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IMPACT OF GUT MICROBIOTA ON THE GUT-BRAIN AXIS IN NEUROLOGICAL DISORDERS AND THE ROLE OF ENDOCRINE AND METABOLIC MARKERS

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Abstract
This review highlights the critical importance of gut microbiota in modulating the gut-brain axis and its consequences for neurological disorders, including autism, Alzheimer's disease, and Parkinson's disease. New evidence is just beginning to suggest that gut microbiota may affect brain functioning through the intricate pathways of the immune, endocrine, and metabolic systems. This review discusses how alterations in gut microbiota composition may lead to aberrant endocrine responses and changes in metabolic markers contributing to neuroinflammation, neurodegeneration, and behavioural symptoms related to these diseases. This study will look at the potential targeting of the gut microbiome as a therapeutic strategy for neurological disorders through bidirectional interactions between the gut and the central nervous system. These findings have relevance toward understanding the gut-brain axis in the search for new biomarkers and development of personalised interventions in better promotion of neurological health.

Keywords Gut microbiota, gut-brain axis, neurological disorders, autism, Alzheimer's disease, Parkinson's disease, endocrine markers, metabolic markers, neuroinflammation, neurodegeneration, and microbiome therapy.

INTRODUCTION

The human gut hosts trillions of microorganisms, known collectively as the gut microbiota, which contribute substantially to maintaining health, especially through the gut-brain axis—a complex communication network linking the gut microbiota with the CNS. This reciprocal pathway is immune, endocrine, and metabolically intertwined, wherein the gut bacteria can modulate mood, cognition, and neurodevelopment. Dysbiosis, or microbial imbalance, has increasingly been associated with neurological diseases such as autism, Alzheimer's, and Parkinson's, in which dysregulation can lead to disease progression.

Certain bacteria produce SCFAs in autism, which have far-reaching implications for social behaviors and stress responses. Alzheimer's disease patients very frequently show decreased SCFAs and increased pro-inflammatory bacteria; hence, dysbiosis has been associated with neuroinflammation and cognitive decline. In Parkinson's disease, gastrointestinal symptoms precede motor issues, with microbial changes affecting dopamine and worsening motor and cognitive symptoms.

The gut microbiota acts on endocrine pathways by controlling cortisol and serotonin levels, which are known to affect mood and the stress response. Metabolically, SCFAs and bile acids derived from microbial activity support neuroprotective functions, and their decline in dysbiosis may

increase neuroinflammation.

These findings are the basis of gut-targeted therapies, such as probiotics, prebiotics, and dietary interventions, to re-establish the microbial balance and improve neurological health. Knowledge of the gut-brain axis might lead to personalized interventions for neurological disorders, hence an improved avenue toward better outcomes in mental and cognitive health.

Literature Review

Recent research has demonstrated how gut bacteria produce key neurotransmitters, including serotonin and gamma-aminobutyric acid (GABA), and short-chain fatty acids (SCFAs), all of which have important functions in brain function. The work of Cryan et al. (2019) in "The Microbiota-Gut-Brain Axis: From Composition to Function" underlines gut microbiota's ability to regulate serotonin production—that approximately 90% of serotonin is synthesized in the gut. Sampson et al. (2016), "Gut Microbiota Regulate Motor Deficits and Neuroinflammation in a Model of Parkinson's Disease", proved that SCFAs, such as butyrate and propionate, produced by gut bacteria, are capable of influencing neuroinflammation by modulating microglial cells in the brain. Mayer et al. (2020) also contribute to this field in "Gut/Brain Axis and the Microbiota: Mechanisms and Clinical Implications," outlining how the feedback loop between the gut and brain works: microbial metabolites communicate with the CNS via the vagus nerve and blood-brain barrier permeability.

In patients with neurological diseases, some studies, for example, appear to reveal extensive significant alterations in microbial composition. Hsiao et al. (2013), in "Microbiota Modulate Behavioral and Physiological Abnormalities Associated with Neurodevelopmental Disorders," demonstrated that patients with autism spectrum disorder (ASD) exhibit different gut microbiota with increased Clostridia and decreased Bifidobacterium, which significantly modulate SCFA levels and associated behaviors. Similarly, Vogt et al. (2017) investigated Alzheimer's disease in "Gut Microbiome Alterations in Alzheimer's Disease," seeing an overrepresentation of pro-inflammatory bacteria—for example, Escherichia—and reduced anti-inflammatory bacteria—for example, Lactobacillus—concluding with an indication of dysbiosis as a correlate of cognitive decline. Keshavarzian et al. (2015), "Colonic Bacteria, Inflammation, and Motor Function in Parkinson's Disease," demonstrated reduced Prevotella in Parkinson's patients, which might have implications affecting dopamine pathways and therefore contributing to motor and non-motor symptoms.

Gut microbiota can even affect endocrine function by regulating the hormones cortisol, serotonin, and dopamine—gonads, thyroids, adrenals—that are critical for mental health. In "The Microbiome-Gut-Brain Axis During Early Life Regulates the Hippocampal Serotonergic System in a Sex-Dependent Manner," Clarke et al. (2013) showed that early-life microbiota could shape serotonin levels, impacting neurodevelopment and behaviour. Needham et al. (2020), in "Gut Microbiota and Cortisol: The Impact of Microbiome on Stress Response in Neurological Disorders," emphasise that dysbiosis may cause an overproduction of cortisol, which can, in turn, exacerbate symptoms of anxiety and depression commonly observed in neurodegenerative diseases. Moreover, Pellegrini et al. (2018), in "Short-Chain Fatty Acids and Bile Acids in the Regulation of Gut-Brain Communication," give a view of how SCFAs and bile acids produced by gut bacteria may influence neuroprotective functions. Fluctuations in these markers have been observed to either increase or decrease the presentation of symptoms, with deficiencies in SCFAs widely associated with neuroinflammation. At the same time, changes in bile acids have been linked to cognitive decline.

METHODOLOGY

A literature review of the role of the gut-brain axis in neurological disorders published between 2010 and 2024 is provided here. According to various studies from PubMed, Scopus, and Google Scholar, gut microbiota contains trillions of microorganisms, which have been implicated in

producing neurotransmitters such as serotonin, gamma-aminobutyric acid (GABA), and short-chain fatty acids (SCFAs), which influence brain function. Dysbiosis, or the alteration in gut microbiota, has been associated with autism spectrum disorder, Alzheimer's, and Parkinson's disease, where evidence suggests specific alterations in the microbiota can modulate the symptoms and course of the disease.

In autism, there is a reduced amount of Bifidobacterium and increased Clostridia, which might contribute to behavioural symptoms. Alzheimer's patients often show an increase in pro-inflammatory bacteria, such as Escherichia, which is associated with neuroinflammation and cognitive decline. Parkinson's research has emphasised a decrease in SCFA-producing bacteria, which may exacerbate dopamine deficits and motor symptoms.

Endocrine markers, such as cortisol and serotonin, and metabolic markers, such as SCFAs and bile acids, all seem major players in gut-brain communication. Evidence has shown that gut microbiota may influence responses to stress, mood, and neuroinflammatory processes, with dysbiosis increasing the severity of symptoms in such disorders. Results have demonstrated that microbiota-targeting therapies, such as probiotics, prebiotics, and dietary interventions, might have tremendous potential as adjutants to treatments for neurological disorders. This evidence points to the gut-brain axis as a promising avenue for novel interventions, opening the way toward more targeted microbiome-focused therapies in neuropsychiatric and neurodegenerative diseases.

Analysis

Research in the gut microbiota of children with Autism Spectrum Disorder (ASD) has shown differences in microbial community structure, which can partly explain the behavioral and cognitive characteristics associated with the disorder. Numerous studies consistently show that children with ASD have reduced gut microbiota diversity compared to neurotypical children, generally characterized by a decrease in beneficial bacterial species such as Bifidobacterium and Prevotella. These are crucial bacteria in producing short-chain fatty acids, such as butyrate, which maintain the integrity of the blood-brain barrier and influence neurotransmitter activity by modulating immune responses. The dysregulation of these bacterial populations disrupts the gut-brain axis and could lead to ASD behaviors (Clarke, G., & Grenham, S., et al. 2013).

It has also been noted that endocrine markers, such as cortisol—the major stress hormone—are altered in ASD cases. Cortisol levels are often elevated in children with ASD, indicating the intensity of their stress response, possibly due to dysbiosis and the intensification of immune activity it may cause. Such elevated cortisol has been associated with some behavioural symptoms of ASD, like anxiety and repetitive behaviours. SCFA deficiencies, particularly of butyrate, further compound the problem by reducing the anti-inflammatory capacity of the gut and impacting the availability of critical neurotransmitters such as serotonin. Such changes in microbial composition and endocrine markers suggest that gut-targeted interventions may have promise for managing symptoms of ASD, as supported by preliminary benefits in several studies using probiotics and dietary modifications to reduce anxiety and improve behavior.

1. Table: Gut Microbiota and Autism Spectrum Disorder (ASD)

Scopes	Microbiota Profile	Effects on Endocrine Markers	Behavioral Implications	Potential Interventions
Microbial Diversity	Lower diversity in children with ASD, marked by reduced	Reduction in SCFA production (e.g., butyrate) which influences	May contribute to anxiety, repetitive behaviors, and impaired social	Probiotic supplementation aimed at increasing

	Bifidobacterium and Prevotella.	cortisol regulation.	interactions.	beneficial bacteria.
Short-Chain Fatty Acids (SCFAs)	Decreased SCFA levels, particularly butyrate, due to lower beneficial bacteria.	SCFAs support anti-inflammatory responses and maintain blood-brain barrier integrity.	Low SCFA levels correlate with increased neuroinflammation and stress responses.	Dietary interventions to boost SCFA-producing bacteria, e.g., fiber-rich diets.
Endocrine Markers	Higher cortisol levels observed, indicating heightened stress.	Increased cortisol may stem from immune activation and dysbiosis.	Elevated cortisol is linked to ASD symptoms like anxiety and stress sensitivity.	Potential use of microbiome therapy to regulate cortisol levels.
Behavioral Symptoms	Imbalance in gut bacteria affecting neurotransmitter precursors.	Impact on serotonin production, affecting mood and anxiety.	Contributes to repetitive behaviors and challenges in social interaction.	Targeted probiotics and serotonin-supportive nutrients.
Gut-Targeted Interventions	Restoration of microbial diversity using specific strains like Lactobacillus.	Probiotics potentially modulate cortisol and other endocrine pathways.	Interventions have shown preliminary reductions in ASD-related behaviors.	Ongoing studies are investigating dietary adjustments and probiotic regimens.

4.2 Alzheimer's disease, the most common form of dementia, has been increasingly linked with changes in gut microbiota composition. Dysbiosis in Alzheimer's patients often presents with increased pro-inflammatory bacteria belonging to the genus Escherichia and decreased beneficial bacteria belonging to the genera Bacteroides and Lactobacillus. This change in microbial populations appears to increase gut permeability, leading to what is commonly referred to as a "leaky gut." Thus, inflammatory cytokines can pass into the bloodstream and possibly into the blood-brain barrier, increasing neuroinflammation, part of the pathology seen in Alzheimer's disease (Cryan, J. F., & O'Riordan, K. J., et al. 2019). Butyrate is a short-chain fatty acid with anti-inflammatory properties that is critical to brain energy metabolism. It has been at lower levels in patients with Alzheimer's disease. Butyrate supports cognitive function by modulating neuroinflammatory responses and maintaining neuronal health. Its deficiency may accelerate cognitive decline since the brain's resilience to neuroinflammation is lowered. Dysbiosis in Alzheimer's patients also affects the metabolism of bile acids, which are increasingly recognized as relevant to Alzheimer's disease due to bile acids' role in signalling pathways implicated in neurodegeneration. Some studies have indicated the potential of gut-targeted therapies, including dietary changes favoring SCFA production and anti-inflammatory microbial populations, in slowing disease progression and alleviating symptoms (Hsiao, E. Y., & McBride, S. W., et al. 2013).

2. Table: Gut Microbiota and Alzheimer's Disease

Scopes	Microbial Changes	Impact on Health	Potential Interventions
Dysbiosis	Increased pro-inflammatory	Increased gut permeability (leaky	Dietary adjustments to increase anti-

	bacteria (Escherichia); decreased beneficial bacteria (Bacteroides, Lactobacillus).	gut), allowing inflammatory cytokines to enter the bloodstream, exacerbating neuroinflammation.	inflammatory bacteria; use of prebiotics and probiotics.
Short-Chain Fatty Acids (SCFAs)	Lower levels of SCFAs, especially butyrate.	Reduced brain energy metabolism and anti-inflammatory effects; lower resilience to neuroinflammation, accelerating cognitive decline.	SCFA-promoting diets (e.g., high-fiber) and supplementation to boost butyrate levels.
Neuroinflammation	Increased systemic inflammation due to gut dysbiosis.	Chronic neuroinflammation contributing to amyloid plaque buildup and neuronal damage.	Anti-inflammatory dietary interventions; ongoing research on microbiota-targeted therapies to mitigate neuroinflammation.
Bile Acid Metabolism	Altered bile acid metabolism associated with dysbiosis.	Disrupted neuronal signaling pathways; possible impact on neurodegeneration.	Therapies focused on restoring bile acid balance, supporting gut microbiota that positively influence bile acid metabolism.

4.3 Parkinson's is a neurodegenerative disease primarily characterized by motor symptoms like tremors and bradykinesia. Still, it also includes a wide variety of non-motor symptoms, such as gastrointestinal disturbances, mood disorders, and cognitive decline. Recently, an imperative relationship between gut microbiota composition and Parkinson's has been highlighted with dysbiosis being widely spread in patients suffering from this disease. In particular, a decrease can be seen in patients in the number of SCFA-producing bacteria, such as Prevotella and Bifidobacterium. This decrease in SCFAs is related to Parkinson's pathology, since these compounds have anti-inflammatory effects and may protect the central nervous system from neuroinflammation, a major contributor to disease progression. (Vogt, N. M., & Kerby, R. L., et al. 2017). In Parkinson's disease, endocrine markers—dopamine in particular—come to the fore because the hallmark of the disease is a depletion of dopamine-producing neurons. Although dopamine itself cannot cross the blood-brain barrier, some of the metabolites produced by gut bacteria do interact with dopamine pathways, indirectly suggesting an influence of gut health on motor and cognitive symptoms in Parkinson's. Dysbiosis has also been linked with gastrointestinal symptoms characteristic of Parkinson's and often predates motor symptoms by years. Such an early onset of gut-related symptoms supports the idea that dysbiosis may be a contributing factor in the initial stages of Parkinson's disease development. Therefore, targeted microbiota therapies, such as probiotics and dietary interventions to increase SCFA levels, are currently under investigation for potential therapeutic benefits in alleviating motor and non-motor symptoms in Parkinson's patients.

3. Table: Gut Microbiota and Parkinson's Disease

Scopes	Microbial Changes	Key Endocrine Markers	Health Impact	Potential Interventions
SCFA Levels	Lower SCFA levels, particularly butyrate, due to reduced microbial diversity.	SCFAs modulate inflammatory markers and support brain health.	Low SCFAs contribute to increased CNS inflammation, impacting motor functions.	Dietary adjustments aimed at boosting SCFA levels, such as high-fiber diets.
Dopamine Depletion	Microbial imbalance may influence dopamine pathways indirectly.	Dopamine (primary hormone impacted in Parkinson's).	Exacerbates motor symptoms and cognitive decline typical in Parkinson's.	Research into gut-brain axis therapies to indirectly support dopamine levels.
Gastrointestinal Symptoms	Associated with dysbiosis often preceding motor symptoms by years.	Gastrointestinal disruptions may indicate early dysbiosis markers.	Gastrointestinal issues increase before motor symptoms, supporting gut-brain link.	Early probiotic intervention may help manage gastrointestinal disturbances.
Non-Motor Symptoms	Gut microbiota influences mood-related metabolites linked to anxiety.	Influences markers related to serotonin and stress response.	Mood disorders and cognitive decline observed in Parkinson's patients.	Interventions aimed at balancing microbiota to manage non-motor symptoms.

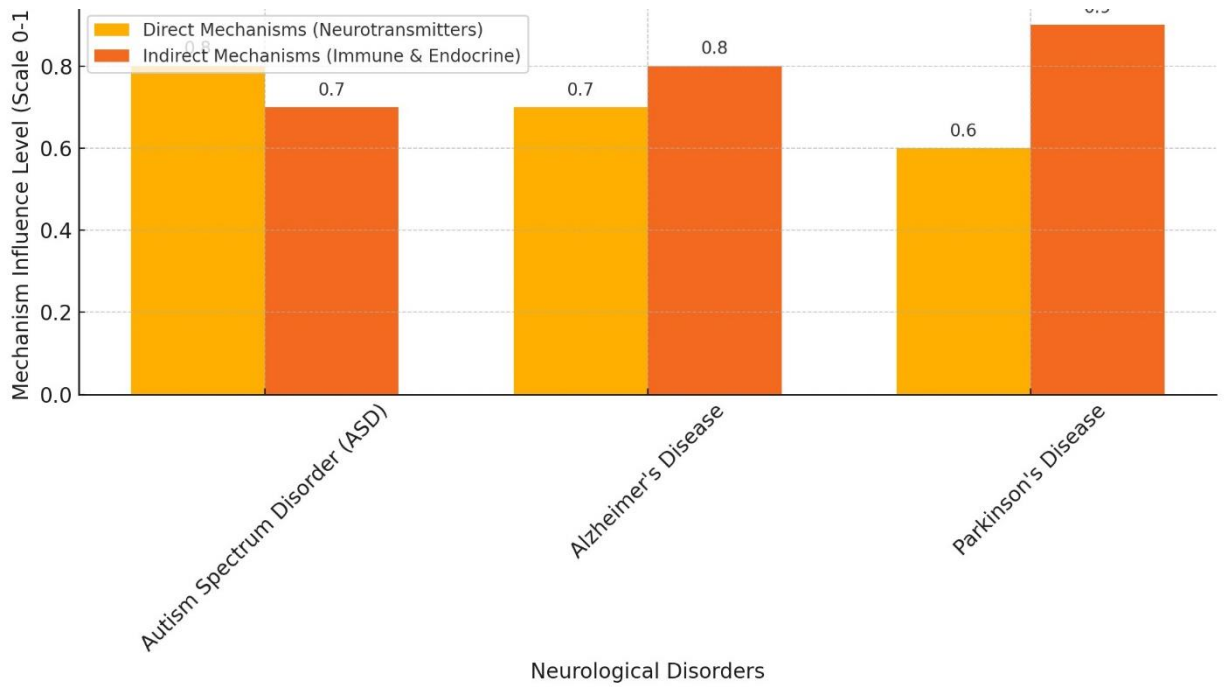
Integrative Perspective on Neurological Disorders and Gut Microbiota

Taken together, emerging studies of the gut-brain axis in neurological diseases emphasize that diversity and proper balance in gut microbiota are very critical to maintaining brain health. Dysbiosis in ASD, Alzheimer's, and Parkinson's diseases is, therefore, likely to affect the brain directly through mechanisms involving altered neurotransmitter production and indirectly by way of pathways involving immune and endocrine signaling. Endocrine and metabolic markers, such as cortisol, SCFAs, and bile acids, have arisen as important factors in the relation between the gut microbiota and the brain. Increased cortisol levels found in ASD and neuroinflammation in Alzheimer's and Parkinson's diseases reveal how gut dysbiosis exacerbates the symptoms of these diseases (Keshavarzian, A., & Green, S. J., et al. 2015).

The consistent finding of SCFA deficiencies in these disorders provides compelling evidence of the therapeutic potential of gut microbiota modulation. Some studies have reported some successes in the use of probiotics, prebiotics, and dietary interventions toward the restoration of microbial balance in alleviating symptoms, reducing neuroinflammation, and improving cognitive and motor functions. However, further studies are needed to provide specific microbial strains and treatment protocols which may be tailored to individuals with these neurological disorders. This increasing body of evidence

suggests that the gut-brain axis might become a target for novel adjuvant strategies in the treatment of neurological disorders, hence paving the way toward microbiome-based interventions in personalized medicine.

Bar chart. Influence of Gut Dysbiosis on Neurological Disorders: Direct and Indirect Mechanisms



DISCUSSION

Gut microbiota, through several mechanisms both direct and indirect, decisively impact CNS function with regard to cognition, mood, and neurodevelopment. Directly, these gut bacteria produce metabolites such as SCFAs like butyrate, acetate, and propionate that cross the BBB and exert health effects within the brain. SCFAs are known to play a vital role in maintaining the integrity of the BBB, preventing neurotoxic compounds from gaining access to the brain. Once inside the brain, SCFAs regulate inflammatory responses by suppressing pro-inflammatory cytokines and supporting the production of neuroprotective compounds. This modulation is critical in controlling neuroinflammatory conditions in which chronic neuroinflammation plays a major role in the pathogenesis of diseases such as Alzheimer's and Parkinson's (Mayer, E. A., & Knight, R., et al. 2020).

Moreover, neurotransmitters and neurotransmitter precursors, such as serotonin, dopamine, and GABA, which are produced by the gut microbiota, influence synaptic plasticity, emotional regulation, and cognitive functions. For example, some 90% of serotonin—a neurotransmitter important for mood stabilization—is produced in the gut; changes in gut microbiota populations may affect serotonin levels. Some studies have been able to indicate that dysbiosis, or microbial imbalance, might lead to lower production of serotonin and thus be connected to mood disorders, anxiety, and depression. For neurological disorders such as

ASD, certain bacteria of the gastrointestinal tract produce SCFAs that are linked with behaviors like repetitive actions and social impairments. The gut bacteria indirectly affect the CNS through activation of immune cells, which in turn influence neuroinflammation.

Dysbiosis can lead to increased gut permeability, often called "leaky gut," thereby allowing LPS and other pro-inflammatory compounds to pass into the bloodstream. These can stimulate immune cells to produce inflammatory cytokines that, in turn, can cross the BBB and promote neuroinflammation. Chronic neuroinflammation has been implicated in the development and progression of neurodegenerative diseases, including Alzheimer's and Parkinson's. These are the ways through which gut microbiota play a multifaceted role in regulating neuroinflammatory responses and synaptic plasticity, raising hopes for therapeutic interventions to restore gut microbial balance. Sampson, T. R., & Debelius, J. W., et al. 2016).

The influence of gut microbiota on brain health has opened new avenues of therapeutic intervention, mostly for neurological disorders. Targeting the gut microbiome represents promising potential in the management and, possibly, the alleviation of symptoms of neuropsychiatric and neurodegenerative disorders. Probiotics, prebiotics, and dietary interventions seem to be the most promising strategies to restore gut microbial balance.

Probiotics, which introduce beneficial bacteria into the gut, have shown promise in studies focused on reduction in anxiety, depression, and cognitive decline.

Specific strains, including Bifidobacterium and Lactobacillus, are more commonly used in probiotic therapies because of their well-documented beneficial effects on mood and immune modulation. Probiotics could restore the balance of the microbiota, increasing SCFAs production and a reduction in inflammation. For instance, in clinical trials, probiotic supplementation has been shown to relieve symptoms in patients with mild cognitive impairment, suggesting that probiotics may prevent further deterioration to dementia (Needham, B. D., & Kaddurah-Daouk, R. 2020). Prebiotics, or dietary fibers that help beneficial gut bacteria grow, are being investigated for the same beneficial effects on mental and neurological health. Prebiotics, by promoting the growth of SCFA-producing bacteria, can contribute to the production of butyrate and other SCFAs involved in supporting brain health by enhancing BBB integrity and reducing neuroinflammation.

Dietary fibers in garlic, onions, bananas, and whole grains act as prebiotics to support microbial diversity and resilience. In the long run, prebiotics are especially useful in dietary strategies since they allow the natural growth of beneficial bacteria without the need for continuous supplementation. Another area of growing interest within the scope of dietary interventions aimed at promoting microbial diversity and resilience relates to supporting brain health. Diets high in fiber, fermented foods, and polyphenol-rich foods (such as berries and green tea) are associated with greater microbial diversity and higher production of SCFAs.

Similarly, fermented foods, such as yogurt, kefir, and sauerkraut, introduce beneficial bacteria directly to the gut, while polyphenols in foods act as prebiotics. The outcome of these interventions seems to give evidence that these interventions not only enhance gut microbiota balance. Still, it might also contribute to improving cognitive function and reducing depressive and anxiety symptoms. In addition to probiotics, prebiotics, and dietary interventions, fecal microbiota transplantation may represent a more direct approach to restoring microbial diversity in cases of severe dysbiosis. During FMT, the gut microbiota is transplanted from a healthy donor into a recipient; this provides an influx of beneficial bacteria.

While it is still experimental in neurological diseases, FMT has succeeded in other diseases, such as infections with Clostridium difficile, and preliminary studies indicate good prospects for applications in neuropsychiatric diseases

(Pellegrini, C., & Antonioli, L., et al. 2018). Such therapeutic approaches underscore the potential of microbiome-centered interventions for treating neurological disorders. Although their mechanisms of action are distinct, they all converge to restore the balance of the microbiota and then enhance neuroprotective effects, reducing neuroinflammation.

The current studies indicate that such therapies could not only alleviate the symptoms but might also contribute to slowing down disease progression, mostly for diseases with a neuroinflammatory solid component, such as Alzheimer's and Parkinson's. A lot more research is needed to fine-tune these therapeutic strategies better, delineating specific strains and protocols for various disorders and developing personalised interventions based on each microbiome profile.

CONCLUSION

The bidirectional communication between the gut and brain is now widely viewed as central to the neurology of disorders. Dysregulation of gut microbiota, or dysbiosis, may act directly on the brain by several pathways, for example, through neurotransmitter production or indirectly via the immune system and endocrine response. This will put the possible importance of this interrelationship in context with diseases such as autism, Alzheimer's, and Parkinson's, in which changes in the gut microbiota are associated with neuroinflammation and loss of cognitive functions.

Therapeutic strategies aiming at the restitution of the microbial balance, therefore, look very promising for the alleviation of symptoms and possibly to slow disease progression. Such treatments, by acting on metabolic markers—for instance, short-chain fatty acids and cortisol—may help reduce neuroinflammation and promote neuronal health. Further research is necessary to understand the functions of endocrine and metabolic markers more precisely in this gut-brain cross-talk. Such insights might help in designing personalised, microbiome-based therapies tailored to an individual's microbial profile, to enhance the strategies for management of and possibly prevention of the progression of such neurological diseases.

REFERENCES

1. Clarke, G., Grenham, S., Scully, P., Fitzgerald, P., Moloney, R. D., Shanahan, F., Dinan, T. G., & Cryan, J. F. (2013). The microbiome-gut-brain axis during early life regulates the hippocampal serotonergic system in a sex-dependent manner. Molecular Psychiatry, 18(6), 666-673. https://doi.org/10.1038/mp.2012.77
2. Cryan, J. F., O'Riordan, K. J., Cowan, C. S., Sandhu, K. V., Bastiaanssen, T. F., Boehme, M., ... & Dinan, T. G. (2019). The microbiota-gut-brain axis: From composition to function. Microbiome, 7(1), 1-27. https://doi.org/10.1186/s40168-019-0479-2
3. Hsiao, E. Y., McBride, S. W., Hsien, S., Sharon, G., Hyde, E. R., McCue, T., ... & Mazmanian, S. K. (2013). Microbiota modulate behavioral and physiological abnormalities associated with neurodevelopmental disorders. Cell, 155(7), 1451-1463. https://doi.org/10.1016/j.cell.2013.11.024
4. Keshavarzian, A., Green, S. J., Engen, P. A., Voigt, R. M., Naqib, A., Forsyth, C. B., ... & Mutlu, E. (2015). Colonic bacteria, inflammation, and motor function in Parkinson's disease. Movement Disorders, 30(10), 1333-1340. https://doi.org/10.1002/mds.26307
5. Mayer, E. A., Knight, R., Mazmanian, S. K., Cryan, J. F., & Tillisch, K. (2020). Gut/brain axis and the microbiota. The Journal of Clinical Investigation, 128(3), 926-938. https://doi.org/10.1172/JCI90912
6. Needham, B. D., & Kaddurah-Daouk, R. (2020). Gut microbiome and stress response in neurological disorders: Cortisol, neuroinflammation, and therapeutic insights. Psychopharmacology, 237(5), 1557-1565. https://doi.org/10.1007/s00213-020-05493-8
7. Pellegrini, C., Antonioli, L., Colucci, R., Blandizzi, C., & Fornai, M. (2018). Short-chain fatty acids and bile acids in the regulation of gut-brain

communication. Neuropharmacology, 136(Part B), 137-146. https://doi.org/10.1016/j.neuropharm.2017.11.011

8. Sampson, T. R., Debelius, J. W., Thron, T., Janssen, S., Shastri, G. G., Ilhan, Z. E., ... & Mazmanian, S. K. (2016). Gut microbiota regulate motor deficits and neuroinflammation in a model of Parkinson's disease. Cell, 167(6), 1469-1480.e12. https://doi.org/10.1016/j.cell.2016.11.018
9. Vogt, N. M., Kerby, R. L., Dill-McFarland, K. A., Harding, S. J., Merluzzi, A. P., Johnson, S. C., ... & Bendlin, B. B. (2017). Gut microbiome alterations in Alzheimer's disease. Scientific Reports, 7, 13537. https://doi.org/10.1038/s41598-017-13601-y