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α-Bisabolol mitigates rotenone-induced dopaminergic neurodegeneration by suppressing oxidative stress, neuroinflammation and apoptosis in rat model of Parkinson's disease

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Parkinson's disease (PD), a chronic age related neurodegenerative disease is characterized by progressive loss of nigrostriatal dopaminergic neurons. Convincing number of studies showed that oxidative stress, neuroinflammation, impaired apoptosis and autophagy leads loss of dopaminergic neurons and accumulation of α-synuclein, a characteristic of Lewy's bodies in PD. There is scarcity of agents to cure, delay, or prevent the onset and progression of the diseases. In recent years, numerous dietary phytochemicals gained interest for their neuroprotective property and among these, α-bisabolol (BSB), a sesquiterpenes from essential oil of *Matricaria chamomilla* (chamomile) and *Salvia runcinata* (sage) generated interest for evaluation in neurodegeneration. In the present study, rotenone (2.5 mg/kg/day for 4 weeks)-induced rat model of PD was used to investigate the neuroprotective potential of BSB that was administered daily for 4 weeks 30 minutes prior to rotenone. Rotenone induced loss of dopaminergic neurons in the substantia nigra, activated microglia and astrocytes and reduced striatal expression of tyrosine hydrolase, a rate-limiting enzyme in the dopamine metabolism. Rotenone also decreased antioxidants, enhanced α-synuclein and induced proinflammatory cytokines. However, BSB treatment attenuated oxidative stress, cytokines, enzymes and apoptosis along with preserving dopaminergic neurons. BSB also reduced activation of microglia and astrocytes as evidenced by Iba-1 and GFAP and α-synuclein accumulation. The findings demonstrate that BSB exert neuroprotective effects against dopaminergic neurodegeneration induced by rotenone and has potential for further development as a therapeutic candidate for PD.

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