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EXPERIMENTAL PAPER

Effect of *Nyctanthes arbor tristis* leaf extract against scopolamine-induced cognitive impairment in rats

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Summary

Nyctanthes arbor tristis (NATE) ethanol extract (150 mg/kg, orally) was evaluated for its protective effect against scopolamine-induced (1 mg/kg i.p.) cognitive impairments in rats using behavioral models like radial arm maze test, Morris water maze test and active avoidance test. NATE effect was evaluated and compared with the standard piracetam (200mg/kg i.p.). NATE ($p<0.005$) significantly reversed the impairment produced by the scopolamine in radial arm maze test. In addition, NATE also decreased the time period taken to find the hidden platform in Morris water maze test and increased number of avoidances in active avoidance paradigm. Acetylcholinesterase activity and thiobarbituric acid levels were significantly ($p<0.005$) decreased along with the rise in activities of superoxide dismutase and catalase. This might suggest that the NATE has protective effect against scopolamine-induced cognitive impairment in rats through acetylcholine muscarinic receptor pathway and also antioxidant activity. No significant changes were found in histopathological studies of brain.

Key words: *Nyctanthes arbor tristis, active avoidance test, Morris water maze test, radial arm maze test, piracetam*

INTRODUCTION

An important function of brain is memory or cognition, without which daily activities are disturbed. According to psychology memory is the ability of an organism to store and recall information along with experiences [1]. The major risk factors that lead to cognitive impairment or memory loss are age, stress, genetic factors and also emotions. This might lead to Alzheimer's disease and other neurodegenerative disorders. Cholinergic nuclei damage in the brain in acetylcholinergic pathway related to memory and learning is the major cause of Alzheimer's disease (AD) [2]. Many attempts have been made in the targeted treatment of AD through the acetylcholinesterase pathway by inhibition of acetylcholinesterase enzyme. Cognitive impairment is achieved by inducing an acetylcholine muscarinic receptor antagonist *i.e.* scopolamine [3].

Nyctanthes arbor tristis L., a sacred ornamental plant belonging to the family *Oleaceae*, also known as Parijata (Night Jasmine) was considered to be one of the five wish-granting plants of Hindu mythology. The phytochemical analysis of the plant revealed the presence of flavonoids, volatile oils, ascorbic acid, mannitol, glycosides and phenols [4]. The leaves extract has many proved pharmacological effects like anti-bacterial [5], analgesic, anti-inflammatory [6], anti-diabetic [7], anti-arthritic [8], antioxidant [9], hepatoprotective [10] and antispasmodic activities [11]. Basing on its antioxidant properties, the present research work has been taken up to find the protective effect of ethanol extract of *Nyctanthes arbor tristis* against scopolamine-induced cognitive impairment in rats.

MATERIALS AND METHODS

Plant material

Plant leaves were collected in May and June from Warangal and authenticated by Dr. Vatsavaya S. Raju, Professor in botany from Kakatiya University, Warangal, India and then voucher specimen was deposited KUW_Acc.No. 1872.

Preparation of plant extract

The leaves were shade dried and then powdered. The powder was taken for maceration with 90% ethanol at a room temperature. The extract was filtered and the residue was excluded. The filtrate was evaporated to dryness and then used. Acute toxicity studies found no deaths up to 2000 mg/kg. The dose of 150 mg/kg was selected for the study [12].

Animals

All experiments were conducted using albino Wistar mal rats (150–200 g), at about 6–8 weeks of age. All animals were procured from Sanzyme Limited, Hyderabad. The animals were maintained with free access to food and water and kept at 25±2°C under controlled 12 h light/dark cycle. Twelve hours before each experiment food was not provided, water was provided *ad libitum*. The care and maintenance of the animals were carried out as per the approved guidelines of the Committee for the Purpose of Control and Supervision of Experiments on Animals (CPCSEA), New Delhi. (Registration Number: 08 / SPIPS / IAEC / 13).

Chemicals

Scopolamine hydrobromide (Buscopan, Boehringer-Ingelheim,USA), acetylcholine chloride, 5,5-dithio-bis2-nitrobenzoicacid, (Ellman's reagent), acetylthiocholineiodide, trichloroacetic acid, thiobarbituric acid (TBA) all were purchased from Sigma–Aldrich (Bangalore, India), Piracetam (Nootropil, UCB India).

Treatment protocol and grouping of animals

Animals were divided into five groups with six rats each. The groups were as follows:

Group 1: normal control group – receiving only vehicle.

Group 2: positive control group – scopolamine-induced (1 mg/kg i.p.) cognitive impaired group.

Group 3: standard treatment group – piracetam (200 mg/kg orally) against scopolamine-induced cognitive impairment.

Group 4: test treatment group – NATE (150 mg/kg orally) against scopolamine-induced cognitive impairment.

Group 5: standard + test treatment group – NATE (150 mg/kg orally) + piracetam (200 mg/kg oral) – against scopolamine-induced cognitive impairment.

Animals were trained for radial arm maze, Morris water maze, and active avoidance test by conducting one daily training trial during which they did not receive any drug. The completely trained animals were chosen for the study. These animals were dosed once a day with respective drugs for eight days along with daily training trial. Scopolamine was given on 8th day 45 min after the treatment. After one hour all animals were tested on radial arm maze performance, Morris water maze and active avoidance test.

Evaluation of NATE effect using radial arm maze test

A radial arm maze was used here for evaluation of working memory in animals. Each arm (50 x 12 cm) of the eight-arm radial maze extends from an octagonal shaped central hub of 30 cm diameter. The platform is elevated 40 cm above the floor, small black metal cups (3 cm in diameter & 1 cm deep) are mounted at the end of each arm that serve as receptacles for reinforces food [3]. The rats of all groups were tested for working memory errors on the 8th day of treatment with respective drugs [13].

Evaluation of NATE effect using active avoidance test

Active avoidance test was conducted in medicraft jumping box. It was divided in to two equal chambers (27×29×25 cm) by Plexiglas partition, with a gate providing access to the adjacent compartment through a 14×17 cm space. Numbers of avoidances were observed on the last day of the treatment with the standard and test drugs [3].

Evaluation of NATE effect using Morris water maze test

Morris water maze test was conducted to find out the memory regaining capacity of brain. Morris water maze consists of a cylindrical tub of 117 cm diameter and 30 cm depth. The tub was filled with water up to 12 cm below the tub rim. A circular platform was placed at a place approximately 1 cm below the water surface. All the animals were trained before the experiment with normal water and then the platform was hidden by mixing with milk [3].

Estimation of biochemical parameters of oxidative stress

Biochemical tests were conducted 24 h after last behavioral test. The animals were sacrificed by inhalation of carbon dioxide. Brains were removed and rinsed with ice-cold isotonic saline. Then, brains were homogenized with ice-cold phosphate buffer (pH 8). The homogenates (10% *w/v*) were then centrifuged at 10,000 rpm for 15 min and the so formed supernatant was used for the biochemical estimations.

Measurement of lipid peroxidation (LPO)

The amount of malondialdehyde (MDA) present in the brain was estimated using TBARS – thiobarbituric acid reactive substances method mentioned by Ohkawa *et al* [14] using spectrophotometer at 532 nm wavelength.

Estimation of superoxide dismutase activity (SOD)

Photo oxidation of o-dianisidine with riboflavin method according to Arutla *et al* was used to estimate the SOD activity in brain by recording the absorbance at 460 nm wavelength using spectrophotometer [15].

Catalase activity

Hydrogen peroxide oxidation by catalase enzyme was estimated at 240 nm wavelength using spectrophotometer according to Luck et al. [16].

Estimation of acetylcholinesterase activity

The acetylcholinesterase activity was estimated using Ellman's method [17]. The esterase activity is measured by providing an artificial substrate acetyl thiocholine. The absorbance was measured at 412 nm wavelength using spectrophotometer.

Estimation of acetylcholinesterase levels using frog rectus abdominus muscle (FRAM)

Acetylcholinesterase levels were assayed using frog rectus abdominal muscle. When brain homogenate was given along with sub maximal dose of acetylcholine, the response of acetylcholine was found to be reduced and this can be attributed to the presence of acetylcholineesterase enzyme in rat brain homogenate. Initially the dose response curve was recorded with different concentration of acetylcholine on muscle preparation by taking 60 seconds as base line and 90 seconds as contact time and 3 minutes for washing. Then the sub maximal dose of acetylcholine is identified and added to graded doses of brain homogenate and incubated at 37°C for 15 min. The incubated preparations were added to tissue bath and responses were recorded on kymograph drum. In the next phase the brain homogenates from scopolamine treated rats, piracetam treated rats, NATE treated rats and NATE+ Standard drug piracetam was added to sub maximal doses of acetylcholine to estimate the activity of acetylcholine in the brain in the presence of drugs listed above and the plant extract [18].

Histopathological studies

The brains of the rats were isolated and stored in the 10% formaldehyde solution untill used. Brains were analyzed to estimate the damage of brain cells. These studies were conducted to know the extent of damage to brain cells caused by scopolamine. Eosin and haematoxylin dyes were used to stain the slides. Images were observed under 40 X of face contrast microscope.

RESULTS AND OBSERVATIONS

Effect of NATE on behavioral parameters

Working memory errors were increased in scopolamine-induced group compared to that of normal control group, which indicates the cognitive impairment achieved. Treatment with NATE (150 mg/kg orally) has shown the protective effect. However, the NATE doesn't have the effect as much as the standard piracetam, shown protective effect in cognitive impairment (tab. 1, fig. 1).

Table 1.

Effect of NATE on working memory errors in radial arm maze test

Group	No. of working memory errors
Control	0.33±0.2
Scopolamine	2.8±0.7*
Piracetam	0.5±0.2
NATE	1.6±0.2#
NATE + piracetam	1.16±0.3

*$p < 0.005$, as compared with the control group
# $p < 0.005$, as compared with the scopolamine treated group
Results are in the form of mean ±SEM. Analysis was carried out using ANOVA, followed by Bonferroni's comparisons. $p < 0.005$ was considered as statistically significant.

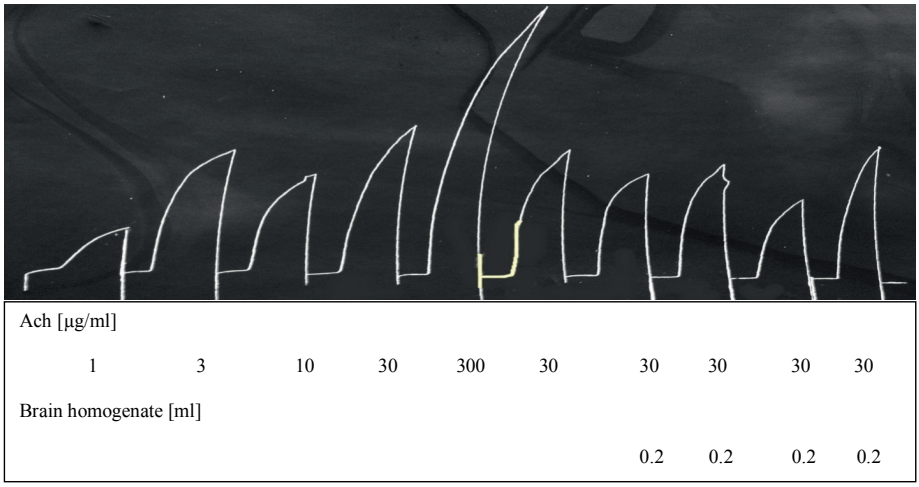

Figure 1.

Control group. Effect of brain homogenate from rats of control group on acetylcholine activity using FRAM preparation

Time taken to find the hidden platform in the scopolamine-induced group has increased, as compared to control group, proving the cognitive impairment. NATE treated group prevented the cognitive impairment by decreasing the time taken for finding the hidden platform. Standard treatment has shown more effect compared to the group received the combination of NATE and standard (tab. 2, fig. 2).

Table 2.

Effect of NATE on time taken to find the hidden platform in Morris water maze test

Group	Time in sec to find the hidden platform
Control	57.5±2.7
Scopolamine	105.0±3.9*
Piracetam	62.0±1.71
NATE	84.6±2.82#
NATE + Piracetam	72.3±2.10

*p< 0.005, as compared with control group
# p<0.005, as compared with scopolamine-treated group.
The values were shown as mean ±SEM. ANOVA one way analysis along with Bonferroni's test was used for statistical analysis. p<0.005 was considered as statistically significant.

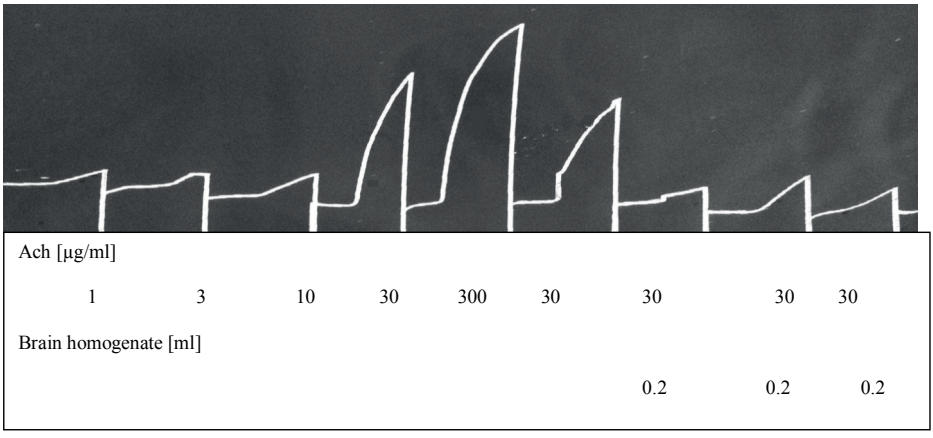

Figure 2.
Scopolamine-induced group. Effect of brain homogenate from rats receiving scopolamine on acetylcholine activity using FRAM preparation

Number of active avoidances reduced in scopolamine-induced group confirms cognitive impairment. NATE treatment has increased the number of avoidances compared to that of scopolamine-induced group, according to table 3 and figure 3.

Table 3.

Effect of NATE on number of avoidances against scopolamine induced cognitive impairment in rats

Group	Numberof avoidances
Control	10.1±0.3
Scopolamine	5.6±0.4 *
Piracetam	8.1±0.3
NATE	7.5±0.5#
NATE + piracetam	9.3±0.2

*$p<0.005$, as compared with control
# $p<0.005$, as compared with scopolamine-treated group. The values are expressed as mean ±SEM. ANOVA one way analysis followed by Bonferroni's test was used in statistical analysis. $p<0.005$ was considered as statistically significant.

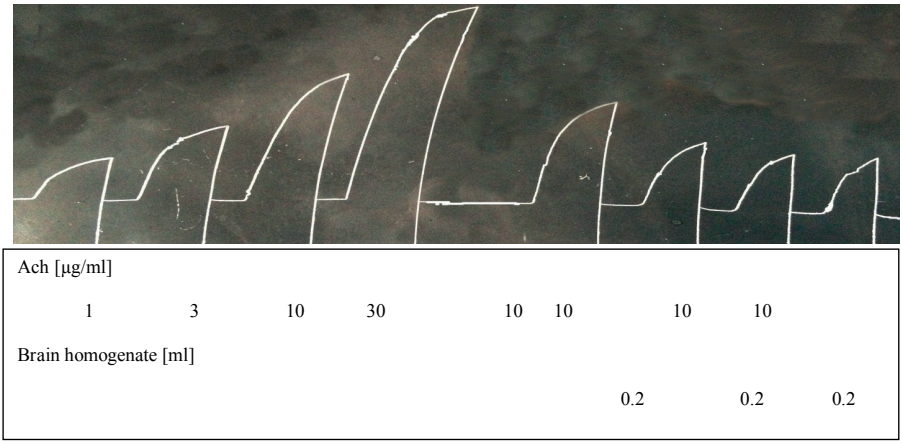

Figure 3.

Piracetam treated group (standard). Effect of brain homogenate from piracetam-treated rats on acetylcholine activity using FRAM preparation

The antioxidant activity of the enzymes such as superoxide dismutase (SOD) and catalase were significantly inhibited in scopolamine-induced group when compared with normal control group. NATE treatment significantly prevented the loss of activity of these antioxidant enzymes when compared to scopolamine given group. Scopolamine induction significantly increased the brain malondialdehyde (MDA) levels compared to control group. NATE treatment significantly prevented the rise in brain MDA levels compared to scopolamine treated group (tab. 4).

Table 4.

Effect of NATE on oxidative stress parameters

Group	SOD (% superoxide ion scavenging activity)	Catalase (% H_2O_2 scavenging activity)	Lipid peroxidation (nmoles/g brain tissue)
Normal control	66.9±2.3	65.76±4.7	22.9±1.19
Scopolamine	29.3±1.4*	44.6±1.15*	47.60±1.05*
Piracetam	59.7±0.4	53.25±1.16	23.79±1.14
NATE	40.3±1.2#	48.42±0.89#	38.52±1.2#
NATE + piracetam	64.39±1.45	61.35±1.90	27.16±0.60

*$p<0.005$, as compared with control group
# $p< 0.005$, as compared with scopolamine-treated group

Effect of NATE on parameters of oxidative stress

Superoxide dismutase activity (SOD)

Superoxide dismutase is an antioxidant enzyme and its activity has been expressed in % inhibition rate. The anti-oxidant activity of the enzyme was inhibited in scopolamine-induced group when compared with normal control group ($p<0.005$). NATE treatment protected the brain against oxidation by preventing inhibition of enzyme activity when compared to scopolamine treated group ($p<0.005$).

Lipid peroxidation (LPO)

Induction with scopolamine significantly increased MDA levels, as compared to control group indicated by lipid peroxidation ($p<0.005$). However NATE treatment (150 mg/kg) prevented the rise in MDA levels indicating antioxidant property in providing protection against oxidative damage induced by scopolamine ($p<0.005$).

Catalase activity

Catalase is an antioxidant enzyme and its activity has been expressed in % inhibition rate. The anti-oxidant activity of the enzyme was inhibited in scopolamine given group when compared with normal control group ($p<0.005$). NATE treatment protected the brain against oxidation by preventing inhibition of enzyme activity, as compared to scopolamine-administered group ($p<0.005$).

Effect of NATE on acetylcholinesterase activity in rat brain

Acetylcholinesterase levels were increased in the scopolamine-induced group compared to control leading to decrease in acetylcholine levels ($p<0.005$). NATE

treatment has succeeded in bringing down the acetylcholinesterase levels and increasing the acetylcholine levels compared to scopolamine treated group ($p<0.005$). NATE treatment is not effective as standard treatment where the levels are close to control group levels.

Effect of NATE on acetylcholine levels using frog rectus abdominus muscle

Bioassay was carried out to find the acetylcholinesterase levels, the antilog concentrations were used to estimate the levels.

In scopolamine-induced group the acetylcholine levels were drastically reduced. Treatment with NATE has prevented scopolamine from blocking ACh levels.

When 0.2 ml of brain homogenate (contains acetylcholineesterase enzyme) from control group animal was added to sub-maximal dose of acetylcholine (30 μg/ml), a fall in the response of acetylcholine was observed.

According to these kymograph readings, as shown in figures 1 to 5 and in table 6, it was clear that the activity of acetylcholine was reduced by the acetylcholineesterase present in brain homogenate and which was drastically reduced in the brain homogenates of scopolamine-treated group. NATE-treated group brain homogenates have shown the increased levels of acetylcholine levels compared to that of scopolamine induced group. However the activity of NATE treated group was smaller, as compared to Standard piracetam.

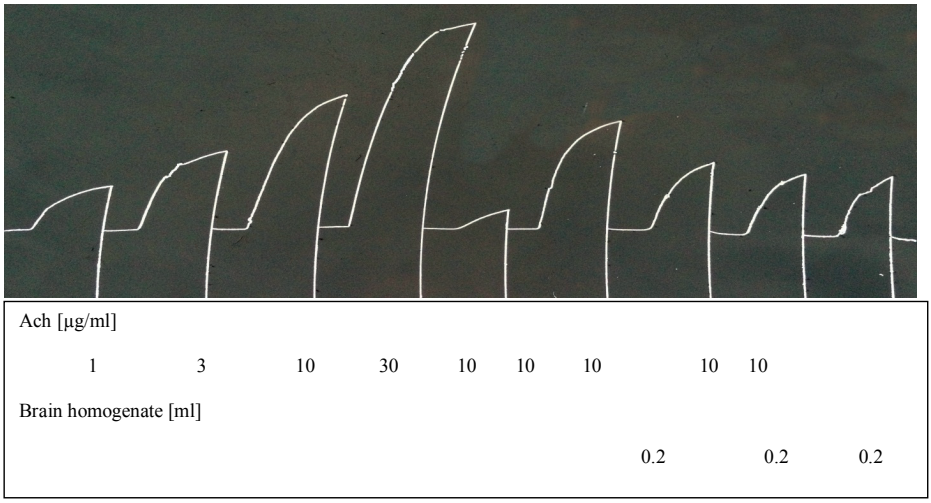

Figure 4.

NATE-treated group. Effect of brain homogenate from NATE-treated rats on acetylcholine activity using FRAM preparation

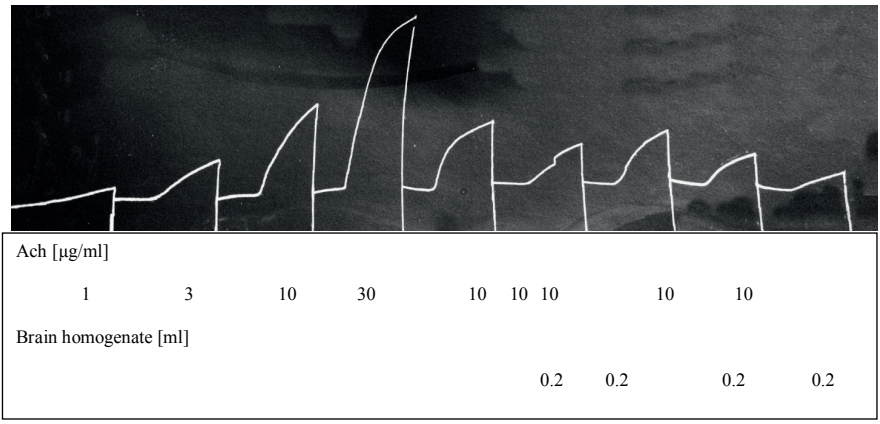

Figure 5.

NATE + standard treatment group
Effect of brain homogenate from NATE and piracetam-treated (standard drug) rats on acetylcholine activity using FRAM preparation

Table 5.

Effect of NATE on acetylcholinesterase activity

Group	nmoles/min/mg protein
Control	120.08±1.8
Scopolamine	214.07±1.5*
Piracetam	117.8±2.2
NATE	169.7±2.04#
NATE + piracetam	127.9±2.3

*p<0.005, as compared with control group
# p< 0.005, as compared to the scopolamine treated group
All values are expressed as mean ±SEM.

Table 6.

Estimation of acetylcholine activity using frog rectus abdominus muscle

Group	Concentrations [μg/ml]
Control	78.62
Scopolamine	17.02*
Piracetam	68.79
NATE	57.42#
NATE + piracetam	63.36

*p<0.005, as compared to control group
# p<0.005, as compared to scopolamine-treated group

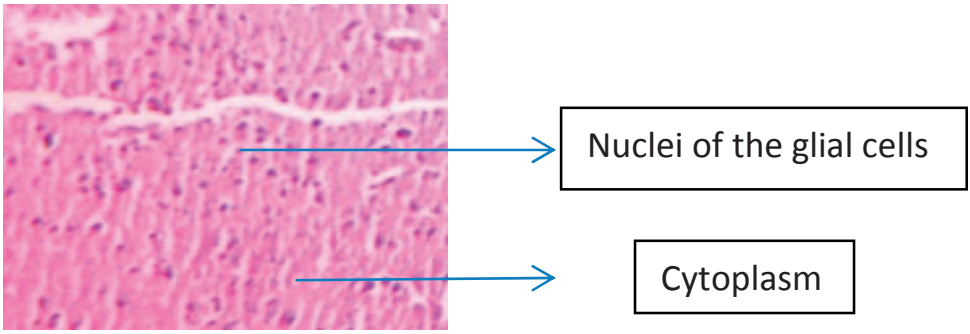

Figure 6.

Control group. The neuronal cells and the cytoplasm present are normal

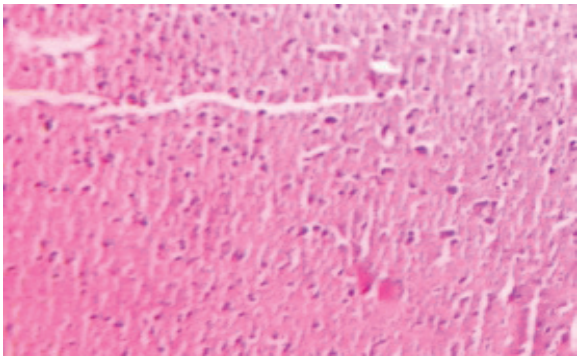

Figure 7.

Scopolamine-induced group. There were no significant changes, as compared to that of control group

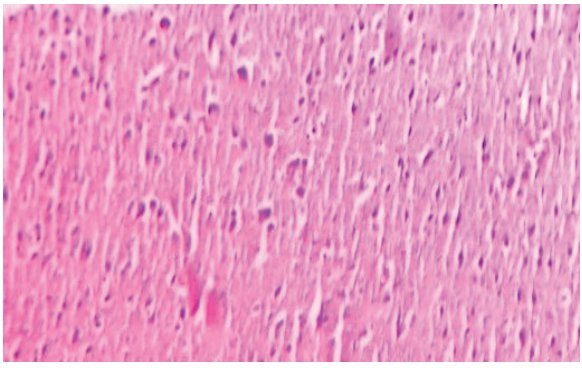

Figure 8.

Standard-treated group (piracetam). The nucleus and the cytoplasm were found to be normal as in control group

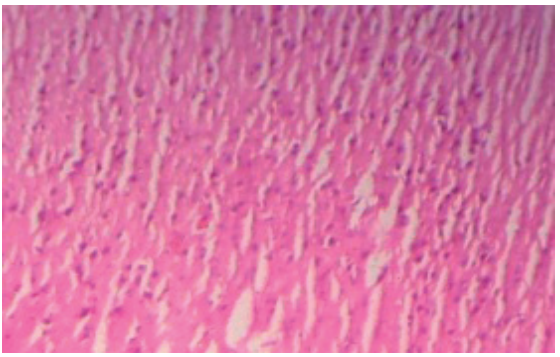

Figure 9.

NATE-treated group. There was no significant change compared to the control and scopolamine treated group

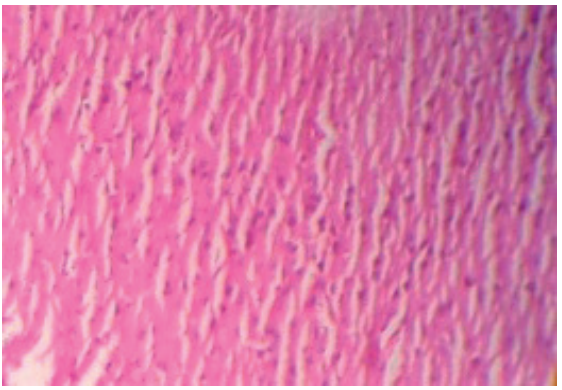

Figure 10.

NATE +standard-treatment group. No changes and cell death were found

Histopathology studies

Analysis of the brain histopathology has not shown any significant changes regarding the damage or death of the brain cells. This result may suggest, that scopolamine does not have effect in causing death to the brain cells.

There were no significant changes observed in any of the treated groups. All the groups have shown the normal condition of the brain.

DISCUSSION

Formation of memory is the most complex process and involves multiple neuronal pathways and neurotransmitters. It is well known that the cholinergic

neuronal system plays an important role in learning and memory in humans and animals [19]. Based on a cholinergic hypothesis, many attempts have been made to reverse cognitive deficits by increasing brain cholinergic activity *via* acetylcholinesterase (AChE) inhibitors.

Nyctanthes arbor tristis (NATE) is an ornamental shrub growing wild in Eastern Asia and some parts of Europe. Recent literature concerning this plant suggests the presence of mannitol, volatile oils, lupeol, ascorbic acid, nyctanthic acid, tannic acid, phenols and flavonoids. Richness in phenolics and flavonoids along with its proven effects in scavenging free radical, was proven in a study conducted by Jitesh et al., 2007 [21]. Its effect against acetylcholinesterase was proved by Verma et al., 2001 [22] it is expected to show positive results in improving memory.

Scopolamine, acetylcholine receptor antagonist, was reported to impair cognitive performances, especially spatial learning and memory. It exerts amnesic effect equally in various behavioral models of memory, including Morris water maze, radial arm maze [20] and produced the same result in our study. Therefore, scopolamine was considered as reliable tool to study neuroprotective effects of plant extract.

In present study, NATE has minimized the time taken to find hidden platform in Morris water maze test. NATE also minimized the number of errors in radial arm maze test and also increased number of avoidances in active avoidance paradigm. This shows the protective effect of NATE against scopolamine-induced cognitive impairment.

Other findings of this study were that NATE improved cognition, decreased malondialdehyde (MDA), and increased activities of catalase and antioxidant activity. A study conducted by Rama KS et al., 2010 has also shown similar results. Bioassay was conducted to estimate the levels of acetylcholine activity in the brain homogenates [18]. This assay proves that NATE has ability to block acetylcholinesterase and shown increase in Acetylcholine levels. This might suggest that NATE works through cholinergic pathway in prevention of cognitive impairment. The standard drug (Piracetam) has shown higher effect in bringing back all the parameters to control group levels. The combination of the standard drug and the NATE has not shown any significant effect. In fact the standard drug's activity was reduced when used in combination. The above facts must be thoroughly studies to uncover the possible mechanism of action of the *Nyctanthes arbor tristis* (NATE).

CONCLUSION

Nyctanthes arbor tristis (NATE) was evaluated for its protective effect against scopolamine-induced (1 mg/kg i.p.) cognitive impairments in rats. Results have proved the beneficial effects of the plant extract. Behavioral models employed shown the efficient memory in rats after treating with NATE extract. Acetylcholinesterase levels have been decreased and the levels of acetylcholine were restored in case of the plant treatment group compared to disease control group. MDA levels were also reduced which is an indicative of protective effect of the plant extract. Percentage

scavenging of peroxide and superoxide dismutase ions was increased, as compared to control group. This study shows that NATE is an acetylcholinesterase inhibitor and also has free radical scavenging activity, which might contribute to its protective effect against scopolamine-induced cognitive impairment. Further studies are needed to ascertain mechanism of action of NATE extract.

REFERENCES

1. Hardeep KS. Experimental Psychology. 1st ed. PHI Learning Private Limited, New Delhi 2010:228.
2. Selkoe DJ. The molecular pathology of Alzheimer's disease. Neuron 1991; 6:487-498.
3. Alikatte KL, Akondi BR, Yerragunta VG, Veerareddy PR, Palle S. Antiamnesic activity of *Syzygium cumini* against scopolamine induced spatial memory impairments in rats. Brain Dev 2012; 34:844-851.
4. Champa R, Chawla S, Mangal M, Mangal AK, Kajila S, Dhawan AK. *Nyctanthes arbor-tristis* Linn (night jasmine): A sacred ornamental plant with immense medicinal potential. IJTK 2012; 11: 427-435.
5. Mahida Y, Mohan JSS. Screening of plants for their potential antibacterial activity against *Staphylococcus* and *Salmonella sp.* Nat Prod Rad 2007; 6:301-305.
6. Bhaduria RS, Bhargava S, Pancholi SS. Evaluation of analgesic and anti-inflammatory activity of *Nyctanthes arbor-tristis* Linn. Int J Pharma World Res 2011; 2(2):1-11.
7. Krishna M, Manish K, Aditi K. Evaluation of hypoglycemic and hypolipidemic activity of *Nyctanthes arbor-tristis* Linn. against streptozotocin induced diabetic rats. Am J Pharmacol Toxicol 2012; 7(1):8-11.
8. Rathore B, Paul B, Chaudhary BP, Saxena AK, Sahu AP, Gupta YK. Comparative studies of different organs of *Nyctanthes arbor-tristis* in modulation of cytokines in murine model of arithritis. Biomed Environ Sci 2007; 20:154-159.
9. Meghashri S, Gopal S. Biochemical characterization of radical scavenging polyphenols from Nyctanthes arbortristis. J Pharm Bioall Sci 2012; 4:341-344.
10. Vishwanatham M, Juvekar AR. Hepatogenerative effects of *Nyctanthes arbor-tristis* Linn. on acetaminophen induced oxidative damage in rats. Int J PharmTech Res 2010; 2:1291-1297.
11. Das S, Sasmal D, Basu SP, Evaluation of CNS depressant activity of different parts of *Nyctanthes arbortristis* linn. Indian J Pharm Sci 2008; 70:803-806.
12. Tripathi S, Tripathi KP. Antistress activity of *Nyctanthes arbor tristis* fruits in rats. Mol Clin Pharmacol 2013; 4(1):53-58.
13. Pattanayak CH, Datta PP. Analgesic activity of *Nyctanthes arbor tristis* leaves in rodents. J Intercult Ethnopharmacol 2013; 2(2):105-112.
14. Okhawa H, Ohishi N, Yagi K. Assay for lipid peroxides in animal tissues by thiobarbituric acid reaction. Analytical Biochem 1979; 95:351-358.
15. Arutla S, Arra GS, Prabhakar CM, Krishna DR. Pro- and Anti-Oxidant Effects of Some Anti-leprotic Drugs in Vitro and Their influence On Super Oxide Dismutase Activity. Arzneim. Forsch Drug Res 1998; 48:1024-1027.
16. Luck H and Bergmeyer HU. Catalase in Methods of Enzymatic Analysis. Ed., Academic Press, New York 1971:885-893.
17. Ellman GL. Tissue sulfhydryl groups. Arch Biochem Biophys 1959; 82:70-77.
18. Manasa S, Sharada V, Raju AB. Assay of screening of Acetylcholinesterase Inhibitors. Int J Chem Sci 2013; 11(1):197-200.
19. Blokland A. Acetylcholine: A neurotransmitter for learning and memory? Brain Res Rev 1996; 21: 285-300.
20. Kumar A, Dogra S, Prakash A. Neuroprotective effects of *Centella asiatica* against intracerebroventricular colchicine induced cognitive impairment and oxidative stress. Int J Alzheimers Dis 2009:1-8.
21. Rathee JS, Shyam AL, Chattopadhyay S. Antioxidant activity of *Nyctanthes arbor tristis* leaf extract , Food Chem 2007:1350-1357.
22. Verma N, Kaur J, Bhatia A. Stimulation of Acetylcholinesterase activity with *Nyctanthes arbor- tristis* leaves extract in the malathion treated immunosuppressed mice. Intern J Environ Studies 2001; 58:645-654.

WPŁYW WYCIĄGU Z LIŚCI *NYCTANTHES ARBOR TRISTIS* NA ZABURZENIA POZNAWCZE INDUKOWANE SKOPOLAMINĄ U SZCZURÓW

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S t r e s z c z e n i e

Badano działanie protekcyjne wyciągu etanolowego z *Nyctanthes arbor tristis* (NATE) (150 mg/kg, podawany dożołądkowo) skierowane przeciwko zaburzeniom poznawczym wywołanym stosowaniem skopolaminy (1 mg/kg i.p.) u szczurów, stosując odpowiednie modele behawioralne takie jak: test labiryntu ramion promienistych (radial arm maze test), test labiryntu wodnego Morrisa i test aktywnego unikania. Wynik NATE został porównany z działaniem piracetamu (200 mg/kg i.p.). W teście labiryntu ramion promienistych NATE ($p<0.005$) istotnie hamował zaburzenia wywołane podaniem skopolaminy. Dodatkowo NATE także skrócił czas potrzebny do wykonania zadań w teście Morrisa i zwiększył sprawność wykonywania zadań w teście aktywnego unikania. Aktywność acetylcholinesterazy i stężenia kwasu tiobarbiturowego zmniejszyły się istotnie ($p<0.005$) równolegle ze zwiększeniem działania dysmutazy ponadtlenkowej i katalazy. To może sugerować, że NATE ma wykazywać efekt ochronny przeciwko zaburzeniom procesów poznawczych u szczurów wywołanym przez skopolaminę zarówno poprzez receptory muskarynowe, jak i poprzez działanie antyoksydacyjne. Badanie histopatologiczne mózgowia zwierząt nie wykazało istotnych zmian.

Słowa kluczowe: *Nyctanthes arbor tristis, active avoidance test, Morris water maze test, radial arm maze test, piracetam*