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NEUROPROTECTIVE POTENTIAL OF MAERUA OBLONGIFOLIA LEAF EXTRACT IN EXPERIMENTAL RAT MODELS

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ABSTRACT: The present study was undertaken to evaluate the neuroprotective potential of Maerua oblong folia leaf extract in scopolamine-induced cognitive impairment in Wistar rats. Memory and learning performance were assessed using standard behavioral paradigms such as Morris Water Maze, Elevated Plus Maze, and Y-Maze. Biochemical estimations were carried out to evaluate oxidative stress and cholinergic function by measuring acetyl cholinesterase (AChE) activity, lipid peroxidation (MDA), and antioxidant enzyme levels including superoxide dismutase (SOD), catalase (CAT), and reduced glutathione (GSH). Scopolamine administration produced significant cognitive deficits characterized by increased escape latency, increased transfer latency, and reduced spontaneous alternation behavior, confirming successful induction of memory impairment. Biochemical analysis revealed elevated AChE activity and MDA levels, along with a significant reduction in endogenous antioxidant enzymes (SOD, CAT, and GSH), indicating enhanced oxidative stress and cholinergic dysfunction. Treatment with Maerua oblongifolia leaf extract showed a dose-dependent improvement in both behavioral and biochemical parameters. The high-dose group demonstrated significant restoration of learning and memory functions, along with normalization of antioxidant status and reduction in AChE and MDA levels, with effects comparable to the standard drug Donepezil. The study concludes that Maerua oblongifolia possesses significant neuroprotective and memory-enhancing activity, possibly mediated through cholinesterase inhibition and antioxidant mechanisms. These findings suggest its potential therapeutic application in the management of neurodegenerative disorders such as Alzheimer's disease, warranting further detailed pharmacological and clinical investigations.

Keywords: Morris Water Maze, Elevated Plus Maze, Y-Maze,Tephrosia purpurea, neuroprotection, cognitive impairment.

INTRODUCTION

NEURODEGENERATION

Neurodegeneration is defined as the progressive loss of structure and function of neurons in the central nervous system. Neurodegenerative diseases have a devastating impact on millions of individuals and their families. Left unchecked, the prevalence of these diseases will grow at an alarming rate1. There is an urgent need to accelerate the search for effective treatments and cures. Many neurodegenerative diseases including Parkinson's, Alzheimer's, and Huntington's occur as a result of neurodegenerative processes. WHO also assures that by 2030, as many as 1 in 5 among us will suffer from neurodegenerative disease. If left unchecked few years from now, more than 12 million will suffer from neurodegenerative diseases. Finding treatments and cures for neurodegenerative diseases is a goal of increasing urgency2.

Common characters of neurodegenerative diseases

The progressivity of neuronal death has also led to speculation on the very long phase of the diseases, which asks key questions concerning early diagnosis and treatment; a stage at

which the clinical signs of the disease are not expressed. It is thus claimed that the earlier the treatment of the disease, the better it would be for limitation of neuronal destruction and clinical expression. Such a consideration emphasizes the search for early events of the diseases and critical putative biomarkers as well as stimulating research programs to promote therapeutic approaches focused at the etiological level leading to true neuroprotection against the diseases.

Neurons

Neurons are the building blocks of the nervous system which includes the brain and the spinal cord. Neurons normally will not reproduce or replace themselves, so when they become damaged or die, they cannot be replaced by the body. Neurodegenerative diseases are incurable and debilitating conditions that result in progressive degeneration and death of nerve cells. This causes problems with movement called ataxias and mental functioning called dementias3.

NEURODEGENERATIVE DISESASES

Neurodegenerative diseases including Parkinson's, Alzheimer's, and Huntington's occur as a result of neurodegenerative processes.

Alzheimer's disease

The most frightening and devastating of all neurological disorders is a dementia that occurs in the elderly. The most common cause of this illness is Alzheimer's disease (AD). The earliest symptoms of AD include forgetfulness, disorientation: to time or place, and difficulty with concentration, calculation, language, and judgment.

Parkinson's disease

Parkinsonism describes a syndrome of which Parkinson's disease is the main cause. Parkinson's disease (PD) is a chronic neurodegenerative disorder characterized by the progressive loss of dopaminergic neurons of substantia nigra pars compact in the ventral midbrain. The loss of dopaminergic neurons, leads to the reduction of dopamine being released into the striatum. This results in bradykinesia, resting tremor, rigidity, and difficulty in initiating movements. Defective nucleoli apparently cause oxidative stress in cells. This can lead to massive cell damage and may be a key prerequisite for the typical nerve damage of Parkinson's disease4.

Amyotrophic Lateral Sclerosis Huntington's disease

Huntington' disease (HD) is now considered one of the most common hereditary brain disorders. It progresses slowly over a 10-to-20-year period and eventually robs the affected individual of the ability to walk, talk, think and reason.HD usually appears between the ages of 30 and 50. It affects both the basal ganglia, which control coordination, and the brain cortex, which serves as the center for thought, perception, and memory.

CAUSES OF NEURODEGENERATION

Protein misfolding

Several neurodegenerative diseases are classified as proteopathies as they are associated with the aggregation of misfolded proteins5.

Alpha-synuclein can aggregate to form insoluble fibrils in pathological conditions characterized by Lewy bodies, such as Parkinson's disease, dementia with Lewy bodies, and multiple system atrophy6.

Tau hyperphosphorylated tau protein is the main component of neurofibrillary tangles in Alzheimer's disease7.

Beta amyloid is the major component of senile plaques in Alzheimer's disease.

Protein degradation pathways

Parkinson's disease and Huntington's disease are both late-onset and associated with the accumulation of intracellular toxic proteins. Diseases caused by the aggregation of proteins are known as proteopathies8.

Programmed cell death

Programmed cell death (PCD) is death of a cell in any form, mediated by an intracellular program. There are, however, situations in which these mediated pathways are artificially stimulated due to injury or disease. Caspases (cysteineaspartic acid proteases) cleave at very specific amino acid residues. There are two types of caspases: initiators and effectors. Initiator caspases cleave inactive forms of effector caspases. This activates the effectors which in turn cleave other proteins resulting in apoptotic initiation.

Neurodegeneration and Parkinson's Disease

Parkinson's disease is one among the neurodegenerative disorder which affects at least 6.5 million people worldwide, irrespective of gender, social, ethnic, economic, or geographic boundaries. Symptoms of the disorder such as tremor, rigidity and bradykinesia develop when about 3/4 of dopaminergic cells

are lost in the substantia nigra9 .Depression and hallucinations are common, and dementia eventually occurs in 20% of patients. At this time, there is no treatment to delay or stop the progression of PD. Rather, the medications currently available aim more towards the alleviation of these symptoms. New surgical strategies may reversibly switch on the functionally damaged circuits through the electrical stimulation of deep brain structures, but although deep brain stimulation is a major advance, it is not suitable for all patients10.It remains therefore necessary to generate new safe medicines by using preclinical models. Selective neurotoxic disruption of dopaminergic pathways can be reproduced by injection of 6-hydroxydopamine (6-OHDA) or MPTP (1-methyl-4- phenyl-1,2,3,6-tertahydropyridine) whereas depleting drugs and oxidative-damaging chemicals may also reproduce specific features of PD in rodents. Unlike MPTP, 6- OHDA lesions cause massive irreversible neuronal loss, and can be uni- or bilateral11. The 6-OHDA lesion model is reliable, leads to robust motor deficits, and is the most widely used after 40 years of research in rats. The loss of dopaminergic input within days, and the functional impairments can be monitored over postoperative weeks and months by rating animal rotations induced by dopaminergic agents2.Parkinson's disease is a progressive disorder of movement that occurs mainly in the elderly. The most important characteristic of PD (Parkinson's disease) is degeneration of the dopaminergic neurons of the substantia nigra pars compacta (SNc). Damage of this region result in movement disorders and the most parkinsonian symptoms are tremor, rigidity, and bradykinesia12.

5. MATERIALS AND METHODS

Collection and authentication of Plant material

The Tephrosia Purpurea, was selected for investigation and were procured from the nearest area of Hyderabad Ranga Reddy District. The plant material was taxonomically identified and authenticated by Dr. Madhava Chetty, Head of Department, Botany, Sri Venkateshwara academia, Tirupathi, Andhra Pradesh.

Preparation of plant extract

Maerua oblongifolia was air dried in shade and extracted with 1:4 ratio ethanol extract, using a Soxhlet extractor for 8 hrs at 55-600C. The supernatant was filtered through Whatman filter paper No.1 and concentrated under reduced pressure using vacuum at 44 ± 100C in a rotavapor (IKA ® RB 10 Rota Evaporator, India) The extract was stored at 220C in a seeded air tight container.

The percentage of extract yield was calculated by using the formula

$$\%\ of\ extract\ yield = \frac{weight\ in\ gm\ of\ extract\ obtained}{weight\ in\ gm\ of\ plant\ material\ taken} \times 100$$

Toxicity Studies

Neuroprotective toxicity studies are performed to evaluate the safety profile of test drugs or plant extracts before assessing their protective effects on the central nervous system. Acute toxicity is commonly conducted according to OECD Guideline 423 (Acute Oral Toxicity – Acute Toxic Class Method). Healthy adult Wistar rats or mice of either sex are fasted overnight and divided into groups. The test substance is administered orally at graded doses (e.g., 5, 50, 300, and 2000 mg/kg body weight). Animals are observed continuously for the first 4 hours for behavioral, neurological, and autonomic changes such as tremors, convulsions, salivation, lethargy, sleep, coma, and mortality, and then periodically for 24 hours and daily for 14 days. Parameters such as body weight, food and water intake, skin and fur condition, eyes, mucous membranes, respiration, and locomotor activity are monitored. At the end of the observation period, animals are sacrificed for gross pathological examination of vital organs including brain, liver, kidney, and heart. The absence of mortality and significant toxic signs indicates that the test substance is safe, and one-tenth of the maximum tolerated dose is selected for further neuroprotective studies.

Animals

For neuroprotective toxicity studies, healthy adult Wistar rat of either sex weighing 150–200 g are used. The animals are obtained from a registered animal house and housed in polypropylene cages (6–8 per cage) under standard laboratory conditions at 22–25 °C with 45–55% relative humidity and a 12-hour light/dark cycle. They are provided with a standard pellet diet and water ad libitum, except during the fasting period prior to dosing. Animals are acclimatized to the laboratory environment for at least 7 days before the experiment. All experimental procedures are carried out in accordance with institutional animal ethics committee guidelines and CPCSEA regulations.

Animal Grouping - Neuroprotective study

Healthy adult Wistar rats are randomly divided into five groups (n = 6 per group) for evaluation of the neuroprotective potential of Maerua oblongifolia leaf extract:

- **Group I:** Normal control (vehicle only)
- **Group II:** Disease control (Scopolamine induced, 1 mg/kg, i.p.)
- **Group III:** Standard drug + scopolamine (e.g., Donepezil 1–3 mg/kg, p.o. + scopolamine 1 mg/kg, i.p.)
- **Group IV**:Maerua oblongifolia leaf extract + scopolamine (e.g., 200 mg/kg, p.o.)
- **Group V**: Maerua oblongifolia leaf extract + scopolamine (e.g., 400 mg/kg, p.o.)

All treatments are given for 7–14 days depending on the experimental design, and scopolamine

Scopolamine Induced

Healthy adult wistar rats are acclimatized to laboratory conditions for 7 days prior to the experiment. Animals are divided into different groups such as control, scopolamine control, standard drug, and test drug groups. The test drug/plant extract and standard drug are administered orally once daily for a predetermined period (e.g., 7–14 days). On the test day, scopolamine is injected at a dose of 1 mg/kg, intraperitoneally (i.p.), 30 minutes before the behavioral assessment to induce memory impairment by blocking central muscarinic receptors. Behavioral tests such as the Morris Water Maze, Y-maze, or Passive Avoidance are then conducted to assess learning and memory. Parameters like escape latency, time spent in the target quadrant, spontaneous alternation, and step-down latency are recorded. The reduction in scopolamine-induced cognitive deficits in treated groups indicates the neuroprotective or memory-enhancing effect of the test substance.

Morris Water Maze [13]

Procedure

Healthy adult Wistar rats are acclimatized to the laboratory environment for at least 7 days before testing. The apparatus consists of a circular pool filled with opaque water maintained at 25 ± 1 °C and divided into four imaginary quadrants. A small escape platform is submerged 1–2 cm below the water surface in the center of one quadrant, and distinct visual cues are placed around the room. During the acquisition (training) phase, conducted for 4–5 consecutive days, each rat is subjected to four trials per day from different starting points and allowed 60–120 seconds to locate the hidden platform. If the rat fails to find the platform, it is gently guided to it and allowed to remain there for 15–20 seconds. Escape latency, path length, and swim speed are recorded. Twenty-four hours after the last training session, a probe trial is performed by removing the platform and allowing the rat to swim freely for 60 seconds. The time spent in the target quadrant and the number of crossings over the previous platform location are measured to assess memory retention.

Elevated Plus Maze [14]

Healthy adult Wistar rats are acclimatized to the laboratory environment for at least 7 days prior to testing. The apparatus consists of a plus-shaped maze elevated about 50 cm above the floor, with two open arms and two closed arms of equal dimensions connected by a central platform. On the training day, each rat is placed individually at the end of an open arm facing away from the central platform. The time taken by the rat to move from the open arm into one of the closed arms is recorded as transfer latency (TL), with a cut-off time of 90 seconds. The rat is allowed to explore the maze for an additional 20 seconds and then returned to its home cage. After 24 hours, the retention trial is conducted in the same manner, and the transfer latency is again recorded. A decrease in transfer latency on the retention day compared to the training day indicates improved learning and memory, while an increase suggests memory impairment.

Y-Maze test [15]

Healthy adult Wistar rats are acclimatized to the laboratory for at least 7 days before testing. The Y-maze apparatus consists of three identical arms (labeled A, B, and C) positioned at 120° to each other. Each rat is placed at the end of one arm and allowed to explore the maze freely for 8 minutes. The sequence and number of arm entries are recorded. An entry is considered when all four paws of the rat enter an arm. Spontaneous alternation behavior is calculated as successive entries into all three different arms (e.g., ABC, BCA, CAB). The percentage alternation is determined using the formula:

$$\% \text{ Alternation } = \frac{\text{Number of alternations}}{\text{Total arm entries} - 2} \times 100$$

A higher percentage of spontaneous alternation indicates better working memory, while a reduction suggests memory impairment.

TO ESTIMATE BRAIN BIOCHEMICAL MARKERS

Acetyl cholinesterase assay (AChE) [16]

Acetyl cholinesterase activity in brain tissue is estimated by Ellman's colorimetric method. A 10% (w/v) brain homogenate is prepared in ice-cold 0.1 M phosphate buffer (pH 7.4) and centrifuged at 10,000 rpm for 15 minutes at 4 °C. The supernatant is used for the assay. In a cuvette, add 2.6 ml of phosphate buffer (pH 8.0), 0.4 ml of brain homogenate, and 0.1 ml of DTNB (5, 5′-dithiobis-2-nitrobenzoic acid). After mixing, add 0.02 ml of acetylthiocholine iodide as the substrate to initiate the reaction. The increase in yellow color due to the formation of Thio nitro benzoate ions is measured spectrophotometrically at 412 nm for 2 minutes at 30-second intervals. The enzyme activity is expressed as µmoles of acetylthiocholine hydrolyzed per minute per mg of protein.

Malondialdehyde (MDA) - Lipid Peroxidation (TBARS method) [17]

Lipid peroxidation in brain tissue is estimated by measuring malondialdehyde (MDA) levels using the thiobarbituric acid reactive substances (TBARS) method. A 10% (w/v) brain homogenate is prepared in ice-cold 0.1 M phosphate buffer (pH 7.4) and centrifuged at 10,000 rpm for 15 minutes at 4 °C. To 1 ml of the supernatant, add 2 ml of TBA–TCA–HCl reagent. The mixture is heated in a boiling water bath for 15 minutes, then cooled and centrifuged. The pink chromogen formed due to the reaction between MDA and thiobarbituric acid is measured spectrophotometrically at 532 nm. The results are expressed as nmoles of MDA per mg of protein.

Superoxide dismutase[18]

Superoxide dismutase activity in brain tissue is estimated by its ability to inhibit the auto-oxidation of epinephrine at alkaline pH. A 10% (w/v) brain homogenate is prepared in ice-cold 0.1 M phosphate buffer (pH 7.4) and centrifuged at 10,000 rpm for 15 minutes at 4 °C. In a cuvette, add 2.5 ml of carbonate buffer (0.05 M, pH 10.2) and 0.1 ml of EDTA, followed by 0.05 ml of the supernatant. The reaction is initiated by adding 0.3 ml of freshly prepared epinephrine solution. The increase in absorbance due to epinephrine auto-oxidation is recorded at 480 nmfor 4 minutes. One unit of SOD activity is defined as the amount of enzyme required to inhibit 50% of epinephrine auto-oxidation, and the results are expressed as units per mg of protein.

Catalase (CAT) [19]

Catalase activity in brain tissue is measured by monitoring the decomposition of hydrogen peroxide. A 10% (w/v) brain homogenate is prepared in ice-cold 0.1 M phosphate buffer (pH 7.4) and centrifuged at 10,000 rpm for 15 minutes at 4 °C. In a cuvette, add 1.9 ml of phosphate buffer and 0.1 ml of the supernatant. The reaction is initiated by adding 1.0 ml of hydrogen peroxide solution. The decrease in absorbance due to the breakdown of H_2O_2 is recorded spectrophotometrically at 240 nm for 1 minute. Catalase activity is expressed as µmoles of hydrogen peroxide decomposed per minute per mg of protein.

Glutathione (Gsh) [20]

Reduced glutathione in brain tissue is estimated using Ellman's reagent (DTNB). A 10% (w/v) brain homogenate is prepared in ice-cold 0.1 M phosphate buffer (pH 7.4) and centrifuged at 10,000 rpm for 15 minutes at 4 °C. To 1 ml of the supernatant, add 1 ml of 10% trichloroacetic acid (TCA) to precipitate proteins and centrifuge. Take 0.5 ml of the clear supernatant and add 2 ml of phosphate buffer followed by 0.25 ml of DTNB solution. The yellow color formed is measured spectrophotometrically at 412 nm. The results are expressed as µg of GSH per mg of protein.

Results

Percentage of yield

$$Percentage\ of\ yield = \frac{Weight\ of\ crude\ extract}{Weight\ of\ dried\ material} \times 100$$

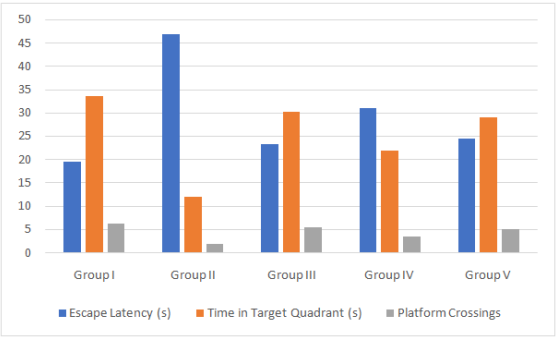
$$Percentage\ of\ yield = \frac{26g}{200g} \times 100 = 13\%$$

200gms of drug dissolved in 800ml of solvent in 1:4 Ratio

Morris Water Maze

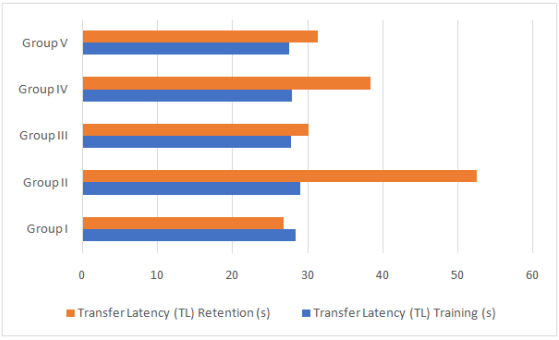
Group	Treatment	Escape Latency (s)	Time in Target Quadrant (s)	Platform Crossings
Group I	Normal control (vehicle)	19.4 ± 1.2	33.6 ± 1.8	6.1 ± 0.5
Group II	Disease control (Scopolamine 1 mg/kg, i.p.)	46.8 ± 2.5	11.9 ± 1.4	1.7 ± 0.2
Group III	Standard (Donepezil + Scopolamine)	23.2 ± 1.6	30.1 ± 2.0	5.3 ± 0.4
Group IV	*Maerua oblongifolia* 200 mg/kg + Scopolamine	31.0 ± 2.1	21.8 ± 1.5	3.4 ± 0.3
Group V	*Maerua oblongifolia* 400 mg/kg + Scopolamine	24.5 ± 1.3	28.9 ± 1.9	4.9 ± 0.4

Scopolamine significantly impaired spatial learning and memory. Donepezil showed marked reversal of cognitive deficits. Maerua oblongifolia extract produced a dose-dependent improvement, with the high dose showing effects comparable to the standard drug.



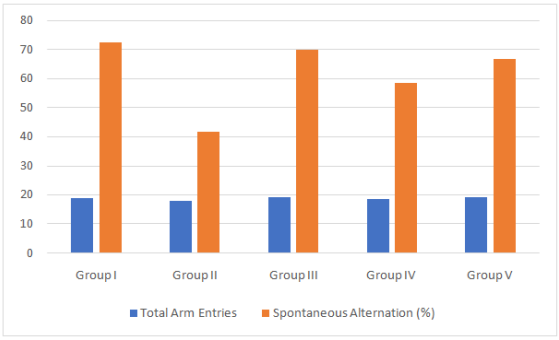
Elevated Plus Maze

Group	Treatment	Transfer Latency (TL) Training (s)	Transfer Latency (TL) Retention (s)
Group I	Normal control (vehicle)	28.4 ± 1.6	26.9 ± 1.4
Group II	Disease control (Scopolamine 1 mg/kg, i.p.)	29.1 ± 1.8	52.6 ± 2.3
Group III	Standard (Donepezil + Scopolamine)	27.8 ± 1.5	30.2 ± 1.7
Group IV	*Maerua oblongifolia* 200 mg/kg + Scopolamine	28.0 ± 1.4	38.5 ± 2.0
Group V	*Maerua oblongifolia* 400 mg/kg + Scopolamine	27.6 ± 1.3	31.4 ± 1.6



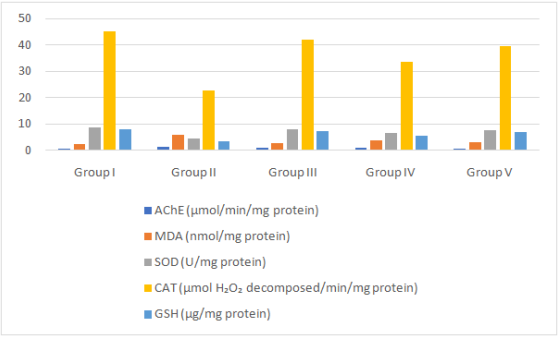
Y-Maze

Group	Treatment	Total Arm Entries	Spontaneous Alternation (%)
Group I	Normal control (vehicle)	18.6 ± 1.2	72.4 ± 2.5
Group II	Disease control (Scopolamine 1 mg/kg, i.p.)	17.9 ± 1.4	41.6 ± 2.1
Group III	Standard (Donepezil + Scopolamine)	19.2 ± 1.3	69.8 ± 2.3
Group IV	*Maerua oblongifolia* 200 mg/kg + Scopolamine	18.5 ± 1.1	58.3 ± 2.0
Group V	*Maerua oblongifolia* 400 mg/kg + Scopolamine	19.0 ± 1.2	66.7 ± 2.2



TO ESTIMATE BRAIN BIOCHEMICAL MARKERS

Group	Treatment	AChE (µmol/min/mg protein)	MDA (nmol/mg protein)	SOD (U/mg protein)	CAT (µmol H_2O_2 decomposed/min/mg protein)	GSH (µg/mg protein)
Group I	Normal control (vehicle)	0.48 ± 0.03	2.10 ± 0.12	8.6 ± 0.41	45.2 ± 2.10	7.8 ± 0.35
Group II	Disease control (Scopolamine 1 mg/kg, i.p.)	0.92 ± 0.05	5.80 ± 0.25	4.1 ± 0.30	22.6 ± 1.40	3.2 ± 0.20
Group III	Standard (Donepezil + Scopolamine)	0.55 ± 0.04	2.45 ± 0.15	7.9 ± 0.38	41.8 ± 1.90	7.1 ± 0.30
Group IV	*Maerua oblongifolia* 200 mg/kg + Scopolamine	0.67 ± 0.04	3.60 ± 0.18	6.2 ± 0.35	33.5 ± 1.60	5.4 ± 0.25
Group V	*Maerua oblongifolia* 400 mg/kg + Scopolamine	0.52 ± 0.03	2.70 ± 0.14	7.4 ± 0.36	39.6 ± 1.80	6.8 ± 0.28



Discussion

In the present study, the neuroprotective potential of Maerua oblongifolia leaf extract was evaluated in scopolamine-induced cognitive impairment in Wistar rat using behavioral models such as Morris Water Maze, Elevated Plus Maze, and Y-Maze along with biochemical estimations. Scopolamine (1 mg/kg, i.p.) produced significant impairment in learning and memory,

evidenced by increased escape latency, increased transfer latency, and decreased spontaneous alternation behavior, confirming successful induction of cholinergic dysfunction and memory deficits. Biochemical analysis showed a marked increase in acetylcholinesterase (AChE) activity and lipid peroxidation marker Malondialdehyde (MDA), indicating enhanced cholinergic breakdown and oxidative stress. A significant reduction in antioxidant defense enzymes such as Superoxide dismutase (SOD), Catalase (CAT), and Reduced glutathione (GSH) further confirmed oxidative neuronal damage induced by scopolamine. Treatment with Donepezil significantly reversed both behavioral and biochemical alterations, validating the experimental model. Importantly, Maerua oblongifolia leaf extract demonstrated a dose-dependent neuroprotective effect. The high-dose group showed significant improvement in learning and memory parameters across all behavioral models, along with restoration of antioxidant enzyme levels and reduction in AChE and MDA levels. These effects suggest that the extract may enhance cholinergic transmission and attenuate oxidative stress-mediated neuronal damage. The observed neuroprotective activity of Maerua oblongifolia may be attributed to its photochemical constituents such as flavonoids, tannins, and phenolic compounds, which are known for their antioxidant and free radical scavenging properties. Overall, the findings indicate that Maerua oblongifolia possesses significant neuroprotective potential against scopolamine-induced cognitive dysfunction, possibly through modulation of cholinergic activity and oxidative stress pathways.

Conclusion

The present study demonstrates that the leaf extract of Maerua oblongifolia exhibits significant neuroprotective activity against scopolamine-induced cognitive impairment in Wistar rats. Behavioral assessments using Morris Water Maze, Elevated Plus Maze, and Y-Maze confirmed that the extract improved learning and memory deficits in a dose-dependent manner. Biochemical findings further supported these results, showing that the extract reduced acetyl cholinesterase activity and lipid peroxidation while restoring antioxidant enzyme levels, including SOD, CAT, and GSH. These effects suggest that the neuroprotective action may be due to both cholinergic enhancement and antioxidant mechanisms.

Maerua oblongifolia leaf extract demonstrates promising memory-enhancing and neuroprotective potential, indicating its possible usefulness in the management of neurodegenerative disorders such as Alzheimer's disease. Further detailed molecular and clinical studies are recommended to confirm its therapeutic application.

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