18th National and 3rd International Conference of Iranian Biophysical chemistry

هجدهمین همایش ملی و سومین همایش بین المللی بیوشیمی فیزیک ایران

25-26 Des, 2024, University of Hormozgan

۶-۵ دی ماه ۱۴۰۳، دانشگاه هرمزگان

Gut Microbiota and Parkinson's Disease: A Double-Edged Sword

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Abstract

Parkinson's disease (PD) is a neurodegenerative disorder characterized by the abnormal accumulation of α-synuclein (α-Syn) in the brain. Recent research highlights the significant role of the gut microbiota, the diverse community of microbes living in the intestines, in modulating α-Syn pathology. This review explores the bidirectional communication along the microbiota-gut-brain axis, focusing on the impact of two gut microbiota metabolites—functional bacterial amyloids (FuBA) and vitamins—on neurodegenerative diseases, particularly PD. FuBA, proteinaceous structures produced by bacteria, contribute to PD pathogenesis by promoting α-Syn aggregation and biofilm formation, which are crucial processes in the development and progression of PD. On the other hand, vitamins, essential micronutrients produced by the gut microbiota, offer neuroprotection through their anti-amyloidogenic, antioxidant, and anti-inflammatory properties. Vitamins such as B vitamins and vitamin K can help reduce oxidative stress, promote neurogenesis, and modulate immune responses, all of which are essential for maintaining brain health. Understanding the complex interplay between the gut microbiota, α-Syn aggregation, and neurodegeneration provides valuable insights into the pathogenesis of PD. Targeting the gut microbiota with therapies aimed at modulating FuBA production or enhancing vitamin synthesis could represent promising avenues for the prevention and treatment of PD. By manipulating the gut microbiome, it may be possible to influence α-Syn aggregation, reduce neuroinflammation, and improve overall brain function in individuals at risk for or diagnosed with PD.

Key words: Gut microbiota, functional bacterial amyloids, vitamin, α-Synuclein, Parkinson’s disease.