Original Article

Neuroprotective Effect of *Bauhinia variegata* Leaf Extract against Colchicine: An Experimental Study on Cognitive Dysfunction and Biochemical Alterations in Mice

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ABSTRACT

Objectives: To study the protective role of *Bauhinia variegata* ethanolic leaf extract induced by colchicine cognitive dysfunction and the damage of oxidative in Swiss albino mice; to examine the neuroprotective consequence of *Bauhinia variegata* by conducting behavioral memory tests and to estimate the biochemical parameters by using brain homogenate. **Methods:** The research was conducted on colchicine induced model for a period of 28 days (4 weeks). Behavioral research was conducted using Elevated plus maze, Passive evasion Paradigm, Morris water maze and Actophotometer; thus biochemical parameters of brain homogenate such as acetyl cholinesterase (AChE), total protein, lipid peroxidation (MDA) and glutathione (GSH) were estimated. Swiss albino mice (48) were distributed into eight sets, each constituting 6 mice. Class I got carboxy methyl cellulose 1 per cent w/v. Group II piracetam was administered i.p. (200 milligrams / kg). Group III obtained 1 mg / kg of colchicine i.c.v. Group IV and V were provided with 200 and 400 mg / kg of ethanolic *Bauhinia variegata* leaf extract. Group VI was administered Piracetam (200 mg / kg, i.p.) to mice each day for 28 days continuously. Colchicine 1 mg / kg, i.p., at 60 min after 28th day piracetam injection was injected. Group VII and VIII received 200 and 400 mg / kg oral ethanolic *Bauhinia variegata* leaf extract for 28 consecutive days, and colchicine (1 mg / kg) was injected i.c.v at 90 min after extract administration on the 28th day. All the data and information were examined and analyzed through the use of ANOVA which is One-way then by a check by Tukey's test. **Results:** The *Bauhinia variegata* leaf extract showed substantial reduction in raised plus maze and morris water maze transfer latency. It showed a marked increase in passive avoidance paradigm transfer latency. The administration of ethanolic *Bauhinia variegata* leaf extract (200mg/kg and 400mg/kg) indicated a substantial increase ($P<0.001$) in acetylcholinesterase and GSH levels, a significant reduction ($P<0.001$) in total protein, NO and MDA. **Conclusion:** This study shows ethanolic leaf extract of *Bauhinia variegata* has a neuroprotective effect against memory loss caused by colchicine.

Key words: *Bauhinia variegata*, Neuroprotective, Alzheimer's disease, Colchicine.

INTRODUCTION

The disease of Alzheimer is a chronic and liberal neurological condition that slowly causes loss of memory, behavior change, personality and ability to think. Dementia in older people is primarily caused by Alzheimer's disease. Duration between symptom initiation and death takes about 8.5 years. Approximately 15 million world population suffer from Alzheimer's disease.[1] The disease of Alzheimer characteristically affects folks around age 65 years.[2] More free radical generation mostly damages the central nervous system. The excessive formation of free radicals can lead to neuronal

Submission Date: 07-09-2020;
Revision Date: 07-03-2021;
Accepted Date: 29-08-2021

DOI: 10.5530/ijper.55.3s.187

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destruction to DNA, membrane lipids and proteins. There is a marked decline of cholinergic neurotransmission in elderly people due to decreased levels of acetylcholine in the brain.[3] The most important cause of dementia is Alzheimer's disease (AD) where neuron loss occurs in distinct areas of the brain.[4] AD is characterized by neurotic plaque formation that includes amyloid β protein. Cholinergic cell failure occurs in the forebrain and acetylcholine that is responsible for the development of dementia.[5] Pollutants, stress and hereditary predilection are the key risk factors for neurodegenerative disorders.[6] The conventional medicine is commonly used for the deterrence and prevention, diagnosis and management and treatment of various ailment and diseases. The treatment by herbal plants of different diseases depends on observations and previous experiences found in books or taught verbally.[7] Neuroprotection involves the therapeutic strategies which can delay or cure neuronal damage. Nowadays, herbalism is as reliable, healthy and cheaper as it is in trend.[8]

Bauhinia variegata named Mountain Ebony (English), Rakta kanchan (Marathi), Kachnar (Hindi) belongs to the family Leguminosae (Caesalpinioideae). It is a medium-sized, tree which seasonally shed its leaves found in Himalayas, at an altitude of 1800 m across India. Leaves are curved at apex, connate for about two-thirds up, wider, leaflets are ovate firmly sub-coriaceous, pubescent beneath when young and intensely cordate with two leaflets. Flowers are distinctive in colour, lateral, sessile, stamens 5, absence of staminodes, flat fruits; rough glabrous dehiscent seeds, seeded 10-15.[9] It is growing in all of India and China. It is a reliable kind of greenhouse that grows in the Himalayas at an altitude of 1800 m.[10] *Bauhinia variegata* Linn. is customarily used in ulcer bronchitis, intestinal worms, fungal infection, diarrhea, dysentery, hepatic disorders, diarrhoea, leprosy, skin disease, wounds, bacterial infection and tumors.[11,12]

METHODS AND MATERIALS

The material of plant

Bauhinia variegata leaves were picked and collected from Shri Ram Murti Smarak college campus and identified by Prof. A.K. Jaitly, The plant science department. A voucher specimen (specimen number- RU/PS/2016/415) of the gathered sample has been deposited for future reference in the institutional herbarium.

Extracts preparation

The *Bauhinia variegata* leaves were initially washed in tap water, followed by drying in shade and finally powdered. The powder obtained was stored and packed into the column of Soxhlet and extracted for 24 h using petroleum ether (60-80°C). The obtained marc was extracted sequentially with chloroform (50-60°C) followed by ethanol extraction (68-78°C) for 24 h. The extract was collected was concentrated on a water bath at 50°C. The extracted dry powder was kept at room temperature. The harvest of the extract of petroleum, chloroform extract, methanol extract, ethanol extract and water extract was found to be 9.50 percentile (w/w), 7.65 percentile (w/w), 8.95 percentile (w/w), 8.50 percentile (w/w) and 0.30 percentile (w/w). Throughout the experimental analysis the ethanolic leaf extract was used.

Drug Treatment

The test of pharmacological made the extract obtained to be suspended at doses of 200, 400 mg / kg p.o. distilled water which double comprises carboxy methyl cellulose (1 percent w/v CMC). The prescriptions and dosages were administered to every mouse in sets and group 4,5,7,8 based on earlier researches of the ethanolic extract of *Bauhinia variegata* extract. Up to the end of the study period there was no mortality due to medication. During the course of the treatment, the drug excerpt and extract of *Bauhinia variegata* caused no abnormality or death.

Animals

Animals were collected from Animal House, Pharmacy Department, SRMS CET (Pharmacy), Bareilly, U.P. Animals were approved for this study by the Institutional committee responsible for Animal Ethics of the Pharmacy Department, SRMS CET (Pharmacy), Bareilly, U.P. Approval number (715/PO/Re/S/02 / CPCSEA). Swiss albino strains of undeveloped healthy adult mice of both sexes in equivalent numerical for every group (n=6) were taken for the study. The weight differences of the used animals were kept nominal at the start of the research and did not exceed ± 20 per cent of the average weight of each and every species. Mice usually weighed 25-30 gm.

The experimental animal house temperature of 22°C was maintained. Relative humidity must be between ranges from 50 to 60%. The lighting was artificial; the sequence was 12 light hr, 12 dark hr. We used traditional laboratory diets *ad libitum* provided with drinking water. Animals of the same group had been kept together in one cage. Healthy young adult mice were indiscriminately assigned to the control, standard and treatment sets and group. The animals were marked by labeling at the base of the tail and accustomed in their cages for at least 5 days before the research was initiated.

Chemicals and Drugs

Drugs: Sigma Aldrich had purchased piracetam and colchicine.

Chemicals: Ethyl acetate, petroleum ether, methanol, ethanol and chloroform were got from the laboratory of Central Drug House.

Vehicle

Bauhinia variegata extract (BVE) has been suspended in 1 % w/v CMC and administered through oral means to mice. Colchicine and Piracetam were liquefied distinctly in normal saline and injected i.c.v. and i.p. respectively. The Capacity administered orally and i.p. injection was 1ml/100 g of mouse.

Studies with serious toxicity

Bauhinia variegata ethanolic extract has been tested for severe oral toxicity in compliance with revised OECD guidelines No. 425. When given by oral route in dosages of up to 2000 mg / kg, the extract was without any toxicity in mice. Therefore, the experiment used 200 and 400 mg / kg of ethanolic leaf extract.

Group I: It was a control set. The vehicle was orally administered for 28 successive days and the transferal of latency was assessed on 28th and again on 29th day after 90 min of administration.

Group II: It denoted the positive control set. Piracetam was given to the underdeveloped mice for 28 consecutive days, and transferal of latency was calculated and on 28th day after 60 min of administration, and again on 29th day after 24 hr.

Class III: This was a negative control group. Colchicine was injected i.c.v to underdeveloped mice, and latency transferal being was assessed 45 min after inoculation and also again after 24 hr (i.e. on the 29th).

Class IV and V: BVE was orally given to the underdeveloped mice for 28 successive days. TL was reported on 28th day after 90 min of exposure and similarly after a period of 24 hr on the 29th day.

Group VI: Young mice received inoculation of piracetam for 28 successive days. Colchicine 1 mg / kg, i.p., at 60 min after 28th day piracetam injection was given in. TL was reported after 45 min of colchicine administration and once more on the 29th day after 24 hr.

Group VII, VIII: BVE were orally given to the underdeveloped mice for 28 consecutive days and colchicine (1 mg/kg) was inoculated i.p. to small aged mice at 90 min. after ad-ministration of excerpt on day 28th. TL was reported 45 min. once the inoculation is done and after 24h.

Behavioral Models which are Exteroceptive

Elevated plus maze

The structure consists of a 10x10 cm-sized central platform attached to two 50x10 cm open arms and two 50×40×10 cm-sized closed arms and raised 50 cm above the floor. The research was using mice weighing 20 to 25 g. The experiment was carried out in 2 steps. On day 14, the day of acquisition testing, each rat was fixed at last position of an open arm pointing away from the middle point. The period of time consumed to through only one locked arms was noted and documented then later calculated as transferal latency [TL]. All four legs were counted as entry within the closed arm. For each mouse the cut-off time was 180 s. Animals who did not reach the closed arms during the time of cut-off were excluded from analysis. Retention testing was carried out on the 15th day, and transfer latency was reported in the same manner as previously stated. Shortening of transfer latency depicted improvement of memory.[6]

Step through Passive avoidance paradigm

The long term memory was tested using the passive avoidance model. This apparatus was composed of a small chamber connected through a guillotine door to a larger chamber. The smaller chamber, as it was illuminated with a 7W/12V bulb, was also called light chamber. Mice were initially given acquisition trial and subsequently given retention trial after 24 hr followed by IInd, IIIrd and IV retention trials on successive days. In the acquirement test experiment, each mouse was positioned at a maximum distance from the guillotine door in the smaller room. We have noticed the time span the mouse had taken to reach the darker room. For the study, the mice which did not reach the door within a cut-off period (90s) were not used. The door was shut automatically after the mouse come into the dark section and an unavoidable foot shock of 1 mA for 1 sec was recorded. Within 10s the mouse had been detached from the dark room. This procedure has been replicated with standard, control and test drugs. The rise in latency step-by - step was considered as learning.[13]

Morris water maze

The MWM was utilized for assessing rodent spatial memory and learning. It consists of a large round black container with a thickness of 120 cm, 50 cm height, filled with water at 26±2°C up to a deepness of 30 cm. The spherical pond was separated into four equal quadrants and an 8 cm^2 platform was flooded 1 cm underneath the impervious and opaque surface of one of the quadrants in the middle. The platform's position was held steady throughout the mission. To hide the

position of the flooded platform the water was made colored which are non-toxic.

The mice were released one by one inside the water and permitted to find the platform for 120 sec. Animals were exposed to 2 testing a day for 4 days with an inter-trial period of 20 min, and the time latency to locate the target was low (< 10 sec). During through trial mice's escape latencies were registered. The mean of the parameter was done for each and every period of testing and for every rat. When the platform was found by the mouse it was permitted to be on it for 10 sec. If the mouse didn't trace the platform within 90 sec, it was positioned for 10 sec on the platform and detached from the pool afterwards. Mice were initially given acquisition trial and were given retention trial after 24 hr followed by IInd and III retention trials on successive days. Day by day in trial 1 the decrease in escape latency denotes long-standing reminiscence or reference recollection, while that from testing or trial 1 to trial 2 and 3 represents working memory or short-lived reminiscence.[14,15]

Actophotometer

Actophotometer comprises six built in photo-sensors and 4 optical counters showing the operation of the locomotive activity. It indicated the behavior digitally. Most locomotive movements in man and animals are affected by CNS acting drugs. The locomotive activity (horizontal activity) can be measured easily by an actophotometer that functions on photoelectric cells that are linked to a counter in circuit. When the beam is cut off by animal of light falling on the photo cell, a count is registered. An actophotometer may possess either a round or a square region where the animal roam about.

We initially weighed and numbered the mice, then switched on the equipment and placed each and every mouse in the activity birdcage individually for 10 min. We noted down all the animals basal activity score.[16]

Biochemical Analysis

On the 28th day after Colchicine injection the biochemical parameters for oxidative stress such as NO, GSH, MDA and AChE were calculated in mouse's brain.

Brain tissue preparation

Using ether anaesthesia the mice were killed. After the cranium was cut and the brain was removed. The brain was cleansed using regular (chilled) saline solution. 10 percent (w/v) homogeneous brain sample was obtained with 0.03 M sodium phosphate buffer (pH 7.4) at 10 strokes at 2000 rpm. NO, GSH, MDA and AChE may be measured using a homogenized preparation of brain tissue.

Scavenging activity of Nitric oxide

Marcocci *et al*., 1994 coined away of probing nitric oxide searching behavior using Griess reagent using the method. 2 ml of sodium nitropruside is dissolved in 0.5 ml of phosphate of pH 7.4 which was combined with 0.5 ml of extracts of varying concentrations (50-200 µg/mL). The blend was nurtured and incubated for 150 min at 25°C. Instead, 0.5 mL of the hatched solution was mixed at room temperature for 5 min with 1 mL of Naphthylethylenediamine dichloride (0.1 percent w / v) with 0.5 mL of Griess reagent [(1.0 mL of sulfanilic acid reagent (0.33 percent of 20 percent glacial acetic acid)]. The mix was then hatched at the surrounding temperature for 30 min and its absorption was taken at 546 nm. By following this equation the inhibition percentile of Nitric Oxide was found as follows:

NO radical percentile inhibition = $(A_0 - A_1)/ A0 \times 100$

where

A_0 - absorbance before reaction

A_1 - the absorbance after reaction[17]

MDA Measurement

MDA is a test of peroxidation of the lipids. MDA can be measured spectrophotometrically using standard 1, 1, 1, 3, 3-tetraethoxypropane, using the technique defined by Colado *et al*., 1997. MDA is typically articulated by nanomoles per mg protein. For 10 min homogenized brain tissue was centrifuged at 700 g. Approximately 500 µl of homogeneous brain tissue in phosphate buffer (pH 7.4) was added to 300 µl 30% trichloroacetic acid (TCA), 150 µl 5N HCl, and 300 µl 2% (w / v) 2-thiobarbituric acid (TBA), respectively. The mix was then boiled at 90°C for 15 min by placing aluminum foil in the mouth of the test tube. The tubes were withdrawn after 30 min and held in ice-cold water for 30 min. The supernatant was obtained in pink colour. The 12,000g mixture was centrifuged for 10 min, the resultant supernatant was spectrophotometrically calculated at 532 nm.[18]

GSH Measurement

GSH was calculated by its 5, 5'-dithiobis (2-nitrobenzoic acid) reaction (Ellman, 1959) yielding a yellow chromophore that was analyzed spectrophotometrically. GSH is a protein expressed in µg /mg. For 10 min homogenized brain tissue was centrifuged at 700 g. 500 µl of brain homogenate was combined with 10% trichloroacetic acid (500µl) and then centrifuged for protein separation

at 2000 g for 10 min at 4°C. 100 µl of supernatant was added to 2 ml of 0.1 M phosphate buffer (pH 7.4), 0.5 ml of 5, 5-dithiobis (2-nitrobenzoic acid) (DTNB) and 0.4 ml of deionized water, followed by shaking the mixture onto the vortex. The absorption was taken within 15 min, at 412 nm.[19]

Acetylcholinesterase (AChE) activity

Acetylcholinesterase action is considered as a marker for the brain's prolonged depletion of the cholinergic system of brain. The Ellman's test conducted quantitative analysis of the acetylcholinesterase levels in the brain. 0.1 ml of DTNB, 0.1 ml of acetylthiocholine iodide, 0.05 ml of supernatant and 3 ml of 0.01 M sodium phosphate buffer (pH 8) were taken using this process. The absorbance shift at 412 nm was assessed at a 30 s interval for 2 min. Outcomes got calculated through the utilization of the chromophore's molar extinction coefficient (1.36 x 10^4 $M^{-1}cm^{-1}$) and expressed as acetylcholine hydrolyzed / min / mg protein micromoles.[20]

$$R = \frac{\delta OD \times \text{Volume of Assay} \times 1000}{E \times \text{mg protein}}$$

Where, R is the rate of enzymatic action in 'micro' mole of acetylthiocholine iodide hydrolyzed per minute per mg of protein.

δ OD is the variation or change in absorbance per minute

E is the coefficient of extinction (1.36×10^4 $M^{-1}cm^{-1}$).[21,22]

Protein estimation

In all brain samples, protein was calculated utilizing Lowry's technique where bovine serum albumin (BSA) (1 mg / ml) was used as standard.[23]

Reagents

1. Alkaline solution
   a) 2% (w/v) Sodium carbonate in 0.1 M NaOH.
   b) 1% (w/v) Copper Sulphate
   c) 2% Sodium Potassium tartrate
   Working alkaline solution: 48ml of A + 1ml of B + 1ml of C
2. Stock std. Bovine Serum Albumin (BSA) - 1mg/ml
3. Working standard BSA (1000µg/ml) diluted the stock 20 times.
4. Folin-Phenol reagent (ice-cold) diluted with equal amount of water at the time of use.

Test Method:

0.1ml of supernatant was added to 0.9ml DDW and 5ml of working alkaline reagent. The mixture was well mixed and then incubated at the temperature of the surrounding for 10 min. Following this, 0.5 ml Folin-phenol reagent was applied and hatched at the temperature of the surrounding for thirty minutes. The absorbance had been calculated against blank at 750 nm. A standard and typical curve (50-1000µg) was then plotted accompanied by an estimate of the sample's protein content as mg/ml.[23]

Statistically Done Analysis

All of the findings were represented as an average ± SEM and assessed by ANOVA which is One-way then by Tukey's multiple *post-hoc* evaluation study. A '*P*' value of < 0.05 has been recognized as statistically important. Graph Pad prism software analyzed data.

RESULTS AND DISCUSSION

Elevated Plus Maze Test

The result of the *Bauhinia variegata* leaf excerpt and extract on elevated plus maze apparatus is shown in Figure 1. The Original Transferal Latency was conducted on the 14th day; i.e., acquisition trial, and no significant variation was found. Outcome are characterized as average ± SEM (n=6), * P<0.05, * * P<0.01, * * * P<0.001when ANOVA which is one way then by Tukey's tests compared with control group. In the leaf extract an increase in transmission latency was found and therefore nootropic activity was proven. The 400 mg/kg dosage of the *Bauhinia variegata* leaf excerpt showed substantial improvement in the mice's transfer latency. This shows the action on the leaf by nootropic excerpt of *Bauhinia variegata*.

Step-through Passive avoidance paradigm

The effect of *Bauhinia variegata* leaf excerpt and extract on step-through passive avoidance paradigm is shown in Figure 2. The acquisition trial was performed and no substantial difference was observed. Outcomes are characterized as average of ± SEM (n=6), * P<0.05,** P<0.01,*** P<0.001 when ANOVA which is one way then by Tukey's trial is applied to liken with control group. In the leaf extract a decrease in transmission latency was observed suggesting nootropic activity. The 400 mg / kg dosage of the *Bauhinia variegata* leaf excerpt showed substantial reduction in the mice's transfer latency. This shows the possible nootropic activity of the leaf excerpt of *Bauhinia variegata*.

Morris water maze

The effect of *Bauhinia variegata* leaf extract on morris water maze is shown in Figure 3. Outcomes are characterized as average of ± SEM (n=6), * P<0.05, * * P<0.0 1, * * * P<0.001

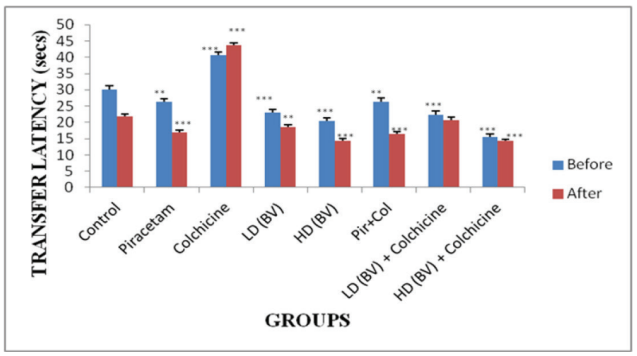

Figure 1: Impact of *Bauhinia variegata* extract and excerpt on Elevated plus maze.

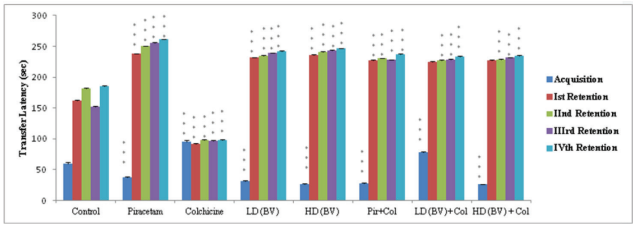

Figure 2: Effect of *Bauhinia variegata* extract on Passive avoidance paradigm.

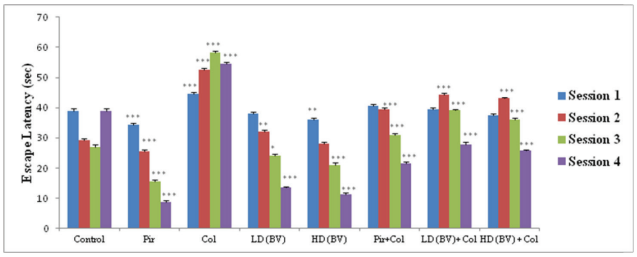

Figure 3: Impact of *Bauhinia variegata excerpt and* extract on Morris water maze.

when ANOVA which is one way then by Tukey's trial is applied to liken with control group. In the leaf extract an increase in escape latency was observed suggesting nootropic behavior. The 400 mg / kg dose of the *Bauhinia variegata* leaf excerpt showed substantial improvements in the mice's escape latency. This shows the possible nootropic activity of the leaf extract *Bauhinia variegata*.

Actophotometer

The effect of the *Bauhinia variegata* leaf extract on an actophotometer is shown in Figure 4. Results are characterized as an average ± SEM (n=6), * P<0.05, ** P <0.0 1, *** P<0.001when ANOVA which is one way then by Tukey's trial is applied to liken with control group. The leaf extract did not show any CNS depressant effect on mice.

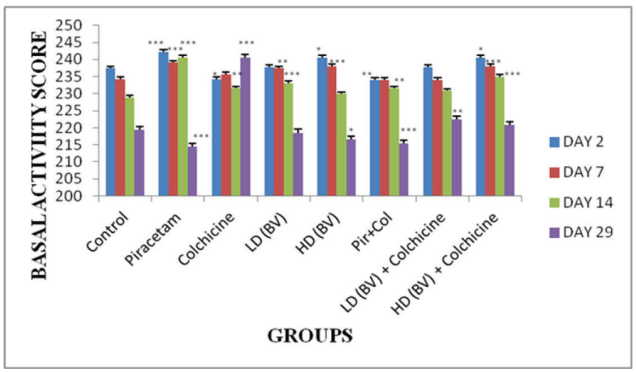

Figure 4: Impact of *Bauhinia variegata* extract on Actophotometer.

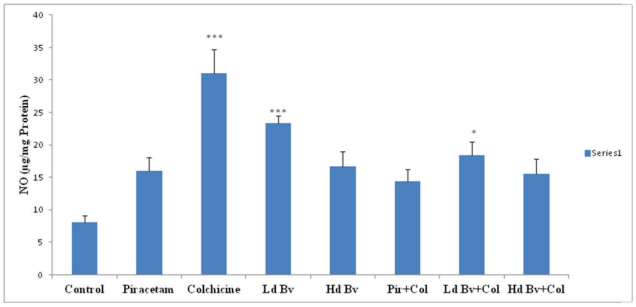

Figure 5: Impact of ethanolic extract and excerpt of *Bauhinia variegata* on NO level.

Estimation of NO

Figure 5 shows the effect of leaf extract of *Bauhinia variegata* on NO level of mice's brain homogenate. The mice were sacrificed on the 29th day, and the brain homogenate was prepared to predict NO level changes. Outcomes are denoted as MEAN ± SEM (n=6), * P <0.05,** P<0.01,*** P<0.001when ANOVA which is one way then by Tukey 's tests likened with control group. In the leaf extract a large decrease in the amount of NO was observed, thus proving nootropic activity.

Estimation of MDA

The effect of *Bauhinia variegata* leaf extract on MDA level of mice's brain homogenate is shown in Figure 6. The mice were sacrificed on the 29th day, and the brain homogenate was prepared to predict improvements in the amount of MDA. Outcomes are denoted as average ± SEM (n=6), * P<0.05,** P<0.01,*** P<0.001 when ANOVA which is one way then by Tukey 's trial to liken with control set. A substantial decrease in the amount of MDA was found in the leaf extract and thus nootropic activity was confirmed.

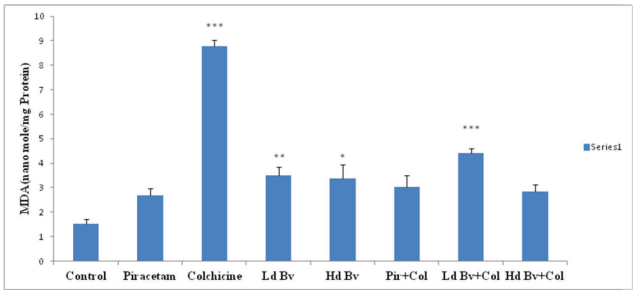

Figure 6: Impact of ethanolic excerpt and extract of *Bauhinia variegata* on MDA level.

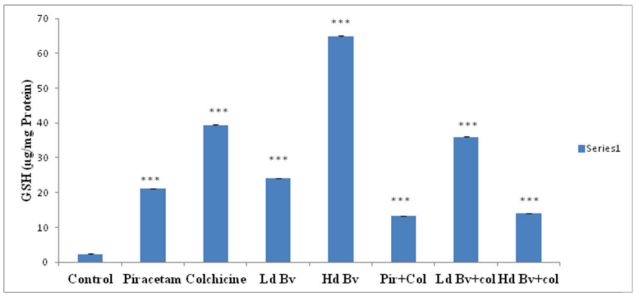

Figure 7: Impact of ethanolic excerpt and extract of *Bauhinia variegata* on GSH level.

Estimation of GSH

The effect of *Bauhinia variegata* leaf extract on GSH level of mice's brain homogenate is shown in Figure 7. The mice were sacrificed on the 29th day and the brain homogenate was prepared to measure GSH level changes. Outcomes are denoted as MEAN ± SEM (n=6), * P<0.05,** P<0.01,*** P<0.001when one way ANOVA then by Tukey 's trial likened with control set. The leaf excerpt and extract showed a large increase in GSH level, thus proving nootropic activity.

Estimation of AChE

The effect of *Bauhinia variegata* leaf extract on AChE level of mice's brain homogenate is shown in Figure 8. The mice were sacrificed on the 29th day, and brain homogenate was prepared to estimate improvements in the amount of AChE. Outcomes are calculated as average ± SEM (n=6), * P<0.05, * * P<0.01, * * * P<0.001when ANOVA which is one way then by Tukey 's trials and tests likened with control collection or group. In the leaf extract an increase in AChE level was observed suggesting nootropic activity.

Alzheimer's ailment and disease is a gradual onset neurodegenerative condition. A satisfactory drug for complete cure of Alzheimer's disease is yet to be developed in the allopathic system of medicine. Now we should look forward to treating this illness with the herbal medicines. In the current research, *Bauhinia variegata*

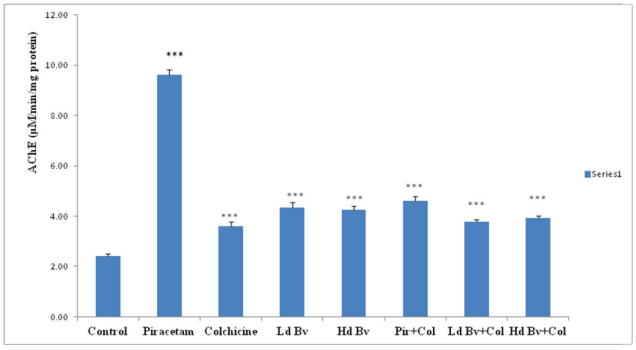

Figure 8: Consequence of ethanolic excerpt and extract of *Bauhinia variegata* on AChE level.

extract for 28 days was given orally which showed an improvement in mice's learning behavior. Throughout this analysis, the higher dose of *Bauhinia variegata* extract (400 mg / kg) significantly enhanced mice's memory as there was a decrease throughout Latency transferal in the case of raised plus maze system and increased Transfer Latency in the case of passive avoidance testing in contrast with the control set or group. In the case of morris water maze study, there was a decline in the latency Seepage as opposed to the control set. The locomotive operation indicated no symptoms of CNS distress. *Bauhinia variegata* extract pretreatment for 28 days sheltered and sheltered the animals from the shortfalls of the memory generated by colchicine. These findings indicate the potential role of *Bauhinia variegata* extract in neuroprotecting.

Reactive oxygen species (ROS) are the root cause of decline related to age in cognitive presentation that could be involved in elderly people developing Alzheimer's disease. *Bauhinia variegata* has antioxidant properties. *Bauhinia variegata* extract's neuroprotective activity is related to its antioxidant properties, due to which the susceptible neurons are subjected to less oxidative stress leading to reduced neuronal harm and enhanced neuronal function. The symptoms of dementia are impairment in neuronal transmission, decline in brain acetylcholine levels and neuronal circuit degeneration in the affected brain areas. From the present analysis it can be inferred that the *Bauhinia vareigata* ethanolic excerpt and extract at a dosage of 400mg /kg possesses nootropic activity comparable to the regular Piracetam medication. Pretreatment with *Bauhinia vareigata* substantially reduced latency transferal in raised plus maze, improved latency transferal in passive avoidance study and decreased the escape latency in morris water maze. The *Bauhinia variegata* ethanolic leaf extract decreased NO, MDA and increased the level of

GSH, AChE. Hence the *Bauhinia vareigata* ethanolic leaf extract has significant nootropic activity.

ACKNOWLEDGEMENT

We thank Shri Ram Murti Smarak, Department of Pharmacy, College of Engineering and Technology (Pharmacy), Bareilly, U.P. to provide chemicals and other facilities for conducting the study. The work is dedicated to my guide and co-guide.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

ABBREVIATIONS

AChE: Acetyl cholinesterase; **MDA:** Malonyldialdehyde; **GSH:** Glutathione; **ANOVA:** Analysis of variance; **IAEC:** Institutional Animal Ethics Committee; **CPCSEA:** Committee for the Purpose of Control and Supervision of Experiments on Animals; **rpm:** Rotation Per Minute; **BV:** *Bauhinia variegata*.

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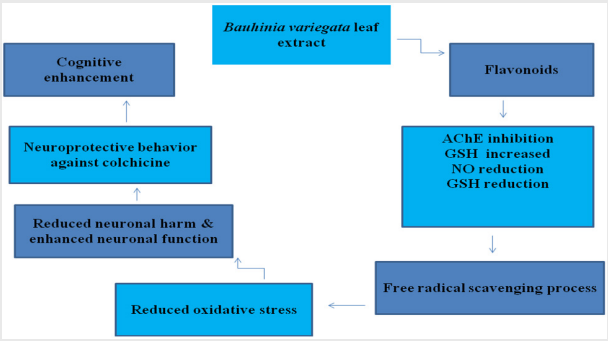
PICTORIAL ABSTRACT



SUMMARY

In Swiss albino mice the neuroprotective effect of *Bauhinia variegata* leaves was tested by a model of neurotoxicity caused by colchicine. Diverse behavioural and biochemical parameters have been measured in animals exposed to colchicine. Colchicine administration resulted in substantial memory deficits, which were also biochemically supported as the brain encountered increased oxidative stress and elevated acetylcholinesterase (AChE). In colchicine treated animals, pretreatment with ethanolic extract of *Bauhinia variegata* leaves significantly improved memory as well as decreased oxidative stress, AChE in the brain. The extract used has been shown to have neuroprotection as proof of improvement in the acetyl cholinesterase, lipid peroxidation, glutathione and protein neurological ranking. This may be due to the presence of flavonoids in *Bauhinia variegata* leaf.

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Cite this article: Khare P, Kishore K, Sharma DK. Neuroprotective Effect of *Bauhinia variegata* Leaf Extract against Colchicine: An Experimental Study on Cognitive Dysfunction and Biochemical Alterations in Mice. Indian J of Pharmaceutical Education and Research. 2021;55(3s):s798-s806.