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PHYTOCHEMICAL EVALUATION AND CNS ACTIVITIES OF *TRADESCANTIA SPATHACEA* ON ALBINO MICE

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Abstract:

Depression is considered as affective mood disorder which is characterized by change in mood, lack of confidence etc. depression is the most prevalent disorder and the symptoms associated with depression changes the neurotransmitter levels in brain such as norepinephrine, seritonine and dopamine. Tradescantia spathacea is the plant selected to be used as a test drug in experimental animals. After selecting the plant, it has been dried and powdered, which was futher extracted using methanol as a solvent in a soxhlet apparatus. The evaluation of CNS activity has been done on mice of four groups by following standard procedures with the help of experimental methods such as Muscle Relaxant Property By Rotarod Apparatus, Anti Anxiety Effect By Elevated Plus Maze Apparatus and Anti Depressant Activity By Using Actophotometer to evaluate the potency of the drug and to know its activity. After comparing the test drug (Diazepam) with standard (Plant extract), results have shown that the extract has CNS activity. Therefore, it is concluded that Tradescantia spathacea extract has strong CNS activity on mice.

Key Words: *seritonine, dopamine, Tradescantia spathacea, Anti-Depressant Activity, and standard drug (Diazepam).*

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INTRODUCTION:

Anti-depressant Activity

Depression is considered as affective mood disorder which is characterized by change in mood, lack of confidence, lack of interest in surroundings, has been estimated to affect 21% of world's population and it may range from mild to severe depression, which is called as psychotic depression (WHO, 1998)[1]. According to the World Health report, approximately 450 million people suffer from a mental or behavioral disorder (WHO, 2001).

Today depression is the leading cause of suicides. It is to be estimated that 3000 people lost their lives in each day and 1 million people lost their lives yearly due to suicide (WHO, 2002)[2]. Depression is the most prevalent disorder and the symptoms associated with depression changes the neurotransmitter levels in brain such as norepinephrine, serotonin and dopamine (Gold et al., 1988).

Antidepressants are drugs used for the treatment of major depressive disorder and other conditions, including dysrhythmia, anxiety disorders, obsessive compulsive disorder, eating disorders, chronic pain, neuropathic pain and, in some cases, dysmenorrhea, snoring, migraines, attention-deficit hyperactivity disorder (ADHD), substance abuse and sleep disorders. They can be used alone or in combination with other medications but only when prescribed.

Antidepressants that belong to the same class of antidepressant produce similar side effects. Antidepressants may cause withdrawal symptoms if abruptly discontinued. Withdrawal symptoms include nausea, vomiting, dizziness, headache, irritability, sleep disturbance, nightmares, psychosis, and seizures.

All adolescents anti-depressants have a warning about use in children and. Antidepressants increased the risk of suicidal thinking, and suicidal behavior in short-term studies in children and adolescents with depression and other psychiatric disorders. Anyone considering the use of antidepressant in a child or adolescent must balance this risk of suicide with the clinical need for the drug. Patients who are started on therapy should be closely observed for clinical worsening, suicidal thoughts or unusual changes in behavior.

Classification of Antidepressants

1) Tricyclic antidepressants (TCAs)
2) Tetracyclic antidepressants.
3) Selective serotonin re-uptake inhibitors (SSRIs)
4) Serotonin and norepinephrine re-uptake inhibitors (SNRIs)
5) Serotonin receptor modulators (SRMs)
6) Monoamine oxidase inhibitors (MAOIs)
7) Lithium Salts

Indications mainly relieve the symptoms of depression or bipolar disorder such as depressed mood, worthlessness, lack of motivation or concentration.

Antidepressants are also used for anxiety disorder, phobic disorder (such as social phobia or agoraphobia), panic attack, obsessive-compulsive disorder, bulimia nervosa, nocturnal enuresis, chronic pain, neuropathic pain and post-traumatic stress disorder.

Mechanism of Action

According to the monoamine hypothesis of depression postulates a deficiency in serotonin or norepinephrine neurotransmission in the brain. Most of the currently used antidepressants work by slowing the removal of both norepinephrine and serotonin from the brain, thus increasing the availability of these neurotransmitters. As a result, they are efficacious for patients whose depression is caused by the imbalance of either norepinephrine or serotonin. Selective serotonin reuptake inhibitors

1. In common with the three better known SSRIs, fluoxetine, paroxetine and sertraline, both citalopram and fluvoxamine have a selective effect on the serotonin reuptake pump. This causes an initial increase in serotonin only at the cell body and the dendrites, not at axon terminals
2. The immediate consequence is to inhibit the rate of firing of serotonin neurons (and the release of serotonin) by an actionat $5HT_{1A}$ somato-dendritic auto receptors.
3. Longer-term exposure to serotonin eventually causes down regulation of these $5HT_{1A}$ auto receptors and disinhibition of serotonin release at axon terminals.
4. The delay in producing the increase in serotonin at the terminals is usually taken as the reason for the delayed onset of action of the SSRIs.
5. The increased release of serotonin at the axons, in the presence of an inhibited serotonin reuptake pump[3,4] increases availability of serotonin to postsynaptic serotonin receptors. These receptors may eventually down regulate.

6. The down regulation of postsynaptic serotonin receptors also occurs during long-term treatment with

tricyclic antidepressants and monoamine oxidase inhibitors.

7. For some SSRIs, chronic administration is also associated with a down regulation of postsynaptic B_1 adrenoceptors, but this has not been observed for citalopram, fluoxetine or fluvoxamine. While this effect is common to other antidepressants, including nefazodone and venlafaxine, it may not be necessary for clinical efficacy. The effect of SSRIs on serotonin neurotransmission may be sufficient to explain their antidepressant effects.

8. The increased availability of serotonin at serotonin receptors in the central nervous system and elsewhere can explain many of the adverse effects of this class of medication. Stimulation of $5HT_3$ receptors is probably responsible for nausea, gastrointestinal discomfort, diarrhoea and headache, which often occur at the start of treatment.

9. Similarly, agitation, akathisia, anxiety, panic attacks, insomnia and sexual dysfunction may be related to an action at $5HT_2$receptors. Sexual dysfunction may also be due to disinhibition of the descending serotonin pathway from the brain stem through the spinal cord to neurons mediating spinal reflexes such as ejaculation and orgasm. The increased serotonin release inhibits sexual functioning.

10. *Venlafaxine:* Like the SSRIs, venlafaxine has acute pharmacological effects on the reuptake of serotonin by presynaptic nerve terminals. It has a simultaneous effect on noradrenaline reuptake and some weak effects on dopamine reuptake

11. The combination of the effects on the reuptake mechanisms appears to be responsible for the antidepressant action of the drug.[5]

12. The reuptake effects of venlafaxine are dose dependent. At low doses (<150 mg/day), the drug acts like the SSRIs. At intermediate to high doses, the additional effects on noradrenaline reuptake become important.

13. In this respect, venlafaxine can be regarded as analogous to the older tricyclic antidepressants, with the exception that down regulation of postsynaptic b1 receptors occurs following single and repeated doses of venlafaxine (tricyclics cause b1adrenoceptor down regulation only after repeated doses).

14. A possible clinical correlate of this pharmacological effect is a faster onset of action of venlafaxine, although this has not been systematically demonstrated in appropriately designed studies.

15. *Venlafaxine* has little `in vitro' affinity for muscarinic cholinergic, histamine H_1 and adrenergic receptors, suggesting a more favourable adverse effect profile when compared to tricyclic antidepressants. Nausea, agitation, sexual dysfunction and insomnia at low doses of venlafaxine are probably mediated by effects on postsynaptic serotonergic receptors. At intermediate to high doses, additional adverse effects such as raised blood pressure and headache are observed in some patients. These effects are probably due to an action on adrenergic receptors.

16. *Nefazodone: It* has a unique pharmacological effect. It acts as a potent and selective antagonist of postsynaptic $5HT_{2A}$receptors. In addition, there is a moderate effect on presynaptic reuptake of both serotonin and noradrenaline. Both actions of the drug appear to be necessary for its clinical effect, but $5HT_{2A}$ antagonism is probably the main action[6]. Chronic administration of nefazodone results in a down regulation of cortical $5HT_{2A}$ receptors as well as b1adrenoceptor down regulation. Together, these actions of the drug are thought to increase serotonergic neurotransmission particularly at postsynaptic $5HT_{1A}$ receptors.

17. In vitro receptor binding studies show that nefazodone has little or no affinity for a range of other receptors including muscarinic cholinergic, histamine H1, GABA-A and dopamine D_1 and D_2 receptors. These data suggest that the drug is likely to lack some of the adverse effects common to tricyclic antidepressants. Blockade of $5HT_2$ receptors probably accounts for some of the adverse effects of nefazodone including somnolence, asthenia and the rare event of visual streaking (palinopsia).

18. Formation of a metabolite, m-chlorophenylpiperazine (mCPP), which acts as a non-selective agonist at 5HT2A, 2C and 5HT3 receptors, accounts for a number of adverse effects of nefazodone. Systemic exposure to mCPP is low (=<8%) under most circumstances, but it may be substantially increased in patients with a genetic deficiency of cytochrome $P_{450}{}^2D_6$ or when prior SSRI administration has inhibited this isoenzyme.

Pharmacology

The pharmacology of antidepressants is not entirely clear.

1. The earliest and probably most widely accepted scientific theory of antidepressant action is the monoamine hypothesis (which can be traced back to the 1950s), which states that depression is due to an imbalance (most often a deficiency)of the mono amine neuro transmitters (serotonin, norepinehrine and dopamine)[7]

2. It was originally proposed based on the observation that certain hydrazine anti-tuberculosis agents produce antidepressant effects, which was later linked to their inhibitory effects on monoamine

oxidase, the enzyme that catalyses the breakdown of the monoamine neurotransmitters.
3. All currently marketed antidepressants have the monoamine hypothesis as their theoretical basis, with the possible exception of agomelatine which acts on a dual melatonergic-serotonergic pathway. [8]
4. Despite the success of the monoamine hypothesis it has a number of limitations: for one, all monoaminergic antidepressants have a delayed onset of action of at least a week; and secondly, there are a sizeable portion (>40%) of depressed patients that do not adequately respond to monoaminergic antidepressants.[9][10]
5. Further evidence to the contrary of the monoamine hypothesis are the recent findings that a single intravenous infusion with ketamine, an antagonist of the NMDA receptor a type of glutamate receptor produces rapid (within 2 hours), robust and sustained (lasting for up to a fortnight) antidepressant effects.
6. To overcome these flaws with the monoamine hypothesis a number of alternative hypotheses have been proposed, including the glutamate, neurogenic, epigenetic, cortisol hypersecretion and inflammatory hypothesis.[9-12]

Side effects of different antidepressants are as follows:

1. Tricyclic & tetracyclic antidepressants: Dizziness, headache, sweating, tremor, somnloence, palpitation, dry mouth, constipation, blurred vision, difficulty passing urine, and orthostatic hypotension. Other less-common adverse effects include seizure, liver dysfunction, ECG changes and abnormal blood count.
2. SSRIs: Nausea, vomiting, gastrointestinal discomfort, somnolence, dry mouth, tremor, headache, sweating, sexual dysfunction and weight loss, etc. Occasionally, some patients may experience excitement, anxiety, insomnia, restlessness or seizure.
3. SNRIs: Similar to SSRIs. May cause hypertension at high doses.
4. SRMs: Somnolence, tremor, headache, constipation, weight gain and hypotension. Some patients may develop seizure, abnormal liver function tests and blood disorder, etc.
5. MAOIs: Dizziness, headache, nervousness, gastrointestinal disturbance, etc. May interact with tyramine-rich food or drinks, as a consequence inducing sweating, vomiting and hypertensive crisis. E.g. pigeon, alcoholic beverages, cheese, chicken and beef liver, chocolate or cheese, etc.
6. Lithium salts: Bitter taste, dry mouth, tremor, polyuria, fatigue and weight gain. Other less-common side effects include hyperthyroidism, hypothyroidism, ECG changes, raised anti-diuretic hormone concentrations, renal failure or leucocytosis.

Commonly-used antidepressants

1. TCAs: Amitriptyline, Imipramine, Clomipramine, Nortriptyline, Desipramine, Dothiepin, Doxepin, Trimipramine, Melitracen.
2. Tetracyclic antidepressants: Mianserin, Maprotiline.
3. SSRIs: Fluoxetine, Paroxetine, Sertraline, Fluvoxamine, Citalopram, Escitalopram.
4. SNRIs: Venlafaxine/ Venlafaxine XR.
5. MAOIs: Moclobemide, Phenelzine, Isocarboxazid, Tranylcypromine.
6. Lithium salts: Lithium carbonate.

Cautions when taking antidepressants Patients with or have a history of suicidal behavior should avoid taking SSRIs. Do not expect the antidepressants to work right away.

Aim and Objectives of the Study

The main objective of the present work was to evaluate and ensure the CNS activity of from *Tradescantia spathacea* plant against various models.

Plan of work with following objectives.

1. Identification and authentication of plant *Tradescantia spathacea* by a renowned taxonomist.
2. Collection of *Tradescantia spathacea* and its shade drying.
3. Preliminary phytochemical screening for the detection of various phytochemical constituents.
4. CNS activity of from *Tradescantia spathacea* plant against various models.
   A. antidepressant activity - actophotometer
   B. skeletal muscle relaxant - rotarod apparatus
   C. Anti anxiety activity - elevated plus maze

Materials and Methods
Materials
List of equipments:
- Heating mantel.
- Hot air oven.
- Camera Lucida.
- Compound Microscope.
- Tail suspension and forced swim apparatus.

List of chemicals:
- Ethanol 95%.

- Water for injection.
- 1% ferric chloride solution.
- 1% aqueous sodium chloride solution.
- Sulphuric acid.

Methods

Collection and Authentication of Plant Material:
The plant *Tradescantia spathacea* was collected in the month of Feb 5th 2016 from medicinal gardens of Geethanjali college of Pharmacy in Cheeryal, Hyderabad, Telangana State, India. The plant specimen was identified by Prof. Rana Kausar, Dept. Of Botany, Osmania University, Hyderabad, Telangana State, Voucher No: 0364. *[Annexure-1]*

Preparation of Extract
The powdered plant (60gm) was extracted at ambient temperature (50 - 60ºC) successively with Methyl Alcohol. The powder was taken in a Soxhlet apparatus with Methanol as Solvent is kept undisturbed for 72hrs. The crude extract from the soxhlet RBF was further air dried to get a fine powdered extract. The extract was subjected to preliminary phytochemical investigation. And subjected for Anti-depressant activity.

Preliminary Phytochemical tests
We have performed the following preliminary phytochemical tests and identify the chemical groups present in the plant extract.

Tests for tannins
Ferric chloride test: Extracts mixed with 1 % ferric chloride solution gives blue, green or brownish green colour.

Tests for flavonoids
Sodium hydroxide test: About 5 mg of the compound is dissolved in water, warmed and filtered. 10% aqueous sodium hydroxide is added to 2 ml of this solution. This produces a yellow coloration. A change in color from yellow to colorless on addition of dilute hydrochloric acid is an indication for the presence of flavonoids.

Tests for steroids
Salkowaski Test: Chloroform solution of the extract when shaken with concentrated sulfuric acid and on standing yields red colour.

Tests of Alkaloids
Hager's Test (Saturated solution of picric acid): The acid layer with Hager's reagent gives yellow precipitate.

Tests for Glycosides
Sodium hydroxide reagent: Dissolve a small amount of alcoholic extract in 1 ml water and add sodium hydroxide solution. A yellow colour indicates the presence of glycosides.

Detemination of Transverse Section, Stomatal index and Stomatal number

Transverse Section of Leaf

Procedure:
- The fresh leaves are thoroughly washed under tap water to remove any external dirt or mud.
- Any one leaf is taken and is cut into small pieces; these pieces are placed in a watch glass containing water.
- From these pieces one or two pieces are isolated and heated in a beaker containing methanol solution.
- Due to this heating, the leaves tend to become colorless by losing their pigments.
- The discolored leaf is taken and T.S of this leaf is done is using a blade.
- The sliced part of leaf is then placed over a glass slide.
- A drop of glycerine is added to the slide and is closed with a cover slip.
- The slide is washed under tap and dried using a filter paper.
- This slide is focused under microscope to observe the arrangement of epidermal layers and their cells.

Stomatal Identification

Procedure:
- To identify the type of stomata present in the leaf of Rhoeo discolor, the upper and lower epidermis of the leaf are separated using the orthodox techniques.
- The separated layers are now heated with methanol to remove excess of water content.
- The Upper/lower epidermal tissue layer is sliced into required shape after treatment with methanol and is placed over a glass slide.
- The slide with leaf tissue is now stained using safranin for the purpose of clear identification of stomata under microscope.
- The slide is covered with a coverslip and is focused at 10x magnification to identify any presence of stomata.
- The identified stomata are magnified to 45 x to clearly understand the type of stomata present in the plant leaf.

Stomatal Index and Stomatal Number

Procedure:
- The upper and lower epidermal regions are separated using common techniques
- The separated layers are heated using methanol and are isolated, treated with safranin and focused under microscope.

- Draw a square of known dimensions by means of stage micrometer & camera lucida on drawing paper.
- Replace the stage micro meter by cleared leaf preparation, focused under same magnification and trace the epidermal cells & stomata by looking through it.
- Examine successive adjacent fields until about 400 cells are counted & calculate the stomatal number using formula.

$$\textbf{Stomatal index} = \frac{S}{E+S} X\ 100$$

Experimental Animals:

Albino mice (40-50 g) of either sex, total of 26 animals were used (grouped as 6 animals in one group i.e., 4 groups). All the animals were approved by the ethics approval committee of the institute (Reg No: 1648/PO/A/12/CPCSEA). The animals were fed with commercially available rat pelleted diet. Water was allowed ad libitum under strict hygienic conditions. The animals were housed at room temperature in a well ventilated animal house under 12hr light/dark cycles.

CNS Activity

Depression is one of the most common psychiatric disorders. The drugs that are used in the treatment of depression are called as anti-depressants.

There are many experimental methods that are used in pharmacological studies to identify the CNS activity of a drug. Some of them can be stated as follows.

1. Antidepressant activity - actophotometer
2. skeletal muscle relaxant - rotarod apparatus
3. Anti-anxiety activity - elevated plus maze

Anti-depressant activity – Actophotometer:-

- Weigh the animals and number them.
- Turn the equipmet check and make sure that all photo cells are woking for accurate readings and place individually each mouse in the activity cage for 10minutes. Inject chlorpromazine (1ml/100gm) and after 30mins reset each mouse for activity scores for 10mins.
- Note the difference in activity before and after chlorpromazine injection. Calculate percentage decrease in motor activity.

$$\%\ \textit{decrease in locomotor activity} = \frac{Before - After}{Before} x\ 100$$

Skeletal Muscle Relaxant - Rotarod Apparatus

- Weigh the animal and number them.
- Turn on the rotarod. Select an appropriate speed (20-25rpm).
- Place the animal one by one on rotating rod. If the rod is divided into several compartments. One place more than one mouse at a time.
- Note down the fall off time when the mouse falls from the rotating rod. A normal untreated mouse generally falls within 3-5mins.
- Inject diazpem to all the animal after 30mins. Repeat the experiment as done in step-3. Note the fall off time
- Compare the fall off time of the animals before and after diazepam.

Anti anxiety activity - Elevated plus maze

- Weigh and number the animal. Divide them into two groups containing 4-5 animals one group is used as control and other for drug treatment.
- Place animals individually in the centre of the maze, head facing towards open arm and then start the stop watch. Note the following parameters for 5mins.
  - First preference of mice to open (or) closed arm.
  - Number of entries in open and enclosed arm (An armentary is defined as entry of four paws into arm).
  - Average time each animal spends in each arm.

$$\textbf{Average time} = \frac{\textbf{total duration in arm}}{\textbf{number of entries}}$$

- Inject diazepam to test group. After 30mins place the animal individually in centre of maze and all parameters described under step-2.
- Compare the preference of an animal to open (or) enclosed arm. Average time spent in an open arm, number of entries in a open arm in each group.

RESULTS AND DISCUSSION:

Tests for Identification

Results of phytochemicals analysis

Name of identification test	Type of chemical group	Result with plant extract
Ferric chloride Test	Tannins	-
Sodium Hydroxide Test	Flavonoids	+
Salkowaski Test	Steroids	-
Hager's Test	Alkaloids	+
Sodium Hydroxide reagent Test	Glycosides	+
Foam Test	Saponins	-

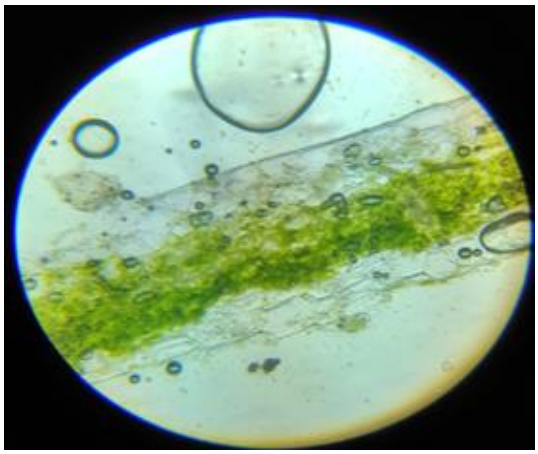
+ Presence ; - Absence

Observation

The plant *Tradescantia spathacea* was found to have the chemical constituents Alkaloids, Flavonoids & Glycosides.

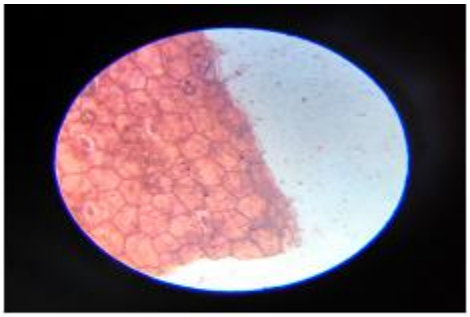
Transverse Section of Leaf

Observation: The Transverse section of plant was performed and the observation is shown in the fig

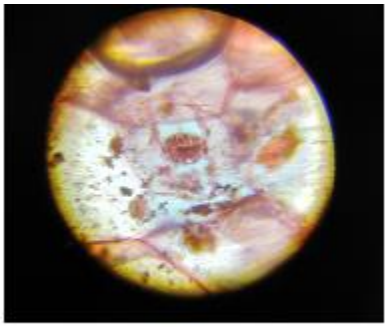


The Transverse Section Of Leaf With Upper, Lower Epidermal And Middle Lamina Regions of Leaf.

Stomatal Identification:



Epidermis Of Leaf Showing Stomatal Presence



Plant With Parasitic Stomata

3(a) Epidermal Layer of Leaf, 3(b) Parasitic Stomata of Tradescantia Leaf.

Observation

The plant was found to contain parasitic stomata (i.e., surrounded by two guard cells)

Stomatal Number:

Calculations:

Square 1

No. of Stomata: 5

No. of Epidermal cells: 26

Square 2

No. of Stomata: 2

No. of Epidermal cells: 21

Square 3

No. of Stomata: 6

No. of Epidermal cells: 30

Square 4

No. of Stomata: 6

No. of Epidermal cells: 18

$$\textbf{Stomatal Number} = \frac{5 + 2 + 6 + 6}{4} = 4.75$$

Stomatal Index

$$\text{Stomatal index} = \frac{S}{E+S} X\ 100 = \frac{19}{95+19} X\ 100 = 16.66$$

Where, S is the No. of Stomata, E is the No. of Epidermal Cells.

Observation

The Stomatal index of the plant was found to be ***16.66***.

The Average Stomatal number was found to be ***4.75***

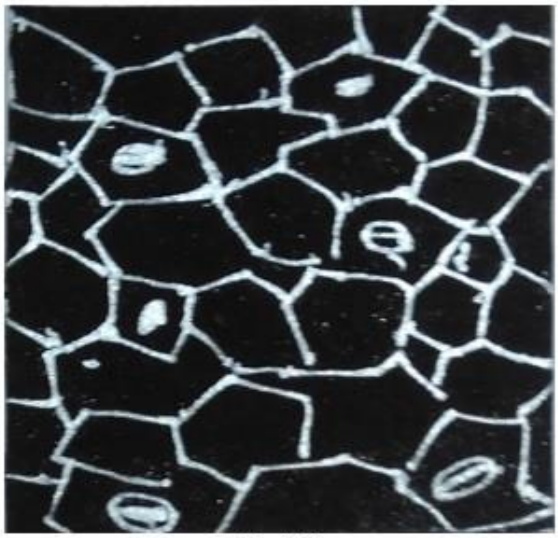


Fig. 4(a)

Fig.4(b)

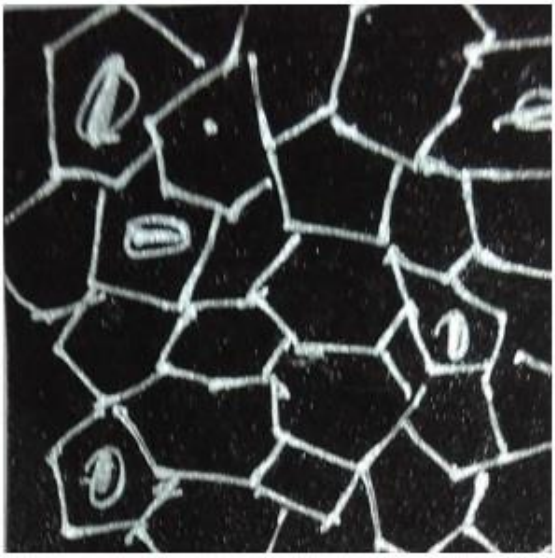


Fig.4(c)

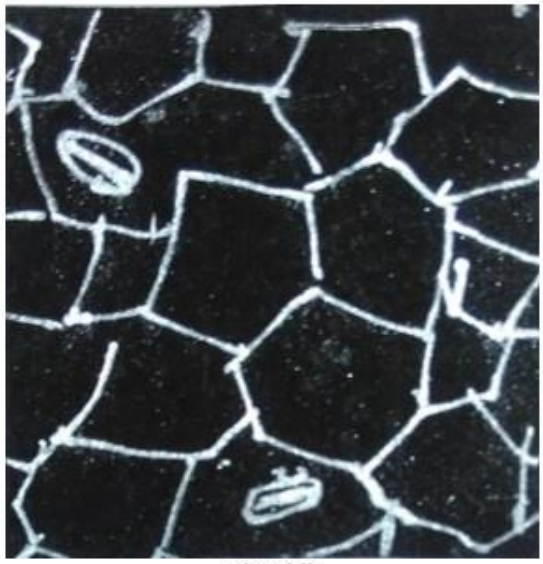


Fig.4(d)

4 (a), 4(b), 4(c), 4(d), Showing Stomatal squares Drawn using Camera Lucida

CNS Activities:-

Anti-anxiety Activity - Elevated plus maze

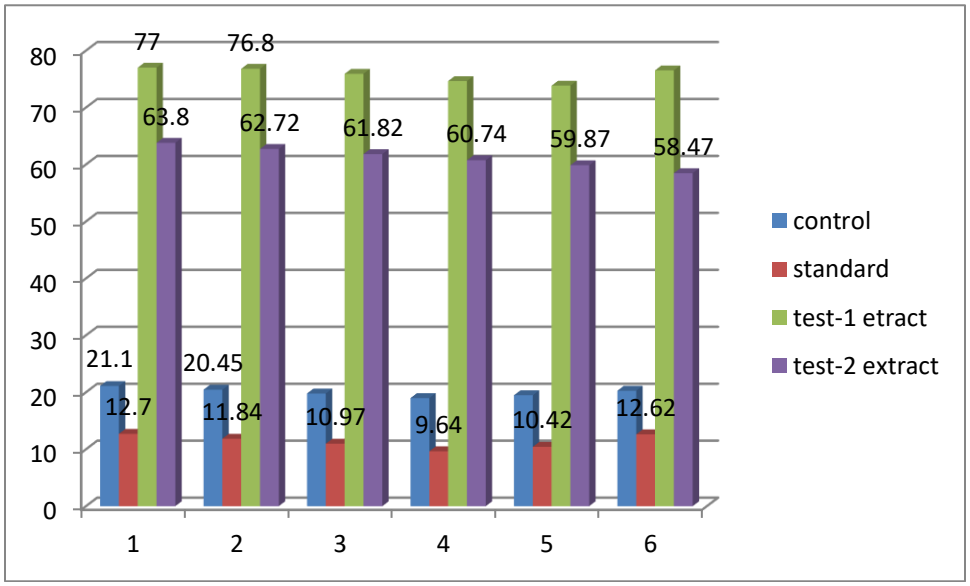
Effect of tradescantia spathaceae on immobility period (secs) of mice using elevated plus maze apparatus.

Groups	drug	Dose(mg/ml)	Closed arm	No of entries in open arms	Open arm	Avg time spent in open arm
control	saline	__	8.71±1.01	6 time	90.3	15.05
	diazepam	10mg	85.20±3.37	4 times	10.33	2.582
standard	plant extract	250mg	85.18±3.06	5 times	11.33	2.832
test -1 extract test – II extract	plant extract	500mg	30.65±1.28	6 times	64.33	16.082

Antidepressant Activity – Actophotometer

Effect of Tradescantia Spathaceae on Immobility Period (Secs) of Mice Using Actophotometer

Groups	drug	Dose mg/ml	Basal response	30 mins	% decrease in time
Control	Saline	__	934	20.01±0.75	21.1%
Standard	Diazepam	10mg	471	11.36±1.23	12.7%
Test-I Extract	Plant extract	250mg	487	75.79±1.28	77%
Test-Ii Extract	Plant extract	500mg	373	61.23±1.94	63.8%

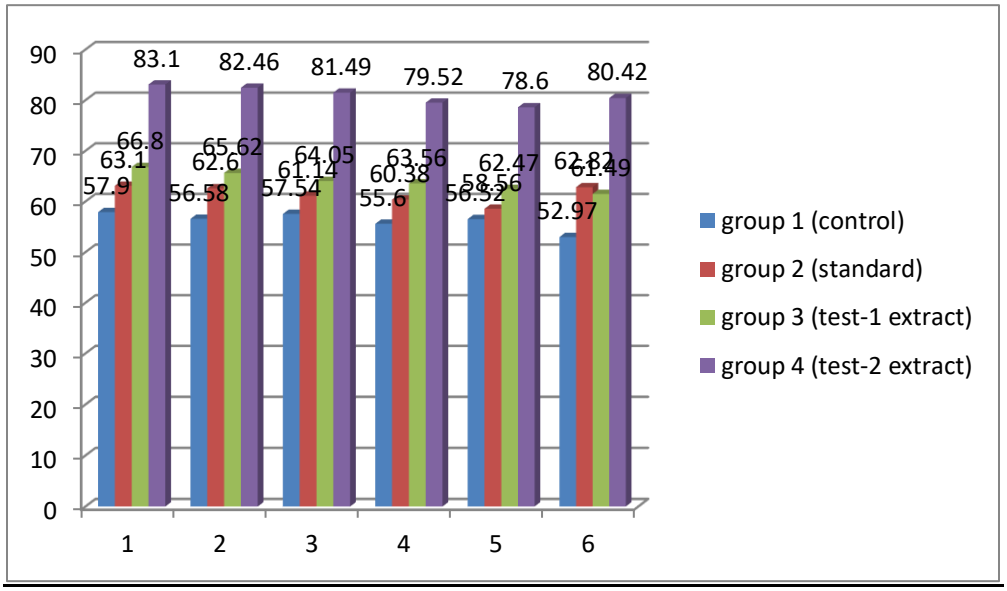


Graph of Antidepressant Activity – Actophotometer Table-2

Skeletal Muscle Relaxant - Rotarod Apparatus

Effect of Tradescantia Spathaceae on Immobility Period (Secs) of Mice Using Rotarod Apparatus

Groups	drug	Dose mg/ml	Basal response	30 mins	% decrease in time
Control	Saline	__	7mins.18secs	20.01±0.75	57.9%
Standard	Diazepam	10mg	8mins.24secs	11.36±1.23	63.1%
Test-I Extract	Plant extract	250mg	9min.04secs	75.79±1.28	66.8%
Test-Ii Extract	Plant extract	500mg	3mins.02secs	61.23±1.94	83.1%



Graph Of Skeletal Muscle Relaxant - Rotarod Apparatus Table-3

DISCUSSION:

The prevention and management of stress disorders remains a major clinical problem. Hence it is very important to address these problems and find effective remedies. Though several drugs are available, all are associated with some limitations and there is an urgent need for alternative medications for these disorders [43]. To treat a wide variety of nervous disorders, *Tradescantia spathacea* is a known choice for more than hundred years. In this work, it was demonstrated that the administration of different doses of the methanolic extract of *Tradescantia spathacea* in rats and mice was able to induce antidepressant effects. In CNS activities, the extract can decrease the immobility time in rats with mild sedative effect. It was found that *Tradescantia spathacea* can produce antidepressant like activity at a dose of 100mg and 200mg/kg body weight in a dose dependent manner. The decrease in the immobility time is accompanied with the increase in swimming time. Previous demonstrated that many neurotransmitters were involved in the pathophysiology of depression. Numerous studies have demonstrated that antidepressant drugs such as Thioxetine, Imipramine stimulated the action of serotonin and act by inhibiting the reuptake of biogenic amines in CNS. These drugs were widely used as antidepressant drugs and agreed with studies in animal models, such as CNS activites[44]. An

antidepressant drugs reduce the exploratory behaviour depending upon the concentration. At present, the study revealed that the methanolic extract of Rhoeo discolor significantly reduces the number of head dippings and number of line crossings were the indicator of exploratory behavior. The findings from the present investigation indicate that *Tradescantia spathacea* possesses significant antidepressant activity as shown by its mitigating effects on different experimentally induced stress models in rats and mice.

CONCLUSION:

The phytochemical analysis has revealed that the plant *Tradescantia spathacea* consists of phytochemical constituents which are Alkaloids, Flavonoids & Glycosides. The study of Antidepressant activity- actophotometerskeletal muscle relaxant - rotarod apparatus Anti-anxiety activity - elevated plus maze have confirmed that the *methanolic* extract of *Tradescantia spathacea* has the CNS activity as it significantly reduces the immobility time and increases the exploratory behavior during depression in animal models.

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