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The Role of Gut Microbiota in Neurodegenerative Diseases

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Abstract

Introduction: The gut microbiota is a key component of the gut-brain axis, and its dysbiosis promotes neuroinflammation. The microbiome plays a significant role in the pathogenesis of neurodegenerative diseases. This article focuses on Alzheimer's and Parkinson's diseases.

Materials and methods: PubMed and Google Scholar databases were searched using the keywords: "gut microbiota," "neurodegenerative diseases," "probiotics," and "fecal microbiota transplantation." A selection of relevant materials was then analyzed.

Aim of the study: The aim of this study is to investigate the impact of the gut microbiota on the development of neurodegenerative diseases, with a particular focus on Parkinson's and Alzheimer's, and to explore the possibility of slowing their progression or improving their treatment by subjecting the microbiota to change.

Description of the state of knowledge: Targeting the gut microbiota represents a novel avenue for managing NDDs. Therapeutic strategies focusing on microbiota modulation, including probiotics and FMT, show significant potential. Further research is essential to validate these approaches and optimize personalized treatments for AD and PD.

Results: Gut microbiota dysbiosis was found to significantly contribute to the progression of AD and PD diseases by promoting neuroinflammation, disrupting the blood-brain barrier, and altering neurotransmitter levels. Probiotics, particularly Lactobacillus and Bifidobacterium strains and FMT showed potential in restoring microbial balance and reducing inflammation and improved cognitive and motor functions. Despite these promising findings, clinical evidence remains limited, and further research is necessary.

Keyword: gut microbiota, neurodegenerative diseases, probiotics, fecal microbiota transplantation

Introduction:

There are approximately 100 trillion microorganisms in the human digestive tract, primarily bacteria, but also viruses, fungi, and protozoa. The microbiome encodes 3 million genes, in comparison, the human body consists of 23,000 (1). The diversity of the microbiome between individuals is enormous, individuals that are approximately 99.9 % identical in terms of genome can differ by approximately 80-90% in terms of their gut microbiome, which can be used to better select personalized therapies (2). Metagenomics studies increasingly provide evidence that imbalances in the gut microbiota (dysbiosis) play a key role in the development of neurodegenerative diseases (NDDs) through the gut-brain axis (MGBA). Changes in the microbiota's composition—whether depletion or excess of specific species—can contribute to the progression or even the onset of these diseases. To better understand the relationships between the microbiome and NDD symptoms, and to precisely identify the key microorganisms responsible for these processes, further scientific research using functional validation methods is necessary to expand the existing body of evidence (3).

1. Influence of the microbiota on the development of neurodegenerative diseases

There are bidirectional communication channels between the gut and the brain that involve neural, inflammatory and hormonal mechanisms. This communication is modulated directly by the vagus nerve and indirectly by neurotransmitters produced by the microbiome, such as GABA, norepinephrine and dopamine, as well as metabolites such as short-chain fatty acids (SCFAs), secondary bile acids (2BAs) and tryptophan metabolites.

The gut microflora can influence the permeability of the blood-brain barrier by modulating the expression of tight junction proteins. Key mechanisms in GBA include neural pathways such as the vagus nerve, which acts as a major channel for the transmission of signals from the gut to the brain, influencing processes such as mood regulation and immune response.

Bacterial neuroactive metabolites, in ways that are not yet fully understood, influence the ageing process and the development of neurological disorders by acting on neuroendocrine cells, resulting in their release of hormones. In addition, they affect the immune function of dendritic cells and microglia (4,5). It has also been proven that the microbiota affects the development and differentiation of immune cells as well as the production of immunological mediators such as IgA, invariant natural killer T cells, and regulatory T cells (6).

Additionally, studies indicate that vitamins A, B, D, and E exhibit neuroprotective properties in the context of Alzheimer’s disease (AD) and Parkinson’s disease (PD). The gut microbiome

plays a crucial role in the metabolism of these vitamins, supplying approximately 30% of the daily requirement for B-group vitamins through the activity of specific gut bacterial strains. These vitamins can also influence the composition of the microbiota, as confirmed by a study showing that serum levels of $1,25(OH)_2D$ are associated with a 5% increase in alpha diversity and a 2% increase in beta diversity of the microbiota (7,8). Vitamin A deficiency may lead to impaired cognitive functions, increased accumulation of beta-amyloid (Aβ) in the brain and gut, as well as reduced levels of brain-derived neurotrophic factor (BDNF) and GABA receptors in the cerebral cortex. These phenomena are linked to lower gut microbiota diversity and a decrease in Lactobacillus bacteria in mice with a model of AD (APP/PS1). This suggests the existence of a bidirectional relationship between vitamins and the microbiota, which is significant in the development of neurodegenerative diseases. These findings indicate the potential benefits of vitamin supplementation in preventing and treating these conditions by modulating the gut microbiota and improving brain function. A deeper understanding of these relationships may lead to the development of new, precise therapeutic strategies that incorporate both diet and the microbiome (9). Furthermore, an interesting aspect is that, in patients with AD, the expression of tight junction proteins, such as claudin, occludin, and zonula occludens-1, is altered. This leads to increased gut permeability, allowing endotoxins to more easily enter the bloodstream and subsequently the central nervous system (10).

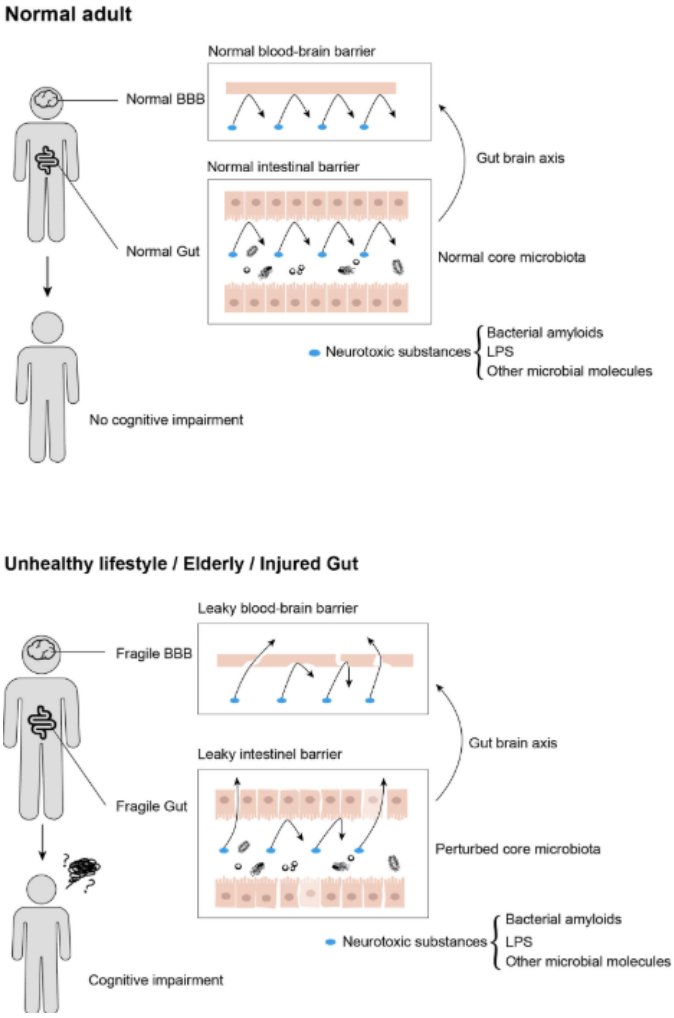

Fig. 1. Neurotoxic substances generated by gut microbiota contribute to the progression of cognitive impairment by traveling from the gut to the brain. This process relies on the intestinal barrier and the blood-brain barrier, which act as critical checkpoints. Under normal circumstances, these barriers prevent the harmful substances from passing through. However, factors such as aging, unhealthy lifestyle choices, and conditions like acute or chronic enteritis can weaken the integrity of these barriers. When this happens, the neurotoxic substances can breach these defenses, reach the brain, and potentially lead to cognitive decline (11).

The microbiome is established during the first three years of life, a process that begins in the prenatal period but can be modulated throughout life by factors such as diet, medication, and stress. The gut microflora plays a crucial role in the normal development and maintenance of brain function. Researchers suggest that disturbances in its homeostasis may contribute to the

development of neurological diseases, including AD, PD, multiple sclerosis, autism spectrum disorders, and stroke (5).

1. Alzheimer's disease

AD, a twofold increase in the level of bacterial lipopolysaccharide (LPS) in the superior temporal lobe neocortex and a threefold increase in the hippocampus have been observed. In some AD patients, up to 26-fold higher levels of LPS, a critical driver of the CNS inflammatory response, have been detected (12).

The gut microbiota of AD patients produces increased levels of tryptophan via the shikimate pathway compared to healthy individuals, which may significantly contribute to the onset of dementia (13). Furthermore, amyloidogenic proteins can induce molecular and cellular changes, promoting aggregation, biofilm formation, tissue invasion, and colonization by various bacterial strains, including Escherichia coli, Salmonella enterica, Salmonella typhimurium, Bacillus subtilis, Mycobacterium tuberculosis, and Staphylococcus aureus.

Of particular relevance to the pathogenesis of AD is the endotoxin produced by E. coli, which enhances the formation of amyloid-beta (Aβ) plaques. These plaques, along with neurofibrillary tangles composed of hyperphosphorylated tau protein, drive neuroinflammation, synapse loss, and neuronal death (14,15). Other bacterial products, such as *E. coli* pili proteins and nucleic acids, have also been detected in the brains of AD patients and are more prevalent in these individuals compared to the general population (16,17). The above data prove the influence of the gut microbiota on the aetiology of AD, so that by influencing the microbiota, the progression of this disease can be slowed down.

2. Parkinson's disease

PD affects more than 1% of individuals over the age of 65 and results from an interplay of genetic and environmental factors (18). Changes in the gut microbiota also play a key role in the pathogenesis of the disease. Patients with PD exhibit alterations in microbiota composition, including an increased abundance of bacteria such as *Akkermansia*, *Lactobacillus*, *Catabacter*, and *Bifidobacterium*, alongside a decreased presence of *Prevotellaceae*, *Faecalibacterium*,

Roseburia, and *Lachnospiraceae*. These changes are associated with increased intestinal permeability and inflammation, which may contribute to the progression of the disease.

Additionally, reduced production of SCFAs, which protect dopaminergic neurons, may exacerbate neurodegeneration in PD patients. The gut microbiota also influences the efficacy of Parkinson's treatment, particularly levodopa, the primary medication used in therapy. Gut microbes metabolize levodopa, potentially affecting its bioavailability and therapeutic effectiveness. Studies have identified bacteria such as *Enterococcus faecalis* and *Eggerthella lenta* as contributors to the metabolism of levodopa and dopamine, highlighting the importance of gut microbiota in the development of personalized treatment strategies for PD.

Emerging research suggests that environmental factors, such as pesticide exposure (e.g., rotenone), may trigger Parkinson's symptoms by altering gut microbiota and promoting the accumulation of α-synuclein in various regions of the central nervous system. Furthermore, genes associated with intestinal diseases, such as *CARD15* and *LRRK2*, may increase the risk of developing PD, suggesting shared genetic pathways between PD and conditions like Crohn's disease (19–21).

3. **Probiotics and prebiotics**

Probiotics and prebiotics, by modulating the composition of the intestinal microflora in ways not yet fully understood, offer therapeutic potential in reducing nervous system inflammation, protecting against cognitive decline, and strengthening the intestinal barrier. Modulating the gut microbiota with probiotics and prebiotics has shown promise in alleviating motor symptoms and nervous system inflammation.

Clinical studies suggest that probiotics, particularly those containing *Lactobacillus* and *Bifidobacterium* strains, can improve symptoms such as constipation, motor function, sleep quality, and cognitive function in PD (20). For example, *Lactobacillus acidophilus* EG004 has demonstrated cognitive-enhancing effects, possibly due to its production of butyrate, a compound that influences brain function by modulating neurotransmitter levels and promoting neurotrophic factors.

Among emerging probiotics, *Clostridium butyricum* has shown protective effects across neurodegenerative conditions, including PD and AD. This strain is known to enhance gut integrity and decrease inflammation, indirectly supporting brain health. Animal studies suggest it mitigates cognitive decline and motor dysfunction by restoring gut microbiota balance and reducing neuroinflammation.

Akkermansia muciniphila has been linked to improved cognitive function in AD models and is being studied as a potential biomarker for early PD diagnosis. However, preliminary findings indicate it may induce α-synuclein aggregation, necessitating further research. Similarly, *Faecalibacterium prausnitzii*, another butyrate producer, influences the Th17/Treg cell balance, inhibiting inflammation and potentially preventing neurodegeneration.

The effects of *Bacteroides fragilis* vary by strain. Non-enterotoxigenic strains exhibit anti-inflammatory properties and improve intestinal barrier integrity, whereas enterotoxigenic strains may promote brain inflammation, particularly in AD. In PD mouse models, *Lactobacillus plantarum* PS128 has been shown to alleviate dopaminergic neuronal death and motor deficits (22). While probiotics hold promise, they should be used as part of a comprehensive treatment approach rather than a standalone therapy for neurodegenerative diseases. Long-term studies highlight the benefits of probiotics like *Bifidobacterium* and *Lactobacillus*, but more research is needed to fully understand their potential and limitations(23). For example, taking probiotics for at least 12 weeks in appropriate doses has been shown to improve cognitive function in individuals with mild cognitive impairment or AD.

The consumption of kefir (2 ml/kg body weight daily for 90 days) has also been associated with improvements in memory, language function, visuospatial abilities, and abstract thinking, alongside reductions in inflammatory and oxidative stress markers. Despite these promising effects, there remains insufficient evidence from randomized clinical trials confirm the long-term neuroprotective effects of probiotics at different stages of AD (24–26). Further large-scale, long-term studies are needed to better investigate the potential of probiotics in preventing and treating neurodegenerative diseases.

4. Faecal microbiota transplantation

Faecal microbiota transplantation (FMT) involves transferring minimally processed faeces from a healthy donor to a recipient's gut to treat disorders associated with gut microbiota dysbiosis. FMT was first studied as a treatment for recurrent *Clostridioides difficile* infection, achieving an impressive success rate of nearly 90%. It has proven to be an effective alternative to antibiotics like vancomycin and fidaxomicin (27–29). FMT has been shown to restore the gut microbiome of patients with CDI to a profile similar to that of healthy individuals (30). PD mouse models, administering 200 μl of a healthy faecal suspension daily for seven days significantly reduced gut microbiota dysbiosis. This intervention also led to decreased SCFAs, increased neurotransmitter levels of dopamine and serotonin, and reduced activation of the TLR4/TNF-α inflammatory pathway in both the gut and brain. These findings suggest that FMT may alleviate nervous system inflammation (31). FMT has demonstrated potential in the treatment of neurodegenerative and neurological diseases, although the evidence varies by condition. In PD, animal studies and some clinical reports in humans suggest beneficial effects. In AD, however, the effects of FMT have so far been observed only in animal studies, underscoring the need for further clinical research (11,32). A large randomized, double-blind clinical trial is essential to better understand the efficacy of FMT in treating and potentially slowing the progression of PD and AD.

5. Results and Discussion

Early diagnosis and personalized therapy that considers the microbiota may help slow the progression of neurodegenerative diseases and improve patients' quality of life. Probiotic use has been associated with reduced β-amyloid accumulation, suggesting that such interventions could play a crucial role in counteracting the early stages of neurodegeneration in AD (3,33). In PD, probiotics, particularly those containing *Lactobacillus* and *Bifidobacterium* strains, have been shown to alleviate gastrointestinal symptoms such as constipation, improve motor function, enhance sleep quality, and support cognitive function.

Further randomized, double-blind studies are needed to comprehensively investigate the role of gut microbiota, probiotics, and FMT in the development and progression of neurodegenerative diseases.

6. Summary

The gut microbiota plays a critical role in the development and progression of NDDs like AD and PD through mechanisms involving the gut-brain axis. Dysbiosis contributes to neuroinflammation, blood-brain barrier disruption, and altered neurotransmitter production. Therapies targeting microbiota modulation, such as probiotics, prebiotics, FMT, show potential in alleviating symptoms, improving quality of life, and slowing disease progression. Although promising, more large-scale, randomized clinical trials are needed to validate these approaches and develop effective, personalized treatment strategies for NDDs.

Disclosure

The authors report no disclosures.

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