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NEURODEGENERATIVE DISORDERS: ROLE OF MEDICINAL PLANTS IN TREATMENT

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ABSTRACT: Neurons are the nervous system's basic components that involve the spinal cord and brain. Neurons do not usually replicate or substitute them because the body cannot replace them as they get impaired or die. Neurological conditions are untreatable and disabling diseases that result in irreversible deterioration/death of the human brain's nerve cells or neurons. Treating these conditions with extended synthetic drug administration can lead to serious side effects. So, herbal therapy is preferable over traditional therapies. Using herbal remedies can provide significant potential benefits in preventing and treating Neurological disorders. Herbal medicines have been gaining much attention for their therapeutic value over many years. Herbal plants have been used to treat and manage various ailments from ancient times due to their economic availability and lesser side effects than conventional anti-parkinsonian drugs. The current review incorporates research on medicinal plants, which has demonstrated the ability to transcend the development of neurological disorders and highlights the significance of traditional plant species in the role and mechanisms involved in neuroprotection. Ayurveda is regarded as an ancient-age traditional medicine with substantial use of herbs and herbal supplements proven to diagnose different conditions.

INTRODUCTION: Neurons are the nervous system's basic components that involve the spinal cord and brain. Neurons do not usually replicate or substitute them because the body cannot replace them as they get impaired or die. Neurodegenerative disorders are untreatable and chronic illnesses that lead to the gradual deterioration/extinction of nerve cells in the brain. This triggers movement problems (called ataxias) or mental control ("Dementias") [1].

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The degenerate neurons aren't replaced, resulting in cognitive impairment and neuro-degenerative problems, which lead to depression, schizophrenia, Alzheimer's disease, dementia, epilepsy, cerebral ischemia, and Parkinsonism [2]. Such disorders include different pathological or structural characteristics affecting neurons in different brain regions.

Neurodegenerative disorders are ramped up by how we live our everyday lives. According to a new survey by the Indian Council of Medical Research (ICMR), the percentage of reported deaths owing to lifestyle-associated had risen from 37.09 percent in 1990 to 61.8 percent in 2016. Therefore, there's still an enormous need to establish methods for its elimination as there are growing numbers of people

who suffer from related diseases every day. Ayurveda is regarded as an ancient-age traditional medicine with substantial use of herbs and herbal supplements proven to diagnose different conditions. Much beyond established variations in pathogenic processes of human disorders, neurodegenerative disorders, recognized as the process that leads to the progressive degradation of functional characteristics of neurons before apoptosis, reflect the central point in this category of disorders and promote scientific interests in attempting to explain specific pathological processes and attain relevant interventions [3]. The precise reason for multiple neurological disorders is a healthcare mystery. The widely researched factors of neurological conditions involve environmental causes, oxidative stress, inflammation, anomalous neuronal protein accumulation, mitochondrial deficiencies, protein degradation, and medical history [4]. Different classical preparations for neurological disorders are mentioned in the Ayurvedic Formulary of India (AFI), which also offers a database of records on a single crop, animal, and mineral products, including their original names with Common counterparts for simple identification [5]. The present analysis draws together literature on plant origin that has shown potential in overcoming the progression of neurological disorders and emphasizes the importance of traditional medicinal plants on neuroprotective roles and mechanisms involved.

Neurological Disorders: There are several neurological system disorders; some are listed in **Fig. 1.**

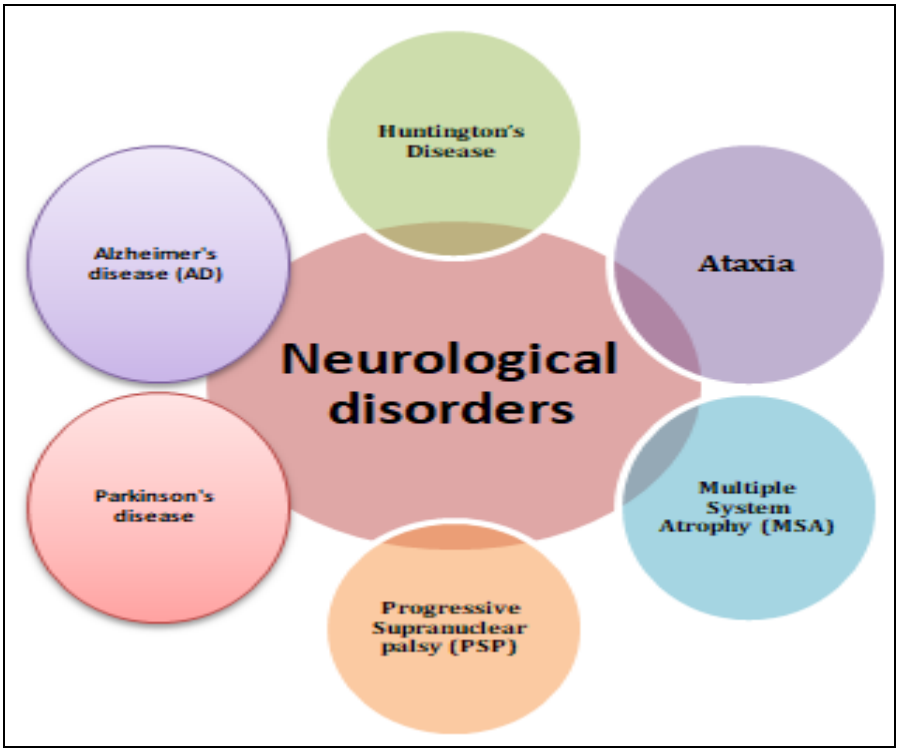

FIG. 1: NEUROLOGICAL DISORDERS

Alzheimer's disease (AD): Alzheimer's disease (AD) was initially known as presenile dementia, which denotes an inherited psychiatric disorder with a lack of critical ability to interact with social functioning. This is correlated with regional brain shrinkage and neuron loss, particularly in the hippocampus and the basal forebrain. The beta-amyloid peptide (BAP) plays an important role in AD growth. While synthetic drugs cannot cure AD, but can be handled with them to some level. Numerous researches have shown that antioxidant compounds, like beta-carotene, vitamin C, and vitamin E, are effective in the free radical scavenging produced during this disease progression. Memory failure is believed to stem from a lack of a nerve transmitter, acetylcholine. The level of such a transmitter in the brain could be improved by hindering the enzyme activity, acetylcholinesterase, which fragments or disintegrates the transmitter material. Synthetic,

medicinal products which restrict the collapse of transmitter acetylcholine could postpone disease progression [6].

Parkinson's disease: Parkinson's disease is a developmental infection affecting slow motion and body stiffness. The psychiatric diagnosis involves bradykinesia (experiencing problems triggering movement), rest tremors, hypokinesia, and rigidity, with non-motor symptoms including autonomic psychosis disorder, and depression. Other signs include rigid posture of the face, gait, psychological changes, repetitive motions, loss of blinking, autonomic dysfunction, micrography, seborrhoea and neurological, sensory effects, and atrophy of the muscles.

It is defined by neuronal failure in substantia nigra and other brain areas, often connected to intracellular protein inclusions such as Lewy bodies. Depleting dopaminergic neurons, contributes to dopamine being released into the striatum getting limited.

The main suspect is mitochondrial dysfunction and oxidative attack. The latest treatment available for PD focuses mainly on Levodopa, which can delay the progression of the disease to a certain degree but has several adverse effects [7-9].

Huntington's disease: The infection is characterized *via* an autosomal dominantly inherited CAG trinucleotide repeat amplification on chromosome 4 in the huntingtin (HTT) gene. Consequently, mutated huntingtin (mHTT) proteins *via* an unusually large polyglutamine loop can be produced. Someone with more than 39 CAG repeats seemed to acquire the illness, whereas those with 36 to 39 repetitions have decreased susceptibility. Whenever the genome is transmitted through the paternal way, it's also possible that even a husband with a CAG repeat sequence throughout the average range will produce a kid with an enlarged pathogenic repeated length.

It's because male sperm has more repetition variation and more repetition lengths than somatic tissue. A verified genetic makeup of HD, a confirmed genetic test, and the beginning of motor dysfunction are used to make the diagnosis. The score goes between 0 (without motor irregularities indicative of HD) - 4, with either a score of 4 indicating motor start [10-12].

Ataxia: Ataxia, described as poor control of voluntary muscular action, seems to be a clinical finding rather than an illness, as well as the root because it must be researched. It might be the patient's primary concern or one of several clinical presentations. Cerebellar impairment or reduced vestibular or proprioceptive afferent stimulation to the cerebellum are generally the causes.

The position of the lesion within the cerebellum is frequently associated with signs and indications. Ipsilateral signs and symptoms are caused by lateralized cerebellar lesions, while diffuse cerebellar abnormalities generate more widespread symmetrical signs. Injuries in the cerebellar lobe cause limb ataxia. Truncal and gait ataxia is caused by vermis injuries, with limbs remaining largely unaffected. Inquiries about work, possibly hazardous exposures to substances, sexual exploits, drug addiction, and excessive drinking can all be part of the social and historical record. A background of substance abuse might point to chemicals or intoxication. Poisoning can occur due to workplace heavy metal exposure or chemicals [13].

Multiple System Atrophy (MSA): MSA is now an uncommon and deadly neurological condition characterized by a varied mix of parkinson's disease, cerebellum abnormalities, and cognitive impairment. The pathological characteristic has been the formation of accumulated α-synuclein in oligodendrocytes, resulting in glial cytoplasmic inclusion, defining MSA as a synucleinopathy alongside dementia.

Before clinical trials, pre-clinical modeling was utilized for several years to explore the mechanisms that underlie MSA development, discover new therapeutic targets, and verify the most effective drugs. Such systems were first created to mimic coupled nigral and striatal degradation with toxic substances used to simulate Parkinson's and Huntington's disease. Autonomic abnormalities occur as the initial sign in a substantial proportion of MSA cases, in either conjunction with it or independently of motor symptoms [14].

Progressive Supranuclear Palsy (PSP): The pathological substances of the latest entity of progressive supranuclear palsy (PSP), also recognized as Steele-Richardson-Olszewski syndrome, were identified as immense degeneration of subcortical neurofibrillary primarily found throughout the subthalamic nucleus, globus pallidus, cerebellar dentate nucleus, and substantia nigra. The clinical manifestations of such PSP subgroups are particularly distinct during the first 2 years after onset.

It has still been regarded as the typical and most common clinical manifestation of PSP-tau disease, accounting for 50% more than PSP patients with post-mortem diagnoses. PSP-P has been the second most prevalent form, accounting for up to one-third of all PSP occurrences. PSP-PAGF, cortical PSP variations (PSP-CBS, PSP-PNFA, PSP-bvFTD), and recently discovered PSP-C are all uncommon, accounting for fewer than 5% of all PSP instances. Anecdotal data from published studies suggests that zolpidem, a GABA agonist, can enhance motor skills, dysarthria, and visual problems. Selective serotonin reuptake inhibitors (SSRIs) are excellent treatments for depression, obsessive-compulsive disorder, and mood changes, but they may exacerbate apathy. In people with PNFA, memantine can give symptomatic relief [15].

Ayurvedic Treatment Favoured Over Prescription Medications: Ethnopharmacology, a knowledge-driven method of drug development, plays a part in drug development centered on natural or conventional awareness of the pharmaceutical or toxicological effects in the human population from animals, plants, and fungi [16]. Over 119 commercially accepted medicines are actually extracted from medicinal herbs. Of these, 74 percent have been identified through chemical detection of the components necessary for patient therapeutic use. Such 119 herbal medicines are made commercially from more than 90 plant species. With more than 25,000 organisms on the planet, their comprehensive study will produce more effective medicines against common viruses [17].

Ethno-pharmacological method for drug research is found to be highly effective for developing new drugs ranging from digitalis to vincristine. The most critical step in identifying medicines from plant sources is choosing the most appropriate materials based on ethnomedicinal and ethnobotanical applications. **Table 1** shows some medicinal herbs widely used for their neuroprotective impact. Also, various plants which show neuroprotective activity are listed in **Fig. 2.**

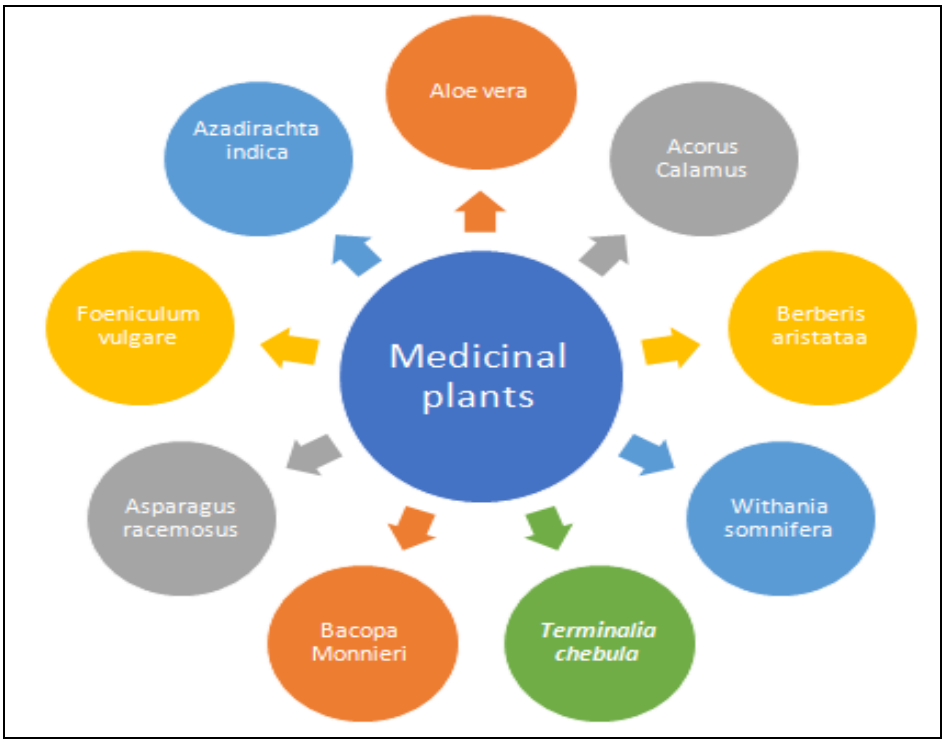

FIG. 2: MEDICINAL PLANTS UTILIZED IN THE TREATMENT OF NEUROLOGICAL DISORDERS

TABLE 1: MEDICINAL HERB WITH NEUROPROTECTIVE MECHANISM OF ACTION

Medicinal Herb	Family	Constituents	Mechanism of action	References
Aloe vera	Liliaceae	Chrysophanol, aloe-emodin emodin anthrones (aloin A and B), aloe resin	AV also preserved mitochondrial structure and activity in mouse brains. AV at 10 mg x L(-1) showed a therapeutic effect on NaN3-induced dysfunction of the mitochondrial role (P < 0.01).	18
Terminalia chebula	Combretaceae	chebulinic acid, ethyl gallate, ArjunglucosideI, punicalagin chebulosides I and II, arjungenin, gallic acid,	It confers an antagonist of acetylcholinesterase but had proposed the development of such a plant as a tool for treating Alzehmier's	19
Bacopa Monnieri	Plantaginaceae	Stigmastanol, Bacoside A, D- Mannitol, Stigmasterol b-Sitosterol, Betulinic acid, Bacoside,	It suppressed cholinergic degeneration and demonstrated a cognitive effect in the rat	20
Foeniculum vulgare	Apiaceae	Anethole, methyl chavicol and fenchone	It increased the SDL when mice were treated with the passive paradigm of avoidance, signifying their potential anti-amnesic activity.	21
Withania somnifera	Solanaceae	Withasomniferols A to C, dehydrowithanolide R, withaferin A, & withanone withasomidienone, Withanolides A to Y, withasomniferin A,	Withanamides are shown to scavenge freely generated radicals during the initiation and progression of Alzheimer's.	22

***Aloe vera*:** *Aloe vera* is a perennial, evergreen, and succulent herb grown worldwide, particularly in medicinal and agricultural applications. It consists of aloenin, aloe resin, aloe emodin, aloin A, aloin B and chrysophanol [23, 24]. Aloe-emodin is stated to boost memory deficits in mice caused by scopolamine [25]. The utilization of aloin as an alternative treatment for vascular complications has proven to lower reactive species and minimize calcium ions + output, which really is liable for neurons depolarization and mortality [26]. *Aloe vera* gel displayed anti-inflammatory and antioxidant effects once administered to rats with an injury to the sciatic nerve [27].

Terminalia chebula: In ayurvedic medicines, *Terminalia chebula* (Combretaceae, widely known as Haritaki) is being utilized to show neuroprotective behaviour. It constitutes of chebuloside (glycosides), gallic acid, ellagic acid, chebulinic acid and chebulagic acid [28, 29]. The fruit pericarp constitutes Triphala, commonly utilized in the formulations of ayurveda. Fructus chebulae, fruit extract (methanolic -70 percent), has indeed been illustrated by protecting from neuronal degeneration to rescue brain ischemia. Promising findings were demonstrated *in-vivo* and *in-vitro* studies [30]. After treatment with the extract, microglial death, lower concentrations of cellular nitric oxide, and MDA (malondialdehyde) are reported. An interruption of oxidative and inflammatory pathways may be the fundamental theory. Its constituents have already shown their impacts on specific sites by providing neuroprotective action in several cells [31, 32].

***Bacopa monnieri*:** *Bacopa monnieri* is a unique herbal herb belonging to family Scrophulariaceae. It is a neuronal tonic that enhances intelligence and intellect and has a cognitive function effect [33, 34]. Its formulation consisting of 43 percent dry leaf extract, with 20 percent bacoside, reduced the generation of reactive oxygen species and 8-iso-PFG2α and thus reducing the stress of oxidation incited by amyloid peptide, H_2O_2 and basal in the studies on rat [35].

In a different study, the dosage-related increases in activities of peroxidase of glutathione, catalase, and superoxide dismutase in the hippocampus, striatum and cerebral cortex were observed as with *Bacopa monnieri* extract (82 ± 0.5 percent) once orally administered in the concentrations of 5 and 10 mg per kg [36]. *Bacopa monnieri* increases memory and

cognitive decline in rats once taken orally. Immunohistology of superoxide dismutase and histological changes were reported in the hippocampus (CA1 area) [37]. The 5 percent (w/w) saponins extract comprises bacopaside I, bacopaside II, bacopasaponin C, bacopasaponin X and bacoside demonstrates enhanced cognitive capacity in model of disorder of Alzheimer's and neuroprotective effects [38, 39]. Bacoside-treated rats showed significant improvements in the grades of antioxidants (enzymatic and nonenzymatic), suggesting that the *Bacopa monnieri* antioxidants state of the mouse brain is strengthened by the plant [40]. *Bacopa monnieri* extracts inhibited the development of oxygen radicals concentrations and suggested crucial security in dopaminergic cytotoxicity (N27 cell lines) mediated by 3-nitropropionic acid. Extract of plants with 55 percent of bacosides showed a protective effect on the memory obstruction caused by ischemia and decreased the size of the ischemic brain infarct (ISB) [41]. In addition, it enhanced the effect of catalase activity and reduced peroxidation of lipid and activity of nitrate. Cultures of neurons on treatment with *Bacopa monnieri*, defended neurons against cell toxicity caused by β-amyloid [42].

The extract shielded cultured cells from glutamate-induced excitotoxicity because glutamate-medium toxicity could not be suppressed. The plant extract showed a decline in the action of lipoxygenase and peroxidation of lipid induced by hydrogen peroxide in Alzheimer's animal illness model (Bl6 mice) [43]. *Bacopa monnieri* induces neuroprotective functions and decreases β-amyloid residues in the same model. The extract has also demonstrated a beneficial impact on Parkinson's disorder caused by rotenone in cell lines of PC-12 [44].

***Asparagus racemosus*:** *Asparagus racemosus* methanol extracts reduced cytokine degrees, malondialdehyde (as the indicator for peroxidation production), and nitrous oxides with essential increases in glutathione, superoxide dismutase, and catalase. Its root extract (100 mg per kg) has healed neurodegeneration (specific to region) and showed a dosage-dependent increase in memory following histopathological studies in albino rats [45]. There has been a marked reduction in the duration of the transfer period and a major increase in AchE (acetylcholinesterase) in histopathological recognition indicating neuroprotective, cholinergic and antioxidant characteristics of *Asparagus racemosu* [46]. EuMil (pharmaceutical formulation) has also been used to rebuild a difference in the amount of dopamine, 5-hydroxytryptamines, and nor-adrenaline (100 mg per kg per oral for fourteen days) containing standard extracted *Asparagus racemosus* for stress-related issues, has been found [47].

The results in levels of growth factor of pro-brain, NCF (nerve growth factor), glutathiones, glutathione disulfide, malondialdehyde, lactic dehydrogenase, and cell viability studies were significant together for *Asparagus racemosus* and *Withania somnifera* [48]. Ovariectomized female Wistar rats demonstrated a substantial increase in the levels of sex hormones (ERα and ERβ), as well as an improvement of neurotrophic input variables (brain-derived), in the region of the frontal cortex and hippocampus. A high expression of sex hormones and an increase in brain-related neurotrophic control factors can be supplied as evidence for the protective effect of an *Asparagus racemosus* ethanol extract [49, 50]. A substantial benefit is observed just after Mentat supplementation (BR-16A), which includes an *Asparagus racemosus* leading to pentylene-tetrazole threshold reduction in rats and mice caused by the withdrawal of ethanol. For the neuroprotective effect in Alzheimer's, sarsasapogenin, saponin (steroidal) of *Asparagus racemosus*, was researched [51]. The notable impairment on acetylcholinesterase, beta-secretase 1, monoamine oxidase-B and butyrylcholinesterase, main enzymes relevant to Alzheimer's pathophysiology, has been demonstrated by sarsasapogenin. Sarsasapogenin had an immense protective effect on cells of PC12 at the time of cytotoxic effects interceded by H_2O_2 and Aβ42 in the study. These findings indicated that sarsasapogenin might be used to treat various pathogenic components of Alzheimer's as a multiple target-driven ligand and as a rational leading agent [52].

***Foeniculum vulgare*:** Foeniculum extract decreased amnesia and memory losses in aging-induced mice. *Foeniculum vulgare* extract has shown acetylcholine inhibition and has substantially increased the duration in the rodents

in the model of exteroceptive behavioural [53]. Fennel volatile oils exposure reduces anxiety and depression caused by beta-amyloid (1-42) and suggests that additional clinical usage can occur [54]. In the experimental model of *Foeniculum vulgare*, there have been enhancements to Parkinson's [55]. Therapeutically, intake of fennel in overweight females lowered body mass, reduced serum Aβ proteins, and improved brain abilities [56]. The expression patterns of oxidant markers of stress (Peroxiredoxin-6 and dismutase of superoxide), an isoform of APP (695 and 770), and the anatomical degradation of neural cells caused by Pb were also enhanced [57].

***Azadirachta indica*:** *Azadirachta indica* has been shown to minimize neurotoxicity induced by cisplatin in rats, frequently referred to as Neem in northern India, and has a protective role on hypoperfusion and reperfusion of ischemia in cerebral cortex [58, 59]. In Parkinson-induced functionally impaired individuals, extracts were proven to be neuroprotective, anti-apoptotic, and anti-oxidative [60, 61]. A standard indica leaf (complete bitterness 4.3 percent), as demonstrated in anti-apoptotic, antioxidant, and anti-inflammatory research, was neuroprotective in the significant injury of nerves in rats [62].

***Picrorhiza kurroa*:** Ayurvedic plant, *Picrorhiza kurroa*, seems to have a potential for vitiligo photochemotherapy. *Picrorhiza kurroa*, apocynin is seen to be *in-vivo* neuroprotective. In an animal model of colitis induced chemically, this also demonstrates a preventive role [63-64]. It facilitates long-term memory rehabilitation, aids in hippocampal neuroprotective effects, and decreases the activation by the glial cells after transient globalized brain ischemia in rats. Numbers of significance in plasma (C_{max}) are 502, 104, 244 ng/ml with a half-life of six, eight, and fourteen hours, respectively, of apocynin, picrosides II and I [65, 66]. *Picrorhiza kurroa* often avoids memory impairment by hindering the activation of NLRP3 inflammatory and the expression of BACE1 in 5xFAD mouse [67]. Plant iridoid medicinal attributes in diseases such as alzheimer's disorders have lately been assessed separately [68, 69].

***Berberis aristata*:** Berberine has already been demonstrated to provide neuroprotection by Nrf2 increased expression and antioxidant activity and stimulate Akt /PI3K signalling in SH-SY5Y cells to relieve the rotenone-mediated cell toxicity. In mouse models of Alzheimer's, neuroprotective activities of berberine were also reported [70, 71]. The beneficial role of Berberine nanostructures towards neurodegenerative changes triggered by LPS was demonstrated [72]. Berberine gives neuroprotective effects by blocking inflammatory cytokines in the treatments of cerebral ischemia [73, 74]. The underlying proprieties of the mediated CA1 neurons of the Aß neuroinflammation were changed, berberine also showed a preventive role. Several forms berberine neurons are covered and recently updated. Authors considered it to be a possible contender to fight neurological disorders [75, 76].

***Withania somnifera*:** *Withania somnifera*, also recognized as Ashwagandha, is also an herbal medication. It improves memory capacity, is an immune booster, is neuroprotective, and shows anti-stress, antioxidant, and anti-inflammatory action. Raut et al. researched the dosage tolerance, protection, and behaviour of *Withania somnifera* and proposed a mean dose tolerance of 750–1250 mg per day [77, 78]. *Withania somnifera* acts on NADPH-d by hindering corticosterone secretion and stimulating choline acetyltransferases which enhance serotonin in the hippocampus. *Withania somnifera* active ingredients like withanolide VI, withanolide IV and withanolide A, can repair the post- and pre-synapses and include neural axons and dendrites regeneration [79]. Many species of plants have been used to cure various diseases, and their medicinal effect is demonstrated through extracts as a raw and semi-purified types. The anti-stress behaviour of *W. somnifera* derivatives treatment in Wistar rats and psychological distress caused by distortions were shown in study [80].

***Acorus calamus*:** *Acorus calamus* Linn. (Acoraceae), commonly called Vacha in Sanskrit, seems to be an aromatic mid-term perennial herb used in the Ayurveda medical systems. The rhizomes of the vegetation are brown, circular, bent, and have a small nod. The leaves have a sword-like shape that is considerably thicker and has curved borders [81].

The calamus oil extracted from the rhizome were tested for MES, minimal clonic seizure (MCS), and pentylenetetrazol (PTZ), models at various dosages. At 300 mg/kg, oil was found to be neurotoxic, despite its effectiveness in the MCS testing at 6 Hz. The oil has been shown to have a protection index of 4.65 [82]. The open field test (OFT) was used to assess the anxiolytic effect of A. calamus tensarin medication in mice. At each of the three dosing levels (50, 100, and 200 mg/kg). An increase in rearing, passage number, and duration employed by mice provided an anxiolytic effect in a dose-dependent manner [83].

The ethanolic extract was tested for memory and learning activities (doses of 25, 50, and 100 mg/kg,). Male levels were employed as participants using the shuttle box and Y maze test. The results revealed an increase in evidence of spatial recognition [84]. It also can potentially increase dopaminergic neuron activity by boosting external dopamine levels and tyrosine hydroxylase expression in substanianigra, and hence may contribute to Parkinson's disease. This also boosts DJ-1 expression of genes throughout the striatum, making it neuroprotective against Parkinson's disease [85].

Effects of Medicinal Plant on Learning and Memory: Memory is a person's ability to remember perceptual stimulation, activities, details, and so on., sustain these across a short and long period , and retrieve the same as needed at a subsequent time. Learning is just the act of gaining information more about the environment, and memory may be described as storing the knowledge gained that could be recalled if or when needed [86]. Memory disruptions may vary from moderate to extreme and gradual or instant. Most studies have shown that learning and memory play a key role in the cholinergic system. Reduced cholinergic neurons and decreased development of choline acetyltransferase in the hippocampus and cerebral cortex are associated with reports of Alzheimer's disease (AD) [87]. Plants involved in learning and memory are shown below in **Table 2.**

TABLE 2: PLANT USED FOR LEARNING AND MEMORY

Medicinal Plant	Study	References
Hypericum perforatum	Concurrent administration of Hypericum in mice as well as its bioactive constituents, enhanced passive memory through shuttle box.	88
Lavandula officinalis	Treatment with essential lavender oil substantially reduced neural defects, stroke volume, MDA levels, carbonyl and reactive oxygen species in rats and showed a significant neuroprotective effect.	89
Ginkgo biloba (G. biloba)	AChE activity within the brain is strongly suppressed by plant extract. The reduction in AChE activity reflects an improvement in basal acetylcholine level.	90
Polygala tenuifolia (P. tenuifolia)	*P. tenuifolia* demonstrated the ameliorative effect on the decrease in the accumulation of passive avoidance caused by scopolamine by improving the cholinergic system.	91
Melissa officinalis (M. officinalis)	*M. officinalis* has acetylcholine receptor activity in the CNS and activates cognitive performance after acute administration.	92
Celastruspaniculatus (C. paniculatus)	A slight rise in AChE activity in the hippocampus happens when the specimen is administered with Celastrus seed oil 200 mg/kg body weight.	93

Other Plants: Several species used in conventional neurological condition treatments have to be investigated phytochemically and compared with current studies. The listing of species with identified neuroprotective activity is given in **Table 3.**

TABLE 3: HERBAL PLANTS HAVING POTENTIAL FOR NEUROPROTECTIVE ACTION

Medicinal Plant	Activity reported	Parts involved	References
Trichosanthes dioica	Root showing neuropharmacological activity.	Rhizome	94
Tinospora cordifolia	Neuro-regenerative action towards excitotoxicity induced by glutamate, neuro-inflammation is suppressed.	Stems	95-97
Sida cordifolia	In Parkinson, ameliorative action.	Whole plant	98
Semecarpus anacardium	Neuroprotective action induced by stress.	Fruits	99
Premna mucronate	Neuroprotective action by apigenin and luteolin.	Whole plant	100
Pluchea lanceolate	Protection towards neuroinflammation induced by LPS in cells of glial in rats, protects neurotoxicity induced	Leaf	101-103

	by aluminium chloride in mice, protects injury of ischemia induced by endothelin.		
Alhagipseudalhagi	Neuroprotective action by alkaloids and flavonoids.	Whole plant	104-106
Fumaria indica	Extract of the leaf shows an antianxiety effect and prominent activity on dysfunctions of cognitive is reported.	Leaf	107-108

CONCLUSION: The use of herbal medicines has been gaining much attention over many years for their therapeutic value. Herbal plants have been used to treat and manage various ailments from ancient times due to their economic availability and lesser side effects than conventional anti-parkinsonian drugs. Recent findings have reported that various plants like *Acorus calamus, Aloe vera, Azadirachta indica*, *Asparagus racemosus, Bacopa monnieri, Terminalia chebula,* and *Withania somnifera* are beneficial in preventing the neurodegeneration in PD and in AD. The active constituent's present in the plant, like Alkaloids, Polysaccharides, Vitamins, Tannins, Phenolic acid, and Flavonoids, are well-known antioxidants and can prevent oxidative damage to the neurons in the substantial nigra and other parts of the brain cortex. Apart from preventing neuronal damage, some plant constituents can enhance the synthesis and secretion of dopamine and acetylcholine, as required in PD and AD. In the current review, plants used for neurodegenerative diseases have been discussed along with their active constituents. Various phytoconstituents and derivatives of plant species showed the beneficial role of neurodegenerative diseases. Although several studies have reported the utility and value of herbal plants in the therapy of neurodegenerative disorders, there are still several unanswered questions regarding their efficacy in neurodegenerative disorders. Since the presence of bioactive compounds is the major concern for the therapy of neurological disorders to establish the exact mechanism of medicinal plants in neuronal damage and protection, further In-vivo pre-clinical studies should be designed to understand their actual mechanism in the prevention of neurodegeneration.

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