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INTEGRATED *IN-VITRO* & *IN-VIVO* PHARMACOLOGICAL EVALUATION OF ANTI-PARKINSONIAN ACTIVITY OF COMBINED ETHANOLIC EXTRACTS OF *PHYSALIS MINIMA* AND *PERONEMA CANESCENS*

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Keywords:

Parkinson's disease, Neurodegeneration, *Physalis minima*, *Peronema canescens*, Haloperidol, Levodopa

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ABSTRACT: This study investigates the anti-Parkinsonian properties of ethanolic extracts derived from *Physalis minima* and *Peronema canescens*. Efficacy was assessed using a combination of *in-vitro* biochemical assays and *in-vivo* behavioural screening models in Swiss albino mice. Parkinson's disease symptoms were induced and subsequently evaluated using the body swing test. Oral administration of the extracts, individually and in combination, noticeably reduced behavioural abnormalities associated with Parkinsonism. Treated groups exhibited significant increases in monoamine neurotransmitters, specifically dopamine (DA), compared to control groups (DA levels approximately 0.35 vs. 0.05). Furthermore, the extracts enhanced the endogenous antioxidant defence by elevating crucial biomarkers, including glutathione (GSH), glutathione peroxidase (GPX), catalase (CAT), and superoxide dismutase (SOD). These biochemical findings were complemented by histological investigations of brain tissue. The results indicate that both *Physalis minima* and *Peronema canescens* extracts possess considerable neuroprotective and anti-parkinsonian activity, demonstrating an effect comparable to conventional standard pharmaceutical treatments like Levodopa. The study concludes that these plant extracts warrant further investigation as potential natural therapeutic agents for managing Parkinson's disease symptoms.

INTRODUCTION: Parkinson's disease (PD) is a chronic and progressive neurodegenerative disorder, currently the second most common worldwide, characterized primarily by the loss of dopaminergic neurons in the brain's Substantia nigra region [1].

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This neuronal degeneration leads to a significant dopamine deficit, resulting in characteristic motor symptoms such as tremors, rigidity, and bradykinesia (slowness of movement) [2].

While current pharmacological treatments, notably levodopa, remain highly effective for symptomatic relief, they do not halt disease progression and are often associated with long-term motor complications like dyskinesias. The complex pathophysiology of PD, involving oxidative stress, neuroinflammation, mitochondrial dysfunction, and the accumulation of misfolded alpha-synuclein proteins into Lewy bodies, highlights the urgent

need for novel therapeutic agents, particularly those with neuroprotective capabilities [3]. Medicinal plants and their derived natural products have emerged as a promising source for developing alternative and adjunctive therapies in PD management due to their multifaceted mechanisms of action, including potent antioxidant and anti-inflammatory properties.

Traditional medicine systems have long utilized botanical compounds to manage various neurological conditions. The bioactive constituents in these plants, such as flavonoids, alkaloids, and phenolic compounds, offer potential avenues to target the underlying pathological pathways of PD, such as scavenging free radicals, inhibiting inflammatory mediators, and promoting neuronal survival. Preclinical studies have identified numerous phytochemicals that demonstrate significant neuroprotective effects in animal and cellular models of PD by modulating key signaling pathways like the Nrf2/ARE antioxidant system and inhibiting apoptosis.

This paper reviews the pharmacological and traditional uses of two such plants: *Physalis minima (Pygmy Groundcherry) and Peronema canescens (Sungkai). Physalis minima, a species of* the Solanaceae family [4], is rich in withanolides and flavonoids and has been reported to possess anti-inflammatory, antioxidant, and acetylcholinesterase inhibitory activities [5, 6,] suggesting a potential role in improving cognitive function and reducing neuronal damage. The anti-inflammatory activities of its withanolides, for instance, are noted for acting on NF-κB, STAT3, and HO-1 pathways in cellular models [8].

Peronema canescens, belonging to the Lamiaceae family [4], contains valuable diterpenoids and phenolic compounds, employed traditionally for malaria, fever, and immune system support [4, 9]. Recent studies highlight its potent immunomodulatory, anti-inflammatory, and antidiabetic activities, linking specific compounds to mechanisms like DPP-4 inhibition in diabetic models [9, 10, 11]. This combination aims to boosts the therapeutic power, reduce side effects and explore the existing knowledge on the phytochemical profiles, report pharmacological activities, assessing their potential as sources of neuroprotective agents to supplement current therapeutic strategies for Parkinson's disease, while also considering challenges such as bioavailability and the need for rigorous clinical validation.

Aim and Objectives:

Aim: The study is aimed at evaluating the anti-parkinsonian activity of the combination of ethanolic extracts of *Physalis minima* and *Peronema canescens* using rats as an experimental model.

MATERIALS AND METHODS:

Collection & Authentication of Plant Material: The leaf powder of *Physalis minima* and *Peronema canescens* is obtained and authenticated by the botanist Dr. K. Madhava Chetty, Assistant Professor, Department of Botany, Sri Venkateswara University, Tirupati, Andhra Pradesh, India.

Chemicals & Materials Required:

Apparatus: Porcelain jars, alluminium foil, muslin cloth, beakers, petri dish.

Chemicals: 99% ethanol & other required chemicals to carry out phytochemical screening tests.

Drugs: Plant test drug, standard drug (Levodopa) & toxicant (Haloperidol).

Dose Preparation: Levodopa 1mg/kg of animal i.e., 0.25mg dissolve in 1ml of normal saline (0.9 w/v) at PH-7 *Haloperidol 1mg/kg i.e., 0.25mg dissolve in 1ml of normal saline (0.9 w/v) at PH-7

Animals Required: Swiss Albino mice weighing around 18-25 g.

Plants Extraction by Maceration Technique: The leaves of *Physalis minima* and *Peronema canescens* were dried at room temperature before the extraction procedure was carried out. Then the dried plants were crumbled into powder form and transferred in jars, ethanol was added, stirred regularly for 7 days. The jars are wrapped with aluminium foil near their mouth. After one week, the plant powders are sieved using a muslin cloth. After filtration, evaporation of ethanol gave a thick consistency to the plant mixture. Thus, plant extracts are ready for experimentation.

FIG. 1: EXTRACTION BY MACERATION TECHNIQUE

Phytochemical Screening Tests: Phytochemical evaluation of ethanolic extracts of plants were carried out to check the presence of biologically active constituents for anti-Parkinson's activity [12].

Sample Size Determination: The sample size for this study was calculated using power analysis (G*Power, version 3.1) with 80% power ($\alpha = 0.05$) and an expected medium effect size (Cohen's f = 0.25). Based on this analysis, each group required 6 animals to detect statistically significant differences in lipid parameters among the experimental groups. This resulted in a total of 54 animals across 9 groups for the main study (6 animals per group × 9 groups).

Acute Toxicity Studies: OECD guidelines 423 (Up and Down Procedure) were followed for conducting toxicological information to determine the safe dose. The animals were split into 2 groups of 3animals each, then the biological extract was given orally in increasing doses of about 100, 200, 500, 1000, and 2000 mg/kg of body weight. After 48 hrs animals are looked for death/toxicity signs. No differences were observed in the body, skin, fur/eyes of the animal after giving plant doses [13].

Experimental Animals: Experimental studies conducted at Shadan Women's College of Pharmacy. Swiss albino mice were kept under a good amount of light i.e 12 hrs of light and dark cycles. Animals are provided with a regular pellet diet, water. The whole experiment was carried out according to ethical standards & CPCSEA guidelines IAEC-07/SES-2025/41/106.

Experimental Design:

Inducing Parkinson's disease: Disease is induced in mice by giving a toxic dose of the drug to animals i.p. The toxic control was prepared freshly in normal saline, given at a single dose. The development of Parkinson's is confirmed by animal screening models, and the efficacy of plants is also determined.

The use of 54 Swiss Albino mice was done. It is divided into 9 groups of 6 mice each.

TABLE 1: GROUPING OF ANIMALS

Groups	Treatment	Dose & Route
G-1	Normal saline	10ml/100g p.o
G-2	Toxic control	1mg/kg i.p
G -3	Toxic control+ Standard drug	1ml/kg i.p

G-4	Toxic control+E.E of Plant-1	200mg/kg p.o
G-5	Toxic control+ E.E of Plant-1	400mg/kg p.o
G-6	Toxic control+E.E of Plant-2	200mg/kg p.o
G-7	Toxic control+ E.E of Plant-2	400mg/kg p.o
G -8	Toxic control+E.E of Plant-1+E.Eof Plant-2	200mg/kg p.o
G-9	Toxic control+E.E of Plant-1+E.Eof Plant-2	400mg/kg p.o

Plant 1: Ethanolic extract of *Physalis minima*. Plant 2: Ethanolic extract of *Peronema canescens*. Toxic control: Haloperidol Standard drug: Levodopa.

Experimental Methods:

Gas Chromatography (GC-MS):

Principle: Gas chromatography and mass spectrometry combines two analytical techniques of separating mixtures by GC and identifying components by MS-GC separates compounds within a temperature limit. Modern GC instruments involve use of an oven, and by adjusting the temperature, compounds can be analysed by retention time. MS is paired with GC, undergo ionisation and generate charged fragments. These ions are sorted and even unstable components are also detected.

Procedure: The sample for identification is passed from the GC entry point, where it is vaporised and passed into the chromatography column by gas.

After passing through the column, sample gets separated.

Later, the column passes into heat transfer, where compounds are converted into ions and detected by charge-to-mass ratio.

Applications:

- It is used for food service analysis.
- It's involved in chemical and elemental analysis.
- It is used in environmental assessment.
- It is helpful for refinery purposes.
- It's found in forensic analysis [14].

***In-vivo* Screening Methods:**

Elevated Body Swing Test:

- In this method, Animals are divided into groups as mentioned earlier.
- Rats in the Test group receive anesthesia with pentobarbital (60mg/kg i.p).
- Haloperidol is injected.
- After 7 days, 14 days, 21 days behavioural testing is carried out.
- The control group animals are placed in a box but without inducing agent.
- After the required time period all the animals are subjected for testing.
- The rat is gently lifted by the base of the tail and held at 2.5cms above the testing surface, swings are recorded.
- Before another swing the animal must be in a vertical position.
- The swings are recorded for 60sec.

FIG. 2: BODY SWING TEST

Evaluation: The total number of swings is recorded. The no. of left side swings and the number of right-side swings are recorded, compared with standard [16].

Biochemical Analyses of Brain Tissue: Following behavioral assessments on day 22, the animals were sacrificed, and their brains collected for various biochemical evaluations.

Tissue Preparation (Homogenization): Collected brain samples were homogenized in a phosphate buffer. The resulting mixture was subjected to high-speed centrifugation (between 15,000 and 25,000 rpm) for 25 minutes. The clarified supernatant was carefully collected and reserved for subsequent analytical tests.

Measurement of Lipid Peroxidation (MDA Assay): Lipid peroxidation was assessed by quantifying the concentration of malondialdehyde (MDA), a key biomarker of oxidative damage. The tissue homogenate was combined with acetic acid (20% concentration), dodecyl sulfate (8% concentration), and thiobarbituric acid (0.8% concentration). This reaction mixture was heated to 90°C for 60 minutes, subsequently cooled, and then centrifuged at a speed of 1500–2000 rpm for 15 minutes. Absorbance readings were taken using a spectrophotometer set at 532 nm. MDA production levels were calculated and reported in nmol/g of tissue.

Quantification of Reduced Glutathione (GSH): The level of reduced glutathione (GSH) was measured using a standard colorimetric method. A sample of the homogenate (0.1 mL) was mixed with 10% trichloroacetic acid and Ellman's reagent (dithiobis-nitrobenzoic acid prepared in a sodium citrate and phosphate buffer solution). After mixing, the solution was centrifuged at 2000 rpm for 15 minutes. The absorbance of the resulting supernatant was measured at 412 nm using a UV spectrophotometer. Results were presented as nmol GSH per gram of tissue.

Superoxide Dismutase (SOD) Activity Assay: Superoxide dismutase (SOD) activity was determined based on its capacity to inhibit the reduction of nitroblue tetrazolium (NBT) by superoxide radicals. A reaction mixture containing NBT, phosphate buffer, supernatant, and xanthine oxidase was incubated. Enzyme activity was calculated based on the degree of NBT reduction inhibition, where one unit of SOD activity corresponds to the amount of protein that inhibits NBT reduction by 50%. Results were expressed as units per milligram of protein.

Catalase (CAT) Activity Assay: Catalase activity was assessed by measuring the rate of hydrogen peroxide H_2O_2 degradation. To a cuvette containing phosphate buffer (2 mL) and H_2O_2 (1 mL), 0.5 mL of the brain supernatant was added. The decrease in absorbance was monitored at 240 nm over a 30-second interval using a spectrophotometer. Activity was calculated as mmoles of H2O2oxidized per minute per milligram of protein.

Neurotransmitter Profiling: The concentrations of key neurotransmitters and their metabolites including dopamine (DA), 3,4-dihydroxyphenylacetic acid (DOPAC), homovanillic acid (HVA), and 5-hydroxyindoleacetic acid (5-HIAA) were determined using commercially available Rat Enzyme-Linked Immunosorbent Assay (ELISA) kits. All assays were conducted strictly according to the manufacturer's provided protocols.

Neuroinflammatory Marker Analysis: The levels of specific pro-inflammatory cytokines, namely Interleukin-6 (IL-6), Interleukin-1 beta (IL-1), and Tumor Necrosis Factor-alpha (TNF-alpha), were measured using respective commercial ELISA kits. Protein was extracted from the brain homogenate, and samples were loaded into antibody-coated ELISA plates. The concentration of each cytokine was quantified following the standard assay procedures [17].

Histopathological Evaluation: Mice were made unconscious and dissected by using chloroform as anaesthetic. For lab examination isolated brains are preserved in formalin solution. Histopathological assessment is done to evaluate parameters.

RESULTS:

Calculation of Percentage Yield:

***Physalis minima*:** Weight of plant ethanolic extract: 130gms; Weight of plant crude extract: 500gms.

Formula used for calculation of % yield is:

$$\text{Wt of sample / Wt of crude extract} \times 100$$

$$\% \text{ yield} = 130 / 500 \times 100 = 26\%$$

Hence, % yield of *Physalis minima* is 26%.

***Peronema canescens*:** Weight of plant ethanolic extract: 122gms, weight of plant crude extract: 500gms.

Formula used for calculation of % yield is: Wt of sample/Wt of crude extract ×100

% yield = 120 / 500 × 100 = 24.4%. Hence, %yield of *Peronema canescens* is 24.4%.

TABLE 2: PHYTOCHEMICAL SCREENING RESULTS OF *PHYSALIS MINIMA* AND *PERONEMA CANESCENS*

S. no.	Phytoconstituents	Tests	E.E of *Physalis minima*	E.E of *Peronema canescens*
1	Alkaloids	Dragendroff's test	+	++
		Hager's test	++	-
		Wagner's test	-	+
		Mayer's test	++	++
		Barfoed's test	+	-
2	Glycoside	Legals test	-	++
		NaOH 10% test	+	+
3	Carbohydrates	Benedict's test	+++	++
		Molisch test	+	-
		Seliwanoff's test	+	+
4	Flavonoids	Bortrangers test	+++	+
		Shinoda test	+	+
		Ferric cl test	++	+++
5	Terpenoids	Salkowski's test	++	+++
		Bromine H2O test	++	++
6	Tannins	L. Burchards test	+++	+++
7	Sterols	Salkowski's test	+	++
		Foam test	++	-
8	Saponins	Sodium OH test	+	+
9	Coumadin's	lodine test	+++	++
10	Phenols	Ferric cl test	++	++
		Pb acetate test	+	+

GC-MS Results:

GCMS Graph of *Physalis minima*:

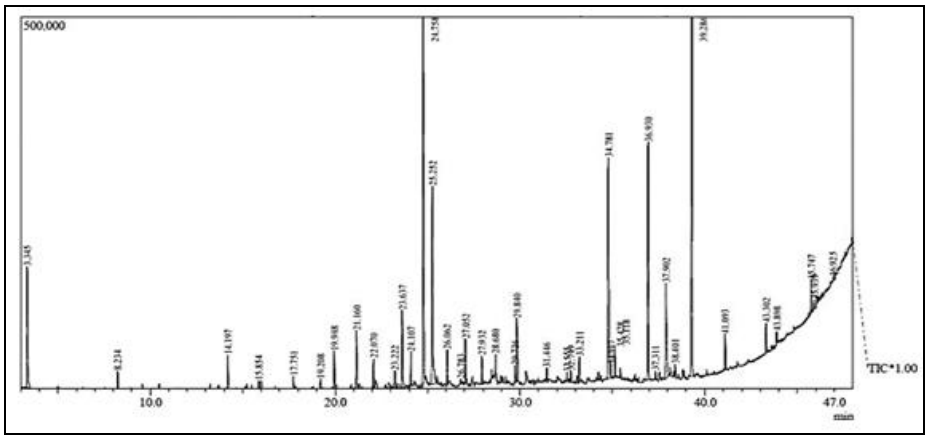

FIG. 3: PHYTOCHEMICAL TENTATIVELY IDENTIFIED BASED ON RETENTION TIME MATCHING IN ETHANOLIC EXTRACTS OF *PHYSALIS MINIMA* BY GC-MS

TABLE 3: PHYTOCHEMICALS TENTATIVELY IDENTIFIED BASED ON RETENTION TIME MATCHING IN ETHANOLIC EXTRACTS OF *PHYSALIS MINIMA* BY GC-MS

S. no.	Retention Time	Database	Applications
1	8.718	Withanolides	Parkinson's, Anti-cancer, Antiinflammatiory
2	9.878	Gallic acid	Neuroprotective, Anti-oxidant, anti-inflammatory
3	13.948	Ellagic acid	Psychotic, Anti-oxidant, anti mutagenic
4	17.719	Tocopherol	Various neurological disorders, vit E
5	17.896	Phytol	Neuroprotective, anxiolytics

GC-MS Graph of *Peronema canescens*:

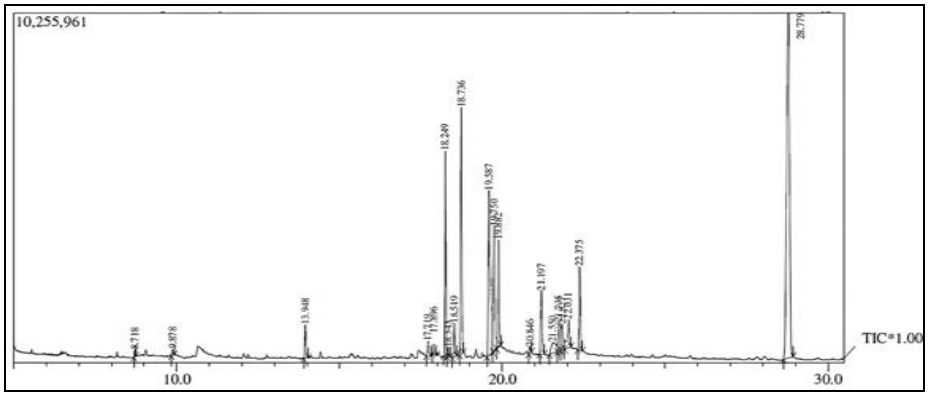

FIG. 4: PHYTOCHEMICAL TENTATIVELY IDENTIFIED BASED ON RETENTION TIME MATCHING IN ETHANOLIC EXTRACTS OF *PERONEMA CANESCENS* BY GC-MS

TABLE 4: PHYTOCHEMICAL TENTATIVELY IDENTIFIED BASED ON RETENTION TIME MATCHING IN ETHANOLIC EXTRACTS OF *PERONEMA CANESCENS* BY GC-MS

S. no.	Retention Time	Database	Applications
1	8.718	Kaempherol	Neurodegeneration, Anti-cancer
2	9.878	Cleodeanediterpinoids	Parkinson's, antimicrobial, antitumor
3	13.948	Apegenine	Treat hypertension, psychotic issues
4	17.719	Squalene	Neuroprotection, skin protection
5	17.896	Catechol	Anti-psychotic, anti-oxidant, chelating agent

In-vitro Results:

Effects of Plant Extract in Haloperidol Induced Parkinson's disease:

TABLE 5: EFFECTS OF PLANT EXTRACT IN HALOPERIDOL INDUCED PARKINSON'S

Groups	Doses	SOD (µg/g)	Catalase (mg/g)	GSH (µu/mg)	MDA (µmole/mg of protein)
Normal control (Vehicle)	1 mL/kg	6.362 ± 0.007	548.337 ± 0.007	1.807 ± 0.003	173.143 ± 0.012
Disease control (Haloperidol)	1 mg/kg	3.928 ± 0.006	398.810 ± 0.005	0.747 ± 0.004	309.755 ± 0.008
Standard (L-Dopa + Carbidopa)	100 mg/kg +25 mg/kg	5.725 ± 0.002***	484.460 ± 0.010***	1.268 ± 0.005***	210.675 ± 0.005***
Extract	200 mg/kg	5.167 ± 0.003***	401.067 ± 0.007***	1.078 ± 0.005***	310.017 ± 0.004**
Extract	400 mg/kg	5.353 ± 0.003***	424.958 ± 0.008***	1.160 ± 0.004***	263.262 ± 0.011***
Extract	600 mg/kg	6.315 ± 0.004***	430.665 ± 0.010***	1.218 ± 0.003***	241.955 ± 0.011***

The values are expressed as mean ± SEM, (*n*=6). ***p << 0.001 *vs*. normal control. **p << 0.001 *vs* disease control group.

In-vivo Results:

Physiological Parameters:

Body Weight:

TABLE 6: EFFECTS OF ETHANOLIC EXTRACTS OF PHYSALIS MINIMA AND *PERONEMA CANESCENS* ON BODY WEIGHT

Groups	Body Weight		
	7th Day	14th Day	21st Day
G-1	223.41±2.91	233.33±1.93	244.45±3.91
G -2	206.33±2.23	196.50±2.47	184.83±2.83
G-3	220.43±3.45	220.83±4.69	222.33±3.47
G -4	212.45±0.05	200.33±0.05	204.33±0.33
G-5	213.33±0.07	212.22±0.06	215.22±0.75
G-6	215.42±0.09	214.32±0.07	217.32±0.33
G-7	216.33±0.07	216.45±0.09	219.22±0.56
G -8	217.35±0.08	217.75±0.66	221.33±0.75
G-9	218.32±0.07	219.33±0.33	222.00±0.38

The above values are expressed in mean ±SEM *P≤0.05,**P≤0.01,***P≤0.001 compared to control group of mice (done by two-way ANNOVA).

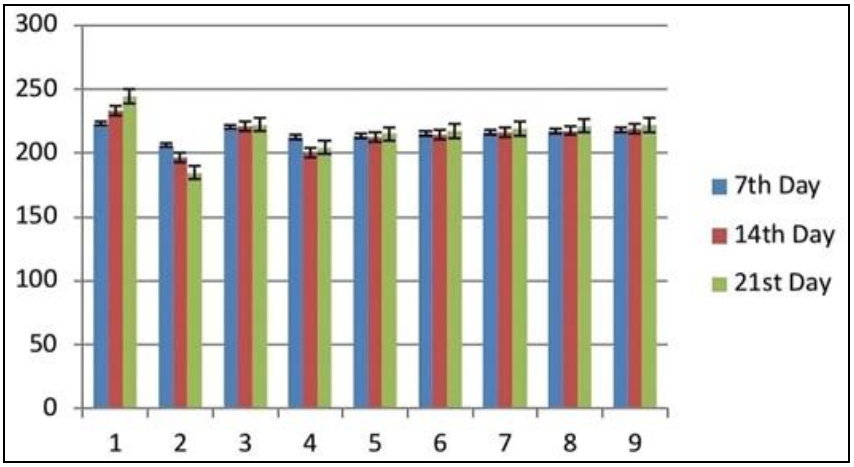

FIG. 5: SHOWING EFFECTS OF E.E.P.M & E.E.P.C. ON BODY WEIGHT

Biochemical Estimations in Blood Sample:

TABLE 7: IMMUNOMODULATORS LEVELS IN BLOODS

Groups	TNF-Alpha (pg/ml)	IL-6 (pg/ml)	IL-10 (pg/ml)
G-1	17.06±3.67	33.00±5.63	819±9.9
G-2	33.09±4.83	176.60±2.70	96.11±3.3
G -3	19.40±3.62	70.49±1.13	533.21±4.1
G-4	30.80±2.98	117.20±1.92	183±3.4
G-5	28.75±0.05	103.02±0.05	429±0.05
G -6	26.55±0.85	90.51±0.06	436±0.06
G-7	22.06±0.07	78.63±0.28	490±0.07
G-8	20.55±0.09	75.55±0.03	491±0.66
G -9	19.01±0.07	81.06±0.08	500±0.75

The data was analysed by 2way ANOVA test & expressed as means ±SEM,*P≤0.01 is compared to non stressed+standard vehicle groups, **P≤0.001 compared to the stressed+control group.

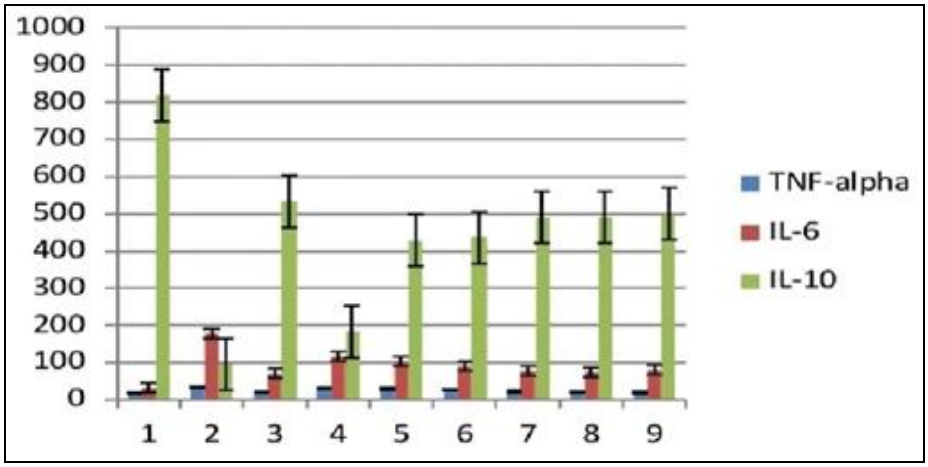

FIG. 6: SHOWING EFFECTS OF E.E.P.M & E.E.P.C AND THEIR COMBINATION IN *MICE* ABOUT TNF, IL LEVELS

TABLE 8: EFFECTS OF ETHANOLIC EXTRACTS OF *PHYSALIS MINIMA* AND *PERONEMA CANESCENS* AND THEIR COMBINATION IN *MICE* ABOUT DA, GSH, GPX LEVELS

Groups	Dopamine Levels (pg/ml)	GSH (mM)	GPX (U/DL)
G-1	0.573±0.04	52.8±0.6	19.64±0.3
G-2	0.05±0.03	13.5±0.4	8.26±0.2
G -3	0.35±0.03	43.2±0.1	17.47±0.4
G-4	0.22±0.04	21.32±0.5	10.33±0.5
G-5	0.28±0.02	28.36±0.39	12.48±0.6
G-6	0.30±0.03	32.66±0.32	14.66±0.7

G-7	0.31±0.23	38.75±0.33	16.33±1.2
G -8	0.32±0.55	40.2±0.23	16.98±1.3
G -9	0.39±0.7	41.33±0.54	17.00±1.4

The above data was analysed by two way ANOVA test and expressed as means ±SEM,*P≤0.01 is compared to non stressed+standard vehicle groups, **P≤0.001 compared to the stressed+control group.

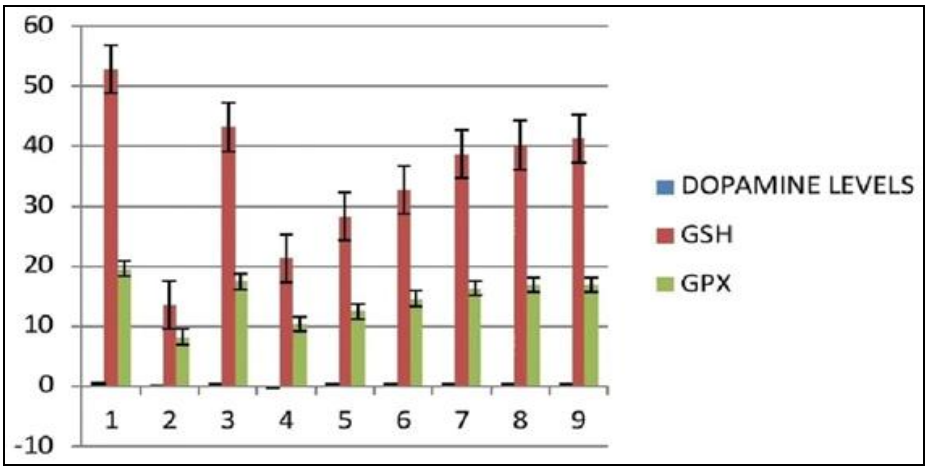

FIG. 7: SHOWING EFFECTS OF E.E.P.M & E.E.P.C AND THEIR COMBINATION IN MICE ABOUT DA, GSH, GPX LEVELS

Biochemical Estimations in Anti-Oxidant Activity:

TABLE 9: EFFECTS OF ETHANOLIC EXTRACTS OF *PHYSALIS MINIMA* AND *PERONEMA CANESCENS* AND THEIR COMBINATION IN *MICE* ABOUT ANTI-OXIDANT LEVELS

Groups	CAT (U/mg)	SOD (U/ml)	TBARS (nmol/mg)
G-1	26.70±0.52	52.23±0.061	17.7±0.22
G-2	12.01±0.61	14.44±0.35	42.2±0.44
G-3	24.97±0.57	47.57±0.27	21.8±0.27
G-4	16.33±0.78	20.35±0.33	30.33±0.45
G -5	18.45±0.33	29.18±0.61	28.45±0.32
G-6	20.46±0.39	30±0.33	24.66±0.33
G-7	22.36±0.29	35.09±0.35	22.32±0.67
G-8	23.46±0.45	39.33±0.48	20.39±0.32
G -9	24.40±3.33	40.33±0.45	20.00±0.39

The above data was analysed by two way ANOVA test and expressed as means ±SEM,*P≤0.01 is compared to non-stressed + standard vehicle groups, **P≤0.001 compared to the stressed+control group.

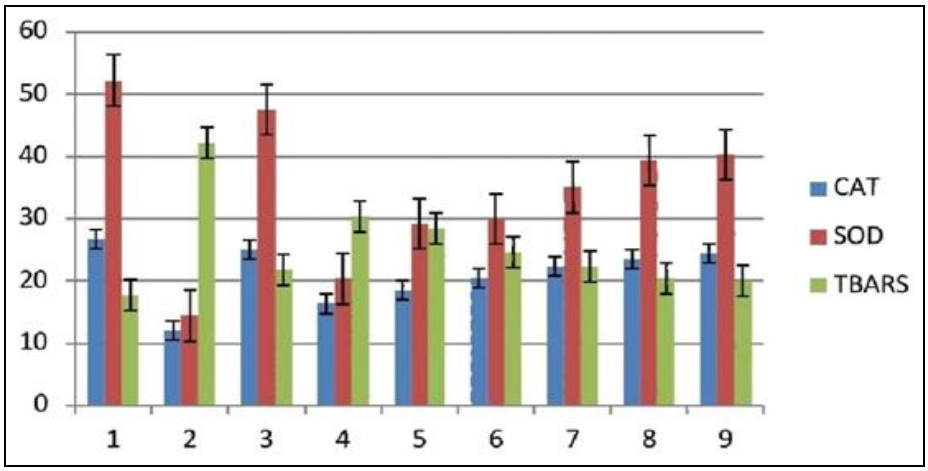

FIG. 8: SHOWING EFFECTS OF E.E.P.M & E.E.P.C AND THEIR COMBINATION IN MICE ABOUT ANTI-OXIDANT LEVELS

Histopathological Analysis:

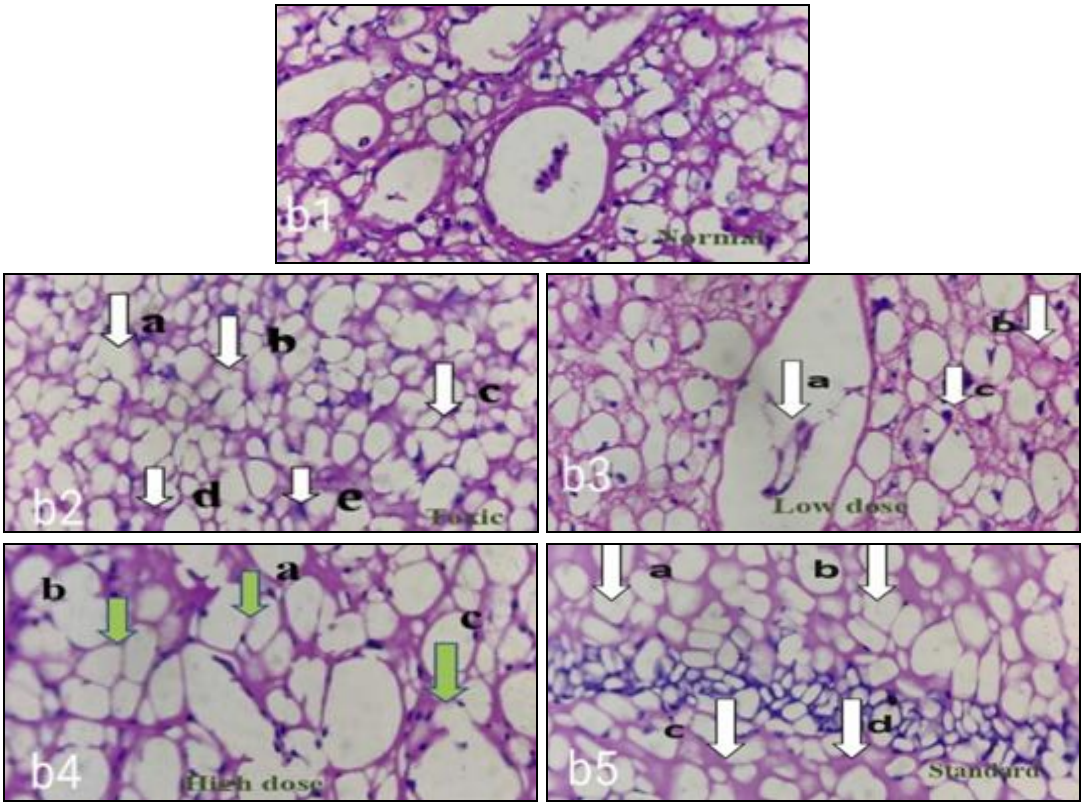

FIG. 9: CHANGES IN SUBSTANTIA NIAGARA OF BRAIN CELLS. B 1: Represents Normal control group, B 2: Represents Toxic control (Haloperidol) group, B 3: Represents E.E.P.M (plant 1) group, B 4: Represents E.E.P.C (Plant 2) group, B 5: Represents Standard control (Levodopa) group

The above photograph shows the changes in substantia nigra of the brain viewed at 400× Magnification and pathological sections are stained with Hematoxylin and Eosin (H&E). Histopathology of brain reveals ,(B1) There are no signs for neurodegeneration in normal control group, (B2) In toxic group neuronal damage, structural damage is observed,(B3) Upon treatment with E.E.P.M slight change in structure & growth occurs,(B4) Treatment with E.E.P.C leads to improvement in form, shape observed,(B5) Standard drug treatment leads to increase in pigmentation, firm structure is seen.

DISCUSSION: The use of various medicinal plants &herbs for the treatment leads to the development of healthcare system. In this study, anti-Parkinsons activity of *Physalis minima* & *Peronema canescens* is carried out. Ethanolic extraction of both plants were obtained by the maceration. Albino mice are animal model used, exposed to toxicant to induce PD (Haloperidol). Thus, the botanical extracts are evaluated by screening models, compared with standard drug. The animals are divided into 9 groups containing 6mice in each. The groups involves normal, toxic, standard, low & high doses of plant extract and lastly, combination of both plants. Before doses, ATS (acute toxicity studies) is carried out. After dosing behavioural, biochemical estimations has done by collecting blood samples, dissection & isolation of brain is performed for further results of study.

CONCLUSION: The present research evaluates ethanolic extracts of *P. minima* and *P. canescence* for their potential Anti-Parkinsonian effects. The phyto-chemicals tests shows positive response for phenols, flavonoids, Sterols, terpenoids, coumarins for Anti-parkinsons activity as according to previous research. *In-vivo* assessment using Body Swing Test is demonstrated. The effectiveness of plant & their combinations showed almost close response to standard drug. The behavioural & biochemical estimations reveals increase in the levels of dopamine, IL, GSH, CAT, SOD by plants *P. minima* & *P. cenescens* and results are similar to the standard drug. Histopathological assessment of treated mice brains, shows the decrease in loss & degeneration of neurons of substantia Niagara region which shows effectiveness of plants.

Hence, this study can be concluded with an impression that these plants exhibits great potential which can be utilised in future treatment of PD and other such brain related disorder.

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