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Neurodegeneration and Gut Microbiota Pathophysiological Links and Therapeutic Opportunities

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ABSTRACT
The vagus nerve, sympathetic and parasympathetic fibres, and the hypothalamic–pituitary–adrenal (HPA) axis are among the neural, endocrine, and immune pathways that mediate the intricate bidirectional communication network known as the microbiota gut brain axis (MGBA), which connects the central nervous system (CNS) and the gastrointestinal tract. Neurological diseases (NDs) like anxiety, depression, autism spectrum disorder, Parkinson's disease (PD), and Alzheimer's disease (AD) have been increasingly associated with dysbiosis, or changes in the makeup of the gut microbiota. Dysbiosis affects the start and course of disease by causing neuroinflammation, microglial activation, compromised blood–brain barrier integrity, and the generation of neurotoxic metabolites. In addition to producing neuroactive substances like short-chain fatty acids (SCFAs), which control behaviour, synaptic plasticity, and brain health, the gut microbiota also influences immunological function through chemokines and cytokines. Probiotics, prebiotics, symbiotic, dietary modification, and faecal microbiota transplantation are examples of emerging treatment approaches that target the MGBA and have the potential to improve cognitive performance, restore microbial balance, and lessen neuroinflammation in neurodegenerative diseases. This review uses the terms "gut–brain axis," "microbiome dysbiosis," "neurodegeneration," "Alzheimer's disease," and "Parkinson's disease" to synthesise information from PubMed, Scopus, Ovid Medline, Cochrane Review, and Google Scholar up until July 2025. The results emphasise the therapeutic potential of gut microbiota changes and emphasise their critical involvement in neurodevelopment, ageing, and neurodegeneration. Clinical translation is still lacking, though, and more thorough, long-term human research is needed to clarify causal links, improve intervention techniques, and use the MGBA to treat neurodegenerative illnesses.

Keywords: *Microbiota–Gut–Brain Axis (MGBA), Neurodegenerative Diseases, Gut Microbiota Dysbiosis, Neuroinflammation, Parkinson's Disease, Alzheimer's Disease, Probiotics and Prebiotics*

1 INTRODUCTION

The central nervous system (CNS), enteric nervous system (ENS), and gut microbiota are all involved in the intricate communication network known as the microbiota-gut-brain axis (MGBA). Neurological, endocrine, and immunological mechanisms mediate this two-way connection. As the primary neurological channel connecting the stomach and brain, the vagus nerve is essential, and the hypothalamic-pituitary-adrenal (HPA) axis uses endocrine signals to control stress reactions [1-3]. Cytokines and chemokines are immune system components that respond to microbial signals to promote communication. Short-chain fatty acids (SCFAs), neurotransmitters, and neuromodulators are among the metabolites produced by the gut microbiota that affect behaviour and brain function. Numerous neurological conditions, such as anxiety, depression, Parkinson's disease, and Alzheimer's disease, have been connected to dysbiosis, or an imbalance in the gut microbiota. According to recent research, gut microbiota may influence neuroinflammation, which in turn may impact microglial activation and central nervous system health. Probiotics and prebiotics together, known as synbiotics, have shown potential in balancing the gut flora and reducing neurological symptoms [4]. Neurodegenerative illnesses may be managed using therapeutic approaches that target the MGBA, such as dietary changes and microbiota

modification. To completely comprehend the processes and therapeutic uses of the MGBA in brain health and illness, further thorough study is necessary. Research on the factors that promote communication between the central nervous system (CNS) and the enteric nervous system (ENS) has demonstrated that this two-way system functions between the CNS and the gastrointestinal tract (GIT) [5]. Through the vagus nerve and spinal afferent fibres connected to the extrinsic nerves related to the gut, the sympathetic and parasympathetic fibres establish a connection between the gastrointestinal tract (GIT) and the central nervous system (CNS). The physiological alimentary functioning associated with glucogenesis is regulated by the Hypothalamic Pituitary Adrenal (HPA)-axis [6]. The apparent association between intestinal health and neurodevelopmental disorders (NDs) is supported by data from several studies. The crucial role of the MGBA in a variety of metabolic diseases, mental illnesses, and neuronal degeneration syndromes, including anxiety, depression, autism, Parkinson's disease, and Alzheimer's disease, has unquestionably been clarified by experimental data. Glioblastoma (GBM) has been linked in recent research to neurological disorders through the microbiota-gut-brain axis (MGBA). Numerous studies have suggested that a GBM imbalance may directly or indirectly impact diseases of the central nervous system (CNS), perhaps affecting microglial-induced neuroinflammation and neurodegeneration [7-9]. There are several routes that connect the brain and GBM in both directions. The composition of the gut microbiota is unique and harmonious, and it functions in a dynamic manner. Numerous studies have clearly shown that MGBA may be the cause of neurological disorders, with even little adjustments to these parameters potentially leading to their development [10]. Notably, by promoting the synthesis of chemokines and cytokines, changes in the microbiota have a major impact on how the immune system functions. Additionally, the gathered study data on the MGBA has demonstrated that many metabolic pathways support the connection between intestinal cells, the ENS, and the central nervous system. Consequently, a number of biochemicals and hormones play a critical role in controlling the relationship between the brain and the gastrointestinal tract (GIT), which has prompted a great deal of study on synbiotics. The use of synbiotics to treat neurodegenerative diseases has been spurred by these arguments. By lowering inflammation, the microbiome has a potent effect that improves cognitive function in people with neurodegenerative illnesses. Probiotics may therefore enhance these people's cognitive capacities. In conclusion, via altering immune system reactions, the gut microbiota may play a major part in the emergence of a number of brain-related illnesses [11-14].

2 GUT MICROBIOME

Initiatives like the Human Microbiome Project and the development of methods like FMT have sparked renewed interest in the topic of gut microbiome in recent years. These investigations have significantly increased our understanding of the ways in which unrelated endogenous processes of illnesses or phenotypes interact with systems such as the gut-brain axis [15]. The body's normal bacterial ecology is complicated, and research has shown that a variety of factors, such as environment, age, and food, may influence this microbial population. Complex carbs and fibres are metabolised by the intestinal microbiota [16-18]. This procedure indirectly suggests involvement from a metabolic pathway since it directly affects the host's energy balance and metabolic health. The immune system's maturation and development are also influenced by the gut flora. It is a crucial defence mechanism against external microorganisms because it increases resistance to infections and modifies systemic immunity. Additionally, the production of conjugate bile acids and vital vitamins by the gut microorganisms' aids in the breakdown of fat. The fermentation of food fibres into short-chain fatty acids (SCFAs), a form of host signalling molecule and energy source, is an example of the metabolic function of the gut microbiota. Throughout the human lifespan, there are significant changes that are frequently attributed to the body's evolving demands, such as maintaining physical balance and preventing illness [19-21]. One's gut microbiota makeup is readily expressed, with variations in lifestyle, environmental exposures, and, most importantly, food habits resulting from regional and cultural disparities. The usage of medications, stress levels, sleep habits, and physical activity are only a few of the numerous other variables that influence the gut microbiota. These elements alter the microbial population, which in turn affects its makeup and capabilities [22]. It is becoming more well acknowledged that the gut-brain axis serves as a conduit for communication between the central nervous system (CNS) and the digestive system. This connection is associated with complex neurological connections. Given this, the vagus nerve plays a vital role in bridging the gap between the gut and the brain, making it vital for comprehending the neurological underpinnings of mood disorders and opening the door to novel treatments [23]. Hormonal signalling, a crucial pathway

in gut-brain communication, is how the gut bacteria work. For instance, changes in tryptophan metabolism linked to the gut microbiota impair serotonin activity in the brain and are a contributing factor to mood disorders that are accompanied by gastrointestinal dysfunction. Additionally, this type of modulation might increase one's capacity for emotional and cognitive processing. SCFAs and other signalling molecules are essential for brain-gut communication [24-27]. These compounds' artificial production might be a novel therapeutic target for neurodegenerative diseases. When gut bacteria digest dietary fibre, SCFAs are created. They have a major effect on general health and cognitive performance. By encouraging neurogenesis, SCFAs maintain the integrity of the blood-brain barrier (BBB) and have an impact on neuroinflammation through receptor-mediated pathways. These metabolites are also important regulators of cell proliferation, BBB function, and neuroinflammation. They are intricately connected, influence cognitive function, and guard against a number of brain disorders [28-32].

3 NEURODEGENERATIVE DISORDERS

Progressive loss of neurones or other neurological cells inside the central nervous system is the primary cause of neurodegenerative disorders. These neurodegenerative illnesses can have a wide range of pathologies and symptoms, and the majority of them have different origins and processes. Neurones are particularly vulnerable to harm because of these cell losses [33-38]. The BBB's selective permeability limits the admission of outside chemicals, making treatment planning more difficult. The majority of therapies require a polar component to help with bloodstream transport, but they must not be too polar in order to pass through the BBB's lipid bilayer. Furthermore, the BBB is extremely susceptible to damage and functional deterioration since its regenerating capability is far slower than that of any other bodily systems [39-42]. Progressive cognitive decline and everyday functional impairment are hallmarks of dementia, especially Alzheimer's disease. Although it is one of the leading age-related causes of mortality in the US, little is known about its possible causes and available therapies. According to current theories, extracellular amyloid plaques and intraneuronal neurofibrillary tangles composed of tau proteins and amyloid-β peptides are the main components of Alzheimer's disease pathogenesis. In addition to interfering with synaptic processes essential for memory and cognition, these toxic deposits cause synapse loss by impairing axon and dendritic maintenance or neurone death [43-44]. All of these symptoms are difficult to identify in their early stages and frequently coexist with typical aging-related characteristics. Another example is Parkinson's disease, which is typified by motor symptoms including stiffness, sluggish movement, and balance issues that make movement increasingly difficult as neurodegeneration progresses. The illness is hypothesised to be caused by malfunctioning of dopaminergic neurones in different areas of the brain, which makes diagnosis challenging due to the vast variety of symptoms. One of the most prevalent pathologies in Parkinson's disease is the degradation of neurones in the substantia nigra pars compacta. Movement disorders result from the disruption of dopamine signalling in the striatum, which in turn affects motor cortex and basal ganglia circuits. These disruptions have broad societal and economic repercussions and can have a major impact on quality of life. Research on treating Parkinson's disease and other neurological diseases is a top priority (see **Figure 1**) [45-46].

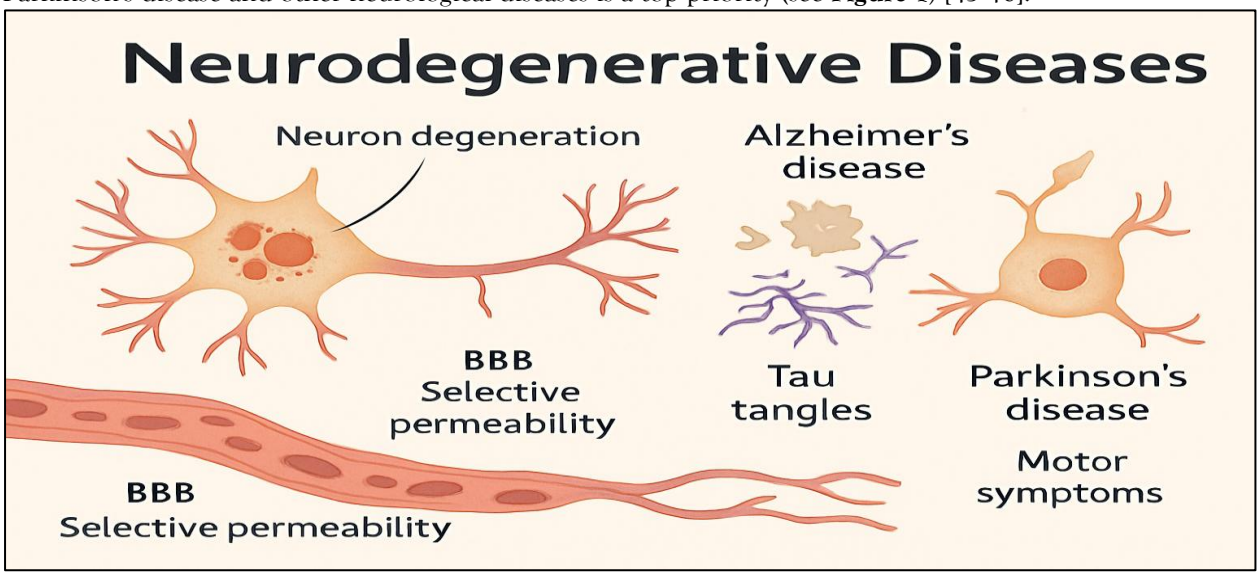

Figure 1. Neuron degeneration, BBB challenges, and key features of neurodegeneration

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4 MOLECULAR PATHWAYS OF COMMUNICATION

The gut microbiota is influenced by the central nervous system (CNS) via complex molecular pathways that include immunological, endocrine, and neurological signals. The vagus nerve, which acts as a direct conduit between the brain and the gut, is the primary means of neural communication. Signals that control gut motility, secretion, and permeability are sent via the vagus nerve. For example, the brain may change gut function by sending signals through the vagus nerve when it senses stress [47]. Another important channel is the hypothalamic-pituitary-adrenal (HPA) axis. The hypothalamus releases corticotropin-releasing hormone (CRH) in reaction to stress, which causes the pituitary gland to release adrenocorticotropic hormone (ACTH). The adrenal glands are subsequently stimulated by this hormone to generate corticosteroids, including cortisol. By altering the gut environment and immunological responses, elevated cortisol levels can change the makeup of the gut microbiota. Another important player in this communication is the immune system. CNS activity can affect cytokines, which are signalling molecules secreted by immune cells [48]. For instance, the CNS may control the synthesis of pro-inflammatory cytokines such as interleukin-6 (IL-6) and tumour necrosis factor-alpha (TNF-α) during an immunological response, which can impact the function and balance of the gut microbiota. The complicated connection between neurones, glial cells, and epithelial cells is known as the neuronal-glial-epithelial axis, and it is essential for preserving homeostasis in the central nervous system (CNS) [49]. Rapid communication across the central nervous system is made possible by the electrical impulses that neurones convey. Glial cells, which include oligodendrocytes, microglia, and astrocytes, maintain the blood-brain barrier, control synaptic activity, and support neuronal function. In order to protect neural tissue from toxins and infections, epithelial cells—especially those that make up the blood-brain barrier—control the flow of chemicals between the circulation and the brain. Signalling substances like as growth factors, cytokines, and neurotransmitters mediate the interaction between various cell types. Neurodegeneration, neuroinflammation, and conditions like multiple sclerosis and Alzheimer's disease can result from disruptions in this transmission. Maintaining brain health and creating specific treatments for CNS disorders need an understanding of these intricate relationships [50]. On the other hand, there are several ways in which the gut microbiota might affect the central nervous system. Neurotransmitters that can affect brain function, including serotonin, dopamine, and gamma-aminobutyric acid (GABA), are produced by gut microbes [51]. Furthermore, microbial fermentation of dietary fibres produces short-chain fatty acids (SCFAs), which have neuroactive properties and can pass across the blood-brain barrier. These SCFAs have the ability to alter neuronal activity and inflammation in the brain. Neurological disorders are associated with dysbiosis, or an imbalance in the gut microbiota [52]. For instance, alterations in gut microbiota in Parkinson's disease may result in increased intestinal permeability, which would enable inflammatory chemicals to enter the circulation and travel to the brain, so intensifying neuroinflammation and neurodegeneration. The restoration of microbial balance and reduction of inflammation are the goals of interventions like probiotics, prebiotics, and dietary changes, underscoring the significance of preserving a healthy gut microbiota for brain health. The intricate and vital connection between gut health and brain function is highlighted by this reciprocal exchange of information between the gut bacteria and the central nervous system (see **Figure 2**) [53-56].

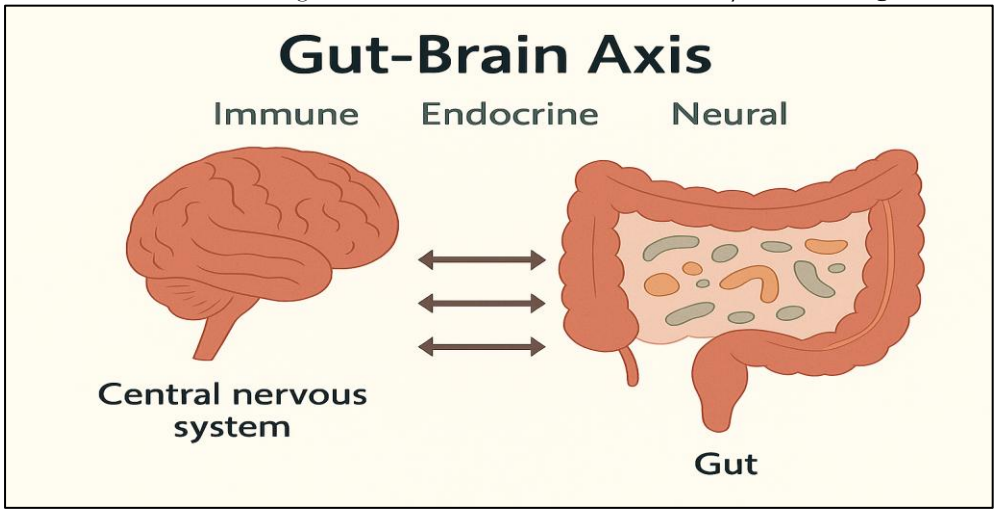

Figure 2. Bidirectional gut-brain communication via immune, endocrine, and neural pathways

5 DYSFUNCTION OF THE GUT-BRAIN AXIS

Through a number of pathways, dysbiosis—an imbalance in the gut microbiota—can have a major effect on brain health. A key component of this link is the gut-brain axis, a network of bidirectional communication between the central nervous system and the gastrointestinal tract. This axis can be upset by dysbiosis, which can result in neurological conditions. The synthesis and control of neurotransmitters is one important route. Neurotransmitters necessary for brain function, such as serotonin and gamma-aminobutyric acid (GABA), are produced by the gut bacteria. These neurotransmitter levels can be changed by dysbiosis, which can exacerbate mood disorders including anxiety and sadness. Furthermore, dysbiosis may weaken the gut barrier, leading to increased intestinal permeability- a condition known as "leaky gut." This promotes neuroinflammation by enabling pro-inflammatory chemicals and microbial metabolites to enter the circulation and pass across the blood-brain barrier [57-61]. Neurodegenerative illnesses including Parkinson's and Alzheimer's are known to be influenced by chronic neuroinflammation. Additionally, because dysbiosis can result in an overreactive immunological response, the immune system is crucial. Pro-inflammatory cytokines may be released as a result of this increased immunological activity, which may affect behaviour and brain function and perhaps cause diseases like multiple sclerosis and autism spectrum disorder. All things considered, the intricate relationship between gut microbiota and brain health emphasises how crucial it is to keep the gut microbiome balanced in order to prevent and treat neurological illnesses. A "broken stomach" is a phrase used to describe severe disturbances in gastrointestinal function that can have a dramatic effect on the central nervous system (CNS) [62]. Often referred to as the "second brain," the stomach is essential to mental and neurological function as well as general wellness. A "broken stomach" usually refers to illnesses that affect the gut's capacity to function normally, such as dysbiosis, leaky gut syndrome, or persistent gastrointestinal disorders. The varied collection of bacteria that live in the digestive system, known as the gut microbiota, is disturbed by several disorders. Because of the two-way connection along the gut-brain axis, studies have demonstrated that a healthy gut microbiota is crucial for preserving central nervous system function. This axis includes immunological, hormonal, and neurological processes [63-67]. Dysbiosis, for example, can result in the overproduction of neurotoxins and pro-inflammatory cytokines, which can cross the blood-brain barrier and cause neuroinflammation and neurodegenerative illnesses like Parkinson's and Alzheimer's. Furthermore, a large amount of neurotransmitters, including serotonin, are created in the gut and can be impacted by gastrointestinal disorders. Mood problems like anxiety and sadness may result from this. Thus, preserving gut health is essential for safeguarding central nervous system activities and averting neurological illnesses [68-72]. Disturbances in gut-brain communication have been increasingly associated with dysbiosis, an imbalance in the gut microbiota that contributes to a number of neurological and psychiatric conditions. There seems to be some evidence between Parkinson's disease with dysbiosis. According to studies, gastrointestinal problems and motor symptoms are frequently associated with changed gut microbiota compositions in Parkinson's disease patients. The part dysbiosis plays in autism spectrum disorders (ASD) is another example. According to research, children with ASD frequently have different gut microbiota profiles than children who are neurotypical [73-77]. This might have an impact on behaviour and neurodevelopment by producing neuroactive metabolites. Major depressive disorder (MDD) has also been linked to dysbiosis. There may be a link between gut microbiota and depression because a study showed that transferring microbiota from MDD patients into germ-free animals caused depressive-like behaviours. These instances demonstrate the vital role that gut microbiota plays in brain function and the possibility of using dysbiosis as a target for therapeutic treatments in neurological and psychiatric disorders [78].

6 ALTERED GUT MICROBIAL PROFILE & NEURODEGENERATIVE DISORDERS

6.1 ROLE OF MICROBIOME AND ALZHEIMER'S DISEASE

Cognitive decline, memory loss, difficulties with verbal and motor activities, and dementia are all symptoms of Alzheimer's disease, which is typified by the buildup of Aβ proteins as senile plaques in the extracellular spaces of the brain and hyperphosphorylated β-tau proteins forming neurofibrillary tangles inside neurons [79]. According to recent studies, several bacteria from the gut microbiota's genera-Streptococcus, Staphylococcus, Salmonella, Mycobacteria, Klebsiella, Citrobacter, and Bacillus may be able to create Aβ proteins. Neuroinflammation may be exacerbated by these proteins' subsequent misfolding and accumulation into Aβ aggregates in the central nervous system. Microbial diversity decreased in a recent study that examined the gut microbiomes of 25 people with Alzheimer's disease.

Bacteroidetes increased whereas Firmicutes and Actinobacteria were significantly reduced. Lachnospiraceae, Ruminococcaceae, and Bacteroidaceae were the most common families among Alzheimer's disease patients. There is a clear correlation between the pathogenic alterations and these microbial shifts. In older individuals with Alzheimer's disease, Cattaneo et al. found that the levels of inflammatory species like Escherichia increased while those of anti-inflammatory bacteria like Eubacterium rectale decreased [80-83].

6.2 ROLE OF MICROBIOME AND PARKINSON'S DISEASE

Parkinson's disease is characterised by the accumulation of Lewy bodies in midbrain dopaminergic neurones, which are mostly made up of ubiquitin and alpha-synuclein. According to animal models, gut dysbiosis causes aberrant α-synuclein deposition to start in the enteric nervous system or olfactory bulbs. The substantia nigra pars compacta in the lower brainstem, the dorsal motor nucleus of the vagus nerve, and other locations in the central nervous system are reached by this aggregation via synaptic transmission. Parkinson's disease patients had lower numbers of bacteria that produce short-chain fatty acids and higher levels of pro-inflammatory cytokines, CD3+ T lymphocytes, and Toll-like receptor 4 in their colons [84-86].

6.3 ROLE OF MICROBIOME AND MULTIPLE SCLEROSIS

Axonal injury and progressive demyelination result in neurodegeneration through autoimmune processes in multiple sclerosis, a neurodegenerative disease. Clinical manifestations include ataxia, muscular spasms, paralysis, tiredness, bladder and bowel dysfunction, and deficits in sensory, visual, autonomic, and cognitive functions. According to recent data, gut dysbiosis plays a significant role in the neuropathophysiology of multiple sclerosis. In particular, pro-inflammatory bacteria, which control immune cells such as regulatory T cells, interleukin 10-secreting CD4+ T cells, regulatory B cells, tolerogenic dendritic cells, and suppressive macrophages, are more prevalent in the relapsing/remitting form of multiple sclerosis than anti-inflammatory bacteria. According to clinical research, people with multiple sclerosis have a different gut microbial makeup than people in good health. When compared to healthy controls, patients with relapsing-remitting multiple sclerosis have lower levels of the bacteria Parabacteroides distasonis and Faecal [87-90].

6.4 ROLE OF MICROBIOME AND AMYOTROPHIC LATERAL SCLEROSIS (LOU GEHRIG'S DISEASE)

A neurological condition called amyotrophic lateral sclerosis, formerly known as Lou Gehrig's disease, is characterised by the progressive death of motor neurones in the brain and spinal cord, which leads to paralysis and muscular weakness. Neurones and glia in amyotrophic lateral sclerosis contain misfolded proteins, especially ubiquitinated cytoplasmic inclusions [91-94]. The pathophysiology is linked to glutamate excitotoxicity, mitochondrial dysfunction, redox imbalance, alterations in RNA metabolism, and dysregulated autophagy. Amyotrophic lateral sclerosis and intestinal dysbiosis are strongly linked, according to recent research. In particular, changes in the makeup of the gut microbiota, poor metabolism, a modified immunological response, and the generation of neurotoxins by Clostridium species that can cause brain damage are linked to the pathogenesis of amyotrophic lateral sclerosis [95-97]. According to metagenomics research, gut dysbiosis is indicated by a decreased Firmicutes/Bacteroides (F/B) ratio in individuals with amyotrophic lateral sclerosis. Furthermore, in comparison to healthy controls, the genus Dorea is substantially more abundant, whereas the genera Oscillibacter, Anaerostipes, and Lachnospiraceae are significantly less abundant [98].

6.5 ROLE OF MICROBIOME AND HUNTINGTON'S DISEASE

Involuntary motions of the head, neck, limbs, and face are hallmarks of Huntington's disease, a neurological movement condition brought on by genetic anticipation of the cytosine-adenine-guanine repeat in the Huntington gene. Clinical manifestations include skeletal muscular atrophy, gastrointestinal disorders, unintentional weight loss, and cognitive, mental, and motor deficits. The degeneration of neurones in the cerebral cortex, putamen, and caudate is the initial pathogenic characteristic [99]. Transgenic rats and Huntington's disease patients have been shown to have altered gut metabolites, which may indicate that alterations in the gut microbiota occur prior to the development of the illness. Unintentional weight loss was a result of gastrointestinal malfunction in rat models of

Huntington's illness [100]. At 12 weeks of age, a recent study using the R6/1 transgenic mouse model of Huntington's disease revealed a changed composition of gut microbes, with higher levels of Bacteroidetes and lower levels of Firmicutes. Despite increased food intake and motor impairments, this gut dysbiosis is linked to poor weight growth [101].

7 BIOMARKERS AND DIAGNOSTIC ADVANCES

Microbiome-based biomarkers provide personalised medical strategies, illness progression tracking, and early identification. Microbial signatures imposed by the aberrant nature of neurodegenerative disorders may be indicative of altered gut microbiome instability, which most likely occurs during the onset or course of these diseases [102]. They might be used to monitor patients with neurodegenerative diseases as powerful, noninvasive diagnostic tools. However, the gut microbiota plays a crucial role in regulating neurodegenerative illnesses, and the central nervous system (CNS) affects the microbiome's state and vice versa. Neurodegenerative diseases can interfere with this kind of two-way communication between the stomach and the brain. Similar mechanisms, such as autonomic nervous system failure, which is common in Parkinson's disease, might account for disruptions in gut motility and, therefore, in the gut environment that alter the makeup of the microbiome [103-106]. Increased gut permeability and altered microbial diversity due to neuroinflammatory and stress hormone dysregulation seem to be hallmarks of Alzheimer's disease. How changes in the brain affect gut microbiota may be explained by this intricate relationship between the gut microbiome and the central nervous system. According to more recent research, the microbiome does in fact include recognisable indicators for neurodegenerative illnesses. For instance, alterations in the gut microbiota are linked to clinical characteristics and might indicate the course of a disease as well as how well a therapy is working. Other genera have also been linked to an increased incidence of amyotrophic lateral sclerosis (ALS). However, Alzheimer's and Parkinson's illnesses are inversely correlated with glutamine levels and other metabolites produced in the gut microbiota [107-109]. These results provide credence to the idea that neurodegenerative disease start or progression might be predicted using identifiable early microbiome alterations. We can now study the intricate microbial communities in the human body thanks to recent developments in microbiome profiling tools. High-throughput investigations of microbial communities at extraordinary depths have been made possible by next-generation sequencing technology, which has transformed microbiome research through metagenomics and meta transcriptomics. The study of metabolites in biological systems, or metabolomics, has shed light on the ways in which microbial metabolites affect host physiology and disease conditions. The combination of technology, namely bioinformatics and computational tools, has directly improved the sensitivity and accuracy of microbiome analysis and its potential as a diagnostic tool. These advancements have given us a better understanding of how the microbiome affects health and illness and have created new opportunities for the development of diagnostics and treatments based on the microbiome [110-112].

One of the biggest obstacles to creating microbiome biomarkers for therapeutic use is standardising and verifying microbiome analysis. More uniform high-throughput analysers and a framework that maintains information security while integrating with other laboratory procedures are necessary due to the complexity of the microbiome itself. Additionally, a method such as the enzyme-linked immunosorbent test (ELISA) has a systemically and randomly variable quality, which increases mistakes and necessitates stricter control over assay performance for clinical usage. Although the development and validation of host serologic microbial biomarkers in illnesses, especially inflammatory bowel disorders, have advanced to a unique position in diagnosis and prognosis, standardisation and interpretation remain challenges [116-120]. Interpreting the rich data provided by microbiome research presents another difficulty. The diversity of microbial communities and their interactions with the host makes it tough to glean therapeutically useful insights from microbiome data. Due to a number of stages in the production of the next generation library sequencing, amplicon-based marker gene techniques used in microbiome studies are prone to bias or inaccuracy. The fact that a strong and consistent protocol for sample preparation should immunise the microbiome markers from research into clinical application is also significant [121].

8 FUTURE PERSPECTIVES

In order to evaluate the work in neurodegeneration, the MGBA alterations will soon be used in experimental phases. When considering MGBA as a restorative goal against neurodegenerative

infections, many investigations highlight the potential drawbacks of antibacterial treatment and the significance of the microbiome. These systems might not be corrective, so we want to focus on preventive strategies [122-126]. For instance, beneficial supplements that maintain a healthy stomach environment and provide metabolites that reduce irritation and hepatic encephalopathy that reduces stoppage and improves the health of the stomach microbiome. However, a number of findings have revealed that the pathophysiology of several brain disorders (AD, PD, HD, and ALS) is significantly influenced by the gut microbiota. However, further research is needed for more accurate confirmation [127-130]. Along with historical data from the individual individuals, a number of study models demonstrated that the gut microbiota is linked to the onset development of central nervous system disorders. Maintaining a healthy microbiota or altering the whole stomach microbial population through the use of FMT (Faecal Microbiota Transplantation) is made easier by the key microbiota-involved therapy method, which involves a necessary measure of a few bacterial strains or chemicals. These therapies do have several limitations, nevertheless, including a lack of feasible plans, fluctuating relevant research, portion normalisation, and a limited number of human data. Furthermore, several studies have suggested that addressing the stomach microbiota may be a useful strategy for slowing the progression of neurodegenerative diseases. For the time being, we can think of stomach microbiota as a potential strategy for not-too-distant future treatments [131-140]. There is a lot of scientific interest in the therapeutic potential of treatments that target the microbiota-gut-brain axis (MGBA) in the treatment of neurological illnesses. According to this expanding body of research, altering the gut microbiome may have an impact on mental health and brain function. It is impossible to overestimate the need of conducting extensive clinical studies to support these assertions, even in light of encouraging first results [141-143]. These studies are necessary to determine which patient populations will benefit most from these therapies, implement standardised methods, and validate the safety and effectiveness of these interventions. Because gut microbiota composition and neurological conditions are complex and multifactorial, large-scale clinical trials offer the statistical power required to identify significant effects and reduce the risk of type I and type II errors. These errors are especially relevant in MGBA research [144-146]. The generalisability of findings depends on the consistency of outcomes across many populations and environments, which these experiments may assist determine. For example, a number of studies have suggested that probiotics and prebiotics may help with illnesses including autism spectrum disorders (ASDs), anxiety, and depression. Small sample numbers, inconsistent outcome measures, and research design variability, however, restrict how definitive these results. The meticulous evaluation of patient selection criteria is a crucial component in carrying out extensive clinical studies. This entails establishing inclusion and exclusion criteria that represent the variability of the clinical condition under research while guaranteeing a homogenous study group. For example, it's critical to stratify patients in probiotic studies for depression according to the severity of their disease, length of sickness, and any ongoing medications [147-160]. This classification guarantees that findings are not distorted by confounding variables and aids in identifying which patient subgroups are most likely to gain from the intervention [161-165]. Furthermore, it is essential to create standardized intervention methods. The probiotic strains or symbiotic (prebiotics and probiotics) that are used, their doses, and the length of therapy should all be specified in these protocols. For data from many research and meta-analyses to be adequately compared, these factors must be consistent. The way these treatments are developed and delivered should also be taken into account, since these factors can have a big influence on how effective [166-168]. For instance, because to differences in their rates of survival through the gastrointestinal track, probiotics in encapsulation may have different effects than those in yoghurt or other dietary matrices. Clinical trials pertaining to MGBA should use multifaceted, comprehensive outcome measures that record both objective and subjective changes in neurological and mental symptoms. Examples of such metrics include neuroimaging investigations, gut microbiota composition study, standardized clinical evaluations, and biomarkers of inflammation and neuroplasticity. These outcome measurements can aid in the identification of biomarkers predictive of response to treatments and offer insights into the processes behind the observed effects. Furthermore, using patient-reported results can provide important insights into the perceived advantages and enhancements in quality of life. In summary, whereas MGBA-targeted therapies show promise, further clinical studies are clearly needed to verify these strategies. Standardized intervention procedures, thorough outcome measurements, and patient selection criteria should all be carefully taken into account while designing these studies. In addition to improving the validity and repeatability of study results, addressing these important factors will direct future investigations and

therapeutic uses in this rapidly developing area. The alteration of the gut-mind hub and its influence on neurodegeneration are now the subject of a few clinical investigations [169-172]. In order to better understand MGBA and neuroinflammation, the experts are looking at a few animal models of mind-related disorders.

9 CONCLUSION

A crucial connection between the gut microbiome and the central nervous system, the microbiota gut-brain axis (MGBA) affects neurological health by modulating the immune system, altering neuronal signaling, and producing metabolites. Even while our understanding of its processes has advanced significantly, there are still important gaps in our knowledge of its specific function in the pathophysiology and pathogenesis of neurological illnesses connected to the immune system and neurodegeneration. The therapeutic potential of the MGBA is demonstrated by data from infection investigations, probiotic therapies, antibiotic treatments, Faecal microbiota transplantation, and germ-free models. Preclinical research yields the majority of discoveries, nevertheless, and extra care must be used when extrapolating these results to humans. There is an urgent need for extensive, carefully monitored clinical studies to verify effectiveness, prove causation, and pinpoint disease-specific reactions. Prebiotics, probiotics, symbiotics, dietary changes, and microbiome-based therapies that target the MGBA present a potential approach to the management and prevention of diseases including Parkinson's and Alzheimer's. Additionally, developments in microbiome research might make it easier to create noninvasive biomarkers derived from the microbiome for early diagnosis and prognosis. In order to fully realize the promise of the MGBA and convert mechanistic discoveries into safe, efficient, and customised therapeutic approaches for neurodegenerative diseases, this review emphasizes the necessity of multidisciplinary cooperation in medicinal chemistry, neurology, and drug development.

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