



***Influence of Hesperidin combined with Sinemet
on genetical and biochemical abnormalities in
rats suffering from Parkinson's disease***

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Declaration

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Dedication

*To my father, my mother, my
brother, my sisters*

&

My special friends

*For their love, encouragement, help
and prayers that made studies
possible and to them I owe
everything.*

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Influence of hesperidin combined with sinemet on genetical and biochemical abnormalities in rats suffering from Parkinson's disease

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Abstract

Nowadays, the incidence of neurologic diseases has increased number than previous. Parkinson's disease (PD) is a progressive disabling neurodegenerative disorder characterized by severe difficulties with body motions and associated with autonomic dysfunction, depression, and dementia. Oxidative stress is thought to play an important role in the pathogenesis of PD and oxidative damage characterizes proteins, lipids, and DNA in the substantia nigra pars compacta of PD patients. Scientists believe that PD may result from a combination of genetic and environmental factors. Pesticides represent one of the primary classes of environmental agents that associated with PD. To date, L-dopa is the most effective medication for controlling PD symptoms, although long-term treatment can enhance oxidative stress and accelerate the degenerative process of residual cells. Thus, the inhibition of oxidation of L-dopa and the inhibition of reactive oxygen species formation are important strategies for neuroprotective therapy. Therefore, efforts are made not only to improve the effect of L-dopa treatment for PD, but also to investigate new drugs with both antioxidant and neuroprotective effects. Hesperidin (HDN), a

naturally occurring flavonoid presents in fruits and vegetables, has been reported to exert a wide range of pharmacological effects including antioxidant, antihypercholesterolemic, antihyperglycemic, anti-inflammatory, anticarcinogenic and neuroprotective actions. Chlorpyrifos (CPF) was used in this study as an animal model of PD. Model of CPF-induced Parkinsonism in rats produced neurotoxicity, oxidative stress, hyperlipidemia, hyperglycemia and DNA damage. Rats were orally administrated with different doses of CPF to determine its LD₁₀₀ and LD₅₀ which were found to be 8mg/Kg b. wt. and 1.26 mg/Kg b. wt., respectively. Seventy male rats were used in this study and divided into seven equal groups. After 6 weeks, the following groups were studied, control group, CPF group, HDN group, Sinemet group, CPF+HDN group, CPF+sinemet group, and CPF+HDN+sinemet group. In the present study, the treatment of parkinsonism with HDN alone or combined with sinemet provided a neuroprotection effect when given early in the course of the disease. In conclusion, HDN could be recommended as a disease-modifying therapy when given alone or combined with L-dopa in the course of Parkinson's disease.

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