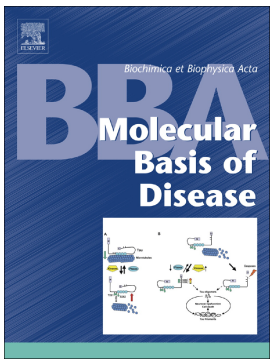

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Mini review

Astroglial and microglial contributions to iron metabolism disturbance in Parkinson's disease

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Abstract

Understandings of the disturbed iron metabolism in Parkinson's disease (PD) are largely from the perspectives of neurons. Neurodegenerative processes in PD trigger universal and conserved astroglial dysfunction and microglial activation. In this review, we start with astroglia and microglia in PD with an emphasis on their roles in spreading α-synuclein pathology, and then focus on their contributions in iron metabolism under normal conditions and the diseased state of PD. Elevated iron in the brain regions affects glial features, meanwhile, glial effects on neuronal iron metabolism are largely dependent on their releasing factors. These advances might be valuable for better understanding and modulating iron metabolism disturbance in PD.

Key words: Astroglia; microglia; iron; alpha-synuclein; Parkinson's disease

Introduction

Parkinson's disease (PD) is a common neurodegenerative disorder characterized by progressive degeneration and loss of dopaminergic neurons in the substantia nigra pars compacta (SNpc). The neuropathological hallmark of PD is characterized by the aggregated alpha-synuclein, with an ascending pattern from the lower brainstem, e.g. dorsal motor nucleus of the vagus nerve and olfactory bulb, and then up to midbrain, finally in the cortex [1, 2]. Prion-like ability of α-synuclein got much attention in these anatomically interconnected regions, as well as extracellular α-synuclein led to the cell-to-cell transmission in the neighboring neurons and glia [3, 4]. Several risk factors as advanced aging, genetic susceptibility or environmental factors might act synergistically or additively in PD pathogenesis [5-9]. Based on these risk factors, iron accumulation was reported in acquired neurodegenerative disorders such as Alzheimer's disease (AD) and PD and in genetic disorders such as neurodegeneration with brain iron accumulation (NBIA) and Friedreich ataxia (FA) [10-12]. Histological and quantitative changes of iron were demonstrated in both PD patients and classic animal models. Epidemiological studies also reveal that iron exposure might be related to a high risk for developing PD [13-16]. Potential mechanisms have been offered to explain why this disturbance in iron metabolism occurs, as well as how elevated iron behaviors leading to dopaminergic neuron degeneration in PD.