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Quality Control of Mitochondria in Parkinson's disease (PD) Using ATP and Bradford Assays

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ABSTRACT

Parkinson's disease is a heterogeneous, multifactorial and often complex disease characterized by motor impairment due to the presence of Lewy bodies and prominent degeneration of dopaminergic neurons in the substantia nigra. Although the specific pathogenesis involving PD remains under investigation, mitochondrial dysfunction has been widely accepted as one of the major pathogenic pathways underlying the development of PD. Based on the hypothesis that depiction of HtrA2 (serine protease gene, mitochondrial precursor) might contribute to an increase in mitochondrial stress and transcriptional up regulation of the nuclear stress response CHOP gene. The present study aimed to analyze through laboratory based research the role of HtrA2 and CHOP in the transmission of stress signaling and the consequent activation of mitochondrial quality control in Parkinson's disease using ATP and Bradford assays.

Keywords: Parkinson's disease, Lewy bodies, Mitochondrial dysfunction, Bradford assays

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