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EVALUATION OF *in-vitro* ANTI PARKINSON'S ACTIVITY OF ETHANOLIC POD EXTRACT OF *Cyamopsis tetragonoloba (L).* IN NEUROTOXICITY CELL CULTURE MODEL

[1] Shunmuga Sundaram N, [2] V. Jenila Jose Jancy,

[1] PG Scholar , [2] Professor & Head,

[1,2] Department of Pharmacology,

[1,2] S.A Raja Pharmacy College, Vadakangulam, Tirunelveli, Tamil Nadu - 627116

Abstract: Parkinson's disease (PD) is a progressive neurodegenerative disorder characterized by selective degeneration of dopaminergic neurons and oxidative stress–mediated cellular damage. The present study aimed to evaluate the in-vitro anti-Parkinson's activity of ethanolic pod extract of *Cyamopsis tetragonoloba* (L.) using a neurotoxicity cell culture model. Phytochemical screening of the extract revealed the presence of flavonoids, alkaloids, phenolics, and saponins, which are known for their antioxidant and neuroprotective potential. In vitro neurotoxicity was induced in cultured neuronal cells using 6-hydroxydopamine (6-OHDA), and the protective effect of the extract was assessed through cell viability (MTT assay), intracellular reactive oxygen species (ROS) generation, and morphological analysis. Treatment with the extract significantly improved cell viability, reduced oxidative stress, and preserved neuronal morphology in a dose-dependent manner when compared with the toxic control. The findings suggest that the ethanolic pod extract of *Cyamopsis tetragonoloba* possesses promising neuroprotective potential, possibly due to its antioxidant phytoconstituents, and may serve as a natural lead for developing adjunct therapy against Parkinson's disease.

Keywords: *Cyamopsis tetragonoloba*, Parkinson's disease, neuroprotection, oxidative stress, cell culture.

Introduction

The central nervous system (CNS) of the human body is a network of more than 100 billion distinct nerve cells that direct our movements, detect our environment, and establish The brain and spinal cord make up the central nervous system (CNS). Neurons, which make up the CNS's functional units, are special in that they can transmit and store information while simultaneously being prone to damage. The brain and spinal cord collectively known as the central nervous system (CNS), serve as the body's control system, regulating thought, movement, sensations, and emotions. The CNS has hundreds of different neuroanatomical areas, many of which are referred to as "nuclei," and is commonly under sampled in microscopy. It is crucial for pathologists to recognise this diversity, become familiar with fundamental neuroanatomical landmarks, and understand how different brain regions respond differently to physical and/or chemical trauma, excitotoxicity, and other stresses. The pathologist must be able to distinguish between glial cells that are normal and those that are even more numerous in order to interpret cytologic alterations.There are anatomically distinct subtypes of each of the two categories of neurons, "small neurons" and "big neurons," which are used to describe neurons in general . Stroke, traumatic brain injury (TBI), spinal cord injury (SCI), and retinal degeneration are all examples of CNS injuries. These conditions affect millions of people worldwide and affect people of all ages. In the paediatric population, CNS injuries are mostly traumatic or congenital, whereas in the adult population, the injuries are traumatic or degenerative.

The CNS contains multiple neuron subtypes that form an intricate network of connections with surrounding cells such as other neurons and glia, as well as a complex organization that is difficult to replicate with current repair strategies.The importance of inflammation in neurodegenerative illnesses of the central nervous system (CNS), such as Alzheimer's disease, amyotrophic lateral sclerosis, Parkinson's disease, and the classic neuroinflammatory disease multiple sclerosis is becoming more widely acknowledged.

PARKINSON'S DISEASE

Parkinson's disease (PD) is a progressive neurodegenerative disorder both motor and non-motor manifestations. While current therapies focus on symptom management, research into disease modifying treatments offers hope for future breakthroughs. Early diagnosis and multidisciplinary care are crucial for optimizing patient outcomes. Primarily affecting movement due to the degeneration of dopaminergic neurons in the substantia nigra pars compacta (SNpc) of the midbrain. It is the second most common neurodegenerative disease after Alzheimer's, with an estimated prevalence of over 10 million people worldwide. PD is characterized by motor symptoms such as tremors, bradykinesia (slowed movement), rigidity, and postural instability, as well as non motor symptoms including cognitive decline, sleep disturbances, and autonomic dysfunction.

Pathophysiology:

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S.No	Pathological Mechanism	Description
1.	Loss of Dopaminergic Neurons	The degeneration of dopamine producing neurons in the substantia nigra pars compacta (SNpc) results in dopamine depletion in the striatum, disrupting the basal ganglia circuitry responsible for motor control. Clinical symptoms typically emerge after 60–80% of these neurons are lost.
2.	Lewy Bodies and Alpha Synuclein Aggregation	The accumulation of Lewy bodies, which consist mainly of misfolded alpha synuclein, is a hallmark of Parkinson's disease (PD). These protein aggregates propagate in a prion like fashion, driving disease progression.
3.	Neuroinflammation and Oxidative Stress	Microglial activation and mitochondrial dysfunction contribute to oxidative stress, amplifying neuronal injury. While genetic mutations (e.g., SNCA, LRRK2, PARKIN, PINK1) are implicated in familial PD, environmental exposures are associated with sporadic cases.

Table 1: Pathological Mechanism, Description of Parkinson's disease

Clinical Features of Parkinson's Disease

Parkinson's disease presents with both motor and non-motor symptoms. The hallmark motor features can be remembered by the TRAP Tremor, typically a resting "pill rolling" tremor, is often an early and noticeable sign. Rigidity refers to increased muscle tone, often manifesting as cogwheel rigidity during passive limb movement.

Akinesia or bradykinesia, characterized by slowness in initiating and executing movements, is a core feature. Postural instability develops in later stages, leading to impaired balance and an increased risk of falls. In addition to motor symptoms, Parkinson's disease involves several non-motor features. Cognitive impairments, including memory loss and executive dysfunction, may progress to Parkinson's disease

dementia (PDD). Psychiatric symptoms such as depression, anxiety, and hallucinations often medication induced are common. Autonomic dysfunction includes issues like constipation, orthostatic hypotension, and urinary incontinence. Sleep disturbances, particularly REM sleep behavior disorder (RBD) and excessive daytime sleepiness, are also frequently observed.

Diagnosis of Parkinson's Disease:

Parkinson's disease (PD) is primarily a clinical diagnosis, based on a detailed medical history and neurological examination. The UK Parkinson's Disease Society Brain Bank Criteria are commonly used and require the presence of bradykinesia along with at least one additional motor symptom, such as tremor or rigidity.

While clinical assessment remains central, imaging studies can aid in the diagnostic process. A DaTscan (Dopamine Transporter SPECT) is particularly useful in distinguishing PD from other movement disorders by visualizing dopamine transporter activity. MRI is often employed to exclude structural brain abnormalities such as stroke or tumors that may mimic PD symptoms. Additionally, research is ongoing into potential biomarkers, including alpha synuclein seed amplification assays (SAA) in cerebrospinal fluid or blood, which may improve diagnostic accuracy.

Treatment Strategies and Prognosis of Parkinson's Disease:
Pharmacological Therapy:

1. Levodopa + Carbidopa: Most effective treatment; increases brain dopamine levels. Long term use may cause dyskinesia's and motor fluctuations ("on off" phenomenon).
2. Dopamine Agonists (e.g., Pramipexole, Ropinirole): Used in early PD; may lead to impulse control disorders.
3. MAO B Inhibitors (e.g., Eelegiline, Rasagiline): Delay dopamine breakdown; provide mild symptom relief.
4. COMT Inhibitors (e.g., Entacapone): Prolong L DOPA effect by inhibiting peripheral metabolism.
5. Anticholinergics (e.g., Trihexyphenidyl): Help control tremor; limited by cognitive side effects, especially in elderly.

Surgical Intervention:

1. Deep Brain Stimulation (DBS) Electrodes implanted in STN or GPi improve motor symptoms in advanced PD.

Emerging Therapies:

1. Alpha synuclein Immunotherapy (e.g., Prasinezumab), Targets misfolded protein aggregates.
2. Stem Cell Therapy: Experimental use of dopaminergic neuron transplantation.
3. Gene Therapy: Targets GDNF to support neuron survival.

Prognosis: PD is progressive and incurable, but individualized treatment can greatly improve quality of life. With optimal care, life expectancy is near normal, although disability increases in advanced stages.

Importance of Exploring Plant Based Neuroprotective Agents:

Plant-based neuroprotective compounds offer several advantages over conventional synthetic drugs, making them promising candidates for neurodegenerative disease management with their multi-target mechanisms, where phytochemicals simultaneously modulate various pathways, including anti-inflammatory, antioxidant, and anti-apoptotic processes, unlike single-target synthetic drugs that often address only one aspect of neurodegeneration. Additionally, plant-derived compounds generally exhibit lower toxicity, with well-tolerated safety profiles, as seen in natural substances like curcumin and resveratrol. Their potent antioxidant and anti-inflammatory properties are particularly valuable, as they counteract oxidative stress and neuroinflammation—two major contributors to neuronal damage. Furthermore, these compounds are highly accessible and cost-effective, with many neuroprotective plants being widely available and deeply rooted in traditional medicine systems, allowing for easier integration into therapeutic strategies. This combination of broad-spectrum activity, safety, affordability, and cultural acceptance underscores the potential of plant-based compounds in neuroprotection.

Plant-based compounds have demonstrated significant neuroprotective effects through various mechanisms such as flavonoids (e.g., quercetin and epigallocatechin gallate (EGCG) from green tea), act as powerful antioxidants by scavenging reactive oxygen species (ROS), reducing oxidative stress in neuronal cells. Additionally, compounds like sulforaphane from cruciferous vegetables activate the Nrf2 pathway, which boosts the production of endogenous antioxidants like glutathione. Anti-inflammatory actions, curcumin from turmeric inhibits the NF-κB pathway, reducing pro-inflammatory cytokines such as TNF-α and IL-6, while ginsenosides from ginseng prevent excessive microglial activation, which is often implicated in neuroinflammation.

In terms of neuroprotection, compounds like bacopa monnieri enhance the expression of brain-derived neurotrophic factor (BDNF), promoting neuron survival, while Withania somnifera (ashwagandha) inhibits caspase-mediated apoptosis, preventing neuronal death. Additionally, EGCG and resveratrol have shown promise in modulating protein aggregation, specifically inhibiting the toxic aggregation of alpha-synuclein in Parkinson's disease and amyloid-beta in Alzheimer's disease. Plant-based compounds like Coenzyme Q10 support mitochondrial function, improving energy metabolism and protecting against mitochondrial dysfunction, a significance of neurodegenerative diseases.

Plant-based neuroprotective compounds

Plant / Compound	Plant / Source	Potential Benefits
Curcumin	Turmeric	Anti-inflammatory, reduces amyloid plaques (AD)
Resveratrol	Grapes, berries	Activates sirtuins, enhances autophagy
EGCG	Green tea	Antioxidant, prevents α-synuclein aggregation (PD)
Ginkgo biloba	Ginkgo leaves	Improves cerebral blood flow, cognitive function
Bacopa monnieri	Brahmi	Enhances memory, supports neurogenesis
Ashwagandha	*Withania somnifera*	Reduces stress, protects dopaminergic neurons
Cannabinoids	Cannabis	Neuroprotective, anti-inflammatory

Table 2: Plant-based neuroprotective compounds

Cyamopsis tetragonoloba (L).

Cyamopsis tetragonoloba (L). commonly known as guar, is a drought-tolerant legume traditionally cultivated in arid and semi-arid regions of India and Pakistan. Various parts of the guar plant have been used in folk medicine to treat ailments such as inflammation, diarrhea, dysentery, and diabetes. The seeds are the primary source of guar gum, a galactomannan polysaccharide widely employed as a natural thickening and stabilizing agent in food, pharmaceutical, and industrial applications. Beyond its industrial utility, guar exhibits significant phytochemical potential, containing bioactive compounds such as flavonoids, saponins, tannins, phenolic acids, and galactomannans, which contribute to its antioxidant, antimicrobial, antidiabetic, and hypocholesterolaemia properties. Recent studies suggest that guar gum may aid in glycemic control, lipid regulation, and gastrointestinal health, positioning it as a promising candidate for nutraceutical and functional food development. The plant's adaptability to harsh climates, coupled with its pharmacologically active constituents, highlights its dual value as both a traditional remedy and a phytochemical-rich resource in modern health sciences.

Figure 1: *Cyamopsis tetragonoloba (L).*

Kingdom	Plantae
Division	Magnoliophyta (Angiosperms)
Class	Magnoliopsida (Dicotyledons)
Order	Fabales
Family	Fabaceae (Leguminosae)
Genus	Cyamopsis
Species	C. tetragonoloba (L.) Taub.

Table 3: Scientific Classification

Pharmacological Activities of *Cyamopsis tetragonoloba (L).*

1. **Antioxidant Activity:** Contains flavonoids and polyphenols that scavenge free radicals. Protects cells from oxidative stress.
2. **Antidiabetic Activity:** Guar gum delays glucose absorption in the intestine. Helps in lowering postprandial blood sugar levels. Improves insulin sensitivity.
3. **Hypolipidemic / Antihyperlipidemic Activity:** Reduces total cholesterol, LDL, and triglycerides. Increases HDL levels, mainly attributed to dietary fibers in guar gum.
4. **Gastro protective and Laxative Effects:** Guar gum promotes bowel movement and helps relieve constipation. Forms a protective mucilage in the GI tract.
5. **Antimicrobial** Activity: Some extracts show inhibitory effects against bacteria and fungi. Activity attributed to phytoconstituents like flavonoids and saponins.
6. **Anti-inflammatory Activity:** Reduces inflammation markers. Useful in treating inflammatory bowel disease and joint inflammation.
7. **Anti-obesity Activity:** Guar gum increases satiety and delays gastric emptying. Helps reduce calorie intake and manage weight.
8. **Cardioprotective** Effects: Improves lipid profile and arterial health. Reduces risk factors associated with cardiovascular disease.
9. **Prebiotic Activity:** Fermentation of guar gum in the colon supports beneficial gut microbiota. Enhances gut health and immunity

METHODOLOGY

Plant Collection And Authentication:

The fruit of *Cyamopsis tetragonoloba (L).* was collected and authenticated.

1. The dried pods were grinded in room atmospheric condition, and the powdered pods were stored in an air tight container.
2. 100g of powdered pods were taken in condenser and 500ml of ethanol were taken in(RBF). After each cycle, the plant material was refluxed for 16 hours at 60°C to 100°C.
3. After each solvent each cycle, extracts were collected, evaporator and chilled to room temperature.
4. After drying, extracts were kept at 5°C in airtight container for one to two days.

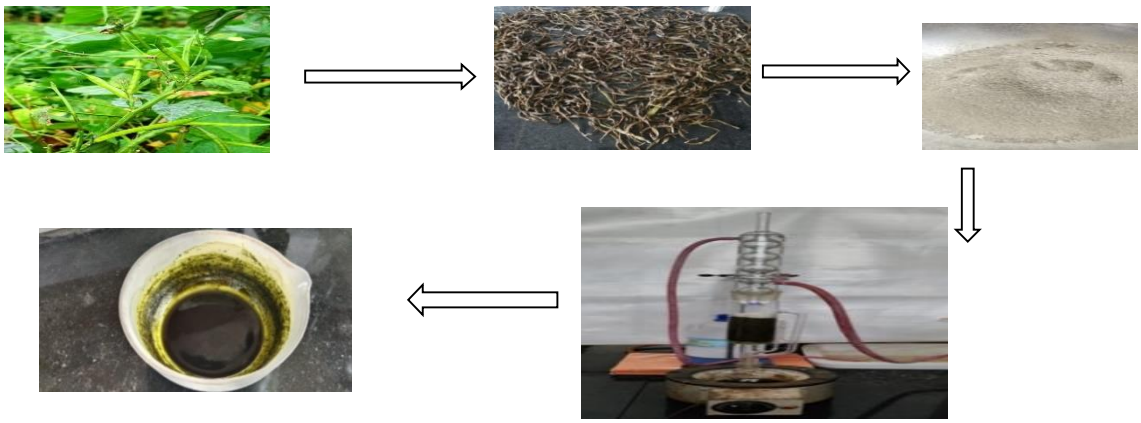


Figure 2: Picture explanation of Soxhlet extraction of *Cyamopsis tetragonoloba (L).*

Qualitative phytochemical analysis:

The pod extract of *Cyamopsis tetragonoloba (L).* subjected to the following chemical tests for the identification of its various active constituents.

Phytochemical Screening:

The phytochemical screening for ethanolic extract was done by using qualitative analysis. The qualitative test for alkaloids, flavonoids, saponins, steroids coumarins, and terpenoids was performed by using the following procedure

Alkaloids :

To 1 ml of extract was added with 2-3 drops of dragendroff‟s reagent .The appearance of orange or reddish orange in colour indicates the presence of alkaloids .

Terpenoids :

To 1ml of the extract was mixed with 2 ml of chloroform and concentrated H2SO4 (3ml) was carefully added to form a layer. A reddish brown colouration of the interface was formed to indicates positive results for the presence of terpenoids.

Flavonoids :

To 1ml of the extract was dissolved in diluted NaOH and HCL was added. A yellow solution that turns colourless indicates the presence of flavonoids.

Steroids:

To 1ml of extract was discovered in 10ml of chloroform and equal volume of concentrated sulphuric acid was added. The upper layer turns red and sulphuric acid layer showed yellow with green fluorescence. This indicates the presence of steroids.

Saponins:

To 1ml of extract was diluted with distilled water up to 3 ml. the suspension was then shaken in a test tube for 15 min formation of a two layer of foam indicated the presence of saponins.

Phytochemical Screening:

Phytochemical screening of the pod of *Cyamopsis tetragonoloba (L).* showed the presence of phenolics, flavanoids, saponins, terpenoids, tannins, amino acids, steroids and absence of alkaloids.

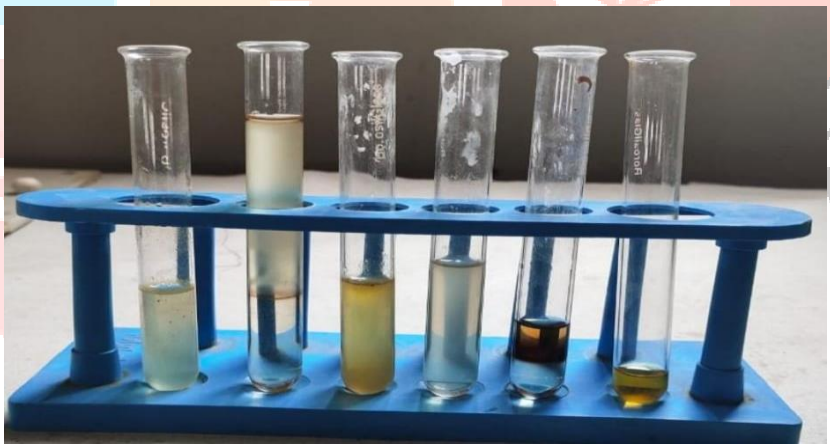


Figure 3: Phytochemical screening of ethanolic extract

Phytochemicals	**RESULT**
Steroids	+
Terpenoids	+
Saponin glycosides	+
Glycosides	+
Flavonoids	+
Tannins	+

Table 4: Phytochemical screening of *Cyamopsis tetragonoloba (L).*

GC-MS Analysis for *Cyamopsis tetragonoloba (L).* Pod Extracts

Ethanolic pod extract of *Cyamopsis tetragonoloba* (L). were prepared by using soxhlet extraction method for relevant period of time. All the extracts were stored in a container for further GC-MS analysis. GC-MS analysis of *Cyamopsis tetragonoloba* (L).pod extracts provides detailed insights into the phytochemical composition of this leguminous plant, commonly known as guar. The analysis helps identify various bioactive compounds, including fatty acids, esters, alcohols, hydrocarbons, and other secondary metabolites that may contribute to the plant's nutritional, medicinal, or industrial value. Through the separation and detection capabilities of gas chromatography and mass spectrometry, GC-MS enables the precise characterization of volatile constituents present in the pod extracts. These findings can be useful in evaluating the potential antioxidant, antimicrobial, and therapeutic properties of guar pods, supporting their application in food, pharmaceutical, and cosmetic industries. Furthermore, such analysis aids in quality control, standardization, and further phyto pharmacological research on C. tetragonoloba.

PEAK	RETENTION	START TIME	END TIME	m/z	AREA	AREA %	HEIGHT	HEIGHT	A/H
1	10.004	9.975	10.035	TIC	27214	5.7	15910	6.68	1.71
2	13.931	13.9	13.965	TIC	75659	15.85	42031	17.64	1.8
3	17.545	17.515	17.575	TIC	38636	8.09	22568	9.47	1.71
4	20.786	20.755	20.815	TIC	29286	6.13	18368	7.71	1.59
5	23.584	23.56	23.615	TIC	16043	3.36	10668	4.48	1.5
6	28.347	28.325	28.375	TIC	8972	1.88	4487	1.88	2
7	30.411	30.385	30.44	TIC	10789	2.26	5950	2.5	1.81
8	32.318	32.295	32.36	TIC	11164	2.34	7405	3.11	1.51
9	34.117	34.075	34.16	TIC	23690	4.96	11649	4.89	2.03
10	34.868	34.815	34.925	TIC	54612	11.44	22024	9.24	2.48
11	34.935	34.925	34.945	TIC	1690	0.35	2296	0.96	0.74
12	35.808	35.78	35.84	TIC	25543	5.35	12195	5.12	2.09
13	37.393	37.36	37.44	TIC	34416	7.21	14432	6.06	2.38
14	37.515	37.44	37.525	TIC	10853	2.27	2596	1.09	4.18
15	37.905	37.895	37.92	TIC	1679	0.35	1932	0.81	0.87
16	38.216	38.2	38.235	TIC	1648	0.35	2876	1.21	0.57
17	38.345	38.31	38.36	TIC	6728	1.41	4575	1.92	1.47
18	38.45	38.435	38.505	TIC	8207	1.72	2646	1.11	3.1
19	38.621	38.585	38.64	TIC	7670	1.61	3507	1.47	2.19
20	38.67	38.64	38.105	TIC	9709	2.03	4105	1.72	2.37
21	38.76	38.715	38.78	TIC	8772	1.84	3332	1.4	2.63
22	38.903	38.855	38.95	TIC	40724	8.53	13168	5.53	3.09
23	39.065	39.05	39.105	TIC	6560	1.37	3259	1.37	2.01
24	39.12	39.105	39.145	TIC	3473	0.73	2697	1.13	1.29
25	39.16	39.145	39.25	TIC	13697	2.87	3555	1.49	3.85

Table 5 : GC - MS Analysis

Ethanolic extract of guar seeds was analyzed by gc-ms and results are shown in table 5. Nine compounds were 4-dihydroxymandelic acid-tetratms, cyclohexasiloxane, dodecamethyl- 2,2,4,4,6,6,8,8,10,10,12,12-dodecamethylcyclohexasiloxane, toxy-1,1,1,7,7,7-hexamethyl-3,5,5-tris(trimethylsiloxy)tetrasiloxane, benzoic acid, 2,5-bis(trimethylsiloxy)-, trimethylsilyl ester benzoic acid, 2,5-bis[(trimethylsilyl)oxy], 1-penten-3-one, 1,5-bis[2-[(trimethylsilyl)ethynyl] phenyl]- $$ 1,5-bis (2-trimethylsilylethynylphenyl)pent, penten-3-one, 1,5-bis[2-[(trimethylsilyl)ethynyl]phenyl]- $$ 1,5 bis(2trimethylsilylethynylphenyl)pent, 1,2-benzenedicarboxylic acid, dioctyl ester, 1,2-benzenedicarbonic acid, dioctyl ester, cyclononasiloxane,octadecamethyl2,2,4,4,6,6,8,8,10,10,12,12,14,14,16,16,18,18-octadecamethylcyclonona,

chlorine dioxide, dimethyl-flubendazole, 3,6-pyridazinedicarboxylic acid, 1,4-dihydro-4-methyl-4-(1-methyl-1h-indol-3-yl)-, dimethyl ester dime.

MTT Cytotoxicity Assay

In the present study, the systematic experimental steps in order to determine the potential cytotoxicity of drug at different concentrations by MTT assay. It is shown that a decreasing absorbance at 540 nm in the cells treated with increasing concentration of the drug in comparison to the control cells without any treatment. A decreased absorbance in the cells treated with drug suggesting cytotoxicity. MTT assay significantly helps the researchers to determine whether any of the test compounds has cell toxicity or proliferative activity. There are many advantages of MTT assay in particularly its simplicity and effectiveness, which make it more suitable to assess the anti- Parkinson activities of any test samples at preliminary levels. From the given result the MTT assay determines that the tested samples concentration from 500 – 10 µg/ml decreases with increasing OD value at 570 nm.

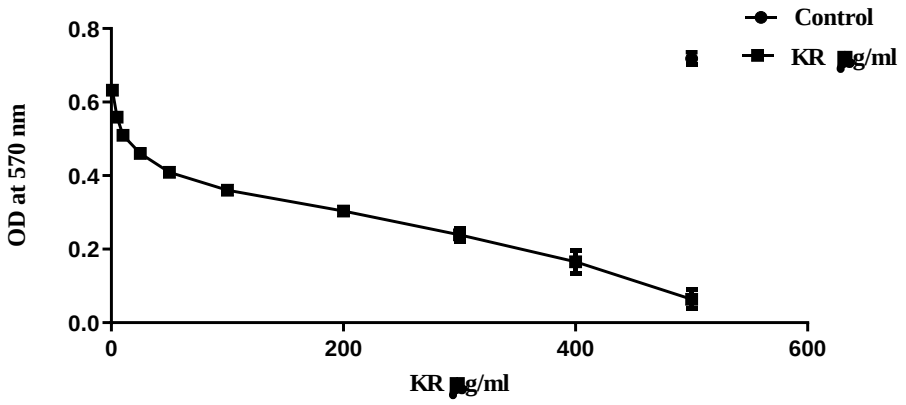
• The % of cell viability using MTT assay were calculated as the tested sample concentration from 500-1 µg/ml decreases with increase in cell viability.

• Cell viability response in IC50 value of tested sample is 81.12 µg/ml

The potential of the extract is to determine the cytotoxic effect was investigated as a part of enquiry into the mechanism of Anti-Parkinson effect. The maximum inhibition absorbed at 500 µg/ml. IC50 value of tested sample is 81.12µg/ml.

S. No	Tested sample concentration (µg/ml)	OD Value at 570 nm (in triplicates)		
1.	Control	0.732	0.699	0.725
2.	500 µg/ml	0.093	0.046	0.051
3.	400 µg/ml	0.132	0.173	0.192
4.	300 µg/ml	0.246	0.253	0.219
5.	200 µg/ml	0.294	0.304	0.313
6.	100 µg/ml	0.346	0.363	0.371
7.	50 µg/ml	0.406	0.396	0.425
8.	25 µg/ml	0.473	0.465	0.443
9.	10 µg/ml	0.496	0.512	0.522
10.	5 µg/ml	0.573	0.561	0.544
11.	1 µg/ml	0.621	0.641	0.636

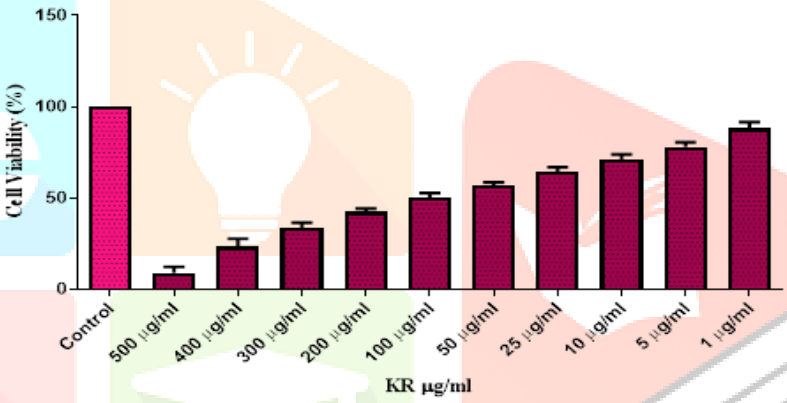
Table 6 : MTT Assay Result



Cell Viability (%)

S. No	Tested sample concentration (µg/ml)	Cell viability (%) (in triplicates)			Mean Value (%)
1.	Control	100	100	100	100
2.	500 µg/ml	12.7049	6.58083	7.03448	8.7734102
3.	400 µg/ml	18.0328	24.7496	26.4828	23.088396
4.	300 µg/ml	33.6066	36.1946	30.2069	33.336006
5.	200 µg/ml	40.1639	43.4907	43.1724	42.275683
6.	100 µg/ml	47.2678	51.9313	51.1724	50.123835
7.	50 µg/ml	55.4645	56.6524	58.6207	56.91251
8.	25 µg/ml	64.6175	66.5236	61.1034	64.081513
9.	10 µg/ml	67.7596	73.2475	72	71.002353
10.	5 µg/ml	78.2787	80.2575	75.0345	77.856894
11.	1 µg/ml	84.8361	91.7024	87.7241	88.087545

Table 7 : Cell Viability

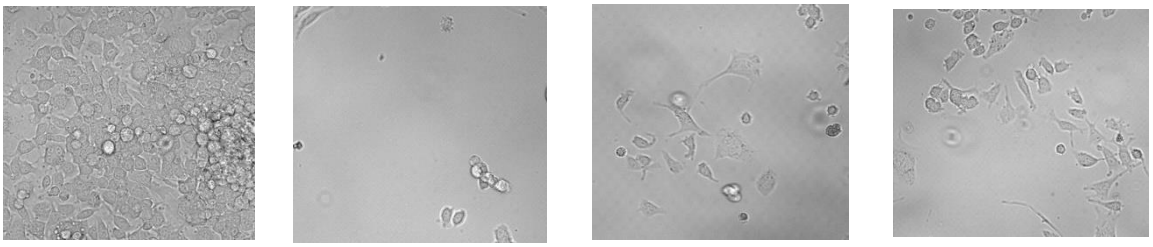


IC50 Value of tested sample: 81.12 µg/ml

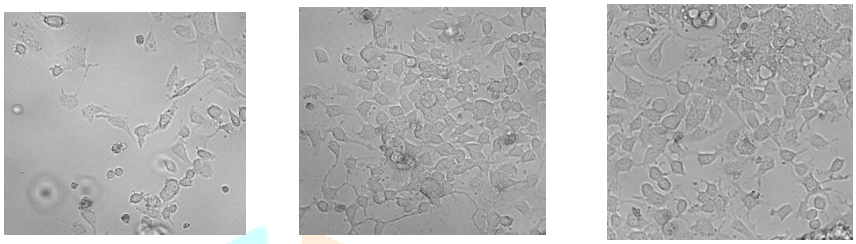
log(inhibitor) vs. normalized response - Variable slope		
Best-fit values		
LogIC50		1.909
HillSlope		-0.8092
IC50		81.12
Std. Error		
LogIC50		0.04605
HillSlope		0.07099
95% Confidence Intervals		
LogIC50		1.815 to 2.003
HillSlope		-0.9546 to -0.6638
IC50		65.29 to 100.8
Goodness of Fit		
Degrees of Freedom		28
R square		0.9284
Absolute Sum of Squares		1947
Sy.x		8.338
Number of points		

Table 8: Cell line studies data

Images of control cells and treated cells



Control cells 500 µg/ml 300 µg/ml 100 µg/ml



50 µg/ml 10 µg/ml 1 µg/ml

Conclusion

The present study demonstrated that the ethanolic pod extract of *Cyamopsis tetragonoloba* (L.) exhibits significant in-vitro anti-Parkinson's activity in a neurotoxicity-induced cell culture model. The extract effectively mitigated oxidative stress, stabilized mitochondrial function, and improved neuronal viability, thereby suggesting its neuroprotective potential. These findings indicate that the phytoconstituents present in *C. tetragonoloba* pods may play a crucial role in attenuating dopaminergic neuronal damage, a hallmark of Parkinson's disease.

The *Cyamopsis tetragonoloba* extracts obtained were subjected to phytochemical investigation and its was found that contains Steroids, Terpenoid, Saponin glycosides, Glycosides, Flavonoids, Tannins. GCMS analysis of *Cyamopsis tetragonoloba* in plant extract were analysed and identified various phytochemical compound. In -vitro Anti-Parkinson's activity was evaluated by using the Neuro 2a (Mouse neuroblast cell) method. Cell viability response in IC50 value of tested sample is 81.12 µg/ml.

Overall, the study highlights *C. tetragonoloba* as a promising natural source for the development of novel therapeutic agents against Parkinson's disease. However, further investigations including bioactive compound isolation, in-vivo validation, and mechanistic studies are essential to substantiate its pharmacological efficacy and clinical applicability.

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