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IN VITRO ANTIOXIDANT ACTIVITIES AND *IN VIVO* ANTI-NOCICEPTIVE AND NEUROPHARMACOLOGICAL ACTIVITIES OF *MIMOSA PUDICA*

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ABSTRACT

Mimosa pudica is the herb that shows sensation on touch. It has been identified as Lajjalu in Ayurveda and has been found to have anti-asthmatic, aphrodisiac, analgesic, anti- inflammatory and antidepressant activities. The aim of this study was to evaluate i*n-vitro* antioxidant activity and *in vivo* biological activity considering anti-nociceptive assessment & neuropharmacological study include open field and swimming test. There is no scientific report on analgesic activity and neuropharmacological activity of *Mimosa pudica*, therefore the present study was undertaken to examine the possible *in-vitro* antioxidant activity of the extracts of *Mimosa pudica* was performed using DPPH free radical scavenging, cupric reducing antioxidant capacity, total antioxidant capacity, total phenol and total flavonoid content determination assays. The result of tests for *in-vitro* antioxidant activity indicates that petroleum ether extract shows maximum antioxidant activity. This plants leaf extracts also exhibit significant anti-nociceptive activity and neuropharmacological activity.

Key words: *Mimosa pudica*, free radical scavenging, total phenolic content, writhing test, neuropharmacological action.

INTRODUCTION

Herbal remedies have been used for decades and centuries. Before the discovery and availability of modern synthetic drugs, humans were completely dependent on medicinal herbs for prevention and treatment of diseases. In the history of ancient civilizations, the use of medicinal herbs for curing diseases has been documented. The drugs were used in crude forms like decoction, infusion, tincture and poultice. *Mimosa pudica* (Fam. Mimosaceae) locally known as Lozzaboti. The plant has very sensitive leaves which fold on touching and has reddish roots. The useful parts of this plant are roots, leaves and flower heads. The whole plant is used medicinally in ayurvedic folk medicine and its photochemical studies revealed the presence of minosine, orientin, isoorientin, β- sterol, D-pinitol, norepinepherine, crocetin, tannins and turgorins. In our country this plant is grown in rainy season and this plant is also called as touch me not plant at the same it should have a various names in different languages like in English sensitive plant, in Hindi lazonthi, in Tamil tottasiningi. Leaves and stems of the plant have been reported to contain an alkaloid mimosine, leaves also contain mucilage and root contains tannins [1]. *Mimosa pudica* is used for its anti diarrhoeal [2] ,anti-convulsant[3] and cytotoxic properties[4] .The plant also contains turgorins, leaves and roots are used in treatment of piles and fistula. Paste of leaves is applied to hydrocele. Cotton impregnated with juice of leaves is used for dressing sinus. Plant is also used in the treatment of sore gum and is used as a blood purifier [1].

Remote peoples are used in the treatment of inflammation, diabetic, fever, piles and various diseases.They are useful in vitiated conditions of pitta, leucoderma, vaginopathy, metropathy, ulcers, dysentery, inflammations, burning sensation, hemorrhoids, jaundice, asthma, fistula, small pox, strangury, spasmodic, affections and fevers. The

leaves are bitter, sudorific and tonic, and are useful in hydrocele, hemorrhoids, fistula, scrofula, conjunctivitis, cuts and wounds and hemorrhages.

The whole plant is used internally for vesicle calculi and externally for odema, rheumatism, myalgia and tumors of the uterus [5]. Literature survey on *Mimosa pudica* suggest various therapeutic use of plant reported such as urolithiasis[6], ovulation, vibriocidal[7] , antidepressant[8] ,estrogenic and antiestrogenic activities[9] anti implantation and antiestrogenic activity [10], effects on oestrous cycle and ovulation[11], hyperglycemic[12] anticonvulsant activity[13] hyaluronidase and proteasem activities[14] .

MATERIALS AND METHODS

Plant materials: The whole plants of *Mimosa pudica* were collected during August, 2012, from Kotbari, Mainamoti, Comilla. Then the plant sample was submitted to the National Herbarium of Bangladesh, Mirpur, Dhaka. One week later its voucher specimen was collected after its identification (Accession No.:- 37879).

Preparation of extract: The sun dried and powdered leaves (300 gm) of *Mimosa pudica* was successively extracted in a Soxhlet extractor at elevated temperature using 200 ml of distilled petroleum ether (40-60)°C which was followed by n-hexane, ethanol, chloroform and methanol. All extracts were filtered individually through filter paper and poured on Petri dishes to evaporate the liquid solvents from the extract to get dry extracts. The dry crude extracts were weighed and stored in air-tight container with necessary markings for identification and kept in refrigerator (0-4)°C for future investigation.

DPPH Free radical scavenging activity: The free radical scavenging activity of the leaves extractives of *Mimosa pudica* was evaluated by the DPPH method[15]. DPPH was used to evaluate the free radical scavenging activity (antioxidant potential).

Cupric reducing antioxidant capacity (CUPRAC): The assay was conducted according to Apak *et al.*, 2004 [16]. 0.025 ml of plant extract or standard of different concentrations solution, 1 ml of copper (II) chloride solution (0.01 M prepared from $CuCl_2.2H_2O$), 1 ml of ammonium acetate buffer at pH 7.0 and 1 ml of neocaproin solution (0.0075 M) were mixed. The final volume of the mixture was adjusted to 4.1 ml by adding 0.6 ml of distilled water and the total mixture was incubated for 1 hour at room temperature. Then the absorbance of the solution was measured at 450 nm using a spectrophotometer against blank. Ascorbic acid and BHT was used as a standard.

Determination of total antioxidant capacity: The total antioxidant capacity was evaluated by the phosphomolybdenum[17] . 0.025 ml of extract and sub-fraction in ethanol, ascorbic acid used as standard (12.5-200 µg/ml) and blank (ethanol) were combined with 0.3 ml of reagent mixture separately and incubated at 95°C for 90 minutes. After cooling to room temperature, the absorbance of each sample was measured at 695 nm against the blank. The antioxidant activity is expressed as the number of equivalents of ascorbic acid and was calculated by the following equation:

$$A=(c\times V)/m$$

Where, A = total content of Antioxidant compounds, mg/gm plant extract, in Ascorbic acid Equivalent c = the concentration of Ascorbic acid established from the calibration curve, mg/ml, V = the volume of extract in ml, m = the weight of crude plant extract, gm.

Total phenolics analysis: Total phenolic content of leaf extractives of *Mimosa pudica* was measured employing the method involving Folin-Ciocalteu reagent as oxidizing agent and gallic acid as standard [18].

Determination of total flavonoids content: Aluminum chloride colorimetric method was used for flavonoids determination[19]. 1 ml of the plant extracts/standard of different concentration solution was mixed with 3 ml of methanol, 0.2 ml of aluminum chloride, 0.2 ml of 1 M potassium acetate and 5.6 ml of distilled water. It remained at room temperature for 30 min; the absorbance of the reaction mixture was measured at 415 nm with spectrophotometer against blank. Methanol served as blank. The total content of flavonoid compounds in plant methanol extracts in quercetin equivalents was calculated by the following equation:

$$C=(c\times V)/m$$

Where; C = total content of flavonoid compounds, mg/gm plant extract, in quercetin equivalent, c = the concentration of quercetin established from the calibration curve in mg/ml, V = the volume of extract in ml and m = the weight of crude plant extract in gm.

Animals and treatment: Swiss albino mice weighing 20-30 g of either sex were used for the research. The mice were purchased from the Animal Research Branch of the International Centre for Diarrhoeal Disease and Research, Bangladesh (ICDDR,B)

Mohakhali, Dhaka. The animals were maintained under standard hygienic conditions (temperature 27 ± 1.0 C, relative humidity 55-65% and 12 h light/ 12 h dark cycle) and had free access to feed and water ad libitum. The animals were acclimatized to laboratory condition for one week prior to experiments.

Measurement of analgesic activity of Mimosa pudica (acetic acid induced writhing test): The acetic acid writhing test in mice as described by Koster *et al.,* 1959 [20] was employed with slight modification. Mice were divided into 4 groups of 6 mice each. The first group was given 10ml\kg of 1% Tween 80 i.p. and served as control. Group 2 received Diclofenac Sodium 100 mg\kg of body weight and served as standard, groups 3, 4 received methanol & ehanol extract of leave of *Mimosa pudica* 100, 150 mg\kg of body weight. Thirty minutes later after oral administration, each mouse was injected intraperitoneally with 0.7% acetic acid at a dose of 10 ml/kg body weight. Full writhing was not always completed by the animal, because sometimes the animals start to give writhing, but they do not finish it. This incomplete writhing was taken as a half writhing. Accordingly, two half writhing were considered as one full writhing. The number of writhing responses was recorded for each animal during a subsequent 5 min period after 15 min IP administration of Acetic acid and the mean abdominal writhing for each group was obtained.
The percentage inhibition was calculated using the formula
% Inhibition = [1- (No. of writhing of standard or sample/ No. of writhing of control)] X 100

Neuropharmacological study (open field test): According to Gupta *et al.,* 1971[21] open field was performed and test to monitor behavioral responses in mice that were placed in a novel and bright arena. Rodents tend to stay away from brightly illuminated areas. The experiment also assesses a range of anxiety-induced, locomotor activity and exploratory behaviors. The animals were divided into 5 groups of 6 mice each. The first group was given 10ml\kg of 1% Tween 80 orally and served as control. group 3 received methanol extract of leaves of *Mimosa pudica* 150 mg\kg of body weight, group 4 received ethanol extract of leaves of *Mimosa pudica* 150 mg\kg of body weight and group 5 received chloroform extract of leaves of *Mimosa pudica* 150 mg\kg of body weight, while the group 2 was given 2mg Diazepam per kg body weight orally and served as standard. The test was carried out according to the technique described by Gupta *et al.,* 1971[21] with slight modification. The open field apparatus is made of hardboard (60cmx60cm; 40cm walls).Blue lines drawn on the floor divide the floor into 36 squares (10cm x 10cm squares alternatively colored black and white and Central Square (10cm x 10cm) in the middle clearly marked. The number of squares visited by the animals was calculated for 2 min, at 0, 30, 60, 90, 120 and 150 min subsequent to oral administration of the xperimental crude extracts.

Neuropharmacological study (Forced swimming test, FST): According to Porsolt *et al.,* 1978[22] swimming test was performed. Animals were randomly divided into 5 groups (n=6). The first group was given 10ml\kg of 1% Tween 80 orally and served as control. group 3 received methanol extract of leaves of *Mimosa pudica* 150 mg\kg of body weight, group 4 received ethanol extract of leaves of *Mimosa pudica* 150 mg\kg of body weight and group 5 received chloroform extract of leaves of *Mimosa pudica* 150 mg\kg of body weight, while the group 2 was given 2mg Diazepam per kg body weight orally and served as standard. The forced swim test was carried out on mice individually forced to swim in an open acquire water tank apparatus (29cm x 19cm x 20cm), containing 9 cm of water at 25±1 °C; the total duration of immobility during the 6-min test was scored as described. Each mouse was judged to be immobile when it ceased struggling and remained floating motionless in the water, making only those movements necessary to keep its head above water. The duration of immobility was recorded. Decrease in the duration of immobility during the FST was taken as a measure of antidepressant activity.

RESULTS AND DISCUSSION

DPPH Free radical scavenging activity: All the extractives of *Mimosa Pudica* were subjected to free radical scavenging activity using DPPH by using ascorbic acid (ASA) as reference standard **(Table 1 & Figure 1).**

In this investigation, the methanol extract (ME) showed significant free radical scavenging activity with IC_{50} value of 35.33 µg/ml.

Cupric reducing antioxidant capacity (CUPRAC): Reduction of Cu^{2+} ion to Cu^{+} was found to rise with increasing concentrations of the different extracts. The standard ascorbic acid, BHA and BHT showed highest reducing capacity. Among the extracts the methanol extract of *Mimosa Pudica* showed maximum reducing capacity that is comparable to ascorbic acid and BHT **(Figure 2).**

Total antioxidant capacity: Total antioxidant capacity of the different extracts of *Mimosa pudica* was evaluated by the phosphomolybdenum method and was expressed as ascorbic acid equivalents (AAE) per gram of plant extract. Total antioxidant capacity of the test samples was calculated using the standard curve of ascorbic acid (y = 0.001x - 0.023; R^2=0.994) **(Figure 3)** Petroleum ether extract of *Mimosa pudica* was found to possess the highest total antioxidant capacity **(Table 2)**. Total antioxidant capacity of the extracts was found to decrease in the following order: Petroleum ether extract > Methanol extract > Ethanol extracts **(Table 2).**

Total phenolics analysis: All the extractives of *Mimosa Pudica* were tested for total phenolic content. The total phenolic contents of the test fractions were calculated using the standard curve of Gallic acid (y = 0.006x - 0.002; R^2 = 0.996) **(Figure 4).** Petroleum ether extract of was found to contain the highest amount of phenols. Phenol contents of the extracts were found to decrease in the following order: Petroleum ether extract > Methanol extract > Ethanol extracts **(Table 3).**

Total flavonoid content: Aluminium chloride colorimetric method was used to determine the total flavonoid contents of the different extracts of *Mimosa pudica*. Total flavonoid contents was calculated using the standard curve of quercetin (y = 0.318x - 0.306; R^2 = 0.815) **(Figure 5)** and was expressed as quercetin equivalents (QE) per gram of the plant extract. Petroleum ether of *Mimosa pudica* was found to contain the highest amount of flavonoid **(Table 4).** Flavonoid contents of the extracts were found to decrease in the following order: Petroleum ether extract > Methanol extract > Ethanol extracts **(Table 4).**

Acetic acid induced writhing method: The result of Acetic acid induced writhing method with leaf extracts of *Mimosa pudica* is shown in **Table 5.** Acetic acid induced writhing model represents pain sensation by triggering localized inflammatory response. Such pain stimulus leads to the release of free arachidonic acid from tissue phospholipids[23]. The constriction response of abdomen produced by acetic acid is a sensitive procedure to evaluate peripherally acting analgesics. It has been associated with prostanoids in general, for example, increased levels of PGE_2 and PGF_{2a} in peritoneal fluids [24, 25] as well as lipoxygenase or cyclo-oxygenases products[26, 27] and acid sensing ion channels[28] . **Table 5** represent the effect of different extracts of *Mimosa pudica* in Acetic acid induced writhing test. Methanol and ethanol extracts inhibited writhes in a dose dependent manner. But ethanol extract at 100 mg/kg showed highest inhibition (25.08%) which is even higher than the standard drug (29.32%).

Neuropharmacological study (open field test): This experiment was performed to assay general locomotor activity levels. After investigation with leaf extracts of *Mimosa pudica* following data was observed **(Table 6).**

According to Hall, 1936[29]originally proposed that measuring aspects of rat behavior in a contained arena would indicate the emotional reactivity of the subjects. Many reports have validated open field tests as useful measures of emotional reactivity[30, 31] e.g., reviewed by Sandnabba, 1996[32] for Turku aggressive mice; others have not found differences in open-field activity despite differences in other anxiety measures e.g., MHC-congenic[33]. Nevertheless, the open-field test remains a standard behavioral assay reported in the literature[34]. The standard Open field test is commonly used to assess locomotor, exploratory and anxiety like behavior in laboratory animals(rats/mice)[35]. The open field test is designed to examine responses of mice or rats to a new and unfamiliar environment (novel environment).Rodents demonstrate anxiety, fear and curiosity when placed in a new environment[36]. In response to the novel environment the rodents tend to explore the surrounding. The exploration capacity might be considered to be an index of anxiety although it is difficult to separate it from motor activity [36]. However, rodents are also fear to go to the open and illuminated space which is also a sign of anxiety. So the novel environment induces anxiety and fear in rodents which is clearly demonstrated by their rearing, grooming, defecation, locomotor, and so on. These parameters are well utilized to assess anxiety and fear in rodents. Inhibition of such behaviors is indicative of centrally acting depressant or sedatives [37]. **Table 6,7,8,9** represents the effect of different extracts of *Mimosa pudica* on various parameter of Open field test. Chloroform extract decreased movement of rodents in a dose dependent manner but could not reach significance; whereas clonazepam decreased movement significantly Clonazepam also decreased standing significantly. But extracts failed to exert any effect on standing and entrance in the center of the open field. The effect of higher dose of methanol 150 mg and dose of chloroform 150 mg on defecation was like clonazepam. The results show that the methanol and chloroform extract has not the ability to relieve stress and had an anxiolytic effect on the rodents like clonazepam did. On the other hand, lower dose of

methanol and both dose of chloroform decreased defecation which cannot support the previous results.

Neuropharmacological study (Forced swimming test): Forced Swimming test was performed to evaluate the effect of antidepressant effect of *Mimosa pudica* leaf extracts on mice. After investigation with leaf extracts of *Mimosa pudica* following data was observed **(Table 10).** Literature revealed that the FST was designed by Porsolt *et al*., 1978[22] as a primary screening test or antidepressants. It is still one of the best models for this procedure. This is a low-cost, fast and reliable model to test potential antidepressant treatments with a strong predictive validity. However, the low face and construct validities should not forbid the use of this model for neurophysiological studies. It has a great sensitivity with all the antidepressant classes and all the mechanisms of action of treatments could be determined, but clinical correlations should be considered very carefully. When rodents are forced to swim in a confined place, they tend to become immobile after vigorous activity (struggling). This stressful inescapable situation can be evaluated by assessing different behavioral strategies and immobility during the test could be an efficient adaptive response to the stress [38]. The development of immobility when the rodents are placed in an inescapable container of water reflects the cessation of persistent escape directed behavior. The CNS depressant effect of the extracts may be attributed to chemical constitute other than flavonoids and alkaloids because flavonoids are responsible for the decrease in immobile phase in the swim test[38] and so does alkaloid as well[39].

CONCLUSION

All three extracts showed antioxidant activity but pet-ether extracts showed highest antioxidant activity. After pharmacological studies with leaf extracts of *Mimosa pudica* for analgesic activity and neuro pharmacological investigation, plant leaf has significant analgesic activity and neuro pharmacological action.

Table 1: IC_{50} values of different extract in DPPH free radical scavenging assay

Sample/standard	IC_{50} (µg/ml)
Methanol	21.23
Ethanol	35.33
Pet-ether	15.02
Ascorbic acid	13.99

Table 2: Total antioxidant capacity of the different extracts of *Mimosa pudica*

Extract	Total Flavonoid Contents (mg/gm, Ascorbic Acid Equivalent
Methanol	67.6 ± 2.790
Ethanol	64.4 ± 5.223
Pet-ether	79.2 ± 4.722

Values are the mean of duplicate experiments and represented as mean ± SD

Table 3: Total phenol contents of the different extracts of *Mimosa pudica*

Extract	Total Flavonoid Contents (mg/gm), Gallic
Methanol	15.67 ± 3.578
Ethanol	10.73 ± 3.312
Pet-ether	26.556 ± 3.312

Values are the mean of duplicate experiments and represented as mean ± SD.

Table 4: Total flavonoid contents of the different extracts of *Mimosa pudica*

Extract	Total Flavonoid Contents (mg/gm, Quercetin Equivalent
Methanol	24.902 ± 2.268
Ethanol	25.94 ± 2.742
Pet-ether	26.556 ± 3.312

Values are the mean of duplicate experiments and represented as mean ± SD.

Table 5: Effect of different extracts of *Mimosa pudica* in acetic acid induced writhing test

Samples	Doses (mg/kg)	No. of Writhing	% of Inhibition
Control	10 ml/kg of 1% Tween 80	3.07 ± 0.77	-
Std (Diclofenac)	150	2.17 ± 0.90	29.32
Methanol	100	2.63 ± 1.88	14.33
	150	2.43 ± 0.27	20.84
Ethanol	100	2.3 ± 0.28	25.08
	150	2.56 ± 0.27	16.61

Number of writhing values are mean ± S.E.M., (n=6)

Table 6: Effect of different extracts of *Mimosa pudica* in Open Field test (Movement)

Samples	Doses (mg/kg)	-30 mins	+30 mins	+60 mins	+90 mins	+120 mins	+150 mins
Control		16.33±11.57	25.17±5.05	31.17±10.59	34.17±19.77	31.5±15.46	18.3±11.63
Clonazepam	2	4.33±3.09	24.5±5.06	27±5.97	22.83±6.04	24.17±3.29	16.33±7.50
Methanol Extract	100	5.33±2.43	18.33±7.43	23.36±7.36	21.16±8.66	25±3.36	28.33±6.82
	150	6.16±1.21	24.83±9.54	30.33±8.75	34±6.73	32.33±11.4	33.16±10.49
Ethanol Extract	100	0.66±0.74	4±1.15	3.16±1.86	3.16±1.86	4.16±1.34	3.83±1.46
	150	0.16±0.37	4.33±1.69	3.16±1.34	3.5±2.06	3.16±1.07	3±1.30
Chloroform Extract	100	2.66±0.74	20.83±9.28	16.83±8.76	16±4.47	10.5±6.42	12.83±4.25
	150	3.16±1.21	20.66±11.16	13.16±6.98	12.5±13.42	7.5±8.38	11.66±15.18

Values are mean ±S.E.M. (n=6)

Table 7: Effect of different extracts of *Mimosa pudica* in Open Field test (Standing)

Samples	Doses (mg/kg)	-30 mins	+30 mins	+60 mins	+90 mins	+120 mins	+150 mins
Control		0.17±0.37	2.83±2.19	3.5±1.98	3.83±2.19	2.83±2.27	2.17±1.57
Std(Clonazepam)	2	0.17±0.37	6.83±1.46	5±1	5.17±1.34	4.16±0.69	4.67±2.05
Methanol Extract	100	0.83±0.68	6.33±2.05	5.83±1.21	4±0.57	5.16±1.95	4.66±2.21
	150	0.16±0.37	3.5±0.95	4.33±1.37	4.5±1.25	4±0.81	4.5±1.89
Ethanol Extract	100	0.66±0.74	4±1.15	3.16±1.86	3.16±1.86	4.16±1.34	3.83±1.46
	150	0.16±0.37	4.33±1.69	3.16±1.34	3.5±2.14	3.16±1.06	3±1.29
Chloroform Extract	100	0.5±0.5	3±0.81	2.5±1.25	2.33±1.10	2.16±1.34	2.16±1.34
	150	0.16±0.37	3±1.63	2±1.39	2.5±2.14	1.66±1.06	1.83±1.21

Values are mean + S.E.M., (n=6)

Table 8: Effect of different extracts of *M. pudica* in Open Field test (Centre)

Samples	Doses (mg/kg)	-30 mins	+30 mins	+60 mins	+90 mins	+120 mins	+150 mins
Control		1±0	0.17±0.37	0.5±1.6	0.33±0.99	0.5±1.6	0.5±1.05
Std(Clonazepam)	2	1±0	0.17±0.37	0.33±0.47	0.83±0.37	0.17±0.37	0.5±0.5
Methanol Extract	100	0.66±0.74	0.16±0.37	0.33±0.47	0.16±0.37	0±0	0±0
	150	0.16±0.37	0.16±0.37	0.16±0.37	0.5±0.76	0.83±0.89	0±0
Ethanol Extract	100	0.66±0.74	0.33±0.74	0.33±0.47	0.16±0.37	0.33±0.74	0.33±0.74
	150	1.16±0.68	1.33±0.47	0.16±0.37	0±0	0±0	0.33±0.47
Chloroform Extract	100	1.33±1.37	0.5±0.76	0.16±0.37	0±0	0.16±0.37	0.33±0.74
	150	0.66±0.47	0.16±0.37	0±0	0.16±0.37	0±0	0.33±0.74

Values are mean ± S.E.M., (n=6)

Table 9: Effect of different extracts of *Mimosa pudica* in Open Field test (Stool)

Samples	Doses (mg/kg)	-30 mins	+30 mins	+60 mins	+90 mins	+120 mins	+150 mins
Control		0.83±0.68	0±0	0±0	0±0	0±0	1.67±1.21
Std(Clonazepam)	2	0.83±0.68	0±0	0±0	0±0	0.17±0.37	0.17±0.37
Methanol Extract	100	1.33±1.11	0.17±0.37	0.33±0.47	0±0	0.5±0.76	0.17±0.37
	150	0.5±0.76	0.33±0.47	0±0	0±0	0.33±0.47	0.33±0.47
Ethanol Extract	100	0.66±0.47	0.33±0.47	0±0	0±0	0.33±0.47	0.33±0.47
	150	1±1	0±0	0±0	0±0	0.17±0.37	0.17±0.37
Chloroform Extract	100	1.12±0.98	0.17±0.37	0.33±0.47	0±0	0.17±0.37	0.17±0.37
	150	1±1.14	0.17±0.37	0.17±0.37	0.33±0.47	0.67±1.49	0.17±0.37

Values are mean ± S.E.M., (n=6)

Table 10: Effect of different extracts of *Mimosa pudica* in swimming test

Samples	Doses (mg/kg)	Duration of Immobility (s)
Control	—	34.14 ± 3.15
Std (Imipramine)	10	38.25 ± 0.74
Methanol Extract	100	33.33 ± 6.86
	150	39.69 ± 6.12
Ethanol Extract	100	39.47 ± 6.02
	150	39.39 ± 1.97
Chloroform Extract	100	36.72 ± 6.65
	150	37.36 ± 5.46

Values are mean ±S.E.M. (n=6)

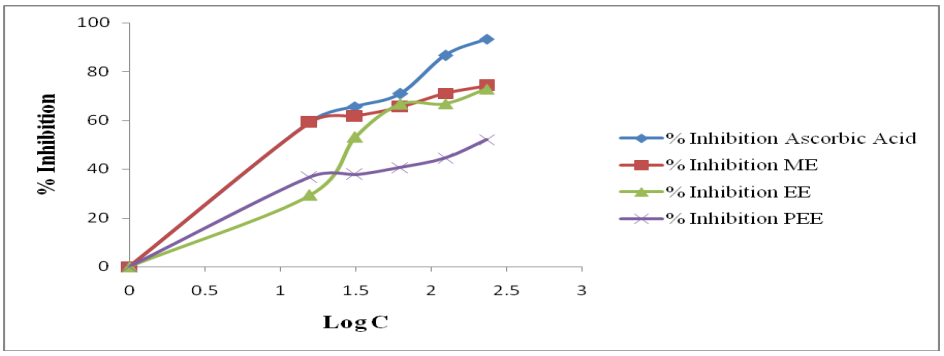

Figure 1: Comparative DPPH radical scavenging activity of extracts and Ascorbic acid

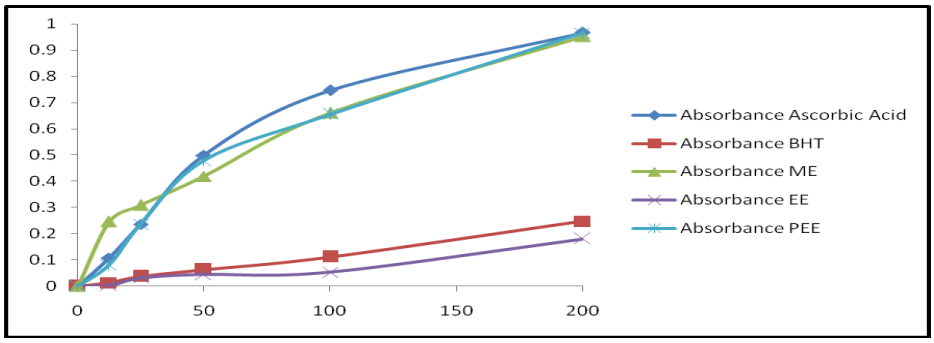

Figure 2: Comparative reducing power of extracts and Ascorbic acid & Butylated hydroxyl toluene (BHT)

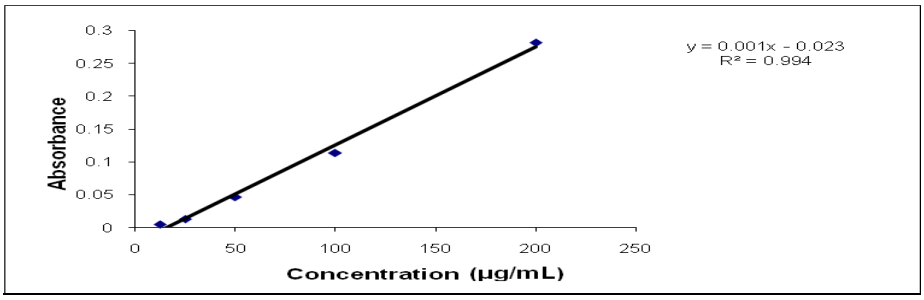

Figure 3: Calibration curve of ascorbic acid

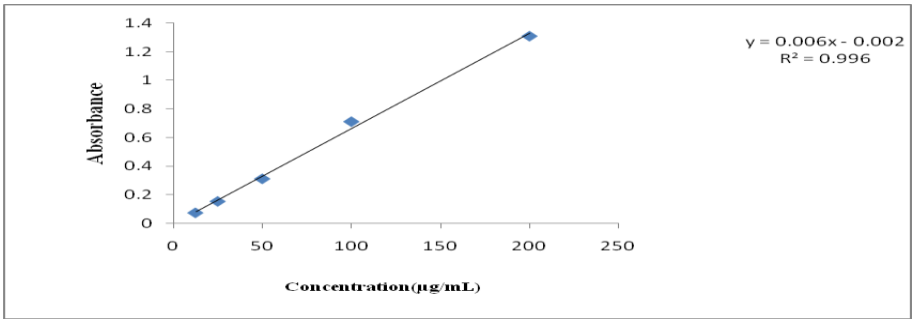

Figure 4: Calibration curve of Gallic Acid

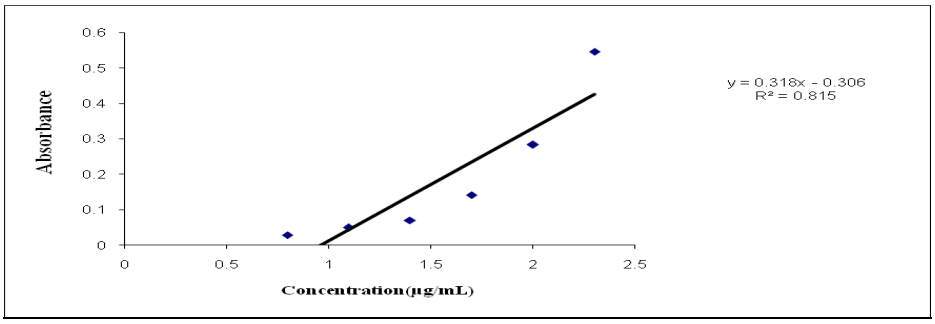

Figure 5: Calibration curve of Quercetin

REFERENCES

1. Ghani A. Medicinal Plants of Bangladesh with Chemical Constituents and uses. 2nd ed., Dhaka, Bangladesh; Asiatic Society: 2003.
2. Balakrishnan K, Sambandam S, Ramaswamy P, Mehta S, Smith KR. J Exp Anal Environ Epidemiol, 2004; 14: S14–S25.
3. Bum EN, Dawack DL, Schmutz M, Rakotonirina A, Herding P. Fitoterapia, 2004; 75(3-4): 309-314.
4. Chowdhury AS, Uddin R, Parvin NM. J App Pharm Sci, 2011; 1(6): 118-121.
5. Sharma PC, Yelne, MB, Dennis TT. Database on medicinal plants Govt. of India, Janakpuri, Delhi; 2001.
6. Joymma S, Rao SG, Hrikeshvan HG, Aroop AR, Kulkarni DR. Indian J. of Exp Bio, 1990; 28(3): 237-240.
7. Akinsinde KA, Olukoya DK. J Diarrhoeal Dis Res, 1995; 13(2): 127-129.
8. Moline J, Bukharovich IF, Wolff MS. Med Hypoth, 2000; 55(4): 306-309.
9. Valsala S, Karpagaganapathy PR. Phyto Res, 2002; 16(2): 190-192.
10. Jamuna Devi Y, Devi SP. Singh HT. Indian Drugs, 2001; 38(8): 414-417.
11. Amalraj T, Ignacimuthu S. Fitoterapia, 2002; 73(4): 351-352.
12. Ngo Bum E, Dawack DL, Schmutz M, Rakotonirina A, Portet C. Fitoterapia, 2004; 75(3-4): 309.
13. Girish KS, Mohanakumari HP, Nagaraju S, Vishwanath BS, Kemparaju K. Fitoterapia, 2004; 75(3-4): 378-80.
14. Winter CA, Risley EA, Nuss GW. Exp BioMed, 1962; 111: 544-547.
15. Braca A, Tommasi ND, Bari LD, Pizza C, Politi M, Morelli I. J Nat Prod, 2001; 64: 892-895.
16. Apak R, Guclu K, Ozyurek M, Karademir SE. J Agri and Food Chem, 2004; 52: 7970–7981.
17. Prieto P, Pineda M, Aguilar M. Analy Biochem, 1999; 269(2): 337-341.
18. Singleton VL, Orthofer R, Lamuela-Raventos RM. Methods in Enzym, 1999; 299: 152–178.
19. Wang SY, Jiao H. J Agric Food Chem, 2000; 48: 5672-76.
20. Koster R, Anderson M, DeBeer E. Fed Proc, 1959; 18: 412–418.
21. Gupta BD, Dandiya PC, Gupta ML. Jpn J Pharmacol, 1971; 21: 293.
22. Porsolt RD, Bertin A, Jalfre M. Eur J Pharmacol, 1978; 51: 291–294.
23. Ahmed F, Hossain MH, Rahman AA. J. Oriental Pharm Exp Med, 2006; 6: 344-348.
24. Ronaldo AR, Mariana LV, Sara MT, Steve P, Ferreira SH, Fernando QC. Eur J Pharmacol, 2000; 387: 111-18.
25. Deraedt R, Jouquey S, Delevallee F, Flahaut M. Euro J Pharmacol, 1980; 61: 17–24.
26. Lcvine ND. J of Protozoo, 1984; 31: 94-8.
27. Dhara AK, Suba V, Sen T, Pal S, Nag Chaudhuri AK. J Ethano pharma, 2000; 72: 265-268.
28. Voilley N. Curr Drugs Targets Inflam Aller, 2004; 3: 71-79.
29. Hall CS. J Comp Psychol, 1936; 22: 345–352.
30. Blizard DA. Behav Genet, 1981; 11: 469–489.
31. Van der Staay FJ, Kerbusch S, Raaijmakers W. Behav Genet, 1990; 20: 51– 62.
32. Sandnabba NK. Behav Genet, 1996; 26: 477– 488.
33. Crabbe JC, Wahlsten D, Dudek BC. Science, 1999; 284: 1670–1672.
34. Walsh RN, Cummins RA. Psychol Bull, 1976; 83: 482–504.
35. Datusalia AK, Kalra P. J Helth Sci, 2008; 54(5): 544-550.
36. Brown S, Birtwistle J, Roe L. Psyc Medi, 1999; 29: 697 -701.
37. Porsolt RD, Bertin A, Jalfre M. Eur J Pharmacol, 1978; 51: 291–294.
38. Butterweck V, Jurgenliemk G, Nahrstedt A. Plnta Med, 2000; 66: 3-6.
39. Silva GN, Martins FR, Matheus ME. J Ethno Pharmacol, 2005; 100: 254-259.