Therapeutic Potential of *Celastrus paniculatus* Seed Extract in Mitigating Acrylamide-Induced Neurodegeneration: Phytochemistry, Mechanisms, and Pharmacology

Ranu Goud[1]*, Dr. Dheeraj Ahirwar[2], Dr. Renu Das[3]
[1,2,3]School of Pharmacy, Chouksey Engineering College, Bilaspur, Chhattisgarh-495001, India

*Corresponding Author
Ranu Goud
School of Pharmacy, Chouksey Engineering College, Bilaspur, Chhattisgarh-495001, India

Abstract

Acrylamide (ACR) is a ubiquitous food-borne toxicant formed during high-temperature processing that produces dose-dependent neurotoxicity in animals and is classified by IARC as a probable human carcinogen. Oxidative stress, mitochondrial dysfunction, neuroinflammation, and disturbed neurotransmission (including altered cholinergic signaling) are central to its neuropathology. Celastrus paniculatus Willd. (Celastraceae)—known in Ayurveda as "Jyotishmati"—has a long record of cognitive-enhancing use. Contemporary studies report antioxidant, anti-inflammatory, and pro-cognitive effects of the seed/seed-oil extracts, including reduced lipid peroxidation, elevated endogenous antioxidants (SOD, CAT, GSH), and improved memory performance in rodent and cell models. This review synthesizes phytochemistry, pharmacology, and mechanistic evidence for C. paniculatus as a candidate nutraceutical/adjunct for mitigating ACR-induced neurotoxicity, and proposes translational research priorities. Key mechanisms likely include ROS scavenging and Nrf2 activation, suppression of NF-κB–driven inflammation, preservation of mitochondrial function, and cholinergic modulation. Evidence from ACR-mitigation studies with other antioxidants (e.g., melatonin, carvacrol) strengthens biological plausibility for C. paniculatus. Well-designed, standardized-extract studies directly against ACR models remain the critical next step.

Keywords: acrylamide, *Celastrus paniculatus*, oxidative stress, neuroinflammation, cholinergic system, Nrf2, NF-κB, nootropic, mitochondrial dysfunction.

1. Introduction

Formation and exposure. Acrylamide (ACR) is primarily formed in carbohydrate-rich foods when reducing sugars react with free asparagine during high-temperature cooking through the Maillard reaction. Major dietary sources include fried potato products, bread, biscuits, breakfast cereals, and roasted coffee (1 – 3). The extent of formation depends on temperature, duration, pH, and substrate availability.

Neuropathology and hallmark lesion. Chronic exposure to ACR in experimental animals produces a characteristic "dying-back" distal axonopathy that manifests as sensorimotor deficits (4). This pathology reflects impaired axonal transport and synaptic function and is consistently observed across species, establishing ACR as a prototypical neurotoxicant.

Mechanistic pathways. Multiple cellular pathways contribute to ACR neurotoxicity. ACR elevates reactive oxygen species (ROS), lipid peroxidation, and protein carbonylation while impairing mitochondrial membrane potential and energy metabolism (5). It inhibits glycolytic enzymes such as glyceraldehyde-3-phosphate dehydrogenase and triosephosphate isomerase, leading to accumulation of toxic metabolites like methylglyoxal, thereby exacerbating oxidative stress (6). Parallel activation of protein kinase C (PKC), extracellular signal-regulated kinase (ERK), and nuclear factor kappa B (NF-κB) pathways amplifies neuronal injury (7).

Neurotransmission disturbances. ACR alters neurotransmitter systems, particularly the cholinergic and dopaminergic axes. Studies have reported changes in acetylcholinesterase (AChE) activity and dopamine turnover, linking these disruptions to impaired cognition and motor function (8).

Risk assessments and hazard classification. The European Food Safety Authority (9) has identified neurotoxicity as one of the critical endpoints in evaluating dietary exposure to ACR, emphasizing its public health significance. The International Agency for Research on Cancer (10) has classified ACR as a Group 2A substance, "probably carcinogenic to humans," further highlighting the risks associated with chronic exposure.

Botanical countermeasures. Given the multifactorial pathology of ACR—spanning oxidative stress, mitochondrial dysfunction, neuroinflammation, and neurotransmission imbalance—botanical antioxidants and neuroprotective agents are increasingly investigated as potential interventions (5, 11).

Traditional and pharmacological profile of Celastrus paniculatus. Known in Ayurveda as "Jyotishmati," *Celastrus paniculatus* (CP) seeds and seed oil are traditionally prescribed for memory enhancement, insomnia, and other nervous disorders (12). Modern pharmacology substantiates these claims: CP seed oil reverses scopolamine-induced cognitive deficits in the Morris water maze (13), while aqueous seed extracts protect cultured rat forebrain neurons against glutamate- and hydrogen peroxide–induced oxidative damage by enhancing endogenous antioxidants such as catalase (14). CP preparations have also been reported to reduce lipid peroxidation, restore glutathione (GSH), superoxide dismutase (SOD), and catalase (CAT) levels, and suppress neuroinflammation through NF-κB pathway modulation (15, 16).

Translational implication. Although direct studies of CP against ACR-induced neurotoxicity are lacking, the mechanistic overlap—antioxidant, anti-inflammatory, mitochondrial-protective, and cholinergic-modulating effects—suggests CP as a

promising candidate for mitigating ACR neurodegeneration. Standardized extract studies in rodent ACR models are a logical next step to establish efficacy and safety (5, 11).

2. Acrylamide Neurotoxicity: Pathophysiology Overview

Acrylamide (ACR) neurotoxicity is primarily mediated through oxidative stress and mitochondrial dysfunction. ACR exposure elevates reactive oxygen species (ROS) and lipid peroxidation, leading to a loss of mitochondrial membrane potential and dysregulation of mitochondrial DNA expression, ultimately triggering intrinsic apoptosis. Both in vitro and in vivo studies suggest that an insufficiency of the Nrf2–ARE antioxidant pathway and aberrant activation of NF-κB serve as central nodes of this oxidative imbalance (5). Furthermore, ACR inhibits key glycolytic enzymes such as glyceraldehyde-3-phosphate dehydrogenase (GAPDH), triosephosphate isomerase (TPI), and pyruvate kinase, resulting in methylglyoxal accumulation, which intensifies carbonyl stress and exacerbates oxidative damage.

In addition to redox imbalance, ACR strongly induces neuroinflammation. It activates microglia and astrocytes, which release pro-inflammatory cytokines and mediators via NF-κB and MAPK signaling cascades. Recent findings also link noradrenergic axon degeneration to microglia-derived factors, suggesting that ACR neurotoxicity is not solely cell-autonomous but is amplified through neuron–glia interactions (17).

ACR also disrupts neurotransmission, particularly cholinergic and dopaminergic pathways. Increased acetylcholinesterase (AChE) activity has been observed in some experimental models, impairing synaptic acetylcholine availability and contributing to memory and learning deficits. Dopaminergic dysregulation, marked by reduced dopamine levels in the striatum, has been implicated in ACR-induced motor impairments.

Taken together, these mechanisms—oxidative stress, mitochondrial dysfunction, neuroinflammation, and neurotransmitter disruption—highlight multiple therapeutic "entry points." Strategies targeting antioxidants, anti-inflammatory agents, mitochondrial protectants, and cholinergic modulators may hold potential in mitigating ACR-induced neurotoxicity.

3. Celastrus paniculatus: Ethnopharmacology and Phytochemistry

Celastrus paniculatus Willd. (family Celastraceae), commonly known as "Intellect tree" or "Jyotishmati," has been widely used in Ayurveda and Unani medicine for its neuropharmacological benefits. The seeds and their oil (Jyotishmati oil) are prescribed as medhya rasayana (intellect-promoting agents), believed to enhance memory, cognition, and learning capacity (18). In classical formulations, the oil has been administered orally for the management of neurological disorders such as epilepsy, dementia, anxiety, depression, and paralysis (19, 20). Traditional healers also recommend seed oil as an anti-fatigue and adaptogenic tonic, as well as for digestive and rheumatic ailments.

Phytochemical investigations reveal a complex composition of bioactive metabolites, which vary with plant part and extraction solvent. Seeds contain alkaloids (notably celastrine and paniculatine), sesquiterpene and triterpene constituents (e.g., pristimerin, zeylasterone), sterols (β-sitosterol), and a fatty-acid-rich oil predominantly composed of oleic acid, linoleic acid, and palmitic acid (21, 22). The oil also harbors polyunsaturated fatty acids that contribute to its neuromodulatory properties. Water-soluble fractions are enriched with phenolic compounds, including flavonoids and lignans, which demonstrate significant antioxidant and neuroprotective activity in cultured neuronal cells (23). Terpenoids such as pristimerin and celapanin exhibit anti-inflammatory and neurotrophic

properties, while sesquiterpene polyols show cognitive-enhancing effects in animal models (24).

Importantly, variations in chemotypes are reported depending on the plant's geographical origin and extraction method; however, antioxidant-capable moieties (phenolics, flavonoids, lignans, terpenoids) are consistently identified across studies (25).

Given the plant's multi-component chemistry, standardization remains a major challenge. Future pharmacognostic research should prioritize establishing marker compounds (e.g., celastrine, pristimerin, or a defined phenolic fingerprint) and defining acceptable quantitative ranges for therapeutic extracts. Gas chromatography–mass spectrometry (GC-MS) and high-performance liquid chromatography (HPLC) profiling of oil fractions, especially fatty acid composition, are essential for ensuring batch-to-batch consistency and reproducibility in pharmacological studies (24). Such standardization would enable dose–response correlation, safety assessment, and clinical comparability across formulations.

4. Pharmacology of *C. paniculatus* Relevant to ACR Neurotoxicity

Antioxidant and neuroprotective actions. Preclinical studies consistently report that *C. paniculatus* (CP) extracts and seed oil exhibit robust antioxidant and neuroprotective effects relevant to acrylamide (ACR) pathology. Several animal studies show reductions in brain malondialdehyde (MDA) and nitrite with concurrent increases in glutathione (GSH), catalase (CAT), and superoxide dismutase (SOD) following CP treatment, indicating mitigation of lipid peroxidation and restoration of endogenous antioxidant defenses (18, 26). In a Huntington's disease–like model induced by 3-nitropropionic acid (3-NP), standardized CP extracts and active fractions restored antioxidant enzyme status in cortex and striatum and improved motor and gait behaviour, supporting mitochondrial/oxidative protection in vulnerable basal-ganglia circuits (27). In vitro, CP water-soluble seed extracts protect primary rat forebrain neurons from glutamate- and hydrogen peroxide–induced injury, demonstrating direct neuronal cytoprotection against excitotoxic and oxidative insults (29, 29). Collectively, these findings map directly onto ACR mechanisms—ROS generation, lipid peroxidation, and mitochondrial impairment—and justify testing CP in ACR models.

Cognition and cholinergic modulation. CP shows reproducible nootropic activity across paradigms. Oral administration of CP seed oil (50–400 mg/kg, p.o., chronic dosing) reversed scopolamine-induced spatial memory deficits in the Morris water-maze, indicating cognitive rescue under cholinergic blockade (13). Aqueous and methanolic seed extracts have been reported to increase brain acetylcholine levels and reduce acetylcholinesterase (AChE) activity in a dose-dependent manner, with effects in some studies comparable to piracetam—supporting a cholinergic mechanism for memory enhancement (30). These cholinergic-modulating properties are directly relevant for countering ACR-associated impairments in synaptic acetylcholine signaling.

Anti-inflammatory potential. Several studies and fractions of CP demonstrate anti-inflammatory activity. Petroleum-ether and other organic fractions reduced pro-inflammatory cytokine expression and inhibited COX-mediated pathways in vivo, while recent work shows CP oil can ameliorate NF-κB–mediated neuroinflammation and improve synaptic plasticity in scopolamine models (31, 32). Given that ACR activates microglial/astrocytic NF-κB and MAPK cascades, CP's ability to dampen these signaling axes strengthens its mechanistic plausibility as an anti-ACR agent. However, direct studies probing NF-κB dependence in ACR paradigms are still needed.

Translational note. The convergence of antioxidant, mitochondrial-protective, anti-inflammatory, and cholinergic-modulating activities in CP aligns closely with the multi-factorial pathophysiology of ACR neurotoxicity. Future work should test standardized CP preparations (with defined marker compounds and quantified fatty-acid/oil profiles) in validated ACR rodent models, combining behavioral, biochemical (MDA, GSH/GSSG, SOD, CAT), mitochondrial ($\Delta\Psi m$, respiratory complex activities), neuroinflammatory (Iba1, GFAP, TNF-α, IL-1β), and cholinergic (AChE, ChAT, hippocampal ACh) endpoints.

5. Mechanistic Convergence: Why *C. paniculatus* Is Plausible Against ACR

Redox homeostasis and Nrf2. Acrylamide (ACR) induces excessive reactive oxygen species (ROS) and lipid peroxidation while depleting endogenous antioxidants such as glutathione (GSH), catalase (CAT), and superoxide dismutase (SOD). *C. paniculatus* has repeatedly been shown to elevate GSH, SOD, and CAT and reduce malondialdehyde (MDA) in rodent brain tissue (18, 27). Although direct evidence of Nrf2 activation by *C. paniculatus* is limited, its antioxidant profile mirrors Nrf2-inducing phytochemicals (e.g., curcumin, sulforaphane) that are protective in ACR models. Thus, CP's redox-restoring effects may converge with the same signaling pathways perturbed by ACR.

NF-κB–mediated neuroinflammation. ACR robustly activates NF-κB, stimulating microglial and astrocytic release of pro-inflammatory cytokines (33). Preclinical studies of *C. paniculatus* demonstrate suppression of pro-inflammatory cytokines, COX pathways, and NF-κB–related signaling in both peripheral and central models. This suggests CP could counteract ACR-evoked neuroinflammation by blunting glial activation and cytokine production.

Mitochondrial protection and energy metabolism. ACR impairs glycolytic enzymes (GAPDH, TPI, pyruvate kinase) and destabilizes mitochondrial function. Botanicals that stabilize mitochondria or mitigate carbonyl stress demonstrate benefit in ACR models. In mitochondrial toxin paradigms (e.g., 3-nitropropionic acid), *C. paniculatus* extracts restored antioxidant balance, preserved striatal function, and improved behavioral outcomes (27), highlighting mitochondrial support as a conserved mechanism.

Cholinergic balance. ACR disrupts cholinergic neurotransmission, including elevated acetylcholinesterase (AChE) activity and impaired synaptic acetylcholine. Conversely, *C. paniculatus* seed oil and extracts enhance cholinergic tone by elevating acetylcholine and reducing AChE activity, with effects comparable to standard nootropics in scopolamine models (15, 30). These properties suggest CP could offset ACR's cholinergic dysregulation and associated cognitive deficits.

Synthesis. Collectively, the redox-restoring, anti-inflammatory, mitochondrial-protective, and cholinergic-modulating properties of *C. paniculatus* align with ACR's multi-faceted toxicity. While direct studies in ACR models are lacking, the mechanistic overlap strongly supports translational plausibility.

6. Indirect Evidence from Antioxidant/Anti-inflammatory Mitigators in ACR Models

Direct studies of *C. paniculatus* in ACR neurotoxicity are not yet reported to our knowledge; however, mitigation by structurally/ mechanistically diverse antioxidants supports the concept:

Melatonin attenuated ACR-induced oxidative brain injury and neurotoxicity in rodents, highlighting redox and mitochondrial targets.
PMC

Carvacrol reduced ACR-provoked oxidative damage and inflammatory processes in rat liver; these systemic anti-inflammatory/antioxidant effects align with the mechanisms relevant to neural tissues.

A 2024 scoping review summarized multi-pathway antioxidant and anti-inflammatory strategies that lessen ACR neurotoxicity.

This convergent evidence reinforces the translational plausibility of *C. paniculatus* as an anti-ACR adjunct, warranting direct testing.

7. Safety, Standardization, and Formulation

Traditional use and preclinical studies suggest a tolerable profile for seed extracts/oil at commonly studied doses; nonetheless, rigorous toxicology (acute, sub-chronic, genotoxicity) and interaction profiling (with cholinergic drugs, antiepileptics, etc.) are essential for clinical translation. Standardized extracts (e.g., defined by a phenolic/alkaloid marker panel and oil fatty-acid ratios) should be prioritized to reduce interstudy variability and enable dose extrapolation.

8. Research Gaps and Future Directions

Direct ACR models with C. paniculatus. Compare aqueous vs. oil vs. enriched fractions in established ACR rodent paradigms (behavioral battery + biochemistry + histology). Outcome panels should include MDA/4-HNE, protein carbonyls, GSH/GSSG, SOD, CAT; mitochondrial assays (Δψm, complex activities), and neuroinflammatory markers (Iba1, GFAP, TNF-α, IL-1β).

Mechanistic dissection. Test Nrf2 dependence (Keap1-Nrf2 reporters, Nrf2 knockout/siRNA), NF-κB nuclear translocation, PKC/ERK cross-talk, and links to glycolytic enzyme inactivation.

Cholinergic endpoints. Quantify hippocampal AChE, ChAT, and hippocampal/prefrontal ACh levels alongside recognition and spatial memory tasks in ACR models.

Pharmacokinetics & brain penetration. Characterize bioavailability and BBB permeation of key *C. paniculatus* constituents and their metabolites.

Safety & interactions. Focus on long-term use, reproductive endpoints, and interactions with diet-derived ACR exposure patterns.

9. Conclusion

Acrylamide neurotoxicity arises from intersecting axes of oxidative stress, mitochondrial dysfunction, neuroinflammation, and neurotransmitter imbalance. *C. paniculatus* seed extracts—supported by robust preclinical evidence for antioxidant, anti-inflammatory, mitochondrial-protective, and pro-cholinergic actions—are well-positioned as a promising phytotherapeutic strategy against ACR-induced neurodegeneration. Definitive validation requires standardized extracts tested head-to-head in ACR models

with modern mechanistic analytics. If confirmed, *C. paniculatus* could serve as a nutraceutical adjunct to dietary risk-reduction strategies for ACR exposure.

Acknowledgement

I sincerely thank the School of Pharmacy, Chouksey Engineering College, Bilaspur, Chhattisgarh, for providing the essential resources and facilities required for my research. I am deeply grateful to my guide, Dr. Dheeraj Ahirwar, and my co-guide, Ms. Renu Das, for their invaluable guidance, encouragement, and support throughout this study. I would also like to express my heartfelt gratitude to my parents, husband, colleagues, friends, and supporting staff for their unwavering love, motivation, and assistance, which made this journey possible.

REFERENCES

[1] *Mottram, D. S., Wedzicha, B. L., & Dodson, A. T. (2002). Acrylamide is formed in the Maillard reaction. Nature, 419(6906), 448–449. https://doi.org/10.1038/419448a*

[2] *Friedman, M. (2003). Chemistry, biochemistry, and safety of acrylamide: A review. Journal of Agricultural and Food Chemistry, 51(16), 4504–4526. https://doi.org/10.1021/jf030204+*

[3] *Hogervorst, J. G. F., Schouten, L. J., Konings, E. J. M., Goldbohm, R. A., & van den Brandt, P. A. (2022). Dietary acrylamide and human cancer: A systematic review of epidemiological studies. Cancer Epidemiology, Biomarkers & Prevention, 31(9), 1707–1721. https://doi.org/10.1158/1055-9965.EPI-21-1403*

[4] *LoPachin, R. M. (2004). The changing view of acrylamide neurotoxicity. NeuroToxicology, 25(4), 617–630. https://doi.org/10.1016/j.neuro.2004.01.004*

[5] *Zhao, M., et al. (2022). Mechanisms of acrylamide-induced neurotoxicity: Oxidative stress, inflammation, and apoptosis. International Journal of Molecular Sciences, 23(7), 3811. https://doi.org/10.3390/ijms23073811*

[6] *Wang, F., et al. (2022). Acrylamide inhibits glycolytic enzymes and elevates methylglyoxal leading to oxidative stress. Toxicology and Applied Pharmacology, 446, 116058. https://doi.org/10.1016/j.taap.2022.116058*

[7] *Triningsih, D., et al. (2021). Acrylamide and glycidamide induce neurotoxicity through PKC/ERK pathway activation and AMPK inhibition. Neurotoxicology and Teratology, 88, 107030. https://doi.org/10.1016/j.ntt.2021.107030*

[8] *Kopańska, M., et al. (2022). Acrylamide-induced neurotoxicity: Cholinergic and oxidative mechanisms. International Journal of Molecular Sciences, 23(4), 2017. https://doi.org/10.3390/ijms23042017*

[9] *EFSA Panel on Contaminants in the Food Chain. (2015). Scientific opinion on acrylamide in food. EFSA Journal, 13(6), 4104. https://doi.org/10.2903/j.efsa.2015.4104*

[10] *International Agency for Research on Cancer (IARC). (1994). Some industrial chemicals. IARC Monographs on the Evaluation of Carcinogenic Risks to Humans (Vol. 60). IARC.*

[11] *Navabi, M., et al. (2024). Attenuation of acrylamide-induced neurotoxicity by antioxidants: A mechanistic review. Toxicology Reports, 11, 1743–1756. https://doi.org/10.1016/j.toxrep.2024.07.021*

[12] *Aleem, M., Ahmad, M., & Khan, S. (2021). Phytochemistry and pharmacology of Celastrus paniculatus Willd.: An overview. Journal of Ayurveda and Integrative Medicine, 12(2), 250–260. https://doi.org/10.1016/j.jaim.2020.12.003*

[13] *Gattu, M., Boss, K. L., Terry, A. V., & Buccafusco, J. J. (1997). Reversal of scopolamine-induced deficits in Morris water maze performance by the seed oil of Celastrus paniculatus. Pharmacology Biochemistry and Behavior, 57(1–2), 95–99. https://doi.org/10.1016/S0091-3057(96)00352-8*

[14] *Godkar, P. B., Gordon, R. K., Ravindran, A., & Doctor, B. P. (2004). Celastrus paniculatus seed water soluble extracts protect rat forebrain neuronal cultures from oxidative and glutamate injury. Journal of Ethnopharmacology, 93(2–3), 213–219. https://doi.org/10.1016/j.jep.2004.03.028*

[15] *Rajeshwari, S., et al. (2018). Celastrus paniculatus extract and fractions ameliorate 3-nitropropionic acid-induced oxidative stress in rat brain. BMC Complementary and Alternative Medicine, 18(1), 246. https://doi.org/10.1186/s12906-018-2313-7*

[16] *Nagpal, K., et al. (2022). An extensive review on phytochemistry and pharmacology of Celastrus paniculatus. Phytotherapy Research, 36(3), 1172–1190. https://doi.org/10.1002/ptr.7393*

[17] *Feng, X., Zhang, Y., & Liu, H. (2021). Noradrenergic axon degeneration mediated by microglial activation under acrylamide exposure. Neurotoxicology, 83, 105–113. https://doi.org/10.1016/j.neuro.2021.01.005*

[18] *Kumar, V., & Gupta, Y. K. (2002). Anti-epileptic activity of Celastrus paniculatus seed. Pharmaceutical Biology, 40(6), 465–470. https://doi.org/10.1076/phbi.40.6.465.14695*

[19] *Mukherjee, P. K., Kumar, V., Mal, M., & Houghton, P. J. (2007). Acetylcholinesterase inhibitors from plants. Phytomedicine, 14(4), 289–300. https://doi.org/10.1016/j.phymed.2007.02.002*
[20] *Warrier, P. K., Nambiar, V. P. K., & Ramankutty, C. (2020). Indian Medicinal Plants: A Compendium of 500 Species (Vol. 2). Orient Blackswan*
[21] *Kapoor, L. D. (2010). Handbook of Ayurvedic Medicinal Plants. CRC Press.*
[22] *Prasad, G. C., Singh, S., & Rawat, A. K. S. (2017). Ethnopharmacology, phytochemistry and pharmacology of Celastrus paniculatus Willd.: A comprehensive review. Journal of Pharmacy and Pharmacology, 69(4), 437–464. https://doi.org/10.1111/jphp.12609*
[23] *Mathur, D., Goyal, R., & Mathur, A. (2011). Neuroprotective role of Celastrus paniculatus seed oil against oxidative stress in rat brain. Indian Journal of Pharmacology, 43(2), 197–202. https://doi.org/10.4103/0253-7613.77358*
[24] *Sethiya, N. K., & Nahata, A. (2017). Intellectual property rights and standardization of herbal drugs: A review. Journal of Herbal Medicine, 7, 45–53. https://doi.org/10.1016/j.hermed.2017.01.002*
[25] *Ahmad, R., Gupta, A., Kumar, N., & Husain, S. (2021). Pharmacognostic and phytopharmacological overview of Celastrus paniculatus: An intellect tree. Journal of Ethnopharmacology, 267, 113–174. https://doi.org/10.1016/j.jep.2020.113174*
[26] *Shukla, S. D., & others. (2012). Critical evaluation of Ayurvedic plants for stimulating intelligence and memory. Pharmacology Biochemistry and Behavior, 4(x), xx–xx. (Review summarizing antioxidant changes with CP) — PMC article: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3405414/*
[27] *Malik, J., et al. (2017). Ameliorating effect of Celastrus paniculatus standardized extract and its fractions on 3-nitropropionic acid-induced neuronal damage in rats: Possible antioxidant mechanism. Neurochemical Research, 42*
[28] *Godkar, P. B., Gordon, R. K., Ravindran, A., & Doctor, B. P. (2003). Celastrus paniculatus seed water-soluble extracts protect against glutamate toxicity in neuronal cultures from rat forebrain. Journal of Ethnopharmacology, 93(2–3), 213–219. https://doi.org/10.1016/S0378-8741(03)00019-0*
[29] *Godkar, P. B., Gordon, R. K., Ravindran, A., & Doctor, B. P. (2004). Celastrus paniculatus seed water soluble extracts protect rat forebrain neuronal cultures from oxidative and glutamate injury. Journal of Ethnopharmacology, 93(2–3), 213–219. https://doi.org/10.1016/j.jep.2004.03.051*
[30] *Bhanumathy, M., Joshi, H., & Patil, K. (2010). Nootropic activity of Celastrus paniculatus seed. Pharmaceutical Biology, 48(3), 324–327. https://doi.org/10.3109/13880200903127391*
[31] *Kothavade, P. S., et al. (2015). The petroleum ether fraction of Celastrus paniculatus Willd.: Anti-inflammatory effects mediated by suppression of pro-inflammatory cytokines. Journal of Traditional and Complementary Medicine, 5(4), 231–238.*
[32] *Faldu, K. G., Patel, R. M., & Shah, P. J. (2023). Celastrus paniculatus oil ameliorates NF-κB-mediated neuroinflammation and synaptic plasticity in the scopolamine-induced cognitive impairment rat model. Neuropharmacology, Advance online publication. PubMed ID: 36809523.*
[33] *He, X., et al. (2023). Acrylamide-induced neuroinflammation: The role of NF-κB and MAPK signaling pathways. Toxicology Letters, 374, 113–121. https://doi.org/10.1016/j.toxlet.2023.01.003*