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Review Paper

Herbal Remedies for Neurodegenerative Disorders: Alzheimer's Disease

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ABSTRACT

Alzheimer's disease (AD) is one of the main healthcare challenges of the twenty-first century, not only affecting millions of people's quality of life but also increasing the burden on the medical community, families, and society. It is a neurodegenerative disorder characterized by learning and cognitive dysfunction, behavioral turbulence, and memory loss and is a major cause of dementia, contributing to 50-60 % of dementia cases in patients above the age of 65. The major pathophysiological changes include accumulation of beta-amyloid plaques (Aβ), highly phosphorylated tau protein, neuroinflammation, GABA neurotransmission disruption, mitochondrial dysfunction, neuronal damage due to free radicals, and decreased concentration of acetylcholine (ACh) and butyrylcholine (BCh)[1]. We synthesize findings from pre-clinical and clinical studies involving key botanicals such as Ginkgo biloba, Bacopa monnieri, and Curcuma longa, salvia officinalis Crocus sativus Withania somnifera, Panax ginseng stablishing the potential of these phytomedicines to offer novel, multi-target therapeutic strategies for AD management.[2]

INTRODUCTION

Alzheimer's disease (AD) is a multifactorial neurodegenerative condition defined by progressive deterioration of cognition, memory loss, and functional impairment. Pathologically, AD is associated with extracellular amyloid-β (Aβ) plaque deposition, intracellular neurofibrillary tangles containing hyperphosphorylated tau protein, synaptic dysfunction, oxidative stress, and chronic neuroinflammation. These complex and interconnected pathological features limit the efficacy of single-target therapeutic agents currently approved for clinical use. [3]Natural products derived from medicinal plants have been incorporated into traditional healing systems, including Ayurveda, Traditional Chinese Medicine (TCM), and Western herbalism, for centuries to manage cognitive disorders and age-

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related decline. These herbal agents often comprise diverse phytochemical constituents such as polyphenols, flavonoids, alkaloids, and terpenoids, which exert antioxidant, anti-inflammatory, anti-amyloidogenic, and cholinesterase-inhibiting effects. Such pleiotropic actions lend themselves to addressing multiple facets of AD pathology concurrently. [4,5]For instance, Ginkgo biloba extract has been widely studied for its capacity to improve microcirculation and reduce oxidative stress in the brain, while Curcuma longa (turmeric) and Crocus sativus (saffron) contain bioactive compounds with demonstrated neuroprotective properties. Other herbs, including Bacopa monnieri, Ashwagandha (Withania somnifera), and several TCM formulations, have shown promise in preclinical models and early clinical investigations. [6,7]

PATHOPHYSIOLOGY

Alzheimer's disease (AD) is a progressive neurodegenerative disorder characterized by multiple inter-related pathological mechanisms. The central feature of AD is the accumulation of amyloid-β (Aβ) peptides, formed due to abnormal cleavage of amyloid precursor protein (APP). These peptides aggregate into toxic oligomers and extracellular plaques, leading to synaptic dysfunction and neuronal damage [8]Another hallmark is tau pathology. Tau protein becomes hyperphosphorylated, detaches from microtubules, and forms neurofibrillary tangles (NFTs) inside neurons. Tau pathology strongly correlates with neuronal loss and cognitive decline .[9]Neuroinflammation plays a crucial role, where activated microglia and astrocytes release pro-inflammatory cytokines and reactive oxygen species, further worsening neuronal injury [10].Additionally, oxidative stress and mitochondrial dysfunction impair cellular energy metabolism and promote neuronal apoptosis. Early synaptic loss and neurotransmitter imbalance, particularly cholinergic deficit, are closely associated with memory impairment [11].Overall, AD pathophysiology involves a complex interaction of amyloid toxicity, tau pathology, inflammation, oxidative stress, and synaptic dysfunction, supporting the need for multi-target therapeutic approaches.

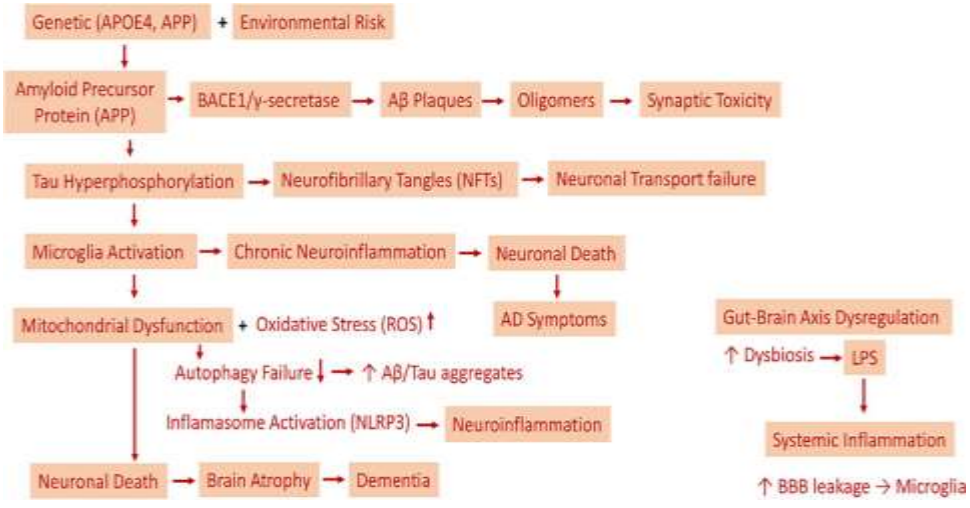

Fig.1. This schematic integrates AD pathways [12]

THE NEED FOR THE MULTI-TARGET THERAPY:-

Today's medicines for Alzheimer's mainly help with symptoms for a short time. They don't actually stop or slow down the disease itself.
Alzheimer's is very complex, so focusing on just one target or one mechanism hasn't been enough. That's why many single-target drugs haven't worked well in real patients.
Scientists are shifting toward treatments that can act on multiple parts of the disease at once-kind of like using a team approach instead of relying on one person. This multi-target strategy offers more hope for truly slowing or modifying the disease.

The Promise of Herbal Medicine :-

Herbal remedies or phytomedicines are naturally rich in a variety of different bioactive compounds, for example, flavonoids, terpenoids, and alkaloids, that can interact with multiple biochemical targets. This characteristic makes them inherently suitable for tackling the complex, multi-factorial pathology of AD. Historically, many traditional systems have used botanicals to enhance memory and cognitive function, providing a basis for modern pharmacological investigation.

TARGETING CORE PATHOLOGIS WITH HERBAL EXTRACT: -

1)GINKGO BILOBA: -

Common name: Maidenhair tree [13]

Active Ingredient: -

The standardized leaf extract, EGb 761®, contains:
• Flavonoid glycosides (22–27%) – mainly quercetin, kaempferol, isorhamnetin.
• Terpene lactones (5–7%) – including ginkgolides (A, B, C) and bilobalide.
• Low ginkgolic acids (<5 ppm, minimized due to toxicity).
These compounds are believed to contribute to antioxidant and neuroprotective properties. [13]

Mechanism of Action:-

Ginkgo biloba extract (EGb 761®) may influence Alzheimer's pathology through multiple pathways:
Antioxidant effects: Scavenges free radicals and reduces oxidative stress linked to neuronal damage.
Anti-inflammatory activity: Reduces inflammation via antagonism of platelet activating factor (PAF).
Neuroprotection & mitochondrial support: Protects neuronal tissue and supports energy metabolism.
Modulation of neurotransmission: Some evidence suggests effects on neurotransmitter systems.
Potential effects on amyloid and tau pathways: Preclinical studies suggest effects on amyloid β aggregation and tau phosphorylation, though clinical significance remains unclear. [14]

Clinical Efficacy:-

Meta-analyses of randomized controlled trials show modest improvements in cognition, activities of daily living, and overall clinical rating with standardized EGb 761® (typically 120–240 mg/day).
Benefits are most noticeable over 22–26 weeks of treatment in patients with dementia and, in some studies, those with neuropsychiatric symptoms.
EGb 761® was generally well-tolerated with no major safety concerns. [15]

2) BACOPA MONNIERI: -

Common name: - Brahmi, widely used in Ayurvedic medicine for memory and cognitive enhancement.

Active Ingredient: -
The major bioactive constituents are bacosides (especially bacoside A and bacoside B), a group of triterpenoid saponins.
Other minor constituents include flavonoids and alkaloids that contribute to neuroprotective activities. [16]

Mechanism of Action:-

Bacopa monnieri exhibits multiple biological mechanisms relevant to Alzheimer's disease, including:
Antioxidant activity: Neutralizes reactive oxygen species and inhibits lipid peroxidation, protecting neurons from oxidative damage.
Cholinergic modulation: Inhibits acetylcholinesterase and increases acetylcholine levels, enhancing cholinergic transmission associated with learning and memory.
Anti-amyloidogenic actions: Reduces aggregation and deposition of amyloid-β peptides, a key pathological hallmark of Alzheimer's.

Anti-inflammatory effects: Suppresses microglial activation and pro-inflammatory cytokines, which contribute to neurodegeneration.

Mitochondrial and cell survival pathways: Evidence suggests improvement in mitochondrial function and modulation of signaling (e.g., GSK-3β/Wnt/β-catenin) reducing apoptosis and tau pathology in experimental models. [17]

Clinical Efficacy:-

Human trials in Alzheimer's disease show mixed and inconclusive evidence. A systematic review of randomized controlled trials found that studies varied widely in Bacopa dose (125–500 mg twice daily) and cognitive outcomes, and overall quality of evidence was very low due to small sample sizes and high risk of bias.
Some trials reported statistically significant improvement in specific cognitive scores compared with placebo or donepezil, but heterogeneity and methodological limitations prevent definitive conclusions. [18]

3) CURCUMA LONGA :-

Common Name:- Curcumin
Active ingredient:- The key bioactive compound of Curcuma longa is curcumin, a lipophilic polyphenol that is primarily responsible for most of turmeric's pharmacological effects. [19]

Mechanism of Action:-

Curcumin exerts multiple mechanisms relevant to Alzheimer's disease pathology:
Antioxidant activity: Curcumin scavenges reactive oxygen species and reduces oxidative stress in neuronal cells.
Anti-inflammatory effects: It modulates neuroinflammatory pathways and inhibits pro-inflammatory cytokines.

Anti-amyloid effects: Curcumin can bind to amyloid-β (Aβ) plaques, inhibit their aggregation, and promote plaque disaggregation, reducing one of the pathological hallmarks of Alzheimer's disease.
Tau and Aβ modulation: Preclinical studies indicate curcumin can attenuate tau hyperphosphorylation and enhance Aβ clearance.
Other molecular targets: Curcumin also affects metal chelation, acetylcholinesterase inhibition, and multiple Alzheimer's-related signaling pathways. [20]

Clinical Efficacy:-

A 24-week randomized, double-blind, placebo-controlled clinical trial in mild-to-moderate Alzheimer's patients using oral curcumin (C3 Complex®) showed it was generally well-tolerated, but definitive cognitive efficacy was not strongly demonstrated in the primary outcomes.
Reviews of clinical and preclinical studies suggest that while curcumin improves cognition and reduces biomarkers related to inflammation and oxidative stress in animal models, translating these effects to humans remains challenging due to poor bioavailability and limited robust human trials. [21]

4) WITHANIA SOMNIFERA :-

Common Name:- Withania somnifera is commonly known as Ashwagandha, Indian ginseng, or Indian Winter Cherry. It is a traditional Ayurvedic herb used for improving cognitive function, memory, and stress resilience.[22]

Active Ingredients:-

The primary bioactive constituents are withanolides (such as withaferin A, withanolide A and D), withanosides, and steroidal lactones, which are believed to mediate its neuroprotective and adaptogenic effects. These compounds exhibit antioxidant, anti-inflammatory, and neuroplasticity-enhancing properties. [23]

Mechanism of Action:-

Withania somnifera acts through several mechanisms relevant to Alzheimer's disease pathology:
Antioxidant and free-radical scavenging — reduces oxidative stress in neuronal tissue, which is linked to neurodegeneration.
Anti-inflammatory effects — decreases levels of pro-inflammatory cytokines and glial activation, mechanisms implicated in Alzheimer's progression.
Anti-amyloidogenic actions — evidence from transgenic mouse models shows reduction in β-amyloid (Aβ) plaque burden, a hallmark of Alzheimer's pathology.
Synaptic protection and neuroplasticity — suggested by promotion of neurite outgrowth and potential modulation of key signaling targets (e.g., TrkB, GSK-3β, BACE-1) in molecular analyses.
Enhanced Aβ clearance — some preclinical studies show Ashwagandha up-regulates liver and brain receptors (such as LRP) that may facilitate removal of amyloid from the brain. [23,24]

Clinical Efficacy:-

Early clinical evidence: A systematic review of clinical studies found that Withania somnifera supplementation improved performance on cognitive tasks, executive function, attention, and reaction time in older adults with mild cognitive impairment or related conditions. However, the clinical evidence is limited and heterogeneous, involving various populations and cognitive endpoints. [22]
Preclinical support: Several animal studies (e.g., in Alzheimer's mouse models) demonstrate improved spatial memory and reduced anxiety/depressive-like behavior along with decreased amyloid plaques following Ashwagandha extract administration. [24]

CONCLUSION

Alzheimer's disease is a multifactorial neurodegenerative disorder involving oxidative stress, neuroinflammation, amyloid-β accumulation, tau pathology, and neurotransmitter imbalance. Herbal agents such as Ginkgo biloba, Bacopa monnieri, Curcuma longa, and Withania somnifera exhibit multi-target neuroprotective actions that address these interconnected pathological mechanisms. Evidence from experimental studies suggests that these herbs possess antioxidant, anti-inflammatory, anti-amyloidogenic, and cognition-enhancing properties.
Although preclinical findings are encouraging, clinical evidence remains limited and inconsistent, largely due to variability in study design, dosage, and formulation. Therefore, while these herbal agents show potential as adjunct or supportive therapies in Alzheimer's disease, further well-designed clinical trials with standardized extracts are required to confirm their efficacy and safety.

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