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The Neuroprotective Efficacy of *Musa acuminata* Colla Flower Extract in a Scopolamine-Induced Rat Model of Alzheimer's Disease

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KEYWORDS

Keywords: Alzheimer's disease, Musa acuminata, scopolamin, acetylcholin esterase, oxidative stress, Morris Water Maze, neuroprotect ion.

ABSTRACT:

Introduction: Background: Alzheimer's disease (AD) is a progressive neurodegenerative disorder characterized by cognitive decline, cholinergic deficit, and oxidative stress. Current treatments offer limited symptomatic relief, necessitating the exploration of alternative therapies. *Musa acuminata* Colla (MAC) possesses diverse phytoconstituents with documented antioxidant and neuroprotective properties.

Objectives: This study aimed to evaluate the neuroprotective potential of the ethanolic extract of MAC flowers (MACEE) against scopolamine-induced AD in a rat model.

Methods: Thirty-six female Wistar rats were divided into six groups (n=6): Normal control, Scopolamine (1 mg/kg i.p.), Scopolamine + MACEE (low, medium, high doses), and Scopolamine + Donepezil (standard). Cognitive function was assessed using the Morris Water Maze (MWM) and Radial Arm Maze (RAM). Biochemical parameters (acetyl cholinesterase (AChE) activity, glutathione (GSH) levels) and histopathological changes in the hippocampus were evaluated.

Results: Scopolamine administration significantly impaired cognitive function, increased AChE activity, decreased GSH levels, and induced neuronal damage. MACEE treatment, particularly at medium (300 mg/kg) and high (400 mg/kg) doses, dose-dependently reversed these effects. MACEE significantly improved escape latency in the MWM and increased correct entries in the RAM, reduced AChE activity, restored GSH levels, and ameliorated hippocampal neuronal damage, with efficacy comparable to Donepezil.

Conclusions: The findings demonstrate that MACEE exerts significant neuroprotective effects against scopolamine-induced cognitive impairment, likely mediated through cholinesterase inhibition, antioxidant activity, and preservation of neuronal integrity. *Musa acuminata* Colla flower extract presents a promising multifunctional therapeutic candidate for Alzheimer's disease.

1. Introduction

Alzheimer's disease (AD) is the most prevalent form of dementia, representing a progressive neurodegenerative disorder marked by the degeneration of brain neurons (Ulm et al., 2021). It is clinically characterized by a decline in memory and at least one other cognitive domain, such as language, understanding, or judgment (Zeinab & Karaman, 2020). The global burden of AD is immense and growing, with prevalence rates estimated to rise from 27 million in 2021 to 135 million by 2050, making it a critical public health challenge (Prince et al., 2015; Javaid et al., 2021).

The pathophysiology of AD is multifactorial, involving several hallmark features. The accumulation of extracellular amyloid-beta (Aβ) plaques and intracellular neurofibrillary tangles composed of hyperphosphorylated tau protein are defining neuropathological lesions (Long & Holtzman, 2019). These pathologies are accompanied by a significant loss of cholinergic neurons in the basal forebrain, leading to a deficit in the neurotransmitter acetylcholine (ACh), which is crucial for learning and memory processes. This "cholinergic hypothesis" is a cornerstone of current pharmacological treatment strategies (Hampel et al., 2018; Craig et al., 2011). Furthermore, oxidative stress, mitochondrial dysfunction, and neuroinflammation are recognized as key contributors to disease progression, creating a vicious cycle of neuronal damage and death (Gella & Durany, 2009; Cadonic et al., 2016).

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Current FDA-approved pharmacotherapies for AD, such as acetylcholinesterase inhibitors (e.g., Donepezil, Rivastigmine) and the NMDA receptor antagonist Memantine, primarily offer symptomatic relief by modulating neurotransmitter systems but do not halt disease progression (Hampel et al., 2019; Farlow, 2004). This limitation has accelerated the search for novel, multi-target therapeutic agents, particularly from natural sources.

Musa acuminata Colla (banana) is a plant with extensive traditional medicinal uses. Various parts of the plant, including its flowers, are rich sources of bioactive phytoconstituents like flavonoids, tannins, saponins, and phenols, which are known for their potent antioxidant, anti-inflammatory, and anti-cholinesterase activities (Nishant Kumar et al., 2021; Sumathy et al., 2011). Given the multi-faceted pathology of AD, a natural extract with a complementary pharmacological profile presents a compelling therapeutic strategy.

Therefore, this study was designed to investigate the neuroprotective potential of the ethanolic extract of *Musa acuminata* Colla flowers (MACEE) in a scopolamine-induced rat model of AD, evaluating its effects on cognitive behavior, cholinergic function, oxidative stress, and hippocampal histoarchitecture.

2. Materials and Methods

Plant Material and Extraction: Flowers of *Musa acuminata* Colla were collected, authenticated (BSI Dehradun, Ref. no. Tech./Herb(Ident.)/2024-2025/812), and extracted using ethanol in a Soxhlet apparatus. The crude extract (MACEE) was stored for use.

Fig: 1. Ethanolic extract of MAC Flower

Fig: 2. Drugs used

Fig: 3) M. acuminate colla Flowers extraction

Fig: 4) M. acuminate colla Flower extract

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Phytochemical Screening: Qualitative analysis confirmed the presence of alkaloids, flavonoids, tannins, saponins, terpenoids, phenols, and steroids.

Animals and Experimental Design: Thirty-six female Albino Wistar rats (150-300 g) were housed under standard conditions. The protocol was approved by the Institutional Animal Ethics Committee (Approval no.1447/PO/RE/S/11/CPCSEA Proposal Number: 103/A)

Animals were divided into six groups:

1. Group 1: Normal saline (10 ml/kg p.o.)
2. Group 2: Scopolamine (1 mg/kg i.p.)
3. Group 3: Scopolamine + MACEE (Low Dose)
4. Group 4: Scopolamine + MACEE (Medium Dose)
5. Group 5: Scopolamine + MACEE (High Dose)
6. Group 6: Scopolamine + Donepezil (5 mg/kg p.o.)

All treatments were administered for 14 days.

Behavioral Assessments:

Morris Water Maze (MWM): To evaluate spatial learning and memory (escape latency, path length).

Radial Arm Maze (RAM): To assess working memory (number of correct entries).

Biochemical Estimations:

Acetylcholinesterase (AChE) Activity: Measured in brain homogenate.

Reduced Glutathione (GSH) Level: Assessed as a marker of antioxidant defense.

Histopathological Examination: Hippocampal tissues were processed, sectioned, and stained with H&E for microscopic evaluation of neuronal damage.

Statistical Analysis: Data are expressed as Mean ± SEM. Statistical significance was determined by one-way ANOVA followed by post-hoc tests. $p<0.05$ was considered significant.

Results

Behavioral Results

MWM Test:** Scopolamine significantly increased escape latency compared to the normal control ($p<0.001$). MACEE treatment dose-dependently reduced this latency, with the high-dose group (20.2 ± 2.08 s) showing performance comparable to the Donepezil group (14.2 ± 1.07 s) (Table 9, Fig. 12).

RAM Test:** Scopolamine reduced the number of correct entries. MACEE administration improved performance significantly, with the high-dose group (14.0 ± 0.37) nearing the performance of the normal and Donepezil-treated groups (Table 10, Fig. 13).

Biochemical Results

AChE Activity: Scopolamine induced a significant rise in AChE activity. MACEE treatment, particularly at medium and high doses, markedly reduced AChE levels, indicating inhibition of the enzyme (Table 11, Fig. 14).

GSH Levels: Scopolamine depleted GSH levels, indicating oxidative stress. MACEE treatment restored GSH levels in a dose-dependent manner, with the high dose showing values similar to the normal control (Table 12, Fig. 15).

Histopathological Results

Histology of the hippocampus revealed severe neuronal damage, shrunken hyperchromatic nuclei, and edema in the scopolamine group. MACEE treatment groups showed a dose-dependent amelioration of these changes, with the high-dose group exhibiting near-normal neuronal architecture, comparable to the protective effects seen with Donepezil (Fig. 16).

Table 1:- Effect of treatment on 14-day escape delay

		MWM	**RAM**	**AChE level**	**GSH level**
S.no.	Groups	**Mean ± S.E.M**	Mean ± S.E.M	Mean ± S.E.M	Mean ± S.E.M
1	Normal Saline	8.5±0.29	16.0±0.37	125.0±3.42	18.0±0.51
2	Scopolamine	59.5± 0.22	4.83±0.31	190.0±7.07	8.0±0.36
3	Scopolamine + Extract low dose	57.5± 0.34	7.17.± 0.31	170.0±3.65	10.0±0.43

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4	Scopolamine + Extract medium dose	30.4± 2.09	9.83 ±0.31	110.0±4.56	13.0±0. 36
5	Scopolamine + Extract high dose	20.2± 2.08	14.0±0 .37	125.0±3.63	17.0±0. 51
6	Scopolamine + Standard	14.2± 1.07	15.17± 0.31	100.0±2.45	20.0±8 6

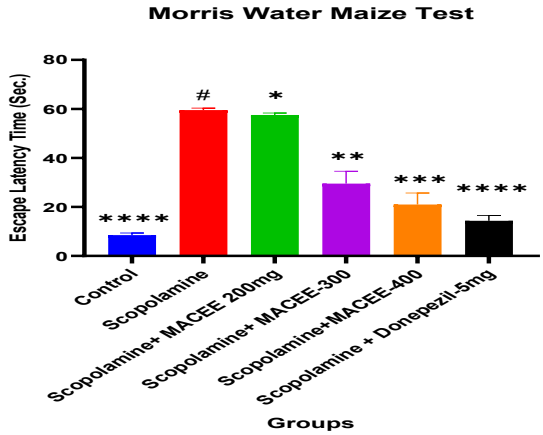

Figure: 5) The Morris Water Maze Test. The escape latency (Sec.) for each experimental group was examined. In comparison to the control group, # indicates statistical significance. The data are displayed as Mean ± SEM. Statistical significance versus the Scopolamine group is indicated by the symbols *, **, *, and ****; greater levels of significance are indicated by more asterisks.

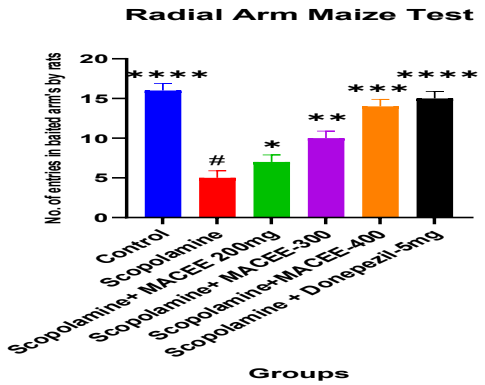

Figure: 6) The Radial Arm Maize Test was used to evaluate the effects of donepezil, MACEE, and scopamine on rats' spatial working memory. The data are shown as mean ± SEM. The study employed a one-way ANOVA to ascertain statistical significance. # p<0.0001 versus the control group; ∗p<0.05 versus the Scopolamine group; ∗∗p<0.01 versus the Scopolamine group; ∗∗∗p<0.001 versus the Scopolamine group; ∗∗∗p<0.0001 versus the Scopolamine group.

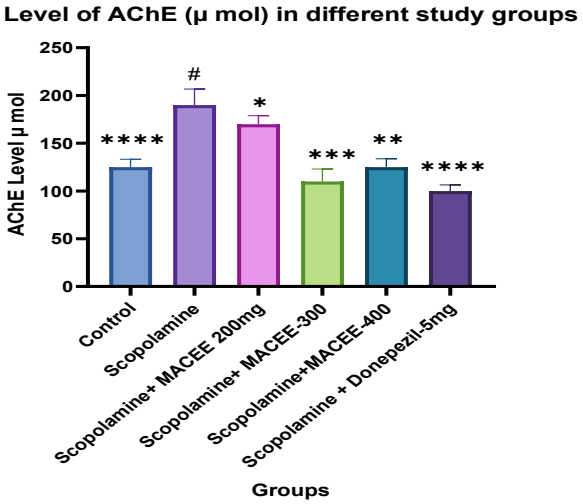

Figure: 7) Level of AChE (µ mol) in different study groups. Data are presented as mean ± SEM. One-way ANOVA was performed, followed by post-hoc tests. Asterisks indicate significant differences compared to the Scopolamine group (P<0.05, P<0.01, P<0.001, ****P<0.0001). '#' indicates a significant difference compared to the Control group (P<0.05).

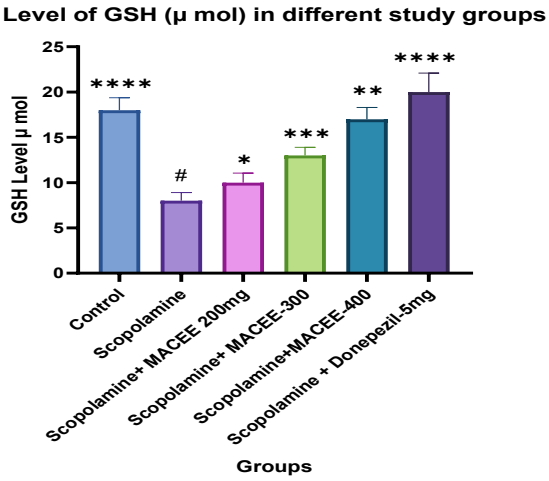

Figure: 8) Level of GSH (µ mol) in different study groups. Data are presented as mean ± SEM. One-way ANOVA was performed, followed by post-hoc tests. Asterisks indicate significant differences compared to the Scopolamine group (P<0.05, P<0.01, P<0.001, ****P<0.0001). '#' indicates a significant difference compared to the Control group (P<0.05).

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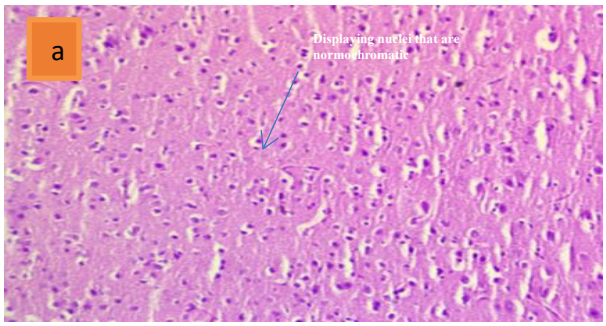
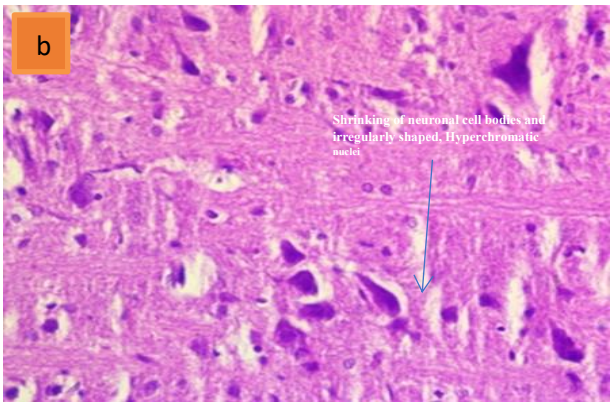
Histopathology of Hippocampus-

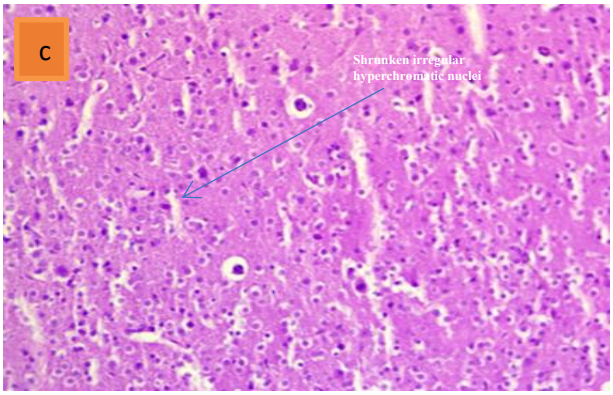
Figure: 9 (A-F) displays the histopathological alterations in the hippocampal region of the brain for each of the six groups under investigation



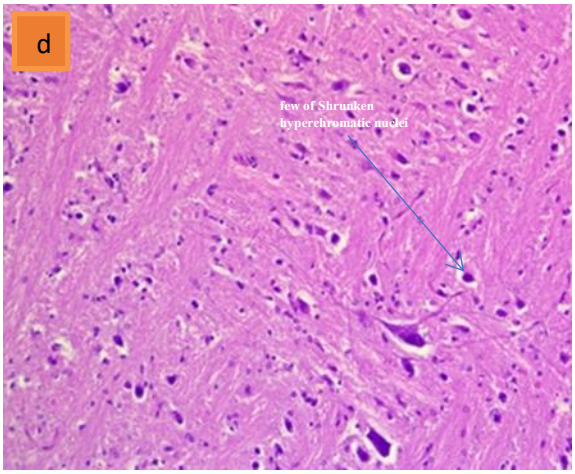
Histopathology of hippocampal- (A-F) at 200X-magnification displays histopathological alterations in the hippocampal area of the brain for each of the six groups under study. a) Normal group: displaying nuclei that are normochromatic.



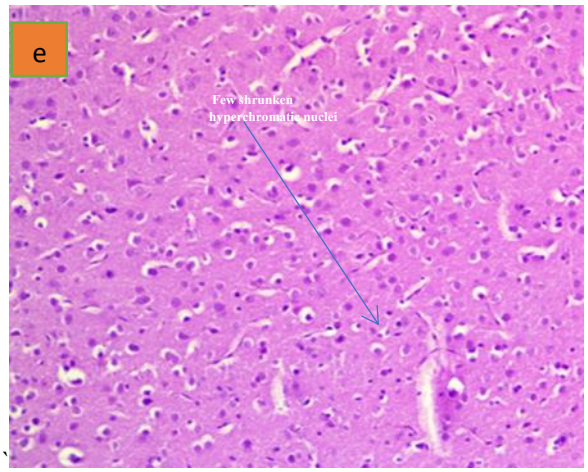
b) Scopolamine: shrinking of neuronal cell bodies and irregularly shaped, hyperchromatic nuclei



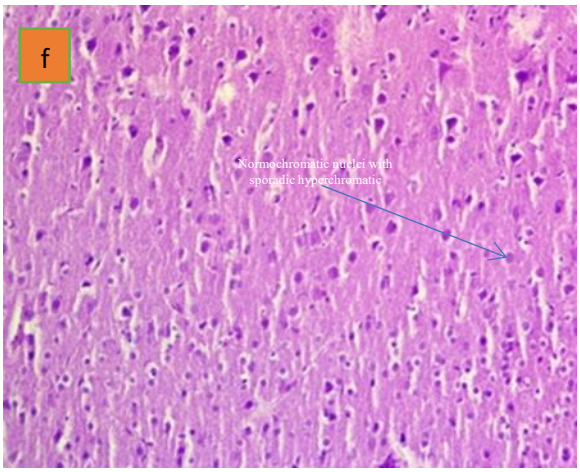
c) Shrunken irregular hyperchromatic nuclei are occasionally observed in the Scopolamine + MACEE (LD) group.



d) A few of Shrunken hyperchromatic nuclei with Scopolamoine + MACEE (MD)



e) A few shrunken hyperchromatic nuclei with Scopolamine + MACEE (HD).



f) Almost normochromatic nuclei with sporadic hyperchromatic nuclei at standard doses of donepezil.

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4. Discussion

The present study demonstrates that ethanolic extract of *Musa acuminata* Colla flowers (MACEE) confers significant protection against scopolamine-induced cognitive deficits, cholinergic dysfunction, and oxidative stress in rats.

Scopolamine, a muscarinic cholinergic receptor antagonist, is well-established for inducing reversible cognitive impairments in rodents that mimic the cholinergic deficit observed in AD (Bhuvanendran et al., 2018). Our results align with this, as scopolamine-treated rats exhibited significant memory deficits in both MWM and RAM tasks, a sharp increase in AChE activity, a decrease in brain GSH, and clear histopathological damage in the hippocampus. The efficacy of the standard drug Donepezil, an AChE inhibitor, in reversing these effects validates our model.

The profound cognitive-enhancing effects of MACEE, as evidenced by improved performance in behavioral paradigms, can be attributed to its phytochemical composition. The presence of flavonoids, tannins, and alkaloids is strongly associated with acetylcholinesterase inhibitory activity (Asaduzzaman et al., 2014). Our biochemical findings confirm this, showing that MACEE significantly reduced scopolamine-elevated AChE activity. By inhibiting AChE, MACEE likely increases synaptic availability of acetylcholine, thereby ameliorating the cholinergic hypofunction and improving memory and learning (Hampel et al., 2018).

Beyond the cholinergic system, oxidative stress is a critical player in AD pathogenesis, contributing to neuronal damage and Aβ toxicity (Gella & Durany, 2009; Praticò, 2008). The observed depletion of GSH in the scopolamine group indicates a compromised antioxidant defense system. MACEE treatment effectively restored GSH levels, highlighting its potent antioxidant capacity. This effect is undoubtedly linked to its high content of phenolic compounds and flavonoids, which are powerful free radical scavengers (Rakesh et al., 2021; Wani, 2017). By mitigating oxidative stress, MACEE helps protect neurons from damage, a effect corroborated by our histopathological findings showing preserved neuronal structure.

The histopathological analysis provides direct morphological evidence of MACEE's neuroprotective efficacy. The reduction in shrunken, hyperchromatic neurons and edema in the hippocampus of MACEE-treated animals indicates a preservation of neuronal integrity. This aligns with studies on other plants, like *Aegle marmelos*, where similar phytoconstituents have been shown to protect against neuronal damage (Raheja et al., 2019).

The multi-target effects of MACEE—simultaneously modulating the cholinergic system and oxidative stress—make it a highly promising candidate for AD therapy. This is a significant advantage over single-target synthetic drugs. The efficacy of the medium and high doses of MACEE was comparable to Donepezil across multiple parameters, suggesting its potential as an effective natural alternative or adjunct therapy.

5. Conclusion

In conclusion, the ethanolic extract of *Musa acuminata* Colla flowers (MACEE) exhibits significant neuroprotective effects in a scopolamine-induced model of Alzheimer's disease. It improves cognitive performance, inhibits acetylcholinesterase activity, enhances antioxidant defense, and preserves hippocampal neuronal morphology. The therapeutic efficacy of MACEE is attributed to its rich profile of bioactive phytoconstituents. These findings scientifically validate the traditional use of *Musa acuminata* and position MACEE as a promising, multi-target therapeutic agent worthy of further investigation for the management of neurodegenerative disorders like Alzheimer's disease.

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