



Original Article

Exploration of Beneficial Effect of Aqueous Extract of *Asparagus racemosus* on Spontaneous Locomotor Activity in Albino Wistar Rats: An Experimental Study

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ABSTRACT

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Background: *Asparagus racemosus* (Shatavari) has been traditionally used for its anti-spasm and locomotor properties. Despite ethnopharmacological evidence, its influence on locomotor activity, requires systematic experimental validation.

Objectives: To evaluate the effect of orally administered aqueous extract of *Asparagus racemosus* (AEAR) at doses of 200 mg/kg and 400 mg/kg on spontaneous locomotor activity in albino Wistar rats using the actophotometer model and to compare results with the standard CNS depressant diazepam.

Methods: Twenty-four albino Wistar rats (150–250 g) were randomly assigned to four groups (n=6): Group I (normal saline; control, 1 ml/100 g p.o. for 21 days), Group II (diazepam, 10 mg/kg i.p. on Day 21), Group III (AEAR 200 mg/kg p.o. for 21 days) and Group IV (AEAR 400 mg/kg p.o. for 21 days). Locomotor activity was measured using the actophotometer as beam-interruption counts over 5 minutes, both before and after treatment. Data were analysed by one-way ANOVA.

Results: Diazepam produced a 58.04% reduction in locomotor activity ($p < 0.001$). AEAR at 200 mg/kg and 400 mg/kg produced dose-dependent reductions of 36.36% and 38.20%, respectively (both $p < 0.001$). All treated groups showed statistically significant reduction in locomotor activity compared to the saline control.

Conclusion: AEAR exhibits significant CNS depressant activity, evidenced by dose-dependent suppression of spontaneous locomotion. These findings provide scientific validation for the traditional use of Shatavari and warrant further phytochemical investigations.

Keywords: *Asparagus racemosus*; locomotor activity; actophotometer; Wistar rat.

INTRODUCTION

The central nervous system (CNS) acts as the primary regulator of physiological, neurochemical, and behavioral activity. Movement disorders often arise from dysfunction of motor control circuits. Dopaminergic and cholinergic pathways in the basal ganglia, cerebellum regulate motor movements¹.

Spontaneous locomotor activity often arise from movement dysfunction leads to motor disorders, as qualified by an actophotometer, which is a validated indicator of central nervous system activity. CNS depressants such as barbiturates, benzodiazepines and ethanol suppress spontaneous motor activity in animal models, whereas stimulants augment it. Movement disorder affects over 3 billion individuals, making the actophotometer assay a standard tool for preliminary screening of centrally acting compounds².

Asparagus racemosus (Asparagaceae), known as Shatavari, is a classical herb listed in both the Indian and British Pharmacopoeias and traditionally employed as a nootropic and adaptogen³. Its bioactive constituents including steroidal saponins (shatavarins I–IV), alkaloids (asparagamine A) and flavonoids (quercetin, rutin) have demonstrated

neuroprotective, nootropic, anxiolytic, and adaptogenic activities preclinically⁴. Notably, lipophilic volatile constituents capable of crossing the blood–brain barrier have been shown to modulate neurotransmitter pathways relevant to sedation and motor control⁵.

However, the specific effect of the aqueous extract of *A. racemosus* (AEAR) on spontaneous locomotor activity has not been systematically evaluated under controlled, graded dosing conditions. The present study addresses this gap by assessing the locomotor-modulatory effects of AEAR at two oral dose levels in Wistar albino rats using the actophotometer, with diazepam as the reference standard.

AIM AND OBJECTIVES

The present study was undertaken to experimentally explore the potential beneficial effect of the aqueous extract of *Asparagus racemosus* (AEAR) in albino Wistar rats.

MATERIALS AND METHODS

Ethical Approval

The study was conducted in the Department of Pharmacology, LLRM Medical College, Meerut (U.P.), India, from October 2024 to September 2025. All experimental procedures were approved by the Institutional Animal Ethics Committee (Approval No. IAEC/LLRM/2024/03, dated 21/08/2024) registered under CCSEA, India (Registration No. 819/Go/ReRcBiBt/S/04/CPCSEA). All experiments were performed in strict accordance with CPCSEA guidelines.

Experimental Animals

Healthy albino Wistar rats of either sex, weighing 150–250 g, were procured from the CCSEA-approved Central Animal House, LLRM Medical College. Animals were housed in standard polypropylene cages under controlled conditions (temperature: 25±2°C; 12-hour light/dark cycle) with unrestricted access to standard rat pellet diet and tap water. Following a 7-day acclimatisation period, animals were deemed suitable for experimental use. Pregnant females were excluded from the study. Dose selection for AEAR was based on previously documented LD₅₀ values in rats, in accordance with OECD-423 guidelines.

Preparation of Aqueous Extract of Asparagus racemosus (AEAR)

Authenticated roots of *Asparagus racemosus* were procured from a local market, washed thoroughly with distilled water to remove adhering debris and shade-dried. The dried roots were powdered using a mechanical grinder. Five grams of the dried powder were subjected to hot continuous percolation with 150 ml distilled water in a Soxhlet apparatus for 4–5 hours. The extract was filtered sequentially through muslin cloth and Whatman No. 1 filter paper, concentrated by evaporation in a water bath and subsequently freeze-dried to yield the crude aqueous extract. A stock solution of 40 mg/ml was prepared in distilled water and used for dose preparation⁶.

Drugs and Chemicals

The aqueous extract of *Asparagus racemosus* (AEAR) was the test drug. Diazepam injection (Vulcan Laboratories Pvt. Ltd.) served as the standard reference drug. Normal (0.9%) saline served as the vehicle control.

Experimental Design and Grouping

Twenty-four animals were randomly assigned to four groups (n=6 per group) as outlined in Table I:

Table I. Experimental Groups and Treatment Schedule for Locomotor Activity Study

Group	Treatment	Dose & Route	Duration
I (Control)	Normal Saline	1 ml/100 g p.o.	21 days
II (Standard)	Diazepam	10 mg/kg i.p.	Single dose on Day 21
III (Test Low)	AEAR	200 mg/kg p.o.	21 days
IV (Test High)	AEAR	400 mg/kg p.o.	21 days

Actophotometer (Locomotor Activity) Assessment

Locomotor activity was assessed using a standard actophotometer (INCO, India), which detects spontaneous motor activity via light-beam interruptions. Each animal was individually placed in the actophotometer chamber and the number of beam-crossing events was recorded automatically by the digital counter over a 5-minute observation period. Exploratory ambulation (all four feet on the floor, crossing between opposing walls) constituted the unit of locomotor count. Baseline counts were recorded prior to treatment initiation. Post-treatment counts were recorded on Day 21, at 30 minutes following drug administration for Group II, and at the corresponding time point for Groups I, III and IV⁷.



Figure 1: Actophotometer

Statistical Analysis

Data are presented as mean $\pm$ Standard Error (SE). Group comparisons were performed using one-way ANOVA to assess significance versus the control group. $p < 0.05$ was considered statistically significant.

OBSERVATIONS AND RESULTS

Baseline Locomotor Counts

All four groups demonstrated comparable baseline locomotor counts prior to treatment, confirming uniformity of experimental animals at the start of the study (Table II). Mean baseline counts ranged from 149.33 ± 3.90 to 155.83 ± 4.76 across groups, with no statistically significant inter-group differences at baseline ($p > 0.05$).

Effect of AEAR on Spontaneous Locomotor Activity

Post-treatment actophotometer counts revealed statistically significant reductions in spontaneous locomotor activity in all treatment groups compared to the normal saline control, which showed no meaningful change between baseline and post-treatment values (150.67 ± 3.36 vs. 148.33 ± 2.85 , respectively).

Diazepam (10 mg/kg, i.p.), the standard CNS depressant, produced the greatest reduction in locomotor activity, with post-treatment counts falling to 62.67 ± 3.89 from a baseline of 149.33 ± 3.90 —representing a 58.04% reduction ($p < 0.001$).

AEAR at 200 mg/kg reduced locomotor counts from 155.83 ± 4.76 to 99.17 ± 3.24 , a reduction of 36.36% ($p < 0.001$). AEAR at 400 mg/kg reduced counts from 149.67 ± 3.77 to 92.50 ± 4.38 , a reduction of 38.20% ($p < 0.001$). A dose-dependent trend in locomotor suppression was thus observed with AEAR, with the higher dose producing a greater absolute and percentage reduction. Full results are summarised in Table II.

Table II. Effect of Aqueous Extract of Asparagus racemosus (AEAR) on spontaneous Locomotor Activity in Albino Wistar Rats (n=6)

Treatment Group	Dose & Route	Beam Interruption Count (Mean $\pm$ SE)		% Reduction p-value	
		Before Treatment	After Treatment		
Normal Saline (Control)	1 ml/100 g p.o.	150.67 ± 3.36	148.33 ± 2.85	1.55	NS
Diazepam (Standard)	10 mg/kg i.p.	149.33 ± 3.90	$62.67 \pm 3.89^{***}$	58.04	< 0.001
AEAR 200 mg/kg	200 mg/kg p.o.	155.83 ± 4.76	$99.17 \pm 3.24^{***}$	36.36	< 0.001
AEAR 400 mg/kg	400 mg/kg p.o.	149.67 ± 3.77	$92.50 \pm 4.38^{***}$	38.20	< 0.001

All values expressed as mean $\pm$ SE; n=6 rats per group. NS = not significant; $^{***}p < 0.001$ vs. control (one-way ANOVA + Dunnett's post hoc test).

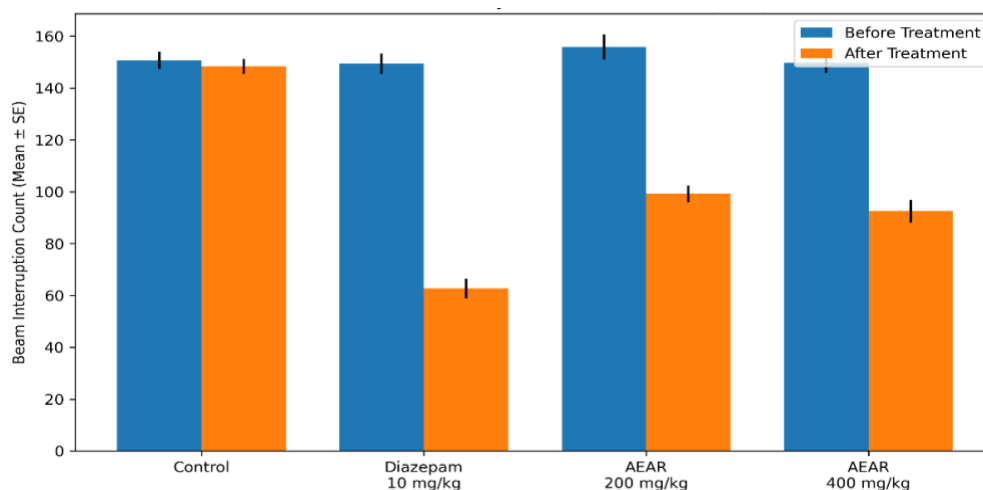


Figure 1: Effect of AEAR on spontaneous locomotor activity (actophotometer) in albino rats (n=6).

DISCUSSION

The actophotometer provides an objective, automated measure of CNS excitability, with reduced beam-crossing counts indicating depressant activity and increased counts indicating stimulation².

Diazepam, the reference standard, produced the expected maximal locomotor suppression (58.04%, $p < 0.001$), consistent with its well-characterised mechanism of positive allosteric modulation at GABA-A receptors enhancing chloride influx, hyperpolarizing neuronal membranes, and suppressing motor excitability⁸.

AEAR at 200 mg/kg and 400 mg/kg significantly reduced spontaneous locomotor activity by 36.36% and 38.20%, respectively ($p < 0.001$), demonstrating a pharmacologically coherent, dose-dependent CNS depressant profile. Although the magnitude was less than diazepam, this is characteristic of plant-derived adaptogens, which tend to exert milder, physiologically modulated locomotor effects.

The primary basis of this activity is likely multifactorial. Steroidal saponins, particularly shatavarin IV, may modulate GABAergic neurotransmission and attenuate HPA-axis-mediated arousal. Flavonoids such as quercetin and rutin are known to interact with benzodiazepine and serotonin receptor sites in vitro⁹. Additionally, the antioxidant activity of AEAR (IC_{50} 85–120 $\mu\text{g/mL}$, DPPH assay) may confer neuroprotection against oxidative dysregulation of motor¹⁰. These findings align with prior reports of monoamine modulation, stress-neurobehaviour attenuation, and acetylcholinesterase inhibition by *A. racemosus* extracts¹¹.

CONCLUSION

The above study concludes that aqueous extract of *Asparagus racemosus* (AEAR) produces significant, dose-dependent suppression of spontaneous locomotor activity in Wistar albino rats at both 200 mg/kg and 400 mg/kg ($p < 0.001$), confirming that it has a CNS depressant effect. Although the effect was of lesser magnitude in comparison to the standard drug diazepam. The consistent dose-response relationship supports the coherent central action, making the experiment credible. Further studies with larger sample size for a longer duration are needed to explore the beneficial effects in detail, offering an opportunity for its clinical development as an effective agent in various neurological disorders with locomotor disabilities.

DECLARATIONS

Conflict of Interest: The authors declare no conflict of interest.

Source of Funding: This research received no specific grant from any funding agency in the public, commercial or not-for-profit sectors.

Ethical Approval: Granted by the Institutional Animal Ethics Committee, LLRM Medical College, Meerut (Approval No. IAEC/LLRM/2024/03; CCSEA Reg. No. 819/Go/ReRcBiBt/S/04/CPCSEA).

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