

ANTI-DEMENTIA ACTIVITY OF METHANOLIC EXTRACT OF MUCUNA PRURIENS SEEDS AGAINST ATROPINE-INDUCED MEMORY IMPAIRMENT IN ALBINO RATS

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ABSTRACT

Background: Dementia is a progressive neurodegenerative syndrome affecting memory, cognition and behaviour, with approximately 57 million people affected worldwide.^[1] Alzheimer's disease, the most common form, involves cholinergic dysfunction, oxidative stress, neuroinflammation and neuronal degeneration. Current drugs provide mainly symptomatic relief and may cause adverse effects, highlighting interest in plant-derived neuroprotective agents. Mucuna pruriens seeds contain L-DOPA, flavonoids, phenolics, alkaloids and saponins with reported antioxidant, anti-inflammatory and neuroprotective effects. **Materials and Methods:** Methanolic extract of Mucuna pruriens seeds (MEMPS) was prepared by Soxhlet extraction and subjected to phytochemical screening. Anti-dementia activity was evaluated in atropine-induced memory impairment in Wistar rats (200–220 g; n=6/group). Animals received atropine (10 mg/kg, s.c.) for 7 days with donepezil (3 mg/kg, p.o.) or MEMPS (200 and 400 mg/kg, p.o.). Memory was assessed on days 1, 2, 4 and 7 using Elevated Plus Maze and Passive Avoidance tests. **Results:** MEMPS produced dose- and time-dependent improvements in transfer latency and step-through latency compared with the disease control. The 400 mg/kg dose showed effects approaching those of donepezil by day 7. Phytochemical screening revealed alkaloids, flavonoids, phenolics, tannins, saponins, glycosides, steroids and carbohydrates. **Conclusion:** MEMPS demonstrated promising anti-dementia activity against atropine-induced cognitive impairment, potentially through antioxidant and cholinergic mechanisms, warranting further mechanistic, biochemical and toxicological studies.

KEYWORDS: Mucuna pruriens; Anti-dementia activity; Atropine-induced amnesia; Elevated Plus Maze; Passive Avoidance test; Donepezil.

1.0 INTRODUCTION

Dementia is a progressive neurological syndrome characterised by decline in memory, learning, reasoning, language and the ability to perform daily activities; it is not a single disease but an umbrella term for several disorders of the brain. The World Health Organization estimates that 57 million people were living with dementia worldwide in 2021, with more than 60% residing in low- and middle-income countries and nearly 10 million new cases arising every year, making it one of the leading causes of disability and dependency among older adults.^[1] Alzheimer's disease (AD) is the most common form, accounting for 60–70% of cases, and is characterised pathologically by extracellular amyloid- β plaques and intracellular neurofibrillary tangles of hyperphosphorylated tau, together with cholinergic dysfunction, oxidative stress, mitochondrial impairment, neuroinflammation and synaptic loss.^[1,2] Among these mechanisms, degeneration of cholinergic neurons projecting from the basal forebrain and the resulting decline in synaptic acetylcholine availability is particularly implicated in the memory and attentional deficits of AD, forming the basis of the cholinergic hypothesis of dementia.^[3,4]

Current pharmacotherapy for dementia remains symptomatic: the acetylcholinesterase inhibitors donepezil, rivastigmine and galantamine, and the NMDA-receptor antagonist memantine. These agents do not halt disease progression and are frequently associated with gastrointestinal, hepatic and other adverse effects on long-term use,^[5] which has sustained interest in plant-derived agents with neuroprotective and cognition-enhancing potential.

Mucuna pruriens (L.) DC. (Fabaceae), commonly known as velvet bean, is a climbing legume used across Ayurvedic, Siddha and Unani systems of medicine.^[6] Its seeds are a particularly rich source of L-DOPA (3–7% of dry seed weight), together with flavonoids, phenolic acids, alkaloids, tannins, saponins and phytosterols.^[6,7] Beyond its established role in Parkinson's disease, where L-DOPA and associated constituents confer neuroprotective and metal-chelating activity,^[8,9,10] *Mucuna pruriens* extracts have shown antioxidant and free-radical-scavenging effects in experimental models,^[11] properties that are mechanistically relevant to the oxidative and inflammatory components of dementia pathology. Direct evidence for its activity in a cholinergic memory-impairment model, however, remains limited.^[12]

Atropine, a competitive antagonist at muscarinic acetylcholine receptors, blocks central cholinergic neurotransmission in brain regions governing learning and memory, including the hippocampus and cerebral cortex, and is a well-established pharmacological tool for producing reversible cognitive impairment to screen candidate anti-dementia agents.^[13,14] The present study used this model to evaluate the anti-dementia potential of the methanolic extract of *Mucuna pruriens* seeds (MEMPS) in Wistar rats, assessed using the Elevated Plus Maze and Passive Avoidance paradigms and benchmarked against the standard drug donepezil, alongside preliminary phytochemical characterisation of the extract.

2.0 MATERIALS AND METHODS

2.1 Plant material and authentication

Mature seeds of *Mucuna pruriens* (L.) DC. were procured from the ICAR–Indian Institute of Horticultural Research, Bengaluru, and authenticated by Dr. Sanmathi Naik A.T.S., Scientist, ICAR–Krishi Vigyan Kendra, Raichur. The seeds were cleaned and shade-dried at room temperature to constant weight, reduced to a coarse powder using a mechanical grinder, and stored in an airtight container until extraction.

2.2 Preparation of methanolic extract of *Mucuna pruriens* seeds (MEMPS)

Powdered seeds (100 g) were subjected to continuous hot extraction with methanol (≈ 300 mL; drug-to-solvent ratio $\approx 1:3$ w/v) in a Soxhlet apparatus at $50\text{--}60^\circ\text{C}$ for approximately 30 h, until the siphoning solvent ran nearly colourless. The extract was filtered, concentrated under reduced temperature ($<40^\circ\text{C}$) over a water bath and dried to obtain the crude extract (MEMPS). Percentage yield was calculated as (weight of dried extract / weight of powdered material) $\times 100$. The extract was stored under refrigeration until further use.

2.3 Preliminary phytochemical screening

MEMPS was screened qualitatively for alkaloids (Mayer's and Dragendorff's tests), flavonoids (Shinoda test, alkaline reagent test), amino acids (ninhydrin test), tannins (ferric chloride and lead acetate tests), phenolic compounds (ferric chloride test), saponins (froth test), terpenoids (Salkowski test), steroids (Liebermann–Burchard test), glycosides (Legal's test), carbohydrates (Molisch's test), reducing sugars (Fehling's test) and sterols (Liebermann's test), following standard pharmacognostic procedures.^[20]

2.4 Experimental animals

Healthy Albino rats of either sex (200–220 g) were obtained from the institutional animal house of V. L. College of Pharmacy, Raichur, and acclimatised for 7 days under standard husbandry conditions ($26 \pm 2^\circ\text{C}$; 45–55% relative humidity; 12 h light/dark cycle) with free access to standard pellet feed and water ad libitum. Animals were fasted overnight before dosing. All procedures were approved by the Institutional Animal Ethics Committee and conducted in accordance with the guidelines of the Committee for the Purpose of Control and Supervision of Experiments on Animals (CPCSEA; institutional registration no. 557/PO/Re/S/02/CPCSEA)^[16] (approval no. VLCP/Phcology/2602).

2.5 Acute oral toxicity and dose selection

Dose selection was based on previously reported acute oral toxicity data for MEMPS (no mortality at doses up to 2000–5000 mg/kg; LD₅₀ > 2000 mg/kg consistent with the acute-toxic-class framework of OECD Guideline 423^[15]). One-tenth (200 mg/kg) and one-fifth (400 mg/kg) of this LD₅₀ were selected as the low- and high-test doses, respectively.

2.6 Induction of atropine-induced memory impairment and experimental design

Animals were randomly allocated to five groups of six ($n = 6$): Group I, normal control (vehicle); Group II, disease control (atropine 10 mg/kg, s.c.); Group III, standard (donepezil 3 mg/kg, p.o., plus atropine); Group IV, MEMPS 200 mg/kg, p.o., plus atropine; and Group V, MEMPS 400 mg/kg, p.o., plus atropine. Atropine was administered subcutaneously once daily for 7 consecutive days to Groups II–V to induce cholinergic memory impairment; Group I received vehicle on the same schedule. Donepezil and MEMPS were administered orally once daily throughout the treatment period.

2.7 Elevated Plus Maze test

Learning and memory were assessed using an apparatus with two open and two enclosed arms set perpendicular around a central platform. On the acquisition trial, the time taken by each animal to move from an open arm into an enclosed arm was recorded as the initial transfer latency;^[17,18] on each subsequent retention trial the same measure was recorded as the transfer latency. Transfer latency was recorded on days 1, 2, 4 and 7. Atropine-treated animals were expected to

show prolonged transfer latency, reflecting impaired acquisition and retention, while effective treatment was expected to shorten it.

2.8 Passive Avoidance test

Learning and memory retention were additionally assessed using a two-compartment (illuminated/dark) passive-avoidance apparatus.^[19] On the acquisition trial, each animal was placed in the illuminated compartment; on entering the dark compartment it received a mild, predetermined aversive stimulus. On retention trials (days 1, 2, 4 and 7), the animal was again placed in the illuminated compartment and the step-through latency (time to re-enter the dark compartment) was recorded.

2.9 Statistical analysis

Data are expressed as mean $\pm$ standard error of the mean (SEM), $n = 6$ per group. Group differences were analysed by one-way ANOVA followed by Tukey's multiple comparison test^[21], with $P < 0.05$ considered statistically significant.

3.0 RESULTS

3.1 Preliminary phytochemical screening

Qualitative phytochemical screening of MEMPS confirmed the presence of amino acids, alkaloids, flavonoids, tannins, saponins, terpenoids, sterols, glycosides, steroids and carbohydrates; reducing sugars were not detected (Table 1).

Table 1: Preliminary phytochemical screening of MEMPS.

Phytoconstituent	MEMPS
Amino acids	Present
Alkaloids	Present
Flavonoids	Present
Tannins	Present
Saponins	Present
Terpenoids	Present
Sterols	Present
Glycosides	Present
Carbohydrates	Present
Steroids	Present
Reducing sugars	Absent

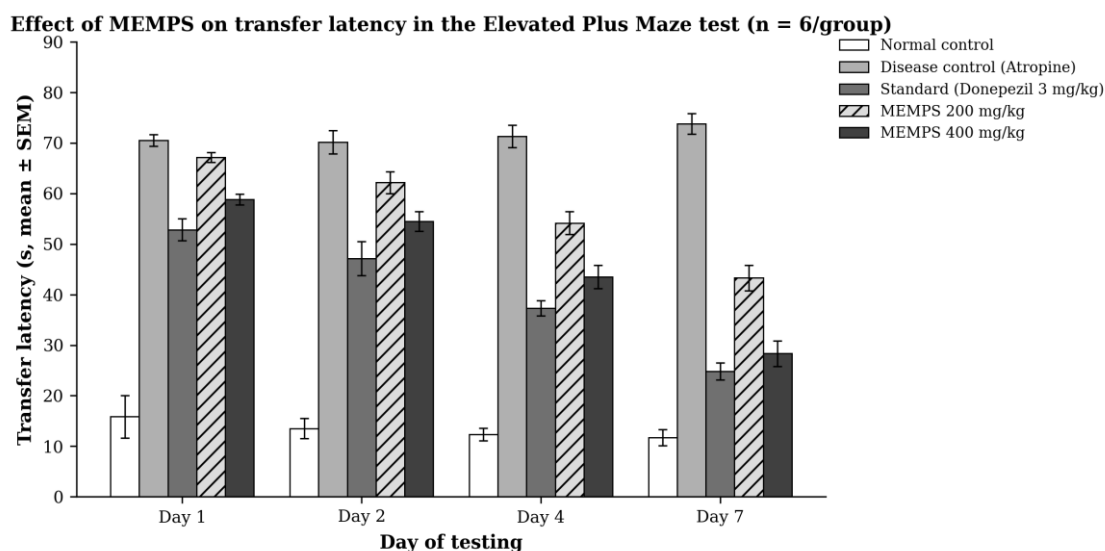
3.2 Effect of MEMPS on transfer latency in the Elevated Plus Maze test

Atropine treatment (Group II) produced a marked, sustained increase in transfer latency relative to the normal control group across all test days (day 7: 73.83 ± 2.06 s vs 11.68 ± 1.59 s), consistent with impaired acquisition and retention of learning. Both MEMPS-treated groups showed a dose-related reduction in transfer latency relative to the disease control group that became more pronounced with repeated dosing: by day 7, transfer latency had fallen to 43.33 ± 2.53 s at 200 mg/kg and to 28.34 ± 2.51 s at 400 mg/kg, compared with 24.83 ± 1.70 s in the donepezil-treated group (Table 2, Figure 1). The 400 mg/kg dose produced a consistently greater reduction in transfer latency than the 200 mg/kg dose at every time point.

Table 2: Effect of MEMPS on transfer latency (s, mean $\pm$ SEM, n = 6) in the Elevated Plus Maze test.

Day	Normal control	Disease control (Atropine)	Standard (Donepezil 3 mg/kg)	MEMPS 200 mg/kg	MEMPS 400 mg/kg
Day 1	15.83 $\pm$ 4.214****	70.50 $\pm$ 1.147	52.83 $\pm$ 2.167****	67.17 $\pm$ 0.980*	58.83 $\pm$ 1.078****
Day 2	13.50 $\pm$ 1.979****	70.17 $\pm$ 2.330	47.17 $\pm$ 3.371****	62.17 $\pm$ 2.171 ^{ns}	54.50 $\pm$ 1.945****
Day 4	12.33 $\pm$ 1.256****	71.33 $\pm$ 2.186	37.34 $\pm$ 1.498****	54.17 $\pm$ 2.272****	43.50 $\pm$ 2.320****
Day 7	11.68 $\pm$ 1.585****	73.83 $\pm$ 2.056	24.83 $\pm$ 1.701****	43.33 $\pm$ 2.525****	28.34 $\pm$ 2.512****

Values are mean $\pm$ SEM; n = 6 animals/group. Statistical comparison by one-way ANOVA with Tukey's post-hoc test [insert group-wise significance levels from the authors' own analysis].

**Figure 1: Effect of MEMPS on transfer latency in the Elevated Plus Maze test across days 1, 2, 4 and 7 (mean $\pm$ SEM, n = 6/group).**

3.3 Effect of MEMPS on step-through latency in the Passive Avoidance test

A comparable pattern was observed in the Passive Avoidance test. The disease control group showed the highest step-through latency of all groups at every time point (day 7: 36.17 $\pm$ 2.21 s), while the normal control group showed the lowest and most stable latency throughout (day 7: 13.50 $\pm$ 2.13 s). MEMPS treatment produced a dose- and time-related shift in step-through latency toward the normal-control range, from 31.17 $\pm$ 0.83 s (200 mg/kg) and 26.17 $\pm$ 0.60 s (400 mg/kg) on day 1 to 16.67 $\pm$ 1.84 s and 13.67 $\pm$ 1.12 s, respectively, by day 7; the donepezil-treated group followed a comparable trajectory (8.67 $\pm$ 1.05 s on day 7) (Table 3, Figure 2).

Table 3: Effect of MEMPS on step-through latency (s, mean $\pm$ SEM, n = 6) in the Passive Avoidance test.

Day	Normal control	Disease control (Atropine)	Standard (Donepezil 3 mg/kg)	MEMPS 200 mg/kg	MEMPS 400 mg/kg
Day 1	13.50 $\pm$ 1.979****	36.50 $\pm$ 2.156	18.83 $\pm$ 0.703****	31.17 $\pm$ 0.833*	26.17 $\pm$ 0.600****
Day 2	14.33 $\pm$ 1.850****	35.17 $\pm$ 1.740	16.17 $\pm$ 1.046****	28.17 $\pm$ 1.249*	24.67 $\pm$ 1.476****
Day 4	13.50 $\pm$ 2.363****	36.17 $\pm$ 1.778	13.34 $\pm$ 1.054****	23.67 $\pm$ 1.453***	18.34 $\pm$ 1.647****
Day 7	13.50 $\pm$ 2.125****	36.17 $\pm$ 2.212	8.67 $\pm$ 1.054****	16.67 $\pm$ 1.838****	13.67 $\pm$ 1.116****

Values are mean $\pm$ SEM; n = 6 animals/group. Statistical comparison by one-way ANOVA with Tukey's post-hoc test [insert group-wise significance levels from the authors' own analysis]. The direction of change for this parameter should be re-confirmed against the exact apparatus and protocol used (see accompanying notes to the authors).

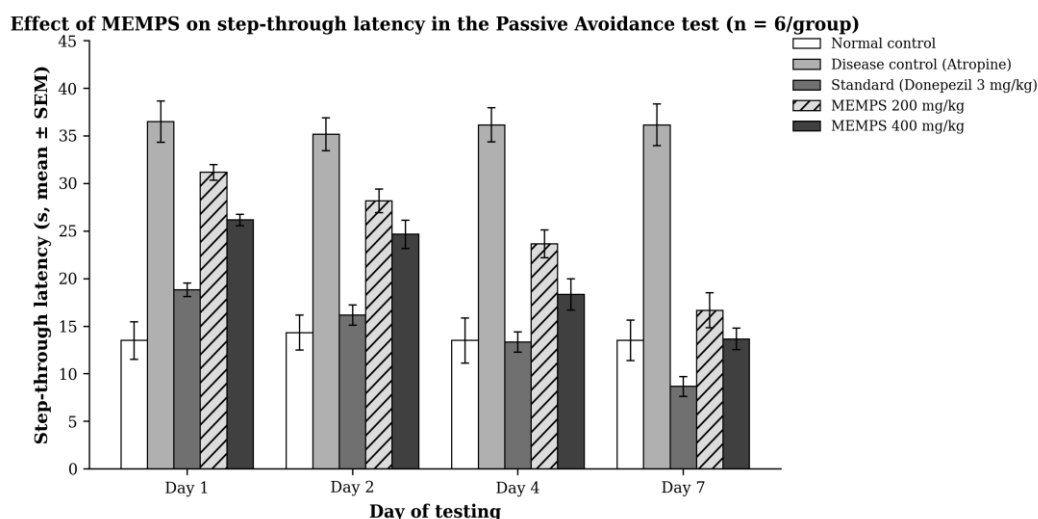


Figure 2: Effect of MEMPS on step-through latency in the Passive Avoidance test across days 1, 2, 4 and 7 (mean $\pm$ SEM, n = 6/group).

4.0 DISCUSSION

Atropine competitively blocks central muscarinic acetylcholine receptors, disrupting cholinergic neurotransmission in the hippocampus and cerebral cortex and thereby producing a reversible model of cognitive impairment that is widely used to screen candidate anti-dementia agents.^[3,4] In the present study, repeated atropine administration clearly impaired learning and memory in both behavioural paradigms, confirming successful induction of the model.

Treatment with MEMPS attenuated this atropine-induced impairment in a dose-related manner in both the Elevated Plus Maze and Passive Avoidance tests, with the higher dose (400 mg/kg) consistently outperforming the lower dose (200 mg/kg) and, by day 7, approaching the effect of the standard drug donepezil. This is consistent with the possibility that MEMPS supports cholinergic neurotransmission and/or protects cholinergic neurons from atropine-associated dysfunction, although the present behavioural data cannot on their own distinguish a cholinergic mechanism from a more general neuroprotective or antioxidant action.

The activity observed is plausibly related to the phytoconstituents confirmed on preliminary screening. L-DOPA, the principal bioactive constituent of *Mucuna pruriens* seeds, crosses the blood–brain barrier and is converted to dopamine, and has separately been shown to have antioxidant and metal-chelating properties in experimental models of neurodegeneration.^[9,10] Flavonoids and phenolic acids present in the extract are established free-radical scavengers^[11] and could plausibly limit the oxidative stress that contributes to cholinergic and synaptic dysfunction in dementia.^[2] These mechanisms are consistent with, but were not directly tested by, the present study: the antioxidant and neuroprotective potential of MEMPS was inferred from its phytochemical profile and the existing literature on *Mucuna pruriens* rather than measured biochemically here, and would benefit from direct confirmation (for example, brain or serum markers of oxidative stress, or acetylcholinesterase activity) in future work.

The improvement produced by MEMPS 400 mg/kg approached, but in most comparisons did not fully match, that of donepezil, suggesting that the extract's activity may be more modest than that of a selective acetylcholinesterase inhibitor, or may act through complementary, slower-onset mechanisms. These findings are in line with previous

reports of neuroprotective and antioxidant activity of *Mucuna pruriens* in models of Parkinson's disease and oxidative injury,^[10,12] and extend this evidence to a cholinergic memory-impairment model relevant to dementia.

This study has several limitations. Only two doses of MEMPS were evaluated; biochemical and histopathological correlates of neuroprotection were not measured; and the acute toxicity data used for dose selection were drawn from the literature rather than generated in this work [see accompanying notes to the authors]. Further studies incorporating biochemical markers of oxidative stress and cholinergic function, histopathological assessment, and pharmacokinetic and chronic toxicity evaluation are needed before the therapeutic potential of *Mucuna pruriens* in dementia can be more firmly established.

5.0 CONCLUSION

The methanolic extract of *Mucuna pruriens* seeds (MEMPS) produced a dose-dependent improvement in learning and memory in the atropine-induced model of cognitive impairment in Wistar rats, as assessed by the Elevated Plus Maze and Passive Avoidance tests, with effects at the higher dose approaching those of the standard drug donepezil. Preliminary phytochemical screening confirmed the presence of alkaloids, flavonoids, phenolic compounds, tannins, saponins, glycosides, steroids and carbohydrates, phytoconstituents that may collectively underlie the observed activity through antioxidant and cholinergic-modulating mechanisms. These findings provide preliminary experimental support for *Mucuna pruriens* seeds as a candidate source of neuroprotective, cognition-enhancing agents, warranting further biochemical, mechanistic, pharmacokinetic and toxicological investigation, and eventual clinical evaluation.

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CONFLICT OF INTEREST

The authors declare no conflict of interest.

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