Extended Abstract

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The Role of the Gut in Parkinsons Disease

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Abstract

The GM also has effects on neurological outcomes via many mechanisms, like metabolite production and therefore the gut-brain axis. Emerging evidence has gradually indicated that GM dysbiosis plays a task in several neurological diseases, like paralysis agitans (PD), Alzheimer's disease , depression, and MS . In this review, we aim to summarize what is known regarding the correlation between the GM and PD pathologies, including direct, and indirect evidence.

Introduction

Parkinsons disease (PD) is the second most common neurodegenerative condition worldwide and is characterized by dopamine deficiency and Lewy body deposition composed of abnormal alpha-synuclein in the surviving neurons of the substantia nigra [1,2]. While the motor features of PD are well documented its pre-motor features are increasingly becoming recognized with constipation the most frequently reported. This is often associated with small gut intestinal bacterial overgrowth and Helicobacter pylori which are known to worsen motor symptoms [3]. It is because of this the possible role of the gut in PD pathogenesis is being investigated as described by Braaks Hypothesis. [4]. Within the gut lies the microbiome, home to an estimated 100-trillion bacteria, two-thirds of which are unique to each individual and are inherited maternally at birth [2,3]. While 50% to 60% of these bacterial species are yet to be cultured it was thought that their function was to aid digestion and vitamin synthesis however in recent years the existence of a bidirectional gut-brain axis mediated by the vagus nerve which incorporates autonomic/enteric nervous systems and overlapping endocrine/immune systems has been recognized [2,3]. This relationship is evident by the identification of cholinergic anti-inflammatory pathways in the CNS to the gut and the role of gut bacteria in microglia maturation [5,6]. The enteric nervous system consists contains a significant proportion of dopaminergic neurons [6]. In addition, half of the body's dopamine production is made by gut bacteria with gut Lewy- body burden correlating with vagal nerve distribution.

Keywords

Parkinson's disease, gut microbiota, enteric nervous system, gut-brain axis, microbiota-targeted therapies, fecal transplant

Discussion

That is already being investigated. Indeed, unlike other accepted Parkinson Disease treatments such as Deep Brain Stimulation (DBS), this is a relatively safe option which may exhibit synergism with existing treatments. Finally, it seems we may have a promising direction in which to try and curtail the Parkinson disease burden.

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At present the mainstay of PD treatment is Levodopa. This aims to replace the deficiency of dopamine within CNS and minimize symptoms. Currently, it is the single best treatment however it is known the bioavailability of levodopa differs significantly amongst PD patients with its effectiveness waning with time with increasing 'off' time periods reported by patients. Levodopa has recently been shown to be metabolized in the jejunum to dopamine by Enterococcus and Lactobacillus which express the enzyme tyrosine decarboxylase it is thought that this explains the increased levodopa doses required to ameliorate symptoms in late disease. This highlights the need to understand gut biology in order to maximize the effects of therapy in the management of PD. Dietary changes, therefore, affecting such populations, however, may be key in affecting microbiota populations and PD progression.

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