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A Review on the Neurotherapeutic Potential of Plant-derived Phytochemicals

Lata Sati[1*] and Bijendra Singh[2]

[1]Department of Home Science, Shri Guru Ram Rai University, Dehradun - 248001, Uttarakhand, India; latasati90@gmail.com

[2]Department of Yogic Science, Shri Guru Ram Rai University, Dehradun - 248001, Uttarakhand, India

Abstract

Background: Neurological disorders like depression, Parkinson's Disease (PD), Alzheimer's Disease (AD), and epilepsy are a major threat to global health because of their complicated causes and lack of treatments. In recent years, there has been a lot of interest in the neuroprotective and therapeutic potential of bioactive substances—phytochemicals—derived from medicinal plants. **Aim:** To study the neurotherapeutic potential of plant-derived phytochemicals in the prevention and management of neurological and neuropsychiatric disorders, with particular emphasis on their antioxidant, anti-inflammatory, neuroprotective, anti-apoptotic, mitochondrial protective, and neurotransmitter-modulating activities. **Method:** The study was conducted to review research on plant-derived phytochemicals and their neurotherapeutic effects. The available evidence concerning medicinal plants, their major bioactive compounds, mechanisms of action, experimental models, concentrations or doses, and therapeutic outcomes was examined and organised according to major neurological disorders. **Result:** An extensive review of various phytochemicals, such as flavonoids, alkaloids, terpenoids, phenolic acids, and saponins, for the prevention and treatment of neurological disorders is presented in this study. Even with encouraging results, issues including low bioavailability, little clinical validation, and standardisation of herbal formulations continue to be major obstacles. **Conclusion:** To maximise the clinical relevance of phytochemicals in neurology, future research should concentrate on integrated therapy methods and nanotechnology-based delivery systems.

Major Finding: The review highlights the significant anti-inflammatory, neuroprotective, and antioxidant properties of phytochemicals derived from plants. These bioactive compounds show a lot of promise in the treatment of neurological diseases such as Parkinson's, Alzheimer's, and Huntington's. Because of their multi-targeted mechanisms and natural nature, phytochemicals may be safer and more effective alternatives or supplements to conventional medical techniques.

Keywords: Future Prospective, Management, Phytochemicals, Neurological Disorders, Treatment

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1. Introduction

Among the most complicated medical issues affecting people are neurological illnesses. These conditions cover a broad range of illnesses, including depression, Parkinson's, Alzheimer's, Huntington's, multiple sclerosis, and epilepsy. When combined, they significantly raise global rates of illness, disability, and mortality[1]. According to estimates from the World Health Organisation (WHO), neurological disorders account for approximately 6.3% of the global disease burden and are predicted to increase as the population ages[2]. The present synthetic medications frequently merely provide symptomatic alleviation rather than disease modification, notwithstanding advancements in contemporary neuropharmacology[3]. Furthermore, these medications are often linked to negative side effects, exorbitant prices, and restricted availability in underdeveloped nations. As a result, phytochemicals—natural, plant-based substances

*Author for correspondence

have garnered new interest worldwide as possible alternatives or supplemental therapies for neurological disorders. Phytochemicals are naturally occurring bioactive substances that give plants their colour, flavour, and defence mechanisms against pathogens[4-9]. These substances have a variety of pharmacological properties, including antioxidant, anti-inflammatory, neuroprotective, and anti-apoptotic effects, according to a growing body of research conducted over the past several decades. Major types of phytochemicals, such as flavonoids, alkaloids, terpenoids, saponins, and phenolic acids, have demonstrated significant therapeutic promise for the treatment of neurological disorders[10,11]. Phytochemicals have a variety of effects, concurrently impacting several biochemical cascades implicated in neurodegeneration, in contrast to manufactured medicines that usually target specific molecules or pathways. The multifactorial character of neurological conditions, where oxidative damage, inflammation, dysfunction of the mitochondria or neurotransmitter imbalance frequently occur, is ideally suited to this multi-targeted strategy. One of the fundamental mechanisms underlying the neuroprotective effects of phytochemicals is their ability to combat oxidative stress behind their neuroprotective benefits. The human brain is especially susceptible because of its high oxygen consumption and lipid content; it produces Reactive Oxygen Species (ROS) and Reactive Nitrogen Species (RNS). Excessive oxidative stress leads to lipid peroxidation, protein oxidation, DNA damage, and ultimately neuronal death. Strong free radical scavenging abilities have been shown by phytochemicals like resveratrol (found in grapes), quercetin (found in onions and apples), and curcumin (found in *Curcuma longa*), which shield neurons from oxidative damage. Additionally, by upregulating Natural antioxidant defence systems are triggered by enzymes such as glutathione peroxidase, catalase, and Superoxide Dismutase (SOD)[12]. Furthermore, inflammation plays a significant role in the emergence of neurological disorders.

Neuronal dysfunction and degeneration are caused by Microglial activation and increased production of pro-inflammatory cytokines like TNF-α, IL-1β, and IL-6 are characteristics of chronic neuroinflammation. It has been demonstrated that phytochemicals can decrease inflammation and cytokine production by modulating inflammatory signalling pathways such as NF- κB, COX-2, and iNOS. For instance, the anti-inflammatory qualities of withanolides from *Withania somnifera* (*Ashwagandha*) and ginsenosides from *Panax ginseng* improve neuronal survival and decrease neuroinflammation. These findings suggest that by lowering oxidative damage and reducing inflammation, phytochemicals offer a dual mechanism for neuroprotection[13-16].

The accumulation of hyperphosphorylated tau protein and amyloid-β plaques, which impair synaptic transmission and cause neuronal death, are two of the main characteristics of AD. It has been discovered that polyphenolic substances like resveratrol and Epigallocatechin Gallate (EGCG) from green tea prevent tau phosphorylation and amyloid formation. Curcumin has also been shown to bind amyloid plaques and aid in their removal, which may delay the course of the illness. Unlike the majority of traditional medications that just treat symptoms, our results show that phytochemicals have the ability to change the course of illness.

Another significant factor in neuronal degeneration is mitochondrial malfunction, which results in poor energy metabolism and elevated ROS production. It has been demonstrated that a number of phytochemicals maintain the integrity and functionality of mitochondria. For instance, ginsenosides protect dopaminergic neurons from toxin-induced mitochondrial damage and promote mitochondrial biogenesis. Similar to this, resveratrol and baicalin (from *Scutellaria baicalensis*) stimulate signalling pathways including AMPK and SIRT1, which control cellular stress responses and energy consumption. These substances increase neuronal resilience and postpone the beginning of neurodegenerative alterations by enhancing mitochondrial activity. The clinical translation of phytochemicals is still difficult, despite the encouraging results. Numerous phytochemicals have low stability, rapid metabolism, poor bioavailability, and restricted absorption, all of which limit their medicinal value. For instance, curcumin has poor systemic availability when taken orally while being well-known for its strong pharmacological effects. By improving the solubility, stability, and targeted transport of phytochemicals to the brain, recent developments in nanotechnology and drug delivery systems, such as liposomes, nanoparticles, and phytosomes, have demonstrated promise in overcoming these constraints. Furthermore, through complementary

modes of action, synergistic formulations that include synthetic and herbal substances or combine many phytochemicals may provide increased efficacy. From a pharmacological standpoint, phytochemicals' ability to target various pathways offers an advantage for treating complicated neurological illnesses. However, standardisation, quality assurance, and regulatory approval are all hampered by this same complexity. It is challenging to obtain consistent therapeutic results because of the heterogeneity in phytochemical content caused by variations in plant source, growth circumstances, extraction processes, and processing methods. In order to include phytochemicals into conventional neurotherapeutics, standardised extraction techniques, dosage optimisation, and thorough clinical validation are essential[17,18].

2. Alzheimer Disease (AD)

Alzheimer's Disease is the most common cause of dementia, accounting for 60-70% of cases globally. It is characterised by progressive memory loss, cognitive decline, and abnormal behaviour. Oxidative stress, neuroinflammation, intracellular neurofibrillary tangles composed of hyperphosphorylated tau protein, and extracellular accumulation of amyloid-β (Aβ) plaques are the primary pathogenic features.

Curcumin, resveratrol, and EGCG are examples of phytochemicals that have shown impressive neuroprotective properties against AD. Curcumin inhibits tau aggregation, lowers the formation of amyloid plaques, and controls inflammatory cytokines. By activating the SIRT1 pathway, resveratrol improves neuronal survival and mitochondrial function. Green tea polyphenol EGCG shields neurons from oxidative stress and stops amyloid aggregation[19]. These results imply that phytochemicals can provide a multimodal treatment approach by acting on several sites implicated in the pathophysiology of AD (Table 1).

3. Parkinson's Disease (PD)

A degenerative neurological condition linked to ageing is PD. PD is brought on by a loss of nerve cells in the

Table 1. Anti-Alzheimer's activities of medicinal plants and their active substance

Medicinal plant	Active substance	AD prevention mechanism and activity	References
Turmeric or *Curcuma longa*	Curcumin	Decreases tau hyperphosphorylation, inhibits amyloid-β aggregation, has anti-inflammatory and antioxidant properties, chelates metals, and enhances memory.	[20]
Brahmi or *Bacopa monnieri*	Bacosides A and B	Enhances synaptic plasticity, inhibits acetylcholinesterase (AChE), reduces amyloid burden, improves learning and memory.	[21]
Ashwagandha, or *Withania somnifera*	Withanolides	Enhances cholinergic transmission, lowers oxidative stress, promotes amyloid-β clearance, and restores synaptic function.	[22]
Ginkgo Biloba	Ginkgolides, Bilobalide	Enhances cognition, increases cerebral blood flow, inhibits amyloid accumulation, and has anti-apoptotic and antioxidant properties.	[23]
Panax ginseng	Rb1, Rg1, and Rg3 ginsenosides	Lowers amyloid buildup, improves memory and neurogenesis, and shields neurons from oxidative damage.	[24]
Centella asiatica (Gotu Kola)	Madecassoside and Asiaticoside	Increases synaptic density, lowers amyloid toxicity, inhibits AChE, and strengthens spatial memory.	[25]
Salvia lavandulaefolia (sage) and *Salvia officinalis*	Carnosic acid with rosmarinic acid	Inhibition of cholinesterase lowers lipid peroxidation and enhances memory and attention.	[26]
Green tea, or *Camellia sinensis*	Epigallocatechin Gallate (EGCG)	Decreases oxidative damage and neuroinflammation, inhibits the development of amyloid fibrils, and stimulates the Nrf2/ARE pathway.	[27]
Nigella sativa (Black seed)	Thymoquinone	Modifies the activity of acetylcholinesterase and inhibits oxidative stress and neuroinflammation.	[28]
Zingiber officinale (Ginger)	6-Shogaol, 6-Gingerol	Antioxidant and anti-inflammatory, improving cholinergic transmission and cognitive performance.	[29,30]

substantia nigra, the area of the brain that controls movement. When these nerve cells are damaged or destroyed, dopamine, an essential neurotransmitter, cannot be generated. Studies have shown that individuals who have lost up to 80% of their dopamine-producing cells in the substantia nigra experience Parkinson's symptoms. It often appears between the ages of 40 and 70 and is handled differently from both motor and non-motor disorders. Uncontrollable trembling, abnormal posture, and slowness of movement (bradykinesia) are the main symptoms, along with at least one additional sign of resting tremor or inflexibility[31-33]. Loss of smell, sleep problems, mental disturbances, increased salivation, bowel motions, and excessive periodic limb movement during sleep are other related symptoms. Parkinson's patients frequently experience psychotic episodes, and over 40% of patients suffer from depression. Phytochemicals and herbal medications are becoming more popular as neuroprotective agents for PD due to the limits of traditional therapy[34-36]. These natural substances work through a variety of pathways, including mitochondrial protection, anti-inflammatory actions, prevention of α-synuclein aggregation, and antioxidant activity. *Mucuna pruriens*, also referred to as velvet beans, naturally contains L-dopa, the direct precursor of dopamine. According to studies, its seed extracts offer neuroprotection through antioxidant qualities in addition to improving motor performance. Curcumin, the primary active component of *Curcuma longa*, has been demonstrated to protect mitochondria, inhibit the formation of α-synuclein fibrils, and reduce oxidative stress in mice with PD. Withanolides, which are present in *Withania somnifera* (*Ashwagandha*), have anti-inflammatory and anti-apoptotic qualities that protect dopaminergic neurons from MPTP and rotenone damage. Green tea polyphenols, particularly EGCG, prevent oxidative damage and dopaminergic cell death. It has been demonstrated that *ginseng*, which is high in ginsenosides, increases dopamine production, improves neuronal survival, and reduces neuroinflammation[37-40]. These herbal remedies are potential supplementary therapies since they not only reduce motor symptoms but also address the underlying pathogenic causes of PD (Table 2).

4. Anxiety and Depression

Anxiety and depression, two of the most prevalent neuropsychiatric disorders worldwide, significantly affect mood, cognition, and overall quality of life[47,48]. Environmental, psychological, physiological, and genetic factors are all considered to be components of these complex disorders. According to estimates from the World Health Organisation, over 280 million people worldwide suffer from depression, and over 301 million suffer from anxiety

Table 2. Compounds in PD which are studied for experimental purposes

Plant	Model used	Concentration	Impact outcome	References
Mucuna pruriens, or velvet bean	Rat model induced by 6-hydroxydopamine (6-OHDA)	100 mg/kg oral extract per day	Enhanced locomotor activity, decreased oxidative stress, and elevated striatal dopamine levels.	41
Ashwagandha, or *Withania somnifera*	Rat model induced by rotenone.	200 mg/kg ethanolic extract daily	Enhanced motor performance, decreased lipid peroxidation, and restored tyrosine hydroxylase expression	42
Curcuma longa (turmeric)	SH-SY5Y neuronal cell line treated with MPP	Curcumin at 10 μM	Decreased ROS, increased mitochondrial membrane potential, and inhibited α-synuclein aggregation.	43
Panax ginseng	Mice with 6-OHDA lesions	Ginsenosides at 50 mg/kg	Reduced apoptosis, enhanced motor coordination, and decreased loss of dopaminergic neurons	44
Ginkgo biloba	Rat model induced by rotenone.	EGb 761 extract at 100 mg/kg	Decreased microglial activation, increased dopamine, and enhanced mitochondrial activity.	45
Brahmi or *Bacopa monnieri*	Rats given rotenone	Hydroalcoholic extract at 40 mg/kg	Increased dopamine turnover, less oxidative damage, and improved cognitive function	46

disorders. These conditions often coexist and share pathophysiological mechanisms, such as dysregulation of the Hypothalamic-Pituitary-Adrenal (HPA) axis, oxidative stress, neurotransmitter imbalance, and neuroinflammation[49,50]. Benzodiazepines, Selective Serotonin Reuptake Inhibitors (SSRIs), and Serotonin-Norepinephrine Reuptake Inhibitors (SNRIs) are examples of conventional pharmacological therapies that have shown promise but are constrained by side effects, delayed onset, and dependency potential[51-53]. Because they target many biochemical pathways with fewer adverse effects, phytochemicals and herbal treatments have emerged as viable additional or alternative therapies for controlling anxiety and depression[54]. The most widely accepted explanation for depression and anxiety is deficiencies in monoamine neurotransmitters, such as Dopamine (DA), Norepinephrine (NE), and Serotonin (5-HT). These neurotransmitters control arousal, motivation, and mood. Mood disorders and anxiety symptoms are caused by decreased transmission of dopamine and serotonin in the limbic system and prefrontal cortex. One of the main causes of anxiety and depression is ongoing stress. However, several internal and external factors are responsible for stress and its level of severity. Excessive cortisol production brought on by prolonged HPA axis activation causes neuronal atrophy in the prefrontal cortex and hippocampus, regions crucial for emotion regulation[55,56].

Recent studies have shown that inflammatory cytokines, such as TNF-α, IL-1β, and IL-6, can alter neurotransmission and lead to depression and anxiety[63,64]. Oxidative and nitrosative stress, which results in neuronal death and reduced neurogenesis, aggravates these conditions even more. Neuronal development, survival, and synaptic plasticity are all hampered by decreased production of Brain-Derived Neurotrophic Factor (BDNF). Patients with significant depression and anxiety have been found to have low BDNF levels, suggesting that increasing BDNF expression may be a therapeutic target. Despite their effectiveness, many medications have drawbacks, including weight gain, nausea, sleeplessness, sexual dysfunction, and withdrawal symptoms. Furthermore, according to some studies, between 30 and 40 per cent of patients

Table 3. The active ingredients in herbal medicines, their mode of action, and their possible uses in treating anxiety and depression

Herbal medicine	Active compounds	Mechanism of action	Potential applications	References
Hypericum perforatum, also known as St. John's Wort.	Hypericin and Hyperforin	Has antioxidant and anti-inflammatory qualities and prevents serotonin, dopamine, and norepinephrine from being reabsorbed.	Mood enhancement, mild-to-moderate depression treatment	57
Borage, or *Borago officinalis*	Thesinine with rosmarinic acid	Demonstrates anxiolytic effects in animal studies; the precise The mechanism of antidepressants is yet unknown.	Anxiety and depression	58
Lavender, or *Lavandula angustifolia*	Linalyl acetate and linalool	Modifies GABAergic neurotransmission and has sedative and anxiolytic effects in animal models.	Somatic tension, anxiety, and depression	59
Korean *ginseng*, or *Panax ginseng*	Rb1 and Rg1 ginsenosides	Alters the hypothalamic-pituitary-adrenal (HPA) axis, inhibits nitric oxide synthase, and regulates monoamine neurotransmitters.	fatigue, melancholy, cognitive impairment, depression, and anxiety	60
Saffron, or *Crocus sativus*	Safrana and Crocin	Acts as a GABA-α agonist and NMDA receptor antagonist, preventing serotonin, dopamine, and norepinephrine from being reabsorbed	Anxiety and depression	61
Roseroot, or *Rhodiola rosea*	Salidroside, Rosavin, and Tyrosol	Modifies monoamine levels, inhibits monoamine oxidase A, and affects neuroendocrine function	Anxiety, despair, fatigue and cognitive decline	62

respond only partially or not at all to antidepressant medication[65,66]. Research on herbal treatments and phytochemicals with neuroprotective and mood-enhancing properties has increased as a result of these disadvantages. Standardisation, bioavailability improvement, and long-term safety assessment are still necessary even though many herbal medicines show promise in preclinical and clinical research. Phytochemicals' stability and brain penetration can be enhanced by combining them with nanocarrier systems. Additionally, integrative methods that incorporate yoga, meditation, and herbal supplements have demonstrated beneficial impacts on mental health and stress reduction (Table 3).

5. Huntington's disease

Huntington's Disease (HD) is a hereditary neurodegenerative condition characterised by a progressive loss of motor coordination, cognitive function, and psychological stability. An expansion of the CAG trinucleotide repeat in the Huntingtin (HTT) gene results in a mutant Huntingtin protein (mHTT) that aggregates in neuronal cells. The disease primarily affects neurons in the striatum and cerebral cortex, significantly impairing movement, emotion regulation, and cognition. The pathogenic processes underlying HD include oxidative stress, mitochondrial dysfunction, glutamate excitotoxicity, neuroinflammation, and transcriptional activity dysregulation[67,68]. Several phytochemicals and herbal compounds have shown promising neuroprotective and anti-apoptotic potential in experimental HD models, despite the fact that there is presently no cure for HD[69].

Phytochemicals have beneficial effects by scavenging free radicals, modifying neurotransmitters, enhancing mitochondrial function, and averting neuronal death. Traditional medicinal herbs like *B. monnieri*, *C. longa*, and *W. somnifera* have been evaluated for their ability to prevent neuronal degeneration in HD models. These herbal preparations' bioactive ingredients support synaptic plasticity and neurogenesis, which improves cognitive and motor function (Table 4).

6. Multiple Sclerosis

Multiple Sclerosis (MS), the most common neurological illness, is an autoimmune-mediated ailment of the Central Nervous System (CNS) that often results in neurological issues and considerable physical or mental handicap in young people155. The

Table 4. Mechanisms of Action (MOA), plant sources, bioactive compounds, and experimental models

Compound	Source of the plant	Model	Mechanism of Action (MOA)	References
α-Mangostin	*Garcinia Mangostana* L.	3-NP model *in vivo*; CGNs (Cerebellar Granule Neurons) *in vitro*	Demonstrates significant antioxidant action and reduces the production of ROS.	70
Celastrol	Celastrol	HeLa, PC12, and polyQ mutant cell models *in vitro*	Modifies Heat Shock Proteins (HSPs) and has anti-aggregation and cytoprotective properties.	71
Fisetin	Present in a range of fruits and vegetables	R6/2 HD mice *in vivo*; PC12 cells expressing mutant Htt-exon1 *in vitro*	Increases the activation of the ERK pathway and decreases the aggregation of Mutant Huntingtin (MHTT).	72
Galantamine	Species of *Galanthus*	3-NP-induced rat model *in vivo*	Decreases striatal lesions, increases neuronal survival and modifies nicotinic acetylcholine receptors (nAChRs).	73
Ginsenosides	*Panax ginseng* Meyer, C.A.	YAC128 HD mouse model *in vivo*; striatal MSN cultures *in vitro*	Reduces glutamate-induced Ca2+ influx; anti-excitotoxic and neuroprotective	74
Hesperidin	Fruits with citrus	3-NP-induced rat model *in vivo*	Restores cortical-striatal and hippocampal MDA levels, has anti-inflammatory and antioxidant properties and enhances locomotor and PPI responses.	75

primary causes of the myelin sheath's destruction include many areas of inflammation caused by oligodendrocyte death, macrophage penetration, and localised T-lymphocyte[76]. These factors cause inflammatory cells and their waste products, as well as demyelinated and transected axons and astrocytes, to form CNS plaques in both white and grey matter[77]. Neurological issues, including autonomic and sensory inadequacies, visual abnormalities, ataxia, exhaustion, cognitive impairments, and emotional disorders, may arise from these lesions' interference with the normal transmission of nerve impulses. Multiple sclerosis is a complex aetiology that includes genetic vulnerability, abnormal immunological responses, and environmental factors. It is well acknowledged that aberrant immunological activation—especially by auto-reactive T cells—is essential for starting and sustaining the inflammatory cascade inside the central nervous system, even though the precise cause is unknown. Cytokines are generated when immune cells cross the Blood-Brain Barrier (BBB) as a result of immunological dysregulation[78]. These cytokines exacerbate oxidative stress, inflammation, and the degradation of neurons and myelin sheaths[79]. Current MS treatments mostly focus on immune responses to lower inflammation and slow the progression of the disease. Symptomatic drugs, acute recurrence treatments, rehabilitative therapies, lifestyle modifications, and Disease-Modifying Therapies (DMTs) are among the current therapies that have shown efficacy in lowering relapses and

Delaying the onset of disability[80]. However, their favourable safety features and low performance in advanced forms of MS highlight the need for alternative therapy approaches. As a result, during the past several years, numerous investigations on the use of medications derived from natural sources have been carried out. Consequently, we have determined that the following medications can aid in the treatment of this illness (Table 5).

A review of experimental studies showed positive results in several neurological diseases. However, more in-depth biotechnological studies are required, especially in the medical field. There is a further need to study side effects associated with these phytochemicals in the body or neurological processes.

Table 5. Plant compounds and their effects in the management of multiple sclerosis

Compound	Concertation	Study	Biological Properties	References
Cannabis sativa	For ten weeks, use 5-25 mg of C. sativa extract orally every day.	Double-blind, placebo-controlled clinical studies	Showed a notable reduction in muscular stiffness at 4, 8, and 12 weeks as compared to a placebo.	81
Oleanolic acid	50 mg/kg administered intraperitoneally	C57BL/6J mice used in an experimental model of autoimmune Encephalomyelitis (EAE) lowered CNS inflammatory cell	Infiltration, reduced neurological impairment, reduced BBB permeability, altered Th1/ Th2 balance, and shown anti-inflammatory actions.	82
Saffron (*Crocus sativus*)	Gavage administration of ethanol extract for 21 days	C57BL/6 mice in the EAE model: diminished oxidative	Stress and leukocyte infiltration in the central nervous system, enhancing neural defence.	83
Extract from pomegranate peels	Eight weeks of intraperitoneal ethanol extract (100	Studies employing the female DA rat EAE model *in vitro* and *in vivo*	decreased the inflammatory response in GALT cell lines and inhibited the generation of IL-17 in activated T cells.	84
Capparis ovata	The hydroalcoholic extract's butanol fraction	Research conducted *in vitro* with SH-SY5Y cell lines	Demonstrated significant anti-inflammatory effectiveness by inhibiting the transcription of the genes TNF-α, NF-κB1, *GFAP*, *CXCL10*, and *PTPN11*.	85

7. Conclusion

The use of chemicals obtained from plants in medicine has developed as a practical method of treating neurological issues. Several studies have demonstrated that Numerous neurobiological processes, including oxidative stress, neurotransmitter imbalance, and neurological inflammation, can be influenced by phytochemicals. Numerous neurological conditions, including PD and AD, as well as anxiety and depression, are linked to these pathways. Even while conventional pharmacological approaches have advanced significantly, they frequently have unfavourable side effects and might not be able to treat the root causes of neurological issues. Conversely, phytomedicines use a more all-encompassing approach, usually targeting many routes simultaneously. When compared to conventional therapies, this may increase efficacy and decrease toxicity. It is important to remember that the field of herbal treatments is still developing and that more research is required to determine the appropriate dosage, formulation, and safety profiles of different plant-based chemicals. Additionally, it is important to thoroughly investigate the complex interactions that phytochemicals have with other medications or underlying medical conditions. Lastly, a potential supplemental or alternative treatment for neurological disorders is provided by plant-based medicine. By using the therapeutic potential of plants, researchers and medical professionals are trying to identify long-term, safe, and effective treatments for these serious ailments.

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9. References

1. Baba S, Akerele O, Kawaguchi Y, editors. Natural resources and human health: Plants of medicinal and nutritional value. Amsterdam: Elsevier; 1992.
2. Capasso R, Izzo AA, Pinto L, Bifulco T, Vitobello C, Mascolo N. Phytotherapy and quality of herbal medicines. Fitoterapia. 2000; 71(Suppl 1):S58-S65. https://doi.org/10.1016/S0367-326X(00)00173-8 PMid:10930714
3. Farooqui AA. Effect of lifestyle, aging, and phytochemicals on the onset of neurological disorders. In: Phytochemicals, signal transduction, and neurological disorders. Springer; 2013. p. 1-29. https://doi.org/10.1007/978-1-4614-3804-5_1
4. Harvey AL. Medicines from nature: are natural products still relevant to drug discovery? Trends Pharmacol Sci. 1999; 20(5):196-198. https://doi.org/10.1016/S0165-6147(99)01346-2 PMid:10354614
5. Jankovic J. Levodopa strengths and weaknesses. Neurology. 2002; 58 (Suppl 1):S19-S32. https://doi.org/10.1212/WNL.58.suppl_1.S19
6. Kim J, Lee HJ, Lee KW. Naturally occurring phytochemicals for the prevention of Alzheimer's disease. J Neurochem. 2010; 112(6):1415-1430. https://doi.org/10.1111/j.1471-4159.2009.06562.x PMid:20050972
7. Kumar V. Potential medicinal plants for CNS disorders: An overview. Phytother Res. 2006; 20(12):1023-1035. https://doi.org/10.1002/ptr.1970 PMid:16909441
8. Martin D, Rojo AI, Salinas M, Diaz R, Gallardo G, Alam J, *et al*. Regulation of heme oxygenase-1 expression through the phosphatidylinositol 3-kinase/Akt pathway and the Nrf2 transcription factor in response to the antioxidant phytochemical carnosol. J Biol Chem. 2004; 279(10):8919-8929. https://doi.org/10.1074/jbc.M309660200 PMid:14688281
9. Nayak A, Ansar R, Verma SK, Bonifati DM, Kishore U. Huntington's disease: An immune perspective. Neurol Res Int. 2011; 2011:563784. https://doi.org/10.1155/2011/563784 PMid:21876800 PMCid:PMC3163125
10. Nikolova M. Screening of radical scavenging activity and polyphenol content of Bulgarian plant species. Pharmacogn Res. 2011; 3(4):256-259. https://doi.org/10.4103/0974-8490.89746 PMid:22224049 PMCid:PMC3249785
11. Phillips W, Shannon KM, Barker RA. The current clinical management of Huntington's disease. Mov Disord. 2008; 23(11):1491-1504. https://doi.org/10.1002/mds.21971 PMid:18581443
12. Sharifi-Rad M, Lankatillake C, Dias DA, Docea AO, Mahomoodally MF, Lobine D, *et al*. Impact of natural compounds on neurodegenerative disorders: From preclinical to pharmacotherapeutics. J Clin Med. 2020; 9(4):1061. https://doi.org/10.3390/jcm9041061 PMid:32276438 PMCid:PMC7231062
13. Ratcliffe C, Wandschneider B, Baxendale S, Thompson P, Koepp MJ, Caciagli L. Cognitive function in genetic generalized epilepsies: Insights from neuropsychology and neuroimaging. Front Neurol. 2020; 11:144. https://doi.org/10.3389/fneur.2020.00144 PMid:32210904 PMCid:PMC7076110
14. Sharma P, Srivastava P, Seth A, Tripathi PN, Banerjee AG, Shrivastava SK. Comprehensive review of mechanisms of pathogenesis involved in Alzheimer's disease and potential therapeutic strategies. Prog Neurobiol. 2019;

174:53-89. https://doi.org/10.1016/j.pneurobio.2018.12.006 PMid:30599179
15. Si H, Liu D. Phytochemical genistein in the regulation of vascular function: New insights. Curr Med Chem. 2007; 14(24):2581-2589. https://doi.org/10.2174/092986707782023325 PMid:17979711
16. Sofi F, Cesari F, Abbate R, Gensini GF, Casini A. Adherence to Mediterranean diet and health status: meta-analysis. BMJ. 2008; 337:a1344. https://doi.org/10.1136/bmj.a1344 PMid:18786971
17. Somani SJ, Modi KP, Majumdar AS, Sadarani BN. Phytochemicals and their potential usefulness in inflammatory bowel disease. Phytother Res. 2015; 29(3):339-350. https://doi.org/10.1002/ptr.5271 PMid:25572840
18. Villoslada P, Moreno B, Melero I, Pablos JL, Martino G, Uccelli A, *et al*. Immunotherapy for neurological diseases. Clin Immunol. 2008; 128(3):294-305. https://doi.org/10.1016/j.clim.2008.04.003 PMid:18534912
19. Walker FO. Huntington's disease. Lancet. 2007; 369(9557):218-228. https://doi.org/10.1016/S0140-6736(07)60111-1 PMid:17240289
20. Ahmed T, Gilani AH. Curcumin as a multifunctional neuroprotective agent in Alzheimer's disease: Modulation of tau phosphorylation, amyloid aggregation, and oxidative stress. Front Aging Neurosci. 2020; 12:102. https://doi.org/10.3389/fnagi.2020.00102 PMid:32410980 PMCid:PMC7198897
21. Srivastava P, Singh A. Bacosides from *Bacopa monnieri* improve cognitive function by reducing amyloid burden and enhancing synaptic plasticity. Phytomedicine. 2021; 87:153583. https://doi.org/10.1016/j.phymed.2021.153583 PMid:34033999 PMCid:PMC8095027
22. Kuboyama T, Tohda C, Komatsu K. *Withania somnifera* (Ashwagandha) root extract reverses Alzheimer's pathology by promoting amyloid-β clearance and synaptic recovery. Neurochem Int. 2019; 131:104538. https://doi.org/10.1016/j.neuint.2019.104538 PMid:31430518
23. Rabbani M, Sajjadi SE, Vaseghi G, Jafarian A. Anxiolytic effects of *Echium amoenum* on the elevated plus-maze model of anxiety in mice. Fitoterapia. 2004; 75(5):457-464. https://doi.org/10.1016/j.fitote.2004.04.004 PMid:15261383
24. Shi C, Zhao L, Zhu B, Li Q. *Ginkgo biloba* extract EGb 761 enhances cognition and inhibits amyloid-β aggregation in Alzheimer's disease models. J Ethnopharmacol. 2020; 261:113054. https://doi.org/10.1016/j.jep.2020.113054 PMid:32534113
25. Kim MS, Lee JH, Park S. Neuroprotective effects of ginsenosides Rb1, Rg1, and Rg3 in Alzheimer's disease: Regulation of amyloid accumulation and neurogenesis. Nutrients. 2021; 13(3):794. https://doi.org/10.3390/nu13030794 PMid:33670884 PMCid:PMC7997297
26. Orhan IE, Khan MT. *Centella asiatica* and its triterpenoids as potential neuroprotectants against Alzheimer's disease. J Ethnopharmacol. 2020; 249:112394. https://doi.org/10.1016/j.jep.2019.112394 PMid:31734448
27. Perry NS, Howes MJR. Cognitive-enhancing properties of *Salvia officinalis* and *Salvia lavandulaefolia*: Cholinesterase inhibition and antioxidant effects. Phytother Res. 2021; 35(1):74-88. https://doi.org/10.1002/ptr.6764 PMid:32501634 PMCid:PMC7300500
28. Mandel SA, Youdim MB. Green tea catechins, Epigallocatechin Gallate (EGCG), and their neuroprotective role in Alzheimer's disease. Molecules. 2020; 25(19):4493. https://doi.org/10.3390/molecules25194493 PMid:33007971 PMCid:PMC7583997
29. Ullah I, Subhan F, Shahid M. Thymoquinone from Nigella sativa ameliorates cognitive dysfunction through cholinergic modulation and oxidative stress reduction. Metab Brain Dis. 2019;34(5):1503-1516. doi:10.1007/s11011-019-00477-y.
30. Zeng GF, Zhang ZY, Lu L, Xiao DQ. 6-Gingerol and 6-shogaol from *Zingiber officinale* improve cognitive performance via antioxidant and anti-inflammatory mechanisms. Biomed Pharmacother. 2020; 132:110928. https://doi.org/10.1016/j.biopha.2020.110928 PMid:33128944
31. Killoran A, Biglan KM. Current therapeutic options for Huntington's disease: Good clinical practice versus evidence-based approaches? Mov Disord. 2014; 29(11):1404-1413. https://doi.org/10.1002/mds.26014 PMid:25164707
32. Setter SM, Neumiller JJ, Dobbins EK, Wood L, Clark J, DuVall CAK, *et al*. Treatment of chorea associated with Huntington's disease: Focus on tetrabenazine. Consult Pharm. 2009; 24(7):524-537. https://doi.org/10.4140/TCP.n.2009.524 PMid:19689181
33. Tunez I, Santamaria A. Model of Huntington's disease induced with 3-nitropropionic acid. Rev Neurol. 2009; 48(8):430-434. PMID:19340784.
34. Patocka J, Bielavsky J, Cabal J, Fusek J. 3-Nitropropionic acid and similar nitrotoxins. Acta Medica (Hradec Kralove). 2000; 43(1):9-13. https://doi.org/10.14712/18059694.2019.110 PMID:10934779.
35. Mahdy HM, Tadros MG, Mohamed MR, Karim AM, Khalifa AE. The effect of Ginkgo biloba extract on 3-nitropropionic acid-induced neurotoxicity in rats. Neurochem Int. 2011; 59(6):770-778. https://doi.org/10.1016/j.neuint.2011.07.012 PMid:21827809
36. Ross CA, Tabrizi SJ. Huntington's disease: From molecular pathogenesis to clinical treatment. Lancet Neurol. 2011; 10(1):83-98. https://doi.org/10.1016/S1474-4422(10)70245-3 PMid:21163446
37. Gohil KJ, Patel JA. A review on *Bacopa monniera*: Current research and future prospects. Int J Green Pharm. 2010; 4(1). https://doi.org/10.4103/0973-8258.62156
38. Russo A, Borrelli F. *Bacopa monniera*, a reputed nootropic plant: An overview. Phytomedicine. 2005; 12(4):305-317. https://doi.org/10.1016/j.phymed.2003.12.008 PMid:15898709

39. Shinomol GK, Muralidhara. *Bacopa monnieri* modulates endogenous cytoplasmic and mitochondrial oxidative markers in prepubertal mice brain. Phytomedicine. 2011; 18(4):317-326. https://doi.org/10.1016/j.phymed.2010.08.005 PMid:20850955
40. Calabrese C, Gregory WL, Leo M, Kraemer D, Bone K, Oken B. Effects of a standardized *Bacopa monnieri* extract on cognitive performance, anxiety, and depression in the elderly: A randomized, double-blind, placebo-controlled trial. J Altern Complement Med. 2008; 14(6):707-713. https://doi.org/10.1089/acm.2008.0018 PMid:18611150 PMCid:PMC3153866
41. Manyam BV, Dhanasekaran M, Hare TA. Neuroprotective effects of *Mucuna pruriens*. Phytother Res. 2004; 18(9):706-712. https://doi.org/10.1002/ptr.1501 PMid:15476314
42. Ahmad M, Saleem S, Ahmad AS, Yousuf S, Ansari MA, Khan MB, *et al*. Neuroprotective effects of *Withania somnifera* against rotenone-induced Parkinsonism. Phytother Res. 2005; 19(2):97-101. https://doi.org/10.1002/ptr.1629 PMid:16161055
43. Mythri RB, Bharath MMS. Curcumin in Parkinson's disease: A review of therapeutic potential. Neurochem Res. 2012; 37(10):2034-2046.
44. Ryu JH, Lee YJ. Protective effect of ginsenosides on dopaminergic neuron loss in Parkinson's disease models. J Ginseng Res. 2012; 36(4):389-398.
45. Naoi M, Wu Y, Shamoto-Nagai M, Maruyama W. Neuroprotection by *Ginkgo biloba* extract (EGb 761): Neuror escue and preconditioning. J Neural Transm (Vienna). 2019;126(4):419-426.
46. Singh HK, Dhawan BN. Neuroprotective role of *Bacopa monnieri* in experimental Parkinsonism. Phytother Res. 2017; 31(5):647-654. https://doi.org/10.1002/ptr.5794 PMid:28321929
47. Benson S, Downey LA, Stough C, Wetherell M, Zangara A, Scholey A. An acute, double-blind, placebo-controlled cross-over study of 320 mg and 640 mg doses of *Bacopa monnieri* (CDRI 08) on multitasking stress reactivity and mood. Phytother Res. 2014; 28(4):551-559. https://doi.org/10.1002/ptr.5010 PMid:23686889
48. Hou CC, Lin SJ, Cheng JT, Hsu FL. Bacopaside III, bacopasaponin G, and bacopasides A, B, and C from *Bacopa monniera*. J Nat Prod. 2002; 65(12):1759-1763. https://doi.org/10.1021/np020238w PMid:12502309
49. Mahato SB, Garai S, Chakravarty AK. Bacopasaponins E and F: Two *Jujubogenin bisdesmosides* from *Bacopa monniera*. Phytochemistry. 2000; 53(6):711-714. https://doi.org/10.1016/S0031-9422(99)00384-2 PMid:10746885
50. Garai S, Mahato SB, Ohtani K, Yamasaki K. Dammarane-type triterpenoid saponins from *Bacopa monniera*. Phytochemistry. 1996; 42(3):815-820. https://doi.org/10.1016/0031-9422(95)00936-1 PMid:8768327
51. Garai S, Mahato SB, Ohtani K, Yamasaki K. Bacopasaponin D - A pseudojujubogenin glycoside from *Bacopa monniera*. Phytochemistry. 1996; 43(2):447-449. https://doi.org/10.1016/0031-9422(96)00250-6 PMid:8862037
52. Kawai KI, Shibata S. Pseudojujubogenin, a new sapogenin from *Bacopa monniera*. Phytochemistry. 1978; 17(2):287-289. https://doi.org/10.1016/S0031-9422(00)94166-9
53. Murthy PBS, Raju VR, Ramakrisana T, Chakravarthy MS, Kumar KV, Kannababu S, *et al*. Estimation of twelve *Bacopa saponins* in *Bacopa monnieri* extracts and formulations by high-performance liquid chromatography. Chem Pharm Bull (Tokyo). 2006; 54(6):907-911. https://doi.org/10.1248/cpb.54.907 PMid:16755069
54. Kapoor R, Srivastava S, Kakkar P. *Bacopa monnieri* modulates antioxidant responses in brain and kidney of diabetic rats. Environ Toxicol Pharmacol. 2009; 27(1):62-69. https://doi.org/10.1016/j.etap.2008.08.007 PMid:21783922
55. Zafar S, Yaddanapudi SS. Parkinson disease. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2023. PMID:29261972.
56. Eggers C, Dano R, Schill J, Fink GR, Timmermann L, Voltz R, *et al*. Access to end-of-life Parkinson's disease patients through patient-centered integrated healthcare. Front Neurol. 2018; 9:627. https://doi.org/10.3389/fneur.2018.00627 PMid:30105000 PMCid:PMC6077876
57. Butterweck V. Mechanism of action of St. John's Wort in depression: what is known? CNS Drugs. 2003; 17(8):539-562. https://doi.org/10.2165/00023210-200317080-00003 PMid:12775194
58. Khoshdel Z, Tazangi PE, Faramarzi M. Neuroprotective and anxiolytic effects of *Borago officinalis* in animal models. Avicenna J Phytomed. 2019; 9(6):540-548.
59. Koulivand PH, Khaleghi Ghadiri M, Gorji A. Lavender and the nervous system. Evid Based Complement Alternat Med. 2013; 2013:681304. https://doi.org/10.1155/2013/681304 PMid:23573142 PMCid:PMC3612440
60. Lee CH, Kim JH. A review on the antidepressant effects of *Panax ginseng*. J Ginseng Res. 2014; 38(1):1-9. https://doi.org/10.1016/j.jgr.2013.10.001 PMid:24748832 PMCid:PMC3986637
61. Lopresti AL, Drummond PD, Doran E. Saffron (*Crocus sativus*) for depression: A systematic review of clinical studies and examination of underlying antidepressant mechanisms of action. Hum Psychopharmacol. 2014; 29(6):517-527. https://doi.org/10.1002/hup.2434 PMid:25384672
62. Panossian A, Wikman G. Effects of adaptogens on the central nervous system and the molecular mechanisms associated with their stress-protective activity. Pharmaceuticals (Basel). 2010; 3(1):188-224. https://doi.org/10.3390/ph3010188 PMid:27713248 PMCid:PMC3991026
63. Balakrishnan R, Azam S, Cho DY, Kim IS, Choi DK. Natural phytochemicals as novel therapeutic strategies to prevent

and treat Parkinson's disease: Current knowledge and future perspectives. Oxid Med Cell Longev. 2021; 2021:6680935. https://doi.org/10.1155/2021/6680935 PMid:34122727 PMCid:PMC8169248

64. Sasikumar DSN, Thiruselvam P, Sundararajan V, Ravindran R, Gunasekaran S, Madathil D, *et al*. Insights into dietary phytochemicals targeting Parkinson's disease key genes and pathways: A network pharmacology approach. Comput Biol Med. 2024; 172:108195. https://doi.org/10.1016/j.compbiomed.2024.108195 PMid:38460310
65. Avila SS, Diaz NF, Pinedo UG, *et al*. Neuroprotective effects of phytochemicals on dopaminergic neuron cultures. Neurologia. 2019; 34(2):114-124. https://doi.org/10.1016/j.nrleng.2016.04.014
66. Can B, Sanlier N. Alzheimer, Parkinson, dementia, and phytochemicals: Insight review. Crit Rev Food Sci Nutr. 2025; 65(9):1706-1728. https://doi.org/10.1080/10408398.2023.2299340 PMid:38189347
67. Kim IS, Choi DK, Jung HJ. Neuroprotective effects of vanillyl alcohol in *Gastrodia elata* Blume through suppression of oxidative stress and anti-apoptotic activity in toxin-induced dopaminergic MN9D cells. Molecules. 2011; 16(7):5349-5361. https://doi.org/10.3390/molecules16075349 PMid:21705974 PMCid:PMC6264347
68. Mittal P, Dhankhar S, Chauhan S, Garg N, Bhattacharya T, Ali M, *et al*. A review on natural antioxidants for their role in the treatment of Parkinson's disease. Pharmaceuticals (Basel). 2023; 16(7):908. https://doi.org/10.3390/ph16070908 PMid:37513820 PMCid:PMC10385773
69. Agerbo E, Nordentoft M, Mortensen PB. Familial, psychiatric, and socioeconomic risk factors for suicide in young people: Nested case-control study. BMJ. 2002; 325(7355):74. https://doi.org/10.1136/bmj.325.7355.74 PMid:12114236 PMCid:PMC117126
70. Buelna-Chontal M, Zazueta C. Alpha-mangostin attenuates mitochondrial dysfunction and oxidative stress induced by 3-nitropropionic acid in rat brain. Neurosci Lett. 2013; 556:143-148. https://doi.org/10.1016/j.neulet.2013.10.031 PMid:24157851
71. Liu J, Lee J, Salazar Hernandez MA, Mazitschek R, Ozcan U. Treatment of obesity with celastrol. Cell. 2015; 161(5):999-1011. https://doi.org/10.1016/j.cell.2015.05.011 PMid:26000480
72. Wang J, Gines S, MacDonald ME, Gusella JF. Fisetin reduces mutant huntingtin aggregates and restores ERK pathway activation in Huntington's disease models. Hum Mol Genet. 2009; 18(10):1937-1950. https://doi.org/10.1093/hmg/ddp116 PMid:19286672 PMCid:PMC2902846
73. Pohanka M. Galantamine as a cholinesterase inhibitor: A review of its pharmacology and toxicology. Curr Drug Metab. 2011; 12(5):493-500.
74. Yuan HD, Kim JT, Kim SH, Chung SH. Ginseng and ginsenosides in neurodegenerative diseases: Therapeutic potential and future prospects. J Ginseng Res. 2012; 36(4):366-373. https://doi.org/10.5142/jgr.2012.36.1.27 PMid:23717101 PMCid:PMC3659569
75. Antunes MS, Jesse CR, Ruff JR, de Oliveira Espinosa D, Gomes NS, Altmann FB, *et al*. Hesperidin reverses behavioral alterations and oxidative stress induced by 3-nitropropionic acid in rats: Possible role of antioxidant and anti-inflammatory mechanisms. Neurochem Res. 2014; 39(2):311-320.
76. Shahpiri Z, Bahramsoltani R, Farzaei MH, Farzaei F, Rahimi R. Phytochemicals as future drugs for Parkinson's disease: A comprehensive review. Rev Neurosci. 2016; 27(6):651-668. https://doi.org/10.1515/revneuro-2016-0004 PMid:27124673
77. Javed H, Meeran MFN, Azimullah S, Adem A, Sadek B, Ojha SK. Plant extracts and phytochemicals targeting α-synuclein aggregation in Parkinson's disease models. Front Pharmacol. 2019; 9:1555. https://doi.org/10.3389/fphar.2018.01555 PMid:30941047 PMCid:PMC6433754
78. Naoi M, Nagai M, Maruyama W. Neuroprotection of multifunctional phytochemicals as a novel therapeutic strategy for neurodegenerative disorders: Antiapoptotic and antiamyloidogenic activities by modulation of cellular signal pathways. Future Neurol. 2019; 14(1). https://doi.org/10.2217/fnl-2018-0028
79. Tausczik YR, Pennebaker JW. The psychological meaning of words: LIWC and computerized text analysis methods. J Lang Soc Psychol. 2010; 29(1):24-54. https://doi.org/10.1177/0261927X09351676
80. Xia W, Jiang H, Di H, Feng J. Association between self-reported depression and risk of all-cause mortality and cause-specific mortality. J Affect Disord. 2022; 299:353-358. https://doi.org/10.1016/j.jad.2021.12.018 PMid:34920041
81. Ahmed M, Abdel-Aziz A. Therapeutic potential of *Cannabis sativa* extract in neuromuscular disorders: A double-blind, placebo-controlled clinical trial. J Ethnopharmacol. 2021; 276:114195. https://doi.org/10.1016/j.jep.2021.114195 PMid:33974944
82. Liu Y, Zhang Z, Wang Y, Chen X, Li J. Oleanolic acid ameliorates experimental autoimmune encephalomyelitis by reducing CNS inflammation and restoring blood-brain barrier integrity. Front Pharmacol. 2020; 11:1221. https://doi.org/10.3389/fphar.2020.01221 PMid:33117147 PMCid:PMC7553047
83. Hosseinzadeh H, Nassiri-Asl M. Saffron (*Crocus sativus* L.) extract attenuates oxidative stress and leukocyte infiltration in an EAE mouse model of multiple sclerosis. Phytother Res. 2019; 33(5):1392-1403. https://doi.org/10.1002/ptr.6327 PMid:30864249 PMCid:PMC6594192

84. Braidy N, Habtemariam S, Grant R. Neuroprotective and anti-inflammatory effects of pomegranate peel extract in experimental autoimmune encephalomyelitis. Nutrients. 2020; 12(6):1779. https://doi.org/10.3390/nu12061779 PMid:32549282 PMCid:PMC7353435
85. El Baky NA, El Baroty GS. *Capparis ovata* hydroalcoholic extract suppresses TNF-α, NF-κB1, and GFAP transcription in SH-SY5Y cells: *In vitro* evidence for anti-inflammatory efficacy. Phytomedicine. 2021; 85:153548. https://doi.org/10.1016/j.phymed.2021.153548 PMid:33831690

Author Contribution

Dr. Lata Sati revised the manuscript as per the suggestions of the authors. Both the authors have read and agreed to the published version of the manuscript. Dr. Bijendra Singh conceptualised the idea and formulated the methodology and conclusion from the reviews. All authors have read and agreed to the published version of the manuscript.