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Research Article

Pharmacological investigation of *Ocimum basilicum* in rotenone-induced Parkinson's disease rat model

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Abstract

Background: Parkinson's disease is characterised by motor dysfunction and oxidative stress–induced neuronal damage. Conventional therapy with Levodopa and Carbidopa improves symptoms but does not halt neurodegeneration, prompting exploration of natural agents like *Ocimum basilicum*. Objective: To evaluate the neuroprotective effects of *Ocimum basilicum* extract, alone and in combination with levodopa-carbidopa, in a rotenone-induced Parkinsonian rat model. Materials and Methods: Parkinsonian-like symptoms were induced using rotenone (1.5 mg/kg, i.p.) for 28 days. Rats were divided into control, rotenone, *O. basilicum* (200 and 400 mg/kg), levodopa-carbidopa, and combination groups. Motor function was assessed using rotarod, stair ascent, and pole tests. Brain tissues were analysed for malondialdehyde (MDA), nitric oxide, dopamine (DA), and glutathione (GSH) levels, along with histopathological evaluation. Results: Rotenone caused significant motor impairment and oxidative stress. Treatment with *O. basilicum*, especially at 400 mg/kg, significantly improved motor performance ($P < 0.001$), restored DA and GSH levels, and reduced MDA and nitric oxide. Combination therapy showed enhanced effects. Histopathology confirmed reduced neuronal degeneration. Conclusion: *Ocimum basilicum* exhibits significant neuroprotective effects and enhances the efficacy of levodopa-carbidopa, suggesting its potential as an adjunct therapy in Parkinson's disease management.

Keywords: *Ocimum basilicum,* Parkinson's, Neuroprotection, Neurodegeneration.

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Introduction

In ageing communities, millions of people suffer from neurodegenerative diseases like Parkinson's (PD) and Alzheimer's. PD ranks second in chronic and progressive neurodegenerative illnesses (1). The primary cause of Parkinson's disease symptoms is the progressive loss of dopaminergic nerve cells in the substantia nigra pars compacta (SNpc) and the resulting depletion of dopamine (DA) in the striatum (2). PD is clinically diagnosed with symptoms including akinesia, stiffness, bradykinesia, resting tremor, instability in posture, and deficits in sensory-motor integration (3). Although environmental and genetic variables have been suggested, the exact cause of Parkinson's disease remains unresolved. Epidemiological research shows that pesticides, along with other environmental pollutants, may contribute to the onset of idiopathic Parkinson's disease (4). Rotenone is an insecticide (neurotoxin) utilised to produce Parkinson's disease in an animal model (5). In rats, rotenone mimics numerous critical clinical characteristics of Parkinson's disease, including oxidative damage.-synuclein aggregation. Previous studies show that the Degeneration of dopaminergic terminals in the striatum is mediated by oxidative stress (6). Currently, dopamine agonist (levodopa) therapy is a typical treatment for Parkinson's disease; however, prolonged use of this medication may induce unpleasant side effects (7,8).

Consequently, there has been heightened interest in the therapeutic potential of medicinal plants with antioxidant properties, which may mitigate oxidative damage in rodent models of Parkinson's disease. Previous research demonstrates that *Mucuna pruriens*,

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Nigella sativa L.(9), *Vicia faba L.*(10), *Crocus sativus L.*(11), *Curcuma longa*, *Acanthopanax senticosus*, *Bacopa monnieri*, *Centella Asiatica*, *Panax ginseng*, *Oxalis corniculata L.*, *Coriandrum sativum*, *Withania somnifera*, *Ginkgo biloba L.*, *Valeriana officinalis*, *Eclipta alba*, *Delphinium denudatum*, and *Carthamus tinctorius L.* are botanical species utilised in the management of Parkinson's disease (12,13,14).

O. basilicum, commonly known as sweet basil, belongs to the Lamiaceae family. Recent scientific investigations have revealed the therapeutic potential of *O. basilicum*, exhibiting properties including antioxidant (15), anti-inflammatory (16), antimicrobial (17), anticancer (18), and neuroprotective effects (19). Numerous researchers have conducted phytochemical analyses of *O. basilicum* and its specific morphological parts, identifying several vital constituents. These compounds encompass volatile oils such as eugenol, methyl eugenol, coumarins, alkaloids, tannins, anthraquinones, flavonoids, diterpenoids, polyphenols, quinones and sugars (20,21).

Oxidative stress contributes to Parkinson's symptoms, and the plant's antioxidants may help. *O. basilicum* has antioxidant effects in vivo and in vitro. Therefore, this study investigated *O. basilicum's* therapeutic benefits on rotenone-induced Parkinson-like symptoms in animal models.

Materials and methods

Collection and authentication plant

The aerial components of *O. basilicum* were collected from numerous locations in Wardha, India, in September 2022. The plant was later identified and verified by Dr. Vishal N. Patil, a botanist at Dr. R. G. Bhoyar Arts, Commerce & Science College, Seloo, Wardha. A voucher specimen was filed with the file number 01/RGBACSBOTANY/2022-23.

Extraction

400 g of dried aerial parts of *O. basilicum* were subjected to extraction at ambient temperature with 5.0 liters of a 7:3 ethanol-water solution by the maceration process for a 24 h. After filtering the extract with a Buchner funnel, the solvent was removed in a rotary evaporator at 60 °C to produce a concentrated extract, which was subsequently freeze-dried to create a dry powder. The extract was diluted with saline, using a few drops of Tween, to attain the final concentration (22).

Animals

All tests were performed with male and female Albino Wistar rats, which the Committee for the Control and Supervision of Tests on Animals (CPCSEA) permitted under registration number IPER/IAEC/2022-23/13. The animals were housed in polypropylene cages at 24 ± 2°C and an equivalent humidity of 40-60% under a 12-hour light/dark cycle. The studies were conducted in a tranquil setting from 8:00 AM to 12:00 PM. Throughout the study, the animals were provided with a balanced meal and unrestricted access to water. All animals underwent acclimatisation for seven days before the initiation of the experimental research. The Institutional Animal Ethics Committee sanctioned the protocol.

Drugs and chemicals

We received rotenone from TCI Chemicals (India) Pvt Ltd. in Mumbai. Levodopa-carbidopa is sourced from the Bengaluru-based Biocon Research Centre. All of the necessary materials were procured, including thiobarbituric acid, reduced glutathione, and other chemicals from local suppliers, including Hi-Media Chemicals.

Instruments

Neurochemistry was studied using HPLC (Shimadzu, Japan) and UV–visible spectrometer (UV-1800, Shimadzu, Japan). Rota rod (M K lab instrument) was used to record the mean latency for rod retention time.

Experimental design

Thirty adult male Wistar rats were used to avoid hormonal variability associated with the oestrous cycle. Five groups of rats, each including six rats, were assigned randomly. All rats, except the first (Normal) group, were administered Rotenone (1.5 mg/kg/day) intraperitoneal (i.p.) for 28 days to induce Parkinson's disease. The Normal group received solely saline solution as a vehicle. The disease Control group was allowed to recover spontaneously. In contrast, the standard Group received the typical Levodopa-carbidopa (100mg/kg/day +25 mg/kg/day p.o) medication for 14 days via oral administration. Doses of *O. basilicum* extract were selected based on a literature survey (22). Test-I groups received *O. basilicum* extract at 200 mg/kg. In contrast, Test-II groups were administered 400 mg/kg, starting from the 15th day of rotenone treatment and lasting 14 days. Rats underwent training sessions utilising the Rotarod and staircase tests. Motor impairment (muscle rigidity) was documented on days 14, 21, and 28, one hour post-administration of *O. basilicum* extract.

Preparation of dose

O. basilicum extract and essential oil were mixed with saline and 0.5% Tween 80. All solutions were freshly made on the day of the study and administered intraperitoneally (i.p). However, Rotenone was solubilised in DMSO and diluted with pure natural oil, and administered intra-peritoneal (i.p.). Following induction, the standard group of rats received the standard medication, typical Levodopa-carbidopa (100mg/kg/day +25 mg/kg/day), administered orally (p.o) daily upto 14 days.

Behavioural study

Rotarod test

The Rotarod test is a straightforward and dependable method for evaluating rodent motor coordination and balance. A motorised circular rod revolves at a steady or accelerating velocity. This Rotarod study investigated the impact of *O. basilicum* extract on muscle performance. To master their position on a 3 cm diameter rod, which rotated at 25 revolutions per minute, animals underwent two 300-second training trials with a 10-minute interval between them. After these training sessions, a 180-second baseline trial documented the duration of each animal's stay on the Rotarod. Animals that remained upright achieved a maximum score of 180 seconds. Data can be examined by calculating the mean latency to fall (across all trials) per day, the best (or worse) latency per day, and the individual representation of all trials (23).

Staircase test

The three-minute staircase test observes rats to assess their climbing skills in an enclosed five-step stairway. A wooden model staircase encased in glass was used to test vehicle-treated and control (rotenone-treated) rats' muscle strength. Each rat was placed on the floor facing away from the staircase, and a video camera recorded its upward steps and rearings. A rat climbed a step when it placed all four paws on it, and down steps were

registered. The animal's fitness and inquisitive behaviour were measured by the number of steps climbed and the time needed to reach the summit (24).

Estimation of Reduced Glutathione (GSH)

Homogenate brain supernatant natant was combined with trichloroacetic acid (10%, w/v) in an equal proportion. The tubes were established to be homogenate supernatants subjected to centrifugation at 1000 g for 10 minutes at 4°C. 0.5 ml of the resulting supernatant was combined with 2 ml of disodium hydrogen phosphate (0.3 M). A freshly made DTNB [5,5-dithiobis(2-nitrobenzoic acid)] solution (0.001 M; 0.25 ml), diluted in 1% w/v sodium citrate, was incorporated into the solution above, and absorbance was measured by spectroscopic at 412 nm. GSH was quantified as µmol/mg of protein (25).

Estimation of Malondialdehyde

The method is based on Spectrophotometric evaluation of the colour produced by the reaction between thiobarbituric acid (TBA) and MDA. Consequently, 2.5 ml of a 100 g/l trichloroacetic acid solution was mixed into 0.5 ml brain homogenate and was subjected to a boiling water bath for 15 minutes. Subsequently, 2 ml of the homogenate was combined with 1 ml of 6.7 g/l TBA solution within a test tube. The tube was subsequently immersed in a bath of hot water for 15 minutes. The solution was subsequently cooled, and its absorbance was assessed with a spectrophotometer at a wavelength of 532 nm. The concentration of MDA was determined using the absorbance coefficient of the MDA–TBA complex, which is reported in nanomoles per gram of protein (26)

Estimation of Nitric Oxide

The quantification of nitrite in the striatum supernatant, indicative of nitric oxide (NO) synthesis, was conducted via a colourimetric assay employing Griess. Equal quantities of the supernatant and Griess reagent were combined and incubated for 10 minutes at ambient temperature without light. Absorbance was subsequently quantified at 540 nm utilising a spectrophotometer. NO were quantified in nM/mg of protein (27).

Neurochemical Estimation

The concentration of the principal neurochemical (DA) were quantified using high-performance liquid chromatography with fluorescence detection (HPLC-FD). Separation was achieved on a reversed-phase C18 column (150 mm × 4.6 mm, 5 µm particle size). The mobile phase consisted of a mixture of 0.1 M phosphate buffer (pH 3.0) and methanol in a 90:10 (v/v) ratio, delivered isocratically at a flow rate of 1.0 mL/min. The fluorescence detector was set with an excitation wavelength of 280 nm and an emission wavelength of 320 nm for optimal sensitivity. The method was validated for linearity, precision, accuracy, limit of detection (LOD), and limit of quantification (LOQ). Calibration curves for DA showed excellent linearity ($R^2 > 0.99$) within the concentration range of interest. Intraday and interday precision exhibited relative standard deviations below 5%, confirming the method's reproducibility and reliability for dopamine quantification in brain tissue samples. The data were presented in ng/mg of moist tissue (28).

Histopathological Analysis

One rat from each group was euthanised, the brain was excised, and the brain areas were meticulously cleansed to remove superfluous tissue and adipose material. The purified brains were situated in a container of 10% formalin solution. The specimens were dispatched to Accuvet Pathology Lab, Tulsi Park, Katol Road, Nagpur-440013 for slide preparation. Tissue slides were dispatched to Kanere Histopathology Lab, Plot No. 102A Shrihari Nagar, No. 02, adjacent to NIT Garden, Manewada, Nagpur-27 for additional analysis.

Statistical Analysis

Data were processed via Graph Pad Prism 9.5.1 for Windows. Results were presented as Mean ± SEM. A one-way analysis of variance (ANOVA) and Tukey's multiple comparison test were employed to assess the significance of differences among the variables across the groups. P values below 0.05 were deemed statistically significant.

Result Analysis

Effects of *O. basilicum* Extract on Rotarod Test

Table 1 illustrates the impact of several dosages of *O.basilicum* extract on retention time on the rod during the Rotarod test in rats. Rotenone administration markedly reduced the retention duration on the rod (###p<0.001) vs. the regular saline group on days 14, 21, and 28. Treatment with *O. basilicum* extract at a 400 mg/kg dose significantly increased retention time (**p<0.01) compared to the rotenone-treated group. Nonetheless, administration of 200 mg/kg of the extract slightly enhanced retention time (**p<0.05), which was not statistically significant to the rotenone-treated group.

Table 1: Effect of *O. basilicum* extract on retention time on the rotenone-treated group in the rotarod test.

Sr no.	Groups	Treatment	Retention time on Rotarod (sec)		
			Day 14	Day 21	Day 28
1	Normal	Saline solution	152.6 ± 1.8	154.2 ± 1.167	153.26 ± 1.99
2	Control	RT (1.5mg/ kg)	114.8 ± 1.8	78.67 ± 1.783	57.34 ± 1.892###
3	Standard	RT+LD+ CD (1.5+100 +25 mg/kg)	120.4 ± 2.2ns	134.46 ± 1.922	148.76 ± 2.216***
4	Test- I	RT+ OBE (1.5 + 200 mg/kg)	115.2 ± 1.4ns	117.73 ± 2.108	124.32 ± 1.352*
5	Test -II	RT + OBE (1.5+400 mg/ kg)	118.9 ± 2.8ns	123.87 ± 2.963	144.59 ± 2.548**

Each group had six rats (n=6), and the results are shown as mean ± S.E.M. ###p<0.001 when compared to the normal group. *p<0.05, **p<0.01, and ***p<0.001 against the rotenone-treated group. (Rotenone (RT), Levodopa (LD), Carbidopa (CD), and *O. basilicum* extract (OBE).

Effects of *O. basilicum* Extract on Staircase

Table 2 demonstrates the impact of different dosages of *O. basilicum* extract on step-up latency in the staircase trial conducted on rats. The injection of rotenone markedly prolonged the duration spent on the stairs (###p< 0.001) vs. the regular saline group on days 14, 21, and 28. Treatment with *O. basilicum* extract at 400 mg/kg markedly diminished the time allocated to stair navigation (**p<0.01) compared with the rotenone-treated cohort. Nonetheless, administration of the extract at 200 mg/kg did not significantly decrease retention Time on the stairs (**p<0.05) relative to the rotenone group.

Table 2: Effect of *O. basilicum* extract on retention time on the rotenone-treated group in the staircase test

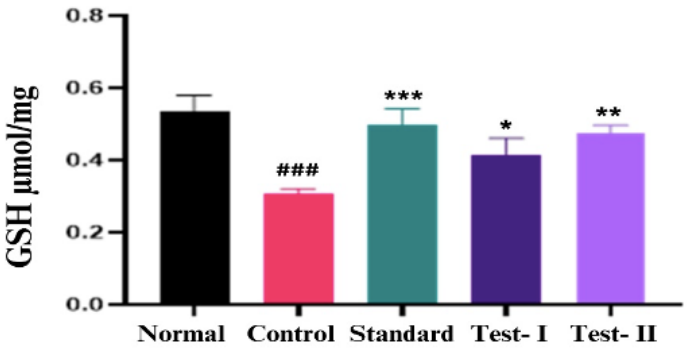
Sr no.	Groups	Treatment	Retention time on staircase (sec)		
			Day 14	Day 21	Day 28
1	Normal	Saline solution	10.14 ± 0.87	9.920 ± 0.790	9.84 ± 0.381^{ns}
2	Control	RT (1.5mg/ kg)	27.52 ± 0.841	30.36 ± 1.08	35.80 ± 0.39$^{###}$
3	Standard	RT+LD + CD (1.5+ 100 +25 mg/kg)	15.17 ± o.819^{ns}	21.10 ± 0.861	23.82 ± 0.755***
4	Test -I	RT+ OBE (1.5 + 200 mg/kg)	24.46 ± 0.819^{ns}	28.93 ± 1.135	34.30 ± 0.395*
5	Test -II	RT + OBE (1.5+400 mg/ kg)	21.68 ± 0.643^{ns}	24.08 ± 0.657	29.32 ± 0.410**

Each group had six rats (n=6), and the results are shown as mean ± S.E.M. ###p<0.001 when compared to the normal group. *p<0.05, **p<0.01, and ***p<0.001 against the rotenone-treated group. (Rotenone (RT), Levodopa (LD), Carbidopa (CD), and *O. basilicum* extract (OBE).

Assessment of reduced glutathione (GSH) concentration in rats.

Figure 1 illustrates the impact of several dosages of *O. basilicum* extract on reduced glutathione concentrations in rats. Rotenone therapy markedly reduced the glutathione levels (###p<0.001) in tissue homogenate relative to the saline group. Administration of *O. basilicum* extract at 400 mg/kg can markedly elevate the concentration (**p < 0.01) of reduced glutathione in rats relative to the control group. Conversely, *O. basilicum* extract (200 mg/ kg) does not significantly restore decreased (**p< 0.05) glutathione levels in rats.

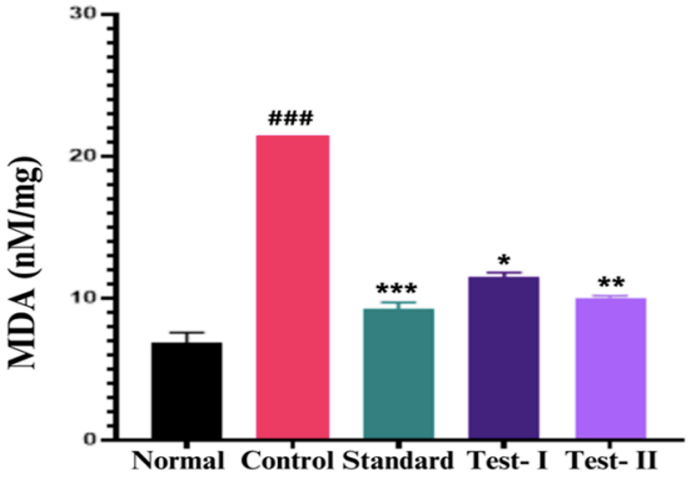
Figure 1: Each group had six rats (n=6), and the results are shown as mean ± S.E.M. ###p<0.001 when compared to the normal group. *p<0.05, **p<0.01, and ***p<0.001 against the rotenone-treated group.



Estimation of Malondialdehyde (MDA) level in rats.

Figure 2 illustrates the impact of varying dosages of *O. basilicum* extract on rats' Malondialdehyde (MDA) concentration. Rotenone administration is markedly elevated MDA (###p<0.001) concentrations in the brain tissue homogenates of rats relative to the Normal group. Treatment with *O. basilicum* extract at 400 mg/ kg can dramatically reduce Malondialdehyde (MDA) levels (**p<0.01) in rats compared to a control group. The *O. basilicum* extract at 200 mg/kg does not significantly reduce rats' MDA levels (**p<0.05).

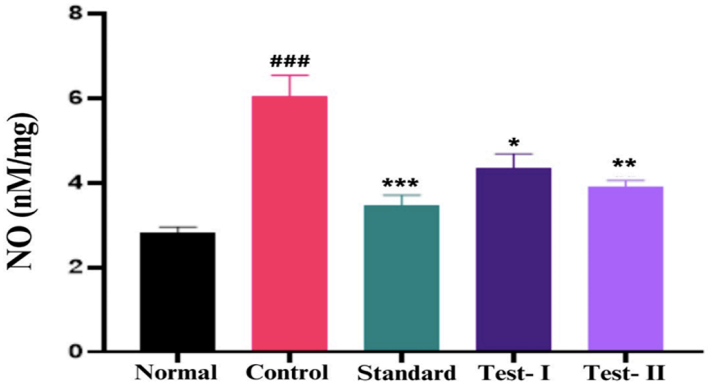
Figure 2: Each group had six rats (n=6), and the results are shown as mean ± S.E.M. ###p<0.001 when compared to the normal group. *p<0.05, **p<0.01, and ***p<0.001 against the rotenone-treated group.



Estimation of Nitric Oxide level in rats.

Figure 3 shows the impact of varying dosages of *O. basilicum* extract on Nitric Oxide levels in rats. Rotenone administration markedly elevated the concentration of Nitric Oxide (###p<0.001) in the brain tissue homogenate of rats relative to the control group. The administration of *O. basilicum* extract at 400 mg/kg can markedly reduce (**p<0.01) nitric oxide levels in rats relative to those in the control group. *O. basilicum* extract at 200 mg/kg does not significantly reduce Nitric Oxide levels (**p<0.05) in rats.

Figure 3: Each group had six rats (n=6), and the results are shown as mean ± S.E.M. ###p<0.001 when compared to the normal group. *p<0.05, **p<0.01, and ***p<0.001 against the rotenone-treated group.

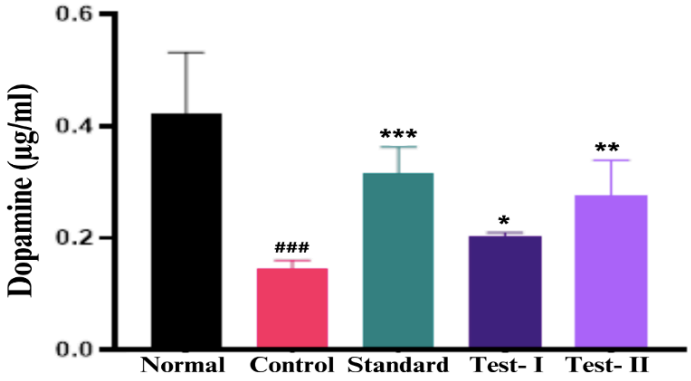


Estimation of Dopamine level in rats

Figure 4 illustrates the impact of varying doses of *O. basilicum* extract on Brain Dopamine levels in rats. The administration of Rotenone treatment resulted in a notable reduction in the concentration (###p<0.001) of Brain Dopamine in tissue homogenate when compared to the Normal group. Treatment with *O. basilicum* extract at a dosage of 400 mg/kg can significantly elevate the levels (**p<0.01) of Brain Dopamine in rats when compared to the control group. The administration of *O. basilicum*

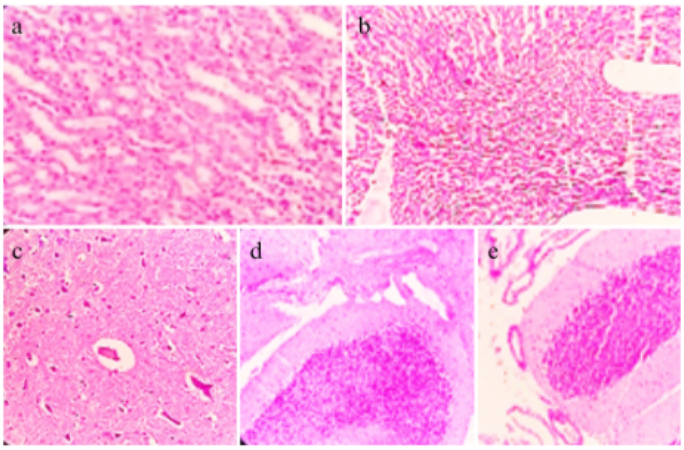
extract at a dosage of 200 mg/kg does not significantly restore Brain Dopamine levels (**p<0.05) in rats.

Figure 4: Each group had six rats (n=6), and the results are shown as mean ± S.E.M. ###p<0.001 when compared to the normal group. *p<0.05, **p<0.01, and ***p<0.001 against the rotenone-treated group.



Histopathological Examination

Figure 5: Histopathological examination of midbrain normal (a), rotenone 1.5 mg/kg/day (b), levodopa-carbidopa (100+25 mg/kg/day) along with rotenone (c), *O. basilicum* extract200mg/kg/day along with rotenone (d), *O. basilicum* extract 400mg/kg/day along with rotenone (e). Observation was done using Hematoxylin and eosin stain; magnification was used 400x.



Sections from the control group (a) show normal neuronal architecture with well-preserved cell bodies, clear nuclei, and absence of degenerative changes. In contrast, the rotenone-treated group (b) demonstrates marked neuronal degeneration characterized by reduced neuronal density, shrunken and pyknotic neurons, cytoplasmic eosinophilia, and disrupted cellular organization. Sections from treatment groups (c–e) reveal varying degrees of neuroprotection. Mild to moderate neuronal degeneration is observed in lower-dose *Ocimum basilicum* treatment (c), with partial preservation of neuronal structure. Higher-dose treatment (400 mg/kg) and combination therapy groups (d–e) show significant attenuation of neuronal damage, with increased number of intact neurons, reduced pyknosis, and improved cellular morphology compared to the rotenone alone treated group.

For quantitative assessment, substantia nigra neuronal degeneration can be expressed as the number of degenerated neurons per microscopic field or by a blinded semi-quantitative scoring system (0 = no degeneration, 1 = mild, 2 = moderate, 3 = severe). The rotenone group would correspond to a higher degeneration score (≈3), whereas treated groups show reduced scores in a dose-dependent manner, with the combination therapy group approaching near-normal levels.

Discussion

Flavonoids and phenolic substances that exist naturally possess antioxidant effects. The plant *O. basilicum* has flavonoids and other phenolic chemicals (29). Hence, The present investigation aimed to assess the protective impact of *O. basilicum* against rotenone-induced rat model of PD. *O. basilicum* is a significant medicinal plant in the Ayurvedic system (30). Traditional uses of this plant include flavouring agents in the food sector, dental and oral goods, and fragrances. It also treats coughs, headaches, parasites, diarrhoea, and skin problems. Basil polysaccharides have been utilised for cancer treatment in traditional Chinese medicine and continue to be prevalent in people's lives (31). Ayurveda employs a personalised methodology in treating Parkinson's disease (32). Previous studies revealed that the plant has significant antioxidant properties and recommended evaluating its potential advantages in neurological disorders based on its ancient use (33). In addition, oxidative stress significantly contributes to the development of rotenone-induced Parkinson's disease pathology (34), so it is pertinent to investigate the advantageous effects of this antioxidant plant within a rotenone-induced Parkinson's like symptoms.

The present investigation revealed that the administration of rotenone to rats resulted in a marked deterioration of motor coordination and muscular tone, as evidenced by the outcomes of many assessments, including the rota rod and stair tests. These results align with those of, who similarly noted neurodegeneration and the beginning of Parkinson's disease after rotenone administration at a comparable frequency (35). *O. basilicum* extract and levodopa-carbidopa markedly enhanced motor coordination in rotenone-treated rats, as evidenced by increased retention durations in the rotarod test and decreased step-up latency in the staircase test. These data indicate that *O. basilicum* extract possesses potential neuroprotective properties that may mitigate motor deficits generated by rotenone

O. basilicum extract significantly diminished rotenone-induced depletion of GSH levels in the midbrain and cortex. A disturbance of the normal antioxidant defence mechanisms, particularly involving GSH, plays a significant role in the cause of PD. A reduced concentration of GSH has been seen in the substantia nigra of individuals with PD (36). A decrease in GSH levels facilitates hydroxyl radical production, which might create a prooxidant environment. This pro-oxidant environment can result in heightened lipid peroxidation, which can be quantified by assessing the levels of TBARS, a byproduct of lipid peroxidation (36).

The oxidative damage resulting from elevated lipid peroxidation and Nitric oxide has been significantly associated with the aetiology of Parkinson's disease (37). The current study demonstrated that *O. basilicum* extracts considerably mitigated the heightened lipid peroxidation and nitric oxide by rotenone, as seen by the TBARS laboratory results.

Moreover, rotenone is a recognised inhibitor of mitochondrial complex I that induces numerous behavioural and neuropathological characteristics of Parkinson's disease due to the degeneration of nigral dopamine neurons. Dopamine is crucial for

motor function in the human body, and its depletion has been associated with the primary symptoms of Parkinson's disease (38,39). The current study observed a significant reduction in dopamine levels in the rotenone-treated group, mitigated by *O. basilicum*. Levodopa and carbidopa exhibit efficacy in Parkinson's disease by elevating dopamine levels in the brain. Consequently, dopamine restoration by *O. basilicum* may serve as a mechanism for mitigating rotenone-induced behavioural changes in rats.

Research indicates that the midbrain is the most sensitive region of the brain to rotenone toxicity (40). Present histological findings indicate neuronal degeneration, suggesting significant damage to the region due to rotenone in the disease control group. The current study demonstrated that *O. basilicum* co-treatment prevents neuronal damage.

The *O. basilicum* has significantly impacted behavioural, biochemical, neurochemical, and histopathological parameters, with the most pronounced effect observed at *O. basilicum* (400 mg/kg). However, *O. basilicum* extract at a 200 mg/kg dose was not statistically significant.

The mechanism underlying the effectiveness of *O. basilicum*in this study may be hypothesised as its antiparkinsonian activity, which could result from its antioxidant effect that mitigates neurodegeneration in Parkinson's disease.

Conclusion

The current study concludes that *O. basilicum* mitigated the behavioural, biochemical, neurochemical, and histological changes generated by rotenone-related oxidative stress impacts. The antioxidant capacity of *O. basilicum* can be understood as it mitigates rotenone-induced oxidative damage in the brains of rats. *O. basilicum* at 400 mg/kg exhibited the most significant benefit across all parameters when administered in conjunction with levodopa and carbidopa. Therefore, it can be inferred that *O. basilicum* may serve as a possible therapeutic agent for Parkinson's disease as a herbal phytoconstituent, providing a foundation for future clinical trials.

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