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Research Article
Anti-amnesic Effect of *Leea indica* Extract in Scopolamine-induced Amnesia of Alzheimer's Type in Rats

[1]Qinghua Chen, [2]Xuemei Chen, [3]Zhenshuai Fu, [4]Souravh Bais and [5]Xunyao Hou

[1]Department of Medical Rehabilitation, Zaozhuang Municipal Hospital of Traditional Chinese Medicine, Zaozhuang, 277000, Shandong Province, China
[2]Innoscience Research Sdn. Bhd., Subang Jaya, 47650, Selangor, Malaysia
[3]Sunshine Union Hospital, Weifang, 261000, Shandong Province, China
[4]Rayat Institute of Pharmacy, 144533, Railmajra, India
[5]Department of Senile Neurology, Provincial Hospital Affiliated to Shandong University, No.324, Jingwu Road, Huaiyin District, Jinan, Shandong 250021, China

Abstract

Background and Objective: Amnesia is one of the major complications associated with memory loss in the patents of neurodegenerative disease like Alzheimer's disease (AD). The present study investigated the potential of *Leea indica* extract in amnesia of Alzheimer's type induced in by scopolamine rats. **Materials and Methods:** Scopolamine was administered at a dose of 3 mg kg^{-1}, i.p. (One time before and after to study anterograde and retrograde amnesia) to rats for four days and then divided into different groups. Group I considered as control, Group II as Negative control, Group III animals represented Piracetam (120 mg kg^{-1}, i.p.) treated group and Group IV were administered the dose of 500 mg kg^{-1} (p.o) of methanolic extract of *Leea indica* (MELI), followed by scopolamine after 45 min. All the animals after 30 min of scopolamine administration were subjected to morris water maze test. On last day of trial, all animals were dissected and subjected to estimate different biochemical estimations like superoxide dismutase (SOD), catalase. AchE, TNF-α and mono amine oxidase (MAO-A and MAO-B) were also estimated. **Results:** Scopolamine produced a marked decline in escape latency time and increased the TNF-α in both retrograde and anterograde amnesia and also effect on various other biochemical parameters of brain and brain tissues. The MELI act as MAO-B inhibitor and effected on TNF-α, acetylcholine esterase enzyme, in cognitive dysfunctions associated with experimental amnesia in rats. The MELI and Piracetam treated animal showed reduction in AchE and elevation SOD and catalase in retrograde and anterograde amnesia. **Conclusion:** So this study concluded that MELI significantly modulated the induced memory deficits, biochemical alterations.

Key words: *Leea indica*, anterograde amnesia, Alzheimer's disease, scopolamine, neurodegenerative, memory deficits

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Corresponding Author: Xunyao Hou, Department of Senile Neurology, Provincial Hospital Affiliated to Shandong University, No. 324, Jingwu Road, Huaiyin District, Jinan, Shandong 250021, China

Competing Interest: The authors have declared that no competing interest exists.

Data Availability: All relevant data are within the paper and its supporting information files.

INTRODUCTION

Neurodegenerative diseases, such as Alzheimer's (AD) and Parkinson's, account for a significant and increasing proportion of morbidity and mortality in the world[1]. The AD is a commonest age related dementia which is distinct from age-related dementia associated with brain infraction[2]. The conventional available medications like anti cholinesterase and NMDA receptor antagonists suffer from various side effects like hallucinations, tremors, nausea, generalised weakness, diarrhea, urinary tract infection, etc. Thus herbal remedies, which offer a safer therapeutic option should be explored[1-4].

Leea indica, a traditional Chinese medicinal plant is the sole member of the family Leeaceae. It is widely distributed from Southern Asia to Northern Oceania[5,6]. Twenty three known chemical compounds were identified by Srinivasan *et al.*[7] in the leaves of *Leea indica* through GC-MS analysis, spectroscopic techniques and co-TLC with standard samples. The identified compounds include 11 hydrocarbons, phthalic acid, palmitic acid, 1-eicosanol, solanesol, farnesol, three phthalic acid esters, gallic acid, lupeol, β-sitosterol and ursolic acid. Plant traditionally used in diarrhea, dysentery, colic, ulcers, skin diseases, vertigo and headache. The leaves and roots of *L. indica* are used to treat diabetes, cardiac diseases and various ailments such as fever, headache, dizziness, soreness, eczema, sprain, leprosy, bone fracture, body pain, muscle spasm, diarrhea and dysentery. The plant has been reported for several activities like cytotoxicity, anxiolytic potential, antioxidant, antiviral, antiprotozoal, thrombolytic, analgesic and phosphodiesterase inhibition inhibitory activities[7-9]. The plant was not reported previously anywhere, for its neuro protective of amnesia of AD type and as per previous literature this plant may contain the significant potential against a memory deficit[8-10]. There are limited drugs, which are approved by USFDA against Amnesia with no side effects so other sources must be investigated to provide an efficient regimen in such diseases. Thus the present work aimed to study the *in vitro* anti-oxidant activity and the effect of methanolic extracts of *Leea indica* (MELI) on memory in scopolamine induced anterograde and retrograde amnesia of AD type in rats.

MATERIALS AND METHODS

Drugs and chemicals: Scopolamine was purchased from Sigma Aldrich (S. and G. Lab Supplies). Piracetam was procured as a gift sample from Sun pharmaceutical Pvt. Ltd. Baddi (H.P.) India. All the chemicals like Dihydrogen Orthophosphate, etc. were used in the study were of analytical grade, procured from Himedia Laboratories (Mumbai).

Plant material: Standardized methanol extract of *Leea indica* leaves was procured from Herbo Nutra, New Delhi, India. The extract dose was prepared using 2% Carboxy Methyl Cellulose (CMC) as suspending agent prior to oral administration.

Animals: Wistar rats and albino mice of either sex weighing 180-200 g and 25-30 g, respectively, were procured from registered breeder and the environmental conditions were maintained. They were housed together in the group of 6 animals each in the propylene cage with standard pellet chow and water. The experimental protocols were approved by as per guidelines provided by the Institutional Animal Ethics Committee and through governing body (IAEC/2015/05).

Acute toxicity studies: LD_{50} was determined according to the guidelines of Organization for Economic Operation and Development (OECD) following the up and down method (OECD guideline No. 423) and fixed dose method (OECD guideline No. 420). Based on these guidelines a limit test was to categories the toxicity class LD_{50} of the compound. The limit test was performed at 2500 mg kg^{-1}, p.o. A dose of 500 mg kg^{-1} (p.o.) was selected for the pharmacological activity.

Phytochemical screening: Phytochemical screening of plant extracts was done by the methods described by Kokate *et al.*[10].

Total phenol quantification: Determination of total phenolic content of freeze-dried extracts of *Leea indica* was performed by the Folin-Cio calteau method[11]. The absorbance was recorded at 725 nm against the reagent blank with a double beam UV/Visible spectrophotometer (EI models No. 5512, Japan). The amount of total phenols was calculated as pyrogallol equivalents from the calibration curve by linear regression.

Estimation of total flavonoid: The aluminium chloride colorimetric technique was used for estimation of flavonoids[12]. The ME fraction (0.5 mL each) was taken (100 mg mL^{-1} of ethanol) in test tube and mixed with 1.5 mL of methanol, 0.1 mL of 10% aluminium chloride, 0.1 mL of 1 M potassium acetate and 2.8 mL of distilled water. The reaction proceeded at room temperature for 30 min and the absorbance was

subsequently measured at 415 nm. The calibration curve was plotted by preparing quercetin standard solution across a range of 10-70 ppm in methanol. The amount of flavonoid was calculated from the standard quercetin graph.

Experimental design: The method described by Dhingra[13] and Saraf[14] was adopted. Animals were divided into four groups consisting of five albino rats per group. Group I animals served as normal control and were administered the vehicle, distilled water (4 mL kg^{-1}, p.o.), Group II animals served as the negative control and were administered the vehicle followed by scopolamine (3 mg kg^{-1}, i.p.) after 45 min. Group III animals served as standard group and received Piracetam (120 mg kg^{-1}, i.p.), followed by scopolamine after 30 min. Group IV animals represented the drug treated group and were administered the dose of 500 mg kg^{-1} (p.o) of methanolic extract of *Leea indica* (MELI), followed by scopolamine after 45 min. All the animals after 30 min of scopolamine administration were subjected to Morris water maze test. Rotarod test was performed to screen the muscle coordination activity of rats before subjecting them to water maze test. Rats showing abnormal swimming pattern in the water maze coupled with low muscle coordination activity on rota rod were excluded from the study.

Experimental design of scopolamine induced retrograde amnesia: To study the effect of the drug extract on retrieval or retention, the animals received only vehicle during the acquisition trial and were subjected to four consecutive trials per day for four days regular to locate the platform in the centre of the water tank. On 4th day, after the drug treatment, the platform was removed and time spent by the animal in the centre of the tank in search of missing platform (TSTQ) was noted as an index of retrieval. The regimen of the treatment is as given in Table 1.

Experimental design of scopolamine induced anterograde amnesia: To study the effect of drug on acquisition, the drug MELI extracts or scopolamine was administered prior to the acquisition trial for 4 days. The animals were subjected to four consecutive trials per day for four days to locate the platform. On 4th day, the vehicle was administered 45 min before the retrieval trial. The platform was then removed and time spent by the animal in the centre in search of missing platform was noted as an index of retrieval (Table 2).

Acetyl choline esterase (AchE) activity: This activity was measured by Ellman *et al.*[15] method.

Estimation of catalase: This activity was determined according to the method described by Luck *et al.*[16].

Estimation of superoxide dismutase (SOD): The SOD enzyme activity was measured by the method described by Yagi[17].

Estimation of TNF-α: The TNF α level was estimated by ELISA kit.

Estimation of MAO-A and MAO-B: Estimation of mono amine oxidase (MAO) activity: It was assayed by the method described by Charles and McEwen [18].

Statistical analysis: The data was expressed as Mean±SEM. In all the tests, the criterion for the statistical significance was set at $p<0.05$ and $p<0.01$. The data for all studies was analyzed using one-way ANOVA followed by Tukey-Kramer Multiple Comparisons test.

Table 1: Experimental design of scopolamine induced retrograde amnesia

	Administration of pharmacological agents	
Groups	Acquisition trial (Day 1-4)	Retrieval day (Day 5)
Control	Distilled water	Distilled water (4 mL kg^{-1}, p.o.)
Negative control	Distilled water	Distilled water+Scopolamine (3 mg kg^{-1}, i.p)
Standard	Distilled water	Piracetam (120 mg kg^{-1}, p.o.)+Scopolamine (3 mg kg^{-1}, i.p)
Test	Distilled water	MELI (500 mg kg^{-1}, p.o.)+Scopolamine (3 mg kg^{-1}, i.p)

MELI: Methanolic extract of *Leea indica*

Table 2: Experimental design of scopolamine induced anterograde amnesia

	Administration of pharmacological agents	
Groups	Acquisition trial (Day 1-4)	Retrieval day (Day 5)
Control	Distilled water (4 mL kg^{-1}, p.o.)	Distilled water
Negative control	Distilled water+Scopolamne (3 mg kg^{-1}, i.p)	Distilled water
Standard	Piracetam (120 mg kg^{-1}, p.o.)+Scopolamne (3 mg kg^{-1}, i.p)	Distilled water
Test	MELI (500 mg kg^{-1}, p.o.)+Scopolamne (3 mg kg^{-1}, i.p)	Distilled water

MELI: Methanolic extract of *Leea indica*

RESULTS

Reducing power assay: The reducing power of sample MELI was increased with an increase in concentration from 10-100 µg. The difference in absorbance of MELI from 10-100 µg was found to be 0.114. Standard ascorbic acid at the concentration range of 2-20 µg showed a difference of 0.677 in absorbance (Table 3).

DPPH free radical scavenging assay: The DPPH scavenging activity of the methanolic extracts was found to increase proportionately with the increase in concentration. The IC_{50} value of MELI was found to be 52.5 µg. The IC_{50} of the standard compound ascorbic acid was found to be 7.2 µg (Table 4).

Total antioxidant capacity, total phenolic content and total flavonoid content of MELI: The MELI showed 256.12±2.12 mg of ascorbic acid equivalents per gram of extract. The phenolic content of MELI was found to be 112.5±0.56 mg of gallic acid equivalents per gram of sample and total flavonoid content was found to be 65.85±3.05 mg of Quercetin equivalents per g of extract (Table 5).

Effect of MELI on retrograde amnesia in scopolamine induced amnesic rats: In the animals of all groups (I-IV) a gradual reduction in ELT was observed during the ongoing acquisition trial period (1-4) during which all the animals received the vehicle alone. On the retrieval day (Day 5) when the platform was removed were found to spend 69.7±5.12 sec in target quadrant. The animals treated with scopolamine alone showed a significant reduction in TSTQ as compared to control group. Piracetam and MELI treated groups reversed the negative effects of scopolamine on memory and significantly increased the TSTQ as compared with a negative control group (Table 6).

Effect of MELI on anterograde amnesia in scopolamine induced amnesic rats: The vehicle treated group (I) animals showed a reduction in ELT from day 1-4. The reduction in ELT was found to be significant on day 2 ($p<0.05$), day 3 ($p<0.05$) and day 4 ($p<0.001$) as compared to day 1. On the last day (5), when the platform was removed, these animals were found to spend 64.5±3.72 sec in target quadrant. The animals treated with scopolamine showed a progressive increase in ELT from day 1-4. The increase in ELT was found to be significant on day 2 ($p<0.05$), day 3 ($p<0.05$) and day 4 ($p<0.001$) as compared to day 1. On the 5th day, after removal of the platform scopolamine was found to significantly ($p<0.001$) reduce the time spent in the target quadrant as compared with vehicle treated control group. The animals treated with Piracetam and MELI reversed the observed effects with scopolamine like it showed a gradual reduction in ELT from day 1-4. The decrease in ELT was found to be significant on day 2 ($p<0.01$), day 3 ($p<0.001$) and day 4 ($p<0.001$) as compared to day 1. Also the animals in Group IV showed significantly ($p<0.001$) increased TSTQ on day 5th as compared to negative control group (II) (Table 7).

Table 3: Effects of MELI with reducing power assay

Ascorbic acid		MELI	
Conc. (µg)	Absorbance	Conc. (µg)	Absorbance
2	0.112±0.12	10	0.098±0.001
4	0.182±0.03	20	0.112±0.003
6	0.256±0.05	40	0.138±0.002
8	0.354±0.02	60	0.145±0.004
10	0.458±0.11	80	0.178±0.003
20	0.789±0.07	100	0.212±0.005

MELI: Methanolic extract of *Leea indica*

Table 4: DPPH scavenging activity of of MELI

Samples	Conc. (µg)	Radical scavenging activity (%)	IC_{50} (µg)
MELI	10	21.58±3.10	52.5
	20	39.45±2.10	
	40	46.54±5.12	
	60	62.45±1.56	
	80	78.32±0.98	
Ascorbic acid	2	23.65±2.12	7.2
	4	36.45±3.02	
	8	52.32±1.02	
	10	61.54±3.01	
	20	82.45±0.56	

MELI: Methanolic extract of *Leea indica*

Table 5: Different quantitative assay values of MELI (Per 50 g of dried extract)

Title	Methanol Ex.
Total antioxidant capacity (mg of ascorbic acid equiv L^{-1})	256.12±2.12
Total Flavonoid contents (Quercetin equiv L^{-1})	65.85±3.05
Total Phenolic contents (Gallic acid equiv L^{-1})	112.5±0.56

MELI: Methanolic extract of *Leea indica*

Effect of MELI on levels of different biochemical parameters in rat's brain: The levels of AchE was found elevated in scopolamine affected rats as compared to control group. The animals treated with piracetam showed a significant reduction in the levels of AchE on 5th day of trial as compared with untreated scopolamine group. The elevated levels of AchE, was declined on treatment with MELI as compared with Scopolamine group. The levels of catalase and SOD were significantly decreased in scopolamine affected rats as compared to a control group. The animals treated with Piracetam showed a significant increase in the levels of catalase and SOD on 5th day of trial as compared with scopolamine-affected group. The reduced levels of catalase and SOD were increased significantly with a dose of 500 mg kg^{-1} of MELI as compared with scopolamine -affected group (Table 8).

Table 6: Effect of MELI on scopolamine induced retrograde amnesia

	Escape latency time (ELT) (sec)				TSTQ (sec)
Groups	Day 1	Day 2	Day 3	Day 4	Day 5
I	68.50±5.21	46.85±2.5	31.54±2.3	19.56±2.5	69.7±5.12
II	47.60±2.1	32.45±1.56	22.23±1.02	16.50±1.2	23.1±1.74
III	78.45±1.32	52.40±1.52	27.56±1.25	15.25±1.5**	38.5±1.98*
IV	57.90±1.2	38.50±1.02	24.50±1.35	18.20±0.98***	35.1±1.08**

Values are expressed as Mean±SEM, *p<0.05, **p<0.01, ***p<0.001, ELT on days 2, 3 and 4 were compared with ELT on day 1, within the group. TSTQ of group II was compared with that of group I, while TSTQ of groups III and IV were compared with group II. Where Group I: Vehicle treated control group, Group II: Scopolamine treated negative control group, Group III: Standard drug (Piracetam) treated group, Group IV: Test (MELI) treated group

Table 7: Effect of MELI on scopolamine induced anterograde amnesia

	Escape latency time (ELT) (sec)				TSTQ (sec)
Groups	Day 1	Day 2	Day 3	Day 4	Day 5
I	71.60±6.21	41.50±3.5	28.54±1.3	22.16±3.5	64.5±3.72
II	61.60±2.5	77.45±1.76	85.13±1.22	96.50±1.2	20.1±1.74
III	78.45±1.32	36.50±1.02	25.54±1.25	22.50±1.4*	48.1±1.98
IV	84.50±3.2	68.50±1.02	44.50±1.35	28.20±0.98**	55.1±1.08*

Values are expressed as Mean±SEM, *p<0.05, **p<0.01, ELT on days 2, 3 and 4 were compared with ELT on day 1, within the group. TSTQ of group II was compared with that of group I, while TSTQ of groups III and IV were compared with group II. Where Group I: Vehicle treated control group, Group II: Scopolamine treated negative control group, Group III: Standard drug (Piracetam) treated group, Group IV: Test (MELI) treated group

Table 8: Effect of MELI on various biochemical parameters in rats' brain

Groups/ Treatment	Anterograde AchE (nM/L/min/g of tissue)	Retrograde SOD (Units/mg of protein)	Catalase (μM of H_2O_2 decomposed/min/mg of protein)	AchE (nM/L/min/g of tissue)	SOD (Units/mg of protein)	Catalase (μM of H_2O_2 decomposed/min/mg of protein)
I	4.20±0.05^{b***}	45.20±1.4^{b***}	2.04±0.123^{b***}	4.10±0.11^{b***}	41.10±1.5^{b***}	1.84±0.013^{b***}
II	9.51±0.29^{a***}	9.26±1.51^{a***}	0.53±0.025^{a***}	7.81±0.29^{a***}	15.16±1.31^{a***}	0.83±0.025^{a***}
III	7.28±0.12$^{a\ ns\ b***}$	43.34±1.54$^{a\ ns\ b***}$	0.95±0.5^{a**b***}	5.78±0.28$^{a\ nsb***}$	43.34±1.54$^{a\ ns\ b***}$	0.85±0.015^{a**b***}
IV	5.24±0.38^{a**b***}	40.07±1.7$^{a\ ns\ b***}$	1.20±0.4^{a***b**}	6.24±0.28^{a**b**}	41.10±3.7$^{a\ ns\ b***}$	1.50±0.018^{a***b***}

Data are Mean±SEM values, n = 8, Data were analyzed by one way ANOVA followed by Tukey- Kramer Multiple Comparisons test, *p<0.05, **p<0.01, ***p<0.001 as compared with inducer, ^{NS}Not significant, ^{a}When compared with control group, ^{b}When compared with negative group, Where Group I: Vehicle treated control group, Group II: Scopolamine treated negative control group, Group III: Standard drug (Piracetam) treated group, Group IV: Test (MELI) treated group

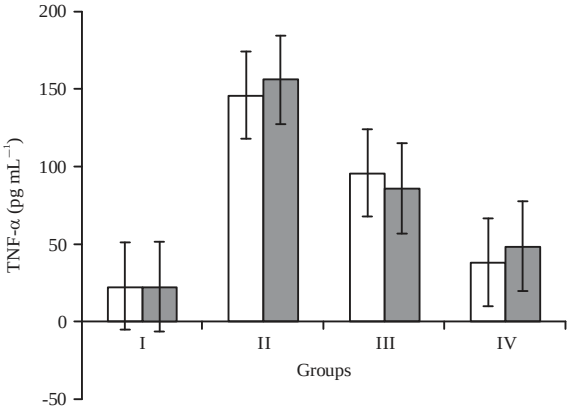

Fig. 1: Effect of MELI on TNF-α level in brain cortex
All values were represented as Mean±SEM, n = 8, Group I: Vehicle treated control group, Group II: Scopolamine treated negative control group, Group III: Standard drug (Piracetam) treated group and Group IV: Test (MELI) treated group

Effect on brain TNF-α level: Rats in group II showed an increase in TNF-α level (Fig. 1) in brain. MELI treated rats showed a significant reduction in TNF-α levels as compared to group II.

Effect of MELI on MAO-A and MAO-B level: The effects of MELI on MAO concentration were shown in Table 9. The MAO and MAO-B activities in a control group were in Anterograde Amnesia 36.05±0.12, 29.5±0.44, 37.8±0.22 and 31.7±0.10 nmol mg^{-1} protein in retrograde amnesia, respectively. MELI at a dose of 500 mg kg^{-1}, (p.o.) showed a marked inhibition in both types of amnesia, MAO-A and MAO-B (Table 9).

DISCUSSION

The present study proved the antioxidant and anti amnesic activity of *Leea indica* in scopolamine induced amnesia of AD type in rats. Cognition is the psychological process of knowing including awareness, perception reasoning and judgement. The AD has been identified as a protein miss folding disease due to accumulation of abnormally folded amyloid beta proteins in the brain of AD patient[19]. Dementia is the progressive loss of memory and intellectual abilities and AD has received utmost attention in the past decade as a major cause of dementia[20]. Central cholinergic system plays an important role in regulation of

Table 9: Effect of MELI on MAO level in brain tissues

	Anterograde				Retrograde amnesia			
Groups/ Treatment	MAO-A (nmol mg^{-1} protein·h)	MAO- A Inhibition (%)	MAO-B (nmol mg^{-1} protein·h)	MAO-B Inhibition (%)	MAO-A (nmol mg^{-1} protein·h)	MAO-A Inhibition (%)	MAO-B (nmol mg^{-1} protein·h)	MAO-B Inhibition (%)
I	36.5±0.12	00	29.5±0.44	00	37.8±0.22	00	31.7±0.10	00
II	24.54±0.14*	40.20	19.1±0.14*	50	21.47±0.14*	33.80	19.5±0.14*	40
III	24.5±0.12*	18	22.2±0.12	21	23.2±0.32*	18	27.1±0.15	24
IV	20.5±0.88**	32	26.2±0.3*	45	21.9±0.88**	32	24.2±0.8*	48

Data are Mean±SEM values, n = 8, Data were analyzed by one way ANOVA followed by Tukey-Kramer Multiple Comparisons test, *p<0.05, **p<0.01, as compared with inducer, Where Group I: Vehicle treated control group, Group II: Scopolamine treated negative control group, Group III: Standard drug (Piracetam) treated group, Group IV: Test (MELI) treated group

cognitive function. Scopolamine, a muscarinic receptor antagonist has been widely adopted to study memory deficits in experimental animals. Recently, several studies reported that scopolamine affected cholinergic system and impairs memory by increasing oxidative stress within the brain[14,21,22]. The Morris water maze is a stabilized model for testing cognitive behavior in animal models and very helpful to analyse the reversal of amnesic effect with investigational drugs because repetitive trials confirm the progress of reversal of amnesia.

In earlier studies, after the normal acquisition period administration of scopolamine on the retrieval day (day-after after acquisition) before the removal of the platform was found to reduce the Time Spent in Target Quadrant (TSTQ) and increase in Escape Latency Time (ELT) as compared to the vehicle control animals. This effect proved that scopolamine impairs the retrieval of memory or produces amnesia[23]. In this study, similar effects observed like significant memory impairment with scopolamine (0.4 mg kg^{-1}) in rats. Piracetam at the dose of 400 mg kg^{-1} (i.p) was reported to reverse the scopolamine induced anterograde and retrograde amnesic effects. Administration of scopolamine after 45 min of piracetam administration during retrieval day (Day 5) and in the acquisition, period (1-4 Day) showed a marked increase in TSTQ and decrease in ELT in both types of amnesia. In a present study, MELI was administered at a dose of 500 mg kg^{-1} based on previous studies where the similar extract of *Leea indica* leaf was proved for its antioxidant activities[6].

In a present study also, both Piracetam and MELI (500 mg kg^{-1}, p.o.) significantly affected both ELT and TSTQ. They gradually decreased the ELT during acquisition trial (Day 1-4) and increased the TSTQ during retrieval trial (Day 5) as compared to scopolamine treated negative control group. This effect showed the piracetam and MELI reversed the scopolamine induced anterograde amnesia by both, increasing the learning and retrieval of memory in animals.

Scopolamine was earlier reported to cause a significant increase in acetyl choline esterase level in the brain of the animal as compared to the vehicle treated control animals[20]. This study also supports the previous findings with an increase in AchE level in the brain of scopolamine treated rats. Piracetam is reported to reduce the level of AchE in the brain of both scopolamine induced retrograde[23] and anterograde[24] amnesic animals. The MELI administration significantly reduced the levels of AchE in test group animals in both retro and anterograde amnesia as compared to the negative control group. Recently, the protective roles of MAO inhibitors in Alzheimer's disease were well explained by Kangatao *et al.*[2] where these compounds proven to restore the memory deficit and this study also supported the observed effects with MELI administration in rats. Phytochemical screening of MELI suggested the presence of alkaloids, terpenoids, sterols, saponins, flavonoids and tannins. The total flavonoids and total phenolic contents of the plants also revealed the presence of appreciable quantities of phenols and flavonoids, which indicates its antioxidant potential, which was further confirmed and evaluated by *in vitro* anti-oxidant assays. A study reveals that flavonoids like Quercetin, Kaempferol, luteolin and apigenin inhibit the β cleavage of APP[25] and proved good Anti-AD and have neuro protective effects[26]. Thus the present study reveals that MELI has potential therapeutic effects on improving memory in amnesia of AD type in animals which either may be due to the inhibition of lipid peroxidation or due to its Anti AchE activity. The *in vitro* assays indicate that the plant is a significant source of natural antioxidants, which might be helpful in preventing the progress of oxidative stress and associated disorders, particularly amnesia or AD. This study will help researchers to explore the effects of *leea indica* extract on other neurodegenerative diseases or especially the diseases modulated by MAO release. This study also creates an urge to investigate the chemical constituents responsible this action.

CONCLUSION

The MELI showed positive effect in treating scopolamine induced amnesia and significantly modulated the induced memory deficits, biochemical alterations. The findings

demonstrated that the memory restorative ability of MELI may be attributed due to its anti-cholinesterase, anti-oxidative and anti-inflammatory potential.

SIGNIFICANCE OF STATEMENT

This study was designed to evaluate the potential role of MELI extract in memory deficits produced by scopolamine and findings of this study suggested the significant MAO inhibition by MELI, which creates further interest among scientific researchers to explore its chemical constituents responsible its action. This study is also a first evidence to report the potential of MELI in modulation of MAO in scopolamine induced amnesia of AD Type in rats.

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