

Assessment of the Neuroprotective Efficacy of *Madhuca longifolia* Extract against Dopaminergic Neurodegeneration

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Abstract: Parkinson's Diseases is a chronic neurological disorder marked by progressive motor and neurocognitive impairments, often accompanied by neurocognitive and behavioural impairments. This study investigates the anti-Parkinson's and neuroprotective potential of *Madhuca longifolia*, a naturally occurring plant-derived flavonoid, using a haloperidol - induced Parkinson's model in wistar rat. Haloperidol (1 mg/kg, i.p.) was administered to induce Parkinson's like symptoms, while *Madhuca longifolia* was evaluated at two oral doses (200 mg/kg and 400 mg/kg) for its dose-dependent effects. Syndopa (levodopa and carbidopa) (100: 25 mg/kg) was used as a standard reference drug. Parkinson's activity was assessed based on motor impairment, Muscle rigidity, and Locomotor Activity. Behavioural outcomes were measured using the rota rod test for motor coordination and balance, wire hang test for muscle rigidity, Open field test for Spontaneous locomotor Activity, Catalapsey test for Posture and Pole test for bradykinesia. In addition, oxidative stress markers such as superoxide dismutase (SOD), Dopamine level, and Nitric Oxide (NO) were evaluated. *Madhuca longifolia* administration significantly delayed Parkinson's Activity and improved both motor coordination and cognitive performance in Haloperidol-challenged rats. Biochemical analysis revealed a substantial restoration of antioxidant enzyme activity and reduced oxidative damage in brain tissues. Histopathological examination showed decreased neuronal degeneration, preservation of hippocampal structure, and reduced gliosis in *Madhuca longifolia*-treated groups compared to Haloperidol controls. These findings suggest that *Madhuca longifolia* exerts marked Anti-Parkinson's and neuroprotective effects, likely through antioxidant and anti-inflammatory pathways. The results highlight Anti Parkinson's potential as a therapeutic

agent in Parkinson's management, especially for mitigating oxidative stress and preserving neuronal architecture.

Keywords: Parkinson's management, Catalapsey, Haloperidol induced Parkinson's model, Hippocampal structure, Locomotor Activity.

1. INTRODUCTION

Parkinson's disease is a disorder of the central nervous system impaired movement, muscular rigidity, and reduced coordination. It happens Because of the degeneration of neurons that produce dopamine in the brain, which causes both movement problems and other issues like mood, sleep, etc. Motor signs include difficulty in moving the body, hardened muscles and in trouble to maintain Proper balance. While non-motor signs include anxiety, sadness, insomnia, and memory loss. (1,2,3) The condition is more frequently observed in older adults, and as the world's population ages, its prevalence is rising. Genetics, environmental pollutants, and harmful cell damage are thought to be main reasons behind its development, even if it's Clear Origin is yet unknown. (4,5,6) It is predicted by the (GBD 2021) No. Of the Patient will be more than doubled up to 2050 (7). So far, medicine has not found a full cure for this illness. Current medical care option, such levodopa and other dopaminergic medications, mostly involved in symptom management but do not halt the course of the illness and it is found that due to uses of this conventional medication on long term, then patient is suffering from the Some unwanted Side effects like Nausea, Dizziness, Dyskinesia, Hallucinations etc. As a result, there is a need to investigate safer and more efficacious treatment alternatives, such as plant-based therapies that may be neuroprotective. (8,9)

Pathophysiology of PD:

Problems in mitochondria are strongly connected to the occurrence of Parkinson's disease. In this state Complex I (NADH: ubiquinone oxidoreductase) doesn't work properly and then, increase in the ROS, ATP Decrease, Oxidative Stress Increased and Dopamine Neuron. (10,11) When Inflammation occurs in the brain then the brain activates their immune cell i.e, is Microglia. When Microglia activates then it is released the TNF- α , interleukins, ROS, increase inflammation, damage dopamine neurons. (12,13) One of the main changes seen in Parkinson's condition is the collection of damaged a naturally occurring protein inside brain cells. When These proteins gather together and form Lewy bodies and are responsible for the information and the collections of this protein is increasing in the brain then it disturbs the normal function

like cleaning the waste material and this makes them weak and unable to function normally, and over time the cells slowly die. (14,15,16)

***Madhuca longifolia* Profile**

Madhuca longifolia, which is also known locally as Mahua, is an important plant that is commonly used in traditional medicine for its health benefits and it comes from the family Sapotaceae and is found in tropical regions of India, especially in states like Uttar Pradesh, Bihar, and Jharkhand (17,18). Previously, it has been stated that several sections of *Madhuca longifolia* have healing properties such as anti-inflammatory, antioxidant, and neuroprotective effects (19,20). Due to these properties, it is considered a good candidate for the study of Parkinson's disorder, which involves neurodegeneration and neuroinflammation (21,22).

2. MATERIAL & METHODS

2.1 Inducing Agent (Haloperidol): Rat with Haloperidol induced PD like symptoms serve as a model for Pd because they show same motor impairment and dopamine blocking effects similar to PD. To cause motor defect, reduced locomotor activity and catalepsy, 1mg/kg of haloperidol was used. It Produced dopamine D2 receptor antagonism in brain. Firstly, Haloperidol Injection was administered to the rat and observe their locomotor activity and catalepsy. (23,24,25)

2.2 Experimental Design

Group 1: Control Group This group received normal saline solution administered orally. The dosage was adjusted according to the body weight of each animal. A total of 6 animals were included in this group.

Group 2: Disease Control Group Animals in this group were administered haloperidol at a dose of 1 mg/kg via intraperitoneal injection (i.p.) daily. This group included 6 animals.

Group 3: Standard Drug Group This group received a combination of levodopa and carbidopa (100:25mg/kg) and haloperidol (1mg/kg), both administered orally on a daily basis. A total of 6 animals were used.

Group 4: Test Drug Group 1 Animals were treated with *Madhuca longifolia* (200 mg/kg) and haloperidol (1mg/kg) administered orally and (i.p) respectively each day, This group also consisted of 6 animals.

Group 5: Test Drug Group 2 This group received *Madhuca longifolia* (400 mg/kg) and haloperidol (1mg/kg) administered orally and (i.p) respectively each day, This group also consisted of 6 animals.

2.3 Catalapsey Test: Five groups of six mice each were used for the catalepsy bar test. One by one, the mice were placed on the catalepsy bar test apparatus, and their immobility and rigidity was observed. All observations made on these animals and the time of catalepsy carefully recorded. Readings taken were in groups. (26,27)

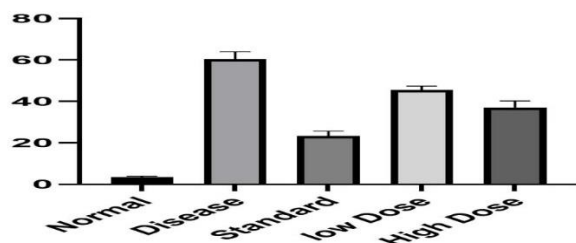


Fig 2.1 Catalapetic Activity

2.4 Open Field Test: This test is used in common behavioral tests in neurodegeneration to evaluate spontaneous locomotor activity in rats. The open field test has an open arena with defined space, where a rat is placed in the center and with squares and allowed to explore freely for five Minutes. The number of squares crossed is counted. Reduced movement indicates motor decline, while increased activity after treatment reflects improvement, this test is used in determining how decline in Neuron health shows effect on locomotor behavior. (28,29)

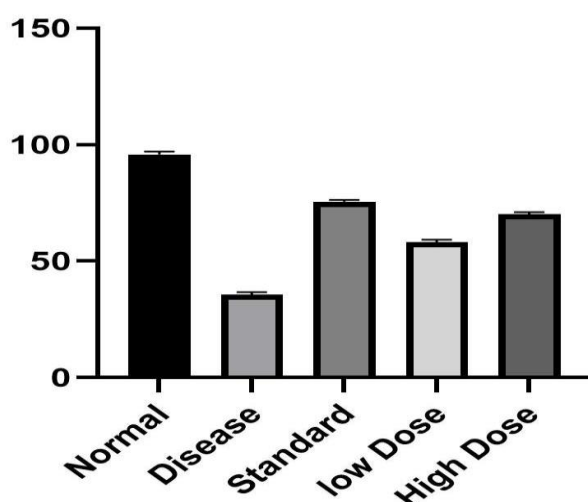


Fig 2.2 locomotor activity

2.5 Pole Test: This is Used to Measure Slowness Of movement in this test the rat was placed head of a vertical pole. The duration of time is noted until it reaches from top to bottom. The longer the time the slower the movement, the shorter the time after treatment the more improvement it shows. (30,31)

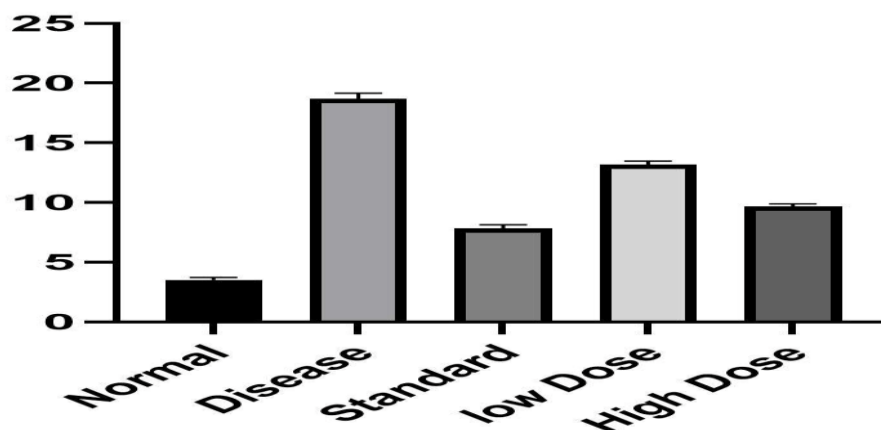


Fig 2.3 bradykinesia activity

2.6 Rota Rod Test: This test is used to study motor coordination and balance. Each rat was placed on a rotating rod set at a 30 RPM. The time for which the animal remained on the rod without falling was recorded in seconds. Each animal was tested three times, and the average value was taken for analysis. A decrease in time indicates motor impairment, while an increase after treatment suggests improvement. (32,33)

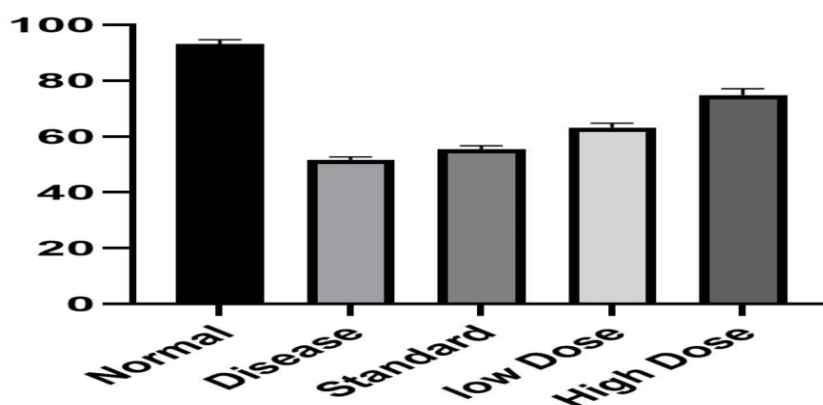


Fig. 2.4 Rotarod Performance (1st day)

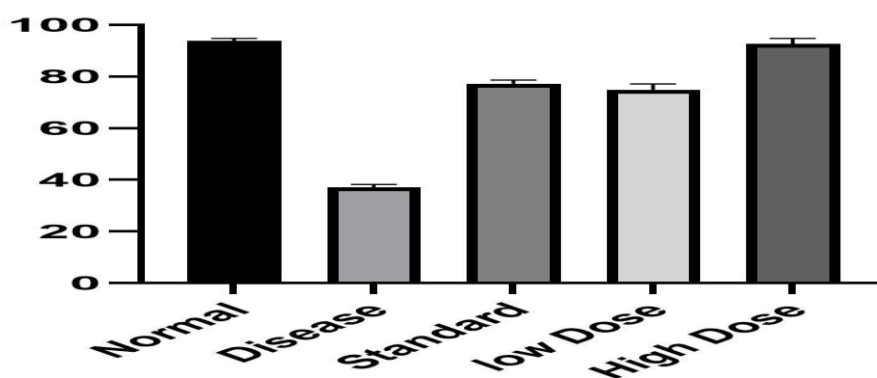


Fig. 2.5 Rotarod Performance (7th day)

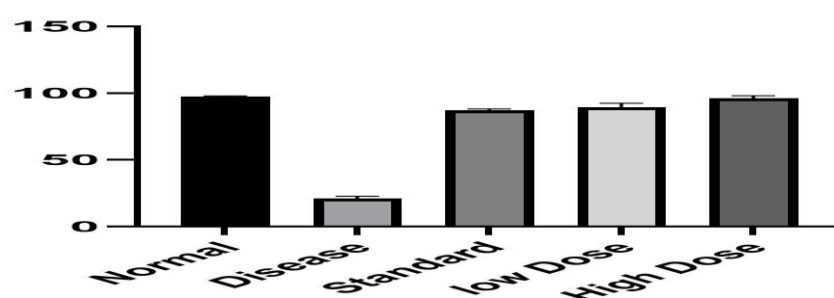


Fig. 2.5 Rotarod Performance (14th day)

2.7 Wire Hang Test: This Test is used to assess the muscle rigidity. In this test rat is placed on hanged wire and noted the time taken to drop rat from the hanged wire. Reduced hanged time tells about muscle weakness while increased hanged time after treatment tells about improvement. (34,35)

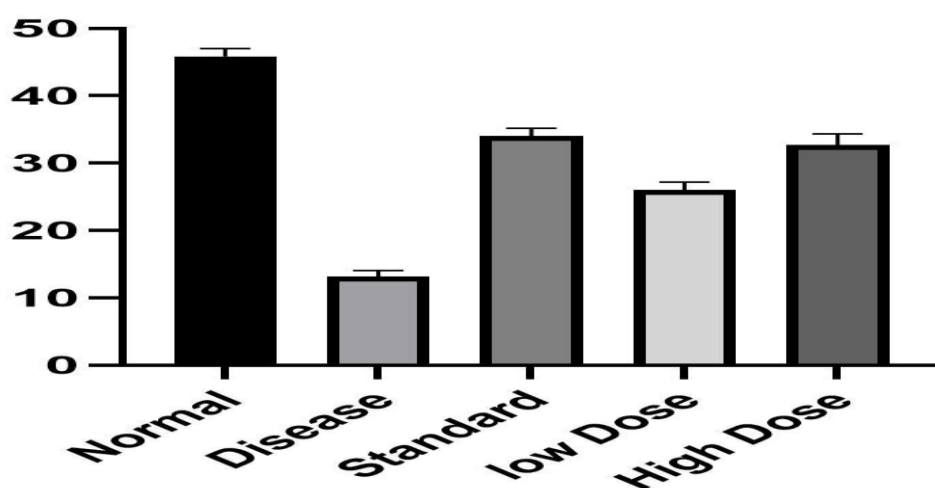


Fig. 2.6 Muscle Weakness

2.8 Estimation Of NO: 2 ml of Griess Regent mixed with 0.1ml homogenate to Produce Pink Coloured then use UV spectroscopy, absorbance of 540nm is to be measured for 2 minutes at 30 to 60 second. The units to be measured in $\mu\text{mol}/\text{mg}$ protein. (36,37)

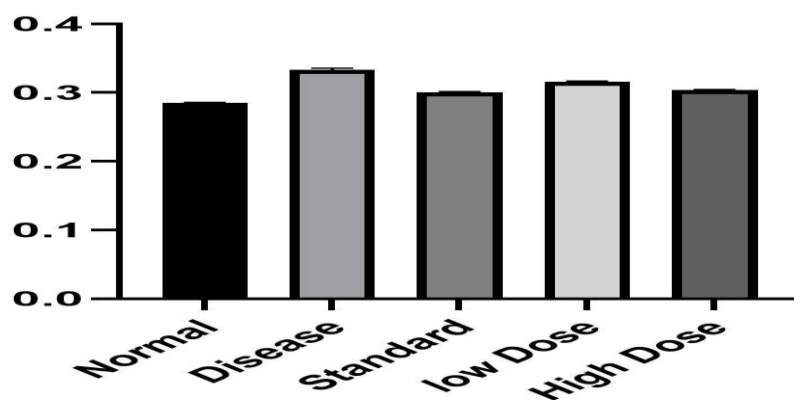


Fig. 2.7 NO Activity

2.9 Estimation Of SOD: 0.5ml of hydroxylamine hydrochloride mixed with 0.1ml homogenate & 2ml of nitazo-bluetetrazolium[NTB], then use UV spectroscopy absorbance of 560nm is to be measured for 2 minutes at 30 to 60 seconds. The units to be measured in U/g protein. (38,39)

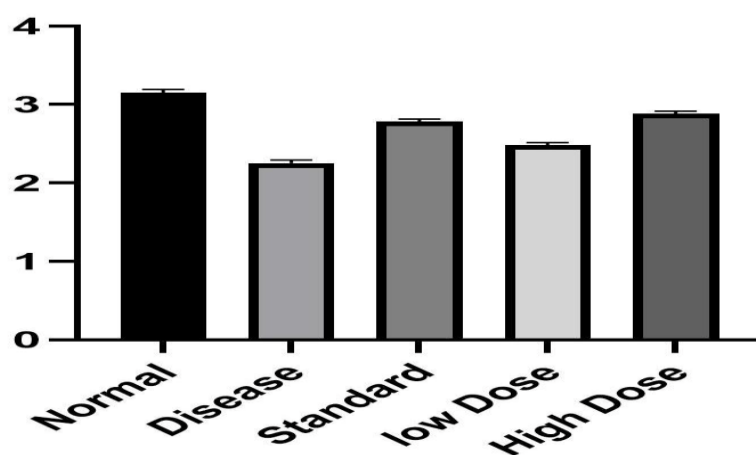


Fig. 2.8 SOD Activity

2.10 Estimation Of GSH: The UV spectrophotometer used to quantify the level of GSH activity, it involves the 0.1ml supernatant in 0.7 ml phosphate buffer solution & 0.2 ML DTNB portion. Absorbance at 412 for 3min at interval of 40s to 60s to form yellow coloured and GSH activity measured U/g protein. (40,41)

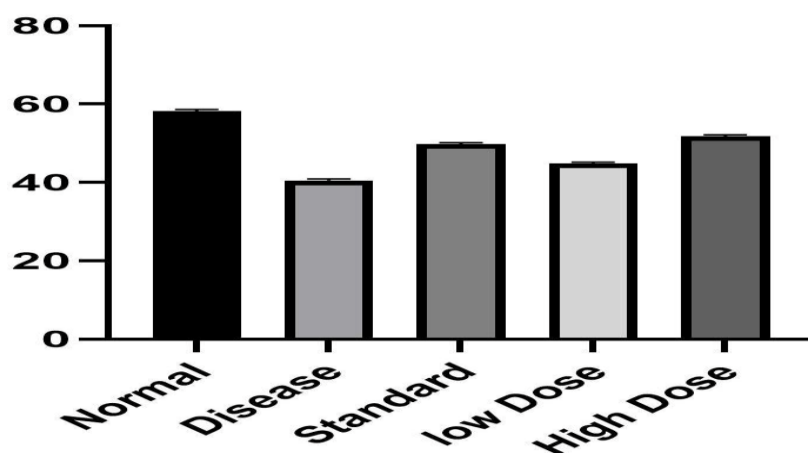


Fig. 2.9 GSH Activity

2.11 Estimation Of Dopamine: The UV-Visible spectrophotometer was used to quantify the level of dopamine. It involves 0.1 ml brain homogenate supernatant mixed with 0.3ml of iodine solution, sodium sulfite and acetic acid. Absorbance at 368 nm was measured for 2 minutes at intervals of 30–60 seconds and dopamine level was expressed as ng/g brain tissue. 42,43)

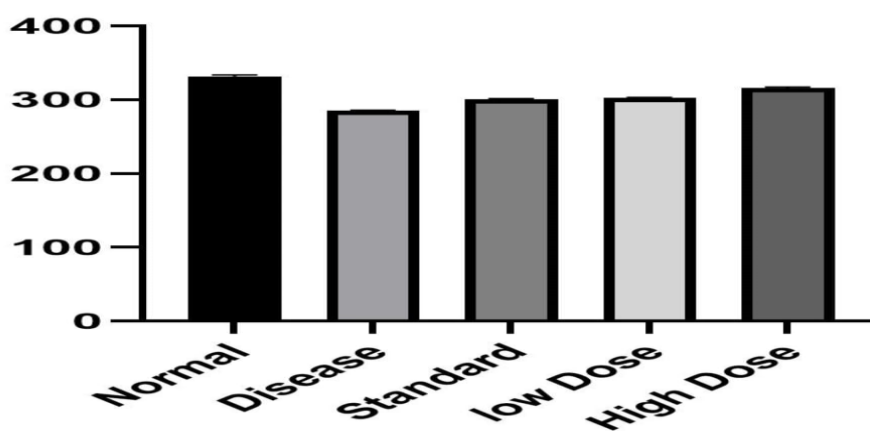
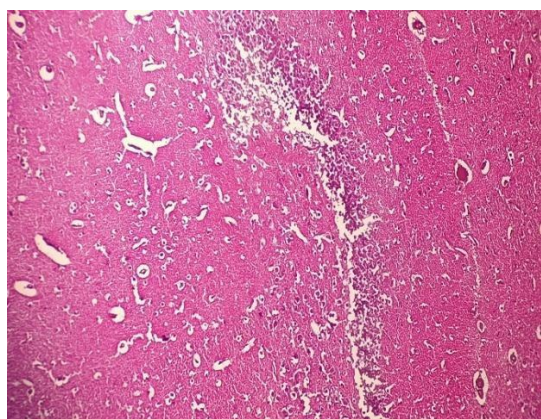
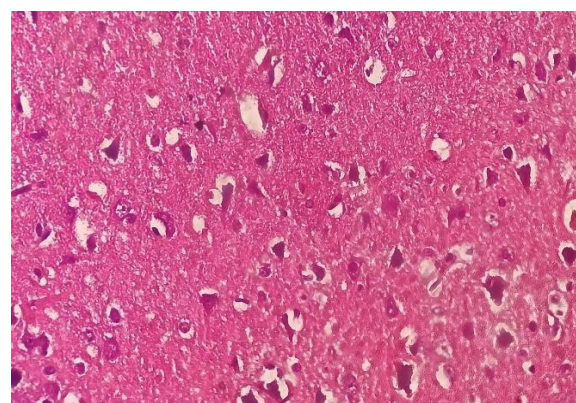


Fig. 2.9 DA Activity

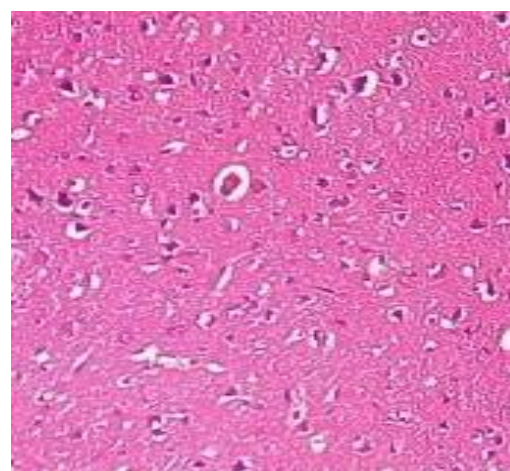
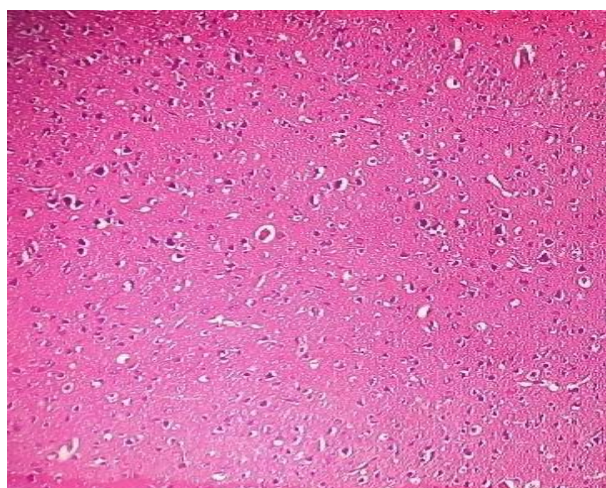
3. HISTOPATHOLOGY BY MICROSCOPIC EVALUATION



Low dose with haloperidol



High dose with haloperidol



Standard with disease control

The histopathological evaluation provided valuable insight into the protective effects of *Madhuca longifolia* against haloperidol-induced neuronal damage. In the disease control group that received only haloperidol, extensive neuronal degeneration was evident. Key features included hippocampal disorganization, neuronal loss particularly in the CA1 region of the hippocampus, which is highly vulnerable to excitotoxic damage. These changes are consistent with the neuropathological patterns commonly observed in chemically induced haloperidol models, where excessive inflammation activity and oxidative stress lead to widespread cellular injury [44,45,46]. In contrast, animals treated with *Madhuca longifolia*, especially at the higher dose of 400 mg/kg, showed significant preservation of hippocampal architecture. The neuronal layers appeared intact, with minimal signs of cellular disruption or inflammatory infiltration. The reduction in oxidative stress and inflammation areas indicates that *madhuca longifolia*

may help prevent glial cell over activation, which is often triggered during oxidative stress activity. Moreover, the lower presence of fragmented or pyknotic nuclei suggests reduced neuronal apoptosis in the test groups [47,48]. Compared to the standard drug syndopa, which also demonstrated a neuroprotective pattern, *Madhuca longifolia* produced comparable preservation of brain tissue with fewer signs of toxicity. The extent of damage seen in the haloperidol-alone group underscores the protective influence of stigmasterol, reinforcing its role in stabilizing neural membranes and possibly modulating excitatory neurotransmitter activity [49, 50]. Altogether, the histopathological outcomes align closely with the behavioral and biochemical findings in this study—supporting the conclusion that stigmasterol mitigates haloperidol induced brain injury by limiting oxidative stress, preserving neuronal structure, and maintaining hippocampal integrity [51].

4. STATISTICAL ANALYSIS

For data reporting, the average mean of the standard of mean, or mean $\pm$ SEM, was utilized; in ANOVA, the difference in gp was compared to the T test. Where the statistical significance of the difference is determined by the p-value.

5. DISCUSSION

This study was conducted to evaluate the neuroprotective potential of *Madhuca longifolia* in a haloperidol-induced epilepsy model using Wistar rats. Haloperidol is widely recognized for inducing Parkinson's activity by altering the excitatory-inhibitory balance in the brain, making it a valuable tool for studying potential anti-Parkinson's agents. As expected, animals treated with haloperidol showed prominent signs of Parkinson's activity, confirming successful induction. However, when treated with *madhuca longifolia*, both at 200 mg/kg and 400 mg/kg doses, a marked reduction in oxidative stress severity and frequency was observed. This suggests that *madhuca longifolia* may offer protective effects against haloperidol-induced neuroexcitation. Biochemical assays further supported these behavioural findings. haloperidol administration significantly reduced superoxide dismutase (SOD) activity, reflecting increased oxidative stress in the brain. Treatment with M.longifolia restored SOD activity, indicating its antioxidant role. In the motor activity test, haloperidol led to noticeable suppression of locomotion due to central nervous system (CNS) disturbance. In contrast, *madhuca longifolia* demonstrated a favorable recovery of motor activity, implying it may help preserve or restore normal CNS function without inducing sedation. The Pole test and wire hang tests were used to assess muscle coordination and motor functions. haloperidol-treated rats showed signs of muscle decline and motor impairment, consistent with previous findings that link dopamine degeneration to neurocognitive deficits. *Madhuca longifolia*

significantly improved performance in both behavioural paradigms, suggesting that it not only prevents dopamine but may also offer cognitive protection and enhance motor retention.

6. CONCLUSION

The findings of this study clearly suggest that stigmasterol possesses significant anti-Parkinson's activity against haloperidol-induced Parkinson's in rats. Both tested doses 200 mg/kg and 400 mg/kg produced protective effects, reducing Parkinson's incidence and associated neurological deficits. In addition to its anti-Parkinson's properties, *Madhuca longifolia* showed strong antioxidant and cognitive-protective effects, demonstrated by improved enzyme profiles, behavioural performance, and preserved brain histology. The neuroprotective effects observed in the *Madhuca longifolia*-treated groups likely stem from its ability to counteract oxidative stress, reduce inflammation, and stabilize cell membranes. Compared to the haloperidol-only group, stigmasterol helped maintain hippocampal integrity, reduced neuronal loss, and minimized cellular disorganization. These properties indicate its potential to limit or prevent long-term damage caused by dopamine degeneration. In conclusion, *madhuca longifolia* shows promise as a natural compound with multiple therapeutic benefits in parkinson's. Its potential role as a neuroprotective and anti-oxidative agent warrants further investigation and may contribute to future drug development targeting Parkinson's disorders and related neurodegeneration.

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