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# *Moringa oleifera* IS PROTECTIVE AGAINST MICROARCHITECTURAL AND NEUROCHEMICAL CHANGES ASSOCIATED WITH CUPRIZONE-INDUCED PREFRONTAL CORTEX NEUROTOXICITY IN FEMALE WISTAR RATS

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## ABSTRACT

Cuprizone administration causes selective damage to axonal myelin sheath and has been used to model demyelinating diseases in neuroscience research. This study aimed at determining the protective effects of *Moringa oleifera* on cuprizone-induced neurotoxicity in the prefrontal cortex (PFC). Sixteen adult female Wistar rats were procured and grouped into 4: Group A was given normal saline, Group B received 0.4% cuprizone diet, Group C was administered with 1.875 mg/ml of *Moringa oleifera* and Group D received a combination of 0.4% cuprizone diet and 1.875 mg/ml of *Moringa oleifera*. All the groups were treated orally for 35 consecutive days after which they were sacrificed. Thereafter the PFC was processed for histological demonstration, while tissue homogenate was used to assay the activity of superoxide dismutase (SOD). Cuprizone administration caused significant reduction in body weight and SOD activities. It also caused an alteration in the microarchitecture and Nissl profile of the PFC. *Moringa oleifera* intervention led to restoration of body weight, SOD levels, Nissl profile and the histology of the PFC. The use of preparations of *Moringa oleifera*, especially the leaf-component, could offer some protective measures to individuals suffering from demyelinating conditions, especially in addressing the associated weight changes and frontocortical dysfunction.

**Key words:** *Cuprizone, Demyelination, Moringa oleifera, Weight, Prefrontal cortex*

## INTRODUCTION

Neurotoxins are chemical substances that are destructive to nervous tissues thereby affecting different parts of the nervous system (Adams and Olivera 1994). The injury, depending on the extent, affects various cellular components, processes and functions, with distinct phenotypic manifestations. Cuprizone (CPZ) is a copper chelator, with selective toxicity against oligodendrocytes, which are responsible for the production of myelin in the central nervous system (Taylor et al. 2010). This activity leads to destruction of myelin sheaths that encapsulate the axons of neurons, thereby adversely affecting neuronal morphology and functions, including axonal impulse conduction and metabolic processes, resulting in different forms of demyelinating conditions, the commonest of which is multiple sclerosis (MS) (Chari 2007). Although the

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