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ROLE OF MEDICINAL PLANTS IN NEURODEGENERATIVE DISEASES

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Abstract:

Neurodegenerative diseases (NDs) such as Alzheimer's disease (AD), Parkinson's Disease (PD), and Huntington's disease (HD) are progressive disorders characterized By selective neuronal loss, protein Misfolding and aggregation, oxidative stress, Mitochondrial dysfunction, and Neuroinflammation. Despite advances in Symptomatic treatments, disease-modifying Therapies remain limited. Medicinal plants And their phytoconstituents have long been Investigated for neuroprotective properties. This review synthesizes current preclinical And clinical evidence on key medicinal Plants—Withania somnifera (ashwagandha), Bacopa monnieri (brahmi), Curcuma longa (turmeric), Panax ginseng (red ginseng), Camellia sinensis (green tea), Coffea arabica (caffeine), and Sesamum indicum (sesamol)—highlighting mechanisms of Action, limitations, and future perspectives Including phytopharmaceutical Development, nanodelivery systems, and Pharmacogenomics. We also discuss Methodological considerations in herbal Research and propose priorities for Translating promising preclinical findings Into clinical therapies.

Keywords : *Neurodegeneration; Medicinal plants; Alzheimer's disease; Parkinson's disease; Huntington's disease; Phytochemicals; Neuroprotection.*

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INTRODUCTION:

Neurodegenerative diseases (NDs) are disorders characterized by progressive malfunction and death of neurons, the basic structural units of the brain and spinal cord that transmit signals to muscles, glands, and other nerves[1]. Since neurons rarely regenerate, their loss leads to irreversible damage. Neurodegeneration results from abnormal protein aggregation in neurons, environmental factors, impaired protein degradation, genetic mutations, mitochondrial dysfunction , and aging [2]. Protein misfolding and accumulation are key pathological features in several NDs, including α-synuclein in Parkinson's disease (PD) [3], amyloid-β (Aβ) in Alzheimer's disease (AD) [4].According to the World Health Organization (WHO), neurological disorders will become the second leading cause of death globally within two decades. Around one billion people are affected annually, including 50 million with epilepsy. Each year, nearly 6.8 million deaths are attributed to neurological illnesses [5], and about 60% of dementia cases occur in low- and middle-income countries. The number of dementia patients is expected to reach 82 million by 2030 and 152 million by 2050 [6].

Between 1990 and 2016, Parkinson's disease affected 2.5 million people, while Alzheimer's disease affected 36 million between 2006 and 2014, including 5.1 million Americans, of whom 200,000 were under 65 years [7]. NDs are often associated with depression and severely impact the quality of life [8,]. Physical manifestations include muscular weakness[9], paralysis, convulsions, and sensory loss[10]. Both acute and chronic NDs are linked to high morbidity and mortality rates [11].

Plants contain potent bioactive compounds that serve as lead molecules with minimal side effects [12]. Their phytochemical composition varies with geography and soil type [13]. Since ancient times, natural compounds have formed the basis of many Ayurvedic and Unani formulations [14].Common medicinal plants in South-East Asia include Murraya koenigii, Moringa oleifera, Ocimum sanctum, Terminalia arjuna, and Azadirachta indica [15]. Several herbs in the Indian subcontinent—such as Curcuma longa, Glycyrrhiza glabra, Huperzia serrata, Bacopa monnieri, and Withania somnifera—exhibit strong neuroprotective and memory-enhancing properties [16].Neuroprotection refers to the preservation of neuronal structure and function against injury or degeneration [17]. With global aging, traditional herbal remedies continue to play a major role in improving memory, learning, and cognitive function [18].

Alzheimer's Disease:

Alzheimer's disease is caused by a combination of genetic, lifestyle, and environmental factors, often inherited and linked to mutations in amyloid precursor protein (APP), presenilin-1 (PS1), and presenilin-2 (PS2) genes that lead to excess beta-amyloid (Aβ) accumulation[19]. Major risk factors include aging, family history, head injury, poor lifestyle, cardiovascular issues, and metabolic disorders such as type-2 diabetes[20]. Both central and peripheral immune responses contribute to disease progression (Figure 1)[21].

Pathophysiology: Aβ and Tau oligomers are toxic to the brain, leading to amyloid plaque and neurofibrillary tangle formation. Aβ is thought to trigger tau toxicity, resulting in paired helical filaments (PHF) and neuronal death, though some evidence suggests.tau damage may also occur independently. Abnormal phosphorylation causes tau to aggregate and disrupt normal brain function[21].

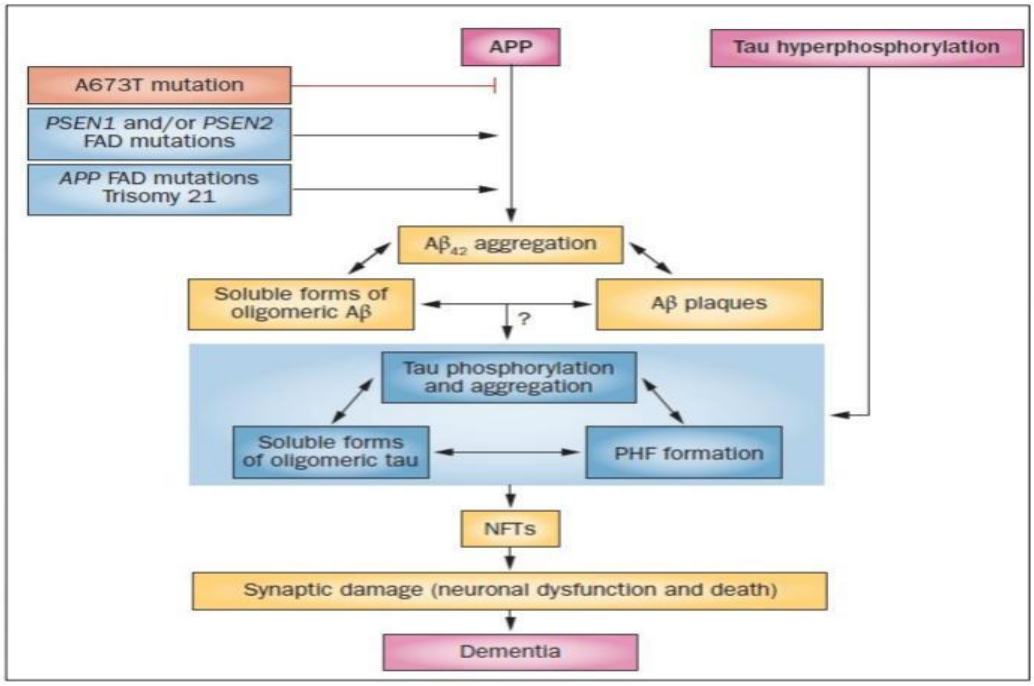

Fig.1 Hypothetical schematic of the amyloid cascade with nomadic suppression causing Dementia.

Medicinal plants used in the Treatment of AD:

1.Ashwagandha (Withania somnifera) - Ashwagandha, also called Indian ginseng or winter cherry, is used to support brain health, boost energy, and act as a nerve tonic[22]. It has antioxidant properties and contains bioactive compounds such as withanolides A–Y, withaferin A, withanone, sitoindosides VII–X[23], and alkaloids that help neutralize free radicals involved in Alzheimer's progression[24]. Withanamides A and C prevent Aβ25–35 fibril formation, while withanolide A promotes neurite growth and repairs axonal and dendritic damage in neurons[25]. In fruit flies, it reduces Aβ toxicity and extends lifespan. In humans, an 8-week trial with 50 mild cognitive impairment patients (300 mg twice daily) improved memory, focus, and decision-making compared to placebo[26].

2.Brahmi (Bacopa monnieri) – Brahmi, a wetland plant used in Ayurvedic medicine, contains bioactive compounds like bacosides, saponins, alkaloids, sterols, and polyphenols, which protect the brain by reducing oxidative stress, lipid peroxidation, and metal toxicity[26]. Human studies indicate that Brahmi (125–320 mg/day) enhances memory, attention, and logical reasoning in older adults and those with mild cognitive impairment, especially when combined with other supplements like Sideritis, astaxanthin, phosphatidylserine, or vitamin E[26].

3.Turmeric (Curcuma longa) – Turmeric, a plant of the Zingiberaceae family, contains curcuminoids (curcumin, demethoxycurcumin, bisdemethoxycurcumin, cyclocurcumin) and turmerone oil, which have anti-inflammatory, antioxidant, and neuroprotective properties. Curcumin reduces oxidative stress, neutralizes reactive oxygen species, lowers Aβ plaque formation, and improves cognitive function in Alzheimer's animal models. Higher doses are more effective, and combining curcumin with piperine enhances absorption and efficacy. Curcumin also binds to metals like copper, zinc, and iron, preventing oxidative damage and amyloid accumulation. Curcumin analogues have been developed to improve bioavailability and blood-brain barrier penetration, showing benefits in cognition and plaque reduction[26].

Parkinson's Disease :

Parkinson's disease is a neurodegenerative disorder caused by progressive loss of dopamine-producing neurons in the substantia nigra, often due to the accumulation of alpha-synuclein from genetic (e.g., PARK-1 mutations) or environmental factors. This leads to impaired basal ganglia function, resulting in motor symptoms like tremors, rigidity, and spasms, as well as non-motor issues such as cognitive decline, sleep disturbances, and mood changes. Neuronal loss

also occurs in the locus coeruleus, basal nucleus of Meynert, and dorsal motor nucleus of the vagus, with Lewy bodies present. Mechanisms include mitochondrial dysfunction, oxidative stress, inflammation, protein misfolding, and gut microbiota changes. The disease prevalence increases with age, affecting 1–2% of those over 60 and more than 4% over 80, often with 60–80% dopamine neuron loss before motor symptoms appear[21].

Pathophysiology:

Parkinson's is a chronic nervous system disorder causing resting tremors, slow movement, stiffness, and balance problems due to the loss of dopamine-producing cells in the substantia nigra.. Other neurotransmitters like serotonin and norepinephrine are also affected. Movement is regulated by direct and indirect pathways between the cortex, striatum, globus pallidus, substantia nigra, subthalamic nucleus, and thalamus. Dopamine modulates these pathways, and its deficiency reduces thalamic stimulation of the cortex, leading to varied motor symptoms. The severity and progression depend on whether tremors or balance problems dominate, with balance-related forms typically having worse outcomes[21].

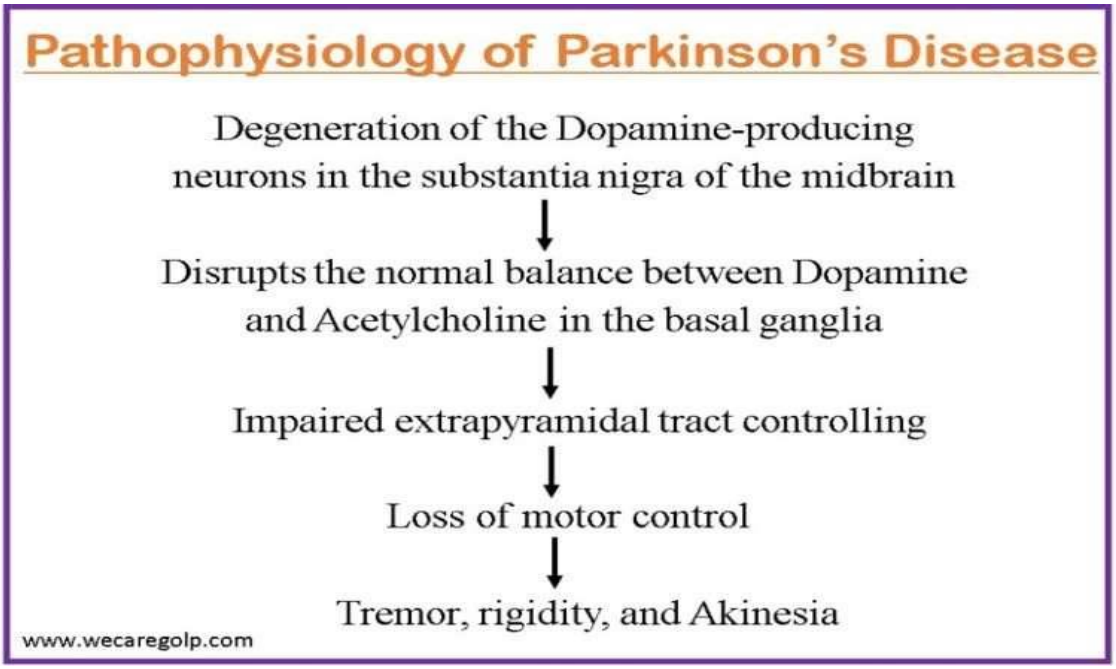

Fig. 2 Pathophysiology of Parkinson's Disease

Medicinal plants used in the Treatment of PD :

1.Red Ginseng (RG) - Brain inflammation in Parkinson's disease (PD) is driven by activated microglia, NF-κB, and increased inflammatory markers, contributing to dopamine neurondamage. Red ginseng (RG) and its components—ginsenosides Rb1, Rg1, Rd, Re, and gintonin—reduce inflammation by lowering IL-6, TNF-α, IL-1β, COX-2, NO, and PGE2, inhibiting microglial overactivation, and protecting dopamine-producing cells in the SNpc and striatum. RG components also prevent alpha-synuclein (a-syn) aggregation, improve coordination, and support TH cell survival, thereby alleviating PD-related brain inflammation and motor deficits[27].

2.Coffea arabica (Caffeine) – Caffeine (1,3,7-trimethylxanthine) is a widely consumed psychoactive substance found in coffee, tea, cocoa, soft drinks, and chocolate. In Parkinson's disease (PD), increased oxidative stress damages the nervous system by weakening antioxidant defenses and promoting lipid peroxidation. Activation of adenosine A2A receptors (A2AR) in the substantia nigra and striatum contributes to neuroinflammation, dopamine reduction, and PD symptoms. Caffeine and other A2AR blockers reduce neurotoxicity, improve motor function, and protect dopaminergic neurons by lowering oxidative stress and excitotoxicity[28].

3.Green tea (Camellia sinensis) – Green tea, made from the steamed and dried leaves of Camellia sinensis, is popular worldwide and contains catechins like epicatechin, epigallocatechin, epicatechin gallate, and epigallocatechin-3-gallate (EGCG). EGCG and L-theanine offer significant neuroprotective benefits in Parkinson's disease, with EGCG being the most

potent. Green tea polyphenols enhance antioxidant enzymes like catalase and SOD, reducing protein and lipid oxidation, including harmful byproducts such as protein carbonyl, 4-HNE, and MDA, which contribute to neuronal damage and synuclein aggregation. EGCG also binds iron to prevent free radical formation and may help degrade synuclein, limiting Lewy body formation. In cellular and animal models, EGCG reduces apoptosis, lowers synuclein levels, decreases MAO-B activity, and protects dopaminergic neurons, while L-theanine boosts ERK1/2 activity and neurotrophic factors like BDNF and GDF. Clinical studies show that consuming green tea with 550 mg polyphenols daily for three months enhances antioxidant defenses and reduces oxidative stress in Parkinson's patients [28].

Huntington's Disease:

Huntington's disease is a progressive neurodegenerative disorder caused by a mutation in the huntingtin gene (mtHTT) on chromosome 4 and follows an autosomal dominant inheritance, meaning a single copy of the faulty gene can cause the disease [21].

Pathophysiology:

Huntington's disease is an autosomal dominant disorder with complete penetrance, caused by mutations in the HTT gene, though the exact mechanisms of damage are not fully understood. The disease primarily affects medium spiny neurons (MSNs) in the striatum, with over 90% of GABAergic MSNs dying, leading to motor dysfunction. Other brain regions, including the cerebral cortex, cingulate gyrus, globus pallidus, cerebellum, amygdala, and hippocampus, are also affected, contributing to movement problems, mood disturbances, and cognitive deficits [29].

Medicinal plants used in the treatment of Huntington's disease :

1.Red ginseng – Huntington's disease (HD) is a progressive neurodegenerative disorder caused by abnormal CAG repeats in the huntingtin (HTT) gene on chromosome 4, leading to mutant HTT (mHTT) accumulation, chorea, cognitive decline, and mental health issues.Neuroinflammation, driven by overactive NF-kB signaling and elevated IL-6, IL-1β, and TNF-α, contributes significantly to HD pathology. Compounds like ginseng extract (RG), ginsenoside Rg1, gintonin, Rg3, and Rf reduce brain inflammation, microglial activation, and mHTT aggregation by modulating pathways such as NF-kB, MAPK, and p53-Bax. These compounds also protect neurons, reduce brain damage, and improve motor and cognitive functions, highlighting their potential therapeutic effects in HD [27]..

2.Ashwagandha (Withania somnifera) – Ashwagandha (Withania somnifera, WS) is a herb from the Solanaceae family widely used in Ayurvedic medicine for longevity and vitality, with antioxidant, anti-inflammatory, immune-modulating, stress-reducing, memory-enhancing, and anticonvulsant effects. Its active compounds, including sitoindosides VII–X and withaferin A, boost antioxidant enzymes and reduce lipid peroxidation, while also inhibiting inflammation via the complement system and lymphocyte proliferation[30]

3.Sesamum indicum Linn.(Sesamol) Sesamum indicum Linn. (Pedaliaceae), or sesame, is used as a health food in India and East Asia. Sesamol, a key component of sesame oil, exhibits strong antioxidant and antitumor activities by enhancing enzyme- and non-enzyme-based antioxidants and modulating the nitric oxide pathway, including reducing iNOS expression.Sesamol also protects against 3-NP-induced Huntington's disease-like behavioral, biochemical, and cellular changes in rodents, improves hippocampal neuron function, and reduces memory loss, oxidative stress, and inflammation. Additionally, it lowers nitrite production and iNOS expression in septic rat livers. These effects suggest sesamol as a potential candidate for HD treatment, though further mechanism-based studies are needed.[30]

Table 1. List of some medicinal plants used in Neurodegenerative Diseases.

Plant Name	Family	Common Name	Compound	Mode of Mechanism and Uses
Withania somnifera.	Solanaceae	Ashwagandha	Withanolides And Withanoles	Exhibit protection against neurons in vivo and in vitro along with in vivo antioxidant and anti-inflammatory activities
Bacopa monnieri	Plantaginaceae	Water Hyssop Or Brahmi	Bacopaside And bacoside	Bacopaside and bacoside enhances activities of

				mitochondrial, reduces accumulation of asynuclein, lessens apoptosis, revamps cognition along with Redox stabilization.
Curcuma longa	Zingiberaceae	Turmeric	Curcumin	Enhanced motor as well as cognitive disability instigated due to 3-NP accompanying a powerful antioxidant activity.
Panax ginseng	Araliaceae	Ginseng	Ginsenosides, Ginseng	Reduces calcium influx, restore homeostasis, acting as a psychic energizer, free radical generation, safeguards nerve cells from dysfunctioning of mitochondria.
Coffea arabica	Rubiacaae	Arabian coffee	Caffeine	Effective in case of loss of dopaminergic neurons in Parkinson's disease.
Camellia Sinesis	Theaceae	Green tea	Polyphenols, catechin [(EC), (ECG), & (EGCG)]	It acts as a redox stabilizer, impedes ROS-NO passage, Chelation of metal and also safeguards DA nerve cells found in the midbrain areas.
Sesamum indicum	Pedaliaceae	Sesame	Sesamol	Protection via nitric oxide mechanism i.e. repression of expression due to inducible nitric oxide synthase (iNOS).Instigation as a result of 3-NP provides protection from memory defacement, inflammation of hippocampus neurons, oxidative stress.

Future perspective:

The exact cause of many neurodegenerative diseases remains unclear. However, herbal medicines show promise due to their antioxidant, anti-inflammatory, and anti-cholinesterase properties. Diseases such as Alzheimer's, Parkinson's, and Huntington's share similar molecular mechanisms leading to cell death and inflammation, making phytochemicals potential therapeutic agents for their treatment.

The effectiveness of herbal medicines is often limited by poor pharmacokinetic properties, which can be improved through advanced drug delivery systems like nanoencapsulation. Nanoformulations enhance targeted delivery, reduce toxicity, and maintain therapeutic efficacy. However, the nanoencapsulation of most herbal medicines is still in its early stages, and methods like electrospinning and electrospraying for neurodegenerative disease treatment remain underexplored. Developing standardized phytopharmaceutical formulations containing multiple bioactive compounds can enhance therapeutic outcomes and bioavailability. Additionally, pharmacogenomic approaches can enable personalized treatments based on genetic variations. Collaboration between traditional medicine and modern science can further advance the development of effective plant-based therapeutics[31].

CONCLUSION:

Alzheimer's disease results from genetic, lifestyle, and environmental factors involving mutations in APP, PS1, and PS2 genes, leading to cortical damage and memory loss. Parkinson's disease arises from aging, genetic, and environmental causes, resulting in dopamine loss in the substantia nigra and symptoms affecting movement and cognition. Huntington's disease is caused by CAG repeat expansion in the huntingtin gene on chromosome 4, producing mutant huntingtin protein that leads to neuronal loss and

cognitive and motor decline. Overall, bioactive compounds from medicinal plants hold great promise for treating such disorders, with ongoing research supporting safe, nature-based, and personalized therapies.

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