Contribution of the nrf2 pathway on oxidative damage and mitochondrial failure in parkinson and alzheimer's disease

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Abstract
The increase in human life expectancy has become a challenge to reduce the deleterious con-sequences of aging. Nowadays, an increasing number of the population suffer from age-associated neurodegenerative diseases including Parkinson's disease (PD) and Alzheimer's disease (AD). These disorders present different signs of neurodegeneration such as mitochondrial dysfunction, inflammation, and oxidative stress. Accumulative evidence suggests that the transcriptional factor nuclear factor (erythroid-derived 2)-like 2 (Nrf2) plays a vital defensive role orchestrating the antioxidant response in the brain. Nrf2 activation promotes the expression of several antioxidant enzymes that exert cytoprotective effects against oxidative damage and mitochondrial impairment. In this context, several studies have proposed a role of Nrf2 in the pathogenesis of PD and AD. Thus, we consider it important to summarize the ongoing literature related to the effects of the Nrf2 pathway in the context of these diseases. Therefore, in this review, we discuss the mechanisms involved in Nrf2 activity and its connection with mitochondria, energy supply, and antioxidant response in the brain. Furthermore, we will lead our discussion to identify the participation of the Nrf2 pathway in mitochondrial impairment and neurodegeneration present in PD and AD. Finally, we will discuss the therapeutic effects that the Nrf2 pathway activation could have on the cognitive impairment, neurodegeneration, and mitochondrial failure present in PD and AD. © 2021 by the authors. Licensee MDPI, Basel, Switzerland.

Author keywords
Alzheimer's disease; Mitochondria; Neurodegeneration; Nrf2 neuro-protection; Parkinson's disease