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Research Article

Neuroprotective Evaluation of Green-Synthesized Silver Nanoparticles of *Magnolia Champaca* and *Origanum Majorana* in A Haloperidol-Induced Parkinson's Disease Model in Wistar Rats

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ABSTRACT

Parkinson's disease (PD), the second most prevalent neurodegenerative disorder globally, is characterized by motor dysfunction due to dopaminergic neuronal loss in the substantia nigra. With an alarming increase in global prevalence, projected to reach 25.2 million cases by 2050, there is an urgent need for disease-modifying therapies. This study evaluates the neuroprotective potential of NP-MCOM, a novel green-synthesized silver nanoparticle formulation derived from *Magnolia champaca* flowers and *Origanum majorana* extracts, in a haloperidol-induced Parkinsonian rat model. Behavioral assessments, including catalepsy, dyskinesia, locomotor, and coordination tests, demonstrated significant, dose-dependent motor improvement at both 200 mg/kg and 400 mg/kg of NP-MCOM, with higher efficacy at the 400 mg/kg dose. Biochemical analyses revealed enhanced antioxidant enzyme activity (SOD, catalase, glutathione), suggesting potent antioxidative properties and mitigation of oxidative stress, a known contributor to PD pathogenesis. NP-MCOM's therapeutic effect was comparable to standard levodopa/carbidopa treatment, highlighting its potential to offer both symptomatic relief and neuroprotection. These findings support the continued development of NP-MCOM as a promising, plant-based nanotherapeutic for Parkinson's disease, warranting further mechanistic and clinical investigation.

INTRODUCTION

Neurodegenerative diseases present serious medical challenges, characterized by the progressive dysfunction of the nervous system.

These conditions involve the gradual deterioration of neural structures, affecting key areas such as the brain or the entire nervous network. Although each disease under this category has its own unique

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symptoms, they all share a common trait: a relentless progression that steadily impairs the nervous system. Their subtle onset often masks the damage occurring within, and by the time symptoms become evident, it is frequently too late for effective medical intervention due to delayed diagnosis. At the heart of neurodegenerative diseases lies the progressive deterioration of neuron structure or function—a process known as neuro-degeneration. This relentless decline affects multiple levels of the brain's neural circuitry, from molecular mechanisms to broader systemic consequences. Unfortunately, the damage to neurons is irreversible, making these diseases currently incurable. Nevertheless, research has highlighted oxidative stress and inflammation as key contributors to the neurodegenerative process. Parkinson's Disease (PD) is relatively rare before

the age of 50, but its prevalence increases significantly with age. Globally, the annual incidence ranges from 5 to over 35 new cases per 100,000 individuals, a variation likely influenced by demographic differences and methodological inconsistencies across studies. A Minnesota-based study using validated diagnoses reported an incidence rate of 21 cases per 100,000 person-years. The incidence of PD rises sharply between the sixth and ninth decades of life. While the overall global prevalence is estimated at approximately 0.3%, it exceeds 3% among individuals aged 80 and older. Mortality rates in PD do not show a marked increase during the first decade after diagnosis, but they double in subsequent years. Advances in healthcare and increased life expectancy have contributed to the growing prevalence of PD over time.

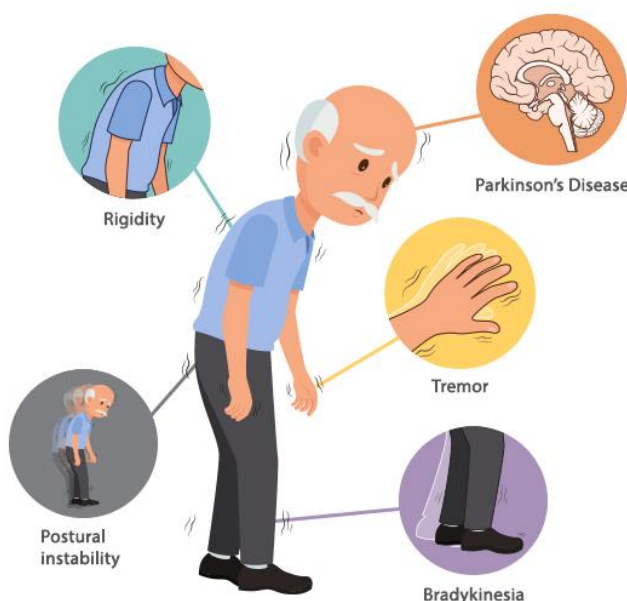


Fig: 1 Parkinson's Disease

1. Plant Profile:

1.1 *Magnolia Champaca* (L.) Baill. Ex Pierre:

2.1.1 Taxonomy:

✚ **Family:** Magnoliaceae

✚ **Genus:** *Magnolia*

✚ **Species:** *champaca*

2.1.2 Botanical Name:

✚ *Magnolia champaca* (L.) Baill. ex Pierre

2.1.3 Synonyms:

✚ *Michelia champaca* L.

✚ *Champaca michelia* (historical usage)

2.1.4 Chemical Constituents of *Magnolia champaca*:

Different parts of the plant—flowers, bark, leaves, seeds, and roots—contain a variety of bioactive compounds. Key classes include alkaloids, essential oils, lignans, flavonoids, terpenoids, and phenolics.

Major Constituents (by plant part):

Flowers:

✚ **Essential Oil** - Linalool, β -Caryophyllene, Methyl eugenol, α -Humulene, Farnesol

✚ **Flavonoids** – Quercetin, Kaempferol

✚ **Phenolic compounds**

Leaves: Flavonoids, Tannins, Saponins, Alkaloids

Bark: Alkaloids (e.g., magnoflorine), Lignans, Triterpenoids, Sterols, Tannins

Seeds: Fixed oils, Fatty acids (palmitic acid, linoleic acid), Proteins, Saponins

Roots: Alkaloids, Lignans



Fig: 2 *Magnolia champaca* (L.) Baill. ex Pierre

2.2 ORIGANUM MAJORANA L:

2.2.1 Synonym:

Origanum majorana has a few botanical synonyms, although it's quite a well-defined species. Some of these may reflect older classifications or variations in taxonomy:

✚ *Majorana hortensis* Moench

✚ *Amaracus majorana* (L.) Benth.

✚ *Origanum onites* var. *majorana* (L.) Nyman

✚ *Marjorana majorana* (L.) H.Karst.

2.2.2 Taxonomical Classification:

✚ **Kingdom:** Plantae

✚ **Order:** Lamiales

✚ **Family:** Lamiaceae

✚ **Genus:** *Origanum*

✚ **Species:** *Origanum majorana*

2.2.3 Chemical Constituents:

Essential Oil Components (Main Bioactive Compounds): Terpenoids: Terpinen-4-ol, γ -

Terpinene, α -Terpinene, Sabinene, Linalool, Carvacrol, Thymol, Cis-sabinene hydrate

Other Phytochemicals: Flavonoids (e.g., apigenin, luteolin), Phenolic acids (rosmarinic acid, caffeic acid), Tannins, Saponins, Ascorbic acid (Vitamin C)



Fig: 3 *Origanum majorana* L.

2. MATERIALS AND METHODS:

3.1 Plant Collection:

Fresh Flower of *Magnolia champaca* (L.) Baill. ex Pierre and Whole Plant of *Origanum majorana* L were collected from the local area of Thiruvannamalai district. It was washed gently with distilled water in order to remove the dust particles may present on the surface of the flower and whole plant. and it was dried under shade for 5 days straight. The stem barks are crushed into smaller pieces and stored in air tight container for further use.

3.2 Green Synthesis of Nanoparticle:

Take 10mg of silver nitrate in 100ml DI-water & set temperature to 80°-90°. The Fresh Flower of *Magnolia champaca* (L.)Baill. ex Pierre and Whole Plant of *Origanum majorana* L extract has been mixed drop wise until the color develops to

light brownish orange. The plant Fresh Flower of *Magnolia champaca* (L.) Baill. ex Pierre and Whole Plant of *Origanum majorana* L extract has been used as a reducing agent and capping agent. The mechanism involved in the synthesis of AgNPs is reduction. The product has been centrifuged using centrifugal apparatus. AgNPs are stored in an air tight container.

3.3 Evaluation of AGNP's:

Specific methods must first confirm the formation of AgNPs before they can be used for their intended application. The most basic method to monitor AgNPs production by visually observing the change in the color of the solution from yellow to brown. A spectrophotometer can further confirm the tracking process and detect nanoparticle peaks in the visible area of the UV-vis spectrum at a wavelength between 400 and 450 nm. Other techniques, including SEM can be used to investigate the size, morphology, dispersion,

and composition of nanoparticles. Moreover, FTIR spectroscopy can help track biomolecules that influence nanoparticle formation and stability.

3.4 Phytochemical Test:

The extracts of Fresh Flower of *Magnolia champaca* (L.) Baill. ex Pierre and Whole Plant of *Origanum majorana* L were subjected to the following preliminary phytochemical analysis.

3.5 Experimental Method:

Thirty male Wistar albino rats were randomly divided into five groups (n=5 per group) to evaluate the neuroprotective potential of NP-MCOM, a green-synthesized silver nanoparticle formulation prepared using the flower extract of *Magnolia champaca* and the whole plant extract of *Origanum majorana*, in a haloperidol-induced Parkinson's disease model.

- ✚ **Group I – Control:** Received only normal saline via oral gavage once daily for 21 days.
- ✚ **Group II – Negative Control:** Received haloperidol (1 mg/kg, i.p.) once daily for 21 days, without any treatment.
- ✚ **Group III – Standard Treatment:** Received levodopa (100 mg/kg) and carbidopa (25 mg/kg) via intraperitoneal injection one hour before haloperidol (1 mg/kg, i.p.) for 21 days.
- ✚ **Group IV – NP- MCOM Low Dose:** Treated with NP-MCOM (200 mg/kg, p.o.) one hour prior to haloperidol (1 mg/kg, i.p.) for 21 days.
- ✚ **Group V – NP- MCOM High Dose:** Treated with NP-MCOM (400 mg/kg, p.o.) one hour prior to haloperidol (1 mg/kg, i.p.) for 21 days.

This protocol was designed to investigate the dose-dependent prophylactic efficacy of NP-MCOM

(green-synthesized silver nanoparticle formulated by flower extract of *Magnolia champaca* and the whole plant extract of *Origanum majorana* in alleviating Parkinsonian symptoms induced by haloperidol in Wistar rats, using standard therapy as a positive control. Certainly, Here's the entire experimental procedure rewritten into a clear, single paragraph format, suitable for inclusion in your Materials and Methods section: Thirty male Wistar albino rats were randomly divided into five groups (n=5 per group) to evaluate the neuroprotective effects of NP-MCOM, a green-synthesized silver nanoparticle formulation derived from the flower extract of *Magnolia champaca* and the whole plant extract of *Origanum majorana*. Group I (Control) received normal saline via oral gavage once daily for 21 days. Group II (Negative Control) was administered haloperidol (1 mg/kg, intraperitoneally) once daily for 21 days without any co-treatment. Group III (Standard) received levodopa (100 mg/kg) and carbidopa (25 mg/kg) intraperitoneally, one hour prior to haloperidol (1 mg/kg, i.p.), for 21 days. Group IV and Group V received NP-MCOM orally at doses of 200 mg/kg and 400 mg/kg, respectively, one hour prior to haloperidol administration (1 mg/kg, i.p.) daily for 21 consecutive days. This protocol was designed to assess the dose-dependent prophylactic potential of NP-MCOM in mitigating haloperidol-induced Parkinsonian symptoms in Wistar rats by comparing it with standard dopaminergic therapy.

3.6 Pharmacological Activity:

- ✚ Catalepsy Bar Test
- ✚ Tardive Dyskinesia (TD) Test
- ✚ Open Field Test
- ✚ Rotarod



✚ Wire Hang Test

✚ Glutathione

✚ Pole Test

✚ Histopathological Study

✚ Beam Walking Test

3. RESULTS AND DISCUSSION:

✚ Assessment Of Serum Superoxide Dimutase

3.1 Preparation Of Nanoparticle:

✚ Assessment of Serum Catalase



Fig: 4 Nanoparticle

3.2 Preliminary Phytochemical Screening:

Table: 1 Preliminary Phyto-chemical Screening

S. No	Constituents	Extract
1.	Alkaloids	+
2.	Carbohydrates	
3.	Protein	+
4.	Terpinoids	
5.	Phenols	+
6.	Tannins	+
7.	Flavonoids	+
9.	Glycosides	+
10.	Saponins	
11.	Fat and Oil	+

4.3 Evaluation of Nanoparticles:

4.3.1 Particle Size & Zeta Potential:

Table: 2 Evaluation of Nanoparticles

S. No	Parameters	Report
1.	Particle Size	431
2.	Encapsulation efficacy (%)	60.9
3.	Drug content (%)	93.5 %
4.	Zeta Potential	-18.3

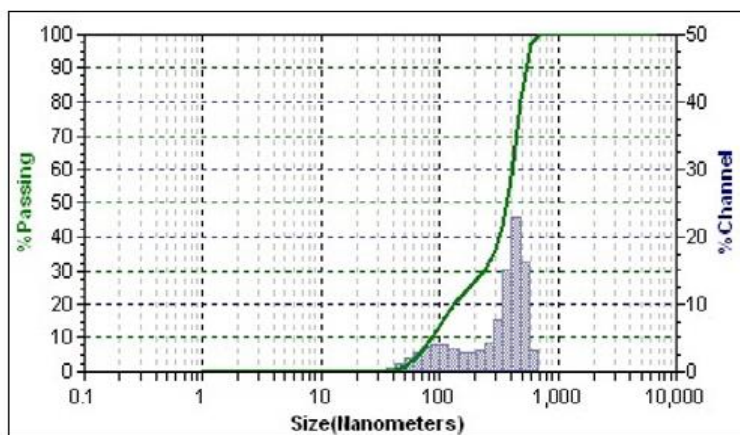


Fig: 5 Particle Size

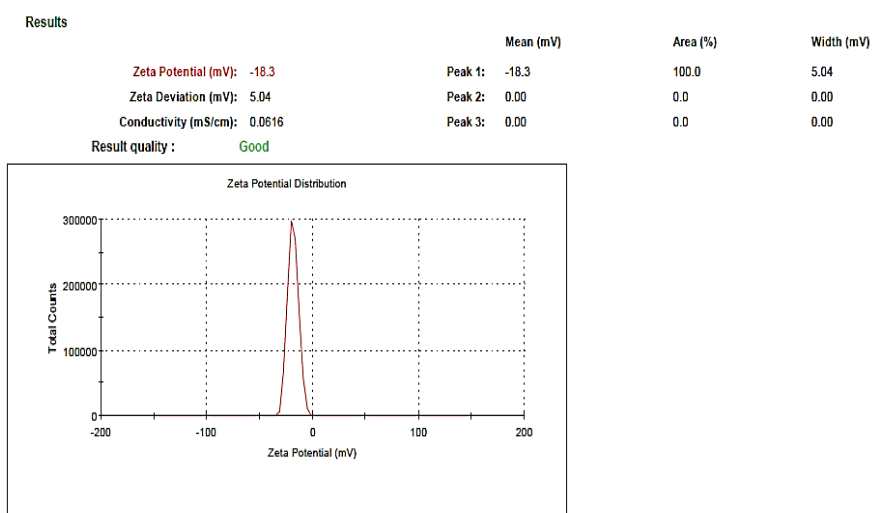


Fig: 6 Zeta Potential

4.3.2 UV Analysis:

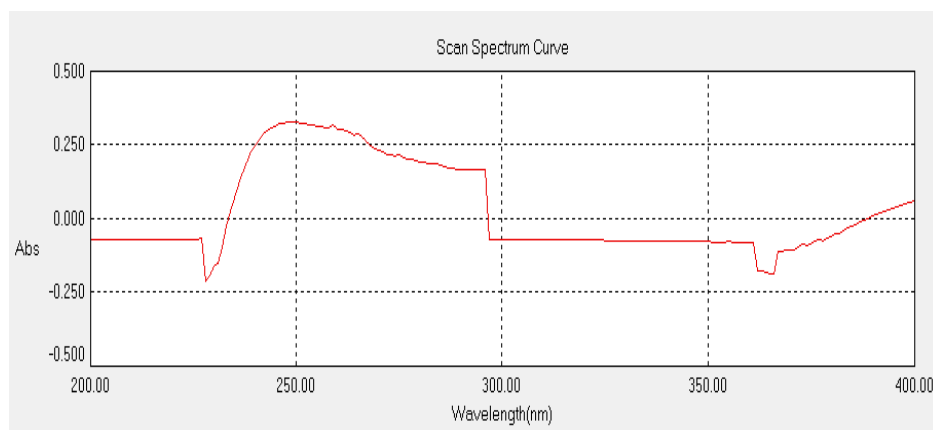


Fig: 7 UV Spectrum Analysis

4.3.3 Surface Morphological Analysis:



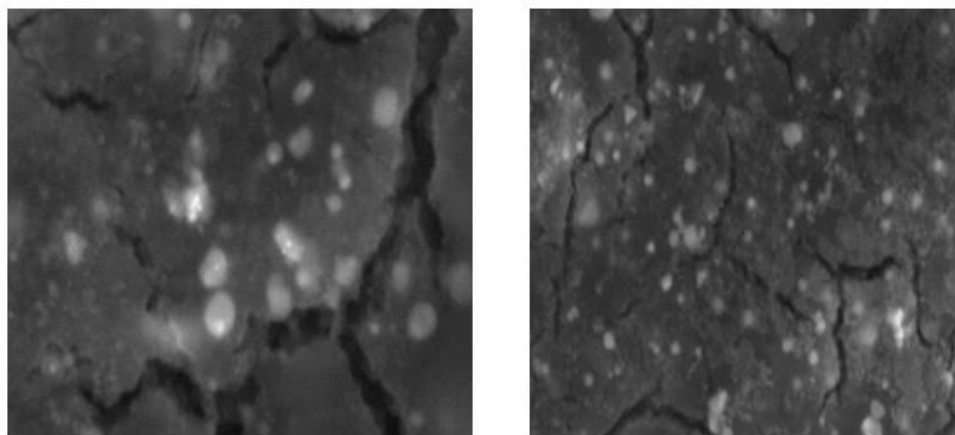


Fig: 8 SEM Analysis

4.3.4 In-Vitro Drug Release Studies:

Table: 3 In-Vitro Drug Release Studies

Time (Hr)	cumulative % drug released	% drug remaining	Square root time	log Cumu % drug remaining	log time	log Cumu % drug released	% Drug released	Cube Root of % drug Remaining(Wt)	Wo-Wt
0	0	100	0.000	2.000	0.000	0.000	100	4.642	0.000
0.5	5.31	94.69	0.707	1.976	-0.301	0.725	5.31	4.558	0.084
1	8.67	91.33	1.000	1.961	0.000	0.938	3.36	4.503	0.139
2	13.65	86.35	1.414	1.936	0.301	1.135	4.98	4.420	0.222
4	29.11	70.89	2.000	1.851	0.602	1.464	15.46	4.139	0.503
6	38.21	61.79	2.449	1.791	0.778	1.582	9.1	3.953	0.689
8	55.32	44.68	2.828	1.650	0.903	1.743	17.11	3.548	1.094
12	73.71	26.29	3.464	1.420	1.079	1.868	18.39	2.973	1.669
14	86.35	13.65	3.742	1.135	1.146	1.936	12.64	2.390	2.252

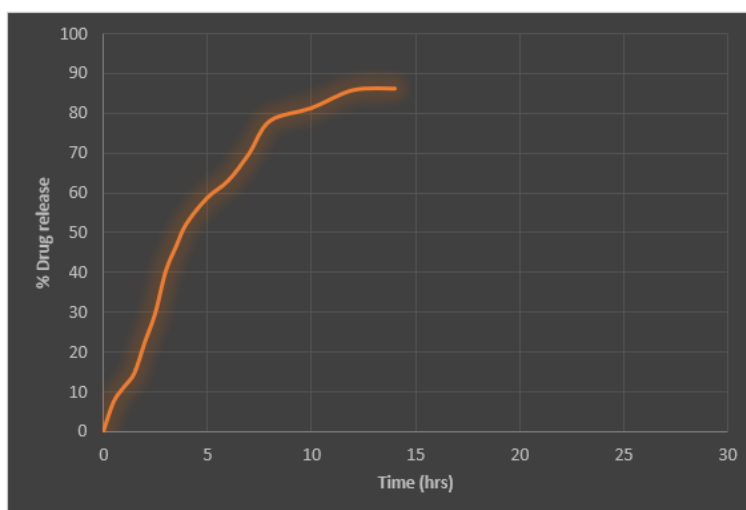


Fig: 9 In-Vitro Drug Release Studies

4.4 Pharmacological Activity:

4.4.1 Effect Of NP-MCOM On Catalepsy Bar Test & Catalepsy Score:

Table: 4 Effect Of NP-MCOM On Catalepsy Bar Test & Catalepsy Score

S.NO	GROUPS	CATALEPSY BAR TEST			CATALEPSY SCORE		
		DAY 07	DAY 14	DAY 21	DAY 07	DAY 14	DAY 21
1	CONTROL	4.46 ± 0.05	3.64 ± 0.05	4.28 ± 0.08	0	0	0
2	NEGATIVE CONTROL	78 ± 5.22 a****	87.4 ± 1.86 a****	107.8 ± 6.11 a****	2.8 ± 0.2 a****	3.4 ± 0.24 a****	4 ± 0 a****
3	STANDARD (L-DOPA)	28.8 ± 1.42 a****b****	15 ± 0.83 a**b****	9 ± 0.70 a ^{ns} b****	1.4 ± 0.24 a****b****	1 ± 0 a****b****	0.4 ± 0.24 a****b****
4	LOW DOSE (NP-MCOM 200mg/kg)	59.8 ± 1.85 a****b****c*** *	43 ± 1.22 a****b****c*** *	30.8 ± 1.49 a****b****c****	2.4 ± 0.24 a****b ^{ns} c****	2 ± 0 a****b****c** **	1.6 ± 0.24 a****b****c*** *
5	HIGH DOSE (NP-MCOM 400mg/kg)	34 ± 0.70 a****b****c ^{ns}	27.4 ± 1.50 a****b****c**	13.2 ± 0.86 a ^{ns} b****c ^{ns}	2 ± 0 a****b****c ^{ns}	1.4 ± 0.24 a****b****c** **	1 ± 0 a****b****c*** *

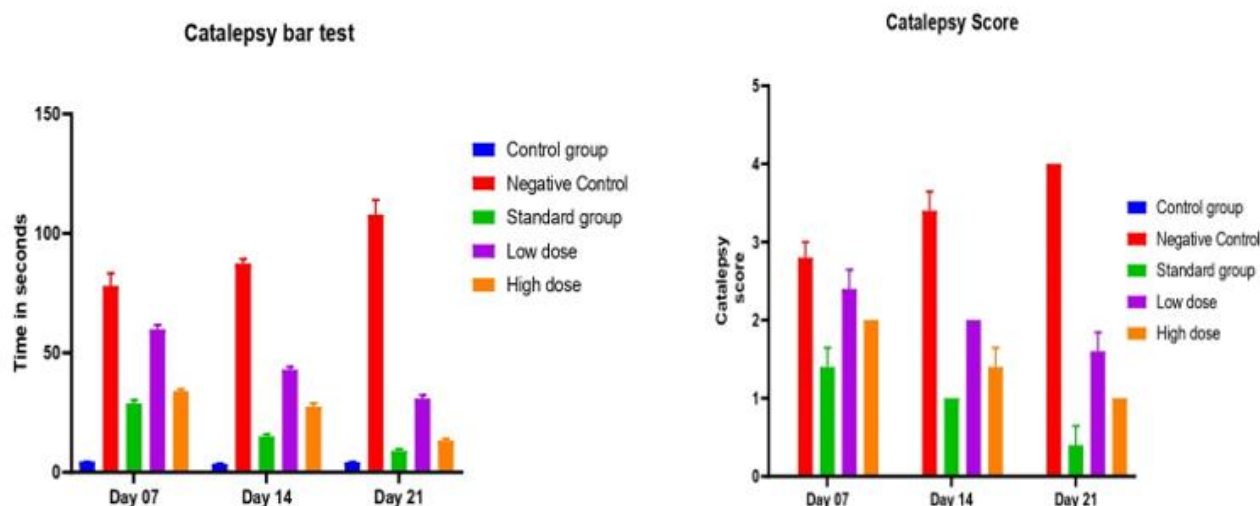


Fig: 10 Effect Of NP-MCOM On Catalepsy Bar Test & Catalepsy Score

4.4.2 Of NP-MCOM On Tardive Dyskinesia Test (Oro-Facial Assessment) & Rota Rod Test:

Table: 5 Effect Of NP-MCOM On Tardive Dyskinesia Test (Oro-Facial Assessment) & Rota Rod Test

S.NO	GROUPS	TARDIVE DYSKINESIA TEST			ROTA ROD TEST		
		DAY 07	DAY 14	DAY 21	DAY 07	DAY 14	DAY 21
1	CONTROL	8.4 ± 0.50	10 ± 0.70	10.4 ± 0.92	212.4 ± 3.50	213.4 ± 3.6	210.2 ± 4.97
2	NEGATIVE CONTROL	95.8 ± 0.73 a****	109.6 ± 1.32 a****	125.8 ± 1.68 a****	105.4 ± 1.63 a****	83.8 ± 1.46 a****	62.4 ± 0.92 a****
3	STANDARD (L-DOPA)	52.6 ± 1.02 a****b****	33 ± 1.09 a****b****	19.6 ± 1.43 a ^{ns} b****	162.8 ± 1.24 a****b****	174.6 ± 1.50 a****b****	210 ± 2.46 a ^{ns} b****
4	LOW DOSE (NP-MCOM 200mg/kg)	76 ± 0.70 a****b ^{ns} c*** *	65 ± 0.83 a****b****c*** *	54.6 ± 0.50 a****b****c**** *	78 ± 0.83 a****b****c****	129.6 ± 3.26 a****b****c****	158.2 ± 1.98 a****b****c*** *

5	HIGH DOSE (NP-MCOM 400mg/kg)	58.8 ± 1.15 a****b**c****	46.6 ± 0.92 a****b****c*** *	31.6 ± 1.20 a***b****c ^{ns}	160 ± 1.70 a****b****c ^{ns}	181.8 ± 1.06 a****b****c ^{ns}	193.2 ± 1.06 a****b****c*** *
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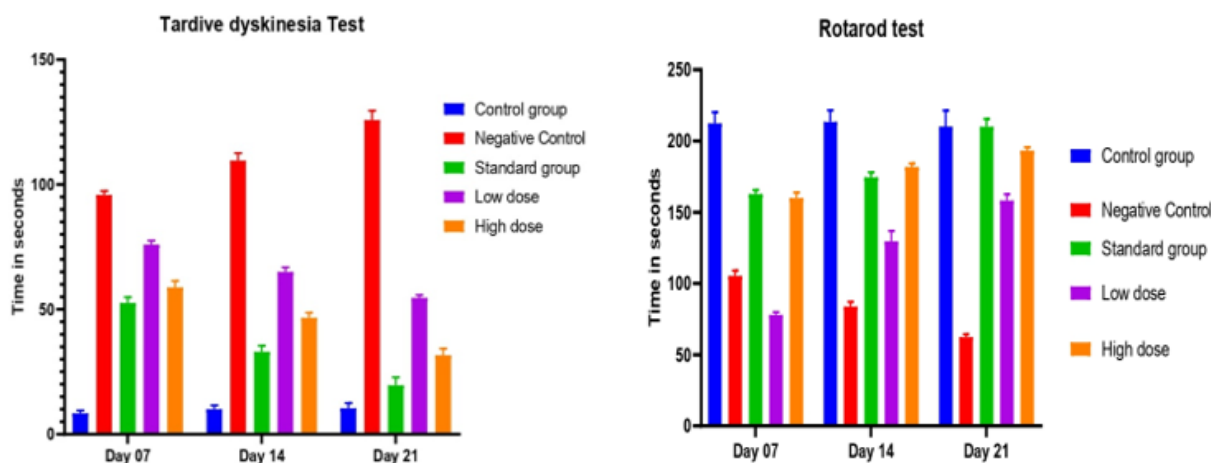


Fig: 11 Effect Of NP-MCOM On Tardive Dyskinesia Test (Oro-Facial Assessment) & Rota Rod Test

4.4.3 Effect Of NP-MCOM On Wire Hang Test & Pole Test (T- Turn):

Table: 6 Effect Of NP-MCOM On Wire Hang Test & Pole Test

S.NO	GROUPS	WIRE HANG TEST			POLE TEST		
		DAY 07	DAY 14	DAY 21	DAY 07	DAY 14	DAY 21
1	CONTROL	119.8 ± 1.31	124 ± 0.89	126.8 ± 0.58	1.84 ± 0.13	2.28 ± 0.07	1.58 ± 0.05
2	NEGATIVE CONTROL	58.6 ± 1.16 a****	43.4 ± 1.20 a****	23 ± 1.78 a****	8 ± 0.31 a****	10.8 ± 0.86 a****	16.2 ± 0.58 a****
3	STANDARD (L-DOPA)	101.6 ± 1.20 a****b****	113 ± 1.64 a****b****	121.4 ± 1.12 a ^{ns} b****	3.9 ± 1.02 a ^{ns} b****	2.4 ± 0.18 a ^{ns} b****	1.2 ± 0.12 a ^{ns} b****
4	LOW DOSE (NP-MCOM 200mg/kg)	72.2 ± 0.96 a****b****c*** *	92.4 ± 1.77 a****b****c*** *	101.4 ± 1.12 a****b****c****	6.4 ± 0.24 a****b*c****	5 ± 0.31 a****b****c****	4.2 ± 0.37 a****b****c*** *
5	HIGH DOSE (NP-MCOM 400mg/kg)	100.8 ± 2.08 a****b****c ^{ns}	113 ± 1.64 a****b****c ^{ns}	121.8 ± 1.06 a ^{ns} b****c ^{ns}	3.8 ± 0.37 a****b****c ^{ns}	3 ± 0.31 a ^{ns} b****c ^{ns}	2.4 ± 0.24 a ^{ns} b****c****

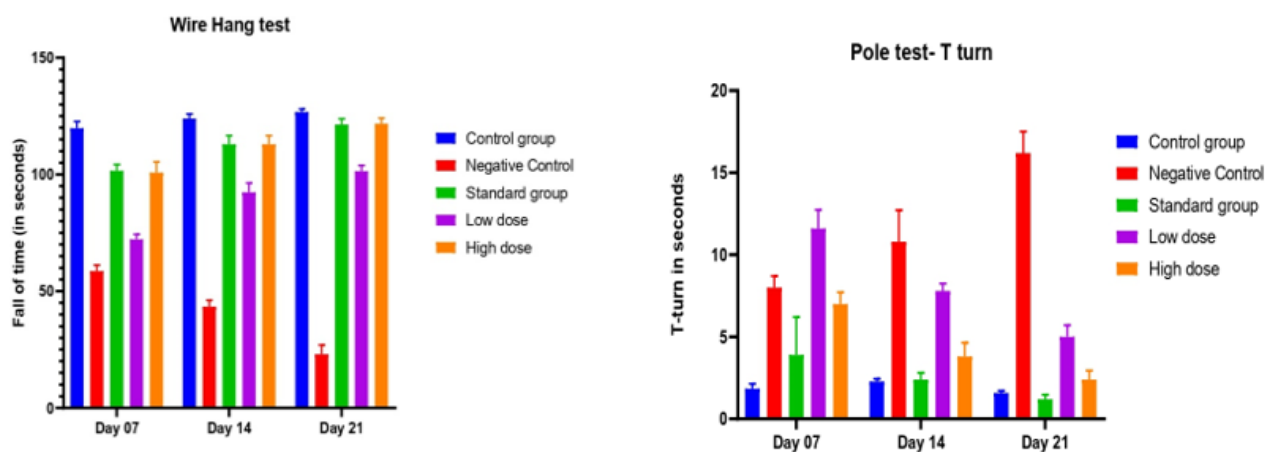


Fig: 12 Effect Of NP-MCOM On Wire Hang Test & Pole Test

4.4.4 Effect Of NP-MCOM On Pole Test (T- Total) & Beam Walking Test:

Table: 7 Effect Of NP-MCOM On Pole Test (T- Total) & Beam Walking Test C

S.NO	GROUPS	POLE TEST			BEAM WALKING TEST		
		DAY 07	DAY 14	DAY 21	DAY 07	DAY 14	DAY 21
1	CONTROL	5.2 ± 0.58	4.6 ± 0.24	4.8 ± 0.37	10.4 ± 0.50	10.16 ± 0.49	11.6 ± 0.50
2	NEGATIVE CONTROL	14 ± 0.44 a ****	17.4 ± 0.87 a ****	25.6 ± 0.67 a ^{ns}	48.6 ± 1.02 a ****	63.2 ± 0.96 a ****	78.6 ± 1.02 a ****
3	STANDARD (L-DOPA)	7.6 ± 0.24 a ** b ****	5.4 ± 0.24 a ^{ns} b ****	3.8 ± 0.37 a **** b ****	21.6 ± 0.50 a **** b ****	14.4 ± 0.50 a **** b ****	9.8 ± 0.37 a ^{ns} b ****
4	LOW DOSE (NP-MCOM 200mg/kg)	10.4 ± 0.24 a **** b **** c ** *	8.8 ± 0.20 a **** b **** c ** *	6.8 ± 0.20 a ** b **** c ****	35.4 ± 0.81 a **** b **** c ** *	24.6 ± 0.50 a **** b **** c ****	15.8 ± 0.37 a **** b **** c ** *
5	HIGH DOSE (NP-MCOM 400mg/kg)	7.4 ± 0.24 a ** b **** c ^{ns}	5.6 ± 0.24 a ^{ns} b **** c ^{ns}	4.6 ± 0.24 a ^{ns} b **** c ^{ns}	25.4 ± 0.24 a **** b **** c ** *	14.8 ± 0.37 a **** b **** c ^{ns}	11.6 ± 0.50 a ^{ns} b **** c ^{ns}

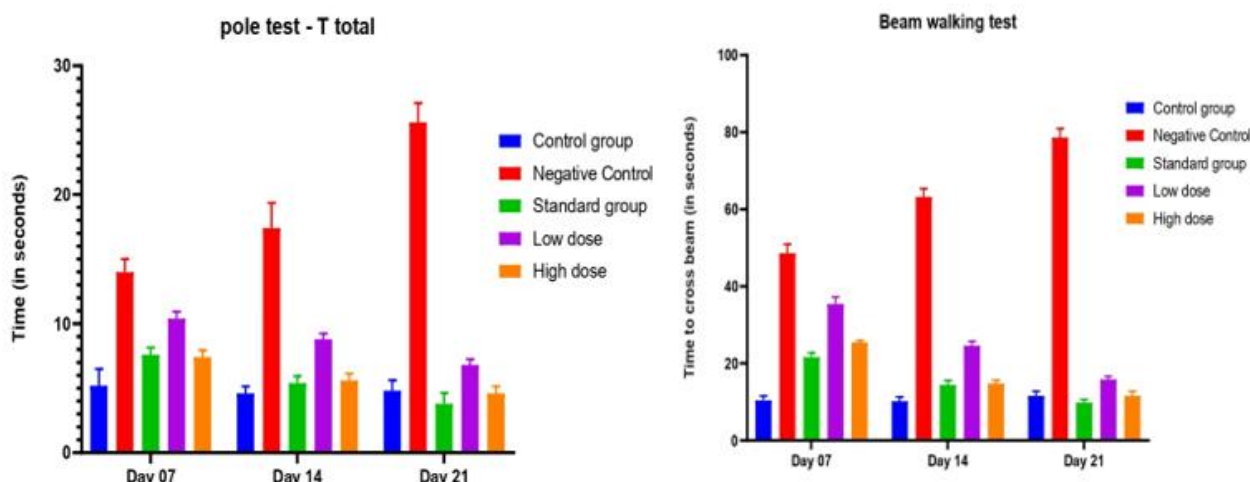
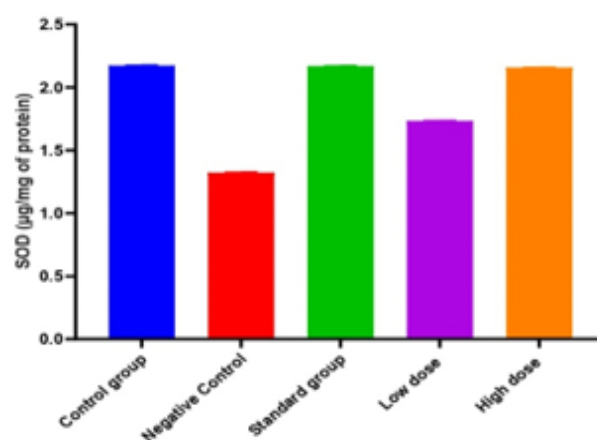
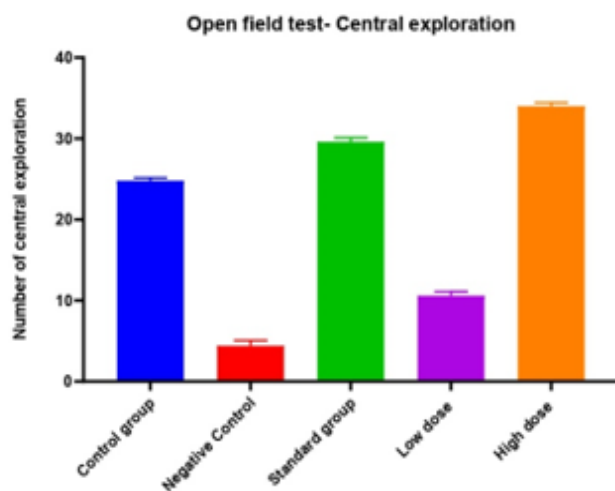


Fig: 13 Effect Of NP-MCOM On Pole Test (T- Total) & Beam Walking Test

4.4.5 Effect Of NP-MCOM On Open Field Test (Central Exploration), Superoxide Dimutase (SOD), Catalase (CAT) & Glutathione (GSH):

Table: 8 Effect Of NP-MCOM On Open Field Test (Central Exploration), Superoxide Dimutase (SOD), Catalase (CAT) & Glutathione (GSH)

S.NO	GROUPS	OPEN FIELD TEST (CENTRAL EXPLORATION)	SUPEROXIDE DIMUTASE (SOD)	CATALASE (CAT)	GLUTATHIONE (GSH)
1	CONTROL	24.80 ± 0.37	2.173 ± 0.25	33.57 ± 0.003	13.34 ± 0.05
2	NEGATIVE CONTROL	4.400 ± 0.67 a****	1.324 ± 0.38 a****	22.44 ± 0.007 a****	8.536 ± 0.05 a****b****c****
3	STANDARD (L-DOPA)	29.60 ± 0.50 a****b****	2.169 ± 0.16 a****b****	31.54 ± 0.007 a****b****	12.85 ± 0.05 a****b****c****
4	LOW DOSE (NP-MCOM 200mg/kg)	10.60 ± 0.50 a****b****c****	1.735 ± 0.24 a****b****c****	25.09 ± 0.008 a****b****c****	10.04 ± 0.01 a****b****c****
5	HIGH DOSE (NP-MCOM 400mg/kg)	34.00 ± 0.44 a****b****c****	2.158 ± 0.18 a****b****c****	29.65 ± 0.005 a****b****c****	12.25 ± 0.07 a****b****c****



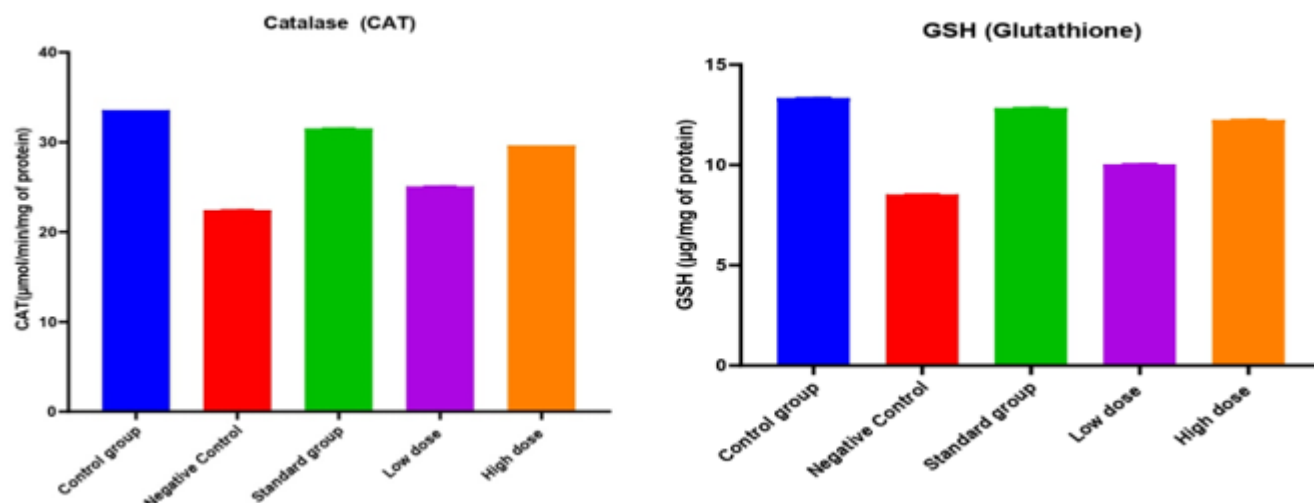


Fig: 14 Effect Of NP-MCOM On Open Field Test (Central Exploration), Superoxide Dimutase (SOD), Catalase (CAT) & Glutathione (GSH)

4.5 Histopathology:

Group I (Control Group): The normal control group (Group I) exhibited an intact histological architecture with well-preserved cellular morphology and tissue organization. No signs of inflammation, degeneration, or necrosis were observed, indicating healthy and uncompromised neural tissue integrity. These findings confirm the suitability of the control group as a baseline for comparison in this study. Overall, the histopathology validates that no adverse effects occurred in untreated healthy animals throughout the experimental period.

Group II (Disease Model – Haloperidol Induced Group): The disease control group showed marked neuronal degeneration with disrupted cellular architecture. Mild congestion in blood vessels and slight hemorrhage were evident, indicating vascular impairment. Additionally, neurofibrillary tangles and plaques were observed in the brain tissues, reflecting hallmark pathological features of neurodegeneration induced by haloperidol (1 mg/kg).

Group III (Standard Group – Levodopa (100 Mg/Kg) + Carbidopa (25 Mg/Kg)): The standard treatment group exhibited significant restoration of healthy, active neurons accompanied by improved vascular nourishment and enhanced blood supply to the brain tissue. Enhanced microvascular integrity contributed to reduced neuroinflammation and facilitated regeneration.

Group IV (NP-MCOM 200 mg/kg): This group showed notable neuronal regeneration with partial restoration of normal neuronal morphology and reduced signs of degeneration. Mild vascular congestion was present but less pronounced than in the disease group, indicating improved microcirculation. Gliosis was also attenuated, suggesting reduced neuroinflammation.

Group V (NP-MCOM 400 mg/kg): Group V exhibited significant histopathological recovery with nearly intact neuronal architecture and minimal degeneration. Vascular congestion and hemorrhagic changes were substantially diminished, reflecting enhanced cerebral perfusion. Marked reduction in gliosis indicated

effective suppression of neuroinflammatory responses.

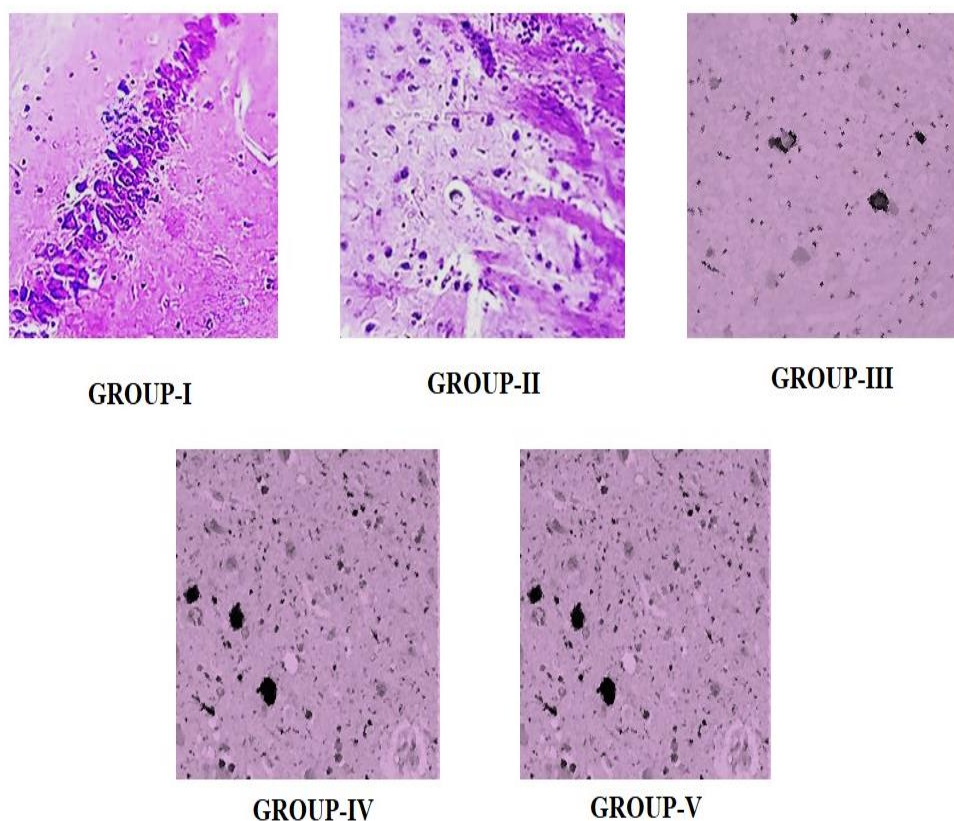


Fig: 15 Histopathology

4. CONCLUSION:

In conclusion, our findings suggest that NP-MCOM holds considerable promise as a neuroprotective agent capable of attenuating both the symptomatic and molecular hallmarks of Parkinson's disease. By significantly improving motor function, enhancing antioxidant defenses, and potentially reducing progressive neurodegeneration, NP-MCOM could contribute substantially to improving patient outcomes and slowing disease progression. These preclinical results provide a strong rationale for further detailed mechanistic studies and clinical trials to evaluate the safety, efficacy, and pharmacokinetics of NP-MCOM in humans.

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