Thunbergia Laurifolia Lind. Extract Alleviates Motor Impairments in Acute MPTP Mouse Model of Parkinson's Disease

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ABSTRACT

Parkinson's disease (PD) is a neurodegenerative disease whereby there is the characteristic progressive degeneration of dopaminergic neurons in the substantia nigra pars compacta (SNpc) leading to motor deficiencies. The 1-methyl-4-phenyl-1, 2, 3, 6-tetrahydropyridine (MPTP), is the neurotoxin that was used to induce the mice to be models of PD. Thunbergia laurifolia Lindl. (TL) is a Thai herbal medicine which is used for reducing the effects of toxins. This study aims to find out whether TL leaf extract can prevent the onset of PD motor impairments due to MPTP by using pre TL and post TL treatments and observing the mouse's latency times on the Rotarod test, the frequency of movements on the Motor Activity Test, and the numbers of the dopaminergic neurons on the tyrosine hydroxylase immunoreaction (THir⁺) The results showed that the latency times and motor activity of the pre TL treated group were significantly higher than that of the pre-vehicle treated group at P < 0.01. On the other hand, there was no difference between those of the post TL and the post vehicle treated groups. The number of THir⁺ neurons of pre TL and post TL treated groups were significantly higher than that of their vehicle

treated groups, yet the number of the pre TL treated group was less different (P < 0.05) than that of the post TL treated group (P < 0.01) when compared with the control. In conclusion, we have determined that the pre-treatment of TL extract in the MPTP mouse model of PD alleviates motor impairments.

Keywords: *Thunbergia Laurifolia* Lind., Motor impairments, Parkinson's disease, Dopaminergic neurons

INTRODUCTION

Parkinson's Disease (PD) is a chronic progressive neurodegenerative disorder that occurs when there is a dopaminergic neuronal loss in the substantia nigra pars compacta (SNpc). It is characterized by motor abnormalities including tremors, muscular rigidity, bradykinesia, and postural abnormalities (Jankovic, 2008). The most common toxin which is used for inducing PD in animal models is 1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine (MPTP) (Philippens et al., 2010; Tieu, 2011; Blandini and Armentero, 2012; Kasahara et al., 2013). This toxin endangers the dopaminergic neurons in the SNpc leading to dopaminergic neurotransmitter synthesis loss occurrences that are found in PD. It was found that the dopaminergic neurons in SNpc in PD had decreased which is indicated by the presence of immunoreactive tyrosine hydroxylase (THir⁺) cells (Kozina et al., 2014; Alam et al., 2017). The motor function and behavior of animals with PD are accessed by Rotarod and Motor Activity Tests which are found to be significantly different from those of normal animals (Hutter-Saunders et al., 2012).

Thunbergia laurifolia Lindl. (TL) or "Rang chuet" is known as a Thai herb medicinal plant which is well known for its detoxification qualities. TL is used to treat poisonings that can occur from insecticides, arsenic, alcohol, chemicals, drugs, food, and water (Junsi and Siripongvutilorn, 2016). Usanawarong et al. (2000) indicated that aqueous TL leaf extract can reduce toxicity in paraquat intoxicated rats. Tangpong and Satarug (2010) also found that TL leaf extract attenuated the neuronal degeneration in the hippocampus and restored memory loss of lead intoxicated rats. Not only is it used detoxify poisons, TL also has several pharmacological properties being an anti-inflammatory, an anti-oxidant, an anti-microbial, and an anti-nociceptive agent (Chan et al., 2011; Boonyarikpunchai et al., 2014). Thongsaard and Marsden (2013) found that TLextract increased extracellular dopamine levels in striatum.

The acute MPTP mouse model of PD was induced by intraperitoneal injections (i.p.) of MPTP at 20 mg/kg of body weight (BW), 4 times every 2 hours, as has been done in previous study (Lee et al., 2017). This model of PD represents the earliest phase of PD which is indicated by having 53% of the dopaminergic neurons compared to that of a normal subject (Pain et al., 2013), and having a 40% to 90% deficiency in dopamine neurotransmitters (Jackson-