INTERNATIONAL JOURNAL OF PHARMACEUTICAL SCIENCES
[ISSN: 0975-4725; CODEN(USA): IJPS00]
Journal Homepage: https://www.ijpsjournal.com

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Review Article

Review on Neuroprotective Herbal Plants

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ARTICLE INFO

Published: 29 Nov 2026
Keywords:
neuroprotection, medicinal plants, phytochemicals, oxidative stress, neuroinflammation
DOI:
10.5281/zenodo.24645919

ABSTRACT

Neurodegenerative disorders, including Alzheimer's and Parkinson's diseases, are associated with progressive neuronal dysfunction and loss. This narrative review summarizes selected medicinal plants and marine botanicals investigated for potential neuroprotective effects: Tinospora cordifolia, Nigella species, Butea monosperma, Sargassum fusiforme, Ecklonia cava, Gynostemma pentaphyllum, Gastrodia elata, Eucommia ulmoides and Withania somnifera. Reported experimental activities include antioxidant and anti- inflammatory effects, modulation of apoptosis and cellular stress pathways, and effects on neurotransmission. Much of the evidence remains preclinical; differences in plant identity, extraction methods, chemical composition, dose and study design limit comparison between studies. The review distinguishes experimental findings from established clinical benefit and highlights the need for authenticated plant material, standardized preparations, pharmacokinetic studies and well-designed clinical trials.

INTRODUCTION

Neurodegenerative diseases are a diverse group of disorders characterized by progressive impairment of neuronal structure or function. Alzheimer's disease and Parkinson's disease are major examples, but the underlying mechanisms are complex and differ across conditions. Oxidative stress, neuroinflammation, mitochondrial dysfunction, abnormal protein handling and altered neurotransmission have all been investigated as contributors to neuronal injury.

Many plant-derived preparations contain mixtures of secondary metabolites, including polyphenols, alkaloids, terpenoids, saponins and glycosides. Such compounds may influence more than one biological pathway in experimental systems. However, activity observed in a cell or animal model does not establish that a preparation prevents or treats a human neurodegenerative disease. Translation is complicated by

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Relevant conflicts of interest/financial disclosures: The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

variable composition, limited oral bioavailability, metabolism, safety considerations and the absence of standardized clinical evidence.

2. Review approach

This manuscript is a narrative review developed from the supplied 10-page student review and a limited supplementary literature check for the additional Withania somnifera section. The original plant-focused scope, photographs, comparison table and conceptual mechanism diagram were retained. The supplied bibliography was not treated as fully verified: many entries lacked complete publication metadata, and these are explicitly identified in the accompanying reference audit. This is not a systematic review; no reproducible database search strategy, protocol registration or formal risk-of-bias assessment was available in the source material.

3. Pharmacological properties of selected neuroprotective plants

3.1.Tinosporacordifolia

Tinospora cordifolia is a climbing medicinal plant used in traditional systems of medicine. The source manuscript attributes potential neuroprotective activity to reduced pro-inflammatory signalling and increased antioxidant defenses. It also describes alkaloids and glycosides as relevant phytochemical groups. The cited records include reports and reviews on immunomodulatory, pharmacological and cognition-related effects. [1–7] These findings should be interpreted cautiously: immunomodulatory activity or general antioxidant effects do not, by themselves, demonstrate disease-modifying neuroprotection. The ability of particular constituents or standardized extracts to reach the brain, their dose-response relationship and their long-term safety require better characterization.



Figure 1: ***Tinospora cordifolia (original photograph from the supplied manuscript).***

3.2. Nigella species

The original manuscript labels this plant Nigella glandulifera, while the cited bibliography and in-text sources predominantly refer to Nigella sativa. These names must not be treated as interchangeable without checking the identity of the plant material used in the cited studies. The literature cited in the source discusses thymoquinone and other constituents of N. sativa, with experimental reports addressing antioxidant activity, memory-related outcomes and models of cerebral ischaemia. [8–14] A taxonomic and source-material check is therefore required before making species-specific conclusions. The evidence summarized here is mainly experimental and does not establish that black-seed preparations prevent or treat human neurodegenerative disease.



Figure 2: ***Nigella plant and seeds (original photograph from the supplied manuscript).***

3.3.Buteamonosperma

Butea monosperma, commonly called flame-of-the-forest, has been investigated for several pharmacological activities. The source manuscript highlights flavonoids and butin and cites studies concerning antioxidant activity, acetylcholinesterase inhibition and antidiabetic effects. [15–21] Acetylcholinesterase inhibition is a pharmacological mechanism relevant to cholinergic neurotransmission, but results from enzyme assays or animal models should not be described as proof of clinical neuroprotection. Further work should establish which plant parts and extracts were studied, quantify marker compounds, assess reproducibility and distinguish direct neuronal effects from indirect effects related to metabolic or inflammatory changes.



Figure 3: ***Butea monosperma (original photograph from the supplied manuscript).***

3.4. Sargassum fusiforme

Sargassum fusiforme is a brown marine alga. The source manuscript discusses polysaccharides and phlorotannin-related constituents and cites studies concerning inflammatory mediators and neurotrophin-related outcomes. [22–28] Marine polysaccharides are structurally diverse, and their biological effects depend on molecular size, composition, extraction and purification. The original claims about synaptic plasticity and neurotrophin regulation should therefore be considered hypothesis-generating unless the specific cited studies directly demonstrate these endpoints. Important research needs include chemical characterization, batch-to-batch standardization, digestion and absorption studies, and confirmation of activity in relevant models.

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Figure 4: ***Sargassum fusiforme (original photograph from the supplied manuscript).***

3.5.Eckloniacava

Ecklonia cava is a brown alga containing phlorotannins, including eckol and dieckol. The source manuscript

cites experimental work on antioxidant activity, reactive oxygen species, acetylcholine-related outcomes and

possible effects on blood–brain barrier integrity. [29–35] These findings provide a rationale for further

investigation but do not establish that extracts are superior to terrestrial polyphenols in clinical use. Claims

about protection of the blood–brain barrier should be linked to the exact model and endpoint used in the

original study. Future studies should report extract composition, purity, dose, exposure, toxicity and

potential interactions with medicines.



Figure 5: ***Ecklonia cava (original photograph from the supplied manuscript).***

3.6.Gynostemma pentaphyllum

Gynostemma pentaphyllum contains dammarane-type saponins commonly referred to as gypenosides. The

original manuscript discusses possible effects on stress-related signalling, the hypothalamic–pituitary–

adrenal axis, glutamate-associated excitotoxicity and calcium regulation. [36–42] These mechanisms are

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biologically plausible research areas, but the cited records as supplied do not provide enough bibliographic

detail to independently confirm every specific claim. The proposed effects on cortisol, hippocampal

protection and neuronal survival should be described as preclinical unless supported by appropriately

designed human studies. Standardized extracts and clearly defined gypenoside profiles are needed to

compare findings across studies.



Figure 6: ***Gynostemma pentaphyllum (original photograph from the supplied manuscript).***

3.7.Gastrodiaelata

Gastrodia elata is used in traditional East Asian medicine and has been investigated for neurological effects.

Gastrodin and parishin are among the constituents discussed in the literature. The source manuscript cites

studies concerning seizure-related conditions, oxidative stress, MAPK signalling and dopaminergic neuronal

models. [43–49] The wording "used to treat Parkinson's disease" in the original text is too strong without

clinical evidence; it has been reframed as investigation in experimental neurological models. Research

should clarify the pharmacokinetics of gastrodin and related compounds, identify active metabolites and

determine whether effects observed in experimental models translate to meaningful clinical outcomes.

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Figure 7: ***Gastrodia elata (original photograph from the supplied manuscript).***

3.8. Eucommia ulmoides

Eucommia ulmoides has been studied for lignans and iridoid glycosides and for possible vascular,

antioxidant and anti-apoptotic effects. The original manuscript links these constituents with endothelial

function, cerebrovascular stability and maintenance of synaptic connections. [50–56] These proposed

mechanisms need to be interpreted in the context of the experimental model and should not be taken as

proof that the plant prevents vascular dementia in people. Further work should distinguish vascular effects

from direct neuronal effects and examine exposure, active constituents, safety and reproducibility.

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Figure 8: ***Eucommia ulmoides (original photograph from the supplied manuscript).***

3.9. Withania somnifera (Ashwagandha) – supplementary section

Withania somnifera (L.) Dunal, commonly known as Ashwagandha, is a medicinal plant used in traditional

Indian systems of medicine. Its roots and other plant parts contain several classes of phytochemicals, including

withanolides. A 2024 systematic review surveyed preclinical and clinical investigations of W. somnifera in

relation to neurodegenerative conditions and described research involving Alzheimer's disease, Parkinson's

disease, Huntington's disease and amyotrophic lateral sclerosis. [57] A 2020 review discusses proposed

neuroprotective mechanisms, including modulation of oxidative stress and inflammation, effects on apoptosis,

and mitochondrial and endothelial function. These mechanisms are supported largely by preclinical work, with

limited clinical investigations for neurodegenerative outcomes. [58] The available literature supports further

investigation of W. somnifera and its withanolides; it does not establish Ashwagandha as a proven treatment or

preventive therapy for Alzheimer's or Parkinson's disease. Extracts may differ substantially in plant part,

withanolide content, extraction method and dose. Clinical research should use authenticated material and

chemically standardized preparations, with adequate monitoring of safety, interactions and long-term

outcomes.

4. Comparative pharmacological overview

Proposed research

mechanisms

Important research gaps

Botanical species

Key constituents discussed

Inflammatory and antioxidant

signalling

Brain exposure; extract

standardization

Alkaloids, glycosides

Tinospora cordifolia

Antioxidant and apoptosis-

related effects

Confirm species identity;

dosage standardization

Thymoquinone (in cited N.

sativa literature)

Nigella spp.

Free-radical scavenging;

acetylcholinesterase inhibition

Reproducible extracts; clinical

relevance

Butea monosperma

Flavonoids, butin

Inflammatory and

neurotrophin-related pathways

Molecular characterization;

bioavailability

Sargassum fusiforme

Polysaccharides, phlorotannins

Antioxidant effects; cholinergic

endpoints

Exposure, interactions, safety

Long-term safety; standardized

Ecklonia cava

Eckol, dieckol

Stress-axis and excitotoxicity

Gynostemma pentaphyllum

Gypenosides

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Key constituents discussed

Proposed

research

mechanisms

Important research gaps

saponins

Botanical species

pathways

Pharmacokinetics; model-to-

human translation

Oxidative stress and MAPK-

related pathways

Gastrodia elata

Gastrodin, parishin

Separate vascular from

neuronal effects

Endothelial and vascular effects

Eucommia ulmoides

Lignans, iridoids

Standardized extracts; robust

clinical trials

Oxidative stress, inflammation,

apoptosis,

mitochondrial

function

Withanolides and other

constituents

Withania somnifera

Table 1: ***Summary of constituents, proposed mechanisms and research gaps. Mechanisms are research***

hypotheses and should not be interpreted as demonstrated clinical efficacy.

5. Mechanism of action of multitargeted neuroprotective agents

The original mechanism-of-action diagram is retained. It presents a conceptual sequence from botanical

sources to phytochemical classes, modulation of inflammatory, antioxidant and autophagy-related pathways,

and possible effects on synaptic function. This should be understood as a general research framework, not as a

single mechanism proven for every plant included in this review.



Figure 9: ***Conceptual mechanism-of-action diagram retained from the original manuscript. Pathway labels represent proposed***

mechanisms, not established outcomes for all botanicals.

6. Critical discussion and limitations

The plants reviewed here contain chemically diverse constituents and have been studied using different

experimental models. A recurring limitation is that the evidence base is uneven: antioxidant activity,

enzyme inhibition or changes in inflammatory markers may be demonstrated in vitro or in animals, while corresponding benefits in patients may remain untested. The terms “neuroprotective”, “memory-enhancing”

and “disease-modifying” should therefore be used precisely and tied to the level of evidence.

Botanical identity is a foundational issue. The original manuscript uses Nigella glandulifera, whereas its listed

references mostly describe Nigella sativa. The correct species should be confirmed from the original study

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material or a taxonomic authority before the manuscript is finalized. Common names and broad plant-family

descriptions are not sufficient substitutes for authenticated voucher specimens.

Other recurring limitations include incomplete chemical characterization, inconsistent extraction procedures,

unclear dose equivalence, limited pharmacokinetic information, possible herb–drug interactions, and

insufficient long-term safety data. These issues are particularly important when discussing complex

preparations intended for older adults or people already taking medicines. This review is narrative and does not

claim systematic or exhaustive coverage of the literature.

CONCLUSION

Medicinal plants and marine botanicals provide a useful source of compounds for investigating pathways

relevant to neuronal injury, including oxidative stress, inflammation, apoptosis, mitochondrial function and

neurotransmission. The plants covered in this review, including the supplementary Withania somnifera

section, show experimental signals that justify further research. However, the evidence summarized here does

not establish these botanicals as proven treatments for neurodegenerative diseases. Future studies should

prioritize authenticated species, standardized extracts, reproducible methods, pharmacokinetic

characterization, safety assessment and well-designed clinical trials. Claims in the final manuscript should

remain proportional to the strength of the evidence.

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HOW TO CITE: Yash Pardhi, Amol Pardhi, Dr. Shriram Bairagi, Review on Neuroprotective Herbal Plants, Int. J. of Pharm. Sci., 2026, Vol 4, Issue 11, 6034-6043. https://doi.org/10.5281/zenodo.24645919