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EVALUATION OF NEUROPROTECTIVE EFFECT OF *Myrmecodia platytyrea* AQUEOUS EXTRACT ON Fe_2SO_4-INDUCED ASTROCYTES (C8-D1A)

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ABSTRACT

Myrmecodia platytyrea was claimed to have antioxidant compounds which have significant pharmacological properties to treat several diseases such as ulcer, diarrhoea, tumour and also in management of cancers. However, not many studies conducted on *M. platytyrea* in the research of discovery of its therapeutic effect especially in treatment of neurodegenerative diseases. Therefore, this research was aimed to study the ability of *M. platytyrea* which possessed antioxidant activity to fight against oxidative stress in astrocyte-induced oxidative stress. Firstly, the cytotoxicity of *M. platytyrea* extract was evaluated using MTT assay on astrocytes (C8D1A) with different concentration of the aqueous extract. After the IC_{50} value was determined, three concentrations were chosen to measure the ability of *M. platytyrea* extract to protect astrocytes against oxidative stress. Astrocytes were induced with both Fe_2SO_4 alone and with the combination of H_2O_2 and Fe_2SO_4. The value of IC_{50} obtained in this study was 501.19±161.11 µg/mL. The percentage of cell death after being treated with 500 µg/mL extract (induced by both hydrogen peroxide and Fe_2SO_4) was lower than the percentage of cell death induced with Fe_2SO_4 alone. *M. platytyrea* may have potential antioxidant activities, however high concentration (500 µg/mL) of *M. platytyrea* suggested a change in mode of action from antioxidant to pro-oxidant. This study implied that *M. platytyrea* is a new potential plant from Rubiaceae family to be discovered as a neuroprotective agent that requires further investigations.

CHAPTER 1

INTRODUCTION

1.1 Introduction

Neuroprotection is a process or mechanism that protects the neuron against injuries or damages. It works on preventing or slowing the progression of the diseases by slowing the rate of neurodegeneration. Neurodegenerative disorders include Alzheimer's disease, Parkinson's disease, and amyotrophic lateral scleroses (ALS) are associated with oxidative stress. According to Shukla, Mishra, & Pant (2011), neuronal damage can occur due to either the increase of oxidative process or the biological system's inability to detoxify the substances causing the decrease of anti-oxidant defense. Since oxidative stress will cause apoptosis in neuron cells, most researchers focused on decreasing the rate of oxidative stress or inhibit the process to protect the neurons (Xian *et al.*, 2012). Oxidative stress occurs when there is imbalance of production of reactive oxygen species (ROS) to its antioxidant defense mechanism favouring to the former.

Human cells are constantly being exposed to environment fortified with oxygen which will continuously generate oxygen free radical that causes oxidative damage (Uttara *et al.*, 2009). Some sources of free radical are from the cells such as hydroxyl radical, superoxide anion, nitric monoxide, and also non free radical entities such as hydrogen peroxide and peroxynitrite. These free radical and non-free radical atoms