Medicinal Plants as Neuroprotective Agents in Neurodegenerative Disorders

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Abstract

Neurodegenerative diseases, including Alzheimer's disease (AD), Parkinson's disease (PD), and Huntington's disease (HD), present significant global health issues, with existing treatments primarily providing limited symptom relief. Consequently, there is an increasing scientific focus on the neuroprotective capabilities of medicinal plants that have been utilized in various traditional healing practices. This review examines the therapeutic properties of Ginkgo biloba, Crocus sativus (saffron), Curcuma longa (turmeric), Mucuna pruriens, and Withania somnifera (Ashwagandha) plants that are abundant in bioactive compounds such as flavonoids, alkaloids, terpenoids, and polyphenols. These phytochemicals possess strong antioxidant, anti-inflammatory, and anti-apoptotic effects, which aid in combating oxidative stress, diminishing protein aggregation, and modulating neurotransmitter levels. Furthermore, plants like Centella asiatica (Gotu Kola) and Bacopa monnieri (Brahmi) have demonstrated potential in improving cognitive function and maintaining neuronal health. Mechanistic investigations reveal that the modulation of acetylcholinesterase activity and the reduction of reactive oxygen species are significant neuropharmacological mechanisms. Although these results highlight the potential of medicinal plants as neuroprotective agents, additional clinical studies are necessary to confirm their safety and effectiveness as either standalone or complementary treatments for neurodegenerative disorders.

Keywords: Neurodegeneration, Alzheimer's, Parkinson's, neuroprotection, Phytochemicals, Neuroinflammation, *Curcuma longa, Bacopa monnieri, Withania somnifera, Ginkgo biloba*

Introduction

Neurodegenerative disorders are mainly defined by the gradual loss of neurons, resulting in the slow decline of cognitive and motor abilities. Among these disorders, Alzheimer's disease (AD) and Parkinson's disease (PD) are the most common, having a

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significant effect on aging populations globally. Current pharmacological interventions primarily aim to alleviate symptoms rather than prevent disease progression. This limited effectiveness is largely due to the blood-brain barrier (BBB), which hinders the passage of therapeutic agents into the central nervous system. Additionally, the pathophysiological processes associated with many neurodegenerative diseases are still not well understood. As a result, researchers have begun to investigate environmental factors such as exposure to heavy metals, pesticides, and air pollutants as possible contributors to neurodegeneration (Dehaghi et al., 2022; Oran et al., 2022).

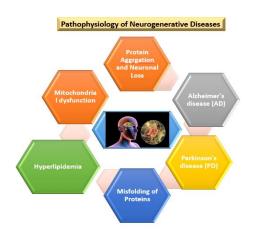


Figure 1. Pathophysiology of Neurodegenerative Diseases:
Alzheimer's and Parkinson's disease

Epidemiological studies indicate a possible connection between environmental factors and an elevated risk of conditions such as amyotrophic lateral sclerosis (ALS), progressive supranuclear palsy (PSP), multiple system atrophy (MSA), Alzheimer's disease (AD), and Parkinson's disease (PD). However, these correlations are frequently inconsistent across various studies, attributed to differences in exposure assessment, population demographics, and diagnostic precision. Neurodegenerative diseases often exhibit overlapping symptoms, which complicates clinical differentiation, especially in instances of Parkinson-plus syndromes (e.g., MSA and PSP). Furthermore, coexisting vascular conditions such as stroke and coronary heart disease can further complicate the diagnostic process. As diagnostic criteria continue to evolve, achieving accurate classification remains a significant challenge, highlighting the necessity for early detection methods and

biomarkers that can facilitate differential diagnosis prior to the onset of irreversible brain damage (Brown *et al.*, 2005; Ashkevari & Ghasemi, 2023; Tural & Şahan, 2023; Ha & Hang, 2024) (Figure 1).

Recent advancements in neuroimaging and neuropsychological assessments have enhanced early diagnostic capabilities, although these techniques still necessitate further refinement. Current studies underscore the significance of incorporating genetic, environmental, and lifestyle factors to gain a deeper understanding of disease origins. There is a growing agreement that extensive longitudinal research is vital for identifying causative elements and enhancing diagnostic accuracy. The rising prevalence of neurodegenerative diseases in the United States and worldwide underscores the pressing need for more effective environmental health policies and focused prevention initiatives (Scheltens *et al.*, 2021; Hoang *et al.*, 2022; Tam *et al.*, 2022; Son *et al.*, 2023). Ultimately, a multidisciplinary strategy that integrates environmental science, neurology, and molecular biology will be essential in tackling these intricate disorders.

The Genetic Basis and Clinical Manifestations of Neurodegenerative Disorders

Neurodegenerative disorders exhibit significant genetic foundations, acting as both inherited characteristics and risk factors in sporadic instances. The identification of genes associated with these diseases and their risk loci has led to significant advancements in medicine, illuminating critical pathways and mechanisms of the diseases. Progress in molecular biology, genomics, and innovative research methodologies has enhanced our comprehension of the genetics underlying neurodegenerative conditions. For instance, the analysis of restriction fragment-length

polymorphism facilitated the discovery of a marker on chromosome 4 related to disease manifestation. In the 1990s, scientists identified causative genes: the APP gene on chromosome 21 was linked to familial Alzheimer's disease (AD), while the SNCA gene on chromosome 4 was associated with Parkinson's disease (PD). These discoveries validated that both conditions can exhibit Mendelian inheritance patterns in certain families, whereas others may be affected by environmental influences and random (stochastic) factors (Pihlstrøm et al., 2018; Gondo & Haryanti, 2024; Linh et al., 2024; Saliev et al., 2024; Zharashueva et al., 2024; Abbas et al., 2025).

Genetic Symptoms in Neurodegenerative Diseases

- Alzheimer's Disease (AD)
- Loss of memory (connected to APP and other genes such as PSEN1, PSEN2)
- Decline in cognitive function
- Alterations in mood
- Challenges in performing tasks
- Parkinson's Disease (PD)
- Tremors (linked to SNCA mutations)
- Stiffness
- Bradykinesia (sluggishness in movement)
- Imbalance
- Challenges with speech and facial expressions

These symptoms, influenced by genetics, frequently intersect, and grasping their genetic origins is vital for timely diagnosis and possible treatment options (Figure 2).

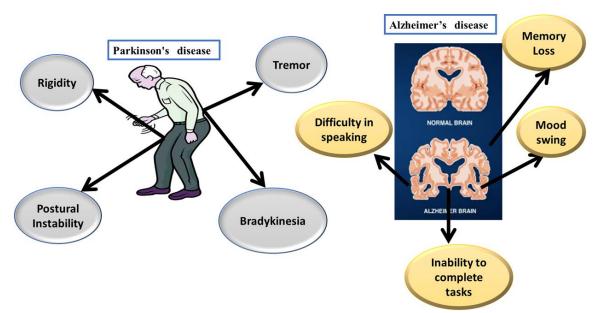


Figure 2. A comparative analysis of the symptoms and neurological alterations associated with Parkinson's disease and Alzheimer's disease. Parkinson's primarily impacts motor functions, such as tremors and rigidity, whereas Alzheimer's primarily affects cognitive abilities, including memory impairment and mood fluctuations.

Nerve Growth Factors

NGF enables its effects through three fundamental cell types: structural fibroblasts, smooth muscle cells, hepatocytes, endothelium, and epithelial cells, in addition to auxiliary Müller cells and astrocytes, and immunological cells, including eosinophils, granulocytes, lymphocytes, mast cells, and antigenpresenting cells. The combination of soluble factors that includes cytokines, neuropeptides, and growth factors enables NGF to induce cell activation and differentiation, along with proliferation, migration, and survival promotion in these cell types. NGF shows great potential in the treatment of numerous human illnesses. This substance functions as a possible therapeutic choice because it affects neuronal growth and survival, along with neuronal activation. Two receptors comprise the biological activity of the insulin-like molecule NGF: p75NTR, as the transmembrane glycoprotein also called "high-affinity receptor", and the tropomyosin receptor kinase with tyrosine kinase activity. The p75NTR receptor interacts with all neurotrophic factors, yet binds Trk receptors differently because NGF engages TrkA, whereas NT-3 connects to TrkC, and BDNF binds to TrkB. Upon receptor binding of the ligand, this family of receptors activates their tyrosine kinase domain, which promotes substrate phosphorylation to enable diverse biological responses such as immune cell development and survival, healing processes, and maintenance functions for neurons. The distribution pattern of these receptors on cellular surfaces establishes the effect of NGF (Abdel-Hadi & Abdel-Fattah, 2022; Akbari, 2022; Perwitasari et al., 2023; Gavioli et al., 2024; Levochkina et al., 2024; Molas-Tuneu et al., 2024). Neurotrophin proteins (NGF, BDNF, NT3, and NT4) exert their effects through both CNS development and aging with normal or pathologic conditions using four mechanisms: Trk receptors, PI3K/Akt-prosurvival gene transcription, MAPK/ERKdifferentiation promoting substrate activation, and IP3-DAGinduced calcium release. Prior to their maturation process, proneurotrophins have low binding affinity to the tumour necrosis factor receptor p75NTR and induce cellular death in both neuronal and non-neuronal cells upon receptor activation. Research shows that the growth factor glial-derived neurotrophic factor (GDNF) protects cell cultures of dopaminergic neurones through its in the transforming membership growth factor (TGFβ) superfamily (Enciu et al., 2011; Özatik et al., 2022; Asar et al., 2023; Petronis et al., 2023).

Role of Oxidative Stress and Inflammation in Neurobiology

Inflammation

The blood-brain barrier (BBB) function forces scientists to consider the brain as an immunologically privileged organ, thus minimizing the analysis of neuro-immune interactions. Research data from preclinical and clinical studies demonstrates that neural-immune communication occurs in two directions through essential neural-immune connections. Medical science experts classify inflammatory processes as weapons that cut both ways. The inflammatory process operates as a protective defense mechanism until it develops into dangerous chronic inflammation, thus becoming a crucial factor in neuropathological events. Neuroinflammation occurs prior to psychotic symptoms; therefore,

scientists consider it significant in understanding psychosis development. Mental disorders display an inflammatory response that produces elevated levels of pro-inflammatory cytokines to define their condition. Clinical research on peripheral procytokines in patients with first-episode inflammatory schizophrenia produced findings of substantial correlations between cytokine levels and disease symptoms. Long-term methamphetamine utilization creates an inflammatory imbalance that results in higher amounts of pro-inflammatory cytokines. The constant activation of the immune system disrupts neurochemical processes that transmit signals due to higher levels of these proinflammatory proteins. Comprehensive research immunological dysfunction in schizophrenia was analyzed by Miller and Goldsmith. The authors analyzed the inconsistent findings from schizophrenia treatment using immunomodulatory therapies and recommended monoclonal antibody immunotherapy with adjunctive effects as potential future strategies. Clinical research on peripheral pro-inflammatory cytokines in patients with first-episode schizophrenia produced findings of substantial correlations between cytokine levels and disease symptoms. Longterm methamphetamine utilization creates an inflammatory imbalance that results in higher amounts of pro-inflammatory cytokines. The constant activation of the immune system disrupts neurochemical processes that transmit signals due to higher levels of these pro-inflammatory proteins. Comprehensive research on immunological dysfunction in schizophrenia was analyzed by Miller and Goldsmith. The authors analyzed the inconsistent findings from schizophrenia treatment using immunomodulatory therapies and recommended monoclonal antibody immunotherapy with adjunctive effects as potential future strategies (Abozor & Abduljawad, 2022; Mickevičius et al., 2023; Alqara et al., 2024; Rawani et al., 2024; Soman et al., 2024).

Oxidative Stress

Scientists define oxidative stress as a condition appearing when the body cannot eliminate reactive products or free radicals or create reactive species. Neurotoxicity and cellular malfunction, together with molecular damage and necrotic and apoptotic cell-death pathways, result from the excessive production of these reactive species. The nitroxyl anion, alongside other nitrogen oxides, belongs to the reactive oxygen species group (such as hydrogen peroxide and superoxide free radicals) that oxidant stress literature mainly focuses on. Mitochondrial production of ATP leads to the generation of ROS. Regular health situations allow the body to maintain redox balance, the state of equilibrium between tissue oxidation and reduction. The medical literature defines oxidative stress as a situation that arises from conflicts between reactive product detoxification capability and reactive species production rates within the human body.

Neurotoxicity and cellular malfunction with subsequent molecular damage alongside necrotic and apoptotic cell-death signaling become possible when reactive species are present in high amounts. RNS such as the nitroxyl anion, alongside other nitrogen oxides, function as ROS, which includes hydrogen peroxide and superoxide free radicals that occupy the majority of reports on oxidative stress in the literature. ROS are generated by the mitochondrial ATP production. Without an abnormality, the body

maintains the redox balance that represents the state of equal oxidation and reduction in tissues. Oxidative stress interacts with a variety of physiological processes in both directions, affecting neurotransmission, neuroinflammation, and homeostatic networks, including the HPA axis. We have discussed the potential role of oxidative stress in theories of psychosis that involve the immune system, dopamine, glutamate, and impaired brain connections. Compared blood indicators of oxidative stress and inflammation in first-episode psychosis (FEP) patients and healthy controls in a comprehensive analysis of cross-sectional studies.

The blood data showed FEP patients had elevated homocysteine and IL-6 and TNF- α concentrations when compared to controls, but they simultaneously presented diminished docosahexaenoic acid and total antioxidant status. Research findings showed that patients with first-episode psychosis demonstrated both elevated pro-inflammatory conditions and decreased antioxidant protection. The DATs that regulate dopamine breakdown become dysfunctional as ROS affects them (Bahamid et al., 2022; Sindhu et al., 2023; Belfiore et al., 2024; Odeh et al., 2024; Upchezhokov et al., 2024; Zangiabadi et al., 2025). Experimental rodent research showed that diminished drug reuptake causes dopaminergic increases in synaptic cleft levels in the nucleus accumbens. Dopamine excess causes auto-oxidation in the brain cells, thus becoming a primary trigger of brain oxidative stress. Dopamine intensifies oxidative stress harm through its reduction of how well antioxidants function. The hippocampus and PFC areas affected by synaptic dopamine participate in dopaminergic effects that lead to cellular pro-oxidation. Research through positron emission tomography imaging demonstrates that higher levels of dopaminergic neurotransmission lead to unhealthy conditions by amplifying the production of reactive oxygen species (ROS). The ability of protective antioxidant enzymes becomes impaired by psychostimulant use. Reactive astrocytes and microglia maintain a cyclic process that results in ROS production and sustained inflammation while killing cells and enhancing the production of pro-inflammatory cytokines. The immune response appears to underlie oxidative stress, especially among psychotic patients. Oxidative stress serves as an example of a bidirectional psychosis mechanism because it triggers neuroinflammation. Research has established that elevated oxidative stress levels function as a mediator of psychosis development in immunological activation conditions. ROS, alongside pro-inflammatory cytokines produced by reactive microglia, can damage the BBB according to scientific evidence. The detection of oxidative stress and neuroinflammatory processes of the immune system during pathological events suggests that this stress behavior both initiates and results from neuroinflammation (Liu et al., 2022; Makhoahle & Gaseitsiwe, 2022; Teleanu et al., 2022; Graefen et al., 2023; İlaslan et al., 2023; Thazha et al., 2023).

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Importance of Medicinal Plants in Neurodegenerative Diseases

American Alzheimer's, Parkinson's disease, and Huntington's disease make up the neurodegenerative diseases that occur more frequently due to humans living longer. Plant extracts have consistently been employed in human history to treat numerous diseases because they possess several therapeutic properties. Scientific researchers now study the neuroprotective effects, along with the biological activities of medicinal plants. Research using animal subjects illustrates that the plant's fruit and root bark possess all three effects, including hypotensive property and hypoglycaemic and antipyretic action. Traditional medicine practitioners in China, along with Japan and Korea, prescribe Lycium chinense Miller for treatment purposes. The plant serves medical purposes for treating neurological conditions and shows effects that reduce aging symptoms. The protective properties of various plant materials against neural cell damage have been extensively researched. Two individual studies proved that extracts from Camellia sinensis and Erigeron breviscapus protected PC12 cells from hydrogen peroxide damage, along with a neuroprotective action demonstrated from Smilacis chinensis plant rhizome in N-methyl-d-aspartate-induced neurotoxicity models. The research showed parallel results when tested on animals with focal cerebral ischaemia (Uddin et al., 2013; AlYousef et al., 2022; Najjar, 2023; Padma et al., 2023).

Plant-Based Interventions in Alzheimer's Disease

Crocus Sativus

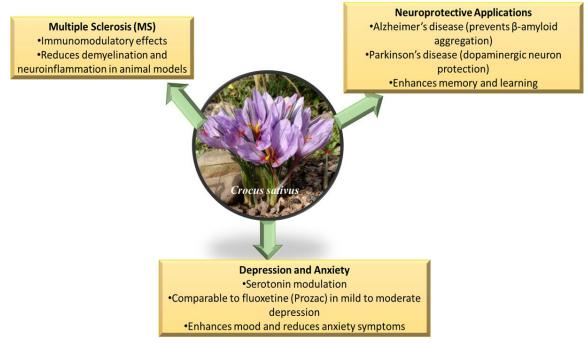


Figure 3. Crocus sativus and its neuroprotective functions against neurodegenerative diseases

Crocus sativus L. stands out among therapeutic plants because of its increasing popularity. Crocus sativus L. belongs to the Iridaceae family and grows throughout Greece, Italy, Spain, Israel, Morocco, Turkey, Iran, India, China, Egypt, Mexico, and other regions. The purpose of this research focuses on the neuroprotective functions of Crocus sativus L., especially in treating Parkinson's disease and Alzheimer's disease. The metabolic redox reactions in cells generate unavoidable byproducts known as reactive oxygennitrogen species, and together they are designated as reactive oxygen-nitrogen species. These species are produced during phagocytosis, oxidative phosphorylation in the respiratory chain of mitochondria, eicosanoid synthesis, and redox reactions that involve metals with variable valence and biotransformation reactions of exogenous and endogenous substrata in endoplasmic reticulum cells (D'Onofrio et al., 2021; Aruta et al., 2023; Gurubasajar et al., 2023; Bergeron et al., 2024; Naseri et al., 2024). Saffron or Crocus sativus has existed as a color and flavoring agent and medicinal herb since the beginning of human civilization. Scientists continue to identify emerging research that demonstrates the pharmacological benefits of saffron alongside its key compounds for CNS therapy applications. Crocus sativus L. has neuroprotective effects on Alzheimer's disease (AD) patients' cognitive impairment. Reviewing the data and mechanisms

underlying saffron-induced therapeutic effects and quantifiable cognitive advantages in AD is the aim of this study. Studies that were preclinical and clinical were found. These criteria were used to choose preclinical in vitro and in vivo studies: 1) creation of the pharmacological profile of saffron on biological or biophysical endpoints; 2) assessment of saffron effectiveness using animal screens as an AD model; and 3) studies lasting at least three months that show that crocin seems to be able to control glutamate levels, lower oxidative stress, and modify tau and Aß proteins. Research investigates how saffron functions as a neuroprotective agent in age-related macular degeneration (AMD) cases (D'Onofrio et al., 2021; Al Hemaidi, 2024). Medical evidence from pharmacological studies demonstrates that saffron supplementation therapy protects the brain through various scientific investigations that link its therapeutic properties to crocins, crocetin, picrocrocin, and safranal compounds. Studies indicate that saffron carotenoids serve as the primary source of antioxidant properties based on the evidence throughout most research. The capacity of saffron metabolites enables them to bind to biomolecules that protect them against free radical damage (Figure 3).

Ginko Bibloba

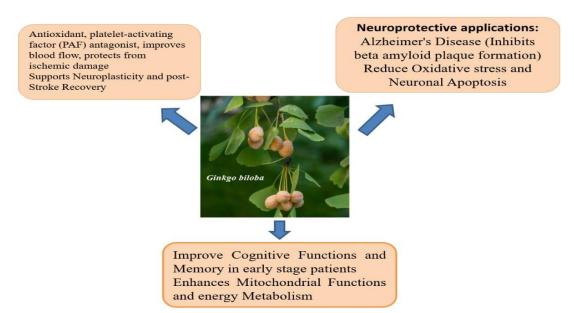


Figure 4. Ginko bibloba and its neuroprotective functions against neurodegenerative diseases

The medical usage of standardised ginkgo extracts contains 6% terpenoids and 24% ginkgo-flavone glycosides. Bilobalide belongs to the group of terpenoids, which also includes ginkgolides A, B, C, M, and J that serve as a group of 20-carbon cage molecules with six 5-membered rings. Ginkgolides function as platelet-activating factor (PAF) antagonists through multiple biological mechanisms. 1 Activating and aggregating platelets, increasing vessel permeability, and causing powerful stomach ulcers and smooth muscle contraction, including bronchial muscle, are additional effects of PAF. Platelet-activating factor directly influences neuronal activity alongside long-term potentiations. Its preventive effects protect brain neurones from peroxidation-induced oxidative damage and reduce both subchronic cold stress effects on receptor desensitisation and neuronal damage from ischemic or electroconvulsive shock situations (Oken, et al., 1998). The antiischaemia and anti-hypoxia effects of Ginkgo support better brain metabolic functions. One of the multiple conditions treated with ginkgo biloba extract includes heart disease, alongside its variant known as coronary heart disease (Figure 4).

Research through meta-analysis confirmed that ginkgo biloba extract leads to improved cognitive performance in patients with

vascular cognitive impairment, although researchers could not determine its effectiveness in the treatment of Alzheimer's Disease patients treatment. Both donepezil medication and ginkgo preparations work toward treating Alzheimer's disease, although they belong to different treatment classes (Li et al., 2023). Medical effects of Ginkgo result from two main active metabolite groups, which include Ginkgo flavones (with quercetin, kaempferol, and iso-rhamnetin glycosides as the principal flavonoids plus biflavonoids ginkgetin/isoginkgetin) and terpene lactones (ginkgolides A, B, C, and J and bilobalide). Pharmacologically active standardized ginkgo leaf extracts are commercially available in the market. The medicinal effects of ginkgo leaf extracts depend on the combined antioxidant capabilities of ginkgolides and the ability of flavonoids and terpenoids to scavenge radicals and suppress platelet-activating factors, and improve blood flow while decreasing platelet aggregation. Scientific evidence suggests that flavonoids protect against the damaging effects of oxidation on lipid membrane structure (Bosch-Morell et al., 2020).

Curcuma Longa



Figure 5. Curcuma longa and its neuroprotective functions against neurodegenerative diseases

Traditional Asian communities have been using curcumin as both food and a medicinal substance since ancient times. Research has proven that curcumin demonstrates three beneficial properties in different studies, which include anticancer effects and antiinflammatory qualities, together with antioxidant properties. Curcumin stands as the most promising treatment material being considered to develop AD therapies because this ancient Indian plant is commonly used in India for curry powder preparation. Modern and Indian medical research demonstrates that this substance effectively addresses a broad spectrum of diseases, including arthritis, liver diseases, colon cancer, breast cancer, haemorrhoids, gastric ulcers, atherosclerosis, and cystic fibrosis. Nerve cell inflammation proves to be an essential cause of Alzheimer's disease, according to recent medical research (Mishra et al., 2008). Medical scientists view curcumin from Indian curry spices as one of the best candidates to create effective treatments for Alzheimer's disease. Studies both in traditional and contemporary medical contexts show extensive usage of curcumin as an ancient Indian herb to treat several diseases, such as arthritis and conditions involving liver diseases and cancer, along with breast cancer, haemorrhoids, gastric ulcers, atherosclerosis, and cystic fibrosis. Proof shows that chronic nerve cell inflammation functions as a leading cause in the development of Alzheimer's disease.

Medical research studies have examined how *curcumin* affects cancer because scientists have confirmed its cancer-influencing properties in tumors. Researchers present evidence that *curcumin* has the potential to treat the development of Alzheimer's disease (AD). Research reveals that curcumin inhibits the activity of $A\beta$ aggregation together with $A\beta$ -induced inflammation and β -secretase, as well as acetylcholinesterase, in in vitro tests. The administration of *curcumin* through the mouth resulted in positive behavioral results during in vivo experiments and inhibited brain

tau phosphorylation and Aß oligomeric aggregation, as well as reduced Aβ brain deposition in AD patients (Hamaguchi et al., 2010). During amyloid-β (Aβ) peptide accumulation, the brain experiences associated inflammatory changes, which cause microgliosis and astrocytosis and lead to the formation of proinflammatory substances. Evidence shows that individuals who regularly take several nonsteroidal anti-inflammatory drugs, including ibuprofen, develop decreased risks of AD symptomatic presentation. Over an extended period of time, NSAIDs have poisonous effects on three essential body organs: the kidneys, liver, and gastrointestinal tract. Therapeutic results from curcumin consumption toward inflammatory processes are robust. Different anti-inflammatory qualities of the treatment make it potentially advantageous for AD management. Adherence of Egr-1 protein and DNA-binding functionalities to THP-1 monocytic cells remains decreased when exposed to curcumin combined with Aβ. Research shows that the amyloid peptide triggers monocyte cytochemokine gene expression through the protein Egr-1. Curcumin prevents the DNA-binding ability of the Egr-1 protein (Figure 5).

Plant-based Interventions in Parkinson's Diseases

Mucuna Pruriens

M. pruriens exists as a climbing legume that originates from eastern India and southern China. Scientists note that various nations have relied on M. pruriens as a food source that ethnic groups have employed historically. Members of distinct ethnic groups across different nations use M. pruriens as a medicinal and nutraceutical product to treat cancer and PD, as well as diarrhoea and helminthiasis, in addition to ulcer management and treatment of infertility, elephantiasis, snake and scorpion stings, and malaria. The plant gains its high nutritional value from its supply of amino acids, which contain arginine, serine, lysine, histidine,

phenylalanine, proline, tyrosine, threonine, and tryptophan. This food source contains an ample variety of minerals, along with fiber components, both at high levels and fatty acids and vitamins. Scientific studies have established the biological strength of L-3,4-dihydroxyphenylalanine (L-DOPA) and two other bioactive components, named ursolic acid and betulinic acid, from the plant. Several other compounds in the plant consist of (Z, E)-7,11-hexadecadien-1-yl acetate, n-hexadecanoic acid (palmitic acid), 9,12-octadecadienoic acid, octadecanoic acid, hexadecanoic acid, 2-hydroxy-1-(palmitoylglycerol), and 2-methyl butanal. Different animal research models confirm how certain bioactive substances generate antidepressant results.

Studies demonstrate that decreased dopamine (DA) signaling throughout different brain locations forming mesocortical and mesolimbic pathways keeps a close relationship to physiological processes which bring about depressive behavior. The plant contains L-3,4-dihydroxyphenylalanine (L-DOPA) and two other vital components, which are ursolic acid and betulinic acid; scientific studies prove their powerful biological effects. The plant contains several substances, including (Z E)-7 11-hexadecadien 1-yl acetate, n-hexadecanoic acid (palmitic acid), 9 12-octadecadienoic acid, octadecanoic acid, and hexadecanoic acid 2-

hydroxy-1-(palmitoylglycerol) and 2-methyl butanal. Scientists have demonstrated specific bioactive chemicals to have antidepressant properties using different animal study models. Scientific investigations show that depressive behavior develops alongside a decrease in dopamine signaling throughout the brain regions which form the mesocortical and mesolimbic pathways (Mata-Bermudez et al., 2024). Scientists have discovered both neuroprotective properties and supportive effects for levodopa anti-PD actions in the individual components of Mucuna seeds such as genistein and gallic acid along with unsaturated acids and nicotine and bufotenin and harmin alkaloids and lecithin. The paper details various components present in Mucuna pruriens seeds regarding their medical potential for Parkinson's disease treatment. A standardized Mucuna seed extract holds great promise to reduce medical expenses while minimizing PD progression because the current treatment combination of levodopa with other medications is very expensive. Mucuna seeds exist in the market at favorable prices as well as easy accessibility. Mucuna pruriens provides effective treatment for Parkinson's disease according to the review due to Ayurveda's full treatment methodology (Kasture et al., 2013).

Withania Somnifera



Figure 6: Withania somnifera and its neuroprotective functions against neurodegenerative diseases

Historically, Ayurveda has adopted ashwagandha, which has the botanical name Withania somnifera (L.) Dunal provides various medical advantages. A wide range of active components in this plant show their anti-inflammatory, anti-diabetic, anticancer, antimicrobial, and neurotherapeutic effects through research involving chemically-treated mice in lab experiments. Researchers use genetically modified Drosophila models to study genetic pathways alongside cellular events happening in situ for various neurodegenerative diseases, including Parkinson's and Alzheimer's. The development of drugs in vitro environments fails to produce effective treatments in clinical trials due to variations in

human genetics and physiological conditions (Murthy *et al.*, 2024) (**Figure 6, Table 1**).

According to Vrindamadhava from 900 AD, this marks the first Ayurvedic treatise to present ashwagandha as medication against diverse neurological diseases, such as Medhya for memory improvement, Kampavatha for Parkinson's patients, and chinta for anxiety, and Nidranasa for sleep disturbances, etc. Studies have shown that different regions of ashwagandha contain over 50 chemical components, and its main biological action originates from withanolide lactones and steroidal alkaloids, which compose

the various pathological symptoms of Parkinson's disease. The combination of ubisol-Q10 with ethanolic ashwagandha extract (ASH) represents two simple, harmless nutraceutical compounds that provide supportive treatment for the multiple biochemical causes found in Parkinson's disease. The combined action of these medications provided better results in medical treatment efforts to stop the progressive neurodegenerative process of Parkinson's disease compared to using them separately. Our research utilized

lower doses of the known neuroprotectant ASH by combining separate drugs that target distinct Parkinson's disease pathologies, rather than previous studies about ASH use alone. The strategies addressed through Ubisol-Q10 treatment did not reach all possible PD mechanisms, but the therapy implemented targeted vital mechanisms. Parkinson's disease exists as a multi-process disease, so neurodegeneration stoppage demands intervention in numerous processes simultaneously (Vegh *et al.*, 2021).

Table 1. Neuroprotective Medicinal Plants and Their Mechanisms of Action in Neurodegenerative Diseases

Medicinal Plant	Scientific Name	Neurodegenerative Target	Key Bioactive Compounds	Mechanisms of Neuroprotection
Turmeric	Curcuma longa	Alzheimer's Disease	Curcumin	Antioxidant, anti-inflammatory, inhibits $A\beta$ aggregation and acetylcholinesterase, reduces tau phosphorylation.
Brahmi	Bacopa monnieri	Alzheimer's Disease	Bacosides	Enhances synaptic transmission, antioxidant effects, improves memory and cognition.
Ashwagandha	Withania somnifera	Parkinson's Disease	Withanolides, steroidal alkaloids	Antioxidant, anti-apoptotic, improves dopaminergic neuron survival, reduces oxidative stress.
Velvet Bean	Mucuna pruriens	Parkinson's Disease	Levodopa (L-DOPA), ursolic acid, betulinic acid	Natural dopamine source, antioxidant, supports neurotransmission, and has antidepressant effects.
Ginkgo	Ginkgo biloba	Alzheimer's & Vascular Dementia	Ginkgolides, bilobalide, flavone glycosides	Antioxidant, platelet-activating factor (PAF) antagonist, improves blood flow, protects from ischemic damage.
Gotu Kola	Centella asiatica	Cognitive Decline	Asiaticoside, madecassoside	Neuroregenerative, antioxidant, enhances synaptic plasticity and cognitive performance.
Goji Berry	Lycium chinense	Aging-related degeneration	Polysaccharides, carotenoids	Anti-aging reduces neuronal damage.

Applications

Neurodegenerative Disorders and The Role of Medicinal Herbs

A distinct feature of the neurodegenerative diseases Parkinson's disease and Alzheimer's disease includes progressive deterioration of neurons that leads to their ultimate death. Many neurodegenerative disorders have origins that healthcare professionals are unable to establish correctly. Neurodegenerative disorders are mainly caused by six different classes of environmental influences, including environmental variables, mitochondrial abnormalities, oxidative stress, inflammation, protein degradation, genetic factors, and protein accumulation in neurones. Neurodegenerative illnesses tend to advance significantly with age as a determining factor. Medical research has employed wolfberry together with ginkgo biloba, resveratrol, and ginseng alongside curcumin, Baccopa monnieri, and Withania somnifera (ashwagandha) as medicinal plants for treating and preventing neurological diseases and their symptoms. When properly processed, natural substances retain their original activity levels suitable for use as therapeutic nanoparticles.

The specific delivery of cells and tissues becomes possible through nanodrug delivery systems, which increase drug bioavailability. A drug delivery system using nanoparticles together with polymeric nanomicelles, as well as complex polymer nanocrystals and nanofibres, transports medicinal plants for neurological illness

treatments. The preventive power of therapeutic herbs operates against neurodegenerative illnesses. These diseases are known as neurodegenerative diseases because they lead to significant neuron reduction along physical and functional lines. People nowadays prefer non-traditional treatment options because various adverse effects increase from contemporary medical approaches combined with disease progression. Medicinal plants demonstrate various cellular and molecular pathways that facilitate their advantageous use in these medical situations. Several neuroprotective capabilities occur in traditional plants, which work by reducing inflammation while improving antioxidative mechanisms and inhibiting the pro-inflammatory functions of tumor cytokines.

The protection of plants from diseases depends mainly on different transcription and transduction pathways (Ratheesh et al., 2017). The healthcare systems around the world face immense costs from the three important neurodegenerative diseases called Alzheimer's disease, Parkinson's disease, and Huntington's disease. These diseases will become more severe when life expectancies continue to increase during the upcoming decades. Medical science lacks any treatment that can cure these illnesses despite offering symptomatic treatments through prescription medications. Medical research proves that oxidative stress functions as either the source or product of other downstream factors leading to neurodegeneration in patients. Proof exists that the application of antioxidants to fight oxidative cellular stress might serve as a

potential therapy for neurological diseases in the nervous system. Scientists have dedicated their research to studying natural antioxidants for their potential as oxidative stress fighters over these past ten years. The outcomes from clinical trials did not lead to effective treatments for neurological issues. Studies on natural extracts for nerve disease prevention or treatment after scientists found these substances have antioxidant properties, along with other helpful biological attributes, demonstrate their worth. The review encompasses expert analysis of both clinical trials and in vitro, along with in vivo investigations, together with the analysis of natural antioxidant potential (Ratheesh *et al.*, 2017).

Limitations

Neurodegenerative disorders (NDDs) represent multiple diseases that damage neurons to produce mental and motor impairment. Many experts now study NDDs because of their complex origin factors, together with multifaceted symptoms, which result in significant social and economic consequences. The observed cell death of brain cells mainly stems from metabolic alterations, which lead to elevated oxidase enzyme activity, together with increased free radicals, mitochondrial dysfunctions, and neuroinflammation. The detection of numerous NDD issues from their initial stages through development has improved through modern diagnostic procedures. Research in NDD focuses on enhancing cellular defense mechanisms and especially on oxidoreductases because scientists aim to discover effective treatments (Zaky et al., 2025).

Challenges and Future Perspective

The application spectrum of plant-derived natural compounds and plant extracts with antioxidant properties extends to in vitro and in vivo studies. The increasing lifespan of people will lead to escalating health problems from these diseases in the future decades. The association between neurodegeneration and oxidative stress exists as both a result and an underlying cause of different pathological events. Medical experts recommend that antioxidants might function as remedies for neurological diseases that help reduce oxidative stress inside nerve cells. The application spectrum of plant-derived natural compounds and plant extracts with antioxidant properties extends to in vitro and in vivo studies. The increasing lifespan of people will lead to escalating health problems from these diseases in the future decades. The association between neurodegeneration and oxidative stress exists as both a result and an underlying cause of different pathological events. Medical experts recommend that antioxidants might function as remedies for neurological diseases that help reduce oxidative stress inside nerve cells. The application of nanotechnology represents a key approach to handling multiple limitations in medical conditions, whereas neurodegenerative disorders stand among the set of diseases it avoids. Nanotechnology represents an important technology that has transformed human life in multiple directions. Multiple nanostructures that include Carbon nanotubes (CNTs) with polymer and lipid nanoparticles and nanoliposomes and nanomicelles combined with vehicle systems based on lactoferrin alongside polylactic-co-glycollic acid and polybutyleyanoacrylate improve medication PK, stability, and drug properties and toxicity profiles. NDs join the millions of people who experience numerous

debilitating diseases. The power of nanotechnology assists medical science in breaking through barriers that exist in numerous disease treatments, especially those affecting neurodegenerative disorders (NDs). This progressive strategy has modified human existence through multiple revolutionary changes. A combination of carbon nanotubes (CNTs), polymer nanoparticles, lipid nanoparticles, nanoliposomes, and nano-micelles with vehicle systems that include lactoferrin, polylactic-co-glycollic acid, and polybutylcyanoacrylate improves the therapeutic qualities of many medications. The medical condition experienced by NDs affects millions of people with multiple chronic debilitating diseases (Ratheesh *et al.*, 2017; Moradi *et al.*, 2020; Puri *et al.*, 2022).

Conclusion

underlying factors contributing neurodegenerative diseases remain largely unknown, which has led to an ongoing exploration of herbal medicine as a viable treatment option. Phytochemicals derived from medicinal plants exhibit encouraging therapeutic properties, primarily due to their capacity to mitigate inflammation, counteract oxidative stress, and inhibit acetylcholinesterase activity—critical elements in the process of neurodegeneration. Conditions such as Alzheimer's disease (AD), Parkinson's disease (PD), and Huntington's disease exhibit shared cellular and molecular pathways, including inflammatory damage, necroptosis, and apoptosis. Nevertheless, the practical use of herbal medicine is frequently hindered by inadequate pharmacokinetic characteristics. Recent innovations in drug delivery systems, especially nanoencapsulation, have enhanced the accuracy and effectiveness of natural compounds by improving target specificity, minimizing toxicity, and decreasing required dosages. New research into electrospinning and electrospraying technologies is broadening the development of fibers and nanoparticles aimed at treating age-related neurodegenerative conditions. Traditional Persian medicinal plants, abundant in bioactive phytochemicals, are among those being studied for their potential to augment standard therapies. these compounds demonstrate neuroprotective potential, additional clinical investigations are necessary to validate their safety and efficacy. In summary, medicinal plants offer a valuable and diverse strategy for addressing neurodegenerative diseases, providing natural alternatives with reduced side effects and the ability to influence multiple disease pathways.

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