RESEARCH PAPER

Evaluation of Nootropic Activity of Gardenia jasminoides on Experimental Rats

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ABSTRACT

The present study was designed to evaluate the nootropic and neuroprotective potential of the ethanolic extract of Gardenia jasminoides leaves (EEGJ) against scopolamine-induced cognitive impairment in experimental rats. Acute oral toxicity studies performed according to OECD guideline 423 demonstrated the safety of the extract up to 2000 mg/kg body weight. Memory-enhancing activity was assessed using Elevated Plus Maze (EPM) and Y-maze behavioral models. Scopolamine administration significantly impaired learning and memory functions, as evidenced by increased transfer latency and reduced spontaneous alternation behavior. Pre-treatment with EEGJ at doses of 200 mg/kg and 400 mg/kg significantly improved cognitive performance in a dose-dependent manner, with the higher dose showing effects comparable to the standard drug piracetam. Biochemical studies revealed that EEGJ significantly reduced brain acetylcholinesterase (AChE) activity and malondialdehyde (MDA) levels, indicating enhancement of cholinergic transmission and reduction of oxidative stress. Histopathological examination of the hippocampus demonstrated that EEGJ protected neuronal architecture against scopolamine-induced neurodegeneration and inflammatory changes. The findings suggest that Gardenia jasminoides possesses significant nootropic, antioxidant, and neuroprotective activities, possibly due to the presence of bioactive phytoconstituents such as flavonoids and phenolic compounds.

Keywords: *Gardenia jasminoides, Nootropic activity, Scopolamine-induced amnesia, Neuroprotection, Antioxidant activity*

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INTRODUCTION

Memory impairment and cognitive dysfunction are major characteristics of neurodegenerative disorders such as Alzheimer's disease, dementia, and age-related neurological decline. These disorders are often associated with oxidative stress, cholinergic dysfunction, neuronal degeneration, and inflammation within the brain **[1]**. Current synthetic nootropic drugs used for the management of cognitive disorders may produce several adverse effects, which has increased interest in the search for safer and effective natural alternatives derived from medicinal plants **[2]**.

Medicinal plants have been recognized as important sources of neuroprotective and cognition-enhancing agents because of their rich phytochemical composition. *Gardenia jasminoides* Ellis, a member of the Rubiaceae family, has long been used in traditional medicine for the treatment of various inflammatory, hepatic, neurological, and oxidative stress-related disorders. The plant contains several biologically active constituents such as flavonoids, iridoid glycosides, phenolic compounds, terpenoids, and alkaloids that may contribute to its therapeutic effects **[3]**.

Oxidative stress and acetylcholine depletion are considered major contributing factors in memory impairment and neurodegeneration **[4]**. Therefore, compounds possessing antioxidant and acetylcholinesterase inhibitory activities may help in improving cognitive function and protecting neuronal cells from damage. Based on these considerations, the present study was undertaken to investigate the nootropic activity of ethanolic extract of *Gardenia jasminoides* leaves using scopolamine-induced amnesia models in experimental rats **[5]**. Behavioral, biochemical, and histopathological evaluations were performed to assess the cognitive-enhancing and neuroprotective potential of the plant extract.

MATERIALS AND METHODS

Identification, collection and authentication of plant material

The leaves of the plant *Gardenia jasminoides* were collected in March 2022 from the Lodhipur region of Uttar Pradesh, District- Moradabad. The plant material was washed and air-dried. Authentication was done by the Scientist in Charge, CSIR – National Institute of Science Communication and Policy Research, New Delhi, India. A

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voucher specimen (Authentication NO.-NIScPR/RHMD/Consult/2021/3971-72-2) was submitted to the department. The authentication letter were shown in Annex no. 1.

Acute Oral Toxicity Study

According to Organization for Economic Cooperation Development (OECD) guideline 423, Ethanolic Extract *Gardenia jasminoides* Leaves at a dose level of 5, 50, 300 and 2000 mg/kg [orally (p.o.)] was used for acute oral toxicity study. EEGJ administered at a dose of 2000 mg/kg body weight did not produce any behavioural abnormalities in the animals. As all tested animals survived, the oral LD_{50} of EEGJ in Wistar rats was found to be 200 mg/kg body weight **[6]**.

Selection of doses

For the assessment of memory enhancing activity by animal models, two dose levels of EEGJ were chosen in such a way that low dose was approximately one-tenth of the maximum dose during acute toxicity studies, and a high dose, which was twice that of one-tenth dose (200 mg/kg and 400 mg/kg).

Memory-Enhancing Activity

Experimental Design

A total of 30 Wistar rats were classified into 5 groups ($n = 6$). All groups except for vehicle control (Group I) were treated with Scopolamine hydrobromide (0.4 mg/kg) intraperitoneally on the eighth day of extract/standard drug treatment to induce amnesia. Transfer latency was recorded after 45 minutes, and the memory of experienced things was evaluated after 1 day in every classification:

I. Group I: vehicle control

II. Group II: Scopolamine control

III. Group III: *Gardenia jasminoides* leaves test sample (200 mg/kg)

IV. Group IV: *Gardenia jasminoides* leaves test sample (400 mg/kg)

V. Group V: Piracetam (100 mg/kg)

Elevated Plus-Maze Method

Elevated plus-maze and Scopolamine-induced amnesia were used for the exteroceptive and interoceptive behavior design, respectively, to monitor the cognitive power and retention of the memory, according to a previously established method with slight modification **[7]**.

The elevated plus-maze having 2 open arms (16 × 5 centimeters), 2 closed arms (16 × 5 × 12 centimeters) raised vertically with the 25-centimeter height was applied. Individual experimental animals were kept at the edge of the open arm facing far from the middle rostrum; then, the total duration for moving from the edge of the open arm to both closed arms using all legs (Transfer Latency, TL) was noted. Those animals that failed to reach inside any closed arm in 90 sec were kindly propelled toward closed arms, and TL was considered to be 90 s. The mice were permitted to search the maze for the next 10 s and afterward come back to the cage. Memory of this experienced work was investigated 1 day after the initial examination day **[8]**.

Transfer latency after 1 day was interpreted in terms of Inflection Ratio (IR), by applying the following equation:

$$IR = (L1 - L0) / L0,$$

Where L_0 is the transfer latency after 1 day, and L_1 is the first transfer latency expressed as seconds.

Y-Maze test

The Y-maze task measures spatial working memory through spontaneous alteration behaviour in rodents. The Y-maze used in this study consisted of three symmetrical arms (33 cm length × 11 cm width × 12 cm height), separated at 120°. For the test, each mouse was individually placed in the triangular centre of the maze and allowed to freely explore it for 8 min **[9]**. The total entry number (arms visits) was used as a measure of locomotor activity, and the degree of spontaneous alternation was estimated by the following calculation:

Percentage of alternation = (Number of alternance)/ (Total number of arms visited-2) X 100.

After the passage of each mouse, the maze was wiped with 70% ethanol to minimize odour clues.

Histopathological Examination

Histopathological examination was carried out to evaluate the microscopic architecture and cellular alterations in the tissue samples. The collected tissues were fixed in 10% neutral buffered formalin, processed through graded alcohol, embedded in paraffin wax, and sectioned into thin slices of approximately 4–5 µm thickness. The sections were stained with hematoxylin and eosin (H&E) and examined under a light microscope (100x) for pathological changes such as inflammation, necrosis, cellular degeneration, edema, hemorrhage, and structural abnormalities. The histological observations were compared between control and treated groups to assess the extent of tissue damage and the protective or therapeutic effects of the treatment **[10]**.

Biochemical Estimation

Collection of Brain Sample

Immediately after behavioural testing (retrieval) on elevated plus mazes, animals were sacrificed by cervical dislocation under light anaesthesia with diethyl ether. The whole brain was carefully removed from the skull. For the preparation of brain homogenate, the fresh whole brain was weighed and transferred to a glass homogenizer and homogenized in an ice bath after adding 10 volumes of phosphate buffer (pH 8, 0.1 M). The homogenate was centrifuged using a refrigerated centrifuge at 3000 rpm for 10 min at 4◦C, and the resultant cloudy supernatant liquid was used for the estimation of brain acetylcholinesterase activity **[11]**.

Brain Acetylcholinesterase Activity

Brain acetylcholinesterase was estimated using the method of Ellman et al., **[12]**. Briefly, 0.4mL of brain homogenate was added to a test tube containing 2.6mL of phosphate buffer. 0.1mL DTNB reagent was added to the above mixture, and absorbance was noted at 412 nm. 0.02mL of acetylcholine iodide solution was added, and again, absorbance was noted 15 min thereafter. Change in absorbance per min was calculated **[13]**.

The rate of hydrolysis of substrate was calculated using the following formula:

R = change in absorbance/min×5.74 × 10−4/C0,

Where R = rate of hydrolysis of acetylcholine iodide/min/mg tissue,

C0 =weight of tissue homogenate in mg/mL.

Malondialdehyde (MDA) Level

The extent of lipid peroxidation in brain tissue was evaluated by measuring malondialdehyde (MDA) levels using the method described by Ohkawa et al., which is widely employed to assess oxidative damage to cellular membranes. This method is based on the reaction of MDA with thiobarbituric acid (TBA) under acidic and high-temperature conditions to form a colored complex. To 0.5 mL of brain supernatant, 1.0 mL of 10% trichloroacetic acid (TCA) was added, and the mixture was centrifuged for 10 minutes. The collected supernatant (0.2 mL) was mixed with 0.1 mL of 8% TBA and heated in a water bath at 80 °C for 40 minutes. After cooling, absorbance was recorded at 532 nm, and MDA levels were expressed as nM/mg protein **[14]**.

4.10 Statistical Analysis

All the data were presented in terms of average ± SD. The mean differences between the different classes were calculated with the help of one-way ANOVA (MS-Excel 2019) and considered significant if $p < 0.05$.

RESULT

Effect of EEGJ on Transfer Latency (TL) and Inflection Ratio (IR)

Scopolamine treatment significantly impaired learning and memory performance by significantly increasing TL in EPM compared with the vehicle control group. Pre-treatment with the EEGJ leaf extract significantly improved the cognitive abilities of animals in a dose-dependent manner. The 200 mg/kg group demonstrated a mild decline in TL over both teaching and retention days. The effect of a 400 mg/kg dose was also a more effective value compared to the standard Piracetam (100 mg/ kg). The animals that received a higher dose of extract also showed a significant decrease in the inflection ratio, suggesting improved learning and memory. The present findings indicate that *Gardenia jasminoides* has an effective attenuation of scopolamine-induced amnesia, with 400 mg/kg exhibiting the most potent nootropic activity.

Table 5.16: Effect of EEGJ on Transfer Latency (TL) and Inflection Ratio (IR)

Group	Treatment	TL (Acquisition, L1) *(sec)*	TL (Retention, L0) *(sec)*	Inflection Ratio (IR)
Group I	Vehicle Control	32.5 ± 2.1	28.4 ± 1.8	0.13 ± 0.01
Group II	Scopolamine Control	78.6 ± 3.5	82.3 ± 4.1	0.04 ± 0.01
Group III	*G. jasminoides* Extract (200 mg/kg)	64.2 ± 3.0*	50.6 ± 2.7*	0.21 ± 0.02
Group IV	*G. jasminoides* Extract (400 mg/kg)	48.3 ± 2.4**	34.8 ± 2.1**	0.28 ± 0.02
Group V	Piracetam (100 mg/kg)	42.1 ± 2.0**	29.6 ± 1.9**	0.30 ± 0.02

5.10.2 Effect of EEGJ on Spontaneous Alternation Behaviour in Y-Maze Test

In the Y-maze test, scopolamine treatment significantly reduced spontaneous alternation behaviour, demonstrating impaired spatial working memory with respect to the vehicle-treated group. Scopolamine control mice showed lower alternation percentage, but no significant difference in the number of total arm entries, indicating cognitive impairment and decreased explorative behavior. The *Gardenia jasminoides* leaf extract pre-treatment dose-dependently enhanced memory function. At the same time, a mild but significant increase in alternation behaviour was obtained among mice receiving a 200 mg/kg dose of EMO, while a substantial enhancement effect (approaching the level of the Piracetam-treated group) was observed in the group supplemented with 400 mg/kg concentration. Total number of arm entries was not different between the extract-treated and Piracetam groups, indicating that higher alternation percentage is the result of improved working memory and not driven by increased locomotor activity. Taken together, the data indicate that the ethanolic extract of *Gardenia jasminoides* Ep inhibits scopolamine-induced spatial working memory impairment and facilitates the 400 mg/kg group, showing an enhanced effect on cognition.

Table 5.17: Effect of EEGJ on Spontaneous Alternation Behaviour in Y-Maze Test

Group	Treatment	Total Arm Entries (Mean ± SEM)	Spontaneous Alternation (%) (Mean ± SEM)
Group I	Vehicle Control	18.6 ± 0.9	72.4 ± 2.8
Group II	Scopolamine Control	14.2 ± 0.7	41.3 ± 2.5
Group III	*G. jasminoides* 200 mg/kg	16.8 ± 0.8	55.6 ± 2.7
Group IV	*G. jasminoides* 400 mg/kg	17.9 ± 0.8	63.8 ± 2.6

Group V	Piracetam 100 mg/kg	19.1 ± 0.9	68.2 ± 2.4

5.10.5.1 Brain Acetylcholinesterase (AChE) Activity

The enzymatic activity of AChE obtained from the brain homogenate was removed immediately after the retrieval test. Significant ($p < 0.01$) increase in AChE activity, indicative of cholinergic dysfunction and memory dysfunction, was observed in the disease/negative control group than in the normal control group. The animals treated with the standard drug showed a significant decrease in AChE activities ($p < 0.01$), resulting in enhancement of cholinergic transmission.

By dose response, EEGJ decreased the AChE activity in a concentration-dependent manner. Both the high dose treatment yielded significantly ($p < 0.05$) lower values than the disease control group, indicating enhancement of cholinergic activity and/or neuroprotection-like effect. These biochemical results are further supported by behavioral observations in the Morris Water Maze, demonstrating learning and memory enhancement as a result of AChE activity attenuation.

Table 5.21: Brain AChE Activity

Group	AChE Activity (µM/min/mg protein)
Normal Control	4.82 ± 0.24
Disease Control	8.65 ± 0.37
Standard Drug	5.12 ± 0.28**
Test Drug – Low Dose	6.94 ± 0.31*
Test Drug – High Dose	5.88 ± 0.27**

5.10.5.2 Brain Malondialdehyde (MDA) Level

The level of malondialdehyde (MDA), a key marker of lipid peroxidation and oxidative stress, was significantly ($p < 0.01$) elevated in the disease/negative control group compared to the normal control group, indicating enhanced oxidative damage in brain tissue. Treatment with the standard drug significantly ($p < 0.01$) reduced MDA levels, suggesting effective attenuation of lipid peroxidation and restoration of antioxidant balance.

Administration of EEGJ produced a dose-dependent reduction in brain MDA levels. Both low- and high-dose treatment groups showed significantly ($p < 0.05$ and $p < 0.01$, respectively) lower MDA levels compared to the disease control group. The high-dose treatment exhibited a more pronounced protective effect, closely approaching normal control values. These findings indicate the antioxidant and neuroprotective potential of EEGJ, which is further supported by improved cognitive performance observed in behavioral studies such as the Morris Water Maze.

Table 5.22: Brain Malondialdehyde (MDA) Level

Group	Treatment	MDA Level (nM/mg protein)
Group I	Vehicle Control	2.15 ± 0.18
Group II	Scopolamine Control	4.98 ± 0.32
Group III	*G. jasminoides* 200 mg/kg	2.46 ± 0.21**
Group IV	*G. jasminoides* 400 mg/kg	3.68 ± 0.27*
Group V	Piracetam 100 mg/kg	2.89 ± 0.24**

Histopathological Evaluation

Microscopic observations of the hippocampal region revealed significant differences between control, scopolamine-treated, and treatment groups. These results provide essential morphological evidence of the neuroprotective effects of EEGJ extracts against scopolamine-induced neuronal damage.

The hippocampal sections of the normal control group exhibited normal neuronal architecture, intact cellular boundaries, and pyramidal cells that were present homogeneously. Nissl's granules of the neurons were prominent and distinct, suggesting normal protein synthesis and neuronal health. There were no signs of eosinophilia, inflammation, or neuronal degeneration, indicating that the hippocampal structure was normal and healthy. This normal structure was used as a control to compare with the neurotoxic groups induced by scopolamine.

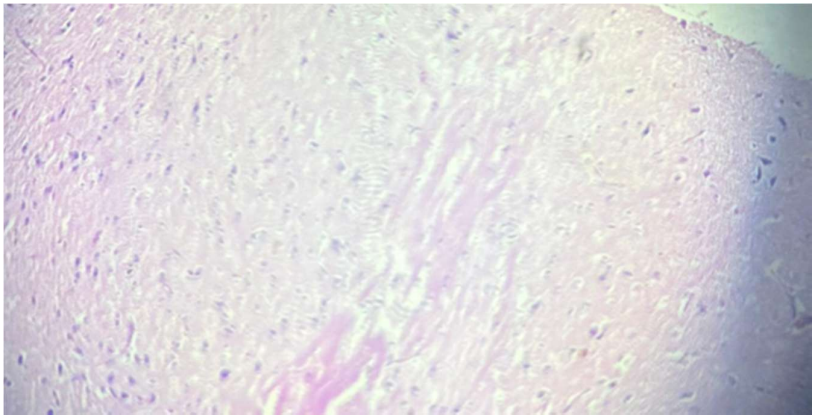

Figure 5.19: Control group microscopic section at 100x

Highly significant histopathological alterations were observed in the rats treated with scopolamine alone, which were represented by evident neuronal degeneration. Shrunken neurons with hyperchromatic nuclei were a major feature of the sections, while Nissl granules appeared disorganized or reduced, indicating compromised neuronal function. The presence of eosinophils and disorganization of the hippocampal layers also suggested inflammation and neurodegeneration. These histopathological alterations indicated successful induction of memory impairment and neuronal damage, proving SCOP as a suitable model for cognitive perturbation.

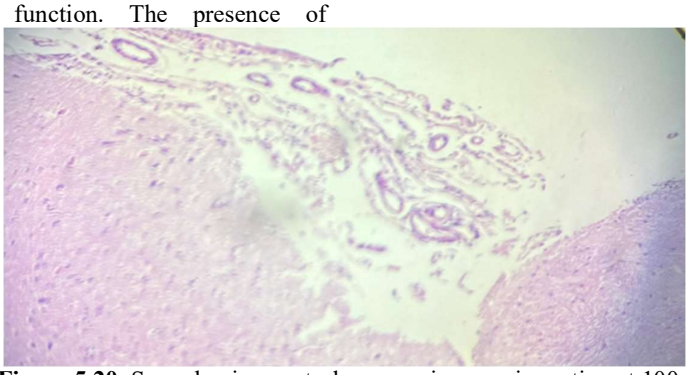

Figure 5.20: Scopolamine control group microscopic section at 100x

However, the neuronal architecture of rats treated with EEGJ at 200 mg/kg was partially recovered in the hippocampal sections when compared to that of their scopolamine-paired controls. Shrunken neurons with mildly hyperchromatic nuclei were fewer, although mild neuronal degeneration was still appreciable. a mild eosinophilia was indicative of an incomplete suppression of the inflammation, and a slight rearrangement of the neuronal architecture indicated a beginning recovery process. These results further suggest that EEGJ at low dosage was mildly neuroprotective, but not enough to completely recover.

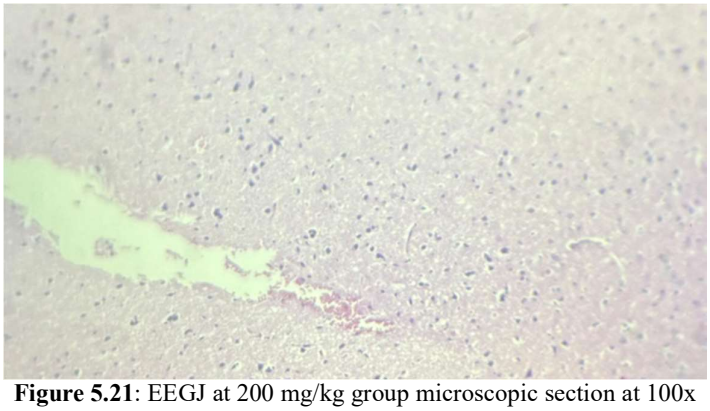

Figure 5.21: EEGJ at 200 mg/kg group microscopic section at 100x

The groups treated with higher doses of EEGJ (400 mg/kg) had a notable histopathological improvement. The majority of neurons seemed to be well-organized with an obvious morphology similar to those in the control group. The granules of Nissl were regular, indicating reestablishment of the synthesis of protein and normal

cellular metabolism. Very low numbers or no eosinophilic cells were seen, indicating potent anti-inflammatory activity. Global decrease in degenerative changes and retained neuronal structure indicated dose-dependent neuroprotective effect of EEGJ extract.

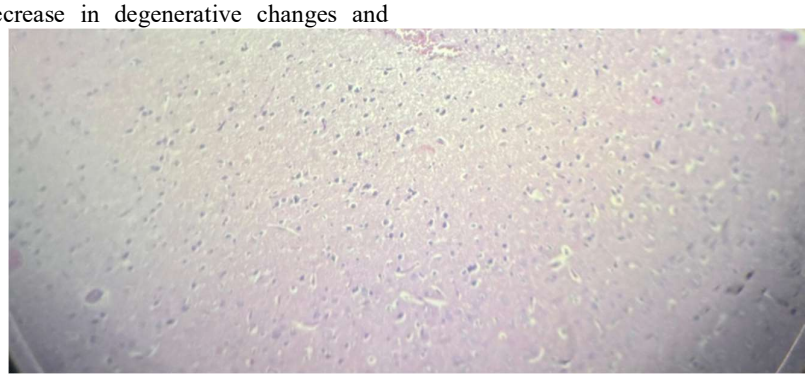

Figure 5.22: EEGJ at 400 mg/kg group, microscopic section at 100x

Piracetam (100 mg/kg), a well-known anti-Alzheimer's drug, almost completely normalized the neuron architecture of the hippocampus. Most of the neurons were well preserved, and their Nissl substance was intact. Eosinophilia was only minimal, which means inflammation had subsided considerably. This confirmed the efficiency of the scopolamine model and standardization comparative study to confirm the therapeutic potential of EEGJ extracts.

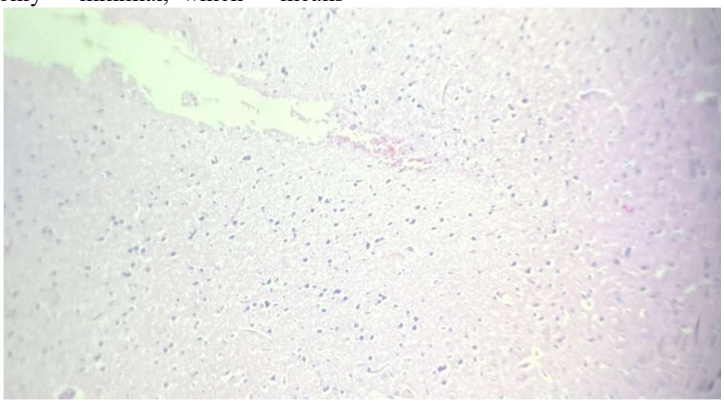

Figure 5.23: Standard piracetam (100 mg/kg) group microscopic section at 100x

The histopathological examination revealed that scopolamine treatment caused obvious nerve damage in mice, such as neuron shrinkage, hyperchromatic nuclei, moderate eosinophilia, and the disappearance of Nissl's granules. By contrast, treatments with EEGJ significantly ameliorated (especially at a dose of 400 mg/kg) the hippocampal morphology, recovered Nissl's granulation, and relieved inflammatory infiltration. The enhancements seen in these treatment groups were similar to those obtained with Donepezil, confirming potent neuroprotection and anti-inflammatory abilities. These histopathological observations are in line with the behavioral and biochemical data and strongly support that EEGJ significantly attenuates scopolamine-induced neurodegeneration in a dose-dependent manner.

CONCLUSION

The present study demonstrated that the ethanolic extract of *Gardenia jasminoides* leaves possesses significant nootropic and neuroprotective activities against scopolamine-induced cognitive impairment in experimental rats. Behavioral studies using Elevated Plus Maze and Y-maze models revealed marked improvement in learning, memory retention, and spatial working memory in extract-treated groups, particularly at the dose of 400 mg/kg. Biochemical analysis showed a significant reduction in brain acetylcholinesterase activity and malondialdehyde levels, suggesting enhancement of cholinergic neurotransmission and attenuation of oxidative stress.

Histopathological examination further confirmed the neuroprotective effect of the extract by restoring normal hippocampal neuronal architecture, reducing neuronal degeneration, and minimizing inflammatory changes. The overall findings indicate that *Gardenia jasminoides* may exert its cognitive-enhancing effects through antioxidant, anti-inflammatory, and cholinergic mechanisms. The study supports the traditional medicinal value of the plant and highlights its potential as a promising natural therapeutic agent for the management of memory disorders and neurodegenerative diseases. Further investigations involving isolation of active constituents and clinical evaluation are warranted to establish its efficacy and safety for therapeutic applications.

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