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Neuroprotective effect of sulforaphane in 6-hydroxydopamine-lesioned mouse model of Parkinson's disease

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Abstract

Parkinson's disease (PD) is characterized by the selective loss of dopaminergic nigrostriatal neurons, which leads to disabling motor disturbances. Sulforaphane (SFN), found in cruciferous vegetables, is a potent indirect antioxidant and recent advances have shown its neuroprotective activity in various experimental models of neurodegeneration. This study was undertaken to examine the effects of SFN on behavioral changes and dopaminergic neurotoxicity in mice exposed to 6-hydroxydopamine (6-OHDA). For this purpose, mice were treated with SFN (5mg/kg twice a week) for four weeks after the unilateral intrastriatal injection of 6-OHDA. The increase in 6-OHDA-induced rotations and deficits in motor coordination were ameliorated significantly by SFN treatment. In addition, SFN protected 6-OHDA-induced apoptosis via blocking DNA fragmentation and caspase-3 activation. These results were further supported by immunohistochemical findings in the substantia nigra that showed that SFN protected neurons from neurotoxic effects of 6-OHDA. The neuroprotective effect of SFN may be attributed to its ability to enhance glutathione levels and its dependent enzymes (glutathione-S-transferase and glutathione reductase) and to modulate neuronal survival pathways, such as ERK1/2, in the brain of mice. These results suggest that SFN may potentially be effective in slowing down the progression of idiopathic PD by the modulation of oxidative stress and apoptotic machinery.

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