KM-34, a Novel Antioxidant Compound, Protects against 6-Hydroxydopamine-Induced Mitochondrial Damage and Neurotoxicity

Fonseca-Fonseca, Luis Arturo

Nuñez-Figueredo, Yanier

Sánchez, Jeney Ramírez

Guerra, Maylin Wong

Ochoa-Rodríguez, Estael

Verdecia-Reyes, Yamila

Hernádez, René Delgado

Menezes-Filho, Noelio J.

Costa, Teresa Cristina Silva

de Santana, Wagno Alcântara

Oliveir

 The etiology of Parkinson?s disease is not completely understood and is believed to be multifactorial. Neuronal disorders associated to oxidative stress and mitochondrial dysfunction are widely considered major consequences. The aim of this study was to investigate the effect of the synthetic arylidenmalonate derivative 5-(3,4-dihydroxybenzylidene)-2,2-dimethyl-1,3-dioxane-4,6-dione (KM-34), in oxidative stress and mitochondrial dysfunction induced by 6-hydroxydopamine (6-OHDA). Pretreatment (2 h) with KM-34 (1 and 10 ?M) markedly attenuated 6-OHDA-induced PC12 cell death in a concentration-dependent manner. KM-34 also inhibited H2O2 generation, mitochondrial swelling, and membrane potential dissipation after 6-OHDA-induced mitochondrial damage. In vivo, KM-34 treatment (1 and 2 mg/Kg) reduced percentage of asymmetry (cylinder test) and increased the vertical exploration (open field) with respect to untreated injured