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Neuroprotective mechanisms of the standardized extract of *Bacopa monniera* in a paraquat/diquat-mediated acute toxicity

Manjeet Singh[a], Ven Murthy[b], Charles Ramassamy[a],

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Abstract

Parkinson's disease (PD) is one of the most common age related neurodegenerative disease and affects millions of people worldwide. Strong evidence suggests a role for oxidative stress and mitochondrial dysfunctions in the pathogenesis of PD. Recent epidemiologic and toxicological studies have shown that environmental factors, especially herbicides such as paraquat and diquat represent one of the primary classes of neurotoxic agents associated with PD. The objective of our study was to investigate the neuroprotective effects of the standardized extract of *Bacopa monniera* (*BM*) against paraquat/diquat-induced toxicity and to elucidate the mechanisms underlying this protection. Our results showed that a pre-treatment with the *BM* extract, from 20.0 μg/ml, protected the rat dopaminergic PC12 cell line against paraquat/diquat-induced toxicity in various cell survival assays. We demonstrated that *BM* pre-treatment, from 5.0 μg/ml, could prevent the generation of intracellular reactive oxygen species (ROS), decreased mitochondrial superoxide levels and depolarized the mitochondria. *BM* pre-treatment also increased tyrosine hydroxylase (TH) levels and antioxidant defense systems such as γ-glutamylcysteine synthetase (γ-GCS) and thioredoxin1 (Trx1) levels. Furthermore,