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Impaired H₂ production by gut microbiota and Parkinson's disease. Trends in Endocrinology and Metabolism, 2018; 29 (5): 286-288.

Scientific Paper

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Abstract

Dysbiosis of the gut flora accompanies Parkinson disease (PD), yet no specific cause–effect link has been identified so far. The gut microbiota produce molecular hydrogen (H₂), a ubiquitous molecule recently recognized as a biologically active gas with antioxidant, antiapoptotic, anti-inflammatory, cytoprotective, and signaling properties. Here, we discuss an idea that an impaired production of endogenous H₂ by intestinal microbiota might play a role in PD pathogenesis, with supplemental H₂ debated as a possible therapy for this progressive neurodegenerative disease.

PD remains a perplexing medical disorder that affects approximately one in 500 people, with the cause and the cure generally unknown, even though its clinical phenomenology was first described 200 years ago. It is believed that the PD pathogenesis involves many intrinsic and extrinsic factors, with recent studies shedding new light on the gut–brain axis in neurodegeneration. Even in his seminal report from 1817, Dr James Parkinson broadly described several gastrointestinal symptoms in a patient suffering from ‘shaking palsy’. Many succeeding studies confirmed gastrointestinal dysfunction in PD; various symptoms (constipation in particular) affect up to 80% of PD patients, and may precede the onset of motor impairment by several years. This early involvement of the gastrointestinal system in PD gave rise to several theories that the gut could be the key organ in the initiation and the development of the disease, either via neuroactive substances synthesized by intestinal bacteria or inflammatory mediators that can affect the enteric and central nervous system. Novel DNA-sequencing studies identified a microbial imbalance in the gut of PD patients, with the abundance of several bacteria families positively associated with the severity of motor difficulties. However, which gut-derived factor initiates the pathophysiological cascade in PD stays elusive. H₂, a well-known component of intestinal microbiology, emerges as a possible mediator in the microbiota–gut–brain crosstalk and perhaps fills a missing piece in the PD jigsaw puzzle.

Link: [https://www.cell.com/trends/endocrinology-metabolism/fulltext/S1043-2760\(18\)30027-4](https://www.cell.com/trends/endocrinology-metabolism/fulltext/S1043-2760(18)30027-4)

