ISSN: 2231-3656

International Journal of Pharmacy and Industrial Research (IJPIR)

IJPIR |Vol.16 | Issue 2| Apr - Jun-2026

www.ijpir.com

DOI: https://doi.org/10.61096/ijpir.v16.iss2.2026.524-531

Comparative Review of Antioxidant and Neuroprotective Potential of *Clitoria trenatea* Vs *Celosia Cristata* from Phytochemical to Mechanisms

Naga Jothy S[1*], Sarmith Kumar. R[2]

[1*] *Assistant Professor, Department of Pharmaceutical chemistry, Thanthai Roever College of Pharmacy, Perambalur*

[2] *Under Graduate Student B PHARM, Thanthai Roever College of Pharmacy, Perambalur*

Corresponding author: Naga Jothy. S
Email id: nagajothy011@gmail.com

Published on: 27.04.2026
Published by: Futuristic Publications

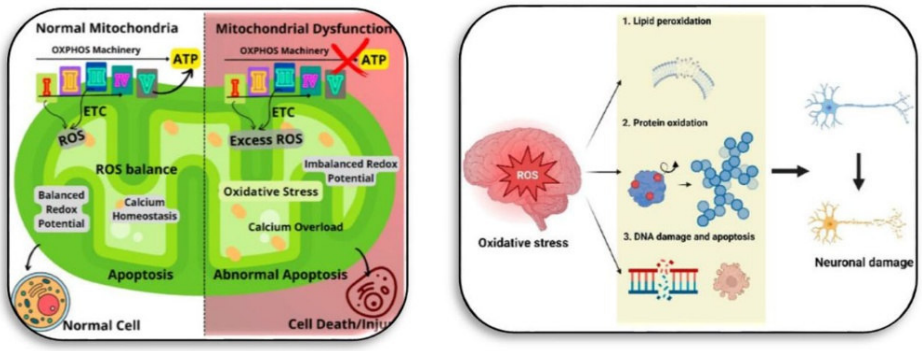
Abstract: Losing brain cells slowly over time defines conditions like Alzheimer's, Parkinson's. Inside these illnesses, harm comes from too much oxidation, swelling in nerve tissue, cell death. Plants used in old healing ways offer compounds that fight such damage safely. Their helpful traits come without harsh side effects often seen in lab-made drugs. One look into *Clitoria trenatea* shows it fights unstable molecules well. Instead of just blocking damage, it helps memory work better. It tweaks how nerves using acetylcholine behave, which matters in thinking clearly. On the other hand, *Celosia cristata* does less direct cleanup. What it does is boost the body's own shields against stress. Swelling and molecular wear go down when this plant gets involved. Both carry natural ingredients -flavonoids, phenols, colorful pigments, bitter alkaloids, foamy saponins, ring-shaped triterpenes. These parts mix together to create useful actions inside living things. Tests in labs and animals back up these effects. Each plant takes a different route but reaches similar ground. Not every herb works the same way even if results seem alike. Pieces of green life shield brain cells by calming swelling and blocking cell death, plus they tend to be well tolerated. Still, confirming their role in treating nerve breakdown demands clearer formulas, better prep methods, deeper molecule matching tests, along with real patient trials.

Keywords: *Clitoria trenatea*, *Celosia cristata*, Antioxidant activity, Neuroprotection, Oxidative stress, Phytochemicals, Neurodegenerative disorders.

INTRODUCTION

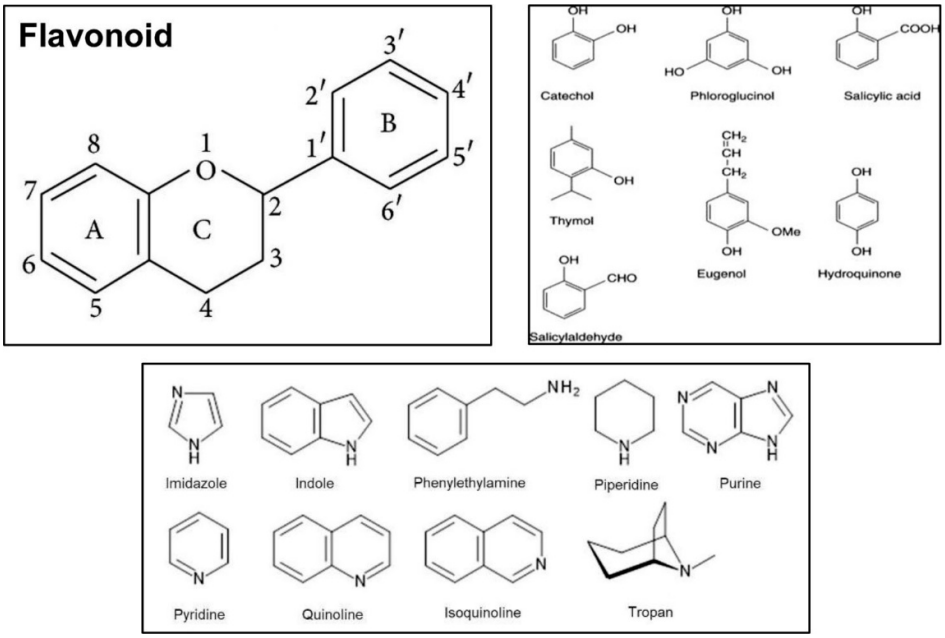
Neurodegenerative disease, as a heterogeneous group of disorder are characterized by slowly progressive losses neurons.[1] The etiology of neurodegenerative disease has not yet been full elucidated, however increased oxidative stress has been suggested as one of the potential common etiology in various neurodegenerative disease. Cumulative oxidative stress may induce cellular damage, impaired of the DNA repair system. [2] Mitochondrial dysfunction all of which have been known as key factors in acceleration of aging process and the development of neurodegenerative disorder.[3] One important antioxidant defense in the brain may be that of keeping intracellular O2 levels as low as possible consistent with normal brain function. [4] Low oxygen level decress that rate of auto oxidation reaction, slow the leakage of electrons from mitochondria electron transport chain and diminish the activity of MOA enzymes.[5] Epidemiological studies seem to indicates that dietary habits with particular respects to vitamin intake, can play an important role in brain aging cognitive impairment and possible in development.[6] *Clitoria trenatea* commonly known as the *Clitoria trenatea* this plant has a widespread distribution across various region. It can be spreaded in the tropical Asian countries such as India, Bangladesh, Butan, Nepal etc. [7] The traditional use of this flower are the roots was used for the treatment of ascetic enlargement of the abdominal viscera, sore throat and skin disease. The seeds and leaves are were widely used as brain tonic and promote memory intelligent. Juice and flower were used in antidote for snake bite.[8] An erect, much branched annual herbals or subshrub 45- 92cm tall, stem slender, glabrous, striped sometime slightly woody. Inflorescence various

branched cock combo like terminal and axillary spikes and this plant is used to dysentery, diorrhea painful mensuration, snake bite.[9]



PHYTOCHEMICAL COMPOSITION OF *CLITORI A TERNATEA* AND *CELOSIA CRISTATA*

The major phytol-constituent found in the plant are the pent acyclic triterpenoids such as taraxerol and taraxerone. Ethanol extract of *Clitoria trenatea* shows presence of terpenoids, flavonoids and tannins which may act as antioxidant. The major phytoconstituent found in *Clitoria trenatea* are the pent acyclic tritepenoids such as taraxerol and taraxerone. [10]



PHYTOCHEMICAL CONSTITUENTS IN *CLITORIA TRENATEA*

Sl.no	Plants parts	Phytochemical	Function	Reference
1.	Leaf	Alkaloids, reducing sugar, flavonoids, steroids	Preventing of neurodegenerative disease and diabetic melllitus. Effectively control the excessive sweating	[11]
2.	Flower	Saponin, tannin, alkaloids, carbohydrades phtosterols	Anti-inflammatory analgesic. Ethanol extracts is used as anti-diabetics	[12]
3.	Root	1,1- diphenyl-2-picrylhydrazyl (DPPH)	Anti-oxidant. The root bark is diuretic and laxative	[13]

4.	Seed	The seed contain nucleoprotein with its amino acid similar to insulin, essential amino-acid, water soluble mucilage.	Seeds are cathartic and root diuretics. Seeds are purgative. Seed are used in swollen joints.	[14]

PHYTOCHEMICAL CONSTITUENTS IN *CELOSIA CRISTATA*

Sl.no	Plants parts	Phytochemical	Function	Reference
1.	Leaf	Flavonoids,	Anti-microbial, anti-diarrhea	[15]
2.	Flower	Polyphenols and tannins	Anti-microbial and Anthelmintic	
3.	Root	Alkaloids	Anthelmintic	
4.	Seed	Saponins	Anti-diarrhea.	

COMPARATIVE ANTIOXIDANT ACTIVITY OF *CLITORIA TRENATEA* AND *CELOSIA CRISTATA*

Antioxidant Activity of *Clitoria trenatea*

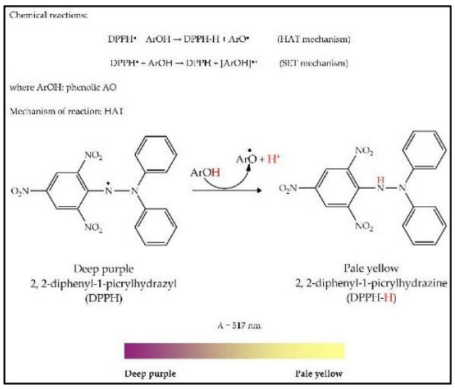
Tests for chemical antioxidants were done to find out how well *Clitoria trenatea* flower petal extracts and a particular eye cream worked as antioxidants. The study focused mostly on free radical scavenging capacity which is a key marker of antioxidant activity.

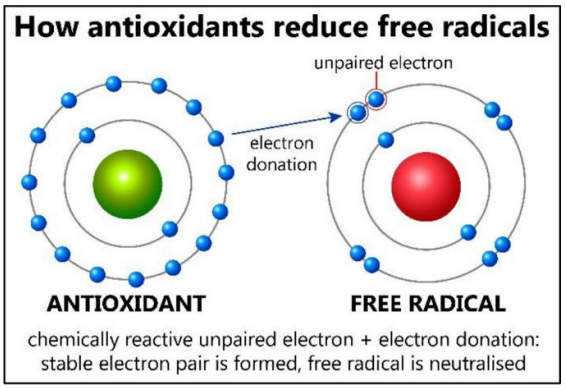
What petals are built from gives them tough defenses. Their ability to shield against damage stood out quickly. Inside each one, chemicals work together - sometimes paired, sometimes alone - to defend cells. Power comes less from any single part but from mixtures special to outer coverings. Scientists did not design this; seasons of bloom refined it gradually

It isn't only anthocyanins - flavonoids matter too. Because they give up hydrogen or electrons, free radicals get neutralized, stopping oxidation before it starts. Even after becoming an eye gel, none of their power was lost. Since they stay stable, the active parts likely hold up through changes.

Flowers fight damage from air exposure because of pigments teaming up with certain plant compounds. What makes them special is how they give away particles that stop destructive molecules in their tracks. Even after turning into a gentle gel for under the eyes, the mix holds its ground - active pieces remain intact through production.

From the first contact, *Clitoria trenatea* neutralizes free radicals instantly. Its strength becomes obvious when seen on the surface of skin. Fast results matter-defense activates without delay. Since it works rapidly, products containing it deliver stronger effects. [16]





Antioxidant Activity of *Celosia cristata*

Through a biologically meaningful system, the flower extract from *Celosia cristata* had its antioxidant effects tested. To mimic cell-level harm triggered by unstable oxygen molecules, researchers applied t-BHP, creating liver cell injury. Despite artificial triggers, responses reflected natural stress pathways.

The treatment significantly reduced signs of oxidative damage, such as lipid breakdown, at the same time boosting internal protective systems - superoxide dismutase, catalase, and glutathione were brought back toward normal. In addition, liver cell membranes stayed intact under exposure, thanks to the stabilizing effect of *Celosia cristata*. Stress markers dropped noticeably when the plant compound was applied.

It appears *Celosia cristata* does not directly neutralize free radicals. Instead, protection arises through a boost in the body's own defenses. Oxidative harm to tissues may be reduced thanks to this shift in internal response. The effect emerges not from direct interaction but via support of natural processes. [17]

ANTI-OXIDANT POTENTIAL

In-vitro antioxidant assay

Chemicals: DPPH solution (200 pM) Phosphate buffer (0.2M, pH 6.6) (Potassium dihydrogen phosphate 0.2M): (NaOH 0.2M): Potassium ferricyanide (1%), TCA (10%), Ferric chloride (0.1%).

DPPH free radical scavenging assay: DPPH free radical scavengirig assay was performed according to method. Percent inhibition of DPPH free radical scavenging activity was calculated using the following formula:

$$\text{DPPH radical scavenged } \% \quad \frac{\text{A cont-A test)}}{\text{A cont}} \text{.i oo}$$

Where, Acont is the absorbance of the control reaction. Atest is the absorbance in the presence of the sample of the exacts.

Determination of reducing power: The total reducing power of roots of blue and white flowered variety of *Clitoria trenatea* was determined according to the method of Oyaizu [18]

NEROPROTECTIVE POTENTIAL.

EXPIRIMENTAL MODEL OF NEUROPROTECTION

Starting off, experimental setups in neuroscience aim to copy key biological events tied to brain disorders - things like cell damage from free radicals, swelling in nerve tissue, overactive signaling, broken energy factories inside cells, and loss of neurons. Instead of just observing, these systems recreate harmful conditions similar to those seen in Alzheimer's, Parkinson's, or stroke-related damage. Through them, researchers get a clearer picture of how protective drugs might work under real disease pressures.

Most lab tests on brain protection fall into three types: grown in dishes, taken from living tissue, or tested within whole animals. Dishes reveal how things work inside cells, plus skip many moral questions. Whole animal studies show real body effects, linking behaviour changes with chemical shifts and tissue damage. Using more than one kind helps confirm results, also shows clearer paths of cause and effect. [19, 20]

IN VITRO NEUROPROTECTIVE STUDIES

Lab studies on brain protection usually work with nerve-like cells - examples are SH-SY5Y, PC12, Neuro-2a, or fresh cortical neurons. Because they react to damage similarly to human brain cells, scientists test how well compounds shield them from harm caused by too much oxidation or overactive signaling. Hydrogen peroxide shows up a lot in these tests; so does glutamate, along with clumps of amyloid-β and poisons like rotenone that disrupt energy factories inside cells

Looking at cell survival, levels of harmful molecules inside cells, energy factory stability, damage to fats, plus signs of programmed death such as caspase-3 activity helps show how well protection works. When brain cells face oxidative harm, natural compounds from plants often help balance chemical reactions, preserving nerve structure along the way. From blue pea flowers, compounds stepped in to shield nerve cells when free radicals attacked, mainly thanks to pigments like flavonoids and anthocyanins. When tested on stressed cells - both brain and non-brain types - plume celosia pulled back damage more strongly as doses rose, revealing its rich stock of plant phenols. [21, 22]

IN VIVO NEUROPROTECTIVE STUDIES

Looking at how well substances protect brain cells means checking behavior along with chemical changes and tissue damage inside living animals. Even today, mice and rats take center stage when studying these protective effects. Instead of just one method, scientists often turn to memory-disrupting setups - such as giving scopolamine to cause forgetfulness or using aluminum chloride to harm nerve cells - then watch how learning holds up.

Starting off with how mice move through watery puzzles, scientists check memory protection clues. Sometimes they watch turns in forked paths to see mental sharpness stay steady. Even skipping shocks after bad memories gives hints about brain shielding. In jars and tubes, researchers track a nerve signal chemical breaking down slowly or fast. When fats in brain cells get damaged by rust-like processes that gets measured too. Enzymes fighting cell wear and tear show up in lab readings often. Now and then, alarm proteins stirring inside gray matter point toward swelling issues deep within. Each number pulled from tissue samples tells part of the story.

Memory stayed sharper in rats given scopolamine when they received *Clitoria trenatea*, while brain enzyme levels bounced back and cell protection systems reset. When metals harmed nerve cells in test models, Celosia plants stepped in - less rusting of tissue occurred, fewer nerves broke down. [23, 24]

Mechanisms of Neuroprotection (Anti-Inflammatory and Anti-Apoptotic)

4.1 Anti-Inflammatory Mechanisms

Tiny brain cells called microglia wake up during inflammation, pouring out harmful chemicals like TNF-α, IL-1β, because they react too strongly. When these signals spread, nerve cells slowly break down over time due to constant irritation. Blocking key routes in this process - like NF-κB or COX-2 - can help shield neurons from harm. This quieting effect slows damage by calming the internal environment inside the brain.

Blue pea plant contains natural compounds that quiet body inflammation by reducing key chemical triggers instead of boosting them. Cockscomb flower fights swelling too, mainly by calming signals between cells while helping nerve coverings stay steady.

4.2 Anti-Apoptotic Mechanisms

When brain cells face too much oxidation, they may die because mitochondria stop working right. This leads to cytochrome-c spilling out, which turns on a chain reaction involving caspases. Some protective substances help prevent cell death in nerves. These helpers boost levels of Bcl-2 while quieting down Bax and lessening caspase-3 actions.

A single plant shows how antioxidants can block cell breakdown, then keeps energy centers working properly. The other does much the same, stopping brain cells from dying too soon through similar pathways. [25, 26]

COMPARATIVE EVALUATION OF NEUROPROTECTIVE ACTIVITY

A glance tells you *Clitoria trenatea* and *Celosia cristata* both guard neurons effectively - though their paths split sharply. What sets *Clitoria trenatea* apart. It lifts cognitive clarity stronger, especially when acetylcholine shifts are tracked during recall tasks. Meanwhile, *Celosia cristata* takes another route: slashing oxidation stress while quieting inflammatory signals, helping nerves endure tough environments.

When tests run side by side - same dose, same species, same lab methods - the results actually mean something. Without matching conditions, comparing treatments leads nowhere fast. [27, 28]

TOXICITY AND SAFETY PROFILE:

Starting off, checking how harmful or safe something is matters a lot when making medicines from plants that protect nerves. Even if people have used herbs for ages, they still need lab tests on cells, animal studies, along with measuring safe dose ranges before being trusted for ongoing brain health uses.

INVIVO CYTOTOXICITY STUDIES

Starting off in a lab dish, tests check how safe plant extracts are for cells. Often, scientists pick nerve-related lines like SH-SY5Y, PC12, or Neuro-2a - sometimes swapping in others such as Vero, HEK-293, or L929. Depending on the amount used, reactions in these cultures show whether harm might occur.

Finding how toxic something is often involves looking at mitochondrial function, whether cells stay intact, or if they survive well - methods like MTT, SRB, neutral red uptake, or watching LDH leak out help spot these signs. When a sample keeps over 70 percent of its cells alive, it usually counts as safe under ISO rules.

From test results, *Clitoria trenatea* extract stays gentle on cells even at different strengths. Though used differently, *Celosia cristata* also barely affects cell health when grown in labs. Both point toward safe profiles under lab conditions. Their behavior supports deeper study without early red flags**.** [29, 30, 31]

INVIVO TOXICOLOGICAL ASSESMENT

Looking at how plant extracts affect living organisms helps spot harmful effects on body systems. These checks also reveal which organs might be impacted. Short-term, medium-length, and longer trials run using guidelines set by OECD - like 423, 425, and 407 - for consistency. Each test follows clear steps to track changes over time. Safety signs emerge through careful observation across different periods. Methods stay steady so results can be compared properly. Through repeated testing, patterns begin to show up clearly. That way, risks get noticed before anything moves forward. Details matter most when outcomes depend on precision. Following these paths ensures nothing gets overlooked too soon

Most lab tests on short-term exposure show how much of a substance leads to death in half the test group plus what immediate harm it causes. Repeated low-level exposure checks track changes over time instead, watching animals' daily weight shifts along with meal patterns. Blood counts appear alongside liver enzymes when scientists review outcomes after several weeks. Organ tissue slides often highlight damage that blood work alone might miss.

Even at high doses, *Clitoria trenatea* didn't cause death or odd behaviour in lab rats. Tests on animals showed it's well tolerated. Celosia species brought similar results - no major harm appeared. Liver and kidneys worked fine during long-term use. Repeated exposure didn't push any warning signs. [32, 33, 34]

THERAPEUTIC SAFETY MARGIN OF EXTRACTS

Starting off, the gap between a helpful amount and a harmful one shapes how useful something can be in real treatment. When this range stretches out, it means the substance works well without getting close to dangerous levels. Sometimes you find it acts strongly even when kept small in quantity. That distance from harm matters more than strength alone. It turns out gentle effects often sit comfortably apart from risky ones. What stands out is how little is needed before any warning signs appear. Safety grows where effectiveness shows up early.

Most plant-based antioxidant compounds tend to be well tolerated, mainly because they do not accumulate strongly in tissues and leave the body slowly over time. Important here - especially when shielding nerve cells - is that such substances can safely remain part of a routine plan across months or years.

Starting low, Clitoria ternatae shows brain protection without crossing into harmful levels. Doses that work sit safely apart from toxic ones, leaving room for safety. Just like that, *Celosia cristata* shields nerve cells while using amounts far below risky thresholds. Its strength lies where harm doesn't follow, making usefulness likely. Protection comes through gentle potency, not force. [35, 36, 37]

FUTURE PERSPECTIVE

Bright blue pea flowers, *Clitoria trenatea*, together with crested cockscomb, *Celosia cristata*, display protective effects against cell damage and brain-related decline in early lab studies. Because of that, deeper investigation is needed to shape these hints into real medical applications. Work ahead must focus on consistent testing methods alongside human trial confirmation. Crafting usable medicines matters just as much as refining blends and delivery forms. Pairing those steps with digital simulations and protein interaction models could sharpen how we build trustworthy remedies from plants like these.

Need for Standardization and Clinical Validation:

One of the most significant obstacles in developing plant-based neuroprotective drugs is the absence of extract standardization. Variability in phytochemical composition resulting from variances in plant source, geographical location, harvesting circumstances, and extraction procedures has a substantial impact on repeatability and therapeutic effects. Standardization using quantitative phytochemical markers and chromatographic profiling (HPLC, LC-MS/MS) is thus required to assure batch-to-batch uniformity.

Following standardization, well-designed clinical validation studies are required to demonstrate the safety and effectiveness in humans. Tolerability and pharmacokinetics should be the primary emphasis of Phase I clinical trials, whereas Phase II studies should include established neuropsychological tests and biomarkers to measure cognitive and neuroprotective results. Without clinical validation, preclinical data is insufficient for therapeutic approval. [38, 39, 40]

Potential for Formulation and Drug Development

Moving beyond basic plant extracts marks a key shift in medicine development. Though some natural compounds protect nerve cells, they often dissolve poorly, break down quickly, and survive digestion weakly - blocking real-world healing use. Tiny carriers like fat-based particles, cell-mimicking bubbles, herb-bound complexes, and microscopic vessels now help more of these substances reach the brain safely.

From *Clitoria trenatea* to *Celosia cristata*, natural compounds offer potential in refining product design. One way forward involves consistent pill formats, another explores nose-based administration - both could boost how well treatments work and how often people stick with them. Tweaking key plant-derived substances through partial lab modification tends to sharpen results without raising risks. [41, 42, 43]

Scope of Molecular Docking and Computational Studies

Starting off, molecular docking plus computational models shed light on how plant compounds interact at the molecular level with brain-related targets. It turns out, these techniques help estimate how tightly natural substances bind, where they sit, and what kind of connections form with proteins tied to nervous system decline - like acetylcholinesterase, monoamine oxidase, NMDA channels, or molecules involved in inflammation. Surprisingly detailed insights come through when simulating these tiny-scale encounters.

Out there among digital models, scanning many plant chemicals at once speeds things up, pointing toward likely candidates worth testing in labs. Instead of guessing, pairing docking methods with checks on how substances move through bodies plus their structural shifts over time sharpens understanding of how long they last and where they bind. When algorithms probe *Clitoria trenatea* or *Celosia cristata*, hidden patterns emerge -

why these plants fight oxidation, calm swelling, protect cells from self-destruction - all slowly shaping smarter ways to build medicines. [44, 45, 46]

CONCLUSION:

Looking into brain health, *Clitoria trenatea* stands out for boosting mental sharpness and influencing key nerve signals. Meanwhile, *Celosia cristata* shines through strong protection at the cell level, calming inflammation and strengthening natural defences. Instead of just listing compounds, think pigments like blues and reds from plants - these fight damage tied to aging brains. Free radicals get neutralised thanks to substances found in both species, working hand-in-hand with the body's own protective tools. Though one leans more toward memory support, the other builds resilience by tuning up how cells respond under stress. Lab tests across models reveal shared paths: less swelling in tissues, fewer cells breaking down too soon. Safety looks promising so far, seen in early animal and dish studies. Still missing? Clear methods to measure active parts consistently. Progress needs smarter delivery forms, deeper look at protein interactions, plus real human trials before any firm conclusions take root.

REFERENCE

1. Lin MT, Beal MF (2006) Mitochondrial dysfunction and oxidative stress in neurodegenerative diseases. Nature 443: 787-795
2. Song P, Zou MH (2015) Roles of reactive oxygen species in physiology and pathology. In: Atherosclerosis: risks, mechanisms, and therapies (Wang H, Patterson C, eds), pp 379-392. John Wiley & Sons Inc., Hoboken, NJ
3. Federico A, Cardaioli E, Da Pozzo P, Formichi P, Gallus GN, Radi E (2012) Mitochondria, oxidative stress and neurodegeneration. J Neurol Sci 322:254-262.
4. Halliwell B, Gutteridge JMC. Free radicals in biology and medicine. 3rd ed. Oxford: University Press, 1999
5. De Groot H, Littauer A. Hypoxia, reactive oxygen, and cell injury. Free Radic Biol Med 1989; 6: 541-51
6. Martin A, Youdim K, Szprengiel A, Shukitt-Hale B, Joseph J. Roles of vitamins E and C on neurodegenerative diseases and cognitive performance. Nutr Rev 2002; 60: 308-26.
7. Sahu, D., Sahu, J. K., Kumar, V., & Tamrakar, S. K. (2023). Phytochemicals and medicinal uses of *Clitoria trenatea*. *International Journal of Plant & Soil Science*, *35*(18), 942-951.
8. Morris JB. Legume genetic resources with novel value added industrial and pharmaceutical use. In: Janick J. (Ed.), Perspectives on new crops and new uses. ASHS Press, Alexandria, VA, USA, 1999;196–201.
9. Rahman, A. H. M. M., & Gulshana, M. I. A. (2014). Taxonomy and medicinal uses on amaranthaceae family of Rajshahi, Bangladesh. *Applied Ecology and Environmental Sciences*, *2*(2), 54-59.
10. Sahu, D., Sahu, J. K., Kumar, V., & Tamrakar, S. K. (2023). Phytochemicals and medicinal uses of *Clitoria trenatea*. *International Journal of Plant & Soil Science*, *35*(18), 942-951.
11. Scalbert A, Manach C, Morand C, Remesy C and Jimenez L (2005). Dietary polyphenols and the prevention of diseases. Critical Review Food Science and Nutrition, 45: 287–306
12. Srivastava V, Khaan S and Banerjee S (2009). An evaluation of genetic fidelity of encapsulation microshoots of the medicinal plant: Cineraria maritime following six months of strong. Plant Cell, Tissue and Organ Culture, 99: 193-198.
13. Braca A, Sortino C, Politi M, Morelli I and Mendez J (2002). Antioxidant activity of flavonoids from Licania linaniaeflora. Journal of Ethno pharmacology, 79: 379-381.
14. Kirtikar KR and Basu BD (1980). Indian Medicinal Plants; Publisher: Bishen Singh, Mahandra Pal Singh; Dehradun, India, 1: 802-803.
15. Gaibimei, P., Yousuf, O., Singh, A., & Devi, N. M. (2018). A study on phytochemical screening of Celosia argentea var. cristata inflorescence extract. *The Pharma Innovation Journal*, *7*(10), 284-287.
16. Kamkaen, N., & Wilkinson, J. M. (2009). The antioxidant activity of *Clitoria trenatea* flower petal extracts and eye gel. Phytotherapy Research, 23(11), 1624-1625.
17. Kim, Y. S., Hwang, J. W., Sung, S. H., Jeon, Y. J., Jeong, J. H., Jeon, B. T., ... & Park, P. J. (2015). Antioxidant activity and protective effect of extract of *Celosia cristata* L. flower on tert-butyl hydroperoxide-induced oxidative hepatotoxicity. Food Chemistry, 168, 572-579.
18. Comparative Evaluation of in *vitro* Antioxidant Activity of Root of Blue and White Flowered Varieties of Cfiiorio *ternatea* Linn
19. Ghosh A, Pahan K. *Neuroimmune pharmacological approaches to neuroprotection.* Int Rev Neurobiol. 2016.
20. Kulkarni SK, Dhir A. *Neuroprotective agents in neurodegenerative disorders*. Drug Discov Today. 2010.
21. Tiwari V, Kuhad A. *Neuroprotective effects of natural products against oxidative stress*. Neurochem Int. 2019.

22. Mukherjee PK et al. *In vitro antioxidant and neuroprotective evaluation of medicinal plants*. Phytother Res. 2012.
23. Kumar A, Prakash A. *Neuroprotective potential of herbal drugs in cognitive dysfunction*. CNS Neurosci Ther. 2014
24. Dhanasekaran M et al. *Protective role of plant antioxidants in neurodegeneration*. J Chem Neuroanat. 2017
25. Glass CK et al. *Mechanisms underlying inflammation in neurodegeneration*. Cell. 2010.
26. Radi E et al. *Apoptosis and neuroprotection*. Curr Pharm Des. 2014.
27. Howes MJ, Perry NS. *Medicinal plants for cognitive enhancement*. Nat Rev Neurosci. 2011.
28. Spencer, J. P. (2010). The impact of fruit flavonoids on memory and cognition. *British Journal of Nutrition*, *104*(S3), S40-S47.
29. Mosmann T. *Rapid colorimetric assay for cellular growth and survival*. J Immunol Methods. 1983.
30. Riss TL et al. *Cell viability assays*. Assay Guidance Manual. 2016.
31. Kamiloglu S et al. *In vitro safety evaluation of plant phenolics*. Food Chem Toxicol. 2020.
32. OECD. *Guidelines for the Testing of Chemicals: Acute Oral Toxicity*. 2001.
33. Hayes AW, Kruger CL. *Principles and Methods of Toxicology*. CRC Press. 2014.
34. Parasuraman S. *Toxicological screening*. J Pharmacol Pharmacother. 2011
35. Ritter, J. M., Robinson, E., Fullerton, J., & Rang, H. P. (2014). Rang & Dale's Pharmacology: with STUDENT CONSULT Online Access. Elsevier Health Sciences.
36. Gutierrez, A., Gamez, R., Noa, M., Mas, R., Valle, M., Mendoza, N., & Garcia, H. (2015). Long-Term (24 Months) Carcinogenicity Study of D-004, a Lipid Extract From Roystonea regia Fruits, in Sprague Dawley Rats. International Journal of Toxicology, 34(2), 138-150.
37. Williamson, E. M., Chan, K., Xu, Q., Nachtergael, A., Bunel, V., Zhang, L., ... & Duez, P. (2015). Evaluating the safety of herbal medicines: Integrated toxicological approaches. Science, 347(6219 Suppl).
38. Patwardhan, B., & Mashelkar, R. A. (2009). Traditional medicine-inspired approaches to drug discovery: can Ayurveda show the way forward?. *Drug discovery today*, *14*(15-16), 804-811.
39. Mukherjee, P. K. (2019). *Quality control and evaluation of herbal drugs: Evaluating natural products and traditional medicine*. Elsevier.
40. Bent, S. (2008). Herbal medicine in the United States: review of efficacy, safety, and regulation: grand rounds at University of California, San Francisco Medical Center. *Journal of general internal medicine*, *23*(6), 854-859.
41. Baishya, R., Hati Boruah, J. L., Bordoloi, M. J., Kumar, D., & Kalita, P. (2019). Novel drug delivery system in phytochemicals: Modern era of ancient science. In *Herbal Medicine in India: Indigenous Knowledge, Practice, Innovation and its Value* (pp. 175-189). Singapore: Springer Singapore.
42. Anand, A., Gautam, P., & Ojha, S. (2024). Application of nanotechnology for herbal medicine development: A review. *Letters in Drug Design & Discovery*, *21*(8), 1325-1333.
43. Alexander, A., Agrawal, M., Uddin, A., Siddique, S., Shehata, A. M., Shaker, M. A., & Shaker, M. A. (2019). Recent expansions of novel strategies towards the drug targeting into the brain. *International journal of nanomedicine*, 5895-5909.
44. Lionta, E., Spyrou, G., K Vassilatis, D., & Cournia, Z. (2014). Structure-based virtual screening for drug discovery: principles, applications and recent advances. *Current topics in medicinal chemistry*, *14*(16), 1923-1938.
45. Ferreira, L. G., Dos Santos, R. N., Oliva, G., & Andricopulo, A. D. (2015). Molecular docking and structure-based drug design strategies. *Molecules*, *20*(7), 13384-13421.