RESEARCH ARTICLE

Nootropic Effect of *Premna mucronata* Roxb Leaves on Scopolamine Challenged Alzheimer's Disease in Rats

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ABSTRACT

The leaves of *Premna mucronata Roxb* were selected for this study due to their ability to enhance cognitive function and memory in Swiss albino rats. Each mouse group was provided with scopolamine (0.4 mg/kg) intraperitoneally per kg body-weight, as well as two split doses of methanol extraction of *P. mucronata Roxb*-MEPM with 200 & 400 mg/kg (oral), for a duration of 7 days. In order to assess memory and learning, researchers employed sophisticated behavioral models such as the Morris water maze and raised plus maze. However, scopolamine is a naturally occurring substance that induces a cognitive state used as an interoceptive model. One-way ANOVA & Dunnett's multiple comparison tests were employed with $p < 0.05$. The outcomes of this study showed that MEPM ($p < 0.05$) enhanced short-term & abstraction remembrance at dosages of 200 & 400 mg/kg. Additionally, there was a remarkable reduction in transfer latency on the sixth and seventh days, indicating improved learning and memory by navigating through the complex maze and reducing the time it takes to escape from the Morris water maze. Results indicate that *P. mucronata Roxb* showed considerable memory-enhancing efficacy in all the screening models tested.

Keywords: *Premna mucronata Roxb*, Learning, Memory, Elevated plus maze, Morris water maze.

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INTRODUCTION

Alzheimer's disease (AD) can lead to a broad & permanent decrease in cognitive function, accompanied by the formation of senile plaque in the Hippocampus.[1] It is the predominant variety of dementia, comprising 60 to 80% of all cases.[2,3] Commencement of dementia before 65 years is noted by rapid development & presence of several cortical utility issues. However, dementia that develops later in life mostly affects memory features and tends to worsen over time.[4] The illness has a slower progression and deteriorates due to heightened somatic cell death.[5] Dementia is a progressive neurodegenerative disease characterized by loss of nerve cells in particular locations.[6] Based on a medical survey, it has been found that dementia or mental impairment might be a substantial unexpected outcome for the Indian population.[7,8] The incidence of dementia can increase rapidly as a person becomes older, and this process of ageing in animal models is linked to a gradual decrease in sensory & motor abilities at regular time points in the brain. Decrease in sensory & motor function is still ascribed to the oxidative damage that results from oxidative stress on proteins, lipids, and nucleic acid.[9] Various antioxidant treatments and flavonoid components have the potential to be beneficial in preventing age-related deficits and protecting brain function.[10,11] Alzheimer's illness is now not treatable. Doctors use medicine to alleviate the symptoms of alertness, migratory behavior, nervousness, sadness, and depression that often occur with AD. Currently, tacrine hydrochloride (HCL) (Cognex), rivastigmine (Exelon), donepezil HCL (Aricept), galantamine (Razadyne), and several other medications are being utilised.[12] They enhance the efficiency of nerve cells that are adversely influenced by Alzheimer's disease. Despite this, the results are temporary and do not address the underlying issue. In order to decelerate the process of protein production, scientists hunted for chemical compounds which impede the "parent" molecule of β-amyloid supermolecule.[13] Research undertaken in adverse situations has demonstrated minor yet essential enhancements in functioning with AD.[14] Scientists are conducting experiments to determine the potential of different antioxidants in safeguarding nerve cells. Researchers have shown that beta-amyloid proteins have the potential to accumulate and potentially cause ototoxicity. Reducing the buildup of proteins will mitigate the extent of damage caused.[15] Herbal medicines, utilized since ancient religious writings, offer a remarkable alternative for exploring and developing contemporary pharmaceuticals. It is well recognized that 80% of medicine ingredients are

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sourced from natural chemicals or herbal items.[16] The Western pharmaceutical industry experienced a surge in the use of herbal medicines for advanced human therapeutics from 1970 to 1980. The use of natural compounds effectively stimulated advancements in the field of pharmacology.[17] Herbal medicine offers several choices to modify the condition and symptoms of an ailment. The teaching and marketing of medicine have seen a change in focus, moving away from the use of medicinal flowers. However, the clinical and commercial significance of these flowers seems to be increasing in areas that are pertinent to health. The plant-based drugs underwent meticulous standardization, and proof of their effectiveness and safety for a specific software system was shown.[18] According to traditional knowledge, the plant possesses a range of medical qualities like anticancer, antioxidant, anti-inflammatory, bactericidal, antifungal, & antifertility effects. Moreover, it is utilized to mitigate anxiousness. The present study was designed to assess whether MEPMs have nootropic effects on the scopolamine mouse model of AD.

Premna mucronata Roxb, also called "Ganiar", "Bari arani", "Agnimatha", "Agethu", & "Agyon", is small, shaggy tree with a stalk that may reach a height of up to 1.2 meters. It belongs to the Verbenaceae family and holds significant ethnobotanical importance.[19] Additionally, *P. mucronata* has medicinal & fragrant characteristics, and it's a significant component in the herbal concoction known as "Dashamula".[20] The bark is used to cure boils, while the leaves are administered topically as a diuretic to treat dropsy. It was found that *P. mucronata* had cardioprotecting effects due to antioxidant content.[21] Additionally, it has larvicidal effects. This plant has been shown to possess antimicrobial, hypocholesterolemic, & wound curing properties, along with anti-inflammatory actions.[22] Recent investigations have demonstrated that the essential oil of *P. mucronata* possesses notable anti-feedant effects.[23] The study investigated the nootropic possessions of the MEPM leaves on an AD animal model induced by scopolamine.

MATERIALS AND METHODS

Collection and Recognition of Plant

Dr. K. Madhava Chetty, Assistant Professor, Department of Botany at Sri Venkateswara University, Tirupati, authenticated the plant. A specimen (Pt 0823&Pt 0754) got preserved in herbarium.

Extraction

A total of 5 kilograms of clean leaves were subjected to shade drying at temperatures ranging from 25 to 35°C for 7 days. The desiccated botanical matter got pulverized using a grinding apparatus. The desiccated botanical powder underwent soxhlet extraction utilizing methanol for extraction. Subsequently, each extract underwent filtration by cotton plugs, followed by filtration (Whatman filter). Filtrates got further intensified, desiccated at low pressure using a rotary evaporator, & finally freeze-dried for a powdered type.

Initial Phytochemical Analysis

Primary and secondary metabolites of *P. mucronata* were analysed in all of its extract/fractions to assure the presence of many 1^{o} metabolites, including amino acids, carbohydrates, proteins, & lipids, as well as 2^{o} metabolites, such as tannins, alkaloids, saponins, phenols, steroids, flavonoids, glycosides, & resins, using established ways.

Animals

For this investigation, a total of 30 healthy, fully developed male Wister albino rats (200–230 g) were obtained from Vab Bioscience, located in Hyderabad, Telangana. The rats were housed in polypropylene cages, with 6 rats per cage. The enclosures were upheld per established environmental protocols, which included a temperature regulation in 25 ± 2˚C, a relative humidity of 60 ± 5%, aand The rats were provided with an ample supply of food and drink. The animals were utilized while ensuring that their welfare was maintained in strict adherence to the laboratory animal standards established by the National Institutes of Health.[22] The experimental procedure received endorsement from the Institutional Animal Ethics Committee under reference number 1447/PO/Re/S/11/CPCSEA-74/A.

Scopolamine-induced Alzheimer's Disease

The objective is to produce memory impairment in rats by administering scopolamine (1-mg/kg) by intraperitoneal injection. Scopolamine is a specific antagonist of muscarinic acetylcholine receptors. The study aims to assess this treatment's behavioral and biochemical consequences on rats. Subjects got categorized into five groupings (n = 6). The control group had distilled water. Group II, the disease group, had only scopolamine (0.4 mg/kg intraperitoneally). Group III was treated with both standard piracetam (400 mg/kg intraperitoneally) and scopolamine (0.4 mg/kg intraperitoneally). Groups IV and V were given 2 doses of MEPM, 200 & 400 mg/kg orally, respectively, along with scopolamine (0.4 mg/kg intraperitoneally). These treatments were administered for of 7 days. Memory activity evaluation was analyzed using the following tests 1-hour after administering the extract. Each test was performed once using the animals. Evaluation of memory function.

Elevated Plus Maze (EPM)

The object comprises two exposed arms with 16 and 5 cm and two enclosed arms with 16, 5, and 12 cm, all of which have been utilised before. The height of the labyrinth was increased to 25 cm. The animals were individually positioned to the end of an exposed arm opposite the platform. The duration it takes for subjects to relocate to one enclosed arm was documented. Transfer latency (TL) refers to the time delay that occurs during data transfer. If any animal failed to enter any of the two closed arms within 90 seconds, it was lightly nudged to any arm, & the chemical element got allocated for a duration of 90 seconds. Afterward, the animals were allowed to freely navigate the maze for an additional 10 seconds before being brought back to their original cage. Following the initial trial,

a 24-hour evaluation was conducted to assess the recall of the learned task. The diagrammatic technique devised by Jaiswal and Bhattacharya (1992) allows us to describe the TL once every 24 hours as the "Inflexion magnitude relation IR". This relation is defined as IR = (L1-L0)/L0,
Where, L0 represents the TL once every 24 hours, and
L1 represents the early TL in seconds.[24]

Morris Water Maze

It is a spherical puddle with a lackluster inside surface that has a width of 120 cm & a height of 50 cm. Pool's depth (35 cm) and volume of 500 mL (milk; 20°C) were sufficient. The puddle got partitioned to four congruent sections, one in each quadrant. Following this, a segment of the puddle got partitioned by a slender frigid platform with a diameter of 6 centimetres and a vertical dimension of 29 cm. During initial study, a 60-second spinning exercise was conducted without the use a flooded platform for the subsequent five days. Subjects were split into two daily trials, with a 30-minute break between them in the centre of the stage. If the subjects were unable to locate the platform within 1.5 minutes, they were allowed to stay in the center for an additional 10 seconds. The subject was transported to its enclosure at home & let to arid naturally. The latency was recorded, which refers to the time required to detect the concealed platform in each trial session. Following the last training session, the rats had a check trial session in which the platform was removed from the puddle. The rats were then allowed to whirl freely for 120 seconds in an attempt to locate the platform. A record was continuously spinning in the puddle quadrant where the platform was previously positioned. The rats got administered scopolamine (0.4 mg/kg) to induce memory impairment 60 minutes post-treatment with test samples. The management cluster was administered normal saline.[25]

Assessment of Biochemical Attributes

Brain tissue assignment

Subjects got euthanized on day 14 following last behavioral exam while in chloroform anesthesia. Cranium was incised & the cerebral cortex got revealed through its posterior aspect. The whole brain got promptly extracted & rinsed with cold normal saline with ice. A 10% (w/v) brain sample homogenate was produced using sodium phosphate buffer (0.03 M) with a pH of 7.4. Homogenate underwent centrifugation at 3000 rpm; for 15 minutes. The resulting supernatant was utilised for measuring activities of SOD & catalysis (CAT), along with determining total GSH substance & MDA point. Furthermore, acetylcholinesterase (AChE) & acetylcholine (ACh) levels were assessed.

Antioxidant Enzymes Analysis

Estimation of GSH

Tissue homogenate was obtained and transferred into phosphate buffer solution (0.1 M) with a pH of 7.4 to measure reduced glutathione (GSH). The procedures were carried out in accordance with the methodology that Shamnas *et al*. had previously delineated.[26] To cause proteins to separate out, a solution of trichloroacetic acid (TBA, 20%) having 1-mM EDTA was added to homogenate in an equal amount. Before centrifugation, the mixture was left undisturbed for 5 minutes. Next, another batch of test tubes was filled with the supernatant (200 mL), and 1.8 mL of Ellman's reagent (5, 5'-dithio bis-2-nitrobenzoic acid; 0.1 mM) was introduced. The reagent was prepared using phosphate buffer solution (0.3 M) & 1% sodium citrate solution. Subsequently, all test tubes increased in volume to 2 mL. Once the whole reaction was finished, the solutions were analyzed at 412 nm compared to blank. The absorbance results were compared to a standard plot derived from identified concentrations of GSH.

CAT analysis

The Hritcu *et al*.[27] approach was employed to quantify the CAT activity. The resulting mix consisted of supernatant (100 mL) and phosphate buffer solution (150 mL) with 0.01 M concentration & pH of 7.0. Following the introduction of 250 L of H_2O_2 at 0.16 M and one minute of incubation at 37°C, the reaction was terminated by the introduction of 1.0 mL of dichromate acetic acid reagent. The spectrophotometer detected the green color that appeared throughout the reaction at a wavelength of 570 nm. Tubes got right away put in a boiling bath with water and kept there for 15 minutes. Simultaneously, control tubes without enzymes were also subjected to treatment. The catalase activity was quantified based on the change in absorbance per unit.

Determination of malondialdehyde

The quantification of determination of malondialdehyde (MDA) (indicator of lipid peroxidation), got performed by thiobarbituric acid test and spectrophotometry.[28] About 200 liters of supernatant were mixed with 1-mL each of 26 millimolar thiobarbituric acid and 50% trichloroacetic acid in 0.1 molar hydrochloric acid for a short period of time. The samples were incubated at 95°C for 20 minutes following vortex mixing. In addition, the samples were centrifugated at 3000 rpm by 10 minutes, after which liquid segments above the sediment were analyzed at a wavelength of 532 nanometers. The reference standard utilized to generate a calibration plot was MDA, & the findings got reported in nmoln per g protein.

Brain neurotransmitter analysis

Quantifying the enzymatic function of AChE and Ach was dobe by acetylthiocholine (ATC). ATC, a man-made substance, was employed to determine the level of AChE activity. Thionitrobenzoic acid (TBA) is generated in the medium through the reaction between thiocholine, which is obtained from the degradation of ATC by AChE, and the -SH reagent 5,5'-dithiobis-2–nitrobenzoic acid. TBA may be detected at 412 nm. The content of TBA was measured using spectrophotometry & served as a straight indication of AChE activity.[29] Stepankova *et al*.[29] utilized the hydroxylamine approach to quantify the concentration of acetylcholine in the whole brain. In a 1:1 volume-to-volume ratio, aqueous hydroxylamine hydrochloride and 3.5 M aqueous KOH were

combined to form the reaction mixture. In order to fully convert ACh into acetohydroxamic acid, the liquid was vigorously agitated for a duration of two minutes. The pH level was further modified by the addition of concentrated hydrochloric acid and water in a 1:2 volume-to-volume ratio. The reddish-brown colour resulting from the addition of ferric nitrate (0.37 M) got detected at a wavelength of 540 nm. Ach got estimated using usual readings.

Histopathology-brain

Following the therapy and behavioral testing, two subjects from each grouping got euthanized using severe anesthesia (CO_2). Brains got then separated & preserved in a 10% formaldehyde solution. The brain was subjected to staining by cresyl violet, and cerebellum & basal ganglia were examined using a light microscope.[30,31]

Statistical Analysis

Data collected from the raised plus maze & MWM experiments got analysed by a one-way ANOVA with Dunnett's multiple comparison tests. $p < 0.05$ was deemed to be significant statistically.

RESULTS

Phytochemical Test

The tests conducted for proteins, amino acids, carbohydrates, alkaloids, triterpenoids, cardiac glycosides, flavonoids, saponins, tannins, phenolic compounds, and steroids were positive, while gums showed negative results.

The effect of MEPM on TL by EPM is reported in Table 1 and Figure 1.

The findings of the raised plus maze is presented in Table 2. Following the administration of scopolamine-induced amnesia, the healthy control group, and the combination of both on the sixth and seventh days, it was anticipated that low doses of MEPM (200 mg/kg) & elevated doses of MEPM (400 mg/kg) would result in highly significant effects ($p < 0.01$) on TL, while low doses would produce slight significance effects ($p < 0.05$). The animals administered with varying dosages saw a substantial decline in their learning & memory capacity on 6th & 7th days, as estimated by their TL. Scopolamine (0.4 mg/kg i.p.) before training resulted in a substantial rise in TL on the sixth and seventh days, suggesting a decline in learning and memory abilities. Piracetam, a commonly prescribed medicine, significantly reduced metal content

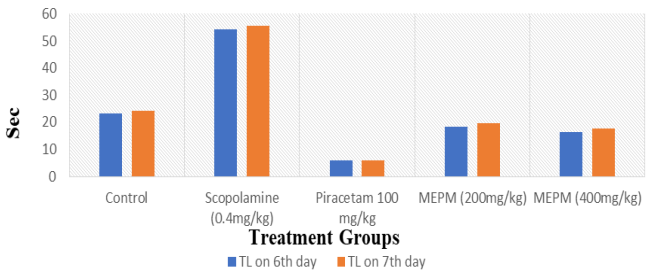

Figure 1: Effect of MEPM on TL by EPM

at 100 mg/kg. Piracetam denoted a more pronounced and statistically significant effect ($p < 0.001$) on learning and memory cin comparison to both standard & control groups, indicating its superior efficacy. Although both MEPM doses led to a significant reduction in TL, the greater dose proved to be more effective than the lower dosage (Table 2, Figure 2).

Morris created the water maze as a navigational challenge using water. The escape delay in scopolamine-evoked animals is significantly increased [(4, 25) = 66.05] (p 0.001) compared to the norm. The F statistic with a value of 4.25 corresponds to a *p-value* of less than 0.001. On the sixth and seventh days, the administration of a modest dosage (50 mg/kg) of MEPM did not show any statistical significance (p 0.8295), (p 0.7727). On the sixth day, administering a greater dosage of MEPM at 250 mg/kg showed a little significance (p 0.0238) and a significant significance (p 0.0024). Regarding learning and memory, the animals who received a higher dosage of MEPM denoted a significant decrease in the time it took them to escape, by 6 to 7 days (Table 3, Figure 3).

A reduction in MDA levels in the brain signifies an augmentation in memory-preserving activity. Table 3 demonstrates that MEPM 400 mg/kg reported a substantial reduction in MDA levels compared to the normal control group. However, the findings for MEPM were not statistically significant at levels of 200 mg/kg. Levels of brain MDA increased in the negative control group treated with scopolamine: Contrarily, piracetam unexpectedly increased levels of MDA in the brain.

When the rats were administered MEPM at all dosages, there was a dose-dependent increase in GSH levels, indicating that it had a beneficial effect on memory and learning. However, rats treated with diazepam exhibited a decrease in GSH levels, as seen in Table 3 and Figure 4.

Table 1: Effect of MEPM on TL EPM

Treatments	*6th day*	*7th day*
Control	23.32 ± 1.54	24.24 ± 1.46
Scopolamine (0.4 mg/kg)	54.43 ± 2.43	55.43 ± 1.32
Piracetam 100 mg/kg	5.82 ± 0.43***	5.92 ± 1.42***
MEPM (200 mg/kg)	18.32 ± 1.34**	19.53 ± 1.54**
MEPM (400 mg/kg)	16.45 ± 1.65*	17.56 ± 1.64*

Results are expressed in mean ± SEM (n = 6); statistical significance at $p < 0.05$*, 0.01** and 0.001***, in comparison to control group

Table 2: Effect of MEPM on escape latency by MWM

Treatments	*6th day*	*7th day*
Control	25.32 ± 2.32	26.42 ± 2.52
Scopolamine (0.4 mg/kg)	38.53 ± 1.62	43.21 ± 0.85
Piracetam 100 mg/kg	11.65 ± 0.91***	12.76 ± 0.32***
MEPM (200 mg/kg)	19.42 ± 0.43**	19.53 ± 1.52**
MEPM (400 mg/kg)	23.32 ± 1.23	25.32 ± 1.34

Results are expressed in mean ± SEM (n = 6); statistical significance at $p <$ 0.05*, 0.01** and 0.001***, in comparison to control group

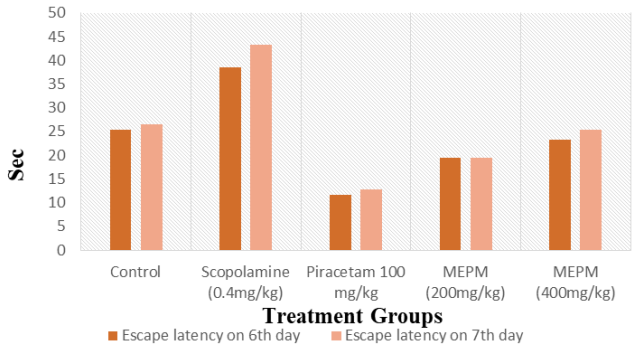

Figure 2: Effect of MEPM on escape latency by MWM

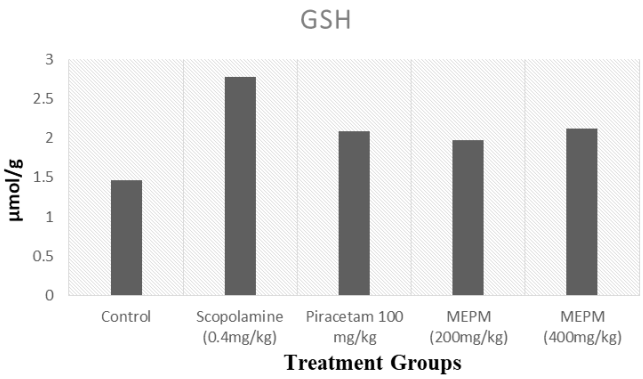

Figure 4: Nootropic effect of MEPM on GSH

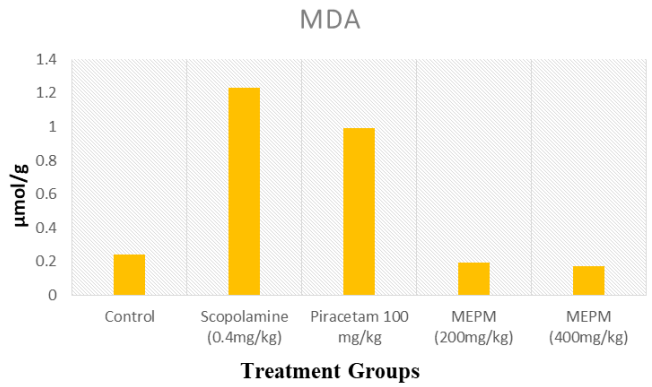

Figure 3: Nootropic effect of MEPM on MDA

Figure 5 indicates changes in antioxidant enzyme activity in the homogenised brain of rats. Rats administered with MEPM at 200 & 400 mg/kg body weight denoted a substantial ($p < 0.05$, $p < 0.01$) elevation in CAT levels.

Table 3 presents the AChE activity in the soluble supernatant (SS) and dense sediment (DS) portions of the homogenized rat brain tissue. The piracetam group had a considerably decreased level of brain AChE activity in both the SS and DS sections of brain tissue homogenate in comparison to disease group ($p < 0.05$, $p < 0.01$). Compared to disease group, the treatment of MEPM 200 & 400 mg/kg b.w. radically reduces the activity of AChE in both the SS and DS areas of rat brain tissue homogenate, as indicated by the *p-value*s of 0.05 & 0.01, correspondingly.

Histopathological Analysis

The histopathological investigation findings are presented in Figure 7. 7A-Normal control simply displays the level of stress-induced neuronal degeneration. 7B- Negative control exhibits vascular deterioration, neuronal degeneration, and infiltration of glial cells. 7C-Regular control exhibits a lower degree of vascular degradation, neuronal degeneration, and glial cell infiltration compared to negative control. 7D-Low dosage of extract demonstrates a reduced level of vascular deterioration, neuronal degeneration, and glial cell infiltration compared to negative control. 7E-High extract dosage exhibits vascular deterioration, neuronal deterioration, and glial cell infiltration, which is less than that observed in negative control. Group II, which was treated with scopolamine, had the most significant pathological alterations in comparison to the other groups. Both the low dosage and high dose of MEPM exhibited a favorable regeneration score in comparison to the other group.

DISCUSSION

The recent focus on ongoing research indicates that MEPM has cognitive-enhancing benefits on scopolamine-induced Alzheimer's disease in rats. Scopolamine, a naturally occurring compound extracted from the Solanaceae plant *Datura stramonium*, negatively affects both the immediate and long-lasting recollection abilities of both animals and human.[32] Hyoscine induces oxidative stress and cognitive impairment by interfering with neurotransmitters in the brain. Thus, the memory impairment caused by scopolamine might be a valuable model for assessing the anti-amnesic effects

Table 3: Nootropic effect of MEPM on brain oxidative status

Treatment	*Biochemical parameters (on day 14)*			
	AChE activity (U/g)	*MDA (µmol/g)*	*GSH (µmol/g)*	*Catalase (units/mg)*
Control	16.9 ± 1.09	0.24 ± 0.016	1.47 ± 0.10	0.71 ± 0.063
Scopolamine (0.4 mg/kg)	25.5 ± 1.23	1.23 ± 0.065	2.78 ± 0.14	0.41 ± 0.054
Piracetam 100 mg/kg	10.41 ± 0.61**	0.99 ± 0.01**	2.09 ± 0.079**	0.63 ± 0.076***
MEPM (200 mg/kg)	13.58 ± 0.58	0.192 ± 0.01	1.98 ± 0.03**	0.57 ± 0.075***
MEPM (400 mg/kg)	11.90 ± 0.73**	0.172 ± 0.02*	2.12 ± 0.01**	0.60 ± 0.053***

Results are expressed in mean ± SEM (n = 6); statistical significance at $p < 0.05$*, 0.01** and 0.001***, in comparison to control group

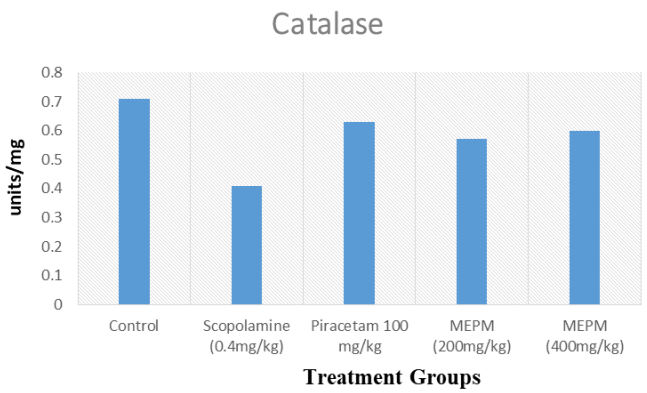

Figure 5: Nootropic effect of MEPM on catalase

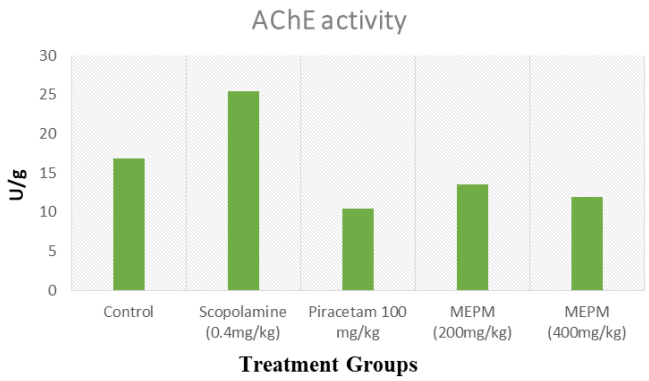

Figure 6: Nootropic effect of MEPM on AChE activity

of the latest medicine. Animals are commonly utilized to evaluate & support the latest findings for dementia.[33] In the present study, rats were given MEPM for seven days before the experiment. The results showed that this pre-treatment effectively counteracted the decrease in the percentage of spontaneous alternation caused by scopolamine. This indicates that MEPM has a considerable positive effect on short-term memory relevant to the specific region being studied. In the present study, a 7-day pre-treatment with MEPM extract effectively reduced the decrease in impulsive interchange caused by scopolamine. This suggests that MEPM has a considerable positive effect on area-related immediate memory. The equine protozoal myeloencephalitis examination was utilized to investigate learning and memory. The EPM is primarily employed for assessing anxiety due to its reliance on rats's inherent dislike to open and crowded environments.[34] In the study of memory, certain retention time metrics from the EPM are utilised. These parameters gauge the duration it takes for an animal to changeover from open arms to enclosed arms. In addition, an animal that was previously exposed to a delicate acquisition trial exhibits a reduced switch latency when entering open hands during the retention experiment. This model has been validated as a widely recognized framework for studying rats' training, learning, and memory processes in recent investigations on various nootropic substances associated with memory impairment in elevated and maze tasks. The results suggest that MEPM has a cognitive-enhancing impact since it enhances memory retention without any factors impairing memory. Animals having learning and memory difficulties due to the effects of scopolamine were treated with piracetam and MEPM for a duration of 7 days.

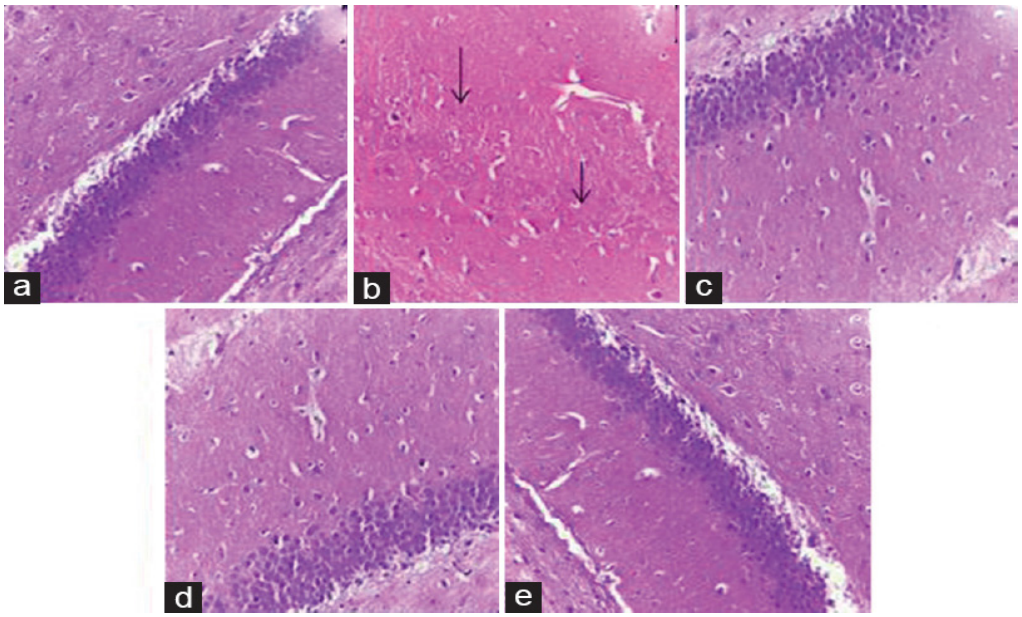

Figure 7: Rat brain tissue homogenate microscopic images (7A-Normal control; 7B- Negative control; 7C-Regular control; 7D-Low dosage of extract; 7E-High dosage of extract)

The verdict cautioned about the possible neuroprotective effects of MEPM. However, the observed rise in inflexion ratio after 24 hours depicted that a learned confront had been preserved for an extended period. MEPM was evaluated using the MWM Test to establish its cognitive boosting benefits on spatial reminiscence & investigate its purpose in rats with scopolamine-induced amnesic impairments. In this work, the administration of MEPM and piracetam to rats with induced amnesia caused by scopolamine resulted in consistently shorter escape latencies compared to animals treated just with scopolamine. This effect was observed for a 7-day training period. Findings indicate that MEPM enhances spatial cognition & memory ability in comparison to memory loss caused by scopolamine. Our previous study's findings indicate that MEPM's phytochemical analysis detected a range of phytoconstituents, such as flavonoids, saponins, and tannins. The saponins complex is widely recognized for its antioxidant and nootropic effects.[35] This provides a sufficient explanation of the extract's approach of conveying emotion. Additional research is required in order to ascertain the distinctive amalgamation of nootropic compounds, clarify the underlying mechanisms that govern spatial learning & memory, scrutinize the impact of aging on spatial guidance, and investigate the potential for nootropic manufacturers to alter specific cognitive processes. Despite thorough empirical and scientific inquiry, the neurochemical foundations of learning and memory are still inadequately comprehended. The relevance of the key cholinergic system is widely established, and it is crucial to consider the location of the different neurochemical systems. Since MEPM successfully reversed the amnesia induced by scopolamine, it is likely that the improvement in learning and memory was a result of the enhanced cholinergic diffusion in the mouse brain.

CONCLUSION

Results indicate that MEPM effectively mitigated the oxidative stress and memory impairment induced by scopolamine. Consequently, it may be concluded that *P. mucronata* has the potential as a beneficial herbal remedy for the treatment of dementia, particularly Alzheimer's type dementia, which is now linked to a decline in age-related cognitive function. However, further research utilizing *P. mucronata* is necessary to explore several advertising possibilities and elucidate the plant's accurate mechanism of action.

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