Isolation of compound and CNS depressant activities of *Mikania scandens* Willd with special emphasis to brain biogenic amines in mice

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*Mikania scandens*, a twining herb that grows as a weed in India and Bangladesh is used as vegetables and is a good source of vitamin A, C, B complex, mikanin, sesquiterpenes, betasitosterin, stigmasterol and friedelin. The present communication reports CNS depressant activities with special emphasis to brain biogenic amines in mice. Ethanol extract of leaves of *M. scandens* (EEMS) was prepared by Soxhalation and analyzed chemically. EEMS potentiated sleeping time induced by pentobarbitone, diazepam and meprobamate and showed significant reduction in the number of writhes and stretches. EEMS caused significant protection against pentylene tetrazole-induced convulsion and increased catecholamines and brain amino acids level significantly. Results showed that EEMS produced good CNS depressant effects in mice.

**Keywords:** Analgesic and anticonvulsant activity, Behavioural profiles, Brain amino acid, Catecholamine, Climbing hempvine, CNS depressant effects, Locomotor activities, Pentylene tetrazole, Sleeping time

*Mikania scandens* Willd (Asteracae) a twining herb with long petiole, opposite leaves and small homogonous flower-heads, grows as a weed in India and Bangladesh¹. It is also used as a vegetable and is a good source of vitamin A, C, B and other active compounds such as mikanin, sesquiterpenes, β-sitosterin, stigmasterol and friedelin. Various parts of this plant have been used in tribal medicine to treat stomach ulcers, gastric problems, inflammation, microbial infections, psychopharmacological problems and carcinogenic manifestations²-⁶.

Considering the referential use of this plant by the tribes in mental disorder, studies on CNS activities of the plant were carried out. Preliminary pharmacological screening showed that ethanol extract of leaves *M. scandens* (EEMS) possessed marked CNS depressant action compared to other extracts of it. However, no work has been reported on the CNS activities as well as mechanism of action of this plant. Keeping this in view, the present study has been undertaken to investigate various CNS activities such as behavioural, locomotor, sedative-hypnotic, analgesic and anticonvulsant effects of EEMS with a special reference to brain biogenic amines in mice to substantiate the folklore claim as well as correlate neurotransmitters activity. Attempts have been made to isolate the compound responsible for such activities.

**Materials and Methods**

**Plant material**—Fresh leaves of *M. scandens* were collected from East Midnapore District of West Bengal, India during November, 2010 as it contains the maximum active constituents at that time. It was authenticated by Dr. HJ Chowdhury, Joint Director, Central National Herbarium, Botanical Survey of India, Howrah, India. A voucher specimen (DKJ 10/2009) has been preserved in the laboratory for further reference. After collection leaves were washed properly with water to remove foreign materials.

**Plant materials and extraction**—The shade dried fresh leaves of *M. scandens* were powdered, sieved (40 mesh size) and extracted successively with petroleum ether (40-60 °C), ethyl acetate and ethanol using a Soxhlet extractor. The extracts were concentrated to dryness in vacuum. The yield of petroleum ether (yellowish Green), ethyl acetate (dark green) and ethanol extract (reddish brown) (EEMS) was 3.71, 10.10 and 14.49% w/w, respectively with respect to dry starting materials.

**Phytochemical screening and isolation of compound**—Small amount of dried extract (EEMS) was appropriately treated to prepare sample solution and then subjected to the phytochemical tests. The

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phytochemical screening of EEMS was performed using the following reagents and chemicals: alkaloids with Dragendorff’s reagent; flavonoids with the use of Mg and HCl; tannins and phenolic compounds with ferric chloride and potassium dichromate solutions; steroids with Libermann Burchard reagent; terpenoids with tin and thionyl chloride; amino acids with ninhydrin solution and saponins with ability to produce suds. Carbohydrates were tested by the use of Molish reagents and conc. H$_2$SO$_4$. These were identified by the characteristic colour changes as per standard procedures$^{17,8}$.

EEMS on preparative TLC using water: ethanol: chloroform: ammonia (5:1:2:2 drop), a compound (A) was isolated.

**Animals**—Adult Swiss albino mice of either sex (22±2 g), 3-4 weeks of age, obtained from local supplier were acclimatized to standard laboratory conditions (55.65% RH, 23.0±2.0 °C and 12:12 h L:D cycle) for one week and given pellet diet and water ad libitum. All experiments were performed between 0800—1200 h to minimize circadian influences. The experimental protocol was subjected to the scrutiny of the Institutional Animal Ethical Committee and was cleared before initiating the experiments. The animals were handled as per guidelines of Committee for the Purpose of Control and Supervision on Animals (CPCSEA), New Delhi, India. For the pharmacological testing, EEMS was dissolved in propylene glycol (PG).

**Toxicity studies**—Acute toxicity study was carried out in mice with different doses (200, 250, 300, 400, 450, 500, 600, 650 mg/kg) of EEMS intraperitoneally (ip) into different group of mice, each containing 10 animals, as per the method described by Litchfield and Wilcoxon.$^9$ The behavioural profiles and mortality were observed for 72 h.

On the basis of LD$_{50}$ value and for the calculation of ED$_{50}$ value and therapeutic index, doses were selected for EEMS and standard drugs in analgesic and PTZ model$^{10,11}$.

**Behavioural effects**—The effects of EEMS (50, 70, and 90 mg/kg, ip) on righting reflex, pinna reflex, corneal reflex, awareness, grip strength, touch and pain responses on mice were observed by conventional methods. Chlorpromazine (5 mg/kg, ip) was used as a reference drug$^{12,13}$.

**Open-field test**—The method used was as described by Gupta et al.$^{14}$ The mice were divided into control (propylene glycol, 5 mL/kg, ip), Standard (diazepam, 1 mg/kg, ip) and test groups (50, 70 and 90 mg/kg, ip), respectively. The animals were placed on the floor of an open field (100 × 100 × 40 cm h) divided into a series of squares. The number of squares visited by each animal was counted for 3 min on 0, 30, 60, 90, 120, 180 and 240 min during the study period.

**Hole-cross test**—This experiment was carried out as described by Takagi et al.$^{15}$ Steel platinum was fixed in the middle of a cage having a size of 30 × 20 × 14 cm. A hole of 3 cm diameter was made at a height of 7.5 cm in the center of the cage. The number of passages of a mouse through the hole from one chamber to another was counted for 3 min on 0, 30, 60, 90, 120, 180 and 240 min after the treatment with control (propylene glycol, 5 mL/kg, ip), diazepam (1 mg/kg, ip) and EEMS (50, 70 and 90 mg/kg, ip), respectively.

**Hole-board test**—In this experiment each mice was placed carefully in the center of the board and the number of holes passed, head dipping and the number fecal boluses excreted recorded for 2 min. The observations were made on 0, 30, 60, 90, 120, 180 and 240 min during the study period after ip injection of control (propylene glycol, 5 mL/kg), diazepam (1 mg/kg) and EEMS (50, 70 and 90 mg/kg), respectively.$^{14}$

**Barbiturate potentiation**—Mice were divided into 4 groups, each group containing 6 mice. The animals of group I served as the control (propylene glycol, 5 mL/kg); groups II, III, and IV received EEMS at a low, medium and high dose (50, 70 and 90 mg/kg, ip respectively). Propylene glycol and the extracts were injected intraperitoneally 30 min prior to the administration of pentobarbitone sodium (40 mg/kg, ip), diazepam (3 mg/kg, ip) and meprobamate (100 mg/kg, ip). The sleeping time was noted by recording the interval between the losses and regaining of righting reflex$^{12,16}$.

**Assessment of analgesic activity**—Acetic acid-induced writhing tests: This method, involved ip injection of freshly prepared 1.2% v/v acetic acid. The number of abdominal constrictions (writhing) and stretching with a jerk at the hind limbs and bending of trunk were counted between 5 and 15 min after administration of acetic acid. For the test, EEMS was administered ip at 30, 50, 70 and 90 mg/kg, in mice. Acetylsalicylic acid (ASA, Cipla Ltd, Mumbai, India), paracetamol (PCM, Tablets India Ltd, Chennai, India) and morphine sulphate (M; Modi-
Hundi Pharma Ltd, New Delhi, India) were used as reference standards at doses of 40, 68, 100 mg/kg; 40, 68, 100 mg/kg; 0.75, 1.15, 1.50 mg/kg ip, respectively to compare with EEMS activity. The decrease in acetic acid induced writhing numbers was calculated. The analgesic activity was expressed in terms of percentage and compared with other reference drugs. The ED$_{50}$ value and therapeutic index of EEMS were also calculated.$^{13,17-19}$

**Assessment of acute anticonvulsant activity**—Pentylene tetrozole induced seizures: EEMS was administered ip in varying doses (30-100 mg/kg) 30 min prior to the administration of pentylene tetrozole (Hi-Media Laboratories Pvt. Ltd., Mumbai, India; 80 mg/kg, ip) in mice. The time needed for the development of unequivocal sustained clonic seizures activity involving the limbs was carefully noted; Isolated myoclonic jerks or other preconvulsive chewing behaviour were not counted. Seizure free state for 1 h was taken as protection.$^{12,20}$

**Determination of brain biogenic amines**—Pentylentetrazole (PTZ) at a dose of 80 mg/kg body wt ip was used as chemo convulsive agent. Pentobarbitone sodium (30 mg/kg) was used as standard. EEMS at 0.050, 0.070, 0.090 g/kg body wt was given to mice ip, 30 min prior to the administration of pentylentetrazole.

Mice were subdivided into following 10 groups of 10 each:

I: Normal saline (0.9% NaCl, w/v, 5 mL/kg, ip).
II: Propylene glycol (5 mL/kg as vehicle, ip).
III: Only pentylentetrazole (PTZ) (80 mg/kg, ip).
IV: Pentobarbitone sodium (30 mg/kg, ip) administered 30 min before pentylentetrazole (80 mg/kg, ip).
V: EEMS (0.050 g/kg, ip) + pentylentetrazole (80 mg/kg, ip).
VI: EEMS (0.070 g/kg, ip) + pentylentetrazole (80 mg/kg, ip).
VII: EEMS (0.090 g/kg, ip) + pentylentetrazole (80 mg/kg, ip).

The injections were given once a week and the experiments were carried out for 6 weeks. Animals from each group were killed by cervical dislocation 30 min after the last dose. The brains were dissected out, weighed and kept on ice for further processing.

**Biochemical estimation for brain biogenic amines**—Brains were homogenized with dry n-butanol and then centrifuged. About 4 mL aliquots of the clear supernatant were extracted with 3 mL of 0.1 M phosphate buffer. Then, after adding 4% EDTA, 0.2 mL iodine solution, 0.5 mL alkaline sulphite and 0.6 mL 5N acetic acid, the solutions were heated and cooled. Standard solutions of 0.1 µg/mL of epinephrine, norepinephrine and dopamine were prepared. The intensities of fluorescence in resulting solutions were determined using a spectrophotofluorometer (Perkin Elmer MPF-44B, USA) at wavelengths of 400/500 and 310/365 for epinephrine, norepinephrine and dopamine respectively.$^{21}$ The concentration of 5-HT in the solution was calculated from the standard curves.$^{22}$

Paper chromatographic method using an undimensional descending technique was adopted for GABA, glutamate and glutamine analysis. The positions of each amino acid in the chromatogram were developed with ninhydrin (90.1%). The eluted portions were analysed using a spectrophotometer ($^{23}$ Systonic M- no 103 at 570 mµ).

**Statistical analysis**—LD$_{50}$ and ED$_{50}$ values of EEMS were calculated with 19/20 (95%) confidence limit using commercial computer and Therapeutic Index (TI) number was calculated by dividing the LD$_{50}$ by the ED$_{50}$ in each case Results were analyzed for statistical significance using one-way ANOVA followed by Dunnett’s test or post hoc Turkey Test (in case of determination of brain catecholamine and amino acids only). For the open-field, hole-cross and hole-board tests, two-way ANOVA followed by Bonferroni post tests was adopted. $P < 0.05$ was considered significant.

**Results**

**Isolation of component**—EEMS on preliminary phytochemical analysis were found to contain steroids, tannin and phenolic compounds, saponins, flavonoids, carbohydrates and amino acids.

Compound A (yield 0.1% with respect to dry starting material) having $R_f$ value 0.7, the $\lambda_{max}$ value 219 nm and characteristics I.R. peaks at 3432.67 cm$^{-1}$ (for –OH, N-H stretching, -CONH$_2$), 3175.22 cm$^{-1}$ (C-H stretching), 1625.7 cm$^{-1}$ (amino compound, C=O stretch amide), 1402.96 cm$^{-1}$ (C-H stretching), 1625.7 cm$^{-1}$ (alkenes, cycloalkenes) molecular wt 127, suggesting the structural similarity with 4-hydroxy 2, 5 diene-hexamide$^{24}$ (Fig. 1). The nature of the compound was suggested by $^1$H-NMR, comparable physicochemical properties and reference data available from literature.
Safety evaluation—In toxicity studies, the EEMS was found to have the LD\(_{50}\) value of 450 (448.4-451.8) mg/kg, ip.

Effect on general behavioral profiles—The results obtained from general behavioural profiles are shown in Table 1 and Figs. 2–4. It was observed that EEMS depressed awareness and alertness, touch and pain responses, grip strength, altered righting, pinna and corneal reflexes when compared to the control (propylene glycol, 5 mL/kg, ip). However, chlorpromazine hydrochloride (standard) produced a significant depression of these responses in comparison with EEMS (Table 1).

EEMS also significantly decreased locomotor activities (Figs. 2–4). The locomotors activity lowering effects were evident in the hole cross and open field test at the 2\(^{nd}\) observation (30 min) and continued up to 7\(^{th}\) observation period (240 min). In the hole-board test the depressant action of EEMS was found for the 2\(^{nd}\) observation (30 min) and continue up to 5\(^{th}\) observation (120 min). The results were also dose dependent and statistically significant.

Barbiturate potentiatio—It was found that EEMS itself did not produce any sedative activity in experimental animals. However, three doses of EEMS (50, 70 and 90 mg/kg) potentiated the sleeping time induced by standard hypnotics viz pentobarbitone (79.40, 117.37 and 149.88\%, low, medium and high dose, respectively), diazepam (81.42, 115.90, and 156.80\%, low, medium and high dose, respectively) and meprobamate (47.08, 67.21, and 92.37\%, respectively) (Table 2).

Analgesic activity—In acetic acid-induced writhing tests, protection produced by EEMS against acetic acid–induced writhing was dose dependent and its ED\(_{50}\) value was found to be 40.35 (33.65-46.75) mg/kg, ip and therapeutic index was 11.15 (Table 3). The ED\(_{50}\) values of morphine, paracetamol and acetyl salicylic acid were found to be 0.82 (0.74-0.91) mg/kg, ip, 55 (46.90-63.22) mg/kg, ip and 57 (47.52-66.50) mg/kg ip, respectively (Table 3).
Anticonvulsive activity—Acute anticonvulsive study: EEMS significantly inhibited the onset and incidence of convulsion against pentylene tetrazole-induced seizures. Protection produced against pentylene tetrazole-induced convulsion was dose dependent and ED₅₀ and therapeutic index of EEMS was determined to be 54 (40.3-67.8) mg/kg, ip and 8.3, respectively (Table 4).

Chronic study: The results related with brain biogenic amines are summarized in Figs. 5 and 6. EEMS significantly increased (compared to vehicle control mice) the levels of epinephrine, nor-epinephrine, dopamine and 5 HT (low dose by 333.3, 322.2, 44.4 and 100%, respectively) in mice brain after six weeks of treatment in a dose dependent manner. The extract also significantly elevated the levels of GABA, Glutamate and Glutamine (low dose by 78.1, 74.1 and 96.3%, respectively) as compared to respective vehicle control (propylene glycol) group.

Discussion
In this study, pentobarbitone, diazepam and meprobamate were used to induce sleep in mice. Benzodiazepines are believed to act at specific binding sites that are closely linked to gamma-aminobutyric acid (GABA) receptors, the binding of benzodiazepines enhancing GABA-ergic transmission. Although the cause of prolongation of diazepam-induced sleeping time is not known, the enhancement of GABA-ergic transmission might be related to its sedative activity. Prolongation of

| Table 2—Effect of EEMS on sleeping time (min) induced by pentobarbitone, diazepam and meprobamate in mice |
| Values are mean ± SE from 6 animals in each group |
| Treatment | Pentobarbitone(40 mg/kg, ip) | Meprobamate(100 mg/kg, ip) | Diazepam(3 mg/kg, ip) |
| Control (PG, 5 mL/kg, i.p.) | 40.3 ± 0.71 | 61.6 ± 0.85 | 74.8 ± 0.87 |
| EEMS (50 mg/kg, i.p.) | 72.3 ± 1.25* | 90.6 ± 1.65* | 135.7 ± 2.01* |
| EEMS (70 mg/kg, i.p.) | 87.6 ± 2.04* | 103.0 ± 2.00* | 161.5 ± 3.00* |
| EEMS (90 mg/kg, i.p.) | 100.7 ± 2.10* | 118.5 ± 3.02* | 192.1 ± 3.74* |

Statistical analysis done by ANOVA followed by post hoc test of significance, Dunnett’s ‘t’ test. *P<0.001 vs vehicle control.

| Table 3—Effect of EEMS (30, 50, 70, 90 mg/kg, ip) on the writhing and stretching induced in mice by ip injection of 1.2% acetic acid (writhing test) |
| Values are mean ± SE from 6 animals in each group |
| Treatment | Dose (mg/ kg, ip) | Number of writhing | Protection(%) | ED₅₀ (mg/kg, ip)# (95 % confidence limit) |
| Vehicle (PG) | 5 mL/kg | 80.31 ± 0.98 | – | – |
| EEMS | 30 | 50.00 ± 1.70* | 37.74 |
| | 50 | 28.60 ± 1.56* | 64.39 | 40.35 (33.65 – 46.75) |
| | 70 | 6.00 ± 0.44* | 92.53 |
| | 90 | A | 100 |
| PCM | 40 | 51.29 ± 1.70* | 36.14 |
| | 68 | 30.99 ± 1.84* | 61.42 | 55 (46.90 – 63.22) |
| | 100 | 7.76 ± 0.51* | 90.33 |
| ASA | 40 | 51.8 ± 1.00* | 35.37 |
| | 68 | 31.99 ± 1.78* | 60.16 | 57 (47.52 – 66.50) |
| | 100 | 9.26 ± 0.91* | 88.47 |
| M | 0.75 | 43.58 ± 1.85* | 45.74 |
| | 1.15 | 24.00 ± 1.20* | 70.12 | 0.82 (0.74 – 0.91) |
| | 1.50 | 6.85 ± 0.87* | 91.47 |
pentobarbitone induced sleeping time might be due to tranquilizing action as well as CNS depressant action. Although the exact mechanism responsible for the sedation action of meprobamate is not clear, it might be due to CNS depressant action or due to enhancement of GABA-ergic transmission. EEMS potentiated significantly the duration of pentobarbitone, diazepam and meprobamate–induced sleep in mice, suggesting probable tranquilizing action as well as CNS depressant action.

Now it is established that inhibition of the touch response, righting reflex, and grip strength is probably produced due to a pronounced CNS depressant action. Reduction of pinna reflex and awareness may be due to synapses block of the afferent pathway or due to overall CNS depressant action. In the present study, the mechanism whereby EEMS depressed awareness, touch and pain responses, righting reflex, pinna reflex, corneal reflex, and grip strength may also be due to synapses block of the efferent pathway or by overall CNS depressant action.

The present study revealed the effect of EEMS on locomotors activities in mice model. Open field, hole cross and hole board methods are common ways to investigate the locomotors activities, which are often used to assess the depressant effects of crude extracts. Locomotor activities are considered as an increase in alertness and decrease in locomotor activities which eventually indicates a sedative effect. EEMS displayed a CNS depressant activity as indicated by the decrease in locomotor activity in the open-field, hole-cross and hole-board tests.
In the present study, EEMS elicited analgesic and anticonvulsant activity in mice in a dose dependent manner and therapeutic index of EEMS was found to be large, thereby indicating its safety margin. The EEMS treatment significantly reduced the number of writhing episodes induced in mice by acetic acid administration suggesting its analgesic activity\(^2\).\(^3\)\(^4\) (Table 4).

Pal et al.\(^1\) and Mazumder et al.\(^3\) found that analgesic activity was probably mediated by inhibition of a post synaptic specific sensitive mechanism either by depleting endogenous levels of nor-epinephrine via dopamine-β-hydroxylase inhibition or by blocking norepinephrine effects at the receptor level. The promising analgesic activity shown by EEMS in the present investigation is probably mediated through the same mechanism as mentioned above. Analgesic and anticonvulsant activities can also be mediated by other mechanisms. The increase of brain serotonin and GABA level may also be responsible for analgesic and anticonvulsant activities\(^2\).\(^3\)\(^4\). It was found that EEMS increased the brain serotonin and GABA level in mice (Figs. 5 and 6). Therefore, analgesic and anticonvulsant activities produced by EEMS may be related to the increased brain serotonin and GABA level in mice\(^2\).\(^3\)\(^4\).

The present results show that EEMS is also effective against pentylene tetrozole induced seizures. It is known that anticonvulsants mediate their action through alteration in various neurotransmitter levels in various regions of the brain\(^1\). In the present study, the biogenic amines were estimated in the whole brain (Figs. 5 and 6). Epinephrine and nor epinephrine are essentially excitatory substances, but both catecholamine often have depressant action\(^2\). Both catecholamine and 5-HT appear to play roles in determining the seizure thresholds for electroshock\(^3\). Dopamine also functions independently as a neuromodulator. It not only increases the level of 5-HT promote sleep, but the melatonin, which is synthesized from 5-HT in the pineal gland and may also occur in other parts of the brain, also have a role in sleep and as a potent inducer of sleep\(^2\). In humans, decreased activity of nor adrenaline and dopamine has been found in some epileptic patients\(^3\). So, the protection offered by EEMS against chemo convulsions in mice probably is due to the increased levels of catecholamines and 5-HT in brain (Figs. 5 and 6). On the other hand, it is well established that GABA (γ-amino butyric acid) protects the mice against the convulsion induced by pentylene tetrozole, etc\(^2\).\(^3\). As far as GABA is concerned, the following facts support its involvement:

(a) lowering levels of GABA in the brain results in the appearance of convulsion\(^4\);
(b) some convulsive drugs found to be GABA antagonists\(^4\);
(c) certain antiepileptic drugs enhance the synaptic action of GABA\(^4\).

In addition to GABA, the increased level of glutamate and glutamine may also be correlated with the anticonvulsive property of EEMS. Increase in the levels of glutamate and glutamine is possibly a result of accelerated conversion of α-ketoglutarate to glutamine acid transmission of glutamine and reduced oxidation of α-ketoglutarate through the succinate pathway\(^1\).\(^2\).\(^3\)\(^4\)\(^5\)\(^6\)\(^7\)\(^8\)\(^9\)\(^10\)\(^11\)\(^12\)\(^13\)\(^14\)\(^15\)\(^16\)\(^17\)\(^18\)\(^19\)\(^20\)\(^21\)\(^22\)\(^23\)\(^24\)\(^25\)\(^26\)\(^27\)\(^28\)\(^29\)\(^30\)\(^31\)\(^32\)\(^33\)\(^34\)\(^35\)\(^36\)\(^37\)\(^38\)\(^39\)\(^40\)\(^41\).

On the basis of experimental evidences, it may be concluded that the catecholamine and GABA systems have a significant role with respect to CNS depressant and anticonvulsive properties of the processed extract.

EEMS enhanced sleeping time, analgesic and anticonvulsant activities and reduced different behavioural reflexes including locomotor activities in treated mice. It can be confirmed from the present discussion that the ethanol extract of M. scandens (EEMS) exhibited strong CNS depressant action, which was further strengthened by the significant increase in the levels of brain biogenic amines in EEMS treated mice (Figs 5 and 6).

Preliminary phytochemical screening reveals the presence of flavonoids, saponin in EEMS. There are reports on the role of flavonoids in analgesic activity primarily by targeting prostaglandins\(^3\).\(^4\). Again, there are also reports on the role of saponins in anticonvulsive activity\(^1\).\(^2\)\(^3\)\(^4\)\(^5\).\(^6\)\(^7\)\(^8\)\(^9\)\(^10\)\(^11\)\(^12\)\(^13\)\(^14\)\(^15\)\(^16\)\(^17\)\(^18\)\(^19\)\(^20\)\(^21\)\(^22\)\(^23\)\(^24\)\(^25\)\(^26\)\(^27\)\(^28\)\(^29\)\(^30\)\(^31\)\(^32\)\(^33\)\(^34\)\(^35\)\(^36\)\(^37\)\(^38\)\(^39\)\(^40\)\(^41\). Therefore, it can be assumed from the above discussion that the analgesic and anticonvulsant activity of EEMS may be due to the presence of flavonoids and saponins in EEMS respectively.

Conclusions

EEMS enhanced sleeping time, reduced different behavioral reflexes and depressed locomotor activities. It showed excellent analgesic and anticonvulsant activities associated with the significant increase in the levels of brain biogenic amines in EEMS treated mice. The present
results demonstrate the potential effectiveness of *M. scandens* (EEMS) as good CNS depressant drug which supports the claim by traditional medicine practitioners as a psychological remedy.

References


