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Neuro-gastroenterology: How the gut–brain axis influences brain disorders.

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Introduction

The gut–brain axis refers to the complex, bidirectional communication network between the gastrointestinal system and the central nervous system. This intricate connection involves neural, hormonal, immune, and metabolic signaling pathways that allow the brain to influence gut physiology and the gut to modulate brain function. Once considered separate entities, the gut and brain are now recognized as deeply interconnected systems, with the gut microbiota—a diverse community of microorganisms inhabiting the digestive tract—playing a central role in this relationship. Over the past decade, research has revealed that alterations in gut microbiota composition and function can impact neurological health, contributing to the onset, progression, or severity of various neurological disorders. This emerging understanding is reshaping perspectives on brain diseases and opening new avenues for diagnosis, prevention, and treatment [1].

Communication within the gut–brain axis is mediated through several key pathways. The vagus nerve, the longest cranial nerve, is a primary conduit for neural signals between the gut and brain. Sensory fibers in the vagus nerve detect chemical and mechanical changes in the gut environment and relay this information to brain regions involved in mood regulation, cognition, and autonomic function. Conversely, efferent signals from the brain can modulate gut motility, secretion, and immune responses. Alongside neural communication, endocrine signaling through hormones such as cortisol, ghrelin, and peptide YY contributes to gut–brain communication, influencing appetite, stress responses, and energy balance. Immune-mediated pathways are equally significant, with gut-associated lymphoid tissue serving as a major immune organ capable of releasing cytokines and other inflammatory mediators that affect brain function. Metabolites produced by gut bacteria, such as short-chain fatty acids, tryptophan metabolites, and bile acid derivatives, can also cross the blood–brain barrier or interact with peripheral nerves, influencing neurotransmission, neuroinflammation, and neuroplasticity [2].

The gut microbiota plays a pivotal role in shaping the gut–brain axis. In healthy individuals, a balanced and diverse microbiota supports digestion, nutrient absorption, immune regulation, and the production of bioactive metabolites. However, dysbiosis—an imbalance in microbial composition—can disrupt these functions and alter gut–brain signaling. Factors such as diet, antibiotics, infections, stress, and aging can shift the composition of the microbiota, reducing

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beneficial bacteria and promoting the overgrowth of harmful species. Dysbiosis has been implicated in the pathophysiology of multiple neurological disorders, including Parkinson's disease, Alzheimer's disease, multiple sclerosis, autism spectrum disorders, and depression [3].

In Parkinson's disease, growing evidence suggests that gastrointestinal symptoms such as constipation often precede motor symptoms by years or even decades. Alpha-synuclein, the misfolded protein that accumulates in the brains of Parkinson's patients, has been detected in enteric neurons, leading to the hypothesis that the disease may originate in the gut and spread to the brain via the vagus nerve. Studies have found distinct alterations in the gut microbiota of Parkinson's patients, including reductions in bacteria that produce short-chain fatty acids, which are known to support intestinal barrier integrity and modulate inflammation. These microbial changes may promote systemic and neuroinflammation, contributing to dopaminergic neuron degeneration. Animal models have further shown that transplanting microbiota from Parkinson's patients into germ-free mice can exacerbate motor symptoms, underscoring a causal role for the gut microbiota in disease progression [4].

Alzheimer's disease, the leading cause of dementia, has also been linked to gut microbiota alterations. Dysbiosis in Alzheimer's patients is associated with increased gut permeability, allowing bacterial endotoxins such as lipopolysaccharides to enter the circulation and trigger systemic inflammation. These inflammatory mediators can cross the blood–brain barrier, promoting neuroinflammation and amyloid-beta aggregation in the brain. Certain gut bacteria can influence the production and clearance of amyloid-beta peptides and tau phosphorylation, both central to Alzheimer's pathology. Experimental studies have demonstrated that modifying the gut microbiota through dietary interventions, prebiotics, or probiotics can reduce neuroinflammation and improve cognitive performance in animal models, suggesting that microbiome-targeted therapies may hold promise for human patients [5].

Conclusion

In conclusion, the gut–brain axis represents a fundamental biological pathway linking gastrointestinal health to neurological function. The gut microbiota plays a pivotal role in this connection, influencing neural, immune, and metabolic processes that can contribute to the development and progression of neurological disorders. Evidence from human and animal studies implicates gut dysbiosis in a wide range of conditions, including Parkinson's disease, Alzheimer's disease, multiple sclerosis, autism spectrum disorders, depression, and anxiety.

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