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NOOTROPIC EFFECT OF POLYHERBAL FORMULATION ON SCOPOLAMINE INDUCED MEMORY IMPAIRMENT IN EXPERIMENTAL ANIMALS

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Abstract
The goal of the current investigation was to ascertain if an ayurvedic polyherbal formulation (PHF) might improve animal models' capacity for learning and memory. Three plant extracts—*Adoxa moschatellina (AM), Alpinia galanga (AG), and Laurus nobilis (LN)*—were suspended to create the produced PHF with additional excipients. Several behavioral paradigms were used to examine PHF's learning and memory in rats with scopolamine-induced memory impairment. The enzymatic levels of MDA (Malondialdehyde), GSH (Glutathione) and AChE (Acetylcholinesterase) in rats were measured by biochemical estimate. The treatment of PHF, according to the results, caused the duration of social investigation trial 2 (SIT2) to be much shorter than SIT1.Plant extracts dramatically reduced the latency time in the fourth and fifth sessions of the water maze test as compared to the first session. The MWM (Morris Water Maze) test showed a reduction in scopolamine-induced memory impairment after the administration of AM, AG, LN, and PHF. In the pole climbing test, the extracts and PHF were similarly successful in cutting down on latency time. Following treatment with extracts and PHF, biochemical measures showed an increase in GSH levels and a decrease in MDA and AChE levels. study's findings support the notion that PHF is a useful formulation for memory and learning processes that protects against scopolamine-induced memory impairment. Furthermore, it is desirable to separate the active ingredients from various plant extracts.

Keywords: *Adoxa moschatellina, Alpinia galanga, Laurus nobilis*, Pole climbing test, Social recognition test, Latency time.

INTRODUCTION

Two important mental functions that take place in the brain's cerebrum are memory and learning. Because memory is used to store and retrieve knowledge after learning, it is a crucial component of all forms of learning.[1] Neuronal networks in the brain use electrochemical signals for learning and memory processes.[2] A link at the neuronal synapse that may change during learning and memory disruption is known as synaptic plasticity.[3] Restructuring neuronal signaling as a result of altered synapse function is anticipated to follow cognitive behavioral treatment.[4] Cognitive behavioral therapy (CBT) causes training-induced neuroplasticity and provides cognitive knowledge that contributes to improved mental health.[5] Medicinal plants have been utilized to cure a variety of

illnesses since ancient times.[6] Because natural medications have fewer adverse effects and more effectiveness, researchers are currently developing many formulations of these drugs.[7] In order to relieve and cure mental illnesses, traditional remedies are utilized all over the globe in the form of unrefined herbal extracts or herbal formulations.[8] *Alpinia galanga* (L.) wild is a well-known medicinal plant that is related to the *Zingiberaceae* family. It has been used in Asian countries to treat a wide range of illnesses, including diabetes mellitus, inflammation, oxidative stress, diarrhea, ulcers, stomach aches, spasms, and microbial and insecticidal infections. It also has a mildly spicy fragrance. In India, it is utilized as a nervine tonic. *A. galanga* belongs to the edible group of plants, and the rhizome of this plant contains a number of active ingredients in the form of essential oils, including methylcinnamate, trans-p-coumaric acid, alpinin, kampheride, and cincole.[9-13]

Adoxa moschatellina, a flowering plant of the *Adoxaceae* family, is more commonly referred to as moschatel. It is a perennial herb that is evergreen and has a distinct, musky scent.1. This plant is often found in shady regions within the habitats, such as rock fissures, and sunny spots along the borders of streams. Even though it is not as good at dispersing seeds, it expands vegetatively and enters new places.2. Short, robust rhizomes of *Adoxa moschatellina* sprout into new shoots and roots it has shown that Flavonoids, terpenoids, iridoid glucosides, cis-3- and trans-2-hexenol glycosides, morroniside, adoxoside, and secologanin are the components because of its antioxidant and free-radical scavenging qualities, which help it function as a neuroprotective agent. Adoxa moschatellina was shown to significantly enhance behavior, memory, and other aging-related neurological issues when different extracts were used in the study.[14]

Laurus nobilis is an evergreen tree that is a member of the *Lauraceae* family. Its historical usage in folk medicine and cuisine has made it well-known. Numerous investigations have been conducted about its chemical makeup and the possible pharmacological effects it demonstrates. This plant is very important clinically because of its purported antibacterial, antifungal, antiviral, biocidal, antidiabetic, antiulcerogenic, neuroprotective, analgesic, anti-inflammatory, and anticholinergic qualities. Terpenoids, phenolics, and fatty acids are the primary chemical constituents of L. nobilis that exhibit bioactive characteristics. In this study, the primary uses of L. nobilis—particularly for the leaves and fruits—are discussed, along with the relationship between their metabolite content and pharmacological and toxicological characteristics.[15]

METHODOLOGY

Drugs and reagents

Donepezil, Scopolamine, Propylparaben, Methylparaben, Tween80 and Sodium carboxymethyl cellulose were procured from Sigma Aldrich, USA. All other solvents were of analytical grade and distilled water was used throughout the study.

Preparation of poly herbal formulation (PHF)

The created PHF included extracts from *Adoxa moschatellina* (AM), *Alpinia galanga* (AG), and *Laurus nobilis* (LN) weighing 0.50, 14.3, and 0.3343 g, respectively. Parabens, Tween 80, and a small quantity of sodium carboxymethyl cellulose were also added, and the mixture was then evenly triturated to create a smooth paste. To get a suspension, the paste was vortexed using a mechanical stirrer set at 500 rpm after being washed with 100 cc of distilled water.

Experimental design and drug administrations

The impact of extracts from *Adoxa moschatellina (AM), Alpinia galanga (AG), and Laurus nobilis (LN)* as well as their PHF was assessed in models of scopolamine-induced memory impairment.

Scopolamine induced memory impairment

On the fifth day, scopolamine was administered to cause impairment in social recognition. A variety of experimental models, including the Morris water maze, and the Social Recognition Test, were used to determine the memory function test. The animals were split up into eleven groups: Group 1 was the control group, which received a vehicle treatment orally (p.o); Group 2 was the toxic group,

which received scopolamine (i/p); Group 3 was Piracetam (200 mg/kg, i/p); Group 4 AM1 extract (100 mg/kg, p.o); Group 5 AM2 extract (200 mg/kg, p.o); Group 6 AG1 extract (100 mg/kg, p.o); Group 7 AG2 extract (200 mg/kg, p.o); Group 8 LN1 extract (100 mg/kg p.o); Group 9 LN2 extract (200 mg/kg, p.o); Group 10 PHF1 extract (100 mg/kg, p.o); Group 11 PHF2 extract (200 mg/kg, p.o). The animals were given medication for five days in a row, with the evaluation of memory function occurring on the fifth day.

Assessment of learning and memory

Social recognition test (SRT)

PHF's effectiveness was assessed in male adult rats weighing between 225 and 250 grams. Scopolamine-induced memory impairment related to social recognition. On the fifth day after the medicine was administered, scopolamine (1.25 mg/kg) models were tested in the cages. The 50–60 g tiny rats were used to provide the social stimulation. The social contact between adult and adolescent rats was observed for duration of five minutes (T1). After two hours, the young rats were taken out of their cages and put back in to measure the social contact time (T2)[16].

Morris water maze (MWM) test

Based on its effectiveness in SRT, the maximum dosage of PHF and individual plant extracts, or 200 mg/kg, was chosen for use in both the current and subsequent animal models. In the scopolamine model (1 mg/kg), male adult rats weighing between 225 and 250 grams were put through the Morris water maze (MWM) test starting on the fifth day. In this model, a 45 cm by 26 cm water pool with four distinct beginning positions (N, E, SE, and NW) was employed. Animals were dropped into the pool from any point, and if they couldn't get out in under 120 seconds, they were sent to the side and left for 30 more minutes. The water maze's escape latency time was used to calculate the index of learning.[17]

Biochemical analysis

Evaluation of MDA level in scopolamine-induced amnesic rat brain

Principle

A malondialdehyde (MDA)-thiobarbituric acid (TBA) adduct is formed as a result of the interaction between MDA and TBA, which is the basis for this particular test. Utilizing this reaction is the most common approach to determining the amount of MDA present in biological materials. Interference, on the other hand, may be a severe concern in some biological samples if it is not dealt with in the suitable manner. The high backgrounds that are often associated with the TBARS response may be dealt with using our test, which offers the most accurate and efficient method yet developed.

A colorimetric test known as the thiobarbituric acid reaction (TBAR) was used to assess the quantity of malondialdehyde (MDA). In summary, brain homogenate and TBA reagent were combined in a test tube and incubated for 60 minutes at 90°C. After chilling on ice cubes, the test tube was centrifuged for 10 minutes at 1000 RPM. In a microplate reader, the supernatant was measured at 534 nm.[18]

Evaluation of GSH level in scopolamine-induced amnesic rat brain

Principle

An oxidized glutathione–TNB adduct (GS–TNB) and the TNB chromophore, which has a peak absorbance at 412 nm, are produced as a result of the interaction between glutathione (GSH) and DTNB, which is sometimes referred to as Ellman's reagent. This reaction lies at the foundation of the test. After that, the disulfide product, which is GS–TNB, is reduced by GR in the presence of NADPH, which recycles GSH back into the process to complete the reaction.The quantity of glutathione that is measured is the sum of the oxidized and reduced glutathione which is present in the sample. This is because GR is responsible for reducing the GSSG that is generated into 2GSH. For example, [GSH]total = [GSH] + 2 × [GSSG]. For the sake of simplicity and consistency in measurement, the rate of change in absorbance (ΔA412 nm min–1) is assumed to be linear.

Furthermore, this linear relationship is linearly related to the total concentration of GSH used in the experiment. It is possible to ascertain the concentration of an unidentified sample by doing calculations based on the linear equation or regression curve that is derived from the standard amount of GSH.[19]

Using this approach, GSH was measured by first making brain homogenate, which was then precipitated in 10% trichloroacetic acid concentration and centrifuged for 5 minutes at 12,000 rpm. After incubation, the absorbance of the supernatant was measured at 425 nm.

Evaluation of AChE level in scopolamine-induced amnesic rat brain

Principle

The Acetylcholinesterase Assay Kit is based on an enhanced Ellman technique, in which the thiocholine that is formed as a result of the action of acetylcholinesterase and 5,5'-dithiobis(2-nitrobenzoic acid) combine to give a yellow hue. The amount of enzyme activity present in the sample is directly proportional to the color intensity of the product that is being tested

As 100µL of Ellman reagent was added to each well of 96 well plates, the amount of acetyl cholinesterase (AChE) was determined. Protein content in brain homogenate was determined after 10µL of brain homogenate was applied to each well containing Ellman reagent and the absorbance was recorded at 456 nm in a microplate reader.[17]

RESULTS

Effect of PHF in SRT behavioral model

The findings showed that SIT2 was much lower than SIT1.It showed good earnings as a result of PHF treatment. On the other hand, the scopolamine-induced group that was not given the test medication did not exhibit any variation in time between trial 1 and trial 2. SIT2 was seen to be significantly lower in the group that received piracetam (200 mg/kg). Treatment with 200 mg/kg of AM, AG, and LN extracts has shown a significant decrease in SIT2 in comparison to SIT1. It suggested preventing memory impairment brought on by scopolamine (Figure 1). PHF prevented scopolamine-induced memory impairment better than individual plant extracts. Additionally, a noteworthy difference was seen in the recognition index between the vehicle, control, and Scopolamine groups, indicating memory impairment (Figure 2). The groups treated with piracetam, individual herbal extracts (200 mg/kg), and PHF (200 mg/kg) had a considerably lower recognition index.

Table1: PHF's effect on the recognition index in amnesia caused by scopolamine

Groups	Recognition index
Control	0.616±0.032
Vehicle	0.515±0.081
Scopolamine	0.936±0.071
Piracetam	0.466±0.052***
AM 100	0.681±0.111*
AM 200	0.563±0.021**
LN100	0.782±0.101*
LN200	0.591±0.041**
AG 100	0.753±0.111*
AG 200	0.562±0.032***
PHF 100	0.705±0.071
PHF 200	0.502±0.043***

Figure 1: PHF's effect on the recognition index in amnesia caused by scopolamine

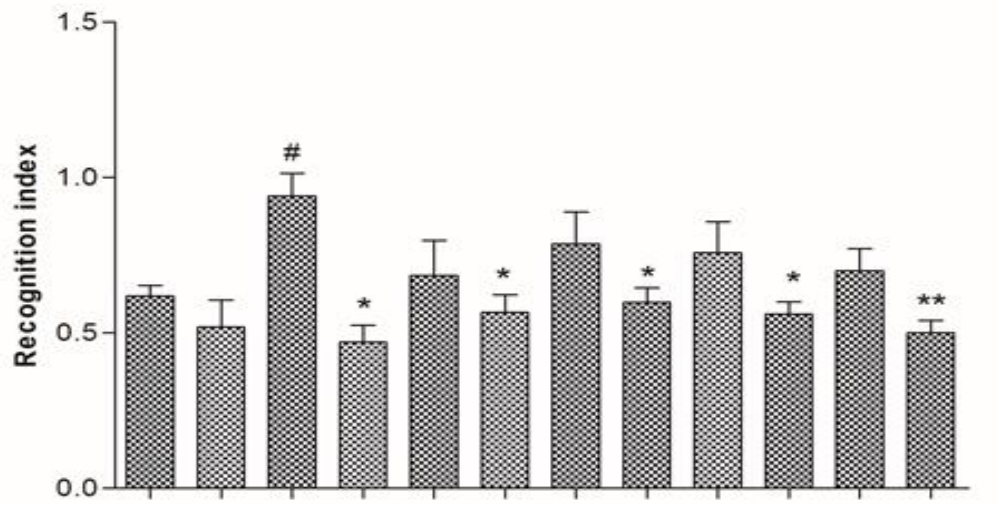
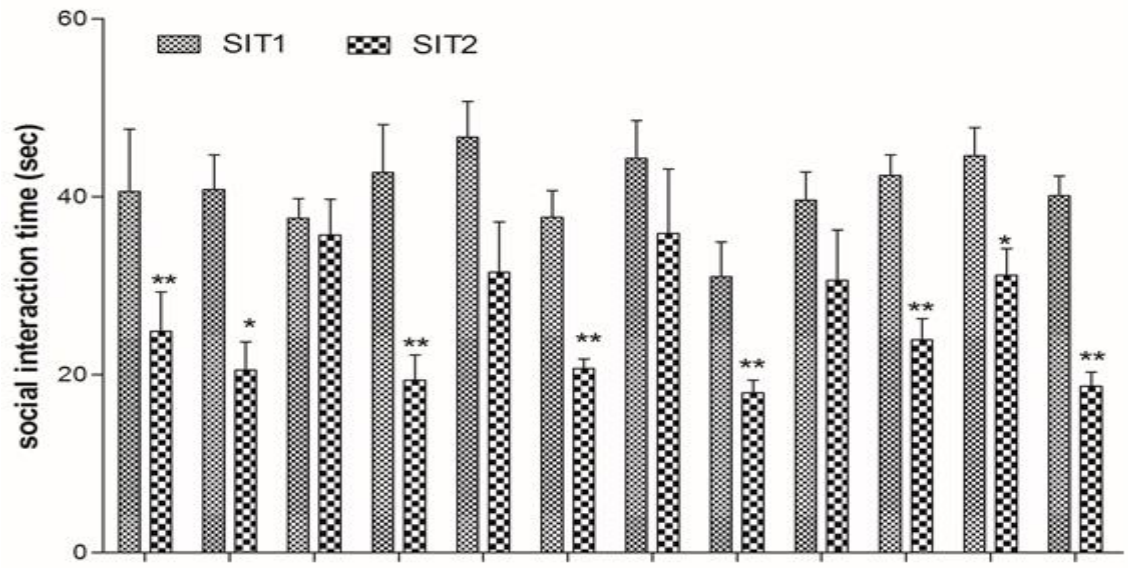


Table.2: Impact of PHF on SRT in amnesia caused by scopolamine

Groups	SIT1	SIT2
Control	39.6±3.0	23.9±4.1
Vehicle	39.8±2.9	21.5±3.2
Scopolamine	36.6±4.2	34.7±4.1
Piracetam	41.7±2.4	20.4±2.4***
AM 100	45.7±5.0	30.5±5.2*
AM 200	35.7±1.0	21.7±1. *1
LN 100	42.3±3.3	34.9±7.4**
LN 200	30.0±2.9	16.0±1.2***
AG 100	36.6±2.2	31.6±5.5*
AG 200	42.4±21.3	22.9±2.1***
PHF 100	41.6±5.2	30.2±3.4
PHF 200	40.1±1.2	17.7±1.2***

Figure.2: Impact of PHF on SRT in amnesia caused by scopolamine



Effect of PHF in MWM behavioral model

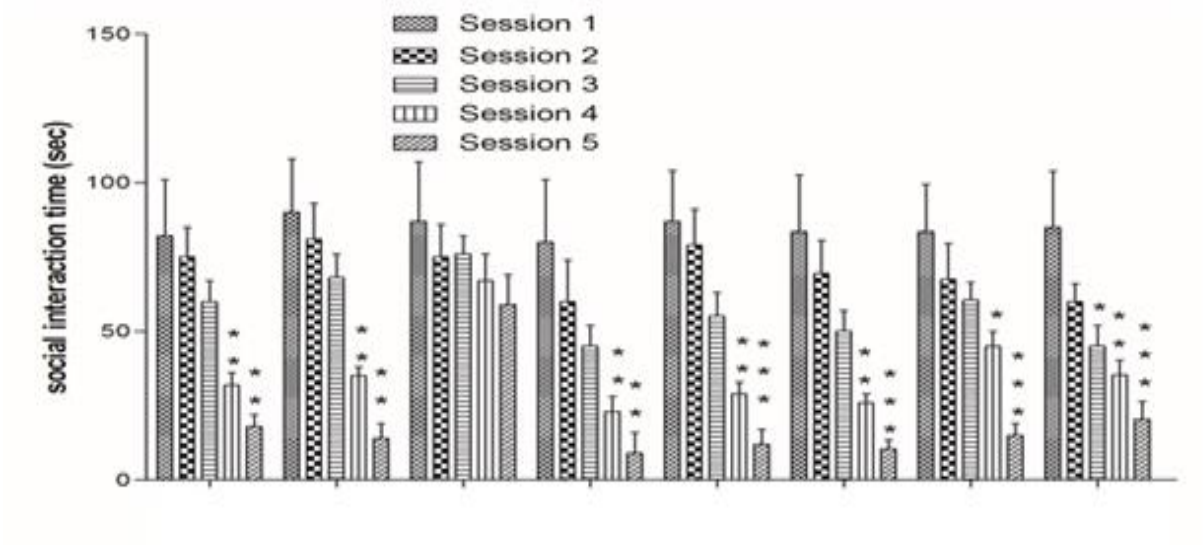
In comparison to session 1, a note worthy reduction in latency time was seen in the control [F(4,20)=7.0] and vehicle [F(4,20)=9.1] groups throughout the fourth and fifth sessions

(Figure 3). All of the water maze sessions were characterized by memory impairment due to the injection of scopolamine. The fourth and fifth sessions' latency times were significantly lower [F(4,20)=6.5] than the first session's due to piracetam treatment.[16] Animals with memory impairment showed amelioration when administered 200 mg/kg of plant extracts. Scopolamine-induced memory impairment was lessened when extracts of AM [F (4, 20) = 9.5], LN [F (4, 20) = 8.2], and GA [F (4, 20) = 7.1] were administered. No, there was no difference seen in the latency times between any group's first and second sessions (Figure 3). In rats with a memory impairment produced by scopolamine, the PHF also dramatically decreased [F(4,20)=6.9] latency starting in session 3, showing that the MWM task was successfully learned.

Table 3: PHF's impact on scopolamine-induced forgetfulness in the Morris Water Maze test device

Groups	Session 1	Session 2	Session 3	Session 4	Session 5
Control	83.0±18	76.0±9	61.0±6	33.0±1	17.0±1
Vehicle	90.0±18	81.0±12	68.0±8	35.0±3	14.0±5
Scopolamine	87.0±20	76.0±11	75.0±5	66.0±8	58.0±11
Piracetam	80.0±21	60.0±14	45.0±7	23.0±5	09.0±7
AM 200	86.0±16	78.0±13	56.0±6	27.0±4	13.0±4
LN 200	82.5±19	68.0±10	51.0±6	24.0±3	11.5±2
AG 200	84.0±15	66.0±10	61.0±5	46.0±6	16.0±3
PHF 200	83.0±19	61.0±6	44.0±7	36.5±5	21.0±5

Fig. 3: PHF's impact on scopolamine-induced forget fulness in the Morris Water Maze test device



Biochemical estimations

Effect of PHF on MDA levels in scopolamine-induced amnesic rat brain

When scopolamine was administered to rats, their hippocampal and cortical MDA levels were significantly higher than those of the vehicle and control groups. MDA levels in both brain areas were considerably lowered by piracetam preventive therapy. The administration of 200 mg/kg plant extracts resulted in a considerable reduction in MDA levels in the hippocampus and cortex of rats treated with scopolamine. MDA levels in several brain areas significantly decreased as a result of PHF therapy (Figure 4).

Fig.4: PHF's effect on MDA levels in rats given scopolamine-induced amnesia

Groups	Cortex (nmol/mg protein)	Hippocampus (nmol/mg protein)
Control	27.4±3.04	22.9±1.14
Vehicle	29.9±3.69	25.1±2.43
Scopolamine	47.6±2.28	49.5±3.56
Piracetam	34.2±2.71	32.0±2.18***
AM 200	36.6±3.17	35.4±1.96**
LN 200	34.1±3.50	34.3±2.42**
AG 200	30.5±1.37	30.5±1.37**
PHF 200	31.4±1.55	26.5±2.04***

The data is shown as mean ± standard error of mean, and it is substantially different at #P < 0.05 compared to the vehicle and control groups and at *P < 0.05 and **P < 0.01 compared to the scopolamine group.

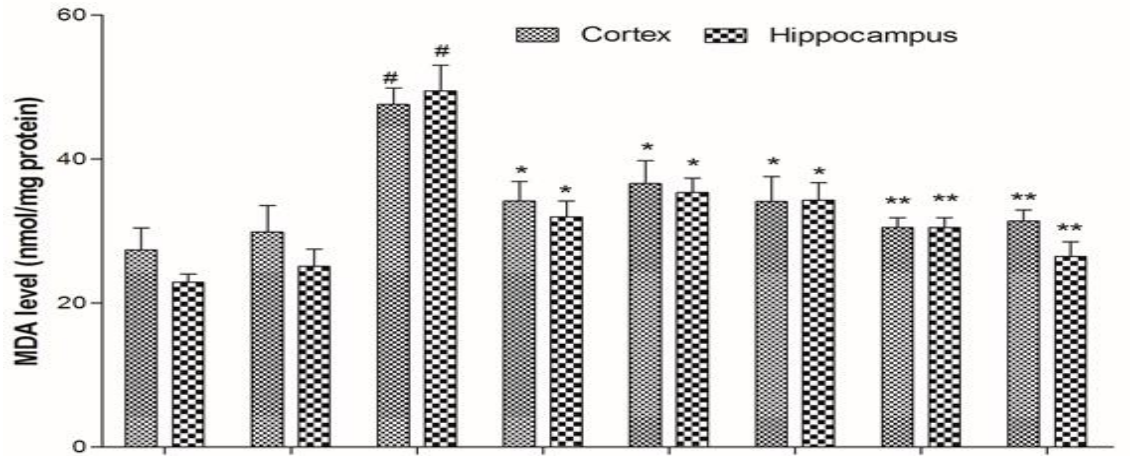


Fig.4: Effect of PHF on MDA level in scopolamine-induced amnesia

Effect of PHF on GSH level in scopolamine-induced amnesic rat brain

Using the calibration curve produced with various glutathione concentrations, the GSH level was measured. GSH levels in brain areas were significantly lower in the Scopolamine group than in the vehicle and control groups. Piracetam dramatically inhibited the scopolamine-induced decrease in GSH levels in the brain and hippocampus, as shown in Figure 5. The drop in GSH levels caused by scopolamine was stopped by the administration of plant extract. In several areas of the brain, AM, LN, and AG extract increased GSH levels. Additionally, PHF at 200 mg/kg increased GSH levels in some brain areas (Figure 5)

Table 5: Effect of PHF on GSH levels in amnesia caused by scopolamine

Groups	Cortex (µ/mg protein)	Hippocampus(µ/mg protein)
Control	145.6±7.09	132.4±8.73
Vehicle	158.3±10.98	133.3±7.20
Scopolamine	117.0±5.60	108.5±4.09
Piracetam	144.4±9.58	136.9±7.85***
AM 200	141.8±8.22	131.0±6.37**
LN 200	152.9±7.20	140.2±9.73**
AG 200	147.0±7.74	130.4±5.08**
PHF 200	177.3±8.01	149.8±9.07***

The data are shown as mean ± SEM and are statistically significant at #P < 0.05 when compared to

the vehicle and control groups, and at *P < 0.05 and **P < 0.01 when compared to the scopolamine group.

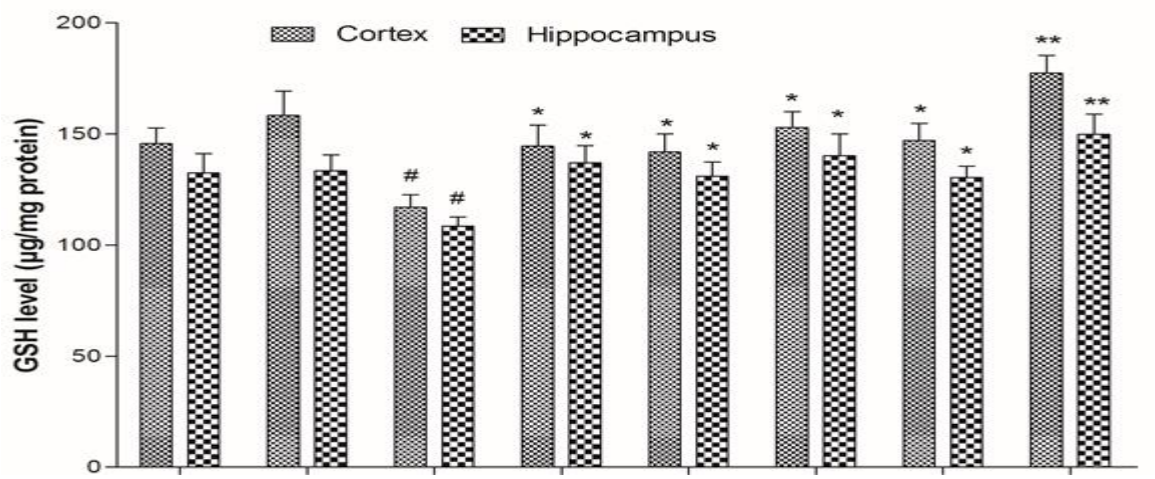


Fig 5: PHF's effect on GSH levels in mice rendered amnesic by scopolamine

Effect of PHF on AChE level in scopolamine-induced amnesic rat brain

In the brain areas of rats with a memory loss produced by scopolamine, there was a considerable increase in AChE activity. AChE activity in many brain areas was considerably decreased by piracetam preventive therapy (Figure 8). The rise of AChE activity caused by scopolamine was inhibited by the injection of plant extract. Animal AChE activity was considerably reduced by AM, LN, and AG extract. AChE activity was likewise decreased in the brain areas by PHF at 200 mg/kg (Figure 6).

Table 6: PHF's effect on acetylcholinesterase levels in amnesia caused by scopolamine

Groups	Cortex	Hippocampus
Control	0.0037±0.0005	0.0041±0.0004
Vehicle	0.0037± 0.0004	0.0040±0.0004
Scopolamine	0.0058±0.0006	0.0062±0.0005
Piracetam	0.0031±0.0006	0.0034±0.0005***
AM 200	0.0037±0.0005	0.0044±0.0003***
LN 200	0.0039±0.0004	0.0042±0.0005***
AG 200	0.0037±0.0004	0.0040±0.0006***
PHF 200	0.0027±0.0003	0.0029±0.0004***

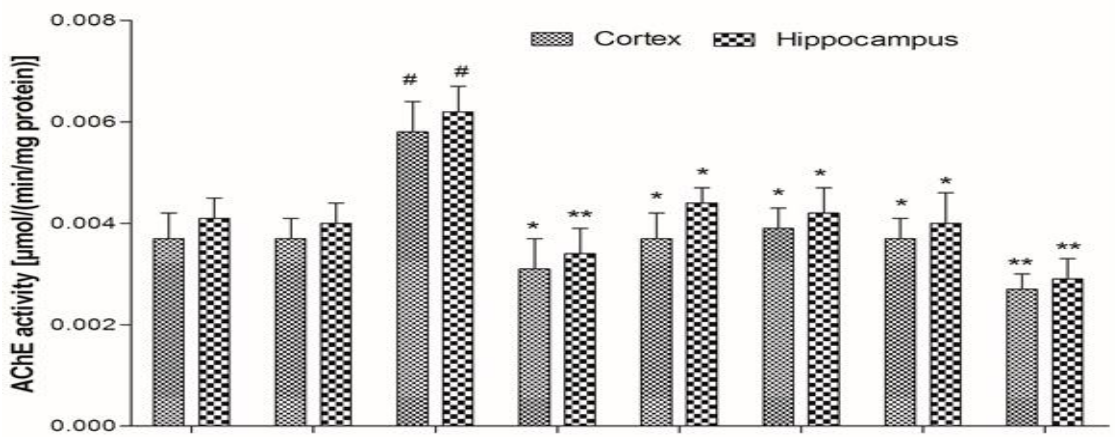


Figure 6: PHF's effect on AChE levels in rats with scopolamine-induced amnesia

The data are shown as mean ± SEM and are statistically significant at #P < 0.05 when compared to the vehicle and control groups, and at *P < 0.05 and **P < 0.01 when compared to the scopolamine group

DISCUSSION

The drug's research team exploited plants as a reliable source of cutting-edge phytoconstituents that may be used to treat a variety of illnesses. There are several chances to develop novel medications for memory impairments thanks to the phytotherapeutic approach.[20] This research assessed a polyherbal formulation (PHF) including three brain-tonic herbs: *Adoxa moschatellina*, *Alpinia galanga*, and *Laurus nobilis*, against experimental models of scopolamine-induced memory loss. We discovered in this cognitive-behavioral research that several animal models' learning and memory processes benefited from this formulation[22-23].

These medicinal plants were chosen for their historic usefulness as nervine tonics and memory enhancers in ancient India. According to reports, Laurus nobilis has been shown to be a neuroprotective herb and to reduce neurotoxicity in the Drosophila model. The work of Malik et al. supports our findings that PHF containing LN as one of the herbal medications decreased scopolamine-induced amnesia.26 Since scopolamine affects rats' and humans' short- and long-term memory functions.[27] Laurus nobilis has also been shown by researchers to be a potent neuroprotective herb that may help prevent tumors, depression, epilepsy, anxiety, and ulcers.Many behavioral research have used neuroprotective medications to counteract oxidative stress in the brain because the production of free radicals signals the advancement of cognitive deterioration [7,17,18,19,] In this investigation, PHF decreased AChE activity in both brain areas in a way that was dose-dependent. The hippocampal region is involved in memory development, organization, and storage as well as the consolidation of information from short-term to long-term memory.[28-29]

CONCLUSION

The study's findings support the hypothesis that PHF prevented oxidative damage, improved cholinergic function, and improved brain functioning to lessen scopolamine-induced amnesia. PHF may thus be a viable option to stop memory loss in some neurodegenerative illnesses.

The in vivo effects of PHF on scopolamine-induced amnesia demonstrated that our findings were consistent with the research conducted [122], which suggests that the herbal extract has the ability to enhance memory by improving the recognition index in a model of scopolamine-induced amnesia. According to Scopolamine hinders memory and learning in animals by blocking the activation of cholinergic neurons in the central nervous system[123]. The rhizomes of A.galanga have shown a neuroprotective effect on mice with amnesia produced by Aβ, as indicated in a study. A combination of many herbal ingredients, including AM,AG, and LN has shown the ability to improve memory in cases of amnesia produced by scopolamine, as reported[30] stated that A.galanga is an effective plant for boosting memory. Furthermore, our investigation found that *L.nobilis* was effective in mitigating scopolamine-induced amnesia, as shown by the data obtained[31].

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CONFLICT OF INTEREST

Nil

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