

Phytotherapy in Dementia: Multi-Targeted Approaches to Neuroprotection and Cognitive Preservation

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Received: 03 May 2025 / Received in revised form: 16 July 2025, Accepted: 17 July 2025, Published online: 25 July 2025

Abstract

Dementia is a progressive neurodegenerative disorder marked by a decline in cognitive function, with Alzheimer's disease being the most prevalent form. Current pharmacological treatments offer only limited symptomatic relief, prompting increasing interest in alternative strategies. Emerging evidence suggests that the plant-derived compounds may play a role as potential adjuncts in the management of dementia. This review explores the neuroprotective properties of selected medicinal plants, including *Ginkgo biloba* (enhancing cerebral blood flow and mitochondrial activity), *Curcuma longa* (inhibiting amyloid-beta aggregation), *Glycyrrhiza glabra* (improving cholinergic transmission), *Panax ginseng* (modulating oxidative stress and amyloid toxicity), and *Camellia sinensis* (reducing oxidative damage and supporting cognitive function). By synthesizing findings from preclinical and clinical studies retrieved from PubMed, Scopus, and Web of Science, this review highlights how these botanicals act on multiple pathological pathways associated with dementia, including oxidative stress, neuroinflammation, amyloid deposition, and cholinergic dysfunction. Although clinical outcomes remain inconsistent due to variations in study design, dosage, and extract standardization, these plants exhibit promising therapeutic potential with favourable safety profiles. The review emphasizes the need for standardized formulations, well-designed clinical trials, and personalized treatment protocols to establish their efficacy and integrate them effectively into dementia care.

Keywords: Dementia, Alzheimer's disease, Neuroprotective, *Ginkgo biloba*, *Panax ginseng*, *Curcuma longa*

Introduction

Dementia is a general term for a range of progressive neurological disorders that significantly affect memory, cognition, behaviour,

and the ability to perform daily activities. According to the World Health Organization, dementia is not a single disease but an umbrella term for various conditions, including Alzheimer's disease (AD), frontotemporal dementia (FTD), Lewy body dementia, and vascular dementia (VD). While aging is the most significant risk factor, dementia is not a normal consequence of aging (Anonymous, 2019; Arvanitakis *et al.*, 2019). The U.S. National Institute of Neurological Disorders and Stroke highlights that dementia is characterized by severe impairment in at least two cognitive domains, such as memory, language, or reasoning, that interfere with day-to-day life (Sheet, 2018).

Historically, the concept of dementia predates the modern medical era. Though the diseases that cause dementia have likely existed since the dawn of humanity, it was only in 1906 that Alois Alzheimer described the condition that now bears his name. Prior to this, cognitive decline in old age was often dismissed as 'senility.' Early medical texts and literary works made vague references to memory loss and behavioural changes in the older peoples, but these were not understood in clinical terms. The early 20th century witnessed breakthroughs in neuropathology that clarified the nature of degenerative dementias, laying the foundation for modern dementia research (Berrios, 1990; Vatanabe *et al.*, 2020). The prevalence of dementia has increased significantly in recent decades, primarily due to longer life expectancies. In 2017, approximately 50 million people worldwide were living with dementia a sharp rise from 35.6 million in 2010 (Sathianathan & Kantipudi, 2018; Gogoi *et al.*, 2023). Projections indicate that this number will grow to 82 million by 2030 and 152 million by 2050, with the greatest burden expected in low- and middle-income countries. Alzheimer's disease is the most common form, followed by vascular and mixed dementias. About 10% of cases involve mixed dementia, often a combination of AD and VD or FTD (Karantzoulis & Galvin, 2011; Prince *et al.*, 2015).

Epidemiological studies have shown that dementia is one of the top five causes of death globally. In 2013 alone, dementia was responsible for 1.7 million deaths, a significant increase from 0.8 million in 1990. Life expectancy after diagnosis varies, typically ranging from 3 to 12 years, depending on age at onset, type of dementia, and other comorbidities. For instance, vascular dementia generally carries a poorer prognosis than Alzheimer's disease (Duthey, 2013; Aburas, 2022; Li *et al.*, 2024b).

Despite advances in diagnosis and symptomatic treatment, dementia remains incurable. Current therapies may temporarily alleviate symptoms or slow disease progression but cannot reverse

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brain damage. The absence of curative treatments underscores the urgent need for further research into the biological, genetic, and environmental factors underlying this devastating condition.

In recent years, there has been growing interest in the use of medicinal plants for the management of dementia, particularly AD, due to their multifaceted pharmacological properties and favourable safety profiles (Gregory *et al.*, 2021; Carpio-Vargas *et al.*, 2023; Mounir *et al.*, 2023). Several traditional medicinal systems, including Ayurveda and Traditional Chinese Medicine, have long utilized plant-based therapies to enhance memory and cognitive function. Notable plants that have been investigated for their neuroprotective effects include *Ginkgo biloba*, *Panax ginseng*, *Curcuma longa*, *Glycyrrhiza glabra*, *Camellia sinensis* (Mantle *et al.*, 2000; Chen *et al.*, 2021; Shahzan *et al.*, 2022; Bulovina *et al.*, 2024; Kadiri & Tiwari, 2025).

These plants exhibit various mechanisms of action relevant to dementia therapy, such as antioxidant, anti-inflammatory, and anticholinesterase activities, as well as modulation of neurotransmitter systems. For instance, *Ginkgo biloba* extract has demonstrated efficacy in slowing cognitive decline and improving memory (Howes & Perry 2011; Singh *et al.*, 2022; Villegas *et al.*, 2022; Tripathi *et al.*, 2024). Likewise, *Panax ginseng*, *Curcuma longa*, *Glycyrrhiza glabra*, and *Camellia sinensis* have shown cognitive-enhancing potential through various neuropharmacological pathways (Lopresti, 2017; Jakaria *et al.*, 2018; Jo, 2018; Cachón-Rodríguez *et al.*, 2024; Nguyen *et al.*, 2024).

These ethnomedicinal plants, used individually or in polyherbal formulations, provide a natural and potentially safer alternative or adjunct to conventional therapies. Their diverse bioactive compounds provide a promising foundation for the development of novel therapeutics targeting the complex pathophysiology of dementia. Therefore, exploring plant-derived compounds remains a vital area of research in the search for more effective, accessible, and holistic treatment options for dementia.

Materials and Methods

Data Collection

The literature for this article was collected through a comprehensive search using major scientific databases, including Google Scholar, PubMed, Scopus, and Web of Science. These search engines were employed to retrieve peer-reviewed articles, clinical studies, reviews, and ethnopharmacological reports related to dementia and the use of medicinal plants. Keywords used in the search included: “dementia,” “Alzheimer’s disease,” “medicinal plants,” “neuroprotection,” “cholinesterase inhibitors,” “antioxidants,” “anti-inflammatory agents,” “*Ginkgo biloba*,” “*Panax ginseng*,” “*Curcuma longa*,” “*Glycyrrhiza glabra*,” “*Camellia sinensis*”.

Role of Herbs in the Management of DEMENTIA

Ginkgo Biloba

Ginkgo biloba, often regarded as a “living fossil,” is one of the oldest surviving tree species, with its origin dating back over 200

million years. It is the sole extant member of the Ginkgoaceae family and has been widely cultivated in China for centuries due to its medicinal value (Jacobs & Browner, 2000; Crane, 2019; Yilmaz *et al.*, 2023). Traditionally, Ginkgo extracts have been used to manage ailments like poor circulation, vertigo, fatigue, and tinnitus (Cybulska-Heinrich *et al.*, 2012). In recent decades, it has gained global recognition for its neuroprotective potential, particularly in age-related cognitive decline and dementia (Çakar *et al.*, 2022; Noor-E-Tabassum *et al.*, 2022).

Phytochemical studies reveal that *G. biloba* contains two major groups of active constituents responsible for its therapeutic properties: flavonoids and terpene lactones. Flavonoids, including kaempferol, quercetin, meletin, and isorhamnetin, exert antioxidant effects and modulate signaling pathways involved in memory and learning (Nakagawa & Yamashita, 2022; Karim *et al.*, 2024; Gamal *et al.*, 2025). Terpene lactones such as ginkgolides A, B, C, and bilobalide (**Figure 1**) exhibit neuroprotective activity by antagonizing platelet-activating factor (PAF) receptors and preserving mitochondrial integrity. Additionally, minor constituents like ginkgotoxin and bilobol have specific biological effects, including neurotoxicity and antibacterial action, respectively (Biemacka *et al.*, 2023; Yahyaeva *et al.*, 2023; Gamal *et al.*, 2025).

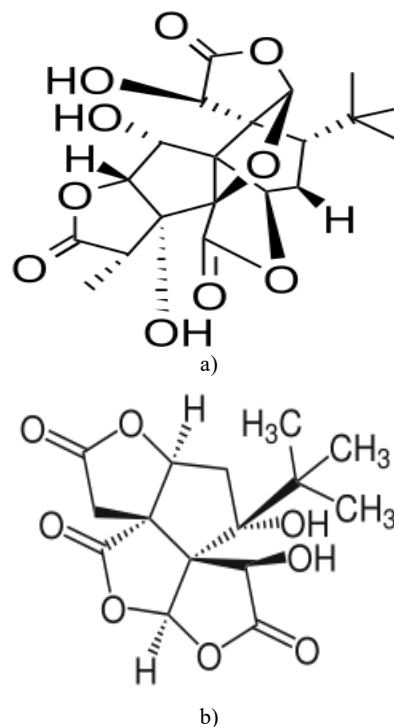


Figure 1. Ginkgolide and Bilobalide

Pharmacologically, *G. biloba* exhibits antioxidant, anti-inflammatory, vasoregulatory, and antiplatelet properties (Gamal *et al.*, 2025). These effects collectively contribute to improved cerebral blood flow, reduced oxidative stress, and enhanced synaptic plasticity, all of which are vital for maintaining cognitive health (Duman & Eken, 2022). The standardized extract EGb 761, containing 24% flavonoids and 6% terpene lactones, is the most

widely studied formulation. It has shown promise in preclinical and clinical studies for preserving cognitive function, protecting neurons from ischemic and toxic damage, and enhancing behavioural outcomes in both AD and VD (Farooqui, 2012). Due to its multifaceted mechanisms and favourable safety profile, *G. biloba* remains one of the few phytomedicines internationally recommended for the management dementia. However further standardized trials are needed to confirm its long-term efficacy.

Curcuma Longa

Curcuma longa L., commonly known as turmeric, is a perennial herb from the Zingiberaceae family. Widely used as a culinary spice and traditional remedy across Asia, particularly in Ayurvedic and Chinese medicine, turmeric has been applied for centuries to treat various ailments, including inflammation, digestive issues, liver disorders, and wound healing. Recent research has focused on its neuroprotective potential, particularly in dementia and AD (Mishra & Palanivelu, 2008; Farooqui, 2018; Tian *et al.*, 2025).

The primary bioactive constituents of turmeric are curcuminoids, including curcumin (**Figure 2**), demethoxycurcumin, and bisdemethoxycurcumin (Sharma & Sharma, 2022). Curcumin (diferuloylmethane) is the major and most studied compound, responsible for turmeric's bright yellow color. It possesses a symmetrical structure with two ferulic acid residues joined by a seven-carbon chain containing a β -diketone moiety. Curcumin also exists in keto and enol tautomeric forms depending on environmental pH. Additionally, turmeric contains essential oils such as turmerone, atlantone, and zingiberene (Oliveira Filho *et al.*, 2021).

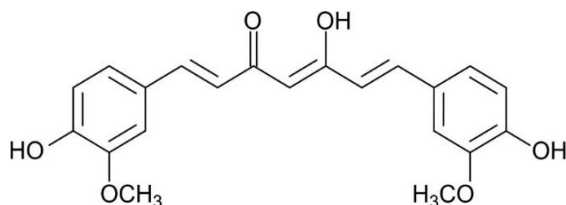


Figure 2. Curcumin

Curcumin exhibits a wide range of pharmacological effects including antioxidant, anti-inflammatory, antimicrobial, anticancer, and anti-amyloidogenic activities (Jyotirmayee *et al.*, 2023; Islam *et al.*, 2024). Its antioxidant properties stem from its phenolic groups and β -diketone moiety, which scavenge free radicals and modulate oxidative stress pathways. It also inhibits key inflammatory mediators such as TNF- α , IL-1 β , and NF- κ B, contributing to its neuroprotective profile (Azzini *et al.*, 2024; Genchi *et al.*, 2024).

Preclinical studies show that curcumin can reduce β -amyloid (A β) accumulation, inhibit tau hyperphosphorylation, modulate synaptic plasticity, and protect mitochondria. It crosses the blood-brain barrier (BBB), binds to A β plaques, and enhances their clearance via phagocytosis. Curcumin also improves memory and reduces oxidative and inflammatory damage in animal models of AD. While clinical data remain limited and inconclusive, curcumin demonstrates potential as an adjunctive therapeutic for managing dementia, especially AD. Improved formulations enhancing

bioavailability are under investigation to support future clinical applications (Chen *et al.*, 2018; Shrifi-Rad *et al.*, 2020).

Glycyrrhiza Glabra

Glycyrrhiza glabra, commonly known as licorice, is a valued medicinal plant from the Fabaceae family, long revered in traditional healing systems such as Ayurveda and Traditional Chinese Medicine. Historically, the roots of *G. glabra* have been used to treat respiratory ailments, digestive disorders, and chronic inflammatory conditions (Batiha *et al.*, 2020; Wahab *et al.*, 2021). More recently, scientific investigations have focused on its neuroprotective potential, particularly in relation to neurodegenerative diseases such as dementia and Alzheimer's disease (Paudel *et al.*, 2020; Sarkar *et al.*, 2023; Verma *et al.*, 2024). The therapeutic efficacy of *G. glabra* is largely attributed to its rich array of bioactive compounds, including glycyrrhizin, glycyrrhetic acid, glabridin, liquiritin, and isoliquiritigenin (Kaur *et al.*, 2013). These phytochemicals possess robust antioxidant, anti-inflammatory, and anti-apoptotic properties that directly counteract key pathological features of dementia.

Among its constituents, glycyrrhizin (**Figure 3**) has shown notable potential in protecting the brain against vascular dementia and other neurodegenerative insults. It exerts neuroprotective effects by reducing oxidative stress, inhibiting lipid peroxidation, and preserving neuronal structure in key brain areas, such as the hippocampus, which plays a crucial role in learning and memory (Guo *et al.*, 2016; Li *et al.*, 2024a). Glycyrrhizin also supports cognitive health by attenuating neuroinflammatory responses, notably by modulating the expression of pro-inflammatory cytokines such as TNF- α , IL-6, and IL-1 β (Song *et al.*, 2025).

Furthermore, it has been observed to protect neuronal cells from glutamate-induced toxicity, a key contributor to excitotoxicity in neurodegeneration, by enhancing cell viability and regulating apoptosis-related genes, such as Bcl-2 and Bax (Yang *et al.*, 2013). Additionally, other flavonoids, such as glabridin, have demonstrated the ability to cross the blood-brain barrier and protect neurons from amyloid-beta toxicity, a hallmark of Alzheimer's pathology.

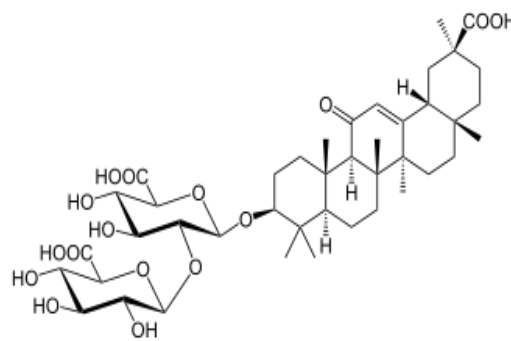


Figure 3. Glycyrrhizin

Animal model studies further suggest that *G. glabra* extract can enhance memory and learning by improving cholinergic transmission and inhibiting acetylcholinesterase activity (Dhingra *et al.*, 2004). Although more extensive clinical studies are needed,

current preclinical evidence and centuries of traditional usage collectively highlight *G. glabra* as a promising natural candidate for dementia management. Its multitargeted action, safety profile, and accessibility support its inclusion in the ongoing exploration of plant-based interventions for neurodegenerative disorders.

Panax Ginseng

Panax ginseng, commonly known as ginseng, is a perennial medicinal plant traditionally used in East Asia, particularly in China and Korea, as a general tonic to enhance vitality and promote longevity (Potenza *et al.*, 2023). Among its various species, *Panax ginseng* C.A. Meyer has received the most attention for its pharmacological properties, especially in relation to cognitive health. Ginseng contains several bioactive constituents including ginsenosides (notably Rg1, Rg2, Rg3), gintonin, polysaccharides, and amino acids, many of which have demonstrated neuroprotective and cognition-enhancing effects in both preclinical and clinical settings (Huang *et al.*, 2019; Liu *et al.*, 2023).

Ginsenosides (**Figure 4**), the primary active compounds, are known to influence multiple pathological pathways relevant to dementia. Ginsenoside Rg3 has been shown to reduce amyloid-beta ($A\beta$) accumulation and improve cognitive function by modulating oxidative stress, neuroinflammation, and apoptosis (Yang *et al.*, 2021; Hwang *et al.*, 2025). Rg1, another well-studied ginsenoside, is believed to protect neurons from $A\beta$ -induced neurotoxicity and oxidative damage, partly through regulation of the p38 MAPK signaling pathway. Both Rg1 and Rg3 also affect cholinergic neurotransmission by modulating acetylcholine receptor activity and inhibiting acetylcholinesterase, mechanisms closely associated with memory and learning (Kim *et al.*, 2018; Jiang *et al.*, 2025).

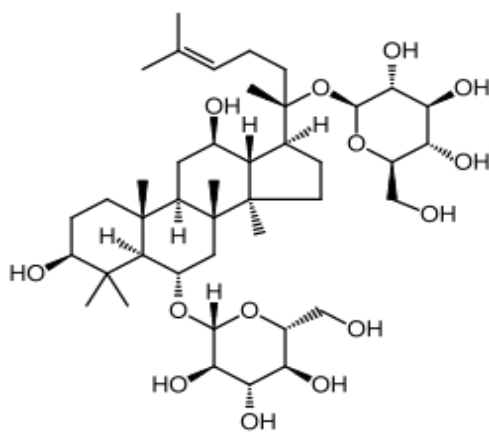


Figure 4. Ginsenoside

Emerging evidence suggests that ginseng extracts improve cognitive performance in individuals with subjective memory impairment (SMI), mild cognitive impairment (MCI), and early AD (58). Ginseng has also demonstrated benefits in VD models by enhancing cerebral blood flow, reducing glial activation, and regulating apoptotic proteins such as Bcl-2 and Bax (Kim *et al.*, 2013). Moreover, gintonin, a recently identified component of

ginseng, contributes to neuroprotection through the activation of lysophosphatidic acid (LPA) receptors, which influences synaptic plasticity.

Although clinical trials evaluating the efficacy of ginseng in AD have produced mixed results, often due to small sample sizes and methodological limitations, its multi-targeted actions make it a promising candidate for integrative dementia therapy. The broad spectrum of neuroprotective activities, coupled with its traditional use and favourable safety profile, supports the continued exploration of *Panax ginseng* as a complementary approach in dementia management.

Camellia Sinensis

Camellia sinensis Kuntze, commonly known as green tea, is among the most widely consumed beverages worldwide and has been recognized for its diverse health-promoting properties, including its neuroprotective potential. Rich in bioactive compounds, particularly polyphenols such as epigallocatechin-3-gallate (EGCG) (**Figure 5**), green tea exhibits potent antioxidant, anti-inflammatory, and anti-apoptotic activities that contribute to cognitive preservation and modulation of neurodegenerative diseases (Aboulwafa *et al.*, 2019; Prasanth *et al.*, 2019).

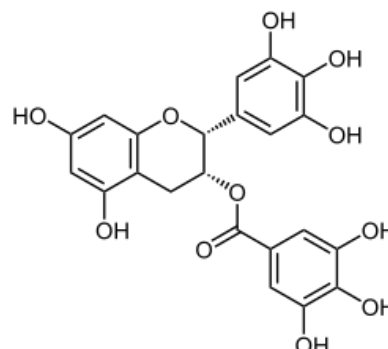


Figure 5. Epigallocatechin-3-gallate

Several studies highlight the role of green tea and its constituents in counteracting key pathological processes involved in dementia, including oxidative stress, amyloid-beta ($A\beta$) toxicity, and cholinergic dysfunction. EGCG and other catechins regulate ROS production, restore mitochondrial membrane potential, and enhance superoxide dismutase activity. L-theanine, a unique amino acid found in green tea, provides neuroprotection by inhibiting ERK/p38 and nuclear factor kappa-light-chain-enhancer of activated B cells (NF- κ B) signaling, thereby reducing $A\beta$ 42-induced memory loss and neuronal death. Additionally, green tea extract improves cognitive function by enhancing the expression of membrane metalloendopeptidase (MME), which facilitates $A\beta$ degradation (Doganoglu & Erbas, 2021; Afzal *et al.*, 2022).

Experimental models demonstrate that green tea ameliorates cognitive impairments in AD, VD, and diabetes-induced cognitive decline. Intrahippocampal and systemic administration of green tea constituents improved learning and memory in rats and protected hippocampal neurons from $AlCl_3$ and ischemia-induced injury. Furthermore, green tea extract inhibits acetylcholinesterase and beta-secretase two critical enzymes implicated in AD pathology

suggesting a dual-action mechanism that enhances cholinergic signaling while impeding A β plaque formation.

Clinical and epidemiological studies also support the cognitive benefits of green tea. While some studies show a strong association between green tea intake and a reduced dementia risk, the results remain partially inconclusive, indicating the need for larger, well-controlled human trials. Nevertheless, green tea's affordability, accessibility, and safety profile make it a viable candidate for dietary interventions aimed at preventing age-related cognitive decline.

C. sinensis shows promise as a multifunctional neuroprotective agent with potential applications in dementia prevention and therapy. Its capacity to modulate multiple molecular targets central to neurodegeneration justifies further exploration in both experimental and clinical settings.

Conclusion

Medicinal plants such as Ginkgo biloba, turmeric, and ginseng exhibit promising neuroprotective effects against dementia through various mechanisms. While preclinical evidence is compelling, clinical applications require standardized extracts and rigorous trials. Their multi-target action and safety profile make them ideal complementary therapies. Future research should focus on optimal formulations and personalized treatment approaches. Integrating these botanicals with conventional care could provide a more comprehensive approach to dementia management. Ultimately, plant-based therapies may bridge current gaps in neurodegenerative disease treatment.

Acknowledgments: The authors are thankful to the Management of RL Jalappa College of Pharmacy, Sri Devaraj Urs Academy of Higher Education and Research, Tamaka, Kolar, Karnataka, India for providing facilities to carry out this work.

Conflict of interest: None

Financial support: None

Ethics statement: None

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