

Nature's Neuroprotectors: The power of herbal remedies in neurodegenerative diseases

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Abstract: Neurodegenerative disorders comprise a group of debilitating conditions characterized by the progressive deterioration of the structure and function of the human nervous system. Several neurodegenerative diseases, including Alzheimer's disease, Parkinson's disease, motor neuron disease, multiple system atrophy, and ataxia, are among the leading causes of mortality and long-term disability worldwide. The fundamental mechanisms underlying these disorders involve genetic mutations, protein misfolding, and the accumulation of toxic protein aggregates that disrupt normal cellular functions. Common clinical manifestations include memory loss, motor dysfunction, behavioral alterations, and cognitive impairment. Current therapeutic strategies primarily focus on symptomatic management and slowing disease progression; however, they do not effectively target the underlying causes of neurodegeneration. Consequently, there is a growing need for novel and more effective therapeutic approaches. Among the various available treatment modalities, herbal medicines are emerging as promising alternatives. Numerous medicinal plants, such as *Panax quinquefolius* L., Indian winter cherry (*Withania somnifera*), Brahmi (*Bacopa monnieri*), *Ginkgo biloba*, and rosemary (*Rosmarinus officinalis*), have demonstrated significant potential in alleviating neurological symptoms and providing neuroprotection in multiple studies. This chapter specifically focuses on the role and therapeutic efficacy of medicinal plants in the management of neurodegenerative disorders.

Keywords: Neurodegenerative disorders, Alzheimer's disease, Parkinson's disease, Medicinal plants, *Ginkgo biloba*, Rosemary.

1. Introduction

Neurons are the fundamental units of the human nervous system responsible for transmitting essential information to various parts of the body. Progressive loss of neuronal structure and function results in a pathological condition known as a neurodegenerative disorder. Among these, Alzheimer's disease and Parkinson's disease are the most prevalent neurodegenerative disorders worldwide [1-2]. Other neurodegenerative disorders include Huntington's disease, amyotrophic lateral sclerosis (ALS), multiple sclerosis, frontotemporal dementia, prion diseases, and Lewy body dementia (LBD) [3-4]. Neurodegenerative disorders have become a global health concern, exerting a profound burden on healthcare systems, society, and the economy. Community care systems face significant challenges in managing these conditions, as their prevalence continues to rise. According to World Health Organisation reports, approximately 82 million individuals are expected to be affected by dementia by 2030, with the number projected to increase to 152 million by 2050. Additionally, nearly 10 million people are currently living with Parkinson's disease, a figure anticipated to double by 2050 [5]. At present, no definitive cure exists for most neurodegenerative disorders. Available therapeutic strategies are largely symptomatic, focusing on managing disease manifestations and improving quality of life rather than addressing the underlying causes of neuronal degeneration. **Figure 1** illustrates the formation of neurofibrillary tangles and their role in triggering neuroinflammation, ultimately leading to cognitive decline and neuronal loss in the brain.

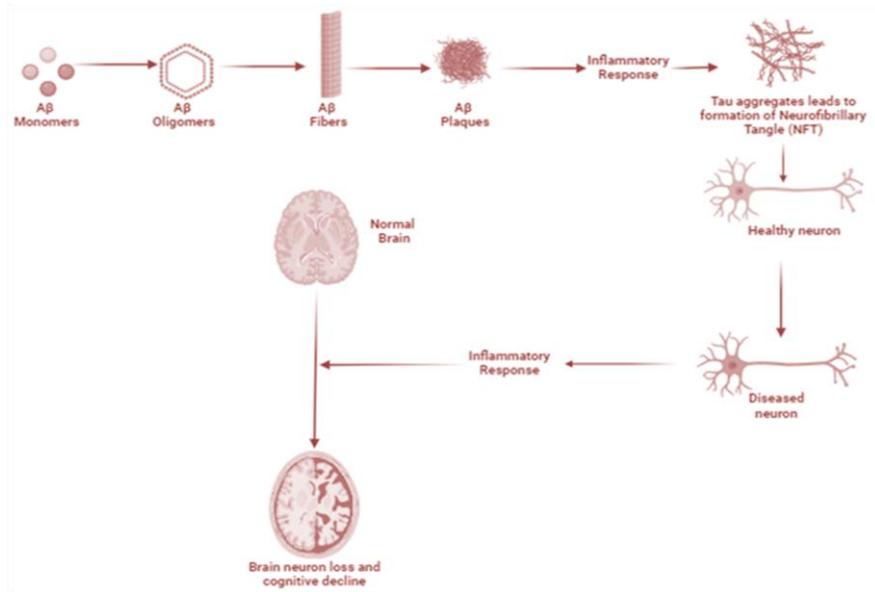


Figure 1: Neurofibrillary tangles produce inflammation leading to cognitive and neuronal loss.

Timely and accurate diagnosis is crucial for implementing interventions that may significantly delay disease progression, which can be achieved through the use of advanced biomarkers and neuroimaging techniques. Furthermore, a comprehensive understanding of the biochemical pathways underlying neurodegenerative disorders is essential for the development of targeted and effective therapeutic strategies [6-7]. **Figure 2** illustrates the common mechanisms involved in neurodegeneration.

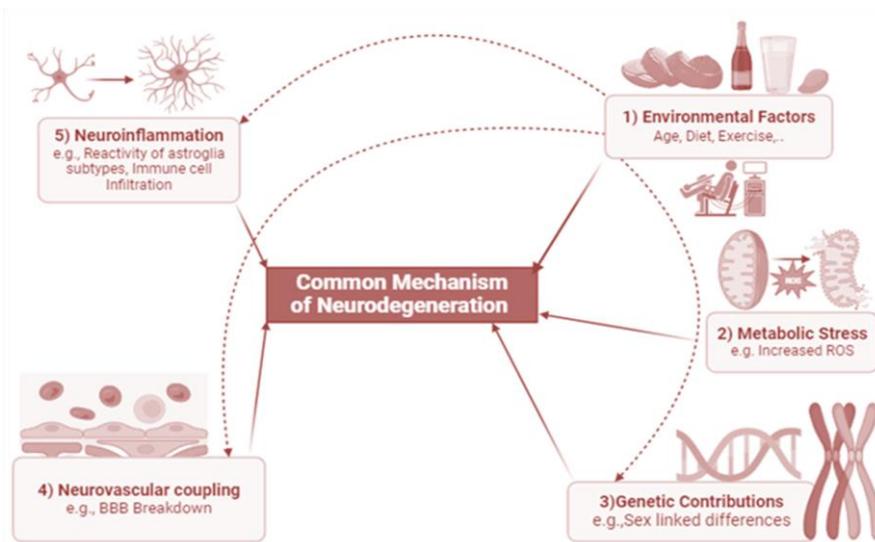


Figure 2: Common neurodegeneration mechanism.

2. A Brief Apprehension of Neuroprotection

Neuroprotection represents one of the most critical approaches for managing neurodegenerative disorders and neurological injuries [8]. It offers the potential to develop novel therapeutic strategies that preserve neuronal structure and function, delay disease progression, and improve patient outcomes. However, achieving effective neuroprotection requires a comprehensive understanding and strategic utilization of multiple neuroprotective mechanisms [9]. Neuronal protection can be strengthened through several key approaches, as discussed below.

2.1 Slowing Disease Progression

Neurodegenerative disorders are characterized by a progressive loss of neuronal number and function [10]. By protecting neurons from damage, disease progression can be slowed, thereby preserving cognitive and motor

functions and ultimately enhancing the quality of life of affected individuals [11]. Several therapeutic strategies contribute to this process, including inhibition of amyloid- β formation and accumulation, stabilization and clearance of tau proteins, antioxidant therapy, analgesic intervention, protection of dopaminergic neurons, and inhibition of α -synuclein aggregation. These approaches collectively support the restoration and maintenance of neuronal integrity and function [11-13].

2.2 Prevention of Neuronal Loss

Neurons are the primary messengers responsible for transmitting information from the brain to the rest of the body. Unlike many other cell types, neurons possess a limited regenerative capacity, making their preservation essential. Neuroprotection can be achieved by maintaining synaptic connectivity, preserving functionally similar neuronal populations, reducing pathological proliferation, delaying symptom onset, and employing disease-modifying therapies. Additionally, regenerative approaches such as stem cell therapy, along with antioxidant treatment, anti-inflammatory agents, and neurotrophic support, play a significant role in preventing neuronal loss [14-16].

2.3 Enhancement of Recovery

Acute conditions such as stroke, traumatic brain injury, and spinal cord injury result in rapid and severe neuronal damage. A primary objective of neuroprotection in these conditions is to limit initial injury and promote endogenous recovery mechanisms, thereby improving overall functional outcomes. Neuroprotective strategies include mitigation of secondary injury, regulation of glutamate and calcium homeostasis, reduction of oxidative stress through antioxidant therapy, enhancement of mitochondrial function, use of anti-inflammatory drugs, gene therapy, stem cell-based interventions, strengthening of synaptic plasticity, promotion of angiogenesis, and stabilization of the blood–brain barrier [16-18]. **Figure 3** illustrates the cellular pathways leading to neuronal death following traumatic brain injury.

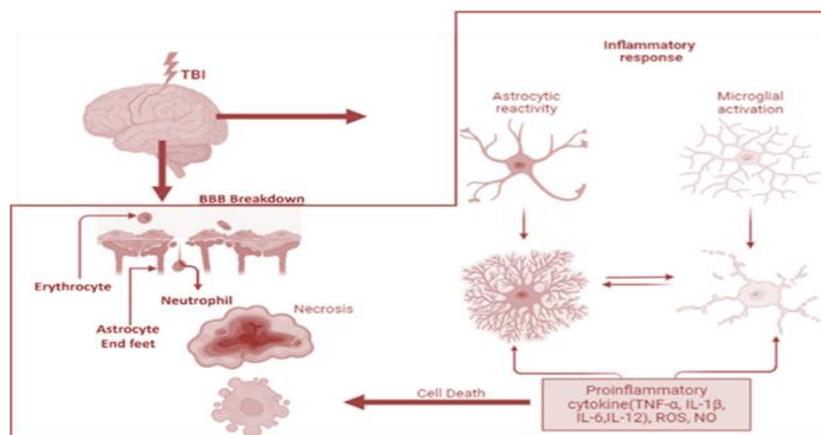


Figure 3: Cell Death occurs Due to Traumatic Brain Injury;

2.4 Improvement of Quality of Life

Neurodegenerative disorders significantly impair cognitive and motor functions, profoundly affecting patients' quality of life. By limiting initial neuronal damage and facilitating repair mechanisms, neuroprotection can substantially improve daily functioning and independence. Cognitive and motor performance can be enhanced by reducing tau pathology and amyloid- β plaque burden, preserving synaptic health, maintaining neurotransmitter balance, protecting neuronal populations, and targeting brain regions such as the hippocampus and amygdala. Additional strategies include safeguarding dopaminergic neurons, supporting overall brain function, and maintaining cognitive and motor abilities. Through these combined mechanisms, neuroprotection plays a vital role in improving quality of life, autonomy, and overall well-being [18-21].

2.5 Reducing Economic Burden

Individuals affected by neurodegenerative disorders require long-term medical care, continuous medication, and assisted support, which collectively impose a substantial financial burden on patients as well as their

families. The availability of effective and affordable therapeutic strategies can significantly reduce healthcare expenditures. Economic burden may be minimized through improved management of chronic conditions, reduction in long-term nursing and hospitalization costs, preservation of cognitive and motor functions, and the use of cost-effective therapeutic interventions. Therefore, the development of novel, affordable, and efficacious treatment approaches is essential not only for alleviating financial strain but also for advancing effective pharmacological management of neurodegenerative disorders [22-24].

3. Significance of Medicinal Plants in Treating Neurodegenerative Disorders

Ancient civilizations possess a rich and diverse history of using natural herbs to treat a wide spectrum of diseases. Traditional medical systems such as Ayurveda, Unani, Siddha, Traditional Chinese Medicine, Traditional Iranian Medicine, and Medieval Islamic Medicine have long utilized medicinal plants to manage various ailments, including neurodegenerative disorders. Medicinal plants are rich sources of biologically active compounds such as flavonoids, terpenoids, alkaloids, and polyphenols, many of which exhibit potent neuroprotective properties. Several plants, including *Curcuma longa*, *Ginkgo biloba*, *Camellia sinensis*, *Withania somnifera*, *Lycopodium serratum* Thunb., *Bacopa monnieri* (water hyssop), and *Rhodiola rosea* (golden root), have demonstrated significant neuroprotective potential in numerous experimental and clinical studies [25-29].

3.1 Antioxidant action

Oxidative stress caused by excessive free radical generation is a major contributor to neuronal damage in neurodegenerative disorders. Several medicinal plants, including *Curcuma longa* and *Ginkgo biloba*, possess strong antioxidant properties that help neutralize free radicals and attenuate oxidative stress, thereby protecting neurons from damage [19, 30-32].

3.2 Anti-inflammatory action

Neuroinflammation is a hallmark feature of neurodegenerative diseases. Medicinal plants such as *Ginkgo biloba* and *Camellia sinensis* have demonstrated significant anti-inflammatory activity, which contributes to the reduction of inflammation-mediated neuronal damage and disease progression [11, 31].

3.3 Neurotransmission Regulation

Certain herbs, including *Bacopa monnieri* and *Lycopodium serratum* Thunb., play a crucial role in enhancing cholinergic neurotransmission, which is essential for maintaining cognitive function. These plants increase acetylcholine levels, thereby improving learning, memory, and overall cognitive performance [33-35].

3.4 Reinforcement of Mitochondrial Function

Plant-derived bioactive compounds from *Withania somnifera* and *Rhodiola rosea* have been shown to improve mitochondrial function, leading to enhanced cellular energy production. Improved mitochondrial efficiency supports neuronal survival, growth, and optimal cellular function, thereby contributing to neuroprotection [36-37].

3.5 Inhibition of amyloid plaques and Tau tangles formation

The accumulation of amyloid- β plaques and tau neurofibrillary tangles represents the pathological hallmark of Alzheimer's disease. Medicinal plants such as *Panax ginseng* and *Centella asiatica* have demonstrated the ability to inhibit the formation and aggregation of amyloid plaques and tau tangles, thereby slowing disease progression and preserving neuronal function [38-40].

4. Important Medicinal Plants and Their Active Constituents

Nature is a rich repository of therapeutic resources, offering a wide array of medicinal plants with remarkable healing potential. Numerous herbs have been traditionally and scientifically recognized for their efficacy in managing neurodegenerative disorders. This section discusses some of the most important medicinal plants and their bioactive constituents that contribute to neuroprotection.

4.1 *Withania somnifera*

Withania somnifera, commonly known as Ashwagandha, Indian ginseng, winter cherry, or gooseberry, is a well-known medicinal plant in traditional medicine. It contains several key phytoconstituents, including alkaloids, tannins, starch, saponins, withanolides, anaferine, and sitoindosides, which exhibit potent anti-stress and rejuvenating properties. Numerous studies have reported its beneficial effects on the brain, including memory enhancement, improvement of cognitive functions, attenuation of inflammation, and promotion of neuronal growth and regeneration [41-43].

4.2 *Bacopa monnieri*

Bacopa monnieri, widely known as Brahmi, Indian pennywort, herb of grace, water hyssop, and gotu kola, is regarded as one of the most prominent neuroprotective herbs. The plant is rich in essential phytoconstituents such as glycosides, flavonoids, alkaloids, saponins, and bacosides A and B. Owing to these compounds, *B. monnieri* enhances memory and learning ability, alleviates stress and anxiety, improves cognitive performance, and regulates neurotransmitter levels [44-46].

4.3 *Panax ginseng*

Panax ginseng, commonly referred to as Asian ginseng, Chinese ginseng, Korean ginseng, or Siberian ginseng, is a well-established medicinal herb with neuroprotective potential. Its major bioactive constituents include ginsenosides, phenolic acids, alkaloids, and polysaccharides. These phytochemicals contribute to its ability to preserve cognitive function, reduce oxidative stress, and exert anti-inflammatory effects, thereby playing a significant role in the management of neurodegenerative disorders [47-49].

4.4 *Ginkgo biloba*

Ginkgo biloba, commonly known as the maidenhair tree, is one of the most widely used herbal remedies for cognitive enhancement. The plant contains bioactive compounds such as ginkgolides, bilobalide, biflavones, and ginkgoghrelins, which are responsible for its memory-enhancing and stress-relieving properties. Additionally, *G. biloba* improves cerebral blood flow, thereby strengthening brain function and offering protection against neurodegenerative disorders [50-51].

4.5 *Rosmarinus officinalis*

Rosmarinus officinalis (rosemary) is native to the Mediterranean region and is well known for its aromatic properties. Due to its pleasant aroma, rosemary is extensively used in culinary practices worldwide. The plant contains several bioactive compounds, including rosmarinic acid, bornyl acetate, camphor, borneol, carnosol, carnosic acid, and 1,8-cineole. Rosemary exhibits strong antioxidant and anti-inflammatory activities and has been shown to enhance cognitive performance, making it a promising neuroprotective agent. Several studies have demonstrated its therapeutic potential against neurodegenerative disorders, with different phytoconstituents exerting distinct biological actions [52-55].

4.5.1 Brain-boosting properties

Rosmarinic acid and carnosic acid are the principal phytoconstituents responsible for rosemary's antioxidant activity, effectively scavenging free radicals and reducing oxidative stress, thereby protecting neurons from injury. Additionally, 1,8-cineole exhibits notable anti-inflammatory effects. Rosemary also promotes neuronal differentiation and growth and enhances learning and memory by modulating cholinergic neurotransmission [56-57].

4.5.2 Prevention of Neurodegeneration

The neuroprotective potential of rosemary against neurodegenerative diseases such as Parkinson's disease, Alzheimer's disease, and dementia has been well documented. Rosemary inhibits the formation and aggregation of amyloid- β plaques, which are responsible for neuronal damage. Furthermore, it helps reduce tau protein tangles, a characteristic pathological feature of Alzheimer's disease [56-58].

4.5.3 Intensification of the Brain Repairing Process

Since neurons are primary targets in neurodegenerative disorders, maintaining their integrity and function is critical. Rosemary has been shown to stimulate neuronal survival and enhance neuronal function, thereby slowing disease progression and supporting brain repair mechanisms [58].

5. Mechanisms of Action of Neuroprotective Ayurvedic Herbs

Ayurvedic herbs exhibit multiple pharmacological properties, including antioxidant, anti-inflammatory, neurotransmitter-modulating, oxidative stress-regulating, and anti-protein aggregation activities, all of which are critically involved in the pathogenesis of neurodegenerative disorders. The combination of various natural herbs possessing these properties can lead to the development of potent neuroprotective agents.

5.1 Anti-oxidant Action

Oxidative stress is one of the major contributors to neuronal damage in neurodegenerative disorders. Several phytoconstituents derived from medicinal plants protect neurons against oxidative stress through multiple mechanisms.

5.1.1 Free Radicals Scavengers

Free radicals, particularly reactive oxygen species (ROS), cause severe damage to neuronal cells by disrupting cellular components. Natural herbs possess antioxidant compounds that neutralize these free radicals, thereby preventing oxidative damage to neuronal structures [59].

5.1.2 Intensification of Endogenous Antioxidant Fortification

The human body contains key antioxidant enzymes such as catalase, superoxide dismutase, and peroxidase that regulate oxidative balance. Plant-derived antioxidant phytoconstituents enhance the activity of these enzymes, leading to efficient neutralization of ROS. For instance, carnosic acid present in rosemary acts as a powerful antioxidant agent by strengthening endogenous antioxidant defense mechanisms [60-62].

5.2 Anti-inflammatory Action through Impeding Pro-inflammatory Cytokines

Pro-inflammatory cytokines such as TNF- α , IL-1 β , and IL-6 play a central role in neuroinflammation. Phytoconstituents like curcumin and rosmarinic acid effectively suppress the production of these cytokines, thereby reducing inflammation and neuronal damage [63-65].

5.3 Altering Neurotropic and Neurotransmitter Functions

Normal brain functioning depends on balanced neurotrophic and neurotransmitter activity. By regulating these pathways, neurodegenerative processes can be attenuated [66-67].

5.3.1 By Targeting Dopamine & Serotonin Functioning

Imbalance in dopamine and serotonin levels is commonly observed in neurodegenerative and neuropsychiatric disorders. Bacosides, the principal phytoconstituents of *Bacopa monnieri* (Brahmi), help maintain the balance of these neurotransmitters and support their optimal functioning in the brain [68-70].

5.3.2 Intensifying levels of Acetylcholine

Acetylcholine is a key neurotransmitter involved in learning and memory. Several herbs contain phytoconstituents that elevate acetylcholine levels or inhibit acetylcholinesterase activity, thereby improving memory and cognitive performance [71-72].

5.3.3 Intensifying Levels of Brain-Derived Neurotrophic Factor and Nerve Growth Factor

Brain-derived neurotrophic factor (BDNF) and nerve growth factor (NGF) play essential roles in neuronal growth, survival, and synaptic plasticity. Herbs such as Brahmi and Ashwagandha have been shown to significantly enhance the levels of these neurotrophic factors, thereby supporting neuronal viability and regeneration [73-74].

5.4 Targeting Oxidative Stress Mechanism

Several medicinal plants contain phytoconstituents capable of attenuating oxidative stress, which directly influences neuronal survival and functional integrity [75-76].

5.4.1 Preventing Alterations in Proteins and DNA

Phytoconstituents protect cellular proteins and DNA from oxidative damage, thereby preserving normal cellular functions and preventing neuronal injury [77].

5.4.2 Staving off Cell Membranes

Cell membranes, composed primarily of lipids and proteins, are particularly vulnerable to oxidative damage. Resveratrol, a phytoconstituent obtained from grapes, effectively inhibits lipid peroxidation, thereby protecting neuronal membranes and maintaining cellular integrity [78].

5.5 Impeding protein Cluster Formation

Protein aggregation, including the formation of amyloid- β plaques and tau tangles, is a hallmark of neurodegenerative disorders. Rosmarinic acid has been shown to inhibit the formation of these protein aggregates and enhance their clearance, thereby slowing disease progression [79-80].

6. Clinical Trials and Research Studies

Several experimental studies have evaluated the neuroprotective potential of rosemary. Administering 20 mg of rosemary extract intraperitoneally to mice daily for five days showed significant improvements in both motor and cognitive functions [81]. In another study, Positive behavioral outcomes were noticed in mice with scopolamine-induced Alzheimer's disease following treatment with rosemary essential oil [82]. Carnosol, one of the major bioactive constituents of rosemary, exhibits strong antioxidant and anti-inflammatory properties. The protective effects of carnosol on SN4741 dopaminergic neuronal cells, highlighting its potential role in neuroprotection [83].

7. Integration of Herbal Remedies with Conventional Therapies

Several studies reported in the literature demonstrate that the integration of naturally derived compounds with conventional pharmacological therapies can produce superior therapeutic outcomes. Such combinations often enhance efficacy while reducing adverse effects.

7.1 Combination of Donepezil and *Ginkgo biloba*

Research has shown that the combination of the conventional drug donepezil with *Ginkgo biloba* extract yields enhanced therapeutic benefits. This combination was found to be more effective in the management of Alzheimer's disease and exhibited fewer adverse effects compared to treatment with donepezil alone [84].

7.2 Combination of Amitriptyline and Curcumin

In one study, the combined administration of amitriptyline and curcumin resulted in a significant reduction in immobility time in both the Forced Swim Test and Tail Suspension Test. This combination improved locomotor activity compared to amitriptyline monotherapy. Additionally, a synergistic antidepressant and anxiolytic effect was observed, accompanied by reduced side effects [85].

7.3 Combination of Diazepam and *Bacopa monnieri* (Brahmi)

An experimental study revealed that both diazepam and Brahmi individually influence the activity of acetylcholinesterase (AChE), potassium, magnesium, calcium, and ATPases, leading to reduced acetylcholine levels. When administered together, these agents exhibited significant synergistic effects in the treatment of epileptic conditions, with a marked reduction in adverse effects [86].

7.4 Combination of Selective Serotonin Reuptake Inhibitors and Rosemary

In a clinical trial involving patients with depression and anxiety receiving selective serotonin reuptake inhibitor (SSRI) therapy, concurrent oral administration of rosemary capsules led to significant behavioral improvement. These findings suggest that SSRIs may be more effective when used as adjunct therapy with rosemary rather than as monotherapy [87]. Collectively, these studies indicate that combining herbal remedies with conventional drugs can provide robust therapeutic strategies for neurodegenerative disorders, offering enhanced efficacy with fewer side effects. Such integrative approaches warrant broader application and further investigation.

8. Challenges Associated with Herbal Remedies

Despite the significant potential of herbal medicines in the treatment of neurodegenerative disorders, several challenges remain. These include issues related to standardization, adulteration, limited clinical trial data, regulatory constraints, marketing difficulties, insufficient research and development, lack of trained personnel and infrastructure, and inefficient cultivation and propagation practices [88]. Government agencies and regulatory authorities must address these challenges to facilitate the development of effective, safe, and standardized herbal therapies, which often demonstrate fewer side effects compared to conventional treatments.

9. Conclusion

Herbal therapeutic approaches hold considerable promise in the management of neurodegenerative diseases, as they enhance cognitive and motor functions while improving overall quality of life. These remedies may also slow disease progression through their multifaceted neuroprotective mechanisms. Continuous and rigorous research on rosemary and other medicinal plants is essential to identify novel and effective treatments for neurodegenerative disorders. Furthermore, the development of integrative therapies combining herbal medicines with conventional drugs may lead to more efficacious treatment strategies, paving the way for improved clinical outcomes in neurological disorders.

10. References

1. Choonara YE, Pillay V, du Toit LC, Modi G, Naidoo D, Ndesendo VMK, Sibambo SR. Trends in the molecular pathogenesis and clinical therapeutics of common neurodegenerative disorders. *Int J Mol Sci.* 2009;10(6):2510–2557.
2. Prusty RK, Begum S, Patil A, Naik DD, Pimple S, Mishra G. Knowledge of symptoms and risk factors of breast cancer among women: a community-based study in a low socio-economic area of Mumbai, India. *BMC Womens Health.* 2020;20:1–12.
3. Hardiman O, Al-Chalabi A, Chio A, Corr EM, Logroscino G, Robberecht W, Shaw PJ, Simmons Z, van den Berg LH. Amyotrophic lateral sclerosis. *Nat Rev Dis Primers.* 2017;3(1):17071.
4. Bano D, Zanetti F, Mende Y, Nicotera P. Neurodegenerative processes in Huntington's disease. *Cell Death Dis.* 2011;2(11):e228.
5. World Health Organization. Neurological disorders: public health challenges. Geneva: World Health Organization; 2006.
6. Stephenson J, Nutma E, van der Valk P, Amor S. Inflammation in CNS neurodegenerative diseases. *Immunology.* 2018;154(2):204–219.
7. Panda S, Kar NR, Padhy RP, Kumar M, De S, Chandel S, Mahapatra C, Jesudasan RE. Design of polylactic acid nanoparticles using central composite factorial design loaded with an anti-inflammatory drug. *Lat Am J Pharm.* 2023;42:3.
8. Bhat SA, Kamal MA, Sastry NY, Ashraf GM. Synopsis on management strategies for neurodegenerative disorders: challenges from bench to bedside in successful drug discovery and development. *Curr Top Med Chem.* 2017;17(12):1371–1378.

9. Petlevski R, Hadžija M, Slijepčević M, Juretić D. Effect of “Antidiabetis” herbal preparation on serum glucose and fructosamine in NOD mice. *J Ethnopharmacol.* 2001;75(2–3):181–184.
10. Adeyemi OO, Akindele AJ, Yemitan OK, Aigbe FR, Fagbo FI. Anticonvulsant, anxiolytic and sedative activities of the aqueous root extract of *Securidaca longepedunculata* Fresen. *J Ethnopharmacol.* 2010;130(2):191–195.
11. Masood A, Nadeem A, Mustafa SJ, O’Donnell JM. Reversal of oxidative stress-induced anxiety by inhibition of phosphodiesterase-2 in mice. *J Pharmacol Exp Ther.* 2008;326(2):369–379.
12. Pandey A, Bani S, Dutt P, Satti NK, Suri KA, Qazi GN. Multifunctional neuroprotective effect of withanone, a compound from *Withania somnifera* roots, in alleviating cognitive dysfunction. *Cytokine.* 2018;102:211–221.
13. Huang JL, Fu ST, Jiang YY, Cao YB, Guo ML, Wang Y, Xu Z. Protective effects of nicotiflorin on memory dysfunction, energy metabolism failure and oxidative stress in multi-infarct dementia model rats. *Pharmacol Biochem Behav.* 2007;86(4):741–748.
14. Medina JH, Viola H, Wolfman C, Marder M, Wasowski C, Calvo D, Paladini AC. Neuroactive flavonoids: new ligands for the benzodiazepine receptors. *Phytomedicine.* 1998;5(3):235–243.
15. Copmans D, Orellana-Paucar AM, Steurs G, Zhang Y, Ny A, Foubert K, Exarchou V, Siekierska A, Kim Y, De Borggraeve W. Methylated flavonoids as anti-seizure agents: naringenin 4’,7-dimethyl ether attenuates epileptic seizures in zebrafish and mouse models. *Neurochem Int.* 2018;112:124–133.
16. Tian J, Fu F, Geng M, Jiang Y, Yang J, Jiang W, Wang C, Liu K. Neuroprotective effect of 20(S)-ginsenoside Rg3 on cerebral ischemia in rats. *Neurosci Lett.* 2005;374(2):92–97.
17. Rangarajan V, Juul SE. Erythropoietin: emerging role in neonatal neuroprotection. *Pediatr Neurol.* 2014;51(4):481–488.
18. Sonntag WE, Ramsey M, Carter CS. Growth hormone and insulin-like growth factor-1 (IGF-1) and their influence on cognitive aging. *Ageing Res Rev.* 2005;4(2):195–212.
19. Yang H, Zeng F, Luo Y, Zheng C, Ran C, Yang J. Curcumin scaffold as a multifunctional tool for Alzheimer’s disease research. *Molecules.* 2022;27(12):3879.
20. Barnes DE, Yaffe K. The projected effect of risk factor reduction on Alzheimer’s disease prevalence. *Lancet Neurol.* 2011;10(9):819–828.
21. Leal-Galicia P, Romo-Parra H, Rodríguez-Serrano LM, Buenrostro-Jáuregui M. Regulation of adult hippocampal neurogenesis exerted by sexual, cognitive and physical activity: an update. *J Chem Neuroanat.* 2019;101:101667.
22. GBD 2016 Neurology Collaborators. Global, regional, and national burden of neurological disorders, 1990–2016: a systematic analysis for the Global Burden of Disease Study 2016. 2019;18(5):459–480.
23. Feigin VL, Norrving B, George MG, Foltz JL, Roth GA, Mensah GA. Prevention of stroke: a strategic global imperative. *Nat Rev Neurol.* 2016;12(9):501–512.
24. Fereshtehnejad SM, Vosoughi K, Heydarpour P, Sepanlou SG, Farzadfar F, Tehrani-Banihashemi A, Malekzadeh R, Sahraian MA, Vollset SE, Naghavi M. Burden of neurodegenerative diseases in the Eastern Mediterranean region, 1990–2016: findings from the Global Burden of Disease Study 2016. *Eur J Neurol.* 2019;26(10):1252–1265.
25. Bhattacharya SK, Satyan KS, Ghosal S. Antioxidant activity of glycowithanolides from *Withania somnifera*. *Indian J Exp Biol.* 1997;35(3):236–239.
26. Aggarwal BB, Gupta SC, Sung B. Curcumin: an orally bioavailable blocker of TNF and other pro-inflammatory biomarkers. *Br J Pharmacol.* 2013;169(8):1672–1692.
27. McKenna DJ, Jones K, Hughes K. Efficacy, safety, and use of *Ginkgo biloba* in clinical and preclinical applications. *Altern Ther Health Med.* 2001;7(5):70–75.
28. Summerlin N, Soo E, Thakur S, Qu Z, Jambhrunkar S, Popat A. Resveratrol nanoformulations: challenges and opportunities. *Int J Pharm.* 2015;479(2):282–290.
29. Russo A, Borrelli F. *Bacopa monniera*, a reputed nootropic plant: an overview. *Phytomedicine.* 2005;12(4):305–317.
30. Singh SK, Srivastav S, Castellani RJ, Plascencia-Villa G, Perry G. Neuroprotective and antioxidant effect of *Ginkgo biloba* extract against Alzheimer’s disease and other neurological disorders. *Neurotherapeutics.* 2019;16(3):666–674.
31. Oken BS, Storzbach DM, Kaye JA. The efficacy of *Ginkgo biloba* on cognitive function in Alzheimer disease. *Arch Neurol.* 1998;55(11):1409–1415.
32. Antona ME, González PM, Ramos C, Cabrera J, Olano C, Morales C, Zago V, Steimetz T, Puntarulo S, Friedman SM. Curcumin exerts a protective effect against obesity and liver injury induced by an atherogenic diet. *Funct Foods Health Dis.* 2021;11(12):673–689.

33. Hanif K, Kumar M, Singh N, Shukla R. Effect of homeopathic *Lycopodium clavatum* on memory functions and cerebral blood flow in memory-impaired rats. *Homeopathy*. 2015;104(1):24–28.
34. Prabhakar S, Saraf MK, Pandhi P, Anand A. *Bacopa monniera* exerts anti-amnesic effect on diazepam-induced anterograde amnesia in mice. *Psychopharmacology (Berl)*. 2008;200:27–37.
35. Sumathi T, Nathiya VC, Sakthikumar M. Protective effect of bacoside-A against morphine-induced oxidative stress in rats. *Indian J Pharm Sci*. 2011;73(4):409–413.
36. Lee DH, Ahn J, Jang YJ, Seo HD, Ha TY, Kim MJ, Huh YH, Jung CH. *Withania somnifera* extract enhances energy expenditure via improving mitochondrial function in adipose tissue and skeletal muscle. *Nutrients*. 2020;12(2):431.
37. Chattopadhyay D, Thirumurugan K. Longevity-promoting efficacies of different plant extracts in lower model organisms. *Mech Ageing Dev*. 2018;171:47–57.
38. Gray NE, Morré J, Kelley J, Maier CS, Stevens JF, Quinn JF, Soumyanath A. Caffeoylquinic acids in *Centella asiatica* protect against amyloid- β toxicity. *J Alzheimers Dis*. 2014;40(2):359–373.
39. Kim DO, Heo HJ, Kim YJ, Yang HS, Lee CY. Sweet and sour cherry phenolics and their protective effects on neuronal cells. *J Agric Food Chem*. 2005;53(26):9921–9927.
40. Zhao HH, Di J, Liu WS, Liu HL, Lai H, Lü YL. Involvement of GSK3 and PP2A in ginsenoside Rb1 attenuation of aluminum-induced tau hyperphosphorylation. *Behav Brain Res*. 2013;241:228–234.
41. Ali A, Maher S, Khan SA, Chaudhary MI, Musharraf SG. Sensitive quantification of six steroidal lactones in *Withania coagulans* extract by UHPLC electrospray tandem mass spectrometry. *Steroids*. 2015;104:176–181.
42. Dar NJ, Ahmad M. Neurodegenerative diseases and *Withania somnifera* (L.): an update. *J Ethnopharmacol*. 2020;256:112769.
43. Lavie D, Glotter E, Shvo Y. Constituents of *Withania somnifera* Dun. Part IV. The structure of withaferin A. *J Chem Soc*. 1965;7517–7531.
44. Devishree RA, Kumar S, Jain AR. Short-term effect of *Bacopa monnieri* on memory—a brief review. *J Pharm Res*. 2017;11:1447–1450.
45. Dhawan BN, Singh HK. Pharmacological studies on *Bacopa monniera*, an Ayurvedic nootropic agent. *Eur Neuropsychopharmacol*. 1996;6:144.
46. Ramasamy S, Chin SP, Sukumaran SD, Buckle MJC, Kiew LV, Chung LY. In silico and in vitro analysis of bacoside A aglycones and its derivatives as the constituents responsible for the cognitive effects of *Bacopa monnieri*. *PLoS One*. 2015;10(5):e0126565.
47. Cho IH. Volatile compounds of ginseng (*Panax sp.*): a review. *J Korean Soc Appl Biol Chem*. 2015;58:67–75.
48. Park J, Cho J. Anti-inflammatory effects of ginsenosides from *Panax ginseng* and their structural analogs. *Afr J Biotechnol*. 2009;8(16).
49. Kim JH, Yi YS, Kim MY, Cho JY. Role of ginsenosides, the main active components of *Panax ginseng*, in inflammatory responses and diseases. *J Ginseng Res*. 2017;41(4):435–443.
50. Dong HL, Lin S, Wu QL, Su RX, Wu ZL, Dong HY, Li HL, Zhang WD. A new bilobalide isomer and two cis-coumaroylated flavonol glycosides from *Ginkgo biloba* leaves. *Fitoterapia*. 2020;142:104516.
51. Youdim KA, Joseph JA. A possible emerging role of phytochemicals in improving age-related neurological dysfunctions: a multiplicity of effects. *Free Radic Biol Med*. 2001;30(6):583–594.
52. Yu MH, Choi JH, Chae IG, Im HG, Yang SA, More K, Lee IS, Lee J. Suppression of LPS-induced inflammatory activities by *Rosmarinus officinalis* L. *Food Chem*. 2013;136(2):1047–1054.
53. Pérez-Fons L, Garzón MT, Micol V. Relationship between the antioxidant capacity and effect of rosemary (*Rosmarinus officinalis* L.) polyphenols on membrane phospholipid order. *J Agric Food Chem*. 2010;58(1):161–171.
54. Lovkova MY, Buzuk GN, Sokolova SM, Kliment'eva NI. Chemical features of medicinal plants. *Appl Biochem Microbiol*. 2001;37:229–237.
55. Begum A, Sandhya S, Vinod KR, Reddy S, Banji D. An in-depth review on the medicinal flora *Rosmarinus officinalis* (Lamiaceae). *Acta Sci Pol Technol Aliment*. 2013;12(1):61–74.
56. Machado DG, Cunha MP, Neis VB, Balen GO, Colla A, Bettio LE, Oliveira A, Pazini FL, Dalmarco JB, Simionatto EL, Pizzolatti MG, Rodrigues AL. Antidepressant-like effects of fractions, essential oil, carnosol and betulonic acid isolated from *Rosmarinus officinalis* L. *Food Chem*. 2013;136(2):999–1005.
57. Sasaki K, El Omri A, Kondo S, Han J, Isoda H. *Rosmarinus officinalis* polyphenols produce antidepressant-like effect through monoaminergic and cholinergic functions modulation. *Behav Brain Res*. 2013;238:86–94.

58. Pengelly A, Snow J, Mills SY, Scholey A, Wesnes K, Butler LR. Short-term study on the effects of rosemary on cognitive function in an elderly population. *J Med Food*. 2012;15(1):10–17.
59. Cantuti-Castelvetri I, Shukitt-Hale B, Joseph JA. Neurobehavioral aspects of antioxidants in aging. *Int J Dev Neurosci*. 2000;18(4–5):367–371.
60. Brigelius-Flohé R. Tissue-specific functions of individual glutathione peroxidases. *Free Radic Biol Med*. 1999;27(9–10):951–965.
61. Ho YS, Magnenat JL, Gargano M, Cao J. The nature of antioxidant defense mechanisms: a lesson from transgenic studies. *Environ Health Perspect*. 1998;106(Suppl 5):1219–1228.
62. Zelko IN, Mariani TJ, Folz RJ. Superoxide dismutase multigene family: a comparison of the CuZn-SOD (SOD1), Mn-SOD (SOD2), and EC-SOD (SOD3) gene structures, evolution, and expression. *Free Radic Biol Med*. 2002;33(3):337–349.
63. Ueda H, Ippoushi K, Takeuchi A. Repeated oral administration of a squeezed ginger (*Zingiber officinale*) extract augmented the serum corticosterone level and had anti-inflammatory properties. *Biosci Biotechnol Biochem*. 2010;74(11):2248–2252.
64. Amaral GP, de Carvalho NR, Barcelos RP, Dobrachinski F, Portella Rde L, da Silva MH, Lugokenski TH, Dias GR, da Luz SC, Boligon AA, Athayde ML, Villetti MA, Antunes Soares FA, Fachinetto R. Protective action of ethanolic extract of *Rosmarinus officinalis* L. in gastric ulcer prevention induced by ethanol in rats. *Food Chem Toxicol*. 2013;55:48–55.
65. Dickmann LJ, VandenBrink BM, Lin YS. In vitro hepatotoxicity and cytochrome P450 induction and inhibition characteristics of carnosic acid, a dietary supplement with antiadipogenic properties. *Drug Metab Dispos*. 2012;40(7):1263–1267.
66. Dumas JA, Newhouse PA. The cholinergic hypothesis of cognitive aging revisited again: cholinergic functional compensation. *Pharmacol Biochem Behav*. 2011;99(2):254–261.
67. Kumar R, Saha P, Lokare P, Datta K, Selvakumar P, Chourasia A. A systematic review of *Ocimum sanctum* (Tulsi): morphological characteristics, phytoconstituents and therapeutic applications. *Int J Res Appl Sci Biotechnol*. 2022;9(2):221–226.
68. Lieberman JA, Bymaster FP, Meltzer HY, Deutch AY, Duncan GE, Marx CE, Aprille JR, Dwyer DS, Li XM, Mahadik SP, Duman RS, Porter JH, Modica-Napolitano JS, Newton SS, Csernansky JG. Antipsychotic drugs: comparison in animal models of efficacy, neurotransmitter regulation, and neuroprotection. *Pharmacol Rev*. 2008;60(3):358–403.
69. Melnik T, Garcia Soares B, dos Santos Puga ME, Atallah ÁN. Efficacy and safety of atypical antipsychotic drugs (quetiapine, risperidone, aripiprazole, and paliperidone) compared with placebo or typical antipsychotic drugs for treating refractory schizophrenia: overview of systematic reviews. *Sao Paulo Med J*. 2010;128:141–166.
70. Meltzer HY, Massey BW. The role of serotonin receptors in the action of atypical antipsychotic drugs. *Curr Opin Pharmacol*. 2011;11(1):59–67.
71. Ahmed F, Urooj A. Anticholinesterase activities of cold and hot aqueous extracts of *Ficus racemosa* stem bark. *Pharmacogn Mag*. 2010;6(22):142.
72. Bores GM, Huger FP, Petko W, Mutlib AE, Camacho F, Rush DK, Selk DE, Wolf V, Kosley RW Jr, Davis L, Vargas HM. Pharmacological evaluation of novel Alzheimer's disease therapeutics: acetylcholinesterase inhibitors related to galanthamine. *J Pharmacol Exp Ther*. 1996;277(2):728–738.
73. Poo M. Neurotrophins as synaptic modulators. *Nat Rev Neurosci*. 2001;2(1):24–32.
74. Lu B, Nagappan G, Guan X, Nathan PJ, Wren P. BDNF-based synaptic repair as a disease-modifying strategy for neurodegenerative diseases. *Nat Rev Neurosci*. 2013;14(6):401–416.
75. Ma T, Klann E. Amyloid beta: linking synaptic plasticity failure to memory disruption in Alzheimer's disease. *J Neurochem*. 2012;120:140–148.
76. Halliwell B. Reactive oxygen species and the central nervous system. *J Neurochem*. 1992;59(5):1609–1623.
77. Freeman BA, Crapo JD. Biology of disease: free radicals and tissue injury. *Lab Invest*. 1982;47(5):412–426.
78. Lu Q, Hao M, Wu W, Zhang N, Isaac AT, Yin J, Zhu X, Du L, Yin X. Antidiabetic cataract effects of GbE, rutin and quercetin are mediated by the inhibition of oxidative stress and polyol pathway. *Acta Biochim Pol*. 2017;65(1):35–41.
79. Atanasov AG, Waltenberger B, Pferschy-Wenzig EM, Linder T, Wawrosch C, Uhrin P, Temml V, Wang L, Schwaiger S, Heiss EH, Rollinger JM, Schuster D, Breuss JM, Bochkov V, Mihovilovic MD, Kopp B, Bauer R, Dirsch VM, Stuppner H. Discovery and resupply of pharmacologically active plant-derived natural products: A review. *Biotechnol Adv*. 2015;33(8):1582–1614.

80. Niranjan A, Prakash D. Chemical constituents and biological activities of turmeric (*Curcuma longa* L.): a review. *J Food Sci Technol*. 2008;45(2):109.
81. Li M, Cui MM, Kenechukwu NA, Gu YW, Chen YL, Zhong SJ, Gao YT, Cao XY, Wang L, Liu FM, Wen XR. Rosmarinic acid ameliorates hypoxia/ischemia induced cognitive deficits and promotes remyelination. *Neural Regen Res*. 2020;15(5):894–902.
82. Satou T, Hanashima Y, Mizutani I, Koike K. The effect of inhalation of essential oil from *Rosmarinus officinalis* on scopolamine-induced Alzheimer's type dementia model mice. *Flavour Fragr J*. 2018;33(3):230–234.
83. Kim SJ, Kim JS, Cho HS, Lee HJ, Kim SY, Kim S, Lee SY, Chun HS. Carnosol, a component of rosemary (*Rosmarinus officinalis* L.) protects nigral dopaminergic neuronal cells. *Neuroreport*. 2006;17(16):1729–1733.
84. Yancheva S, Ihl R, Nikolova G, Panayotov P, Schlaefke S, Hoerr R; GINDON Study Group. Ginkgo biloba extract EGb 761®, donepezil or both combined in the treatment of Alzheimer's disease with neuropsychiatric features: a randomised, double-blind, exploratory trial. *Aging Ment Health*. 2009;13(2):183–190.
85. Patel P, Gohil KJ, Roy SP, Patel N. Investigation of antidepressant and anxiolytic activity of curcumin given alone and in combination with amitriptyline in rats. *Indian J Res Pharm Biotechnol*. 2014;2(3):1173–1177.
86. Komali E, Venkataramaiah C, Rajendra W. Antiepileptic potential of *Bacopa monnieri* in the rat brain during PTZ-induced epilepsy with reference to cholinergic system and ATPases. *J Tradit Complement Med*. 2021;11(2):137–143.
87. Azizi S, Mohamadi N, Sharififar F, Dehghannoudeh G, Jahanbakhsh F, Dabaghzadeh F. Rosemary as an adjunctive treatment in patients with major depressive disorder: a randomized, double-blind, placebo-controlled trial. *Complement Ther Clin Pract*. 2022;49:101685.
88. Rukangira E. The African herbal industry: constraints and challenges. *Erboristeria Domani*. 2001;1:1–23.

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