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Targeting Neuro-inflammation with Herbal Compounds: An Emerging Strategy for Brain Disorder

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Abstract

Neuroinflammation has emerged as a key pathological feature underlying several brain disorders, including Alzheimer's disease, Parkinson's disease, and depression. The sustained activation of microglia and astrocytes leads to the excessive release of pro-inflammatory cytokines, chemokines, and reactive oxygen species, resulting in oxidative stress, synaptic dysfunction, and neuronal loss. Conventional anti-inflammatory therapies often show limited efficacy in the central nervous system due to poor blood–brain barrier penetration and systemic side effects. In recent years, herbal compounds have gained attention as promising modulators of neuroinflammation owing to their multitargeted mechanisms, antioxidant properties, and favorable safety profiles. Phytochemicals such as curcumin, quercetin, naringenin, *Ginkgo biloba*, and ashwagandha exhibit potent neuroprotective actions by regulating pathways such as NF-κB, MAPK, and Nrf2, thereby reducing cytokine production andoxidative injury. This review provides a comprehensive overview of the mechanisms of neuroinflammation and explores how herbal compounds can counteract these processes to prevent or mitigate neurodegenerative and neuropsychiatric conditions. It also discusses challenges in formulation, bioavailability, and clinical translation,

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highlighting the future potential of plant-based therapeutics as an emerging strategy for targeting neuroinflammation in brain disorders.

Keywords: Neuroinflammation, Neurodegenerative diseases, Herbal compounds, Oxidative stress, Microglial activation, Neuroprotection

Introduction

Neuroinflammation is a complex and tightly regulated immune response in the central nervous system (CNS), primarily mediated by glial cells such as microglia and astrocytes[1]. Under normal physiological conditions, these cells maintain neuronal homeostasis, clear cellular debris, and preserve synaptic integrity[2]. However, chronic activation of microglia and astrocytes in response to infections, toxins, misfolded proteins, or neuronal injury can trigger a sustained inflammatory response that disrupts neuronal signaling and accelerates neurodegeneration[3]. This persistent inflammation plays a critical role in the development of neurological disorders, including Alzheimer's disease (AD), Parkinson's disease (PD), multiple sclerosis, and neuropsychiatric conditions such as depression and schizophrenia[4].

The pathophysiology of neuroinflammation involves the release of pro-inflammatory cytokines (TNF-α, IL-1β, and IL-6), chemokines, reactive oxygen species (ROS), and nitric oxide (NO)[5]. These mediators impair mitochondrial function, damage neuronal membranes, disrupt the blood–brain barrier (BBB), and enhance neurotoxicity[6]. Although conventional anti-inflammatory agents, such as corticosteroids and nonsteroidal anti-inflammatory drugs (NSAIDs), can reduce systemic inflammation, their effectiveness in the CNS is limited due to poor BBB permeability and associated adverse effects[7].

In this context, herbal compounds have gained attention as potential modulators of neuroinflammation[8]. Phytochemicals such as curcumin, quercetin, naringenin, Ginkgo biloba, and ashwagandha exhibit anti-inflammatory, antioxidant, and neuroprotective properties by targeting multiple signaling pathways, including NF-κB, MAPK, and Nrf2[9]. This review highlights their therapeutic potential in neuroinflammatory disorders[10].

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Table 1: Herbal Compounds with anti-inflammatory activity

S. No	Herbal Compound (Plant Source)	Scientific Name	Major Chemical Constituents	Anti-inflammatory Mechanism/ Action	References
1	Turmeric	*Curcuma longa*	Curcumin, Demethoxycurcumin, Bisdemethoxycurcumin	Inhibits COX-2 and NF-κB pathway, reduces prostaglandin synthesis	[11]
2	Ginger	*Zingiber officinale*	Gingerols, Shogaols, Zingerone	Suppresses prostaglandin and leukotriene synthesis	[12]
3	Ashwagandha	*Withanias omnifera*	Withanolides, Withaferin A	Inhibits pro-inflammatory cytokines (TNF-α, IL-6)	[13]
4	Tulsi (Holy Basil)	*Ocimum sanctum*	Eugenol, Ursolic acid, Rosmarinic acid	Inhibits COX and LOX enzymes, reduces oxidative stress	[14]
5	Aloe Vera	*Aloe barbadensis miller*	Aloin, Emodin, Polysaccharides	Modulates inflammatory mediators and scavenges free radicals	[15]
6	Boswellia (Indian Frankincense)	*Boswellia serrata*	Boswellic acids (AKBA)	Inhibits 5-lipoxygenase (5-LOX), reduces leukotriene synthesis	[16]
7	Licorice	*Glycyrrhiza glabra*	Glycyrrhizin, Liquiritin, Glabridin	Inhibits COX-2, suppresses cytokine production	[17]

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8	Green Tea	*Camellia sinensis*	Epigallocatechingallate (EGCG), Catechins	Reduces expression of inflammatory mediators via NF-κB inhibition	[18]
9	Neem	*Azadirachta indica*	Nimbidin, Azadirachtin, Quercetin	Suppresses macrophage activation and cytokine release	[19]
10	Amla (Indian Gooseberry)	*Phyllanthus emblica*	Gallic acid, Ellagic acid, Ascorbic acid	Antioxidant and anti-inflammatory via ROS scavenging	[20]
11	Black Pepper	*Piper nigrum*	Piperine	Inhibits pro-inflammatory cytokines (IL-1β, TNF-α) and reduces COX-2 expression	[21]
12	Cinnamon	*Cinnamomum verum*	Cinnamaldehyde, Eugenol	Suppresses NF-κB activation and decreases nitric oxide production	[22]
13	Clove	*Syzygium aromaticum*	Eugenol, Beta-caryophyllene	Inhibits COX and LOX pathways; strong antioxidant and cytokine-modulating effects	[23]
14	Gotu Kola	*Centella asiatica*	Asiaticoside, Madecassoside	Reduces inflammatory markers (TNF-α, IL-1β) and enhances	[24]

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				wound-healing anti-inflammatory pathways	
15	Guggul	*Commiphora mukul*	Guggulsterones (E-Guggulsterone, Z-Guggulsterone)	Inhibits NF-κB signaling and reduces expression of inflammatory genes	[25]

Pathophysiological Role of Neuroinflammation

Neuroinflammation is a complex immunological response in the central nervous system (CNS), primarily mediated by microglia, astrocytes, and infiltrating peripheral immune cells[26]. Under physiological conditions, microglias remain in a resting or surveillant state, maintaining cellular homeostasis and clearing debris. However, in response to brain injury, infection, or the accumulation of abnormal proteins such as amyloid-β or α-synuclein, microglia become activated[27]. This activation occurs in two main phenotypes: M1 (pro-inflammatory), characterized by the release of cytokines such as tumor necrosis factor-α (TNF-α), interleukin-1β (IL-1β), and reactive oxygen species (ROS); and M2 (anti-inflammatory), which promotes tissue repair and releases anti-inflammatory cytokines like IL-10[28].

Persistent M1 activation leads to chronic inflammation, neuronal dysfunction, and cell death, contributing to neurodegenerative diseases such as Alzheimer's disease, Parkinson's disease, and multiple sclerosis[29]. Excess cytokine production can induce a "cytokine storm," increasing oxidative stress and disrupting the blood–brain barrier, allowing peripheral immune cells to enter the CNS. This oxidative stress further impairs mitochondrial function, exacerbating neuronal apoptosis and synaptic damage[30].

Neuroinflammation thus acts as both a trigger and amplifier of neuronal injury, creating a vicious cycle of degeneration. Understanding these molecular and cellular mechanisms is essential for developing targeted therapeutic strategies to regulate microglial activation and control inflammatory responses in neurological disorders[31].

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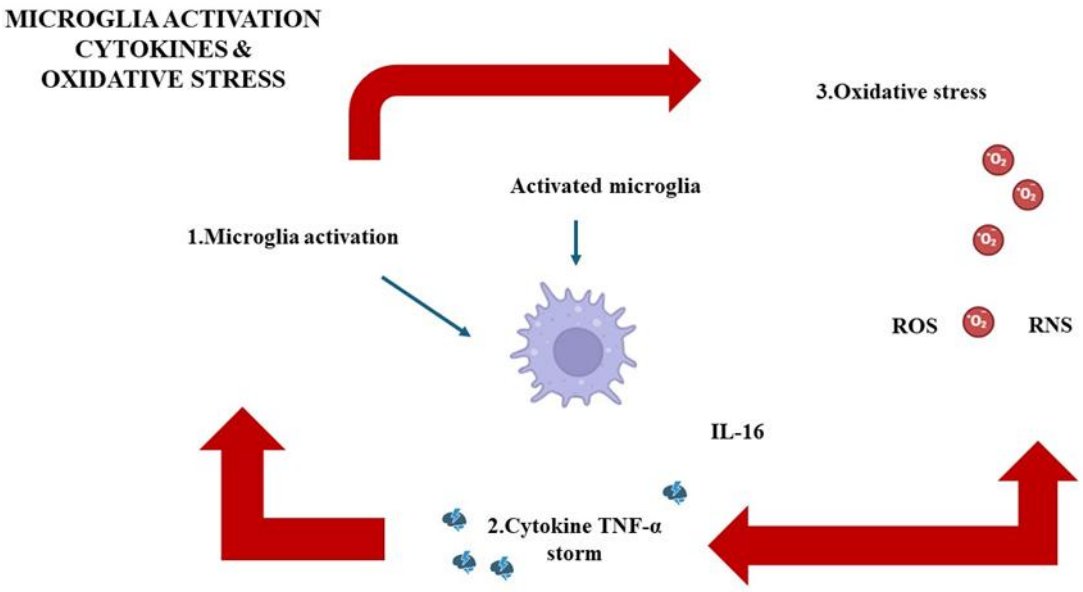

Figure1: Pathophysiological aspect of Activation of Microglia and Cytokines Strom and Oxidative Stress

Herbal Compounds that have Anti-inflammatory potential

Herbal compounds are emerging as promising modulators of neuroinflammation due to their multitargeted actions and natural origin[32]. Various phytochemicals, including curcumin, quercetin, naringenin, Ginkgo biloba extract, and Withania somnifera (ashwagandha), exhibit significant neuroprotective and anti-inflammatory properties[33]. Curcumin, the principal polyphenol of Curcuma longa, inhibits NF-κB and MAPK signaling pathways, thereby reducing the production of pro-inflammatory cytokines and reactive oxygen species. It also activates the Nrf2/HO-1 antioxidant pathway, enhancing neuronal resistance to oxidative stress. Quercetin, a flavonoid found in apples and onions, suppresses microglial activation and reduces inflammatory mediators by inhibiting iNOS and COX-2 expression[34].

Naringenin, abundant in citrus fruits, attenuates neuroinflammation by inhibiting TLR4/NF-κB signaling and enhancing antioxidant defences[35]. Ginkgo biloba extract protects neurons by reducing oxidative stress, improving mitochondrial function, and inhibiting lipid peroxidation. Ashwagandha, through its active withanolides, exerts anti-inflammatory and adaptogenic effects by modulating JAK/STAT signaling and regulating the HPA axis[36].

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These herbal compounds collectively regulate inflammatory pathways, reduce oxidative stress, and promote neuronal survival, making them promising candidates for managing neuroinflammation-associated neurodegenerative disorders[37].

Table 2: Representation of herbal Compounds with Chemical Constituents

S. No.	Herbal Compound (Plant Source)	Scientific Name	Main Chemical Constituents	Pharmacological Actions/Uses	References
1	Turmeric	*Curcuma longa*	Curcumin, Desmethoxycurcumi, Bisdemethoxycurcumin	Anti-inflammatory, antioxidant, antimicrobial	[38]
2	Neem	*Azadirachta indica*	Azadirachtin, Nimbin, Nimbidin, Quercetin	Antibacterial, antifungal, antimalarial	[39]
3	Ashwagandha	*Withania somnifera*	Withanolides, WithaferinA, Alkaloids	Adaptogenic, anti-stress, immunomodulatory	[40]
4	Tulsi (Holy Basil)	*Ocimum sanctum*	Eugenol, Ursolic acid, Rosmarinic acid	Antioxidant, anti-inflammatory, antimicrobial	[41]
5	Ginger	*Zingiber officinale*	Gingerols, Shogaols, Zingerone	Anti-nausea, anti-inflammatory, digestive aid	[42]
6	Amla (Indian Gooseberry)	*Phyllanthus emblica*	Ascorbic acid (Vitamin C), Tannins, Gallic acid	Antioxidant, hepatoprotective, immune booster	[43]
7	Garlic	*Allium sativum*	Allicin, Alliin, S-allyl cysteine	Hypolipidemic, antimicrobial, antihypertensive	[44]
8	Aloe Vera	*Aloe barbadensis miller*	Aloin, Barbaloin, Emodin, Polysaccharides	Wound healing, moisturizing, anti-inflammatory	[45]
9	Guggul	*Commiphora mukul*	Guggulsterones, Myrrhanol A, Myrrhanone A	Anti-hyperlipidemic, anti-inflammatory	[46]
10	Brahmi	*Bacopa monnieri*	Bacosides A and B, Alkaloids, Flavonoids	Memory enhancer, neuroprotective	[47]

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11	Shatavari	*Asparagus racemosus*	Shatavarins (Saponins), Isoflavones, Steroidal saponins	Female reproductive tonic, adaptogenic, anti-ulcer	[48]
12	Giloy (Guduchi)	*Tinospora cordifolia*	Tinosporaside, Berberine, Cordifolioside A	Immunomodulatory, antipyretic, anti-inflammatory	[49]
13	Licorice (Mulethi)	*Glycyrrhiza glabra*	Glycyrrhizin, Glabridin, Liquiritin	Expectorant, anti-inflammatory, gastroprotective	[50]
14	Black Pepper	*Piper nigrum*	Piperine, Essential oils, Lignans	Enhances bioavailability of nutrients, digestive stimulant	[51]
15	Fenugreek	*Trigonella foenum-graecum*	Trigonelline, Diosgenin, Saponins, FiberDiosgenin, Saponins, Fiber	Antidiabetic, digestive aid, hypolipidemic	[52]

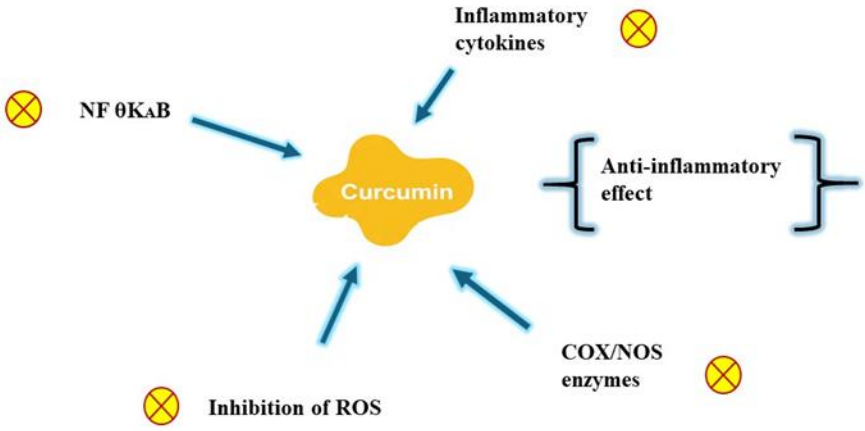

Figure 2: Mechanistic Approach of Curcumin

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Therapeutic Applications

Clinical validationis required [53]. Herbal compounds exhibit significant neuroinflammatory modulatory effects and hold therapeutic potential in various neurological and neuropsychiatric disorders. In Alzheimer's disease (AD), chronic microglial activation and accumulation of pro-inflammatory cytokines accelerate amyloid-β aggregation and neuronal death[54]. Phytochemicals such as curcumin and quercetin have demonstrated the ability to inhibit amyloid aggregation, reduce oxidative stress, and improve synaptic plasticity. Notably, curcumin can cross the blood–brain barrier (BBB) and suppress NF-κB signaling, thereby attenuating the inflammatory environment associated with AD[55].

In Parkinson's disease (PD), neuroinflammation contributes to the degeneration of dopaminergic neurons in the substantia nigra. Compounds like naringenin and Ginkgo biloba extract provide neuroprotection by reducing oxidative stress and preserving mitochondrial function[56]. These agents inhibit glial activation and decrease pro-inflammatory cytokine production, ultimately slowing disease progression.

Beyond neurodegenerative disorders, herbal compounds also show promise in neuropsychiatric conditions such as depression, where inflammation and oxidative stress play key roles[57]. Ashwagandha, known for its adaptogenic and neuroprotective properties, regulates the hypothalamic–pituitary–adrenal (HPA) axis and mitigates inflammation-induced neurochemical alterations[58].

Overall, these findings highlight the potential of herbal compounds as complementary therapeutic agents, although further

Formulation and Delivery Challenges

Despite substantial evidence from preclinical studies supporting the neuroprotective and anti-inflammatory effects of herbal compounds, their clinical translation remains limited[59]. Much of the existing data is derived from in vitro experiments and animal models, while human trials are relatively scarce and often involve small sample sizes or short durations[60]. Nevertheless, several clinical studies have begun to demonstrate the therapeutic potential of these compounds in neurodegenerative and neuropsychiatric disorders[61].

Curcumin, for instance, has shown beneficial effects in patients with mild cognitive impairment and Alzheimer's disease by improving memory and reducing inflammatory biomarkers[62]. Similarly, Ginkgo biloba extract (EGb761) has demonstrated cognitive and neuroprotective benefits, including enhanced cerebral blood flow and antioxidant activity in elderly populations. Ashwagandha supplementation has also been associated with reduced stress-induced inflammation

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and improved mood and cognitive performance[63].

However, significant challenges remain, including variability in herbal extract composition, lack of standardized dosing, and insufficient characterization of pharmacokinetics and metabolism in humans[64]. These limitations hinder consistent therapeutic outcomes and complicate clinical validation. Future progress depends on well-designed randomized controlled trials to evaluate safety, efficacy, and optimized formulations[65].

Emerging approaches such as nanotechnology-based drug delivery, systems biology, and network pharmacology offer promising strategies to enhance bioavailability and understand multitarget mechanisms[66]. Additionally, integrating pharmacogenomics and personalized medicine may

enable tailored phytotherapeutic interventions[67]. Overall, advancing clinical validation and regulatory standardization is essential for translating herbal therapies into effective neuroinflammatory treatments[68].

Conclusion

Neuroinflammation is a key driver of many neurological disorders, contributing to progressive neuronal damage and functional decline. Its pathogenesis involves a network of activated microglia, pro-inflammatory mediators, and oxidative stress, making it an important therapeutic target for slowing or preventing neurodegeneration. In this context, herbal compounds have emerged as promising modulators of inflammatory and oxidative pathways in the brain. Phytochemicals such as curcumin, quercetin, naringenin, Ginkgo biloba extracts, and Ashwagandha act through multiple mechanisms, including suppression of inflammatory signaling cascades, activation of antioxidant defenses, and support of neuronal survival processes. Their multitargeted actions offer advantages over conventional single-pathway drugs. However, limitations involving solubility, bioavailability, and brain penetration continue to restrict their clinical utility. Innovative delivery systemssuch as nanoformulations and phytosome technologiesare helping address these challenges. Overall, herbal-based strategies targeting neuroinflammation hold strong potential for future evidence-based therapies against neurodegenerative and neuropsychiatric conditions

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List of abbreviations

AD	Alzheimer's Disease
ALS	Amyotrophic Lateral Sclerosis
BBB	Blood–Brain Barrier
BDNF	Brain-Derived Neurotrophic Factor
CNS	Central Nervous System
COX	Cyclooxygenase
COX-2	Cyclooxygenase-2
CSF	Cerebrospinal Fluid
DA	Dopaminergic Neurons
DTI	Diffusion Tensor Imaging
EGCG	Epigallocatechin Gallate
EGb761	Standardized Extract of Ginkgo biloba
HD	Huntington's Disease
HO-1	Heme Oxygenase-1
HPA axis	Hypothalamic–Pituitary–Adrenal Axis
IL-1β	Interleukin-1 Beta
IL-6	Interleukin-6
IL-10	Interleukin-10
iNOS	Inducible Nitric Oxide Synthase
JAK/STAT	Janus Kinase / Signal Transducer and Activator of Transcription
LOX	Lipoxygenase
MAPK	Mitogen-Activated Protein Kinase

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MCI	Mild Cognitive Impairment
M1/M2	Microglial Phenotypes (Pro-inflammatory / Anti-inflammatory)
MS	Multiple Sclerosis
NF-κB	Nuclear Factor Kappa-Light-Chain-Enhancer of Activated B Cells
Nrf2	Nuclear Factor-Erythroid 2-Related Factor 2
NO	Nitric Oxide
NSAIDs	Non-Steroidal Anti-Inflammatory Drugs
PD	Parkinson's Disease
PET	Positron Emission Tomography
ROS	Reactive Oxygen Species
TNF-α	Tumor Necrosis Factor-Alpha
TLR4	Toll-Like Receptor 4
TPGS	D-α-Tocopheryl Polyethylene Glycol Succinate
EGb	Ginkgo biloba Extract
HPLC	High-Performance Liquid Chromatography
NPs	Nanoparticles
SLN	Solid-Lipid Nan

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