Gut-microbiota link in Parkinson’s disease: current perspectives

Abstract

Parkinson’s disease (PD) is a metacentric neurodegenerative disorder results with accumulation and aggregation of alpha-synculein (α-Syn) (or alpha-synculeinopathy) in the substantia nigra in the central nervous system (CNS). Contributory factors include pesticide exposure, head injury and agriculture background. PD has been considered to be a non-genetic disorder, however around 15% individuals with PD have first-degree relative who has the disease. Mutations in genes including SNCA, LRRK2 and gluococerebrosidase (GBA) found to be risk factor for sporadic PD. Brain cells could be lost due to an abnormal accumulation of the protein alpha-synculein. This insoluble protein accumulates inside neurons forming inclusions called Lewy bodies. Other cell death mechanisms include proteasomal and lysosomal system dysfunction, but the mechanisms are not fully understood. Brain–gut axis (GBA) refers to central nervous system (CNS) control of the enteric nervous system (ENS) through vagus nerve intervention. PD is characterized by alphasynculeinopathy affecting all levels of the brain-gut axis. Both clinical and neuropathological evidences indicate the neurodegenerative changes in PD are accompanied by gastrointestinal symptoms that may precede or follow the central nervous system impairment. Frequent symptoms in PD include tremor, rigidity, slowness of movement and difficulty with walking. Treatment with L-DOPA (levodopa), with dopamine agonist, medications become less effective and produce complications. Research studies recommend new therapeutic approach in PD based on modification of the gut microbiota with probiotics, prebiotics, or even fecal microbiota transplantation.