

# Neurotoxicological and neuroprotective elements in Parkinson's disease

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SNpc neurons are uniquely at risk from damage by a variety of ROS, including catechol-quinones formed from autoxidation of DA; as well as Tyr O, TyrOOH, H<sub>2</sub>O<sub>2</sub>-.2, NO., HONOO-, and HO.. The high content of Fe<sup>2+</sup> in SNpc neurons further promotes HO. formation. Self-preservation ensures that SNpc cells are endowed with a battery of cellular antioxidants, which deactivate these cytotoxic species. Most notable are catalase, superoxide dismutase; and DT-diaphorase, which catalyzes a 2-electron reduction of catechol-quinones. Intra mitochondrial MAO can serve to protect or to damage SNpc neurons, depending on the cellular environ at any time. Newer treatment approaches towards PD include the addition of nutrients such as vitamin E and addition of antioxidant drugs that may already be in use as antiparkinsonians: deprenyl, amantadine, DA D2 agonists, apomorphine, NMDA- and adenosine A<sub>2A</sub>-antagonists. Finally, the potential of neural transplants is explored in the proposal that Sertoli cells, or