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Researchers Discover a Potential Cause for Parkinson's

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STORY AT-A-GLANCE

- Researchers from the University of Helsinki linked a strain of Desulfovibrio bacteria as a causative agent of Parkinson's disease, which may open new therapeutic avenues to screen for, slow progression or prevent Parkinson's
- Nearly 1 million people in the U.S. live with Parkinson's and 90,000 new cases are diagnosed each year; new findings support past research demonstrating a gut-brain link in Parkinson's disease
- Researchers also believe that neurodegenerative disease could be successfully addressed by activating autophagy, the process of eliminating damaged and dead cells to encourage the growth of new healthy cells
- Alzheimer's disease is another neurodegenerative condition linked to specific gut bacteria, including specific short-chain fatty acids and lipopolysaccharides, the cell walls of dead bacteria
- You can take several steps to help optimize your gut microbiome, including eating traditionally fermented foods to seed your gut with healthy bacteria and feeding them with probiotic soluble and insoluble fiber. Avoid antibacterial soap and products with triclosan, processed foods, conventionally raised meat and antibiotics unless absolutely necessary

In May 2023, researchers from the University of Helsinki linked a strain of Desulfovibrio bacteria as a causative agent of Parkinson's disease.[1] Researchers hope this

breakthrough will enable screening for and removal of the bacteria from the gut and potentially prevent the disease.[2]

Parkinson's disease causes unintended or uncontrollable movements that result from brain damage, the mechanism of which has not yet been confirmed. People notice that symptoms begin gradually and then worsen over time. These movements occur because the cells that make dopamine,[3] a neurochemical that helps coordinate movements, stop working or die.

Although it's known as a movement disorder and the primary symptoms include slowness and stiffness with walking and balance, it also triggers other symptoms such as depression, memory problems and constipation. While the condition is not curable, some treatment options may offer relief.[4]

Because the symptoms can differ from person to person, the treatments must also be customized. Medications are primarily used, but a surgical brain implant delivering mild electric current has offered symptom relief for some people. This is also known as deep brain stimulation. Other experimental treatments include stem cell transplants, neuron repair treatments and gene therapies or gene-targeted treatments.[5]

Harmful Bacteria Are a Possible Cause of Parkinson's Disease

Evidence from the featured study suggests that with testing and removal of the harmful bacteria, doctors may be able to prevent Parkinson's disease, or halt its progression. The key feature in pathology is the aggregation of the neuronal protein alpha-synuclein.[6] Past research has suggested these aggregations could be induced in the gut by pathogenic microbes, which have been associated with Parkinson's disease.

The researchers used fecal samples of 10 patients with Parkinson's and fecal samples from their healthy spouses to look for Desulfovibrio species of bacteria. Isolated strains were fed to nematodes to fuse the alpha-synuclein with a yellow fluorescent protein. These were then used in an animal model and compared against a control bacterial strain.

The goal was to determine how the different strains of bacteria contributed to the progression of Parkinson's disease. The researchers found that the strains of bacteria isolated from patients with Parkinson's disease caused aggregation of the protein associated with Parkinson's.

They also noted that different strains of the same bacteria from healthy individuals did not cause aggregation to the same degree. Commenting on the results of the study, Per Saris from the department of microbiology at the University of Helsinki said:[7]

> *"Our findings make it possible to screen for the carriers of these harmful Desulfovibrio bacteria. Consequently, they can be targeted by measures to remove these strains from the gut, potentially alleviating and slowing the symptoms of patients with Parkinson's disease.*
>
> *Once the Desulfovibrio bacteria are eliminated from the gut, α-synuclein aggregates are no longer formed in intestinal cells, from which they travel towards the brain via the vagus nerve like prion proteins."*

In addition to this bacteria, researchers have also found exposure to the widely used chemical trichloroethylene (TCE) is also linked to Parkinson's disease.[8] One study evaluated more than 340,000 service members stationed at Camp Lejeune and Camp Pendleton. When the data were compared, it showed a 70% higher risk of developing Parkinson's for servicemen stationed at Camp Lejeune between 1975 and 1985.

The risk factor was exposure to TCE in the water supply that had reached limits 70 times higher than the EPA deemed safe. Gary Miller is a neurotoxicologist studying Parkinson's disease at Columbia University. He was not involved in the study, but commented that researchers had suspicions that TCE was linked to a higher risk of Parkinson's disease and these results were "very compelling."

TCE has been used since the 1920s in liquid or vapor form. It was an inhaled surgical anesthetic and an ingredient in several types of cleaning products. One of the researchers commented that the chemical is still used in the U.S. and worldwide and "production has been increasing over the past several years."

The chemical breaks down slowly in the environment and is found in one-third of U.S. drinking water. As Ray Dorsey, a neurologist from the University of Rochester commented, "Almost everyone reading your story likely lives near a site contaminated with TCE."

Gut Brain Link in Parkinson's Disease

According to the Parkinson's Foundation,[9] there are 1 million people in the U.S. who are living with Parkinson's Disease, 90,000 are diagnosed with the disease each year and the incidence of the disease increases with age. A 2018 study[10] published in the Journal of Parkinson's disease demonstrated an association between the development of Parkinson's and your gut microbiome.

This study also focused on alpha-synuclein pathology and, as other studies, found it plays a role in the development of familial and sporadic cases of Parkinson's disease. Because the symptoms are only apparent after brain cells have already been affected, it is difficult to slow the progression of the disease.

Researchers have been looking for ways to detect the condition earlier and the results of the featured study may help positively impact prevention and treatment. Alpha-synuclein is a presynaptic protein linked neural pathologically and genetically to Parkinson's disease.[11]

While it may contribute to symptoms, these cells are toxic to cellular homeostasis and trigger neuronal death, which affects synaptic function. Secreted alpha-synuclein may also have negative effects on neighboring cells, seeding aggregation and contributing to progression. Researchers want to identify pathways that are involved in the transfer of protein from the brain to the gut where it's found in people with Parkinson's disease.

Importance of Autophagy in Parkinson's Disease

Some of the aspects[12] of the disease include suppressing the autosomal-lysomal **autophagy system**, which is a systematic degradation of your body's functional

components due to cell destruction and is characterized by the loss of dopamine-transmitting neurons in a section of the midbrain. By activating autophagy, you can begin repairing the dysfunctional mechanism.

Researchers believe neurodegenerative diseases may be successfully addressed by activating autophagy. The term literally means "self-eating" and refers to the process of eliminating damaged cells by digesting them. In essence, it helps clean out old and damaged cells and encourages the growth of new healthy cells, which is foundational to cellular rejuvenation and longevity.

In a paper[13] published in Nature Reviews Drug Discovery, researchers explain the pathway is involved in a variety of human health conditions, including metabolic disorders, neurodegenerative diseases, cancer and infectious diseases. In 2012,[14] researchers noted that dysregulation of autophagy has been observed in the brains of patients with Parkinson's disease as well as in animal models.

Scientists also recognize that autophagy plays a pivotal role in maintaining neurological health. Two major features[15] of Parkinson's disease pathology are the impairment of autophagy that allows alpha-synuclein accumulation and subsequent degeneration of dopaminergic neurons.

Several studies have examined the potential for measuring autophagy biomarkers for early detection of the disease. A 2019 paper[16] in Current Medicinal Chemistry acknowledges that defects in autophagy, microautophagy, macroautophagy and chaperone-mediated autophagy pathways result in the accumulation of protein aggregates.

These are common features in several neurodegenerative disorders, including Huntington's disease, Parkinson's disease and Alzheimer's. They concluded that in the development of new interventions, researchers need a deeper understanding of the autophagy defects in Parkinson's disease and should also account for the multifactorial nature of the disease process.

Alzheimer's and the Gut Connection

Alzheimer's continues to be a leading cause of death in the U.S. According to the Alzheimer's Association,[17] 1 in 3 seniors dies with Alzheimer's or another type of dementia and, according to the CDC,[18] it is the seventh leading cause of death in the U.S.

While a cure has remained elusive, researchers have made the connection between the gut microbiome and brain health, suggesting the bacteria in the intestines influences brain function and can even promote neurodegeneration.[19]

In one study[20] of 89 people between 65 and 85 years of age, researchers used PET imaging to measure amyloid deposits in the brain and they also measured lipopolysaccharide and short-chain fatty acids. Lipopolysaccharide (LPSs) are dead bacteria, or more specifically, the cell walls of dead bacteria.

Since your immune system perceives LPSs as living bacteria, it mounts an immune defense and raises the inflammatory profile. LPSs have been found in amyloid plaques in the brains of Alzheimer's patients. The study revealed high levels of LPSs and the short chain fatty acids acetate and valerate were also associated with large amyloid deposits in the brain.

Butyrate, another short-chain fatty acid, appeared to have a protective effect as high levels were associated with less amyloid. As in the featured study, which demonstrated specific strains of gut bacteria are associated with the development of Parkinson's disease, this study also demonstrated bacterial products in the intestinal microbiota influence the number of amyloid plaques in the brain.

"Our results are indisputable: Certain bacterial products of the intestinal microbiota are correlated with the quantity of amyloid plaques in the brain," explains Moira Marizzoni, a study author with the Fatebenefratelli Center in Brescia, Italy.[21]

Another study[22] published in March 2023, identified 10 bacterial types that were associated with a higher likelihood of developing Alzheimer's disease. As with the harmful bacteria identified with a higher likelihood of Parkinson's disease, researchers

hope this finding will also lead to new treatments that help to lower a person's risk of developing the disease and potentially slowing the progression.

Optimize Your Gut Microbiome

In the Parkinson's study, researchers found harmful strains of Desulfovibrio were associated with the development of Parkinson's disease and in the Alzheimer's study, the team found individuals with Alzheimer's had a reduced microbial diversity and had 10 bacteria overrepresented and other microbes decreased.

As research teams continue to find stronger connections between gut microbiota and neurological health, it only makes sense to **optimize your gut bacteria** to protect you from these and other health conditions. By reseeding your gut with beneficial bacteria, you help keep populations of pathogenic microbes and fungi in check and keep them from taking over.

One of the easiest ways to optimize your gut is to regularly eat traditionally fermented and cultured foods. This is indisputably the most effective and least expensive way to make a significant impact on your gut microbiome. Healthy choices include cultured grass fed organic milk products such as kefir and yogurt, natto and all kinds of fermented vegetables.

I'm not a big supporter of taking many supplements since I believe the majority of your nutrition should come from food, yet probiotics are the exception if you don't eat fermented foods on a regular basis. Spore-based probiotics, or sporebiotics, can be particularly helpful when you're taking antibiotics and they're an excellent complement to regular probiotics.

Antibiotics indiscriminately kill your gut bacteria, both good and bad.[23] This is why secondary infections and lowered immune function are common side effects of taking antibiotics.[24] Chronic low-dose exposure through food also takes a toll on your gut microbiome, which can result in chronic ill health and increased risk of drug resistance.

In addition to seeding your gut with beneficial bacteria and supporting those bacteria with **prebiotic fiber**, it's also important to avoid things that disrupt or kill your microbiome. These include:

- Antibiotics, unless absolutely necessary
- Conventionally raised meats and other animal products, as these animals are routinely fed low-dose antibiotics, plus genetically engineered and/or glyphosate-treated grains
- Processed foods (as the excessive sugars feed pathogenic bacteria)
- Chlorinated and/or fluoridated water
- Antibacterial soap and products containing triclosan

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