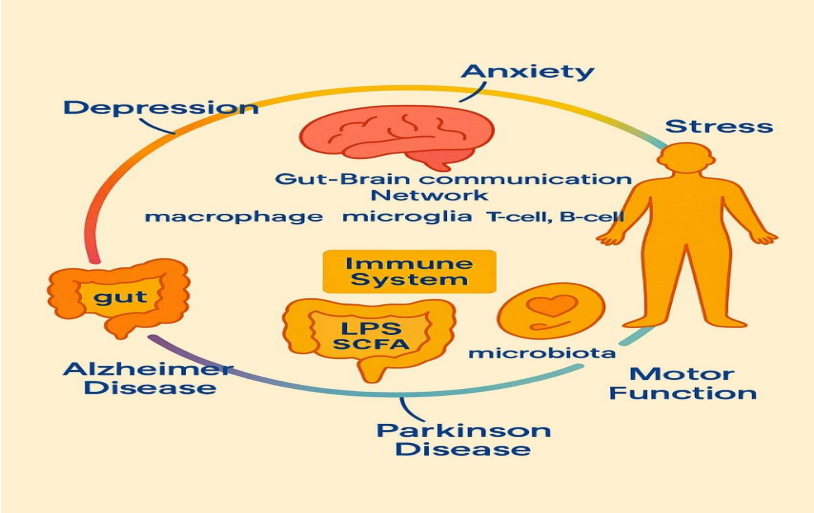

Fig.1: Gut-Brain Communication

GUT-BRAIN AXIS

The intestinal microbiota has recently been found to have an unexpected effect in controlling mood and gastrointestinal (GI) motility. The entire network is now frequently referred to as the "gut connectome" because the microbiome lives in the gut lumen and interacts intimately with the brain and enteric nervous system (ENS). The intricate, two-way communication that occurs within this system is still a mystery to researchers. Through immunological, endocrine, and neural signals, gut microbes can affect the central nervous system (CNS). The gut environment can then be influenced by the brain. For example, stress can cause the release of certain mediators that change the gene expression of bacteria or alter gut function through the sympathetic and parasympathetic nervous systems, affecting immunological responses, motility, and secretion. By controlling intestinal processes including secretion, motility, permeability, and immunological activity—all of which influence the makeup and behavior of gut microorganisms—the enteric nerve system (ENS) plays a crucial part in this conversation. Numerous mechanisms, such as bacterial metabolites, immunological chemicals, paracrine messengers, neurotransmitters, and vagus nerve transmission, are involved in communication along the microbiota–gut–brain axis. A growing body of research indicates that disruptions in these pathways, along with the microbial imbalances they cause, may play a role in the emergence of neurodegenerative diseases like Parkinson's and Alzheimer's, as well as neurological and psychiatric disorders like depression and autism spectrum disorder [5]. However, there is mounting evidence that the gut microbiota is significantly shaped by our food. Diets that are low in fiber and high in sugar and saturated fats can upset the gut's microbial balance. Remarkably, prebiotic therapies, which support good gut flora, have been demonstrated in diabetic rats to lower inflammation and enhance glucose tolerance. Notably, GI function has also been observed to be improved by mood disorder medicines, such as selective serotonin reuptake inhibitors (SSRIs). This knowledge has been expanded by recent research on aerobic exercise. The impacts of obesity, metabolic diseases, poor food, and even some neurological or behavioral disorders may be mitigated by these alterations in gut microbes and their metabolic byproducts. It's still unclear how much exercise affects the relationship between the gut and the brain, but it's becoming clear that the

two are closely related. Thus, the purpose of this review is to present the complex interrelationship between the gut, the brain, and the microbiome, the so-called microbiome–gut–brain axis. Changes in the gut microbiota within this system can affect neural transmission, neurogenesis, and behavior in the central nervous system as well as gastrointestinal processes like secretion, motility, and intestinal barrier integrity. The stomach may influence the brain, and the brain can influence the gut. This is significant since these interactions are reciprocal [6].

MECHANISMS OF GUT MICROBIOTA INFLUENCES ON NEUROPHARMACOLOGY

A. GUT BACTERIA AND MODULATION

The gut microbiome's composition, its reaction to diet and medication, its function in immune regulation and digestion, and its impact on entero-endocrine signaling pathways are the main topics of the majority of current research on the human gut. But as we learn more, especially about the gut-brain axis (GBA), the hypothalamic-pituitary-adrenal (HPA) axis, and the gut's connection to cognitive and neuropsychiatric conditions like autism, depression, and schizophrenia (as reviewed by Dicks et al.), new questions about how gut bacteria affect the production of important neurotransmitters like γ-aminobutyric acid (GABA), dopamine (DA), norepinephrine (NE or noradrenaline), serotonin (5-HT), and histamine are becoming more apparent. Four general categories can be used to classify neurotransmitters: Glutamate, acetylcholine, histamine, dopamine, norepinephrine, and epinephrine are examples of excitatory neurotransmitters. Neurotransmitters that inhibit (such as GABA, serotonin, and dopamine) Dopamine, serotonin, acetylcholine, histamine, and norepinephrine are examples of neuromodulators [7].

B. MICROBIOTA AND NEUROTRANSMITTERS

Glutamate: The primary excitatory neurotransmitter in the brain must be produced locally since it cannot cross the blood–brain barrier. It is produced in the brain by astrocytes and neurons working together, using intermediates from the tricarboxylic acid cycle (TCA). Specialized enteroendocrine “neuropod” cells in the stomach can also produce glutamate and use VGLUT1 transporters to swiftly transmit signals to the brain via the vagus nerve.

GABA: A crucial inhibitory neurotransmitter, GABA is involved in numerous physiological and metabolic processes. It is produced in the brain by GABA-producing neurons, which use the enzyme glutamic acid decarboxylase to convert glutamate into GABA.

Acetylcholine: In both invertebrates and vertebrates, acetylcholine is a crucial neurotransmitter. It is essential to the nervous system because it acts locally to send excitatory impulses between neurons in the peripheral and central nervous systems. Acetylcholine imbalances are closely associated with neurodegenerative diseases, such as Alzheimer's.

Serotonin: Specialized neurons in the brain's raphe nuclei produce the majority of serotonin, and mental health disorders like anxiety and depression are associated with abnormalities in serotonin levels.

Norepinephrine: One important catecholamine that functions as a neurotransmitter in the brain and other parts of the body is norepinephrine. It is essential for maintaining our vigilance, concentration, and readiness to face obstacles. It also triggers the "fight-or-flight" reaction in times of stress or danger. Neurons in the locus coeruleus, which converts the amino acid tyrosine first into dopamine and then norepinephrine, are the main source of norepinephrine in the brain[8].

Dopamine: Similar to serotonin, dopamine plays a critical role in controlling movement and emotional reactions. For general physical and mental well-being, dopamine balance must be maintained. Significant levels of free dopamine and norepinephrine have been detected in the guts of several Clostridium species, while Bacillus oklahomensis has been linked to elevated synthesis of the dopamine metabolite 3,4-dihydroxyphenylacetic acid. Dopamine (and serotonin in some situations) can also be produced by other bacteria, including Morganella morganii, Hafnia alvei, Klebsiella pneumoniae, and Escherichia coli[9].

IMMUNE AND INFLAMMATORY PATHWAYS

There is growing evidence that changes in brain shape are caused by microorganisms in the gut microbiome. First, studies on GF animals showed that the absence of the microbiota affects the anatomy of the brain. Second, mice given certain bacterial strains showed alterations in various brain areas. Additionally, human genomic research on these strains and the brain confirmed the findings' potential relevance. Lastly, preclinical research has shown that antibiotic exposure or a persistent bacterial infection brought on by gut dysbiosis in early childhood or adulthood can have long-term effects on the brain, spinal cord, and ENS. Myelination, dendritic and microglial morphology, BBB permeability and structure, neurogenesis, and synaptic structure and function can all be impacted by the gut bacteria [10]. The innate immune signaling pathway is a key regulatory mechanism in the development of inflammatory

and infectious disorders, which can be brought on by immune system imbalance. It is hypothesized that the innate immune signaling system influences inflammatory and infectious illnesses via regulating the MGB axis [11].

ROLE IN SPECIFIC NEUROLOGICAL AND PSYCHIATRIC DISORDERS

ALZHEIMER'S DISEASE

The gradual, irreversible neurological disease known as Alzheimer's disease (AD) is characterized by behavioral abnormalities, cognitive decline, and memory loss. Among its hallmarks are amyloid-β (Aβ) plaque buildup, neuronal death, synaptic failure, and aberrant tau protein phosphorylation, which causes microtubule instability, calcium imbalance, and neuronal apoptosis. Although the exact causes of AD are still unknown, there is evidence that microbial infections and neuroinflammation are linked to its progression. Infections with bacteria (like Chlamydia pneumoniae and Porphyromonas gingivalis), viruses (like HSV-1), and fungi can cause tau damage and Aβ buildup. Dysbiosis of the gut microbiota has been identified as a possible cause of AD through the gut–brain axis (GBA). Reductions in anti-inflammatory Eubacterium rectale and elevations in proinflammatory Escherichia and Shigella are two examples of the changed microbial composition found in AD models and patients [12]. Gut Microbes' Direct Impact on AD Bacterial metabolites can disperse and move around. structurally, or perhaps cross the BBB and get at the cerebrospinal fluid (CSF) in either a healthy or particular state. But these Toxins or bacterial metabolites may ultimately lead to illness or alter immunological and biochemical aspects of health without infecting or causing sepsis, which is the so-called "leaky gut growth" [13].

PARKINSONS DISEASE

For Parkinson's disease (PD), the second most prevalent neurodegenerative disease in the elderly, the average period from diagnosis to death is 15 years. Although the exact origin of Parkinson's disease is still unknown, the GI tract is the source of numerous toxins that affect the central nervous system and interact with the brain through the vagus nerve's dorsal motor nucleus. These symptoms include mood, cognitive impairment, sleep difficulties, sensory disruption, and gastrointestinal dysfunction. PD patients frequently have abnormalities in their gut microbiome. and the connection between them and brain disorders is being thoroughly looked into. In three separate studies Regarding the composition of the fecal microbiota, there was a decline in microbes Prevotellaceae, Coprococcus, and Firmi- B. cutes, *Clostridium leptum*, and *Clostridium coccoides*. Fragriosis and more Lactobacillaceae representation, Verru- Ruminococcaceae, Lactobacuillus, Bacte, and Comicrobiaceae Proteobacteria, Clostridiaceae, Akkermansia, and roidetes. PD patients' gut microbiome are changed to a pro-inflammatory state because several genes pertaining to "pro-inflammatory" and LPS generation Bacterial taxa such as Ralstonia and Akkermansia were discovered. to be elevated in people who are infected [14]. The development of PD pathology is thought to be significantly influenced by dysbiosis-related impairments, including inflammation, altered immunological responses, and increased intestinal permeability. In view of recent discoveries about the pathophysiology of neurodegenerative diseases in both human and animal models, the current research has therefore suggested altering the gut microbiota as a possible treatment approach.[15].

MULTIPLE SCLEROSIS

Unlike AD and PD, multiple sclerosis (MS) is a condition that primarily affects young people. adults, especially ladies. It is a CNS disorder that demyelinates and has an inflammatory part. Chronic inflammation in both the white and gray tissue is a hallmark of multiple sclerosis. This results in the breakdown of the myelin sheath around the brain and spinal cord. neurons Despite the incomplete understanding of the mechanisms behind MS, it is assumed that immune system dysfunction is the most likely cause of the illness. This condition makes it easier for immune cells, primarily T-cells, to migrate. to the brain's penetration and the nerve system . After entering the central nervous system, T-cells begin to identify myelin as an immune system trigger, leading to increased inflammation, which causes demyelination. Environmental and genetic factors may potentially play a role in the pathophysiology of MS. Early-life obesity reduced blood levels of vitamin D, and in both smoking and enough exposure to sunshine are the recognized and most commonly cited reasons. microbiota. Because microbes can regulate T-cells to influence immunity, the gut microbiome has drawn notice as a significant contributing element to MS pathophysiology[16].

ANXIETY

The emotional state of anxiety is brought on by immunological, endocrine, and neurological processes. Stress, whether from biological, environmental, or psychological sources, can trigger anxiety reactions by triggering the HPA axis or immune system reactions. In comparison to SPF mice with normal microbiota, GF mice exhibit reduced anxiety-like behaviors and greater motor activity. In the GF condition, a decrease

in anxiety-like behaviors is linked to increased 5-HT and tryptophan metabolism as well as long-term regulation of synaptic transmission through decreased expression of synaptophysin and postsynaptic density protein 95. These results suggest that gut microbiota The degree of the HPA axis response is regulated by biota. Pathogenic gut bacteria-induced dysbiosis can encourage and exacerbate anxiety by interfering with the body's immunological and metabolic systems gut-brain-microbiota axis. of neuronal activities, without raising cytokines that promote inflammation. Anxiety-like conduct was linked to elevated expression of c-Fos, a neural activation marker after exposure to *Campylobacter jejuni*, especially in areas of the brain connected to stress regulation and autonomic reactions. There has been a noticeable rise in anxiety-like behaviors. Observing infection with *Citrobacter rodentium*, most likely caused by vagal sensory neurons, but no rise in inflammatory indicators. Likewise, an infection with Trichuris muris has been demonstrated to increase anxiety-like symptoms via metabolic and immunologic processes. Conversely, using probiotics to treat dysbiosis may reduce fear. Certain Bifidobacterium and Lactobacillus species, renowned for their ability to reduce anxiety, including *Bifidobacterium longum, Bifido- Lactobacillus helveticus, Lactobacillus rhamnosus*, or *Bacillus infantis*, have been demonstrated to normalize whether utilized separately or in combination. behavioral characteristics in models of animal anxiety by adjusting immuno GABA receptor regulation and nologic factors [17].

NEUROPHARMACOLOGY THERAPEUTIC APPROACHES THAT TARGET THE MICROBIOTA
PROBIOTICS AND PSYCHOBIOTICS

Probiotics are live, non-toxic yeast and bacteria that are advantageous for the human body when administered, enhance and encourage the equilibrium of microbes, especially in the digestive system. They are mostly made up of *Lactobacillus* and either *Saccharomyces boulardii* or *Bifidobacterium* species. The probiotic strains play a role in a number of physiological processes, such as lowering the intestinal pH, cell- to-cell communication, reducing and stopping the spread of harmful microorganisms and controlling the host's immunological response. "Psychobiotics" are a distinct class of probiotics that may enhance mental and psychological well-being and affect anxiety, mood, cognition, memory, and focus..". Several gastrointestinal hormones, short-chain fatty acids, vitamins, and medication absorption are all linked to GM[18]. It would seem that psycobiotics can alter the local balance of inhibition/excitation to regulate the systemic reactions, as GABA is the primary inhibitory neurotransmitter of the nervous system. Depression, anxiety, and stress Nowadays, psychobiotics are thought to be important components of affective disorders [19].

FECAL MICROBIOTA TRANSPLANTION

FMT is a new and powerful technique for repairing the gut ecology. The microbiomes that are rich in species and metabolites and their products from healthy donors can change the production and boost the diversity of microorganisms of metabolites from the host and certain microorganisms, and change the immunological response of the host[20].We regularly receive questions regarding the potential of FMT for a range of clinical issues, such as IBD, IBS, obesity, anorexia nervosa, systemic autoimmunity, food allergies, eosinophilic disorders of the gastrointestinal tract, as well as neurodegenerative and neurodevelopmental disorders. FMT can be curative in their individual conditions [21].

SYNBIOTICS

The host microbes in this case Show the microbiota that typically lives in the host's gastrointestinal tract, including the intake of microorganisms cultivated outside the body and taken as probiotic supplements. Both parties of microorganisms may be the substrate (prebiotic) that the synbiotic contains. getting ready. As the result of scientific analysis, it was declared that Synbiotics couldn't be just a product made by mixing a probiotic source with a prebiotic substrate. This recommendation is made. The health advantages of consuming synbiotics extend beyond intestinal health, as They may also have an impact on regions other than the gut. The way that synbiotics are currently defined outlines their advantages based on the outcomes of complementing synbiotics taken orally. investigated in human experiments. Studies on interventions have demonstrated that diet consumption contributed to the reduction of irritable bowel syndrome by incorporating synbiotics. diarrhea, inflammatory bowel disorders, metabolic syndrome, and skin issues like dermatitis atopica [22]. Supplementing with synbiotics may help manage the prevalence of non-alcoholic fatty liver disease (NAFLD) and possible problems with the nervous system [23]

PHARMACOLOGICAL INTERACTIONS AND IMPLICATIONS

DRUG METABOLISM BY MICROBIOTA

Throughout their lives, humans coexist with essential symbiotics, which influences the ability to detoxify xenobiotics (such as medications and food ingredients) by cometabolism between microbiota and host. Phase I and Phase II detoxification systems are part of the host. responses. Phase I metabolism, which includes hydroxylation, reduction, and oxidation, is primarily carried out by cytochrome P450 (CYP) enzymes in the stomach, liver, and other organs, which increase the polarity of foreign substances to help excrete them in urine. substances. The conjugation reaction, which includes glucuronidation, is the phase II metabolism. sulfonation as well. The indigenous metabolites and the exogenous chemicals are coupled by mechanisms for transferring host enzymes to enhance their excretion in the urine. Phase II enzymes comprise uridine 5′-diphospho-glucuronosyltransferase (UDP-), sulfotransferase (SULT), and UGT, NAT, and gluctathione S-transferase are examples of glucuronosyltransferases. In light of the given proof that gut microbiota's impact on pharmacokinetics, as it typically regulates the oral medication half-life or bioavailability through microbiota-host cometabolism by changing the ability of enzymes that break down drugs or the expression of genes related to drug metabolism within the tissues of the host. Additionally, each gut microbiota's unique makeup or activity is suitable for be impacted by external variables like food, antibiotics, or the state of health of due to the fact that many illnesses are linked to gut dysbiosis or vice versa [24]. The microbiota can biotransform xenobiotics, such as food polyphenols, medicinal components, and other substances, and it has a strong metabolizing activity [25].

TOXICITY

Numerous accounts have been written about the significance of bacteria in human health. But The degree to which exposure to EDCs results in dysbiosis and impacts human well-being. One area of public health that is pertinent is the impact of environmental contaminants. The microbiota in the gut is susceptible to The cause appears to be changes in its surroundings and the customs of an industrialized culture of its increasing degradation and dysbiosis. Over the past century, there have been numerous shifts: urbanization, globalization, and primary sector industrialization Every every one of them have significantly altered the population's way of life and caused the loss of of ancestor organisms that play vital roles in the human. Hormonal disturbances (EDs), pesticides, heavy metals, and other pollutants affect gut microbes. such as problems of energy metabolism. We are discussing obesity, diabetes, liver illness, infertility, asthma, issues with neurodevelopment, and neurode cancer and generative disorders [26]. It has been shown that exposure to environmental contaminants targets the resident's and host's gut flora, whose disruption may have systemic consequences, such as changes in how the CNS functions through the MGBA Air pollutants, organic solvents, and heavy metals are some of the most thoroughly researched categories of man- Man-made and natural poisons linked to mental health disorders and psychosocial operating [27].

CHALLENGES AND FUTURE DIRECTIONS

FUTURE ASPECTS

Future study will probably concentrate on identifying more individualized therapeutic approaches and dissecting the causal links within the gut-brain axis. Specifically, the complex nature of gut bacteria and metabolites must be taken into account in cellular and animal models used to investigate the influence of gut microbiome on neuropathologies through the gut-brain axis. This will be necessary to align the findings with data from biopsies and postmortem tissue samples as well as human clinical observations. This topic is anticipated to advance because to developments in high-throughput sequencing, bioinformatics, and integrative systems biology techniques, which will clarify the complex connections between microbial communities and the host. Additionally, there is a significant chance of creating novel therapeutic approaches based on microbiome manipulation. From prebiotics, probiotics, and synbiotics to more complex techniques like genetic and microbial engineering, the possible interventions cover a wide range. In order to confirm the efficacy of these treatments and understand the long-term effects of microbiom modification, longitudinal and interventional studies will be essential [28].

METHODOLOGICAL RECOMMENDATIONS

Because DNA sequencing techniques are widely used to describe the composition of the gut microbiota, a high Numerous associations between the microbiome and host visible characteristics, such as brain structure, function, and behavior of the host. However, the majority of these correlations are erratic and unpredictable. span observational research and don't have follow-up confirmation. The inconsistent results also complicate the process of setting up mechanistic validation studies that seek to prove causal relationships. Furthermore, mechanistic validation studies of correlational findings fall behind,

significantly impeding their translation to therapeutic applications and biological understanding. Various methods from microbiology can be blamed for the discrepancies in correlational results. Statistics, bioinformatics, epidemiology, genetics, and other disciplines Strengthening The Organization and Reporting of Microbiome Studies is a checklist that was modified and created by a multidisciplinary working group to help with the reproducibility and replicability of human microbiome research. This checklist offers instructions on how to succinctly and thoroughly present microbiome data, of which choosing possible confounders is a crucial step. Variables that affect both predictors and outcomes are known as confounders, and they must be properly identified before being included in multivariate statistical analysis. Limited understanding of these complex systems makes it difficult to find confounders of links between mental qualities and diverse and complex microbial ecosystems. Because of its complexity, analyzing relationships throughout the MGBA is a difficult task [29].

CONCLUSION

By impacting immunological regulation, neurotransmitter synthesis, and neuroendocrine signaling, the gut microbiota plays a critical role in preserving neurological health via the gut–brain axis. Dysbiosis, or disturbances in microbial equilibrium, is directly linked to mental and neurological conditions like anxiety, multiple sclerosis, Parkinson's disease, and Alzheimer's disease. Therapeutic strategies that restore gut homeostasis and enhance brain function include probiotics, psychobiotics, prebiotics, synbiotics, and fecal microbiota transplantation. Furthermore, drug metabolism, efficacy, and toxicity are all greatly impacted by the microbiota, which makes it a crucial component of individualized neuropharmacology. Even if the evidence is mounting, further investigation and established procedures are required to pinpoint the causes and guarantee trustworthy therapeutic uses. There may be new avenues for the prevention and treatment of neuropsychiatric and neurodegenerative diseases if the gut-brain axis is understood and altered.

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