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Rutin mitigates MPP+ induced neurotoxicity through the regulation of signalling pathways

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Statement of the Problem: Accumulating evidence suggests that apoptosis, autophagy, and dysregulation of signaling pathways are common mechanisms involved in Parkinson's disease (PD) pathogenesis and that development of therapeutic agents targeting these mechanisms may be effective for the treatment of this disease. Rutin, a bioflavonoid, is reported to have pharmacological benefits such as antioxidant, anti-inflammatory, and antitumor activities, however, there are no reports on the activity of this compound in PD models using 1-methyl-4-phenylpyridinium (MPP+). Therefore, we investigated the effects of rutin on apoptosis, autophagy, and cell signaling markers in SH-SY5Y cells treated MPP+.

Methods: Human dopaminergic SH-SY5Y neuroblastoma cells were pretreated with rutin, exposed to MPP+ and then assays were conducted to evaluate cell viability. Western blot techniques were used to investigate apoptosis, autophagy and cell signaling activities. Also, transmission electron microscopy was utilized to examine ultrastructural changes in cells following treatment with rutin and then MPP+.

Findings: Our findings reveal that rutin prevented MPP+ induced changes in nuclear morphology as well as attenuated caspase 3/7 and 9 activities in cells treated with MPP+. Also, rutin effectively regulated cell signaling pathways to protect SH-SY5Y cells from the deleterious effects of apoptosis and autophagy. This was demonstrated by rutin's ability to significantly reduce protein expression levels of cleaved PARP, cytochrome c, LC3-II, and p62 as well as significantly increase protein expression level of full-length caspase 3 in SH-SY5Y cells treated with MPP+. In confirmation of our western blot findings on autophagy, transmission electron images revealed that rutin significantly reduced autophagosomes in SH-SY5Y cells treated with MPP+.

Conclusion and significance of study: Our findings provide experimental evidence highlighting rutin's ability to offer neuroprotection against MPP+-induced neurotoxicity in SH-SY5Y cells and may, therefore, be considered as a promising therapeutic agent for clinical trials in humans.

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