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AMELIORATIVE EFFECT OF *Mucuna pruriens* AND *Camellia sinensis* ON PARKINSON DISEASE

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ABSTRACT

Mucuna pruriens (Mp) and *Camellia sinensis* (GT) are used in folklore practice in the management of persons presenting with movement disorders with claims of improvement in these conditions. This study was carried out to investigate the motor function potentials of *Mucuna pruriens* and *Camellia sinensis* in parkinsonian mice models. Thirty mice were divided into six groups, namely; control, D2, Mp, GT, D2+GT and D2+Mp groups. Haloperidol was administered for 14 days, and subsequently treated with extracts of *Mucuna prupriens* and *Camellia sinsensis*. Motor function test was performed via parallel bar and rotarod tests. On administration of 15 mg/kg haloperidol (D2), decline in motor function was established. Latency of turn time (25 sec) and PBT time (120 sec) were significant ($p < 0.001$ and $p < 0.05$) in haloperidol treatment groups -D2, and –D2 and GT, respectively. There was no significant difference in rotarod test in the entire groups. Significant increase ($p < 0.05$) was observed in oxidative stress and lipid peroxidation in post haloperidol treatment (-D2 + Mp; -D2 + GT) and Mp (alone) treatment groups, compared with control. Lipid peroxidation was significantly ameliorated in GT and –D2 +Mp treatment. Histopathological studies revealed mild pyknosis and patchy intima erosion in the blood vessels in the D2 group. Findings from this study indicate that Mp and GT have the potential to restore motor activities and ameliorate oxidative stress and lipid peroxidation. Therefore, *Mucuna pruriens* and *Camellia sinsensi* treatment may be possible for amelioration of parkinsonism..

Key words: *Parkinson disease, Mucuna pruriens, Camellia sinensis, Parallel bar test, Rotarod test.*

INTRODUCTION

Parkinson's disease (PD), also known as idiopathic or primary parkinsonism, hypokinetic rigid syndrome, or paralysis agitans is a degenerative disorder of the central nervous system mainly affecting the motor system. PD is the second most common neurodegenerative disorder, primarily characterized by bradykinesia, rigidity, resting tremor, and postural instability. These motor signs are mainly due to progressive degeneration of dopaminergic neurons in the substantia nigra pars compacta (SNpc) (Chaudhuri and Schapira 2009). PD could be triggered by chemical, environmental, genetic and neurotrophic factors in which dopaminergic neurons are lost and there is de-pigmentation in the substantia nigra (SN) (Atasoy et al. 2004; Da et al. 2006). The prevalence increases exponentially with age between 65 and 90 years. The mean age of onset is about 65 years. However 5-10% of people who develop PD experience symptoms before the age of 40 (young onset), and juvenile onset is when people experience these symptoms before the age of 20. (Dave 2008). The motor symptoms are collectively called degenera

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