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Melatonin modulates rotenone induced behavioral and mitochondrial dysfunction in Drosophila model of Parkinson's disease

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Parkinson's disease (PD) is a neurodegenerative disorder resulting from progressive loss of dopaminergic neuron in the central nervous system. PD causes tremor, rigidity, postural instability and slowness of movement. Rotenone is widely used toxin that inhibits mitochondrial complex-I causing oxidative damage which contributes to the neural loss in PD. Melatonin is a neurohormone produced by the pineal gland. It is a potent free radical scavenger and a broad-spectrum antioxidant. In this study, our aim was to establish Drosophila as an alternate model for PD by applying different doses of rotenone concentration. Further more, we also studied the neuromodulatory role of melatonin in established Drosophila model of PD. We found flies exhibited dosage dependent impairment in behavioral assays, significant induction of oxidative stress as evidenced marked elevation in mitochondrial reactive oxygen species generation. Western blot analysis showed significant decrease in tyrosine hydroxylase, increase of apoptotic regulator protein Bax and decrease of anti-apoptotic regulator protein Bcl-2 among the flies exposed to rotenone for 7 days. However, exposure to melatonin showed significant decrease in Parkinsonian symptoms. Taken together, exposure of rotenone confirmed that rotenone plays an important role in recapitulates key aspects of PD, which is justified in the Drosophila fly model. The effect of melatonin on the rotenone-induced flies was also seen. Further studies must be done to elucidate their molecular and cellular mechanism pertaining to neuroprotection.

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