



Effect of grewia hirsuta vahl ethanolic root extract on cognition in scopolamine induced amnesia

N.Yamini¹, B.Pushpa kumari², S.Mehathaj³, V.Phani deepthi⁴

¹ Department of Pharmacology, Sri Padmavathi School of Pharmacy

² Department of Pharmacology, Sri Padmavathi School of Pharmacy

³ Department of Pharmacology, Sri Padmavathi School of Pharmacy

⁴ Department of Pharmaceutics, Oil Technological Pharmaceutical Research Sciences, JNTUA

Article History	Abstract
Received: 02-11-2021 Revised: 17-11-2021 Accepted: 25-12-2021	Objective Alzheimer's illnesses are becoming medical nightmares because there is no exact solution and existing nootropic medicines (Piracetam, tacrine, and metrifonate) have significant drawbacks. The goal of this study was to see if the ethanolic root extract of <i>Grewia hirsuta</i> (ERGH) could improve memory in rats who had been given scopolamine.
Keywords Alzheimer's diseases, memory enhancing, Scopolamine.	Materials and procedures At rats, ERGH was given orally in dosages of 200 and 400 mg/kg for 28 days, followed by Scopolamine (18 mg/kg i.p.) from the 25th to the 27th day. The usual nootropic drug was piracetam (200 mg/kg). The elevated plus maze (EPM), Morri's water maze (MWM), and passive avoidance (PA) paradigms are used to assess cognitive functioning. In vivo anti-oxidant activity and brain acetylcholine esterase (AChE) activity were assessed.
*Corresponding Author N. Yamini Email: nemalapalli@gmail.com	Results: At the indicated doses, ERGH extract showed a substantial memory enhancing activity by decreasing the transfer latency in EPM, increasing the escape latency in MWM, and increasing the shock free zone in PA. In scopolamine-induced amnesia rats, pretreatment with ERGH resulted in a significant drop in AChE enzyme, an increase in enzymatic antioxidant, and a decrease in MDA levels.
https://doi.org/10.37022/jpmhs.v4i4.49	Conclusion Because of its several favourable benefits, such as memory-improving properties, anticholinesterase activity, and antioxidant activity, ERGH may prove to be a useful drug in the current study, and it would be important to investigate its potential in the care of Alzheimer's patients.

This article is licensed under a Creative Commons Attribution-Non Commercial 4.0 International License.
Copyright © 2021 Author(s) retain the copyright of this article.



Introduction

The physiological process of knowing, which includes awareness, perception, reasoning, and judgement, is known as cognition. Memory, attention, creativity, and intellect are the four main categories of cognitive functions. A variety of factors can influence it, including ageing, stress, hypertension, and neurodegeneration [1, 2].

Alzheimer's disease (AD) is a slow-onset neurodegenerative brain ailment that causes dementia, strange behaviour, personality changes, and eventually death [3].

The basic origin of Alzheimer's disease is unknown; nevertheless, amyloid and tau protein aggregation, decreased acetylcholine (ACh), and glutamatergic deficiency are thought to be the key pathophysiology of the disease. According to the WHO, dementia of

Alzheimer's disease affects 5% of men and 6% of women over the age of 65 worldwide [4].

Memory formation is the most complicated process, including numerous neural circuits and chemicals. The cholinergic neural system is widely known for its function in learning and memory in both humans and animals. The use of nootropic medicines like piracetam and its equivalents like oxirecetam, aniracetam [6], and metrifonate is based on this medicinal rationale [7]. However, several side effects associated with these medicines, such as loss of appetite, nausea, vomiting, diarrhoea, stomach cramps, headache, dizziness, exhaustion, and sleeplessness, have limited their usage. As a result, newer products are gaining popularity, one of which is medicinal herbs, which are non-toxic, have fewer side effects, and are inexpensive

Grewia hirsuta, a member of the Tillaceae family, is widely used in Ayurvedic medicine to treat fever, heart illness, epistaxis, nervine tonic, expectorant, pain, urinary diseases, and brain tonic. There was an outlook for herbs that have a medical utility in treating memory loss, considering the positive advantages of herbs over allopathic treatment. An attempt was undertaken to explore the positive effects of the plant *Grewia hirsuta* against scopolamine-induced dementia in experimental rats due to a lack of scientific data and a good medicine to treat AD. The goal of this study was to see if an ethanolic extract of *Grewia hirsuta* root could improve memory.

Materials and Methods

Animals

This study employed healthy male Albino rats of the Wistar breed (180-200g) obtained from Raghavendra enterprises in Bangalore. The animals were housed in regular conditions with free access to food and water. Prior to the experiment, the animals were housed in polypropylene cages for one week to acclimate to laboratory settings. The experimental methodology was authorised by the Sri Padmavathi School of Pharmacy's Institutional Animal Ethical Committee (SPSP/CPCSEA/IAEC-1016/a/2014/010).

Acute toxicity studies

Acute oral toxicity testing was carried out in accordance with OECD-423 criteria. Female rats (n=3) were chosen at random and put into four groups. Overnight, the animals were fasted. Ethanolic root extract was given orally to all of the groups at doses of 5, 50, 300, and 2000 mg/kg body weight. The animals were monitored during the first four hours for any unusual signs such

tremors, convulsions, exophthalmus, salivation, diarrhoea, and lethargy, and then for another 14 days. The animals were observed for any changes in behavioural pattern and death at the end of the trial.

Collection of plant materials

Grewia hirsuta plant material was obtained from a local market and certified by B.Sitharam of Tirupathi, India. The plant material was dried in the shade and roughly pulverised. The Maceration process was used to make an ethanolic root extract of *Grewia hirsuta* (ERGH). 250g of root powder was mixed with 750ml (1:3) ethanol and kept at room temperature for 3 days with continuous shaking. It was then filtered and evaporated to leave a solid residue.

Experimental Design

In this investigation, five sets of experimental rats, each with six rats, were used, and the treatment was given for 28 days. The following was the procedure that was devised.

Group I (Normal group) consists of the following individuals: The rat was given the vehicle (1 percent CMC) orally for twenty-eight days in a row.

Scopolamine (18 mg/kg, i.p.) was given to Group II (Control group) for three days in a row, from the 25th to the 27th.

Group-III (Standard group): Piracetam (200 mg/kg, p.o.) was given daily for 28 days, then scopolamine (18 mg/kg, i.p.) was given three days in a row from the 25th to the 27th day.

Group-IV (Test-1): Every day, ERGH (200 mg/kg, p.o.) was given. ERGH (200 mg/kg, p.o.) was given one hour before on days 25 to 27

Group-V (Test-II): ERGH (400 mg/kg, p.o.) was given to rats for a total of 28 days, with ERGH (400 mg/kg, p.o.) given one hour before scopolamine (18 mg/kg, i.p.) was given

Behavioural Parameters

Elevated plus maze (EPM)

The EPM (8, 9) was used to assess learning and memory (cognition) in rats as an exteroceptive behavioural model. The plus maze apparatus was made up of two open arms (50cm10cm40cm) and two enclosed arms (50cm10cm40cm) with an open roof, placed so that the two open arms faced each other. To evaluate the anxiety index in rats, the maze was raised to a height of 50cm above the ground. During the training phase, rats were free to explore EPM; if they did not enter one of the two enclosed arms within 90 seconds, they were gently pushed into one of the two enclosed arms, and the TL was set to 90 seconds. On the 28th day, 1 hour after the

last doses were administered, each rat was placed at the end of an open cage. EPM's arm is angled away from the platform's centre. The time it took a rat to move inside one of the enclosed arms with all four legs was recorded, and the rats were then returned to their home cage.

Passive shock-Avoidance paradigm

Long-term memory tasks are frequently screened using the passive avoidance (8, 9) behaviour paradigm. Rats were given a daily session of five trials of a passive avoidance task in which a 0.7mA foot shock was delivered for a maximum of thirty seconds. The average time for five trials was obtained for the rat housed in the shock chamber to reach the centrally placed shock free zone (SFZ) in 10 seconds. The rats were considered learned and selected for the study if they reached SFZ in less than 10 seconds for three days in a row.

The above-mentioned trained rats were given the necessary medicines every day for the next 28 days, as specified in the experimental design. Rats were given scopolamine injections (18mg/kg, i.p.) on the 25th day, 60 minutes after dosing, to create amnesia, and then evaluated for the passive avoidance response task 60 minutes later. The time it took the rats to reach the SFZ in comparison to the control group was utilised to assess the drug's effect on short-term memory.

Morris water maze

The Morris water maze is a huge circular pool (100 cm in diameter and 35 cm in height) filled with water to a depth of 30 cm. Titanium dioxide was used to make the water opaque. The pool was divided into four equal quadrants at random [10]. A clear Plexiglas platform with an 11cm diameter was submerged 1cm below the surface of the water. The platform was put towards the centre of a quadrant, and rats from one of the remaining three quadrants were released into the water to search for the platform. In each experiment, the time it took to find the hidden platform [escape latency (EL)] was recorded. If the animal did not reach the platform after 90 seconds, it was directed to it. Animals were herded onto the platform. For 30 seconds, the animals were allowed to stay on the platform. The inter-trial period was 1.5 minutes, and between each trial, the rats were towelled and placed under a heating light to avoid hypothermia.

The above-mentioned rats were dosed with the necessary medications every day for the next 28 days, as per the experiment's procedure. On the 25th day, one hour after dosing, rats were given a scopolamine injection (18mg/kg, i.p) to produce amnesia, and then tested for

spatial memory for 60 minutes. The time it took the rat to reach EL was compared to the time it took the control group rats to assess the drug's effect on short-term memory.

Locomotor Activity [11]

An actophotometer was used to monitor locomotor activity. After 1 hour of treatment with the final dose, each rat was individually placed in the actophotometer for 5 minutes to get the basal activity score. CNS stimulant activity was defined as an increase in count, while depressant activity was defined as a decrease in count

Biochemical Estimations

Estimation of AchE levels

Rats from each group were euthanized by cervical decapitation after the learning and memory paradigms were assessed in scopolamine-induced amnesia. The entire brain was extracted and placed in an ice cold phosphate buffer. The brain was homogenised with 0.1M phosphate buffer (pH 8.0) using a glass Teflon homogenizer after being washed in ice cold phosphate buffer. The levels of AchE were determined using Ellman et al's approach, which was slightly modified [12] At 420nm, the sample's change in absorbance per minute was measured spectrophotometrically.

Estimation of oxidative stress markers

Animals were decapitated and cut open at the end of the study to remove the brain. The homogenate was prepared as follows: the brain was weighed, then the homogenate was prepared as follows: The tissue was minced and homogenised in ice-cold 10mM tris HCL buffer (to pH7.4) at a concentration of 10% (w/v) with 25 stokes of tight Teflon pestle of glass homogenizer at a speed of 2500 rpm with 25 stokes of tight Teflon pestle of glass homogenizer. The purpose of the extended homogenization under hypnotic conditions was to rupture the ventricular surface of cells as much as possible, allowing rpm to release soluble protein while leaving only membrane and non-vascular materials in sedimentation form. After that, it was centrifuged. The clear supernatant was extracted and utilised to estimate super oxide dismutase (SOD), reduced glutathione (GSH), catalase (CAT), and lipid per oxidation at 5000 rpm at 20oC temperature (LPO) [13].

Measurement of Superoxide Dismutase

Misra et al approach's was used to determine superoxide dismutase activity. The suppression of epinephrine auto-oxidation was used to test the superante for SOD activity. 0.25 ml ethanol, 0.5 ml chloroform (all reagents refrigerated) were added to 0.5 ml of sample diluted with

0.5 ml of distilled water. The mixture was mixed for 1 minute before being centrifuged for 20 minutes at 2000 rpm. The activity of enzymes in the supernatant was measured. 0.05 ml carbonate buffer (0.05 M, pH 10.2) and 0.5 ml EDTA (0.49 M) were added to a mixture of 0.05 ml carbonate buffer (0.05 M, pH 10.2) and 0.5 ml EDTA (0.49 M). The reaction was started by adding 0.4ml of epinephrine, and the change in optical density/min at 480nm was observed. Units/mg protein change in optical density/min were used to measure SOD activity. The enzyme unit is 50 percent suppression of the epinephrine to adrenochrome transition. SOD in the range of 10 to 125 units was used to create the calibration curve.

Measurement of Reduced Glutathione (GSH)

Ellman's approach was used to determine glutathione levels. To separate the proteins, an equal amount of homogenate (w/v) and 10% TCA were combined and centrifuged. 2 ml phosphate buffer (pH 7.4), 0.5 ml 5, 5'dithiobisnitro benzoic acid (DTNB), and 0.4 ml double distilled water were added to 0.01 ml of this supernatant. Within 15 minutes, the absorbance was measured at 412 nm after the mixture was vortexed. The amount of GSH in each gramme of protein was measured in moles GSH [14].

Measurement of catalase

Claiborne's approach was used to determine catalase activity. In a cuvette containing 1.9 ml of 50 mM phosphate buffer, 0.1 ml of supernatant was added (pH 7). The reaction was begun by adding 1 ml freshly produced 30 mM H₂O₂ to the mixture. At 240 nm, the rate of H₂O₂ breakdown was determined spectrophotometrically. Catalase activity was measured in moles of H₂O₂ used per minute per milligramme of protein [15].

Measurement of lipid peroxidation

Ohkawa's method for measuring TBARS, a measure of lipid per oxidation, was used. In a nutshell, 1 ml of suspension medium was extracted from the 10% tissue homogenate. It was then treated with 0.5 ml of 30 percent Trichloroacetic Acid (TCA) reagent and 0.5 ml of 0.8 percent Thiobarbituric Acid (TBA) reagent. The tubes were wrapped in aluminium foil and placed in an 80°C shaking water bath for 30 minutes. Tubes were removed after 30 minutes and placed in ice cold water for another 30 minutes. These were centrifuged for 15 minutes at 3000 rpm. At room temperature, the absorbance of the supernatant was measured at 540 nm against an adequate blank. 1 mL distilled water, 0.5 mL 30% TCA, and 0.5 mL 0.8 percent TBA make up the blank. Values of TBARS.

The TBARS were calculated as n moles malonaldehyde (MDA)/mg protein [16].

Statistical analysis

The mean and standard deviation (SEM) are used to express the data. One-way ANOVA was used for statistical analysis. Dunnet's test was used to make group comparisons. The significance level was set at p 0.05.

Results

Phytochemical screening

Carbohydrates, tannins, phytosterols, saponins, and flavonoids were discovered in preliminary phytochemical testing on ERGH.

Acute toxicity tests on rats were carried out according to OECD 423 recommendations at dosages of 5 mg/kg, 50 mg/kg, 300 mg/kg, and 2000 mg/kg, p.o. They exhibit typical behaviour, including as attentiveness, grooming, and pain reaction. There were no stereotypes or signs of passivity to be found. Even the animal's motor activity and signals were normal, and there were no indicators of depression. It was also discovered that at a dose of 2000 mg/kg, For the current study to evaluate memory enhancing activity, it can be deemed a safe dose by taking 1/10th of its dose as a low dose (i.e., 200 mg/kg) and double of its dose as a high dose (i.e., 400 mg/kg).

The animals were separated into five groups and given therapy for a total of 28 days, as shown in table 5. On the 25th day, scopolamine (18 mg/kg, i.p.) was given to each group for three days in a row.

a) Effect of ERGH on Transfer latency (TL) in Elevated plus maze

Transfer latency (TL) is a metric used to assess the retrieval mechanism of learning and memory. It is the time it takes for animals to go from open arms to closed arms. When compared to the normal group (22.61±0.427), the control group (121.53 ±0.719) treated with scopolamine (18 mg/kg, i.p) had a substantial increase in TL. When compared to the control group, the groups treated with the conventional drug Piracetam exhibited a significant drop in TL (15.30±0.430). When compared to the control group, the groups treated with low dose (200 mg/kg, p.o) and high dose (400 mg/kg, p.o) of ERGH showed a substantial decrease in TL (14.32±0.376 and 12.37±0.369, respectively). TL had decreased significantly in response to ERGH, and this was dose dependent (Table 01).

b) Effect of ERGH on latency to reach SFZ in passive avoidance

The time it takes to enter the shock-free zone (SFZ) is used as a measure of memory acquisition and retention. When compared to the control group, the test group receiving ERGH at low dose (200 mg/kg, p.o) and high dose (400 mg/kg, p.o) demonstrated a substantial decrease in latency of time to reach SFZ (4.487 ± 0.330 and 3.68 ± 0.472). The latency of time to reach SFZ was significantly reduced by ERGH in a dose-dependent manner. When compared to normal (4.487 ± 0.439), the latency to reach the SFZ in the control group treated with scopolamine (18 mg/kg, p.o) was significantly increased (5.660 ± 0.284). When compared to the control group, the conventional drug Piracetam (200 mg/kg, p.o) exhibited a considerable reduction in latency to reach SFZ (4.403 ± 0.357). (Table-8)

c) Effect of ERGH on Escape latency in Morri's water maze

The time it takes to find the concealed platform in a water maze has been used as a measure of spatial memory.

When compared to the control group, the piracetam (200 mg/kg, p.o) treated group demonstrated a substantial decrease in EL (20.74 ± 17.69). As scopolamine (18 mg/kg, p.o) was given to a control group, the EL increased significantly (77.72 ± 0.386) when compared to the normal group (18.30 ± 0.419).

When compared to the control group, the group receiving ERGH at a modest dose (200 mg/kg, p.o) had a substantial drop in EL (17.69 ± 0.4054). When compared to the control group, the test group treated with ERGH at a high dose (400 mg/kg, p.o.) demonstrated a substantial and dose dependent drop in EL (15.63 ± 0.382). (Table -9).

d) Effect of ERGH on locomotor activity

Locomotor activity is a measure of mental activity wakefulness (alertness). When compared to the normal group (263.7 ± 14.62), the locomotor activity in the control group was significantly lower (132.7 ± 9.659). When compared to the control group, piracetam (200 mg/kg, p.o) and ERGH (200 mg/kg and 400 mg/kg, p.o) showed no significant decrease in locomotor activity (279.2 ± 11.42 , 258.3 ± 4.92 , and 290.7 ± 10.36). (Table-10).

e) Effect of ERGH on Cholesterol levels

Cholesterol turnover appears to be important for amyloid peptide accumulation and clearance in the brain. When compared to normal (58.68 ± 5.947), blood cholesterol levels in the control (77.26 ± 4.249) and piracetam (200 mg/kg, p.o) (74.25 ± 3.350) treated groups were significantly higher. When compared to the control group, ERGH treated at low dose (200 mg/kg,

p.o) and high dose (400 mg/kg, p.o) had significantly lower cholesterol levels (52.73 ± 4.700 and 43.19 ± 2.154), respectively.

f) Effect of ERGH on Triglycerides

When compared to normal (77.09 ± 1.545), triglycerides levels in the scopolamine-treated control group and piracetam (200 mg/kg, p.o) groups were significantly higher (122.2 ± 3.995 and 121.4 ± 13.07). When compared to the control group, the group receiving ERGH at a modest dose (200 mg/kg, p.o) had significantly lower triglyceride levels (80.23 ± 1.411). When compared to the control group, the group treated with ERGH at a high dose (400 mg/kg, p.o) saw a significant drop in triglycerides levels (74.00 ± 1.999).

g) Effect of ERGH on Glucos levels

Scopolamine is a medication that inhibits the energy metabolism of cholinergic nerves by blocking acetylcholine receptors.

When compared to normal (90.24 ± 5.156), glucose levels in the control group treated with scopolamine (18 mg/kg, p.o) were considerably lower (51.154 ± 7.063). When compared to the control group, piracetam (200 mg/kg, p.o) revealed a considerable increase in glucose levels (73.78 ± 13.21).

When compared to the control group, the group treated with ERGH at a modest dose (200 mg/kg, p.o) had a substantial increase in glucose levels (78.23 ± 9.258). When compared to the control group, ERGH at a high dose (400 mg/kg, p.o) revealed a significant and dose-dependent increase in glucose level (85.40 ± 8.400).

h) Effect of ERGH on AchE activity

Scopolamine, by an unknown mechanism, can increase acetylcholinesterase (AChE) activity in brain tissue. When compared to the normal group (0.000723 ± 0.00026), rats in the control group treated with scopolamine showed a substantial increase (0.005802 ± 0.00076) in AChE levels. When compared to the control group, the group treated with the conventional medicine piracetam (200 mg/kg, p.o) demonstrated a substantial decrease (0.00094 ± 0.00061) in AChE levels. When compared to the control group, the test group receiving ERGH at a low dose (200 mg/kg, p.o) demonstrated a significant decrease (0.00123 ± 0.00017) in AChE levels. When compared to the control group, the test group treated with ERGH at a high dose (400 mg/kg, p.o) demonstrated a substantial and dose dependent drop (0.000627 ± 0.00042) in AChE levels.

i) Effect of ERGH on *Invivo* antioxidants

Increased occurrence of cognitive problems is linked to oxidative damage in neurons. It is one of the initial signs of AD onset and progression. When compared to normal, the scopolamine treated control group had significantly lower levels of SOD, GSH, and CAT (2.518 ±0.3154, 2.576±0.449, 7.195±0.352) and significantly higher levels of LPO (3.3560.217). When compared to the control group, the conventional medicine piracetam (200 mg/kg, p.o) exhibited significantly improved levels of SOD, GSH, and CAT (7.137±0.6028, 6.360±0.433, 14.25±0.690) and lower levels of LPO (1.424±0.349). There was significant elevated levels of SOD, GSH, CAT (5.674±0.4457, 5.789±0.261, 17.37±0.421) respectively and decreased levels of LPO (1.643±0.311) was observed in the group receiving ERGH at low dose (200 mg/kg, p.o). The group treated with ERGH at high dose (400 mg/kg, p.o) has showed significant and dose dependent increased levels of SOD, GSH, CAT (7.631±0.3607, 6.914±0.551, 18.84±0.193) respectively and decreased levels of LPO (1.543±0.472) was observed when compare to control group.

Table 01: Effect of ERGH on transfer latency in Elevated plus Maze

S.NO	GROUPS	TRANSFER LATENCY (SEC)	
		TL on acquisition on 27 th day	TL on Retention on 28 th day
1	Normal	24.59±0.323	22.61±0.427
2	Control	127.50±1.675 ^a	121.53±0.719 ^a
3	Piracetam (200 mg/kg)	21.64±0.402 ^b	15.30±0.430 ^b
4	ERGH(200 mg/kg)	18.40±0.434 ^b	14.32±0.376 ^b
5	ERGH(400 mg/kg)	17.49±0.433 ^b	12.37±0.369 ^b

Values are expressed in mean ± S.E.M

a**=p<0.001 when compared to normal group

b***=p<0.001 when compared to control group

Table 2: Effect of ERGH on Time taken to reach SFZ in Passive Avoidance

S.NO	GROUPS	Time taken to reach SFZ	
		on acquisition day(27 th day)	on retention day(28 th day)
1	Normal	5.210±0.409	4.487±0.439
2	Control	6.733±0.260 ^a	5.660±0.284 ^a
3	Piracetam (200 mg/kg)	4.407±0.367 ^b	4.403±0.357 ^b

4	ERGH(200 mg/kg)	4.007±0.117 ^b	4.487±0.330 ^b
5	ERGH(400 mg/kg)	5.040±0.266 ^b	3.68±0.472 ^b

Values are expressed in mean ± S.E.M

a**=p<0.001when compared to normal group

b***=p<0.001 when compared to control group

Table 03: Effect of ERGH on Escape Latency in Morri's Water Maze

S.NO	GROUPS	ESCAPE LATENCY (SEC)	
		EL on acquisition on 27 th day	EL on Retention on 28 th day
1	Normal	20.56±0.353	18.30±0.419
2	Control	80.60±0.443 ^a	77.72±0.386 ^a
3	Piracetam (200 mg/kg)	23.31±0.3281 ^b	20.74±17.69 ^b
4	ERGH(200 mg/kg)	28.67±0.401 ^b	17.69±0.4054 ^b
5	ERGH(400 mg/kg)	19.10±0.6447 ^b	15.63±0.382 ^b

Values are expressed in mean ± S.E.M

a**=p<0.001 when compared to normal group

b***=p<0.001when compared to control group

Table 04: Effect of ERGH on locomotor activity

S.No	Groups	Activity Score	
		0 day	28 th day
1	Normal	251.3±3.21	263.7±14.62
2	Control	223.2±5.381 ^a	132.7±9.659 ^a
3	Piracetam (200 mg/kg)	268.7±1.456 ^b	279.2±11.42 ^b
4	ERGH(200 mg/kg)	259.3±5.228 ^b	278.3±4.92 ^b
5	ERGH(400 mg/kg)	288.3±3.781 ^b	290.7±10.36 ^b

Values are expressed in mean ± S.E.M

a**=p<0.001 when compared to normal group

b***=p<0.001 when compared to control group

Table 05: Effect of ERGH on In vivo antioxidants

S. N O	GRO UP	SOD (μ/mg. protein)	GSH (μg/m g. protei n)	LPO(n m MDA / mg tissue)	CAT (μM H ₂ O ₂ consum ed/mg protein
--------	--------	----------------------	------------------------	--------------------------	--

1	Normal	13.19±0.3443	10.19±0.678	1.536±0.383	12.20±0.609
2	Control	2.518±0.3154 ^a	2.576±0.449 ^a	3.356±0.217 ^a	7.195±0.352 ^a
3	Piracetam (200 mg/kg)	7.137±0.6028 ^b	6.360±0.433 ^b	1.424±0.349 ^b	14.25±0.690 ^b
4	ERGH (200 mg/kg)	5.674±0.4457 ^b	5.789±0.261 ^b	1.643±0.311 ^b	17.37±0.421 ^b
5	ERGH (400 mg/kg)	7.631±0.3607 ^b	6.914±0.551 ^b	1.543±0.472 ^b	18.84±0.193 ^b

Values are expressed in mean ± S.E.M

a=*** p<0.001 when compared to normal group

b=*** p<0.001 when compared to control group

Discussion

Alzheimer's disease (AD) is a neurological disease that causes cognitive impairment. Despite the disease's severity and widespread prevalence, the allopathic medical system has yet to develop a satisfactory antidote. As a result, the current study focuses on investigating the ERGH's memory-enhancing action in a scopolamine-induced amnesia rat paradigm. Because of its facilitative effect on the retention of spatial memory in scopolamine-induced amnesia, the current study demonstrates that ERGH has memory-enhancing action. The TL has decreased, indicating that rats were able to detect the black zone quickly after being exposed to the open arm in the EPM paradigm, indicating that cognition has improved. The SDL is raised in the passive avoidance paradigm when ERGH is administered. This shows that after the animal is in the shock-free zone, it retains memory of the shock. When ERGH is given to a patient with MWM, the EL time is lengthened. This indicates that the animal retains memory in the water after entering the platform. In comparison to the control group, ERGH treated rats showed considerable antioxidant activity, with increased levels of SOD, CAT, and GSH, as well as lower levels of MDA.

Clinical data suggests that cholinergic neural networks play a key role in cognitive deficits associated with Alzheimer's disease, ageing, and neurodegenerative disorders. Piracetam and ERGH dramatically lowered AChE activity in this investigation, indicating that both medications stimulate the cholinergic system. As a

result, the ERGH's memory-enhancing impact can be related to its anti-ChE activity.

Conclusion

In this study, we found that ERGH improves memory by increasing Ach levels and encouraging antioxidant properties, which leads to a reduction in serum cholesterol and, ultimately, enhanced memory (spatial and avoidance). As a result, the ERGH appears to have a good effect on memory, and it would be desirable to investigate the plant's potential in the treatment of cognitive impairment.

Acknowledgement

We sincerely tanks to our organization Sri Padmavathi School of pharmacy

References

1. Ringman JM and Cummings JL, Current and emerging pharmacological treatment options for dementia, *Behavneuro*, 17(2006)5.
2. Lanni C, Lenzken SC and Pascale A, Cognition enhancers between treating and doping the mind, *Pharmacol Res*, 57(2008) 196.
3. Jewart, R. D., Green, J., Lu, C. J., Cellar, J. and Tune, L. E. (2005). Cognitive, behavioural, and physiological changes in Alzheimer disease patients as a function of incontinence medications. *Am J Geriatr Psychiatry*. **13**: 324-328
4. Fratiglioni L, Winblad B, Strauss EV. Prevention of Alzheimer's disease and dementia. Major findings from the Kungsholmen project. *Physiol behaviour* 2007; 92:98-104.
5. Blokland A. Acetylcholine: A neurotransmitter for learning and memory. *Brain Res Rev* 1996; 21:285-300.
6. Hock FJ. Therapeutic approaches for memory impairment. *Behav brain Res* 1995; 66:143-50.
7. Ringam JM, Cummings JL. Metrifonate: Update on a new antidementia agent, *J Clin Psychiatry* 1999; 60:776-82.
8. Joshi H, Parle M. Brahmi rasayana improves learning and memory in mice. *Evid Based Complement Alternat Med* 2006; 3:79-85.
9. Vasudevan M, Parle M, Pharmacological actions of *Thespesia Populanea* relevant to Alzheimer's diseases. *Phytomedicine* 2006; 13:677-87.

10. Morris R: Developments of a water-maze procedure for studying spatial learning in the rat. *J Neurosci Methods*, 1984, 11, 47–60.
11. Turner RA. Depressants of the central nervous system .Screening methods in Pharmacology. New York: Academic press; 1965.p.78.
12. Voss G, Sachsse K. Red cell and plasma cholinesterase activities in microsamples of human and animal blood determined simultaneously by a modified acetylcholine/DTNB procedure. *Toxicol Appl Pharmacol* 1970; 16:764-72.
13. Lowry OH, Rosenburg NJ, Farr AL, Randale RJ. Protein measurement with folin phenol reagent. *J Biol Chem* 1951; 193:265-75.
14. Ellman GL. In: Tissue sulphhydryl groups. *Arc Biochem Biophys* 1959; 82:70-3.
15. Kagiya T, Kagiya S, Phillips MI. Expression of angiotensin type 1 and 2 receptors in brain after transient middle cerebral artery occlusion in rats. *Regul Pept* 2003; 110:241-7.
16. Okhawa H, Ohishi N, Yagi K. Assay of lipid peroxides in animals tissue by thiobarbituric acid reaction. *Anal Biochem* 1979; 95:351-8.