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Chemistry of *Celastrus paniculatus* Seed Oil role in the Management of Parkinson's Disease

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ABSTRACT

Parkinson's disease (PD) is a progressive neurodegenerative disorder characterized by tremors, rigidity, bradykinesia, and postural instability, primarily resulting from dopaminergic neuronal loss and oxidative stress in the substantia nigra. The present study investigates the neuroprotective and antioxidant potential of *Celastrus paniculatus* seed oil (CPS oil) in experimental models of PD. Gas chromatography–mass spectrometry (GC–MS) analysis revealed the presence of bioactive phytoconstituents such as sesquiterpenoids, alkaloids, phenolic triterpenoids, and essential fatty acids, which may underlie its pharmacological effects. CPS oil exhibited moderate DPPH free radical scavenging activity compared to ascorbic acid, indicating significant intrinsic antioxidant potential. Pharmacological evaluations, including assessments of body weight, grip strength (rotarod test), locomotor activity (actophotometer), and skilled motor coordination (staircase test), demonstrated significant improvement in CPS oil-treated groups relative to the disease control. Biochemical assays further revealed elevated levels of antioxidant enzymes—superoxide dismutase (SOD), catalase (CAT), and reduced glutathione (GSH)—alongside reduced lipid peroxidation (LPO), confirming oxidative stress attenuation. These findings suggest that CPS oil confers neuroprotection by enhancing endogenous antioxidant defense and motor function, thereby scientifically validating the traditional Ayurvedic use of *Celastrus paniculatus* as a nervine tonic and highlights its potential as a natural therapeutic candidate for managing Parkinson's disease.

Key words: *Celastrus paniculatus*, Parkinson's disease, antioxidant, GC–MS, oxidative stress, neuroprotection, dopaminergic neurons.

INTRODUCTION

Parkinson's disease (PD) is a progressive neurodegenerative disorder characterized by tremor, rigidity, bradykinesia, and gait disturbances. These motor impairments primarily result from the gradual loss of dopaminergic neurons in the substantia nigra (SN). Current therapeutic strategies for PD largely focus on dopamine replacement therapy, which offers symptomatic relief and improves quality of

life. However, long-term administration often leads to motor complications that are challenging to manage. In addition to motor dysfunction, patients experience several non-motor symptoms such as olfactory loss, sleep disturbances, autonomic dysfunction, and cognitive impairment, which are generally resistant to available pharmacological treatments and contribute significantly to disability. This highlights the urgent need for neuroprotective strategies capable of delaying disease progression and mitigating the onset of these complications[1].

Neuropathological studies have revealed that dopaminergic neuronal loss is part of a broader neurodegenerative process that begins in the brainstem and gradually extends to cortical and subcortical regions. Thus, the course of PD spans several decades, with motor symptoms appearing only at intermediate stages. This slow progression provides a critical therapeutic window during which interventions targeting oxidative stress, mitochondrial dysfunction, and neuroinflammation could prevent or delay irreversible neuronal loss. Despite advances in symptomatic treatment, a major challenge remains—the lack of an effective neuroprotective therapy that can be implemented early in the disease course[2].

Celastrus paniculatus Willd. (Celastraceae), commonly known as Jyotishmati, Malkangni, or Kangani, is classified in Ayurveda as a Medhya Rasayana (nervine tonic). Traditionally, its seeds and seed oil have been used for enhancing memory and cognitive function. Phytochemical investigations have identified numerous bioactive constituents in the seeds and oil, including sesquiterpenoid polyalcohols and esters (malkanguniol, malkangunin, polyalcohol A–D, celapnin), alkaloids (paniculatine, celastrine), phenolic triterpenoids (celastrol, paniculatadiol), fatty acids (oleic, linoleic, linolenic, palmitic, stearic, lignoceric acid), and agarofuran derivatives. Experimental studies have reported that *Celastrus paniculatus* possesses neuroprotective, antioxidant, anti-inflammatory, anxiolytic, and memory-enhancing properties, attributed largely to its free-radical-scavenging and mitochondrial-protective mechanisms[3].

In the present study, CPS oil was evaluated for its neuroprotective and antioxidant potential in experimental models of PD. The study aimed to (i) characterize the phytochemical profile of CPS oil using gas chromatography–mass spectrometry (GC–MS), (ii) assess its antioxidant activity in vitro, and (iii) evaluate its neuroprotective effects through behavioral and biochemical assessments in PD-induced rats. This study provides preclinical evidence supporting the traditional Ayurvedic use of CPS oil and its potential role as a natural neuroprotective agent for the management of Parkinson's disease.

MATERIALS AND METHODS

Materials

Seeds of *Celastrus paniculatus* were procured from a local herbal market in Lucknow, India. The seeds were authenticated and the oil was extracted by cold pressing. All other reagents and chemicals used were of analytical grade and procured from standard suppliers.

Experimental Animals

Male Wistar rats (220–250 g) were used for the study. Animals were obtained from a CCSEA-registered breeder and acclimatized for one week under standard laboratory conditions (temperature: 22 ± 2 °C; relative humidity: 55–65%; 12 h light/dark cycle). Standard pellet diet and water were provided ad libitum. All experimental protocols were approved by the Institutional Animal Ethics Committee (IAEC) of Bilwal Medchem and Research Laboratory Pvt. Ltd., Jaipur (Approval Ref. No.: BMRL/IAEC/2024-37). All behavioral experiments were conducted during the light phase (09:00–17:00 h)[4].

Gas Chromatography–Mass Spectrometry (GC–MS) Analysis

The chemical composition of *C. paniculatus* seed oil was determined using GC–MS (Agilent 7890A GC system coupled with a 5975C mass selective detector). Separation was performed on a DB-5MS capillary column (30 m × 0.25 mm × 0.25 µm film thickness). The sample was diluted with n-hexane (1:10 v/v), and 1 µL of sample was injected in split mode using helium as the carrier gas at a constant flow rate of 1.0 mL/min. The injector temperature was maintained at 250 °C. The oven temperature was programmed as follows: initial 60 °C (held for 2 min), increased to 200 °C at 3 °C/min, and then to 300 °C at 5 °C/min, with a final hold of 10 min. The mass spectrometer operated in electron impact (EI) mode at 70 eV with an ion source temperature

of 230 °C and a mass scan range of m/z 40–650. Chromatographic peaks were identified by comparing their mass spectra with those in the NIST and Wiley libraries. A total of 109 peaks were detected, representing diverse phytoconstituent classes, including sesquiterpenes, alkaloids, triterpenoids, and fatty acids. The relative abundance (%) of each component was calculated from peak area normalization. Representative chromatograms and corresponding mass spectra are presented in Figures 1–2, and major compounds are listed in Table 1 [5–8].

RESULTS AND DISCUSSION

Pharmacological Studies

Body weight was recorded for each animal at the beginning and at the end of the experimental period to assess general health status and the effect of CPS oil treatment. The percentage change in body weight was calculated using the formula:

Percentage change in body weight = [(Final weight – Initial weight) / Initial weight] × 100

Table 1: Antioxidant activity

Conc. (μg/mL)	% inhibition of AS	% inhibition of CPS OIL
100	90.69	46.04
200	91.93	52.34
300	93.02	60.44
400	95.19	70.85
500	96.74	80.93
IC_{50}	5.73	26.44

Table 2: Assessment of body weight

	Initial Body Weight Parameters								
	I	I	I	I	I	I	MEAN	SD	SE
NC	175	178	182	179	184	170	178.00	5.02	2.05
DC	171	173	178	185	170	172	174.83	5.71	2.33
SD	180	185	187	190	195	180	186.17	5.85	2.39
TLD	190	175	179	188	180	176	181.33	6.25	2.55
THD	180	173	181	187	170	171	177.00	6.72	2.74
	Final Body Weight Parameters								
	F	F	F	F	F	F	MEAN	SD	SE
NC	186	187	179	171	185	180	181.33	6.02	2.46
DC	145	148	140	135	150	155	145.50	7.18	2.93
SD	175	160	175	180	175	176	173.50	6.89	2.81
TLD	169	165	170	179	160	175	169.67	6.80	2.78
THD	174	176	172	178	168	165	172.17	4.92	2.01

Table 3: Assessment of Muscle Coordination Using the Rota-Rod Apparatus

	ROTA ROD						MEAN	SD	SE
NC	164	161	151	157	162	167	160.33	5.65	2.30
DC	64	52	56	61	72	60	60.83	6.88	2.81
SD	165	154	150	156	160	168	158.83	6.82	2.79
TLD	122	129	138	126	133	135	130.50	5.96	2.43
THD	156	148	141	152	147	160	150.67	6.80	2.78

Table 4: Locomotor activity

	ACTOPHOTOMETER						MEAN	SD	SE
NC	129	119	122	129	125	136	126.67	6.02	2.46
DC	40	34	38	42	35	48	39.50	5.13	2.09
SD	118	122	128	125	120	124	122.83	3.60	1.47
TLD	100	97	89	99	85	98	94.67	6.15	2.51
THD	116	121	122	120	112	118	118.17	3.71	1.51

Table 5.1: Test of muscle coordination using a staircase

	Staircase (15 min)								
	I	I	I	I	I	I	MEAN	SD	SE
NC	36	39	40	45	47	48	42.50	4.85	1.98
DC	110	108	106	112	110	104	108.33	2.94	1.20
SD	45	44	50	48	53	46	47.67	3.39	1.38
TLD	58	54	59	56	50	58	55.83	3.37	1.38
THD	50	49	53	50	58	44	50.67	4.63	1.89

Table 5.2: Responses of different groups in Initiation Test

	Staircase (15 min)								
	F	F	F	F	F	F	MEAN	SD	SE
NC	39	49	49	48	49	50	47.33	4.13	1.69
DC	113	111	115	118	111	108	112.67	3.50	1.43
SD	60	70	64	61	62	70	64.50	4.46	1.82
TLD	70	68	70	72	76	73	71.50	2.81	1.15
THD	62	65	69	70	71	71	68.00	3.69	1.51

As shown in Table 2 and Figure 3, the disease control group exhibited a significant reduction in body weight compared to the normal control group, indicating systemic stress and dopaminergic neuronal damage associated with Parkinsonian pathology. In contrast, treatment with CPS oil produced a dose-dependent improvement in body weight, reflecting enhanced metabolic and neuromuscular function. The improvement in weight gain among CPS oil-treated groups suggests overall physiological recovery and neuroprotection, comparable to the standard treatment group[9].

Grip Strength

Grip strength was assessed to evaluate muscle tone, balance, and motor coordination, key indicators of neuromuscular integrity affected in Parkinson's disease. The test was performed using a Rota-Rod apparatus, following the method described by Dunham and Miya[10]. Each rat was placed on a rotating rod (20 rpm), and the time (in seconds) that the animal remained on the rod without falling was recorded. Prior to testing, animals were trained for three consecutive days to minimize variability. The mean latency to fall was calculated for each group. Data presented in Table 3 and Figure 4 demonstrate a marked reduction in grip strength and fall latency in the disease control group, indicating severe motor impairment. Conversely, animals treated with CPS oil exhibited a significant, dose-dependent improvement in grip strength and retention time on the rotarod compared to the disease control. This improvement reflects enhanced neuromuscular coordination, balance, and resistance to fatigue. The higher dose of CPS oil (e.g., 400 mg/kg) produced effects comparable to those observed with the standard

Table 6: Assay for antioxidant enzymes in anti-parkinson's disease Various antioxidant measurements in the context of antiparkinsonian effect SOD, CAT, GSH, LPO

			SOD				MEAN	SD	SE
NC	60	63	65	67	62	58	62.50	3.27	1.34
DC	21	23	20	19	18	25	21.00	2.61	1.06
SD	50	55	49	48	53	51	51.00	2.61	1.06
TLD	45	38	39	40	42	43	41.17	2.64	1.08
THD	46	52	51	48	47	42	47.67	3.61	1.48
			CAT				**MEAN**	**SD**	**SE**
NC	58	63	62	58	57	63	60.17	2.79	1.14
DC	30	28	26	29	25	29	27.83	1.94	0.79
SD	51	49	51	53	50	48	50.33	1.75	0.71
TLD	35	37	35	39	42	39	37.83	2.71	1.11
THD	48	45	47	46	45	42	45.50	2.07	0.85
			GSH				**MEAN**	**SD**	**SE**
NC	55	58	54	52	57	58	55.67	2.42	0.99
DC	20	21	25	23	20	26	22.50	2.59	1.06
SD	47	49	46	45	40	48	45.83	3.19	1.30
TLD	39	37	30	32	35	38	35.17	3.54	1.45
THD	42	46	41	43	44	48	44.00	2.61	1.06
			LPO				**MEAN**	**SD**	**SE**
NC	102	108	101	109	103	107	105.00	3.41	1.39
DC	240	238	242	245	237	234	239.33	3.88	1.58
SD	175	180	182	170	181	176	177.33	4.55	1.86
TLD	200	206	204	198	197	204	201.50	3.67	1.50
THD	197	196	186	194	195	185	192.17	5.27	2.15

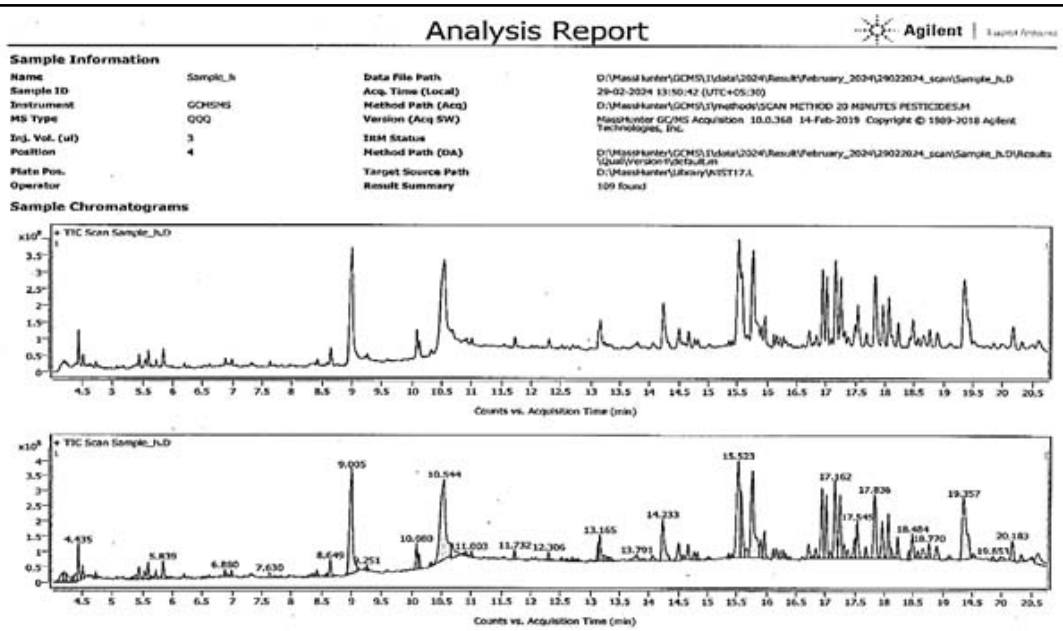

Fig. 1:GC–MS chromatogram of CPS oil

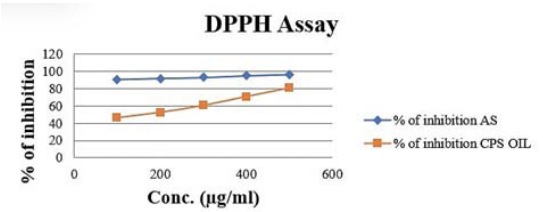

Fig. 2: DPPH radical scavenging activity of ascorbic acid and CPS oil

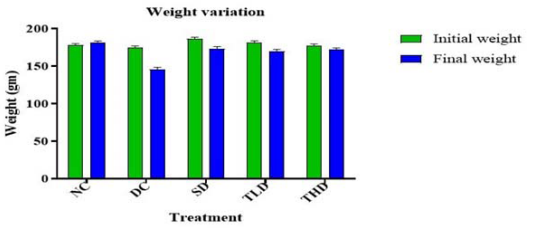

Fig. 3: Responses of Various Groups Comparing Initial vs. Final Body Weight

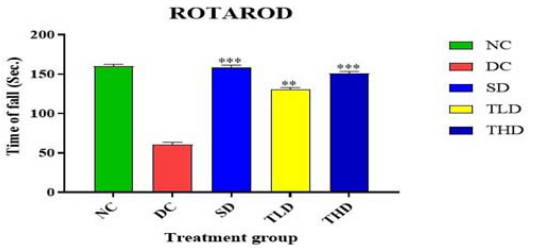

Fig. 4: Responses of Different Groups in the Rota-Rod Test

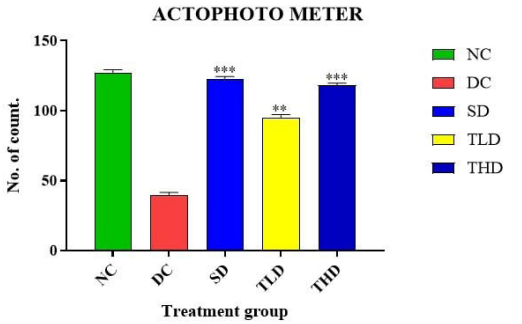

Fig. 5: Responses of Various Groups in the Actophotometer Test

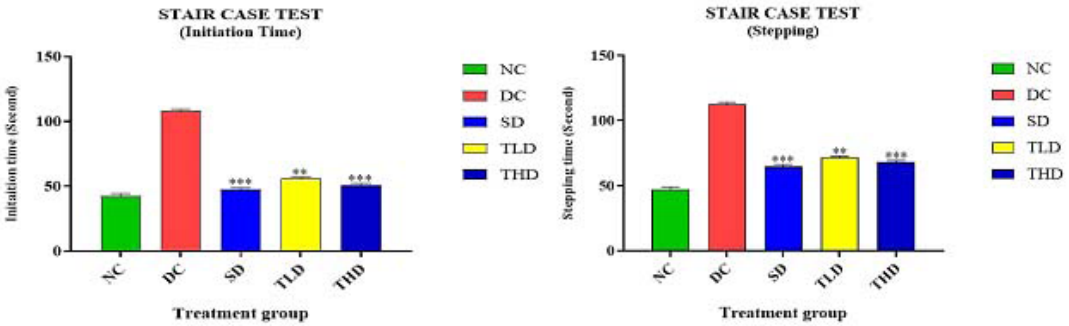

Fig. 6: Responses of different groups in Initiation test and Stepping

drug treatment, supporting its neuroprotective efficacy.

Locomotor Activity (Actophotometer Test)

Spontaneous locomotor activity was evaluated using a digital actophotometer, which measures the movement of animals based on the interruption of infrared light beams positioned above the chamber floor[11]. Each rat was placed individually in the activity cage, and the total number of beam interruptions was automatically recorded for a period of 5 minutes. The apparatus was cleaned with 70% ethanol between trials to eliminate odor cues. Decreased locomotor activity reflects bradykinesia and reduced exploratory behavior typical of Parkinsonian symptoms.

As shown in Table 4 and Figure 5, the disease control group demonstrated a significant reduction ($p < 0.01$) in locomotor activity compared to the normal control, confirming motor impairment due to dopaminergic neurodegeneration. Treatment with CPS oil significantly enhanced locomotor activity in a dose-dependent manner, indicating improvement in spontaneous movement and overall motor function. The higher dose of CPS oil (400 mg/kg) restored activity levels close to those of the standard treatment group, suggesting

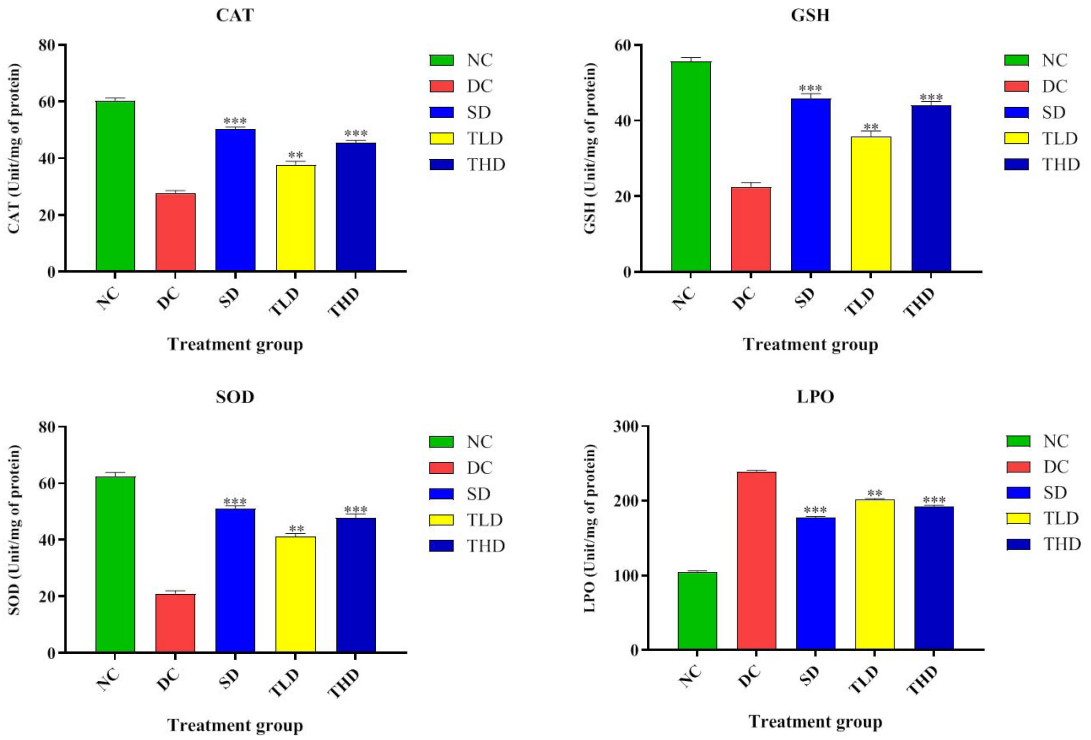

Fig. 7: Antioxidant enzyme levels (SOD, CAT, GSH, and LPO) in experimental groups

effective neuroprotection and partial restoration of dopaminergic tone.

Skilled Motor Coordination (Staircase Test)

The staircase test was employed to assess fine motor coordination, skilled forelimb use, and paw dexterity in experimental animals[12–15]. Each rat was placed in the central compartment of the staircase apparatus, which consisted of two descending staircases on either side, each containing recessed wells holding food pellets. The setup allows for independent evaluation of the left and right forepaws, as pellets can only be retrieved with the corresponding paw.

Animals were subjected to mild food restriction (maintaining 90% of their normal body weight) to enhance motivation. Prior to testing, each rat underwent three training sessions to familiarize them with the apparatus. During the test session, the number of pellets retrieved, displaced, and left untouched within a 15-minute period was recorded. This provides a quantitative measure of reaching ability, coordination, and fine motor performance. As shown in Tables 5.1 and 5.2, and Figure 6, the disease control group exhibited a significant reduction in the number of pellets retrieved and a higher number of pellets displaced or untouched compared to the normal control group ($p < 0.01$). This indicates pronounced impairment in skilled forelimb function, typical of Parkinsonian motor deficits.

Treatment with CPS oil markedly improved performance in a dose-dependent manner, evidenced by an increased number of pellets successfully retrieved and a decrease in pellets displaced. The higher dose of CPS oil (400 mg/kg) showed results comparable to the standard-treated group, demonstrating substantial restoration of motor coordination and paw dexterity. These findings confirm the therapeutic potential of CPS oil in improving fine motor control and mitigating PD-related neuromotor dysfunctions.

Biochemical Estimations

Biochemical estimations were performed to evaluate the antioxidant potential of the test formulations by quantifying endogenous antioxidant

enzyme levels and oxidative stress markers in brain tissue homogenates. The assays included SOD, CAT, and GSH, which represent key components of the enzymatic antioxidant defense system. In addition, LPO levels were measured to assess the extent of oxidative damage to membrane lipids.

These biochemical parameters were determined using standard spectrophotometric methods following established protocols[16–19]. The results were expressed as mean ± SEM and are presented in Table 6 and Figure 6, which demonstrate the comparative antioxidant status among different treatment groups. Collectively, these estimations provided a comprehensive insight into the ability of the test formulation to mitigate oxidative stress and restore redox homeostasis in experimental animals.

The present study demonstrated that CPS oil possesses significant neuroprotective and antioxidant potential in experimental models of PD. GC–MS analysis revealed the presence of bioactive constituents such as sesquiterpenoids, alkaloids, phenolic triterpenoids, and essential fatty acids, all of which are known for their neuroprotective and antioxidative activities. The DPPH assay confirmed the free radical scavenging capacity of CPS oil, which, although lower than that of standard ascorbic acid, still indicated a moderate yet pharmacologically relevant antioxidant activity.

Pharmacological assessments including body weight, grip strength (rotarod test), locomotor activity, and skilled paw use (staircase test) showed marked improvements in motor coordination, muscle tone, and dexterity in CPS oil-treated groups compared to the disease control. These findings suggest that CPS oil effectively ameliorates dopaminergic dysfunction and alleviates Parkinsonian motor deficits.

Biochemical estimations further substantiated these observations by showing a significant restoration of endogenous antioxidant defense systems. Elevated levels of SOD, CAT, and GSH, along with reduced lipid peroxidation (LPO), indicate that CPS oil attenuates oxidative stress, a central pathogenic mechanism in Parkinson's disease. The observed neuroprotection may be attributed to the synergistic action of phytoconstituents, which not only scavenge reactive oxygen species but also enhance mitochondrial function and promote neuronal survival.

Collectively, these findings corroborate the traditional Ayurvedic use of CPS oil as a Medhya Rasayana (memory and intellect enhancer) and provide a scientific rationale for its neuroprotective potential in PD models[20–25].

CONCLUSION

The present study concludes that CPS oil exhibits promising neuroprotective and antioxidant properties, effectively improving motor coordination, muscle strength, and locomotor activity while reducing oxidative stress in experimental models of Parkinson's disease. Its rich phytoconstituent profile contributes to the enhancement of endogenous antioxidant defenses, thereby mitigating neurodegeneration. These findings highlight the therapeutic potential of CPS oil as a natural adjunct in the management of PD. However, further investigations focusing on its molecular mechanisms of action, long-term safety, and clinical validation are essential to translate these preclinical findings into viable therapeutic applications.

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ETHICAL APPROVAL

Approval Ref. No.: BMRL/IAEC/2024-37

Informed consent

Not Applicable

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Conflict of Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper. The authors declare

no conflict of interest among themselves. The authors alone are responsible for the content and writing of this article.

Financial interests

The authors declare they have no financial interests.

Human and animal rights declaration : NA

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