International Journal of Allied Medical Sciences and Clinical Research (IJAMSCR)

ISSN:2347-

IJAMSCR |Volume 9 | Issue 2 | Apr- Jun - 2021
www.ijamscr.com

Research Study *Medical research*

Pharmacological evaluation of polyherbal formulation for neuroprotective effect

Syed Iqra Naznin*, Tarif hussain, Govind Pawar

Assistant Professor, Department Of Pharmacology, Holy Mary Institute Of Technology And Science College Of Pharmacy, Hyderabad, Telangana 501301, India

***Corresponding Author: Syed Iqra Naznin**
Email id: isyedpharma@gmail.com

ABSTRACT

Neurodegenerative disorders share characterized with destruction of neurons and are one of the most difficult diseases to treat worldwide, especially in the elderly patient. The neuroprotective effect and LD_{50} of polyherbal formulation was determined in Albino wistar rat. Rats were subjected treatment with polyherbal formulation and standard drug for a period of 21 days. The nerve conduction was assessed by Tail flick and clip method, Tail immersion method. At the end of the study animals were sacrificed and the samples are used for Biochemical analysis. The phytochemical analysis of Polyherbal formulation shows positive results for glycosides, alkaloids, flavonoids, tannins, terpenoids and phenolic compounds. Acute toxicity tests find no lethality or adverse reactions with elevated dose (2000 mg/kg weight of body). The neuroprotective effects are showed based on the dose.Group VIII (animals received polyherbal formulation at 600 mg/kg b.w, p.o per day). Has significant effect when compared with other groups.

Keywords: Neuroprotective, Polyherbal formulation, Sciatic nerve ligation

INTRODUCTION

The nervous system, which comprises the brain and spinal cord, is made up of neurons, which are the building blocks (Stahl, 2008)[1]. They've been designed to relay data across the body and these highly specialized nerve cells are in charge of communicating information in both chemical and electrical ways, as well as performing a variety of functions in the human body. Sensory neurons transport sensory cell information to the brain across the whole body. Motor neurons transfer input from the brain to the body's muscles. Interneurons communicate information about various neurons in the body.

Neurodegenerative disorders share pathological and pathogenesis characteristics, such as the destruction of neurons, and are one of the most difficult diseases to treat worldwide, especially in the elderly[2]. It influences the CNS, allowing neuron activity to gradually deteriorate. The loss of neuronal cell function is a defining feature of these debilitating and incurable diseases, which are frequently accompanied by atrophy of the affected nervous system structures. They are chronic and incurable diseases that cause nerve cell death and gradual degeneration. This can result in ataxias (problems with movement) or dementias (abnormal mental functioning). Some of the neurodegenerative diseases are Alzheimer's disease, Parkinson's disease, Huntington's disease, Motor neurone disease, Prion disease, spinocerebellar ataxia and Spinal muscular atrophy3-6.

Collection and Identification of the Plant

Carica papaya leaves extract, *Vitis vinifera* Linn. Seed extract, and *Psidium guajava* Linn. Leaf extract were obtained from Hapur's local market (Uttar Pradesh)[22]. Temperatures of 40°C were used to dry the plant materials. It was crushed up and held in closed containers.

Preliminay phytochemical screening

Qualitative Phytochemical Analysis[24]

Phytoconstituents such as alkaloids, glycosides, carbohydrates, phenolics and tannins, phytosterols, fixed oils and fats, proteins and amino acids, flavonoids, saponins, and others were determined in the hydro alcoholic extract of *Carica papaya* Linn., seed extract of *Vitis vinifera* Linn., and leaf extract of *Psidium guajava* Linn.

Principle

Female sex groups were dosed in a stepwise manner with fixed doses of 300 and 2000 mg/kg (Oral administration) (exceptionally, a supplementary fixed dose of 5000 mg/kg may be considered)[26]. Based on a sighting report, the initial dosage level was chosen as the dose that would produce any symptoms of toxicity without causing severe toxic effects or death. Based on the appearance or lack of signs of toxicity or death, additional classes of animals may be dosed at higher set doses.

Selection of animal species

For the sample, female Wister albino rats were chosen. The classification criteria for the animal are based on literature searches of specific LD samples[27]. While there is no variation in sensitivity between the sexes, females are significantly more sensitive in those situations where variations are found. When the test is carried out on men, sufficient justification should be given. Female mice, both nulliparous and non-pregnant, were included. Each animal was between the ages of 8 and 12 weeks when dosing began.

Observations

Since dosing, animals were monitored separately at least once every 30 minutes for the first 30 minutes, then sporadically for the first 24 hours. The special warning was issued for the first four hours and then every day for the next fourteen days. Changes in skin and hair, eyes and mucous membranes, as well as metabolic, circulatory, autonomic, and CNS functions, and behaviour patterns, were all observed. Tremors, convulsions, salivation, diarrhoea, lethargy, sleep, and coma were all closely monitored. According to OECD recommendations, all plant extracts were screened for lethal dose (LD50) and the LD50 cut off value of extracts was determined.

Table:1 Qualitative Phytochemical Analysis

Name of Extract	LD_{50} cut off mg/kg b.w.	Therapeutic Dose
Carica papaya	1.2 mg/kg	120 mg/kg
Vitis vinifera	3 mg/kg	300 mg/kg
Psidium guajava	10 mg/kg	1000 mg/kg

Acute toxicity study of poly herbal formulation

Doses for Therapeutic Use On the basis of OECD recommendations 420, a dose of 200 mg/kg was chosen for the sample. Female rats were dosed in groups of five in a stepwise protocol with set doses of 5, 50, 300, and 2000 mg/kg. The initial dosage level was chosen based on a sighting report that predicted certain symptoms of toxicity but no significant toxic effects or mortality. The acute toxicity analysis of poly herbal formulations was carried out in the manner previously described, and the findings are shown in Table No. 2.

Animals

The rats included in the study were Swiss Albino male rats weighing 120–130 g. prior to the tests, the animals were acclimatised for two weeks in a laboratory environment. They were held in normal conditions of temperature (23 2°C) and humidity (50 10% relative humidity) with 12hour light/dark cycles. The animals were served a normal pellet diet and had free access to tap water (Agro Corporation Private Ltd., Bangalore, India).

Surgical Procedure to induce neuropathy

Animals The rats were subjected to SNL surgery under general anaesthesia with intramuscular injection of ketamine 100 mg / kg and 10 mg /kg xylazine. The rats were put in a prone position and the fur on their hind limbs and midback was shaved. At the dorsa caudal area, the sciatic nerve was exposed. An incision was made 0.5 cm laterally from the animal's midline and continued for 3 cm laterally to the tibio femoral articulation. The femoral biceps and gluteus muscles were separated with blunt dissection forceps to provide access to the sciatic nerve, and half of the left sciatic nerve was ligated with an 8-0 nylon suture at the upper thigh level. Sham surgery was done by exposing the sciatic nerve without ligation [15].

Nociceptive threshold was assessed on 15th day. Then the animals were sacrificed by cervical decapitation, sciatic nerve was collected, homogenized and supernatant was used for biochemical studies.

Development of Poly Herbal Formulation

On the 15th day, the Poly Nociceptive threshold was determined. The animals were then decapitated, the sciatic nerve was collected, homogenized, and the supernatant was used for biochemical experiments. The basis of selection of the individual plant extract was the most effective dose and therapeutic doses were finalised on the basis of Acute Toxicity Studies (OECD 420) guidelines. The Poly Herbal Formulation was prepared in the three different strengths for the purpose of dose optimization and to find out the most effective and safer dose. **(**Table:2)

Table:2 Experimental designs : animals were divided into eight groups of six each

Group – I :	Normal healthy rats received only the water served as normal control
Group – II :	SNL – Sciatic nerve ligation group, animals received vehicle only
Group – III :	(Sciatic nerve ligation group - SNL) animals received *Carica papaya* extract (120 mg/kg b.w, p.o per day)
Group – IV :	(Sciatic nerve ligation group - SNL) animals received *Vitis vinifera* extract (120 mg/kg b.w, p.o per day).
Group – V :	(Sciatic nerve ligation group - SNL) animals received *Psidium guajava* extract (120 mg/kg b.w, p.o per day).
Group – VI :	(Sciatic nerve ligation group - SNL) animals received polyherbal formulation CVP-1 (200 mg/kg b.w, p.o per day).
Group – VII :	(Sciatic nerve ligation group - SNL) animals received polyherbal formulation CVP-2 (400 mg/kg b.w, p.o per day).
Group – VIII :	(Sciatic nerve ligation group - SNL) animals received polyherbal formulation CVP-3 (600 mg/kg b.w, p.o per day).

Evaluation of nociceptive parameters[28]

Tail flick method

The tail flick test, like the hot plat test, assesses an animal's pain tolerance. By studying the response to heat, it is used in basic pain research and to assess the potency of analgesics. The quantitative assessment of an animal pain threshold in the presence of thermal radiation, as well as the estimation of analgesic efficacy. The nociceptive reaction was assessed in terms of the time it took for the tail to exit in response to noxious radiant heating. Specific restraining cages are mounted in which the tail is allowed to hang freely. Before being tested, the animals are given 30 minutes to adjust to their new surroundings. The tail flick analgesiometer was used; the tip of the rat's tail was mounted on a hot metal wire, and the delay of withdrawal was measured manually using a stop watch.

Tail clip method

The method was described by Haffner Each of the five groups received six screened Wister rats. The medication was given 30 minutes before the exam. To cause discomfort, an artery clip was added to the root of the tail (approximately 1 cm from the body). The animal bites the clip or the tail near where the clip is located in response to the noxious stimulus. A stopwatch was used to record the time between stimulus and reaction in 1/10 second intervals. In various periods of therapies, the amount of time before reaction suggests the period of highest operation after dosing (Schleyerbach, 2002). For comparative analysis, the experimental groups' values of latency time prolongation were relative to the control group's values.

Hot Plate Method

Woolfe and MacDonald 148 first mentioned this procedure (1944). The paws of rats are extremely sensitive to heat, even at temperatures that aren't harmful to the skin. Jumping, paw withdrawal, and paw licking are some of the reactions. Following the initiation of centrally acting analgesics, the time it takes for these reactions to occur is extended. In this hot plate procedure, animals from each party were put on a commercially accessible hot plate (Eddys hot plate) with an electrically heated surface. The temperature of this hot plate is held between 55 and 56 degrees Celsius. This may be a heated glass surface or a copper plate. The observation was completed before paw licking or jumping was observed; the cut-off time was set at 10 seconds. The reaction time was measured after the medications and test compound were given orally.

Tail Immersion Method

Hot and cold water Tail Immersion Test

This method was described by Sharma *et al*., 2006 a 15. The treatment was based on the response time of the normal tail withdrawal reflex in rats, which was triggered by immersing the end of the tail in warm (52.51) and cold (100.5) water. The lower portion of the tail was labelled and submerged in a beaker of

freshly filled warm and cold water, about 5 cm from the top. In a short amount of time, the rats responded by pulling their tails down. This reaction was tested two to three times with a 15-minute interval between each measurement in order to achieve two values that varied by no more than 10%. The tail was carefully dried for each determination. Hot water tail immersion took 15 seconds, and cold water tail immersion took 30 seconds.

STATISTICAL ANALYSIS

The data for all of the analyses is interpreted as Mean S.E.M. on regression analysis using one-way ANOVA followed by Duncun's Multiple Range Test, with a significance level of $p< 0.05$.

RESULTS

Table: 3 Preliminary Phytochemical Screening

SL. NO	CONSTITUENT	*Carica Papaya*	*Vitis Vinifera*	*Psidium guajava*
1	Alkaloid	+	+	+
2	Carbohydrate	+	+	+
3	Protein	+	+	+
4	Terpinoids	+	+	+
5	Phenols	+	+	+
6	Tannins	+	-	+
7	Flavanoids	-	-	+
8	Glycoside	+	+	-
9	Saponins	+	+	+

Presence - Absence

Objection recognisation test

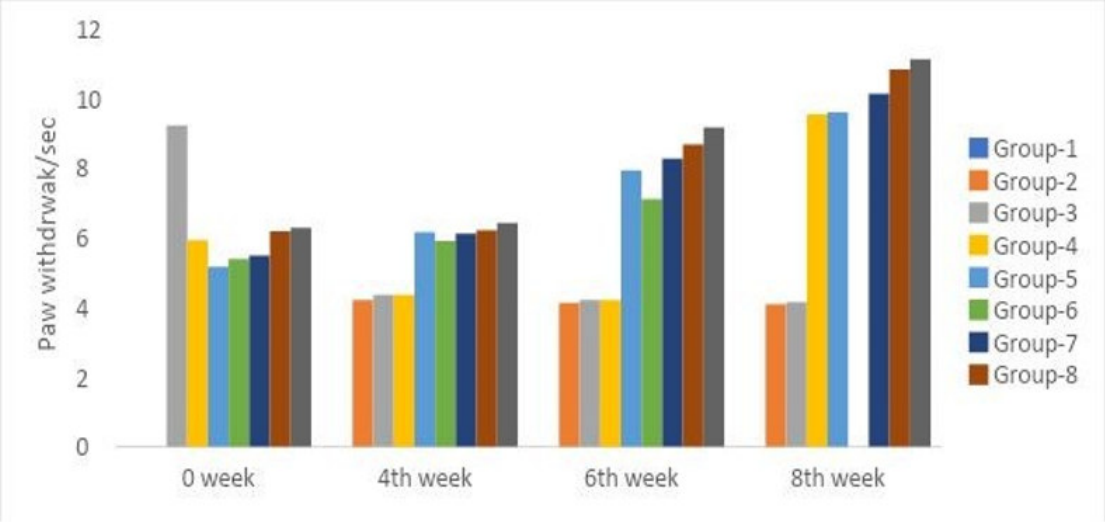
Hot plate test (thermal hyperalagesia)

GROUPS	READ TIME IN SECONDS			
	0TH WEEK	4TH WEEK	6TH WEEK	8TH WEEK
Group -1	5.05 ± 0.84	4.46 ± 0.73	4.20 ± 0.27	3.91± 0.17
Group -2	5.05 ± 0.31	4.31± 0.67	4.11 ± 0.37	4.03 ± 0.54
Group -3	5.05 ± 0.56**	4.50 ± 0.16**	4.96 ± 0.56	9.31 ± 0.16
Group -4	5.05 ± 0.31*	4.60 ± 0.37*	4.70 ± 0.84	9.54 ± 0.22
Group -5	5.05 ± 0.31	5.20 ± 0.18	8.81 ± 0.16	10.87 ± 0.12
Group -6	5.05 ± 0.31	5.42 ± 0.18	9.21 ± 0.16	12.87 ± 0.12
Group -7	5.05 ± 0.31	6.20 ± 0.18	9.81 ± 0.16	14.27 ± 0.12
Group -8	5.05 ± 0.31	6.80 ± 0.18	10.11 ± 0.16	6.17 ± 0.12

Values are expressed SEM

The ANOVA shows most significant i.e. P>0.005

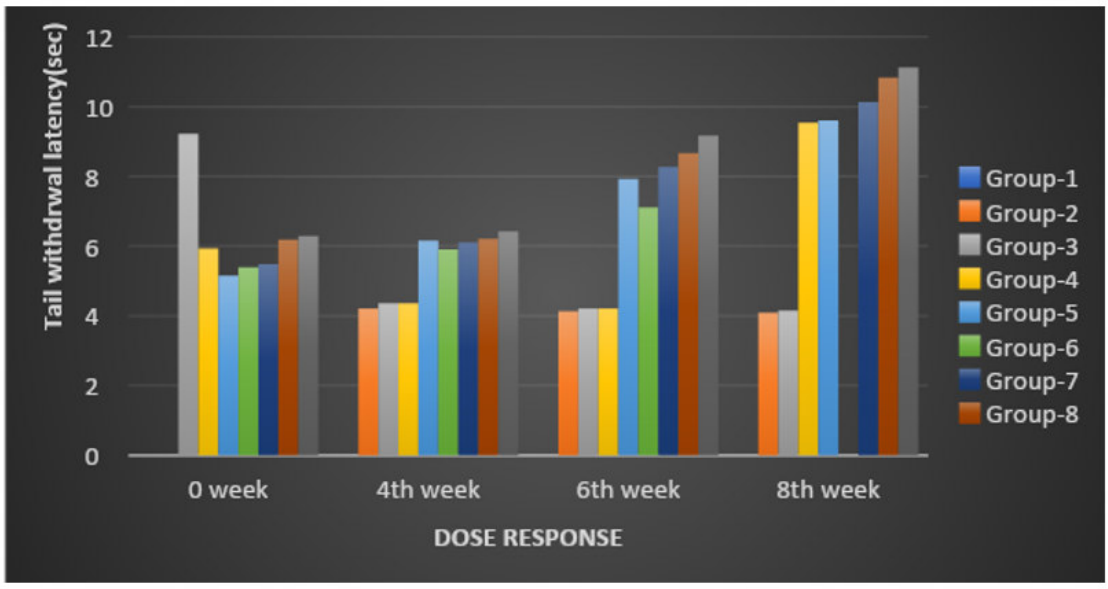
Tail flick test



GROUPS	READ TIME IN SECONDS			
	0TH WEEK	4TH WEEK	6TH WEEK	8TH WEEK
Group -1	4.23 ± 016	4.21 ± 0.27	4.13 ± 0.15	4.09 ± 0.627
Group -2	9.22 ±0.57**	4.36 ± 0.19	4.21 ± 0.15	4.45 ± 6.27
Group -3	5.93 ± 0.27**	6.16 ± 0.31**	7.93 ± 0.96	9.54 ± 0.73
Group -4	5.16 ± 0.16	5.91 ± 0.22*	7.11 ± 0.54	9.6 ± 0.56
Group -5	5.39 ± 0.23 *	6.11 ± 0.56	8.27 ± 0.22	9.93 ± 0.73
Group -6	5.49 ± 0.23 *	6.21 ± 0.56	8.67 ± 0.22	10.13 ± 0.73
Group -7	6.19 ± 0.23 *	6.42 ± 0.56	9.17 ± 0.22	10.83 ± 0.73
Group -8	6.29 ± 0.23 *	7.11 ± 0.56	10.27 ± 0.22	11.13 ± 0.73

Values are expressed SEM

The ANNOVA shows most significant value i.e. group-2 and 3 p>0.001



Effect of hot water tail immerssion test (Thermal hyperalagesia)

GROUPS	READ TIME IN SECONDS			
	0TH WEEK	4TH WEEK	6TH WEEK	8TH WEEK
Group -1	5.05 ± 0.84	4.46 ± 0.73	4.20 ± 0.27	3.91± 0.17
Group -2	5.05 ± 0.31	4.31 ± 0.67	4.11 ± 0.37	4.03 ± 0.54
Group -3	5.05 ± 0.31*	4.60 ± 0.37*	4.70 ± 0.84	9.54 ± 0.22
Group -4	5.05 ± 0.56**	4.56 ± 0.16**	4.96 ± 0.56	9.31 ± 0.16
Group -5	5.05 ± 0.31	5.42 ± 0.18	9.21 ± 0.16	12.87 ± 0.12
Group -6	5.05 ± 0.31	6.20 ± 0.18	9.81 ± 0.16	14.27 ± 0.12
Group -7	5.05 ± 0.31	6.80 ± 0.18	10.11 ± 0.16	6.17 ± 0.12
Group -8	5.05 ± 0.84	4.46 ± 0.73	4.20 ± 0.27	3.91± 0.17

Effect of cold water tail immerssion test (Thermal allodynia)

GROUPS	READ TIME IN SECONDS			
	0TH WEEK	4TH WEEK	6TH WEEK	8TH WEEK
Group -1	9.47 ± 0.67	5.11 ± 0.34	4.24 ± 0.13	1.01± 0.27
Group -2	9.86 ± 0.67	5.36 ± 0.34	4.76 ± 0.13	1.13 ± 0.27
Group -3	6.11 ± 0.27	7.58 ± 0.16	6.53 ± 0.14	5.32 ± 0.21
Group -4	6.34 ± 0.18**	8.56 ± 0.27	11.33 ± 0.18	13.24 ± 0.18
Group -5	6.46 ± 0.13	7.67 ± 0.16*	8.54 ± 0.13	10.16 ± 0.9
Group -6	6.51 ± 0.15	9.08 ± 0.53	10.56 ± 0.38	12.31 ± 0.84
Group -7	6.58 ± 0.15	9.18 ± 0.53	10.86 ± 0.38	12.61 ± 0.84
Group -8	6.62 ± 0.15	9.68 ± 0.53	11.16 ± 0.38	13.21 ± 0.84

DISCUSSION

Diabetic neuropathy is characterised by progressive chronic neuropathic pain that is tingling and burning in nature, as well as hyperesthesia (excessive physical sensitivity, particularly of the skin) and paresthesia (an irregular feeling, usually tingling or pricking (pins and needles), caused chiefly by pressure on or injury to peripheral nerves) with deep aching, which is aggravated by contact[29]. Neuropathic pain is a form of chronic pain caused by injury to or dysfunction of the central nervous system. Both signs of neuropathic pain in its clinical nature include hyperalgesia, allodynia and spontaneous pain[30]. Hyperglycemia contributes to neuronal toxicity because of accelerated glucose oxidation, resulting in antioxidant therapy regulated ROS.

In preliminary EEOA phytochemical analyses, glycosides, alkaloids, flavonoids, tannins, terpenoids and phenolic compounds were revealed[32]. The polyherbal formulation has anti-neuropathic and neuroprotective effects of tannins, phenols and flavonoids, while the herbal drug's alkaloids and glycosides have been shown to be potent antioxidant and nephroprotective agents[33-34]. Acute toxicity tests find no lethality or adverse reactions with elevated dose (2000 mg/kg weight of body) before the end of the study to the non-toxic character of EEOA. The LD50 dose of 2000 mg/kg was regarded as unclassified in accordance with the OECD 423 guidelines (Acute Oral Toxicity[35]: Acute Totoxic Classic Method), and was therefore found to be secure.

SUMMARY AND CONCLUSION

This research examined the neuroprotective role of diabetic neuropathy in rats by EEOA I. The phytochemical study shows that tannins, phenols and flavonoids have been shown to be effective in treating neuropathy. By improving the nociceptive threshold, attenuating behavioral change, and biochemical changes, the protecting effect of the polyherbal formulation against SNL induced neuropathic pain.

The PN is contributing to the alleviation of SNL mediated hyperalgesia, allodynia, inflammation and oxidative stress by anti-allodysis, hyperalgesia and antioxidant capacity. It also seems to be a therapeutic alternative solution for neuropathic patients.

REFERENCES

1. Kochupilai N. Clinical endocrinology in India. Curr Sci. 2000; 79:1061-7.
2. King H, Aubert RE, Herman WH. Global burden of diabetes, 1995-2025: prevalence, numerical estimates, and projections. Diabetes Care. 1998; 21(9):1414-31. doi: 10.2337/diacare.21.9.1414, PMID 9727886.
3. Boulton AJ, Vinik AI, Arezzo JC, Bril V, Feldman EL, Freeman R, Malik RA, Maser RE, Sosenko JM, Ziegler D, American Diabetes Association. Diabetic neuropathies: a statement by the American Diabetes Association. Diabetes Care. 2005; 28(4):956-62. doi: 10.2337/diacare.28.4.956, PMID 15793206.
4. Vinik AI, Mehrabyan A. Diabetic neuropathies. Med Clin North Am. 2004; 88(4):947-99, xi. doi: 10.1016/j.mcna.2004.04.009, PMID 15308387.
5. Obrosova IG. Diabetic Painful and Insensate Neuropathy: pathogenesis and Potential treatments. Neurotherapeutics. 2009; 6(4):638-47. doi: 10.1016/j.nurt.2009.07.004, PMID 19789069.
6. Greene DA, Stevens MJ, Obrosova I, Feldman EL. Glucose-induced oxidative stress and programmed cell death in diabetic neuropathy. Eur J Pharmacol. 1999; 375(1-3):217-23. doi: 10.1016/s0014-2999(99)00356-8, PMID 10443578.
7. Platel K, Srinivasan K. Plant foods in the management of diabetes mellitus: vegetables as potential hypoglycaemic agents. Nahrung. 1997; 41(2):68-74. doi: 10.1002/food.19970410203, PMID 9188186.
8. Kar A, Choudhary BK, Bandyopadhyay NG. Comparative evaluation of hypoglycaemic activity of some Indian medicinal plants in alloxan diabetic rats. J Ethnopharmacol. 2003; 84(1):105-8. doi: 10.1016/s0378-8741(02)00144-7, PMID 12499084.
9. Frankish N, de Sousa Menezes F, Mills C, Sheridan H. Enhancement of insulin release from the beta-cell line INS-1 by an ethanolic extract of Bauhinia variegata and its major constituent rose side. Planta Med. 2010; 76(10):995-7. doi: 10.1055/s-0029-1240868, PMID 20143296.
10. Swain LA, Downum KR. Light-activated toxins of the Moraceae. Biochem Syst Ecol. 1990; 18(2-3):153-6. doi: 10.1016/0305-1978(90)90052-H.
11. Mishra A, Kumar S, Bhargava A, Sharma B, Pandey AK. Studies on in vitro antioxidant and antistaphylococcal activities of some important medicinal plants. Cell Mol Biol (Noisy-le-grand). 2011; 57(1):16-25. PMID 21366958.
12. Zhao Y, Ye W, Boye KS, Holcombe JH, Hall JA, Swindle R. Prevalence of other diabetes-associated complications and comorbidities and its impact on health care charges among patients with diabetic neuropathy. J Diabetes Complications. 2010; 24(1):9-19. doi: 10.1016/j.jdiacomp.2008.09.001, PMID 18930413.
13. Taylor VH, MacQueen GM. Cognitive dysfunction associated with metabolic syndrome. Obes Rev. 2007; 8(5):409-18. doi: 10.1111/j.1467-789X.2007.00401.x, PMID 17716298.
14. Biessels GJ, Kappelle AC, Bravenboer B, Erkelens DW, Gispen WH. Cerebral function in diabetes mellitus. Diabetologia. 1994; 37(7):643-50. doi: 10.1007/BF00417687, PMID 7958534.
15. Singh N, Armstrong DG, Lipsky BA. Preventing foot ulcers in patients with diabetes. JAMA. 2005; 293(2):217-28. doi: 10.1001/jama.293.2.217, PMID 15644549.
16. Simmons Z, Feldman EL, Williams R, Herman W, Kinmonth AL, Wareham NJ. Treatment of diabetic neuropathy. The Evid Base Diabetes Care. 2002:555-76.
17. Wernicke JF, Pritchett YL, D'Souza DN, Waninger A, Tran P, Iyengar S, Raskin J. A randomized controlled trial of duloxetine in diabetic peripheral neuropathic pain. Neurology. 2006; 67(8):1411-20. doi: 10.1212/01.wnl.0000240225.04000.1a, PMID 17060567.
18. Kochar DK, Rawat N, Agrawal RP, Vyas A, Beniwal R, and Kochar SK, Garg P. Sodium valproate for painful diabetic neuropathy: a randomized double-blind placebo- controlled study. Qjm. 2004; 97(1):33-8. doi: 10.1093/qjmed/hch007, PMID 14702509.
19. Dallocchio C, Buffa C, Mazzarello P, Chiroli S. Gabapentin vs. amitriptyline in painful diabetic neuropathy: an open-label pilot study. J Pain Symptom Manage. 2000; 20(4):280-5. doi: 10.1016/s0885-3924(00)00181-0, PMID 11027910.
20. Dworkin RH, O'Connor AB, Backonja M, Farrar JT, Finnerup NB, Jensen TS, Kalso EA, Loeser JD, Miaskowski C, Nurmikko TJ, Portenoy RK, Rice ASC, Stacey BR, Treede RD, Turk DC, Wallace MS. Pharmacologic management of neuropathic pain: evidence-based recommendations. Pain. 2007b; 132(3):237-51. doi: 10.1016/j.pain.2007.08.033, PMID 17920770.
21. Effect of treatment with capsaicin on daily activities of patients with painful diabetic neuropathy. Capsaicin Study Group. Diabetes Care. 1992; 15(2):159-65. doi: 10.2337/diacare.15.2.159, PMID 1547671.
22. Barbano RL, Herrmann DN, Hart-Gouleau S, Pennella-Vaughan J, Lodewick PA, Dworkin RH. Effectiveness, tolerability, and impact on quality of life of the 5% lidocaine patch in diabetic polyneuropathy. Arch Neurol. 2004; 61(6):914-8. doi: 10.1001/archneur.61.6.914, PMID 15210530.
23. Rao UM, Sreenivasulu M, Chengaiah B, Reddy JK, Madhusudhana C. The ayurvedic formulary of India, page no-919. 24. Chetty Herb Med Diabetes Mellitus Rev IJPTR. 2010; 2(3):1883-92.

24. Nikhil K. Sachan, YatindraKumar, SeemaPushkar, R.N. Thakur, Sudhir.
25. Purohit, Prajapati. A hand book of medicinal plants: a complete source book. Longman orient, Indian medicinal plants (a compodium of 500 species). New York: Longman Pvt Ltd.; 2002.
26. Yognarasimhan SN. Medicinal plants of India Vol. 2nd, Tamil nadu: interline publishing pvt Ltd., Banglore& dehradun. India; 2000.
27. Khandelwal. Practical pharmacognosy. 19th ed. Pune: Niraliprakashan; 2008.
28. Husain A, Virmani OP, Popli SP, Misra LN, Gupta MM, Srivastava GN, et al. Dictionary of Indian medicinal. Plants Luck now, India: CIMAP; 1992. p. 546.
29. Joseph, Baby, Raj SJ. Phytopharmacological and phytochemical properties of three Ficus species-an overview. Int J Pharm Biol Sci. 2010:246-53.
30. Agarwal V, Chauhan BM. A study on composition and hypolipidemic effect of dietary fibre from some plant foods. Plant Food Hum Nutr. 1988; 38(2):189-97. doi: 10.1007/BF01091723, PMID 189197.
31. Khan N, Sultana S. Chemomodulatory effect of Ficus racemosa extract against chemically induced renal carcinogenesis and oxidative damage response in Wistar rats. Life Sci. 2005a; 77(11):1194-210. doi: 10.1016/j.lfs.2004.12.041, PMID 15885707.
32. Biswas TK, Mukherjee B. Plant medicines of Indian origin for wound healing activity: a review. Int J Low Extrem Wounds. 2003; 2(1):25-39. doi: 10.1177/1534734603002001006, PMID 15866825.
33. Veerapur VP, Prabhakar KR, Parihar VK, Kandadi MR, Ramakrishana S, Mishra B, Satish Rao BS, Srinivasan KK, Priyadarsini KI, Unnikrishnan MK. Ficus racemosa stem bark extract: a potent antioxidant and a probable natural radioprotector. Evid Based Complement Alternat Med. 2009;6(3):317-24. doi: 10.1093/ecam/nem119, PMID 18955240.
34. Ahmed F, Urooj A. Anticholinesterase activities of cold and hot aqueous extracts of F. racemosa stem bark. Pharmacogn Mag. 2010;6(22):142-4. doi: 10.4103/0973-1296.62897, PMID 20668582.
35. Malairajan P, Geetha Gopalakrishnan, Narasimhan S, Jessi Kala Veni K. Analgesic activity of some Indian medicinal plants. Journal of Ethnopharmacology. 2006;106(3):425-8. doi: 10.1016/j.jep.2006.03.015.

How to cite this article: Syed Iqra Naznin, Tarif hussain, Govind Pawar. Pharmacological evaluation of polyherbal formulation for neuroprotective effect. Int J of Allied Med Sci and Clin Res 2021; 9(2): 251-258.

Source of Support: Nil. **Conflict of Interest:** None declared.