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The Impact of Gut Microbiota on Parkinson's and Alzheimer's Diseases: A Review of Medical Literature

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Abstract

Introduction

Neurodegenerative diseases, such as Parkinson's disease (PD) and Alzheimer's disease (AD), pose a major public health challenge due to their progressive nature and profound impact on patients and healthcare systems. Emerging evidence underscores the key role of the gut microbiota in the pathogenesis and progression of these diseases. This paper examines the current state of knowledge on the impact of the gut microbiota on PD and AD, focusing on mechanisms such as modulation of inflammation, blood-brain barrier integrity, neurotransmitter production and amyloid pathology. Future research should target the potential hidden in the gut to fully exploit the therapeutic potential of the gut microbiota in neurodegenerative diseases.

Aim of the study

This review aims to summarize the current state of knowledge on the impact of the gut microbiota on neurodegenerative diseases, mainly Parkinson's and Alzheimer's disease.

Materials and methods

The PubMed database and articles from the last 10 years were reviewed. Keywords used in the search included “gut microbiota,” “Parkinson’s disease,” “Alzheimer’s disease,” and “gut-brain axis.” Selected studies were then analyzed to obtain information on the mechanisms of action.

Conclusions

Intestinal microflora plays a significant role in the pathogenesis of Parkinson's and Alzheimer's diseases. Modulating it through dietary interventions, probiotics and prebiotics holds promise for new therapeutic strategies. Research on the gut-brain axis and its impact on neurodegeneration will enable the creation of new therapies.

Keywords

Gut microbiota, Parkinson's disease, Alzheimer's disease, neurodegeneration, gut-brain axis, SCFA.

Introduction

Neurodegenerative diseases affect millions of people worldwide. Due to the aging population, their development and risk factors are the subject of many scientific observations. According to studies, the pathogenesis of neurological disorders is linked to the dysregulation of the bidirectional axis connecting the nervous system with microorganisms found in the human gut and the substances they produce. Metabolites produced by the gut microbiota are responsible for increased levels of reactive oxygen species (ROS), which significantly contribute to the increased risk of neurodegenerative diseases[1].It is estimated that around 6.9 million Americans suffer from Alzheimer's disease, with numbers projected to rise to 13.8 million by 2060. [2] Parkinson's disease also has a significant prevalence, affecting approximately 1 million Americans, with numbers expected to double by 2040. As of 2024, it is estimated that over 10 million people worldwide are living with Parkinson's disease. This significant increase reflects the growing burden of Parkinson's disease globally, driven by factors such as aging populations and increased life expectancy (WHO, 2023; Parkinson's Foundation, 2024)

Neurodegenerative Diseases

Neurodegenerative diseases are incurable, progressive, debilitating disorders that cause irreversible, selective loss of nerve cells. They most commonly result in motor disturbances of the pyramidal and extrapyramidal systems, as well as cognitive and behavioral dysfunctions. Neurodegenerative diseases are largely associated with the presence of pathological protein aggregates, leading to the loss of function and death of neurons due to proteotoxic stress, oxidative stress, programmed cell death, and neuroinflammation[3]. The most common neurodegenerative diseases are Alzheimer's disease and Parkinson's disease, characterized by the accumulation of specific proteins. In Alzheimer's disease, tau protein accumulates inside neurons, whereas in Parkinson's disease, alpha-synuclein accumulates in Lewy bodies.[4]

Gut Microbiota

Gut microbiota is an integral part of the human organism and evolves throughout life. From birth to death, continuous changes occur [5]The entirety of the microbiota consists of trillions of bacteria, archaea, fungi, and viruses that inhabit epithelial surfaces of the body, influencing local and systemic processes [6]. Most microorganisms reside in the gastrointestinal

tract, estimated to weigh about 1.5 kg of symbiotic bacteria. The main bacteria include Firmicutes, Bacteroides, Proteobacteria, Actinobacteria, Fusobacteria, and Verrucomicrobia.[7]The most diverse species are found in the colon. The quantity and diversity of microorganisms are influenced by genetic predispositions, environment, and diet.[8]Gut microbiota provides many benefits to humans, including maintaining the integrity of the intestinal barrier, metabolizing bile acids, preventing pathogen invasion, and aiding in the production of serotonin, B vitamins, and K vitamins. [9]

Bacterial metabolites play an important role in maintaining homeostasis in the body, produced during the fermentation of polysaccharides—short-chain fatty acids (SCFAs). The most important SCFAs include butyric acid, propionic acid, and acetic acid. SCFAs are responsible for communication between the microbiome and the immune system, participating in balancing pro-inflammatory and anti-inflammatory reactions. [10]SCFAs are natural ligands for fatty acid receptors 2 and 3, found in various immune and endocrine cells.[11] SCFA concentrations change along the gastrointestinal tract, with the highest concentrations in the proximal and the lowest in the distal sections. Dietary habits, environment, and age influence SCFA levels. About 95% of produced SCFAs are absorbed by intestinal epithelial cells, with only about 5% excreted in feces. [10]

SCFAs have been observed to impact conditions such as type 1 and type 2 diabetes, liver cirrhosis, and atherosclerosis by reducing ROS levels through mitochondrial activity modulation.[10]

Dysbiosis

The composition of gut microbiota can change, leading to an overgrowth of undesirable species, including pathogens, causing dysbiosis, negative imbalance. Dysbiosis is characterized by reduced microbial diversity and an increase in Proteobacteria. Studies indicate that many diseases are associated with gut dysbiosis, including type 2 diabetes, obesity, inflammatory bowel diseases, neurological diseases, and autoimmune diseases [12] The microbiota includes so-called pathobionts, which are symbiotic bacteria that can become pathogenic in large numbers and under certain conditions.[13] Dysbiosis, characterized by disrupted microbiome balance and lack of organism diversity, can affect immunological and metabolic processes. Animal studies have shown that microbiota is linked to various diseases, such as liver diseases, allergies, autoimmune arthritis, and neurological disorders. [10] Dysbiosis impacts the nervous

system through numerous gut-brain interactions, including increased intestinal permeability, allowing pro-inflammatory endotoxins like lipopolysaccharides from commensal gram-negative bacteria to enter the bloodstream. These endotoxins can activate the microglia and pro-inflammatory cytokines, inducing a generalized inflammatory response. The interaction between microbiota and the immune system promotes the development of lymphatic tissue and cells. Dysbiosis disrupts proper immune response with a prevalence of pro-inflammatory cytokines, which can also impair nutrient absorption, leading to malnutrition and vitamins. [14]

The Gut-Brain Axis

The gut-brain axis is a bidirectional network between the gut microbiome and the nervous system. Includes gut microbiota metabolites, central nervous system (CNS), neuroendocrine/neuroimmune system, autonomic nervous system, and enteric nervous system. Signals from the intestines reach the CNS and then smooth muscles. The next step is for the CNS to regulate secretion, absorption and blood flow through the intestines. The descending pathway includes the autonomic nervous system, the enteric nervous system, and the hypothalamic-pituitary-adrenal axis. Ascending pathways include vagal sensory pathways, dorsal root ganglia, cytokines, immune mediators, and microbial and intestinal metabolites.[15]

Microbiota can influence the brain by modulating neurotransmission, including serotonergic, noradrenergic, GABA-ergic, dopaminergic, and glutamatergic systems.[16] Serotonin is produced by Candida, Escherichia, and Streptococcus. Acetylcholine is produced by Lactobacillus, and dopamine by Bacillus and Serratia [1] Enteroendocrine cells in the gastrointestinal tract control the entire communication pathway. The cranial nerve, the vagus nerve, plays a key role in gut-brain communication, and its afferent and efferent fibers transmit signals from the gut to the brain and the opposite side. Another communication pathway involves the immune system, which can regulate the concentration of pro-inflammatory and anti-inflammatory cytokines.[1]. It has been observed that pathological gastrointestinal symptoms may precede the onset of Alzheimer's and Parkinson's disease symptoms by several years.[17]

The Role of Gut Microbiota in the Human Body

Numerous studies have been conducted to explain the role of microflora in the body and its impact on human health and diseases. For example, studies on the pathogenesis of type 2 diabetes have shown that Lactobacillus plantarum can promote vesicle transfer and insulin secretion in pancreatic islet cells.[18] Research also indicates that the composition of the microbiota changes after ischemic stroke. A reduction in Lactobacillus counts was observed in monkeys after stroke, and Lactobacillus supplementation improved cognitive function and alleviated depressive symptoms.[19]

Studies have shown the impact of disruption of the gut-brain axis on the pathogenesis of neurological diseases. Metabolites produced by microorganisms can lead to the production of large amounts of ROS, which is a risk factor for neurodegenerative diseases. Neurodegenerative diseases are largely caused by oxidative damage, increased ROS levels, inflammation of the nervous system and impaired energy metabolism, which also affect the number of microorganisms. An imbalance between the amount of ROS and the amount of antioxidants promotes oxidative stress. Intestinal dysbiosis and inflammation of the nervous system cause neurological disorders. In addition to playing a role in the pathogenesis of diseases, microflora also influences the protective functions of the nervous system through the production of metabolites and polyphenols from dietary fiber. Commensal bacteria modulate mitochondrial activity by transforming cellular ROS. Commensal bacteria produce Formylated peptides that cause inflammation in epithelial cells by binding to G protein-coupled receptors on macrophages and neutrophils, which causes the production of nitric oxide, increasing cellular ROS levels. Lactobacilli and Bifidobacterium convert nitrates and nitrites into NO, which has neuroprotective effects at nanomolar concentrations and is considered a neurotransmitter for noradrenergic and noncholinergic neurons in the gut. However, high concentrations of NO have harmful effects on the body, producing reactive oxygen and nitrogen species, highly reactive hydroxyl radicals, which are an important component of neuroinflammation, neuronal degradation and neurodegenerative diseases.[1]

Animal studies have demonstrated the impact of microbiota on brain development, neurogenesis, and the central and peripheral nervous system through the "gut-brain axis." The role of intestinal microflora disorders is also noticed in diseases such as anxiety, depression, visceral pain, somatic disorders on the autism spectrum, Alzheimer's and Parkinson's diseases. Unfortunately, the mechanisms influencing these disorders are not fully understood and

continue to create opportunities for new research and observations by scientists around the world.[20]

Alzheimer's Disease

Alzheimer's disease is the most common neurodegenerative disease worldwide, characterized by progressive short-term memory loss and cognitive dysfunctions. As the disease progresses, personality changes and cognitive deficits occur. [17] It involves the gradual loss of cortical and subcortical brain tissue, along with the accumulation of amyloid-beta plaques and tau protein neurofibrillary tangles [21]. Alzheimer's disease is divided into early-onset and late-onset forms. The early form is associated with genetic predispositions and familial occurrence, while the late form is considered idiopathic and sporadic. Risk factors for the early form include mutations in the amyloid precursor protein (APP), PSEN-1/2 in tau proteins and tau kinase genes. However, the sporadic, idiopathic form is associated with increased expression of mutant forms of apolipoprotein E (APOE), especially APOE4 mutations. The sporadic form may correlate with vascular dementia, potentially due to the accumulation of damaged proteins.[22]

Parkinson’s Disease

Parkinson’s disease is a progressive neurodegenerative disorder characterized by the loss of dopaminergic neurons in the substantia nigra of the brain, leading to motor symptoms such as tremor, muscle rigidity, and bradykinesia.[22] The disease also involves non-motor symptoms, including cognitive decline, mood disorders, and autonomic dysfunctions. The pathological hallmark of Parkinson’s disease is the presence of Lewy bodies, intracellular aggregates of alpha-synuclein.[23][24]

Mechanisms Linking Gut Microbiota with Neurodegenerative Diseases

The Gut-Brain Axis

The gut-brain axis is a bidirectional communication system between the central nervous system (CNS) and the digestive tract. This communication takes place via neural, hormonal and immune pathways, and is significantly influenced by the intestinal microflora. The vagus nerve plays a key role in transmitting signals between the intestines and the brain. Gut microorganisms can produce neurotransmitters such as serotonin, dopamine and gamma-

aminobutyric acid (GABA), which directly affect brain function[25] Moreover, gut microbiota can influence the hypothalamic-pituitary-adrenal (HPA) axis, which regulates stress responses.[26]

The Influence of the Vagus Nerve on the Gut-Brain Axis

The nervous system includes cranial nerves, one of them is the vagus nerve, whose role is to integrate signals between the brain and the digestive tract, creating the so-called gut-brain axis. It is essential for maintaining homeostasis and regulating various physiological functions such as digestion, intestinal motility, enzyme secretion and immune responses.[25] The vagus nerve runs from the medulla oblongata and runs through the neck, chest, and abdomen. It has afferent and efferent fibers that enable monitoring and regulation of the functions of internal organs. Vagal afferent fibers transmit sensory information from the gut to the brain, influencing perception, emotion, and eating behavior.[27] Many researchers have drawn attention to the use of vagus nerve function as a potential therapy for a variety of conditions, including treatment-resistant depression, epilepsy, and inflammatory bowel diseases. VNS may modulate the activity of the gut-brain axis, reducing inflammation and improving cognitive function.[28]

Inflammatory Processes and Neuroinflammation

A key feature of neurodegenerative diseases is inflammation. Gut dysbiosis can lead to "leaky gut," or increased intestinal permeability, allowing bacterial endotoxins such as lipopolysaccharides (LPS) to enter the bloodstream. LPS can cause systemic inflammation and, by crossing the blood-brain barrier, cause inflammation of the nervous system. This process involves the activation of microglia, immune cells that inhabit the brain, which can increase neuronal damage and contribute to the progression of neurodegenerative diseases.[14]

Microbiota Metabolites:

Short-chain fatty acids (SCFA) are metabolites produced by the fermentation of dietary fiber by intestinal bacteria. SCFAs, including butyrate, propionate, and acetate, have anti-inflammatory and neuroprotective properties. They can modulate the expression of genes involved in inflammation and oxidative stress, strengthen the integrity of the blood-brain barrier, and provide energy to brain cells. Studies have shown that butyrate, in particular,

promotes the differentiation of regulatory T cells, which help maintain immune homeostasis and reduce inflammation.[11]

Impact of Gut Microbiota on Parkinson's Disease

Evidence from Preclinical Studies

Animal models of Parkinson's disease have shown that gut microbiota can influence the severity of motor symptoms and neuroinflammation. A study in mice showed that these germ-free animals lacking microbiota exhibit reduced motor deficits and neuroinflammation compared to conventionally bred mice. When germy mice are colonized with the microbiota of Parkinson's disease patients, they develop more severe motor symptoms and increased neuroinflammation. [29] These findings suggest that gut microbiota can modulate the pathophysiology of Parkinson's disease.

Evidence from Clinical Studies

Study Objective

A study was conducted to examine the composition of the gut microbiota in patients with Parkinson's disease and to identify correlations between specific bacterial families and clinical phenotypes of the disease.

Methods

The study included 152 participants. 80 PD patients and 72 healthy controls. Fecal samples were collected and analyzed using 16S rRNA gene sequencing. Diet and lifestyle data were also collected to account for confounding factors such as age, gender and BMI.

Results

Significant differences were observed in the composition of the gut microbiota between PD patients and healthy controls. Patients with PD showed higher levels of Lactobacillaceae, Enterobacteriaceae and Enterococcaceae and reduced levels of Lachnospiraceae. Lower levels of Lachnospiraceae and higher levels of Enterobacteriaceae were associated with greater disease severity and motor impairment.[30]

Clinical studies have shown significant changes in the composition of the intestinal microflora in patients with Parkinson's disease compared to a healthy control group. A reduction in beneficial bacteria such as Lactobacillus and Bifidobacterium and an increase in pro-inflammatory bacteria such as Enterobacteriaceae. Moreover, the severity of motor symptoms in PD patients has been correlated with the abundance of specific bacterial taxa. Higher numbers of Enterobacteriaceae have been associated with more severe postural instability and difficulty in movement.[31]

Gut Microbiota in Parkinson's

The intestinal microbiota, composed of trillions of microorganisms, plays a key role in maintaining homeostasis and influencing various physiological processes. A reduction in the number of Prevotella and Bifidobacterium genera with a concomitant increase in Enterobacteriaceae and Proteobacteria has often been reported.[31][32] Reduced microbial diversity and altered ratio of Firmicutes to Bacteroidetes. These changes in microbiota composition are thought to contribute to gut inflammation, increased intestinal permeability, and systemic inflammation that may exacerbate neuroinflammation and neurodegeneration in Parkinson's disease.[30]

Impact of Gut Microbiota on Alzheimer's Disease

Evidence from Preclinical Studies

Animal studies have shown that intestinal microflora may influence the deposition of amyloid beta and tau proteins in the brain, which play a key role in the pathogenesis of Alzheimer's disease. For example, mice treated with antibiotics to deplete the gut microbiota show reduced beta-amyloid deposition and improved cognitive function. Additionally, probiotic supplementation has been shown to reduce beta-amyloid levels and improve memory in mouse models of Alzheimer's disease.[33]

Evidence from Clinical Studies:

Purpose of the study

The purpose of this study was to assess the composition of the gut microbiota in patients with mild cognitive impairment (MCI) compared to healthy subjects, identifying specific microorganisms associated with Alzheimer's disease biomarkers.

Methods

The study included 20 patients with MCI and 20 healthy controls. Fecal samples were collected and analyzed using 16S rRNA gene sequencing to assess the composition of the gut microbiota. The study also examined interactions between diet, gut microbiota and AD biomarkers.

Results

The result of the study shows significant differences in gut microbiota composition between patients with MCI and healthy controls. Patients with MCI showed reduced microbial diversity and altered proportions of specific bacteria and fungi. increased levels of Candida and decreased levels of Bifidobacterium. These changes correlated with AD biomarkers such as amyloid-beta and tau.

Conclusions

Dysbiosis of the gut microbiota may influence AD pathogenesis by modulating neuroinflammation and amyloid-beta deposition. The results suggest that interventions targeting the gut microbiota may hold promise for the treatment or prevention of AD.[34]

Clinical studies have shown significant changes in the composition of the intestinal microflora of patients suffering from Alzheimer's disease, compared to healthy people. There is a reduction in beneficial bacteria such as Faecalibacterium and an increase in pro-inflammatory bacteria such as Escherichia/Shigella. Moreover, the severity of cognitive impairment in AD patients has been correlated with changes in specific bacterial taxa. Lower numbers of anti-inflammatory bacteria have been linked to more severe cognitive decline.[35]

Gut Microbiota in Alzheimer's: The gut microbiota of AD patients is characterized by reduced microbial diversity and altered microbial composition, which promotes the induction of inflammation. These changes in the gut microbiota may contribute to systemic inflammation, increased intestinal permeability, and neuroinflammation, which play a role in the pathogenesis of Alzheimer's disease.[36] Altered composition of intestinal microbiota in Alzheimer's disease patients promotes inflammation. These changes in the gut microbiota may contribute to systemic inflammation, increased intestinal permeability, and neuroinflammation. [37]

Potential Therapeutic Interventions

Probiotics and Prebiotics: Probiotics are live microorganisms that confer health benefits when consumed in adequate amounts. Prebiotics are non-digestible food components that promote the growth and activity of beneficial gut bacteria. Clinical studies have shown that probiotic supplementation can improve cognitive function, reduce neuroinflammation, and alleviate motor symptoms in patients with PD and AD. For example, a randomized controlled trial demonstrated that probiotic supplementation for 12 weeks improved cognitive performance and metabolic profiles in AD patients.[38]. Recent research indicates that probiotics may play a role in treating these disorders through the gut-brain axis. Probiotics may influence neuroinflammation, oxidative stress, and beta-amyloid accumulation, key factors in neurodegenerative diseases. Lactobacillus and Bifidobacterium strains have demonstrated the ability to enhance cognitive function and reduce neuroinflammation in models of Alzheimer's disease.[39] It has been noted that probiotics in Parkinson's disease result in improved motor functions and reduced aggregation of the alpha-synuclein protein.[40] Randomized studies have shown that 12 weeks of probiotic supplementation correct cognitive impairment and metabolic profiles in patients with Alzheimer's disease.[41] Another study showed that probiotics alleviated gastrointestinal symptoms.[42]

Diet and Lifestyle: A diet rich in fiber, polyphenols and omega-3 fatty acids maintains the balance of intestinal microflora and reduces inflammation. A Mediterranean diet, rich in fruits, vegetables, whole grains, nuts and olive oil, has been linked to a lower risk of neurodegenerative diseases. The effect of fiber on intestinal microflora and inflammation. Dietary fiber has a probiotic effect. It is fermented by intestinal bacteria to produce short-chain fatty acids (SCFAs) such as butyrate, which have anti-inflammatory properties and support intestinal barrier integrity.[43] Polyphenols have strong antioxidant and anti-inflammatory properties. Slowing

the progression of diseases such as Alzheimer's disease by modulating signaling pathways.[44] Omega-3 fatty acids, in particular DHA, have building and controlling functions for neuronal membranes. Supplementation may have a beneficial effect on the reduction of oxidative stress and inflammation.[45] Regular physical activity has a positive impact on human health, which supports the development of beneficial intestinal bacteria, such as Faecalibacterium prausnitzii, and reduces the number of pathogenic bacteria. It is also associated with increased production of SCFAs and their anti-inflammatory effects.[43]

Studies have analyzed the effects of different types of diets on neurodegenerative diseases pathogenesis. The first study showing that a ketogenic diet (KD) that provides fasting may have beneficial effects on neurodegenerative diseases. KD is a type of high-fat, low-carbohydrate diet that is used to treat drug-resistant epilepsy.[46] Over time, the benefits of this diet have been noted in this amyotrophic lateral sclerosis, traumatic brain injury, cerebral ischemia, neurodegenerative diseases (Parkinson's and Alzheimer's diseases). The KD approach has been proposed as a potential therapeutic strategy for AD, based on targeting underlying into the pathophysiological mechanisms of the disease. Impaired glucose metabolism is common in AD. Reduced glucose uptake in the brain plays a role in the development of AD.[47] Vegetarian, Mediterranean and low protein, high-carbohydrate diets. Recent research shows that early changes associated with Parkinson's disease, such as constipation and anosmia, are consistent with histological findings of changes in the gut, including the presence of Lewy bodies.Constipation is the dominant symptom of Parkinson's disease and appears before the onset of characteristic motor symptoms.[48] Research shows more frequent occurrence of PD in people with chronic inflammation intestinal diseases (IBD) and immune system markers Calprotectin activity is elevated in stool samples from PD patients.[49] Impaired intestinal function and microbarriers Imbalance in the gut can contribute to translocation pro-inflammatory cytokines and endotoxinscolon, which may lead to systemic inflammation. A human study assessed how the Mediterranean diet affects the gut microbiota composition, intestinal permeability and gastrointestinal system function in patients with Parkinson's disease. The study shows that after 5 weeks of the diet, constipation disappeared. Bilophila, which was initially higher in PD, decreased slightly after the dietary intervention. Especially, Roseburia's share was much lower in PD compared with controls at baseline and increased after the intervention. However, there were no differences in markers of intestinal permeability between the control group and the control group.[50] Western diet, which contains large amounts of animal fat proteins and refined carbohydrates is associated with the beginning

of inflammation of the nervous system. A Western-style diet causes an imbalance in the intestinal microflora, which leads to an increase in the permeability of the intestinal barrier, and endotoxins enter the circulatory system.[51] LPS produced mainly by Gram-negative bacteria of the intestines bacteria is linked to diseases such as Alzheimer's disease. Studies show positive correlations between AD and attendance Gram-negative bacteria such as Helicobacter pylori, Porphy romonas gingivalis, Prevotella melaninogenica and Campy lobacter rectus.[52] In studies conducted on rats, it was noticed that changes in the composition of intestinal bacteria, especially the ratio of Enterobacteriaceae to Eubacteria increased in the second week of HFD, and Firmicutes/The Bacteroidetes (F/B) ratio increased in the eighth week.This is related to changes in the intestinal microflora gastrointestinal inflammation and systemic inflammation. Additionally, the study showed that long-term consumption HFD for 40 weeks led to an increase in the level of amyloid plaques in the brain. It has been observed that intestinal dysbiosis leads to increased levels of bacterial amyloid in the peripheral nervous system. Long-term HFD consumption not only induces cognitive functions dysfunction, but also contributes to the progression of Alzheimer's disease, is accompanied by increased levels of amyloid in the brain. They find some The results strongly support the suggested "smart brain" hypothesis. that gut dysbiosis plays a role in the early stages of dementia development, even before it becomes visible.[53]

Other Therapies:. Faecal microflora transplantation (FMT) also offers great therapeutic possibilities. This is done by transferring feces from a healthy donor to a patient with gut dysbiosis. FMT has shown promise in the treatment of gut microbiota-related disorders, including Clostridioides difficile infections.

Preliminary research suggests that FMT may have a beneficial effect on therapies for patients with neurodegenerative diseases by restoring the proper composition of the intestinal microflora. There is still insufficient data and new information is still needed to establish the safety and effectiveness of FMT in the treatment of PD and AD. [54][55]

Conclusions

The latest research clearly indicates the significant impact of intestinal microflora on the pathogenesis and progression of neurodegenerative diseases, such as Parkinson's and Alzheimer's disease. Intestinal microflora influences inflammatory and metabolic processes and neurological functions through the communication mechanisms of the gut-brain axis. Research

that shows the potential impact of probiotics, prebiotics, a healthy diet and fecal microflora transplantation in the prevention, diagnosis and treatment of neurodegenerative diseases. However, further in-depth clinical studies are required to fully understand the mechanisms of action and effectiveness of these interventions.

Author's contribution

Conceptualization, Joanna Jakubiec; methodology, Joanna Jakubiec and Martyna Opatowska; software, Tomasz Kucharski and Julita Gmitrzuk check, Katarzyna Wiśniewska and Marta Piotrowska; formal analysis, Joanna Jakubiec and Zuzanna Malinka; investigation, Joanna Jakubiec and Anna Jachymek; resources, Martyna Opatowska and Tomasz Kucharski; data curation, Julita Gmitrzuk, Katarzyna Wiśniewska; writing – rough preparation, Joanna Jakubiec; writing - review and editing, Joanna Jakubiec and Marta Piotrowska; visualization, Joanna Jakubiec, Zuzanna Malinka and Anna Jachymek; supervision, Julita Gmitrzuk and Martyna Opatowska; project administration, Joanna Jakubiec, Tomasz Kucharski. All authors have read and agreed with the published version of the manuscript.

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