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A COMPREHENSIVE REVIEW ON PHYTOCHEMISTRY AND PHARMACOLOGICAL ACTIVITIES OF *GINKGO BILOBA*

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ABSTRACT

Ginkgo biloba, often referred to as the maidenhair tree, is among the oldest living plant species and has been widely utilized in traditional medicine, especially within Chinese health practices. Herbal remedies remain significant in contemporary medicine because of their availability, healing properties, and impact on drug development. This review gathers information from multiple scientific databases such as Google Scholar, PubMed, and Scopus, employing conventional literature review methods to outline its historical context, classification, structure, chemical composition, and medicinal properties. *Ginkgo biloba* is classified under the Ginkgoaceae family and is noted for its dioecious traits, fan-shaped foliage, and distinctive vein structure, while it physically appears as a tall, deciduous tree with unique leaf formation. The plant holds significant bioactive compounds like flavonoids (quercetin, kaempferol) and terpenoids (ginkgolides and bilobalide), which contribute to antioxidant, anti-inflammatory, neuroprotective, and vasodilatory properties. From a pharmacological perspective, it has shown considerable promise in boosting cognitive abilities, enhancing memory, and treating neurological conditions. Additionally, its therapeutic effectiveness in cerebrovascular and cardiovascular disorders like ischemic stroke, intracerebral hemorrhage, hypertension, myocardial ischemia, and atherosclerosis is ascribed to its vascular and hemorheological characteristics, especially the inhibition of platelet-activating factor (PAF) by ginkgolides, which leads to decreased platelet aggregation and enhanced microcirculation. It further safeguards against ischemia-reperfusion injury by lowering oxidative stress and inflammation and demonstrates moderate efficacy in peripheral vascular issues such as intermittent claudication. In summary, *Ginkgo biloba* shows potential as a medicinal plant, but additional research is necessary to standardize its formulations and confirm its clinical effectiveness.

KEYWORDS: *Gingko Biloba*, Phytochemical, Pharmacological, Morphological.

1. INTRODUCTION

Herbs play a crucial role in modern medicine. Even today, about 60–70% of medicines come from herbs, either directly or by using them as a model to create new drugs. People all over the world trust herbs for their healing power. They play a role not only in treating different illnesses but also in supporting overall health and well-being.[1] Herbal remedies are gaining more attention today because many plants naturally support heart health and may help manage common cardiovascular problems. Garlic (*Allium sativum*) is often used for its ability to help lower blood pressure and cholesterol.[2] Turmeric, especially its active compound curcumin, is also valued for its strong antioxidant and anti-inflammatory effects, which can help protect the heart.[3] Traditional herbs like Arjuna (*Terminalia arjuna*) have been used in Ayurveda for centuries to strengthen the heart and improve overall cardiac function. [4] Hawthorn (*Crataegus oxyacantha*) and *Ginkgo biloba* are known for improving blood circulation and easing symptoms related to mild heart failure.[5,6] Other herbs, including ginger, green tea, fenugreek, flaxseed, ashvagandha and guggul, help reduce cholesterol levels and oxidative stress, both important in preventing heart disease. All these herbs, when used alongside each other, can offer gentle support for the heart and may help improve the effectiveness of conventional medical approaches.[7]

The Ginkgoaceae, a gymnosperm family originating in the Mesozoic Era, is currently represented by a single extant species, *Ginkgo biloba*. This solitary status has earned it the frequent designation of a 'living fossil.' Although the family once encompassed multiple genera that formed widespread forests, identifying this ancient diversity is challenging. Leaf morphology is highly variable and thus an unreliable taxonomic indicator; instead, paleobotanists rely on distinct wood and bark anatomy to differentiate the diverse ginkgo species that thrived in prehistoric times.[8]

The Ginkgoaceae family, is now found naturally only in eastern China. Although its natural habitat is restricted, the "Maidenhair Tree" has become economically significant worldwide as a hardy ornamental street tree, valued for its ability to withstand pollution and its brilliant golden fall colors. Uniquely among gymnosperms, these dioecious trees have thick short shoots and distinctive fan-shaped (flabelliform) leaves with open, forked veins. Their reproductive structures are also peculiar: males produce catkin-like clusters containing paired microsporangia, while females have paired, upright ovules that mature into seeds without a protective ovary, enclosed by a fleshy outer layer (sarcotesta) and a hard inner shell. This taxonomically distinct gymnosperm, sharing only a distant evolutionary link with conifers like pines and firs, has flourished in North America and Europe since its reintroduction centuries ago.[9,10]

Ginkgo biloba (Maidenhair tree) is one of the oldest and most widely used herbal medicines in the world, with a history of therapeutic application dating back thousands of years in ancient China. Renowned for treating memory loss and respiratory ailments, the herb is rich in bioactive compounds, specifically flavonoids and terpene lactones, which exhibit antioxidant, antiplatelet, and hemorrheological properties. Today, it is a top-selling pharmaceutical and dietary supplement globally, marketed for managing cerebrovascular insufficiency, peripheral circulatory deficits, and the progression of Alzheimer’s disease. While popularly used as an alternative therapy for conditions like claudication, clinical consensus suggests its efficacy in these areas is marginal.[11]

2. MATERIALS AND METHODS

This study is based on an extensive review of previously published literature sourced from Google Scholar, PubMed, Scopus, Springer, Nature, ResearchGate, and standard reference books.

3. Taxonomical identification

3.1. Microscopical description

Microscopic examination of *Ginkgo biloba* leaves during the spring, late summer and autumn growth stages reveals that the petiole is differentiated by a greater percentage of lignified vessel elements and rare starch grains, while the lamina is identified by its wavy-walled epidermal cells, anisocytic stomata and calcium oxalate clusters. The size of these crystals, the degree of vessel lignification, and the morphology of the cell walls all change predictably as the plant ages. Additionally, there is an increase in an unnamed yellow globular substance that is proven by chemomicroscopy to be free of phenolics, oils, or starch. These results provide a structural blueprint and developmental chronology that are crucial for ginkgo sample quality assessment and botanical identification.[12] The anatomical composition of this specimen probably *Ginkgo biloba* is marked by the extensive occurrence of phenolic compounds in the chlorenchyma, vascular bundle sheaths, and epithelia of secretory cavities. Its petiole changes from a plano-convex outline near the base to a concave-convex form toward the tip, and the stem during early secondary growth shows a circular cross-section covered by a robust, single-layered epidermis. Within the vascular cylinder, there are slender rays and a uniform xylem made up of tracheids, fibers, and parenchyma of small diameter. On the outside, the leaves are protected by a thick, enduring layer of waxy tubes that mostly stays intact until the shift into late summer and fall{Citation}

3.2. Morphological description

Table 1: Morphological characteristics of *Ginkgo biloba.*

S. No.	Parts	Description	Shape	Size	Ref.
1	Leaves	Ginkgo leaves can be easily recognised due to their distinctive two-lobed shape and unique veining that consistently splits without connecting.	The leaves are fan-shaped which are flat, and irregularly notched.	5 to 10 cm	[12,13]
2	Seed	The seed coat of *Ginkgo biloba* consists of a fleshy, orange-yellow sarcotesta, a hard, whitish sclerotesta, and a membranous endotesta. It changes from a tip that is smooth and prone to cracking to a rough base, displaying an inner layer with opaque and golden-brown areas.	The seed of the *Ginkgo biloba* is shaped like an ellipse or an egg, characterized by a smooth, pointed micropylar end and a broader, rounded chalazal base.	Measuring between 1.4 and 2.5 cm long and 1.1 to 1.6 cm wide.	[15,16]
3	Branches	Ginkgo branches exhibit a dimorphic arrangement consisting of elongated, leaf-spaced growths and shorter, gradually growing spur shoots that produce reproductive structures.	Ginkgo branches are dimorphic, featuring linear long shoots and short, knobby spur shoots with clustered foliage		[17]
4	Root	The root system of *Ginkgo biloba* consists of a deep taproot and a diarch vascular cylinder shielded by a developing exodermis early in its growth. Anatomically, it is characterized by specialized phi cells and Casparian bands that offer mechanical support and control the flow of nutrients.	The *Ginkgo biloba* root is secured by a deep, straight taproot and displays a diarch symmetry (two-poled) in its internal vascular structure.		[18,19]

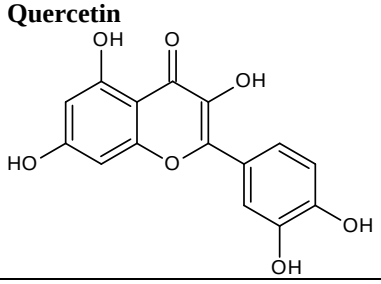
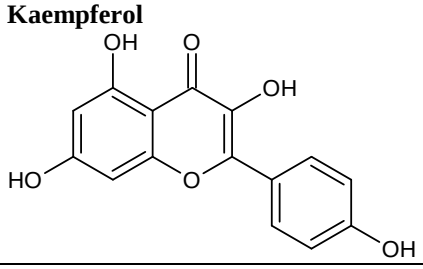
4. Chemical constituents

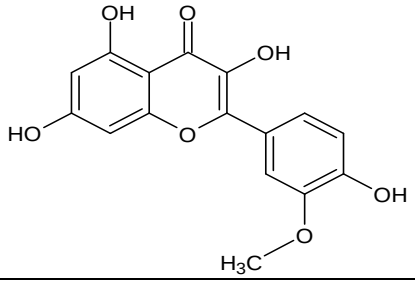
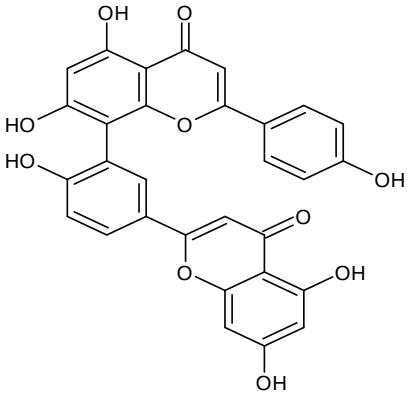
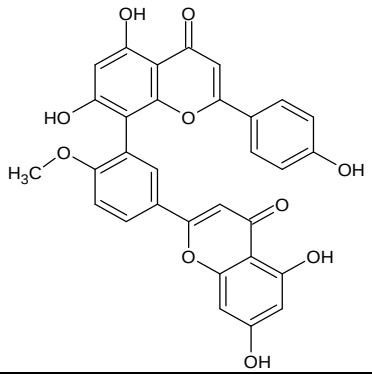
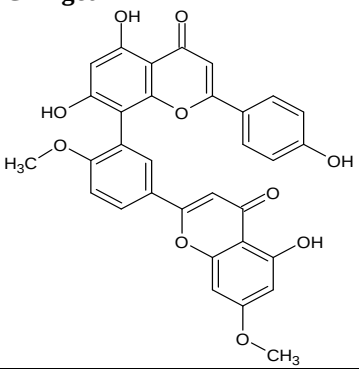
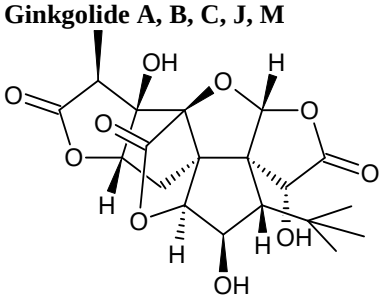
Ginkgo biloba is abundant in complex chemicals and comprises a diverse array of bioactive compounds from various chemical categories. The leaves of *Ginkgo biloba* are abundant in a wide range of phytochemicals that fall into different chemical categories. Di-trans-poly-cis-octadecaprenol is the main polyprenol discovered. Quercetin, kaempferol, isorhamnetin, rutin, luteolin, delphinidin, and myricetin are notable flavonoids. In addition to nitrogen-containing acids, the leaves also contain organic acids, mostly benzoic acid derivatives such ginkgolic acid. There are other biflavonoids such as sciadopitysin, ginkgetin, isoginkgetin, amentoflavone, bilobetin, and 5′-methoxybilobetin. Waxes, steroids, sugars, catechins, proanthocyanidins, phenols, aliphatic acids, cardanols, 2-hexenal, and rhamnose are also present in the leaves. The terpenoid components of *Ginkgo biloba* can be found in the leaves, roots, and bark. The roots primarily consist of triterpenes in the form of sterols. Sesquiterpenes, with bilobalide as a representative, are found in the leaves, roots, and bark. Additionally, diterpenes, known as ginkgolides, are plentiful in these parts and include ginkgolide M alongside ginkgolides A, B, C, and J. These terpenoids are regarded as the key bioactive components of *Ginkgo biloba* and are responsible for many of its therapeutic effects. The seed of gingko biloba include fatty alcohols, long-chain alkanes, fatty acids, carboxylic acids, sterols, diterpenes, triterpenes, flavonoids, and their derivatives.

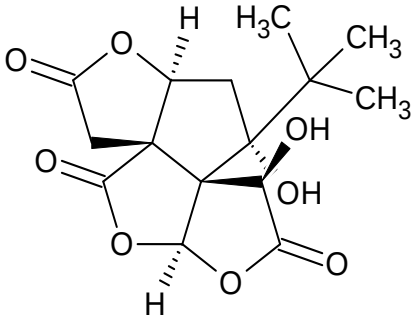
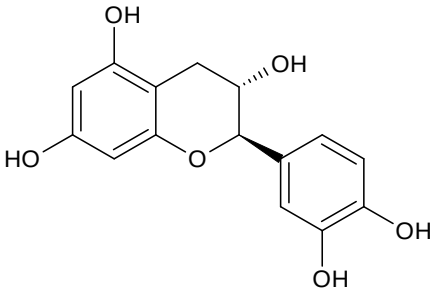
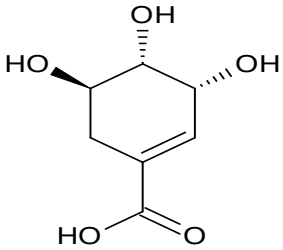
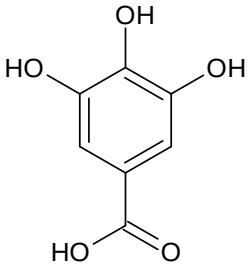
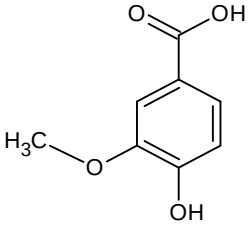
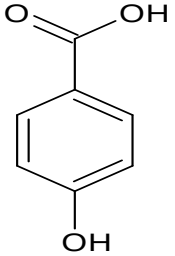
Many of the therapeutic benefits of *Ginkgo biloba* are supported by the chemical constituents' broad spectrum of common pharmacological activities, which primarily include antioxidant and anti-inflammatory properties. These components have been shown to have wide antimicrobial qualities, such as antibacterial, antiviral, and antifungal effects, as well as anticancer and antitumor activities, operating through anti-proliferative, pro-apoptotic, antimetastatic, and chemoprotective mechanisms. Their relevance in neurodegenerative, cardiovascular, and hepatic illnesses is further supported by the substantial neuroprotective, cardioprotective, and hepatoprotective effects that have been reported.

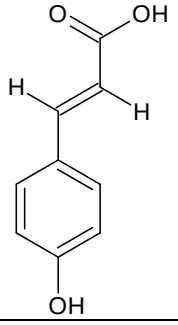
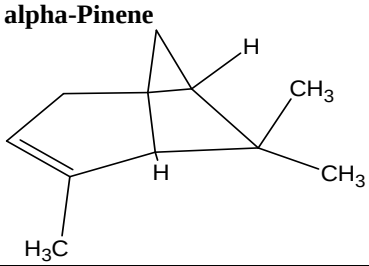
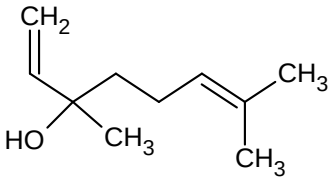
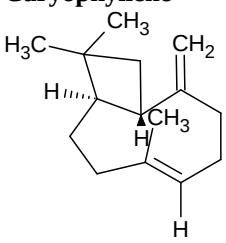
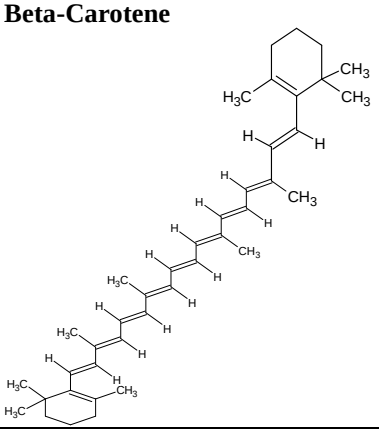
The literature also emphasises organ-protective, immunomodulatory, anti-aging, analgesic, and gastroprotective effects, as well as antidiabetic, hypolipidemic, anti-atherosclerotic, and antithrombotic activities. These findings collectively highlight the diverse therapeutic potential of *Ginkgo biloba* constituents, as summarised in (Table 2).[14-16]

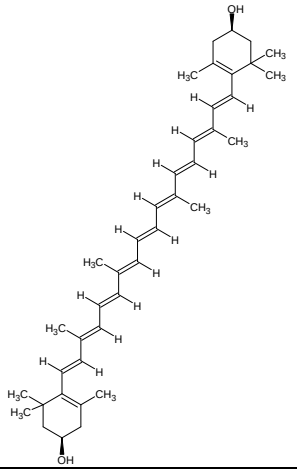
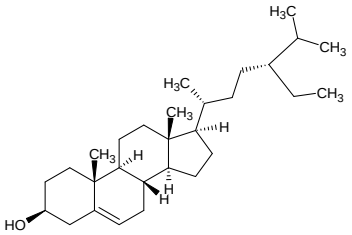
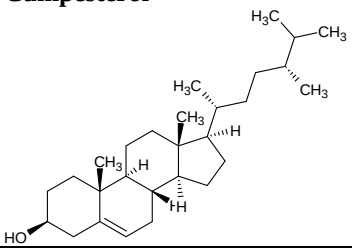
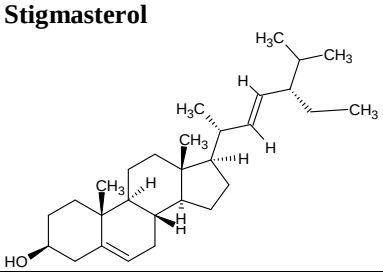
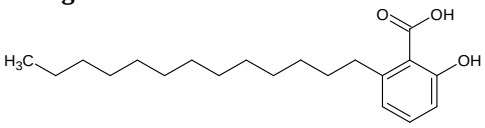
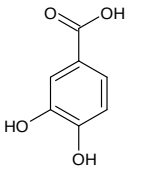
Table 2: Chemical constituents of *Ginkgo biloba.*

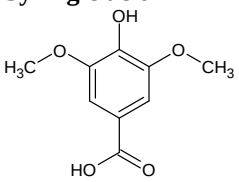
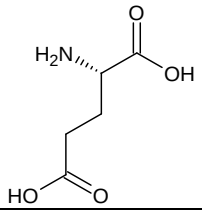
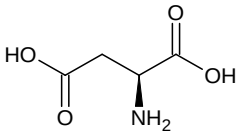
SI. No.	Name of the compound	Nature of the compound	M.F	M.W. g/mol	Activity
1	**Quercetin** 	Flavonoid	$C_{15}H_{10}O_7$	302.23	Anti-cancer, Hepatoprotective, Anti-inflammatory, Anti-oedema, Anti-hemorrhagic, Antioxidant. [17-19]
2	**Kaempferol** 	Flavonoid	$C_{15}H_{10}O_6$	286.23	Anticarcinogenic, Anti-inflammatory, Antibacterial, Antifungal, Anti-diabetic, Antiprotozoal. [20,21]

3	**Isorhamnetin** 	Flavonoid	$C_{16}H_{12}O_7$	316.26	Antioxidant, Antiviral, Anticancer, Antimicrobial, Anti-Tuberculosis, Anti-inflammatory, Hepatoprotective. [22,23]
4	**Amentoflavone** 	Biflavonoid	$C_{30}H_{18}O_{10}$	538.464	Anti-tumour, Anti-inflammatory, Anti-oxidant, Anti-virus, and Anti-fungal, Anti-senescence, Analgesic activities. [24,25]
5	**Bilobetin** 	Biflavonoid	$C_{31}H_{20}O_{10}$	552.48	Antiviral, Antiherpes, Therapeutic agents for HCC treatment, in LIHC treatment. [26,27] Hypolipidemic, Insulin-sensitizing. [34]
6	**Ginkgetin** 	Biflavonoid	$C_{32}H_{22}O_{10}$	~566.52	Anti-cancer, [35] Neuroprotective, Anticancer, Cardioprotective, Anti-inflammatory, Antiviral, diabetic nephropathy. Antibacterial, Antiparasitic. [30-32]
7	**Ginkgolide A, B, C, J, M** 	Terpenoids	GinkgolideJ: $C_{20}H_{24}O_{10}$	424.4 g	Anti-inflammatory, Anticancer, Anti-atherosclerosis, Anti-atherombosis, Hepatoprotective, Neuroprotective. [39]

8	**Bilobalide** 	Terpenoids	$C_{15}H_{18}O_8$	326.3	Neuroprotective effects,[40] Anti-fungal, Anti-inflammatory, Anti-oxidant, Antihyperlipidemic, Anti-proliferative
9	**Catechin** 	Flavonoid	$C_{15}H_{14}O_6$	290.26	Antimicrobial, Anticancer, Antihypertensive, Anticoagulant, Antiulcer, Organ protection. [35-38]
10	**Shikimic acid** 	Organic Acid	$C_7H_{10}O_5$	174.15	Antibacterial, Antiviral, Antioxidant, Anti-inflammatory, Hypolipidemic, Bone protective, Skin protective, Neuroprotective, Antidiabetic.[45]
11	**Gallic acid** 	Phenolic Acid	$C_7H_6O_5$	170.12	Antibacterial, Antiviral, Antioxidant, Anti-inflammatory, Antimicrobial, Anticancer. [46]
12	**Vanillic acid** 	Organic Acid	$C_8H_8O_4$	168.148	Antibacterial, Antiviral, Antioxidant, Anti-inflammatory, Antimicrobial, Anticancer, Anti-allergy, Anti-diabetic. [41,42]
13	**p-Hydroxybenzoic acid** 	Organic Acid	$C_7H_6O_3$	138.12	Antiviral, Antioxidant, Anti-inflammatory, Antimicrobial, Anticancer, Anti-allergy, Antialgal, Antimutagenic, Antiestrogenic, Hypoglycemic, Nematicidal, Anti-platelet aggregating. [43,44]

14	**p-Coumaric acid** 	Phenolic acid	$C_9H_8O_3$	164.160	Anticancer, Antioxidant,[51] Antibacterial, Anti-inflammatory. [52]
15	Long-chain polyisoprenoid alcohols	Polyprenols	H-$(C_5H_8)_n$-OH		Anti-tumour, Anti-hepatitis C virus, Anti-HIV effects, Treats Hypertension, High cholesterol, Diabetes, Gout, Lupus and other immune function disorders, Anti-anaemia. [53]
16	**alpha-Pinene** 	Bicyclic Monoterpene	$C_{10}H_{16}$	~136.23	Antioxidant, Anti-inflammatory, Anti-carcinogenic, Antimicrobial, Apoptotic, Antimetastatic, Antibiotic, Hypoglycemic. [54] [55]
17	**Linalool** 	Monoterpene	$C_{10}H_{18}O$	154.25	Anti-inflammatory, Antimicrobial, Helps in preventing Alzheimer's disease, Anti-cancer, Antioxidant, Sedative, Anxiolytic, Anticonvulsant. [50-52]
18	**Caryophyllene** 	Sesquiterpene	$C_{15}H_{24}$	204.357	Antibacterial, Antioxidant, Gastroprotective, Anxiolytic, Anti-inflammatory, Anticancer, Cytotoxic, Antimicrobial, Hypolipidemic, Neuroprotective, and Cardioprotective. [53,54]
19	**Beta-Carotene** 	Carotenoids	$C_{40}H_{56}$	536.888	Anti-inflammatory, antioxidant, and anti-tumour, Provitamin A activities. [61,62]

20	**Lutein** 	Carotenoids	$C_{40}H_{56}O_2$	568.871	Anti-cancer, Antioxidant, Anti-cataract, Antidiabetic, Hepatoprotective, Cardioprotective, Anti-arthritics, [63] Anti-inflammatory. [58,59]
21	**Beta-Sitosterol** 	Phytosterols	$C_{29}H_{50}O$	414.718	Anti-inflammatory, Inducing Apoptosis, Chemoprotective, Hypocholesterolemic Activity, Angiogenic Effect, Genotoxicity Effect, Analgesic, Anthelminthic, Anti-oxidant, Prostatic Cancer Treatment, Neuroprotection, Anti-diabetic. [60,61]
22	**Campesterol** 	Phytosterols	$C_{28}H_{48}O$	400.691	Antioxidant, Anti-inflammatory, Anticancer, Treatment of Rheumatoid Arthritis. [69,70]
23	**Stigmasterol** 	Phytosterols	$C_{29}H_{48}O$	412.702	Antimicrobial, Antioxidant, Anti-tumour, Anti-inflammation, Anti-diabetic. [65,66]
24	**Ginkgolic acid C13:0** 	Ginkgolic acids	$C_{20}H_{32}O_3$	320.47	Anti-microbial, Anti-inflammatory, Anti-tumor,[73] Antidiabetic, Anti-bacteria, Anti-virus, Anti-fibrosis, and Neuroprotection. [74]
25	**Protocatechuic acid** 	Natural phenolic acid	$C_7H_6O_4$	154.121	Antioxidant, Anti-inflammatory, Antihyperglycemic, Neuroprotective, Pro-apoptotic, Anti-proliferative effects [75] Antibacterial, Anticancer, Antiulcer,

					Antidiabetic, Antiageing, Antifibrotic, Antiviral, Analgesic, Antiatherosclerotic, Cardiac, Hepatoprotective, Neurological, Nephroprotective[76]
26	**Syringic acid** 	Natural phenolic acid	$C_9H_{10}O_5$	198.174	anti-oxidant, antimicrobial, anti-inflammatory, antiendotoxic, neuro and hepatoprotective, prevention of diabetes, CVDs, cancer, cerebral ischemia. [71,72]
27	**Glutamic acid** 	Amino Acid	$C_5H_9NO_4$	147.130	Anticancer agent[79]
28	**Aspartic acid** 	Amino Acid	$C_4H_7NO_4$	133.103	Antimicrobial, [80] Immune support, [81] Neurotransmitter. [82]

5. Pharmacological Activity

5.1. Ischemia

Ginkgo biloba and its bioactive constituents exhibited significant neuroprotective effects across multiple ischemic models, including cerebral ischemia/reperfusion, focal ischemia, cerebral ischemic stroke, permanent ischemia, and perinatal hypoxic-ischemic injury. In vivo studies demonstrated that ginkgetin, ginkgo peptide extract, and 6-hydroxykynurenic acid reduced ischemia-induced neuroinflammation by suppressing the TLR4/NF-κB pathway, decreasing pro-inflammatory cytokines, and attenuating oxidative stress through reduced malondialdehyde levels and enhanced superoxide dismutase activity. In focal ischemia/reperfusion models, G. biloba polysaccharides significantly improved neurological deficits, reduced infarct volume, increased MPO and anti-inflammatory cytokine IL-10 levels, and suppressed IL-1β, TNF-α, nitric oxide, and lipid peroxidation. Mechanistically, G. biloba activated key survival and antioxidant pathways, including Akt, ERK1/2, PKA, EGFR, and PI3K/Akt-mediated Nrf2 and CREB signaling, while inhibiting caspase-3–dependent apoptosis. Additionally, standardized G. biloba extracts enhanced neurogenesis, improved motor and neurological function, and reduced extrinsic apoptotic signaling in permanent ischemia models, while attenuating NLRP3 inflammasome activation in perinatal hypoxic-ischemic injury. Collectively, these findings highlight the broad, multi-target neuroprotective potential of G. biloba in ischemic brain injury which are listed in (Table 3).[83-88]

5.2. Cerebrovascular and neurodegenerative disorders of the CNS

Ginkgo biloba demonstrates significant neuroprotective and cardioprotective effects across a broad spectrum of neurovascular and cardiovascular disorders. In chronic cerebral hypoperfusion, hemorrhagic stroke, intracerebral

hemorrhage, and Alzheimer's disease models, G. biloba reduced neuroinflammation, glial activation, neuronal apoptosis, oxidative stress, and microvascular damage, thereby improving cognitive and vascular outcomes. In cardiovascular disease–associated cerebrovascular conditions, including permanent and transient middle cerebral artery occlusion and acute ischemic stroke, G. biloba preparations improved motor and neurological function by attenuating oxidative stress, calcium overload, mitochondrial dysfunction, and neuronal cell death. Moreover, G. biloba exerted cardioprotective effects in cardiac remodeling, cardiotoxicity, viral myocarditis, abdominal aortic aneurysm, diabetic cardiomyopathy, atherosclerosis, thrombosis, hypertension, ventricular hypertrophy, and myocardial ischemia reperfusion injury through modulation of Akt/Nrf2, CaN/NFAT, and inflammatory signaling pathways, improvement of endothelial and lipid homeostasis, and inhibition of apoptosis and platelet aggregation. These findings support the multi-target therapeutic potential of *Ginkgo biloba* in neurovascular and cardiovascular diseases as presented in (Table3).[83-101]

NEOPLASTIC DISEASES

Ginkgo biloba and its active compounds show widespread anti-cancer effects in various cancer models. Specifically, EGb 761 influences genes involved in steroid production and aromatase, thereby lowering estrogen in postmenopausal breast cancer. Ginkgolic acids, on the other hand, slow pancreatic tumor growth by affecting fat-building pathways. In pancreatic cancer, ginkgolide B boosts gemcitabine's effectiveness and inhibits tumor growth and survival by reducing PAFR and NF-κB signals. Ginkgetin fights prostate cancer by blocking STAT3 signaling. For lung cancer, EGb 761 and ginkgetin reduce proliferation, migration, and invasion by altering AKT/p38 MAPK and STAT3 pathways, disrupting cellular balance, and triggering apoptosis and autophagy. Selenium-enriched *Ginkgo biloba* polysaccharides induce cell death in bladder cancer by affecting proteins that control apoptosis, while EGb 761 halts gastric cancer by causing cell cycle arrest, promoting apoptosis, improving chemotherapy response, and overcoming resistance through ERK1/2 pathway inhibition. Furthermore, ginkgolic acid limits liver cancer by curbing cell changes, migration, and invasion, inhibiting MMP-2/MMP-9 and HGF/c-Met signals. *Ginkgo biloba* extracts also inhibit melanoma by reducing new blood vessel formation and PI3K/Akt/HIF-1α/VEGF signaling, while promoting cell death. Collectively, the available data indicate that G. biloba constituents represent promising therapeutic options for cancer therapy as summarized in (Table 3).[102-111]

GASTROINTESTINAL DISORDER

Compounds derived from *Ginkgo biloba* have demonstrated significant gastroprotective and anti-inflammatory properties in various models of gastrointestinal disorders which are illustrated in (Table 3). Specifically, in models of peptic and gastric ulcers, bilobalide, EGb, and EGb 761 effectively reduced mucosal damage caused by ethanol and ammonia. They achieved this by suppressing pro-inflammatory cytokines, oxidative stress, and apoptosis, while simultaneously protecting gastric mucus, sulfhydryl content, and antioxidant defenses through the regulation of MAPK/NF-κB signaling pathways. Furthermore, in models of colitis and inflammatory bowel disease, EGb 761 and bilobalide helped to alleviate intestinal inflammation. This was accomplished by inhibiting macrophage activation and NF-κB-driven inflammatory responses, ultimately promoting the healing of the intestinal lining. In addition, EGb 761 was found to reduce serum amylase and lipase levels and improve histopathological outcomes in acute pancreatitis models, suggesting a reduction in pancreatic damage. Taken together, these results underscore the therapeutic promise of *Ginkgo biloba* components for managing gastrointestinal inflammatory conditions.[112-116]

NEUROTOXICOLOGICAL DISORDERS

Ginkgo biloba and its derivatives have demonstrated considerable neuroprotective benefits in models of neurotoxicological disorders, effectively shielding neurons from chemically induced damage. Specifically, EGb has been shown to lessen the neurotoxic and dementia-inducing effects of sodium arsenite by bolstering antioxidant defenses and mitigating oxidative injury to neurons. In cases of aluminum-induced neurotoxicity, EGb helped alleviate biochemical and histopathological changes in the brain. Furthermore, *Ginkgo biloba* extract (GBE) enhanced behavioral outcomes, normalized lipid profiles and membrane fluidity, and maintained the structural integrity of neuronal membranes, with significant protective effects observed in the hippocampus and cortex, primarily through antioxidant pathways. Regarding cisplatin-induced neurotoxicity, EGb 761 lowered nitrosative stress, as indicated by reduced levels of nitrate and glutathione, although it did not impact lipid peroxidation. Moreover, in models of fluoride-induced neurotoxicity, EGb (administered at doses of 50–200 mg/kg) substantially improved learning and memory, suggesting it can help reverse cognitive impairments associated with fluoride exposure. Taken together, these results underscore the promising therapeutic applications of G. biloba compounds for experimental neurotoxicological conditions as summarised in (Table 3).[117-119]

METABOLIC DISORDERS

Ginkgo biloba and its derivatives showed notable anti-obesity, anti-diabetic, and hepatoprotective benefits in metabolic illness animals. In addition to improving glucose and lipid homeostasis and maintaining pancreatic β-cell function in diabetes models, EGb and associated compounds decreased oxidative stress, increased lipolysis, and repressed adipogenesis through AMPK activation and inhibition of important adipogenic regulators. Compounds derived from G. biloba reduced hepatic steatosis, inflammation, and liver damage in nonalcoholic fatty liver disease, maintained the integrity of the intestinal barrier, and reduced lipotoxicity, in part by activating antioxidant pathways like Nrf2. All of these results point to the potential for G. biloba components to treat metabolic diseases, details of which are provided in (Table 3).[126,127]

ACUTE NEUROLOGICAL DISORDER

In models of neurological disorders, *Ginkgo biloba* and its standardized extracts, as listed in (Table 3), have demonstrated extensive neuroprotective and neurorestorative properties. Specifically, EGb 761 substantially mitigated early brain damage subsequent to subarachnoid hemorrhage. This was achieved by diminishing neurological deficits, cerebral edema, and neuronal programmed cell death, through the activation of the Akt/Bcl-2 pathway and the suppression of Bax, caspase-3, oxidative stress, inflammation, and NLRP3/TXNIP signaling. Within neuropathic pain models, EGb 761 provided pain relief by reducing oxidative stress and pro-inflammatory cytokines, concurrently augmenting the expression of μ-opioid receptors. In models of epilepsy, standardized G. biloba leaf fractions and EGb decreased seizure intensity, improved cognitive capabilities, and preserved hippocampal neurons by inhibiting mTOR signaling and downregulating P-glycoprotein, with a synergistic effect observed when administered with phenytoin. Furthermore, EGb 761 alleviated tardive dyskinesia by reducing vacuous chewing movements, restoring the Bax/Bcl-2 ratio, bolstering antioxidant defenses, and elevating BDNF levels. In spinal cord injury models, *Ginkgo biloba* isoflavones facilitated neurological recovery by upregulating Bcl-2, promoting neuronal survival, reducing glial scarring, and enhancing locomotor function. Collectively, these results highlight the therapeutic promise of G. biloba derivatives for a variety of neurological conditions.[128,129]

NEUROINFLAMMATION

Ginkgo biloba extract (EGb) showed strong anti-inflammatory effects in neuroinflammation models (Table 3). EGb (100 mg/L) treatment of microglial cells in vitro significantly reduced microglial inflammatory responses by transrepressing the expression of pro-inflammatory cytokine genes, suggesting a strong modulatory effect on microglia-mediated neuroinflammation.[130]

SKELETAL DISORDER

Ginkgo biloba and its standardized extract (EGb) demonstrate significant anti-osteoporotic and chondroprotective effects in skeletal disorder models. Ginkgo constituents enhance osteoblast differentiation by upregulating VEGF expression in MC3T3-E1 cells and promote osteogenic while inhibiting adipogenic differentiation of bone marrow mesenchymal stem cells (BMSCs). In vivo studies further show that EGb improves periodontal bone support, mandibular cortical thickness, and bone mass in ovariectomy-, ageing-, and glucocorticoid-induced osteoporosis models. These effects are associated with modulation of the OPG/RANKL ratio, enhanced osteogenesis in aged MSCs, reduced oxidative stress, and suppression of osteoclastogenesis. Additionally, *G. biloba* extracts protect chondrocytes in osteoarthritis by inhibiting LPS- and IL-1β-induced matrix degradation; EGb reduces MMP-3 production, while GB preserves collagen-II and aggrecan expression via MAPK pathway inhibition, highlighting its therapeutic potential in skeletal degeneration and osteoarthritis, which are depicted in (Table 3).[131]

Table 3: Pharmacological Activity of *Ginkgo biloba.*

SI. No.	Activity	Method used	Dose	Key Outcomes	Ref
Ischemic disease					
01	Cerebral ischemia	In vivo	25, 50, 100 mg/kg	Decrease Pro-inflammatory cytokines Decrease TLR4/NF-κB pathway.	[84]
02	Focal ischemia	In vivo	100, 200 & 400 mg/kg	Neuroprotective effects Improving neurological deficits Increase MPO and SOD activities, and levels of anti-inflammatory cytokine (IL-7 –10) Decrease in Infarct volume, levels of pro-inflammatory cytokines (IL-1β & TNF-α), and MDA content. Decrease NO production	[85]
03	Focal cerebral ischemia	In vivo	16 mg/kg/d	Increase Expression of p-Akt, p-ERK1/2, p-PKA, p-Src, and p-EGFR.	[86]
04	Cerebral ischemic stroke	In vivo	1, 3 & 10 mg/kg	Increase PI3K/Akt-mediated Nrf2 and CREB signalling pathway.	[87]
05	Permanent ischemia	In vivo	100 mg/kg	Increase Neurogenesis in OVX9 mice Increase Androgen receptor expression Increase Grip strength and neurological deficits Decrease the Extrinsic apoptotic pathway	[88]
06	Perinatal hypoxic-ischemic (HI)	In vivo	1, 5, or 10 mg/ kg	Decrease Hypoxic-ischemic brain injury Decrease NLRP3 inflammasome activation	[83]
Cerebrovascular and neurodegenerative disorders of the CNS					
07	Chronic cerebral hypoperfusion (CCH)	In vivo	5, 10, 20 & 40 mg/kg	Increase BCCAo-induced VaD13-like pathology Decrease Glial proliferation, neuroinflammation, and cholinergic deficits	[91]
08	Hemorrhagic stroke	In vivo	45 mg/kg	Decrease Cognitive impairments via regulating the expression of inflammatory factors secreted by microglia.	[92]
09	Intracerebral haemorrhage	In vivo	100 mg/kg	Decrease TUNEL-positive neurons and richer	[93]

	(ICH)			microvascular networks Decrease Meuronal apoptosis in vitro, GSK3β activity and CC3 expression	
10	Alzheimer's disease	In vitro	100 µg/ml	Increase Cell apoptosis and intracellular ROS17 generation Decrease BBB18 leakage, Increase TJ scaffold proteins Decrease RAGE19 in bEnd.3 cells	[94]
11	Permanent middle cerebral artery occlusion (pMCAO)	In vivo	25,50,100 mg/kg	Increased Motor function associated to the reduced neuronal damage Increased Motor function in the sequelae phase of stroke	[95]
12	Middle cerebral artery occlusion (MCAO)	In vivo	5.2, 2.6, 1.3 mg/kg	Increased Neurologic impairment Scavenging free radicals Decreased Free Ca2+ inflow into the cells Protective effect on cerebral ischemia	[96]
13	Acute ischemic stroke (AIS)	In vitro	EGb= 0.1 mg/ml GB = 100 µmol/ml	Neuroprotective effects Preventive effects of EGb on neuronal cell death Increased Function of brain capillary endothelial monolayers after OGD/R injury in vitro	[97]
14	Mesenteric arterioles	In vivo	50, 100 & 200 mg/kg	Protective effect Increased Vascular elasticity and Akt/FoxO3a signaling pathway	[98]
15	Arrhythmia	In vivo	50 mg/l	Increased Threshold doses of aconitine and ouabain Inhibitory effects on DADs and TA induced by ouabain andhigh Ca2+ papillary muscles	[90]
16	Ischemic arrhythmia	In vitro	0.005 to 0.25 mg/ml	hERG-HEK293 cell line Antiarrhythmic effect Decreased IKr and ICa-L currents	[89]
17	Cardiac ventricular remodeling	In vivo	100 mg/kg	Decreased Myocardial remodeling after acute myocardial infarction Decreased Transcription of TGF7-β1, MMP8–2 and MMP-9 genes Decreased Expression of type I collagen, MMP-2 and MMP-9 proteins in myocardial cells	[107]
18	Cardiotoxicity	In vivo	761 100 mg/kg	Cardioprotective effects by removing oxygen free radicals and NOx Regulating inflammatory and vasoactive mediators Decreased Membrane lipid peroxidation	[106]
19	Myocardial ischemia	In vivo	20, 40mg/kg	Increased Cardiac function Decreased Oxidative stress Decreased Inflammatory cascade	[105]
20	Hypertension	In vivo	80 mg/kg	Increased Plasma concentration of losartan Decreased Concentration of Losartan carboxylic acid	[104]
21	Ventricular hypertrophy	In vivo	60 mg/kg	Decreased CaN / NFAT signal pathway Decreased Expression of TRPC6 protein	[103]
22	Myocardial ischemia–reperfusion injury (MIRI)	In vivo	20, 40 mg/kg	Decreased Apoptosis of myocardial cells Protecting the myocardium Increased Akt/Nrf2 pathway Decreased Oxidative stress Decreased Inflammatory reaction	[102]
23	Diabetes cardiomyopathy (DCM)	In vivo	200 and 400 mg/kg	Decreased Cardiomyocyte apoptosis, collagen deposition, and inflammation in diabetic mice Decreased p-JNK15, CHOP, and caspase-12	[101]

				pathways Regulating Serum levels of the proinflammatory cytokines (IL16–6, IL-1β, &TNF-α17), blood glucose, and lipid profiles	
24	Atherosclerosis	In vitro	20 mg/kg/d	Increased Production of clopidogrel active metabolite Alteration in the pharmacokinetics of the clopidogrel active metabolite in high dose	[100]
25	Thrombosis	In vitro	1, 10 & 100 μM	Inhibitory effects on human thrombin	[99]
Neoplastic diseases					
26	Prostate cancer	In vivo	30 mg⁄kg	Antitumor activity Decreased STAT3	[109]
27	Pancreatic cancer	In vitro	1, 2, 5, 10, 20, 50,100 & 200 μM	Anti-tumor effect through targeting pathway driving lipogenesis	[110]
28	Postmenopausal breast cancer	In vitro	10 & 100 μg/ml	Decreased 17β-estradiol and testosterone Decreased Aromatase activity No change in aldosterone or cortisol Potential therapeutic effects on estrogen dependent breast cancer	[111]
29	Cervical cancer (CCa)	In vitro	40.2 ± 1.2 nm	HeLa and SiHa cells Suppressing cancer cell proliferation and inducing apoptosis via upregulating intracellular ROS4 generation and inducing the activation of the caspase-dependent mitochondrial apoptotic pathway	[112]
30	Skin cancer	In vitro	50, 100, 200 mg/kg	Decreased Growth of B16 transplanted solid tumor Decreased Expression of CD34 Decreased MVD14 Dreased Angiogenesis and regulation of PI3K/Akt/ HIF-lα15/VEGF16 signaling pathway	[113]
31	Liver cancer	In vivo	50 mg/kg	Decreased Migration and invasion abilities of HepG2 cells Decreased Expression of invasion-related molecules (MMP11-2 & MMP-9) Preventing EMT12 of HepG2 cells Decreased Invasion and EMT of HepG2 cells Decreased HGF/c-Met-signaling Decreased Tumor growth of liver cancer and prevented EMT In vivo	[114]
32	Lung cancer	In vitro	100 ug/mL	Decreased Migration ability of A549/H441 Decreased HSP1927 expression Decreased AKT and p38 MAPK pathways Decreased HSP27 expression and migratory ability of A549/H441 cells	[115]
33	Bladder cancer	In vitro	50, 100 & 200 μg/ml	Increased Apoptosis through the mitochondriadependent pathway Increased BAX Decreased Expression of Bcl-2, caspase-9,caspase-3 and PARP	[116]
34	Gastric cancer	In vitro	80, 320 & 1.280 μg/ml	Increased Chemotherapeutic sensitivity Reversing the chemoresistance suppressing Increased KSR1-mediated ERK1/2 signaling pathway	[117]
35	Breast cancer	In vitro	40,200 & 1000 mg/ kg/d	Decreased Tumor size and tumor CYP19 mRNA expression Anti-tumor properties	[108]

Gastrointestinal disorder					
36	Acute pancreatitis	In vivo	761 100 mg/kg	Decreased Serum amylase, lipase levels andhistopathologic scores	[119]
37	Peptic ulcer	In vivo	3, 6 mg/kg	Decreased Levels of IL1 –6, IL-1β & TNF-α2 Increased MPO3 level in stomach Increased SOD4 activity	[120]
38	Gastric ulcer	In vivo	8.75, 17.5, 26.25 mg/kg	Decreased Ethanol-induced gastric lesions probably through blockade of cell apoptosis Decreased Lipid peroxidation and preservation of gastric mucus and NP-SH	[121]
39	Colitis	In vivo	120–240 mg	Decreased Activation of macrophages Anti-inflammation effects Decreased Markers of inflammation and inflammatory stress Increased Apoptosis in vitro and in vivo	[122]
40	Inflammatory bowel disease (IBD)	In vivo	100 mg/kg	Decreased TNF-α and NO9 levels, Colonic MPO activity, Colonichydroxyproline content and Serum ceruloplasmin activity Decreased Proinflammatory (MMP10–1, MM	[118]
Neurotoxicological disorders					
41	Sodium arsenite-induced neurotoxicity and dementia	In vivo	100 mg/kg	Antioxidant activity and protective effects against the neurotoxicity induced by sodium arsenite	[124]
42	Aluminum chloride toxicity	In vivo	200 mg/kg	Increased Some biochemical and histological changes observed in the brain and testis of male rats	[125]
43	Aluminium Neurotoxicity	In vitro	100 mg/kg	Decreased Changes in behavioral studies, lipid composition and membrane fluidity Protecting the membrane integrity effectively through antioxidant activity of GBE Increased Affecting on hippocampal and cortical regions of brain in pretreatment group	[123]
Metabolic disorders					
44	Obesity	In vivo	500 mg/kg	Increased Proteome profile and oxidative stress response in the adipose tissue	[127]
45	Diabetes			Increased Insulin secretion, insulin reserve and secretion capacity Decreased Cardiomyocyte apoptosis, collagen deposition, and inflammation Decreased p-JNK3, CHOP and caspase-12 pathways Regulating Serum levels of the proinflammatory cytokines (IL4–6, IL-1β & TNF-α5), blood glucose, and lipid profiles	[126]
Acute neurological disorder					
46	Early brain injury (EBI)	In vivo	10, 50 & 100 mg/kg	Decrease in Neurological dysfunction Decrease Brain water content Decrease Neuronal apoptosis Increase the Akt pathway Increase Bcl-2 levels	[129]
47	Epilepsy	In vivo	50,100 mg/kg	Decreased Seizure severity Increased Spatial cognitive functions and recognition memory Decreased Neuronal damage in thehippocampal pyramidal layer Decreased Anxiety-like behavior and aggression	[128]

Neuroinflammation					
48	Microglial inflammation	In vitro	100 mg/l	Decrease the Microglial inflammatory response by transrepression of the inflammatory cytokine gene.	[130]
Skeletal disorder					
49	Osteoporosis	In vitro	1.25, 5, 20, 80 & 160 µg/l	Increase Osteoblastic differentiation of MC3T3-E1 cells through VEGF2 Increase mRNA expression and secretion levels of VEGF Increase Osteoblastic differentiation of the MC3T3-E1 cells	[131]

Discussion and Future Perspective

The current compilation emphasises *Ginkgo biloba*'s broad pharmacological spectrum and supports its applicability as a multi-target therapeutic agent in a variety of pathological conditions, especially those involving oxidative stress, inflammation, vascular dysfunction, and neurodegeneration. Cardiovascular, neurological, metabolic, neoplastic, and gastrointestinal illnesses are only a few of the many conditions listed, which highlights the pleiotropic nature of its bioactive components and encourages further study in translational and clinical research.

The critical role that oxidative stress and inflammation play in the pathophysiology of disease is a major theme that emerges from this investigation. By scavenging reactive oxygen species (ROS) and triggering endogenous defence mechanisms like the Nrf2 pathway, *Ginkgo biloba*'s flavonoid fraction mainly quercetin, kaempferol, and isorhamnetin contributes significantly to its antioxidant activity. In neurodegenerative diseases like Alzheimer's, Parkinson's, and spinocerebellar ataxia, where oxidative damage and mitochondrial dysfunction are major causes of neuronal loss, this process is especially pertinent. Furthermore, by stabilising mitochondrial membranes, preventing apoptosis, and regulating neurotransmission, terpene lactones such as ginkgolides and bilobalide offer neuroprotection.

The therapeutic benefits of *Ginkgo biloba* for cerebrovascular and cardiovascular conditions such ischaemic stroke, intracerebral haemorrhage, hypertension, myocardial ischaemia, and atherosclerosis are further explained by its vascular and hemorheological properties. Ginkgolides' inhibition of platelet-activating factor (PAF) is essential for decreasing platelet aggregation and enhancing microcirculation. This is especially helpful in cases of ischemia-reperfusion injury, where blood flow restoration ironically makes inflammation and oxidative damage worse. Its minor effectiveness in peripheral vascular illnesses such intermittent claudication may potentially be due to improved endothelial function and blood flow.

It is becoming more widely acknowledged that microglial activation and neuroinflammation are important causes of both acute and chronic neurological diseases. *Ginkgo biloba*'s anti-inflammatory properties, which are mediated via the suppression of NF-κB and pro-inflammatory cytokines, indicate that it may be useful in treating illnesses including epilepsy, neuropathic pain, spinal cord injury, and depressive-like behaviour brought on by LPS. Its significance in neurovascular dysfunction and toxin-induced neurological injury, including heavy metal neurotoxicity (e.g., arsenite, aluminium, fluoride, and cisplatin), is further highlighted by its capacity to alter the integrity of the blood–brain barrier. *Ginkgo biloba* shows encouraging antidiabetic and hypolipidemic benefits in metabolic illnesses such diabetes mellitus, obesity, and nonalcoholic fatty liver disease (NAFLD). Improved insulin sensitivity, decreased lipid peroxidation, and altered inflammatory signalling pathways are probably the mechanisms underlying these advantages. Its potential for systemic treatment is further supported by the cardioprotective effects seen in diabetic cardiomyopathy and ventricular

remodelling. By modifying signalling pathways like STAT3 and NF-κB, the anticancer activities of *Ginkgo biloba* compounds, especially biflavonoids like ginkgetin, are linked to the suppression of metastasis, induction of apoptosis, and reduction of tumour cell growth. Its effects in a number of cancers, including those of the prostate, breast, lung, liver, and stomach, may be attributed to these pathways. These results, however, are still mostly preclinical and need more clinical confirmation.

The clinical effectiveness of *Ginkgo biloba* varies based on the disease, despite these encouraging pharmacological effects. Although there is evidence to support its usage in vascular dementia and cognitive impairment, its benefits in claudication and other disorders are still minimal. Variability in extract composition, dose, study methodology, and patient groups could be the cause of this disparity. In order to ensure repeatability, safety, and therapeutic efficacy, standardised extracts such as those with roughly 24% flavonoid glycosides and 6% terpene lactones with minimum ginkgolic acid content are crucial. Safety and the possibility of medication interactions are other important factors.

Ginkgo biloba's antiplatelet action may raise the risk of bleeding, especially when used with anticoagulants or antiplatelet medications. As a result, in clinical settings, thorough patient selection and monitoring are crucial.

To sum up, *Ginkgo biloba* is a special case of a phytopharmaceutical with a variety of medicinal applications. It is especially useful in complicated, multifactorial diseases like neurodegenerative and cardiovascular disorders since it can target many cellular pathways at once. Nevertheless, more carefully planned clinical trials are required to prove conclusive efficacy for the many illnesses covered. To fully realise its therapeutic promise, future research should concentrate on clarifying specific molecular pathways, refining formulations, and investigating synergistic effects with traditional medicines.

Future research on *Ginkgo biloba* should focus on strengthening clinical evidence across its wide therapeutic spectrum. Although preclinical studies demonstrate efficacy in cardiovascular, neurodegenerative, metabolic, and neoplastic disorders, large-scale, randomized controlled trials are needed to validate its effectiveness, optimal dosage, and long-term safety in diverse patient populations.

CONCLUSION

Ginkgo biloba, often referred to as a "living fossil" in evolutionary terms, possesses considerable pharmacological significance due to its unique combination of flavonol glycosides and terpene lactones. These compounds improve microcirculation and offer neuroprotection by influencing the Nrf2 antioxidant response and functioning as PAF antagonists. Such multi-faceted mechanisms are beneficial in the management of Alzheimer’s, dementia, and ischemic stroke, as well as peripheral circulatory and inflammatory conditions. To guarantee clinical safety, it is crucial to utilize standardized extracts that maintain a balance of active metabolites while removing harmful ginkgolic acids. In conclusion, Ginkgo exemplifies a refined fusion of ancient botanical endurance and contemporary medical applications.

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