

Neuroprotective And Memory Enhancing Effects Of *Withania Somnifera* Extract In Experimental Models

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ABSTRACT:

Objective: The main goal was to assess possible memory-enhancing effects of *Withania somnifera* root extract on Wistar rats.

Materials and Methods: Extract of *Withania somnifera* roots evaluated for memory-enhancement by Morris Water Maze (MWM) and Elevated Plus Maze (EPM). Here, Parameter examined was transfer latency (TL).

Results: There was dose-dependent decline in TL when *Withania somnifera* root extract was administered, as compared to the control.

Conclusion: The observed dose-dependent reduction in transfer latency provides compelling evidence of the extract's potential in countering neurodegeneration and affirming its nootropic capabilities.

Key words: *Withania somnifera* roots, EPM, MWM, Memory, Learning

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INTRODUCTION

Neurodegenerative disorders and cognitive impairments, including Alzheimer's disease, Parkinson's disease, and age-associated memory decline, represent a significant and growing global health burden. These conditions are characterized by progressive neuronal loss, oxidative stress, mitochondrial dysfunction, neuroinflammation, and impaired neurotransmission, ultimately leading to deterioration of cognitive functions such as learning and memory¹. The increasing prevalence of these disorders, coupled with the limited efficacy and adverse effects of currently available synthetic drugs, has prompted the exploration of safer and more effective alternatives derived from natural sources².

Medicinal plants have gained considerable attention due to their multi-targeted mechanisms and favorable safety profiles. Among these, *Withania somnifera* (L.) Dunal, commonly known as Ashwagandha, is a well-known adaptogenic herb extensively used in traditional Ayurvedic medicine for its rejuvenating, anti-stress, and cognitive-enhancing properties³. The pharmacological potential of *W. somnifera* is attributed to its diverse bioactive constituents, including withanolides, sitoindosides, alkaloids, and flavonoids, which exhibit antioxidant, anti-inflammatory, and neuroprotective effects⁴.

Oxidative stress plays a crucial role in the pathogenesis of neurodegenerative disorders by promoting neuronal damage through excessive production of reactive oxygen species (ROS). *W.*

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somnifera has been reported to enhance endogenous antioxidant defense systems, such as superoxide dismutase, catalase, and glutathione, thereby protecting neurons from oxidative damage⁵. Additionally, it modulates neuroinflammatory pathways by inhibiting pro-inflammatory cytokines and reducing microglial activation, contributing to its neuroprotective potential⁶.

Furthermore, *W. somnifera* has demonstrated significant effects on cholinergic neurotransmission, which is critically involved in learning and memory processes. Studies have shown that the extract can inhibit acetylcholinesterase activity, thereby increasing acetylcholine levels in the brain and improving cognitive performance⁷. It also promotes neuronal growth, synaptic plasticity, and regeneration of damaged neurons, which are essential for memory enhancement⁸.

Experimental studies using animal models have provided strong evidence for the neuroprotective and memory-enhancing effects of *W. somnifera*. Behavioral assessments, such as the Morris water maze, elevated plus maze, and passive avoidance tests, have demonstrated improved learning and memory in treated animals⁹. Moreover, its ability to attenuate neurotoxicity induced by various agents further supports its therapeutic potential in neurodegenerative conditions¹⁰.

In this context, the present study aims to evaluate the neuroprotective and memory-enhancing effects of *Withania somnifera* extract in experimental models, with a focus on its underlying mechanisms involving antioxidant activity, modulation of neurotransmitters, and neuroregenerative potential.

MATERIALS AND METHODS

Chemicals and Reagents

Withania somnifera extract (standardized to withanolides content) was procured from a certified herbal supplier. Piracetam was used as the standard nootropic drug, and scopolamine hydrobromide was used to induce amnesia. All other chemicals and reagents were of analytical grade and obtained from standard commercial sources¹¹.

Experimental Animals

Healthy adult Wistar albino rats (150–200 g) of either sex were used. Animals were housed under standard laboratory conditions (25 ± 2°C, 55 ± 5% humidity, 12 h light/dark cycle) with free access to food and water ad libitum.

All experimental procedures were conducted in accordance with CPCSEA guidelines and approved by the Institutional Animal Ethics Committee¹².

Preparation of Extract

The dried roots of *Withania somnifera* were coarsely powdered and extracted using hydroalcoholic solvent (ethanol:water, 70:30) via Soxhlet extraction. The extract was concentrated under reduced pressure using a rotary evaporator and stored at 4°C until further use¹³.

Experimental Design

Animals were randomly divided into five groups (n = 6):

Group I (Normal Control): Normal saline (10 mL/kg, p.o.)

Group II (Negative Control): Scopolamine (1 mg/kg, i.p.)

Group III (Standard): Piracetam (200 mg/kg, p.o.) + scopolamine

Group IV (Test Low Dose): *W. somnifera* extract (100 mg/kg, p.o.) + scopolamine

Group V (Test High Dose): *W. somnifera* extract (200 mg/kg, p.o.) + scopolamine

Treatment was continued for 14 days. Scopolamine was administered 30 min after treatment on the final day to induce amnesia¹⁴.

Evaluation of Memory and Learning

1. Morris Water Maze Test

Spatial learning and memory were evaluated using the Morris water maze. Escape latency time (ELT) during training and time spent in the target quadrant during probe trial were recorded¹⁵.

2. Elevated Plus Maze (EPM)

Transfer latency (TL) was recorded as the time taken to move from an open arm to a closed arm. Initial and retention latencies were recorded on consecutive days to assess learning and memory¹⁶.

3. Passive Avoidance Test

Step-down latency (SDL) was measured as an indicator of memory retention. Increased SDL reflects improved memory performance¹⁷.

Biochemical Estimation

1. Acetylcholinesterase (AChE) Activity

AChE activity in brain homogenate was determined using Ellman's colorimetric method¹⁸.

2. Antioxidant Parameters

Superoxide dismutase (SOD), catalase (CAT), and reduced glutathione (GSH) levels were estimated using standard biochemical methods to assess antioxidant status¹⁹.

3. Lipid Peroxidation (MDA)

Malondialdehyde (MDA) levels were measured as an index of lipid peroxidation and oxidative stress²⁰.

Histopathological Studies

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Brain tissues (hippocampus region) were fixed in 10% formalin, processed, embedded in paraffin, sectioned, and stained with hematoxylin and eosin for microscopic examination of neuronal damage.

Statistical Analysis

All data were expressed as mean \pm SEM. Statistical analysis was performed using one-way ANOVA followed by Tukey's post hoc test. A value of $p < 0.05$ was considered statistically significant.

RESULTS

1. Effect of *Withania somnifera* on Learning and Memory

Table 1: Effect on Behavioral Parameters

Group	ELT (sec) ↓	TL (sec) ↓	SDL (sec) ↑
Control	45 \pm 2.1	20 \pm 1.5	120 \pm 4.2
Scopolamine	85 \pm 3.4***	65 \pm 2.8***	40 \pm 3.1***
Piracetam	40 \pm 2.0####	25 \pm 1.9####	110 \pm 3.8####
WS Low Dose	55 \pm 2.6**	35 \pm 2.2**	90 \pm 3.5**
WS High Dose	48 \pm 2.3####	28 \pm 1.7####	105 \pm 3.2####

(Values are Mean \pm SEM, $n = 6$; *** $p < 0.001$ vs Control; ** $p < 0.01$ vs Scopolamine; #### $p < 0.001$ vs Scopolamine)

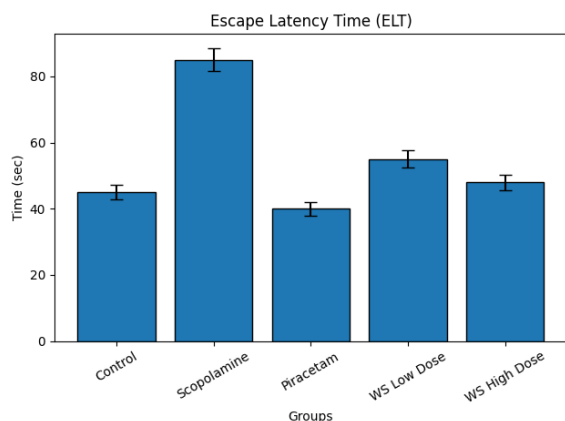


Fig 01: Effect of *Withania somnifera* extract on escape latency time in the Morris water maze.

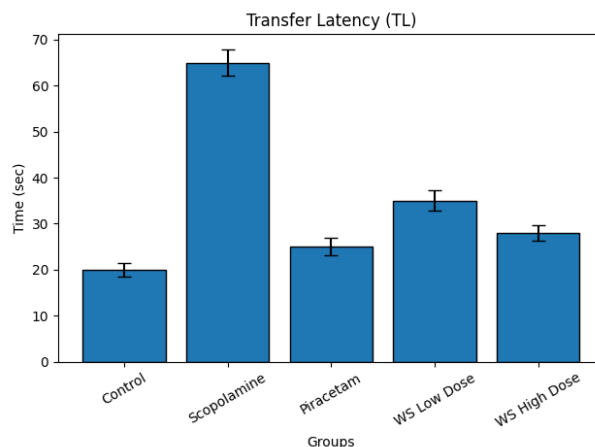


Fig 02: Effect of *Withania somnifera* extract on transfer latency using the elevated plus maze model.

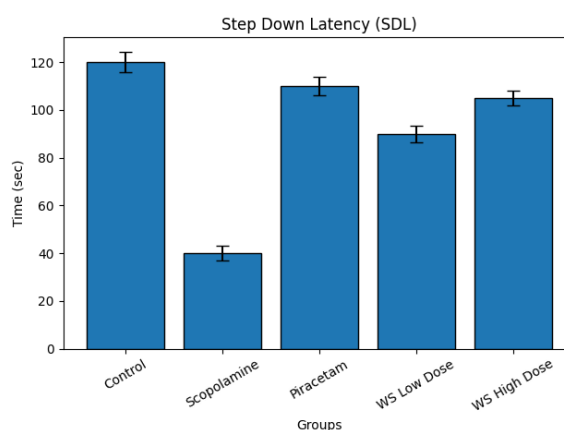


Fig 03: Effect of *Withania somnifera* extract on step-down latency in the passive avoidance test.

In the Morris water maze test, scopolamine-treated rats showed a significant increase in escape latency time (ELT) compared to the control group ($p < 0.001$), indicating impaired spatial memory. Treatment with *Withania somnifera* extract significantly reduced ELT in both dose groups, with the high dose showing results comparable to piracetam.

In the elevated plus maze, scopolamine significantly increased transfer latency (TL), whereas *W. somnifera* treatment significantly decreased TL ($p < 0.01$ and $p < 0.001$), suggesting improvement in learning and retention.

In the passive avoidance test, step-down latency (SDL) was significantly reduced in the scopolamine group, indicating memory impairment. Administration of *W. somnifera* extract significantly increased SDL in a dose-dependent manner, demonstrating enhanced memory retention.

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2. Effect of *Withania somnifera* on Biochemical Parameters

Table 2: Effect on Brain Biochemical Markers

Group	AChE ($\mu\text{mol}/\text{min}/\text{mg}$) ↓	SOD (U/mg protein) ↑	MDA (nmol/mg) ↓
Control	15 \pm 0.8	12 \pm 0.6	2.5 \pm 0.2
Scopolamine	32 \pm 1.2***	6 \pm 0.4***	6.8 \pm 0.3***
Piracetam	18 \pm 0.9###	11 \pm 0.5###	3.0 \pm 0.2###
WS Low Dose	22 \pm 1.0**	9 \pm 0.5**	4.2 \pm 0.3**
WS High Dose	19 \pm 0.8###	10 \pm 0.4###	3.5 \pm 0.2###

Biochemical analysis revealed that scopolamine administration significantly increased acetylcholinesterase (AChE) activity and malondialdehyde (MDA) levels while significantly reducing superoxide dismutase (SOD) levels ($p < 0.001$), indicating oxidative stress and cholinergic dysfunction.

Treatment with *Withania somnifera* extract significantly reversed these alterations. Both doses reduced AChE activity and MDA levels while increasing SOD levels, with the high dose showing effects comparable to the standard drug piracetam.

3. Histopathological Findings

Histopathological examination of hippocampal brain sections from the control group showed normal neuronal architecture. The scopolamine-treated group exhibited neuronal degeneration, cytoplasmic vacuolization, and disrupted hippocampal structure.

In contrast, rats treated with *Withania somnifera* extract showed marked improvement in neuronal integrity with reduced degeneration. The high-dose group demonstrated near-normal histological features, similar to the piracetam-treated group.

The findings of the present study clearly demonstrate that *Withania somnifera* possesses significant neuroprotective and memory-enhancing effects. The improvement in behavioral parameters, restoration of antioxidant defenses, and reduction in cholinergic dysfunction indicate that its mechanism of action involves modulation of oxidative stress and neurotransmitter systems.

DISCUSSION

The present study demonstrates that *Withania somnifera* extract possesses significant neuroprotective and memory-enhancing effects in

experimentally induced cognitive impairment. Scopolamine-induced amnesia is a well-established model for studying cognitive dysfunction, as it mimics cholinergic deficits observed in neurodegenerative disorders such as Alzheimer's disease²¹. In the present study, administration of scopolamine resulted in marked impairment in learning and memory, as evidenced by increased escape latency time (ELT), transfer latency (TL), and decreased step-down latency (SDL), confirming successful induction of cognitive dysfunction.

Treatment with *Withania somnifera* extract significantly ameliorated these behavioral deficits in a dose-dependent manner. The observed improvement in spatial learning and memory (Morris water maze), retention (elevated plus maze), and avoidance behavior (passive avoidance test) suggests that the extract enhances both acquisition and retrieval processes of memory. These findings are consistent with previous reports indicating the cognition-enhancing potential of *W. somnifera* in various experimental models²².

One of the key mechanisms underlying cognitive impairment is cholinergic dysfunction. Scopolamine exerts its amnesic effects by blocking muscarinic acetylcholine receptors, leading to decreased cholinergic transmission²³. In the present study, scopolamine significantly increased acetylcholinesterase (AChE) activity, which further reduces acetylcholine levels in the brain. Treatment with *Withania somnifera* significantly reduced AChE activity, indicating enhancement of cholinergic neurotransmission. This suggests that the extract may act through inhibition of AChE, thereby improving synaptic availability of acetylcholine and enhancing cognitive function²⁴.

Oxidative stress is another critical factor contributing to neurodegeneration and cognitive decline. Excessive production of reactive oxygen species (ROS) leads to lipid peroxidation, protein damage, and neuronal cell death²⁵. In this study, scopolamine administration resulted in increased malondialdehyde (MDA) levels and decreased antioxidant enzyme activity (SOD), indicating oxidative stress. *Withania somnifera* treatment significantly reduced MDA levels and restored SOD activity, demonstrating its potent antioxidant properties.

The antioxidant activity of *W. somnifera* is primarily attributed to its bioactive constituents, particularly withanolides, which are known to scavenge free radicals and enhance endogenous antioxidant defense systems²⁶. Additionally, these compounds have been

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reported to stabilize cellular membranes and prevent lipid peroxidation, thereby protecting neuronal integrity.

Neuroinflammation is also a major contributor to the progression of neurodegenerative disorders. Activated microglia release pro-inflammatory cytokines that exacerbate neuronal damage²⁷. Previous studies have shown that *W. somnifera* exhibits anti-inflammatory activity by downregulating inflammatory mediators such as TNF- α and IL-1 β , thereby contributing to its neuroprotective effects²⁸⁻⁴⁸.

Furthermore, *Withania somnifera* has been reported to promote neuroregeneration and synaptic plasticity. It enhances dendritic growth and neurite outgrowth, particularly in the hippocampus, which plays a crucial role in learning and memory⁴⁹. This neuroregenerative property may contribute to the restoration of cognitive function observed in the treated groups.

Histopathological findings in the present study further support the neuroprotective effect of *W. somnifera*. The extract attenuated neuronal degeneration and preserved hippocampal architecture, indicating protection against scopolamine-induced neurotoxicity. These findings align with previous reports demonstrating structural and functional neuroprotection by *W. somnifera*⁵⁰.

Overall, the results suggest that the neuroprotective and memory-enhancing effects of *Withania somnifera* are mediated through multiple mechanisms, including enhancement of cholinergic neurotransmission, antioxidant activity, anti-inflammatory effects, and promotion of neuronal regeneration. The multi-targeted action of this herb makes it a promising candidate for the management of cognitive disorders⁵¹⁻⁶⁵.

CONCLUSION

The present study demonstrates that *Withania somnifera* extract possesses significant neuroprotective and memory-enhancing effects in experimentally induced cognitive impairment. The extract effectively improved learning and memory, as evidenced by reduced escape latency time and transfer latency, along with increased step-down latency in behavioral models.

Biochemical findings further supported these observations, showing that *Withania somnifera* significantly reduced acetylcholinesterase activity and lipid peroxidation while enhancing endogenous antioxidant defenses. These results indicate that the extract mitigates oxidative stress and improves cholinergic neurotransmission, both of which are critical factors in cognitive function.

Histopathological analysis confirmed the protective effect of the extract on neuronal integrity, particularly in the hippocampal region, which is essential for memory processing. The overall findings suggest that *Withania somnifera* exerts its neuroprotective effects through a multi-targeted mechanism involving antioxidant, anti-cholinesterase, and neurorestorative actions.

Thus, *Withania somnifera* may serve as a promising natural therapeutic agent for the management of cognitive disorders and neurodegenerative diseases.

Future Scope

Further studies are required to isolate and characterize the active constituents responsible for the observed neuroprotective effects of *Withania somnifera*. Detailed investigations on molecular mechanisms, pharmacokinetics, and long-term safety are necessary. Additionally, well-designed clinical trials should be conducted to validate its efficacy in humans, along with the development of advanced drug delivery systems to enhance brain targeting and therapeutic outcomes.

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