Anti-Inflammatory Potential of Medicinal Plants: A Review

Mohini Patel*[1], Reshma Jain[2]

[1]*Assistant Professor, School of Pharmacy, ITM SLS Baroda University, Vadodara-391510, Gujarat, India*
[2]*Assistant Professor, Faculty of Pharmacy, The Maharaja Sayajirao University of Baroda, Vadodara, Gujarat, India*
***Corresponding Author:** mohinipatel2712@gmail.com

Abstract:

Inflammation is a key contributor to numerous health conditions commonly associated with modern lifestyles. Although non-steroidal anti-inflammatory drugs (NSAIDs) are frequently prescribed to manage inflammatory responses, their prolonged use is often linked to adverse effects such as gastrointestinal irritation, skin rashes, ulcers, liver toxicity, and renal complications. Historically, medicinal plants have played a fundamental role in healthcare systems, offering therapeutic potential for a wide range of diseases. Today, these natural remedies continue to serve as valuable resources for pharmaceutical development. Inflammation, a defence mechanism triggered by injury or infection, typically presents as pain, heat, redness, and swelling. This review highlights the anti-inflammatory potential of various medicinal plant species, emphasizing their bioactive constituents and mechanisms of action. By exploring plant-based alternatives, the review aims to support the development of safer and more sustainable anti-inflammatory therapies.

Key Words: *Inflammatory conditions, non-steroidal anti-inflammatory drugs (NSAIDs), side effects, therapeutic herbs, plant-derived anti-inflammatory compounds*

1. Introduction:

1.1 Inflammation:

Inflammation, derived from the Latin word *inflammare* meaning "to ignite," refers to a complex biological response triggered when vascular tissues encounter harmful stimuli such as pathogens, irritants, allergens, or damaged cells. It serves as a protective mechanism aimed at initiating tissue repair and restoring cellular balance. [1]

Clinically, inflammation is characterized by signs such as redness, swelling, heat, pain, and sometimes loss of function, typically occurring at the site of injury or infection. It is considered a central feature of many pathological processes but is also an essential defensive response that promotes wound healing and recovery of tissue integrity.

The inflammatory process can be activated by internal disruptions (e.g., tissue injury, hormonal imbalances, organ dysfunction) or external triggers (e.g., microbial invasion, allergens, and environmental toxins). Factors like obesity, dietary components, and pollutants can also contribute. Innate immune cells detect these threats through antigen receptors, initiating chemical signalling cascades. When the regulatory control of this process fails, it can lead to prolonged inflammation and tissue damage. [2]

1. 2 Types of Inflammation

Inflammation is generally classified into two forms:

1. **Acute Inflammation** – short-term and self-limiting
2. **Chronic Inflammation** – long-lasting and often progressive

Acute inflammation is the body's initial defence mechanism, presenting with classic symptoms such as pain, redness, heat, swelling, and reduced function at the affected site. These signs result from increased blood flow, vascular permeability, and sensitized nerve endings. Typically, this response is tightly regulated to balance tissue protection and damage prevention.

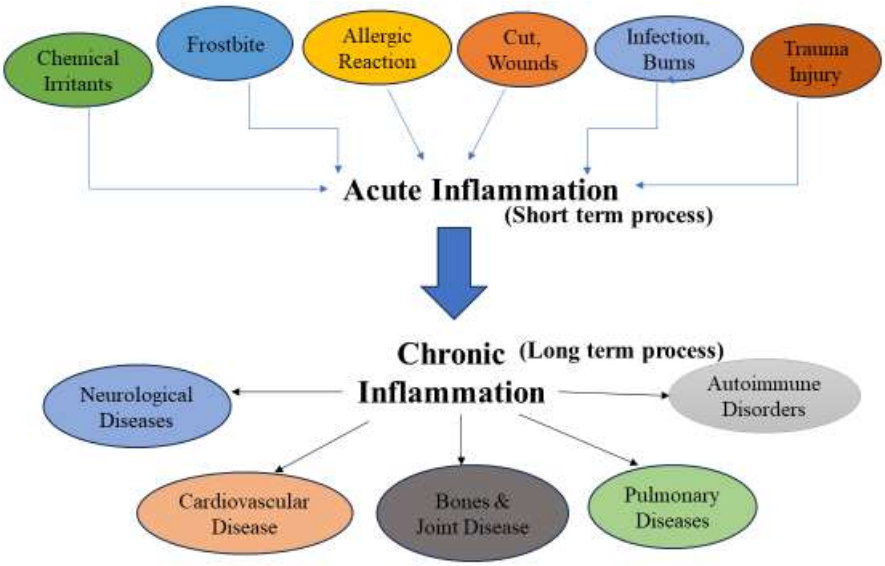

Figure 1: Some factors cause inflammation and lead to Different disease

In contrast, chronic inflammation develops over a longer period and is often linked to ongoing tissue injury and repair. It plays a role in the pathogenesis of various diseases, including autoimmune disorders (e.g., rheumatoid arthritis, multiple sclerosis), psoriasis, inflammatory bowel disease, and even cancer. Chronic inflammation is recognized as a key contributor to tumour development and progression in several types of cancers.

Persistent, low-grade inflammation is commonly associated with many chronic illnesses. Despite its widespread impact, managing chronic inflammatory conditions remains a significant challenge due to the lack of consistently effective and safe treatments, especially for diseases like inflammatory bowel disease. [3]

The inflammatory process is mediated by numerous biochemical signals, including cytokines and other molecules such as histamine, prostaglandins, leukotriene, interleukins (e.g., IL-1, IL-8), tumour necrosis factor-alpha (TNF-α), interferon-alpha (IFN-α), and serotonin (5-HT). These substances initiate and coordinate complex signalling pathways, resulting in an organized immune response to harmful stimuli. [1]

1.3 Biochemical Basis of Inflammation

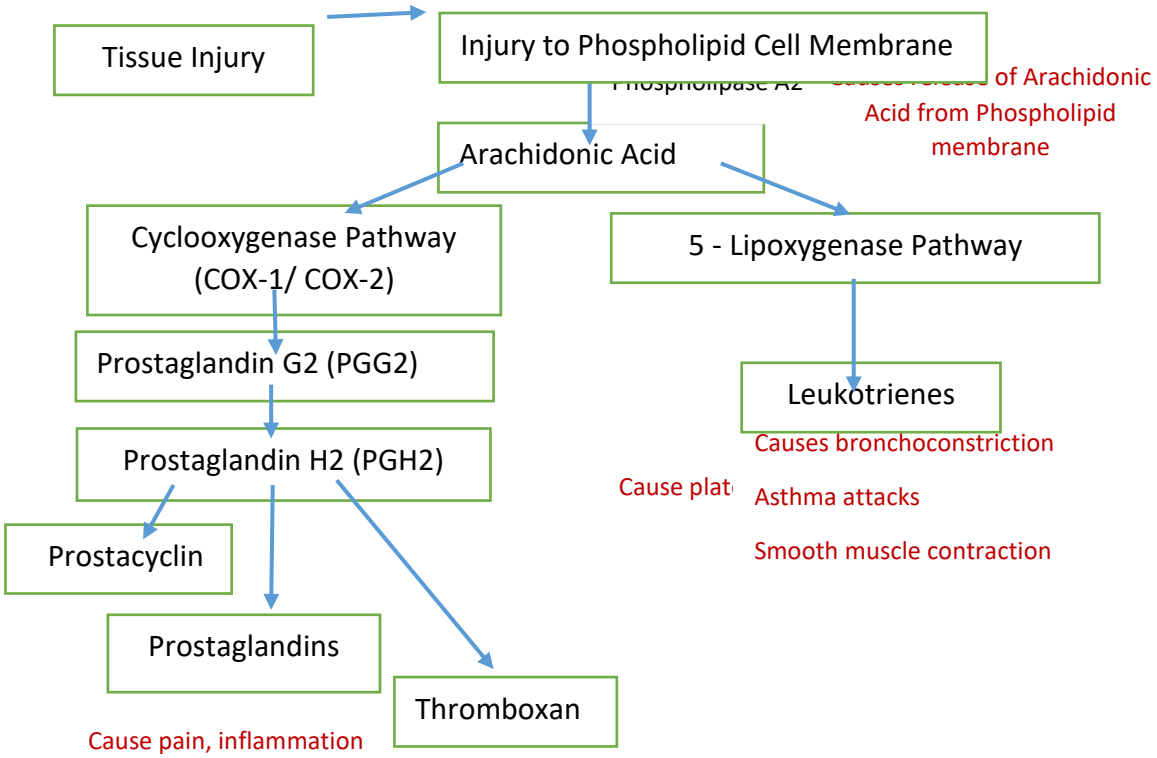

Figure 2: Inflammation Process

Inflammation involves the local synthesis of key mediators, notably prostaglandins (especially PGE_2) and leukotrienes, both derived from arachidonic acid. This fatty acid, stored within cell membranes as a phospholipid, is enzymatically released and metabolized via cyclooxygenases (COX) and lipoxygenases pathways. Specifically, COX-2 converts arachidonic acid into prostaglandins, which play a critical role in initiating and sustaining inflammation following tissue injury.

Pro-inflammatory symptoms such as pain, swelling, fever, and tissue dysfunction are mediated by molecules like prostaglandin E_2 (PGE_2) and nitric oxide (NO). These mediators are regulated by inducible nitric oxide synthase (iNOS) and COX-2, both of which are up regulated during inflammation. [2, 4]

At the molecular level, one of the central signalling pathways driving inflammation is the nuclear factor-kappa B (NF-κB) pathway, activated through mitogen-activated protein kinases (MAPKs). This pathway leads to the expression of major pro-inflammatory cytokines,

including tumour necrosis factor-alpha (TNF-α), interleukin-1 beta (IL-1β), and interleukin-6 (IL-6), all of which amplify the inflammatory response. [4]

1.4 Inflammatory Mediators

Inflammatory mediators are biologically active substances produced by immune and tissue cells that orchestrate the body's response to injury or infection. Key categories include eicosanoids, cytokines, vasoactive agents, and free radicals.

1.4.1 Eicosanoids

Eicosanoids are lipid-derived signalling molecules synthesized from arachidonic acid, a component of membrane phospholipids. Their production involves enzymes such as cyclooxygenases (COX-1 and COX-2) and lipoxygenases (5-LOX, 12-LOX). The 5-LOX enzyme, discovered in 1976 in rabbit leukocytes, is primarily found in myeloid cells like monocytes and neutrophils and plays a vital role in inflammatory and immune responses.

Cyclooxygenases convert arachidonic acid into pro-inflammatory prostaglandins and prostanoids. COX-1, present in most mammalian cells including platelets and neurons, contributes to both homeostasis and inflammation. COX-2 is typically induced during inflammation. Prostaglandins, such as PGE_2 and PGD_2, regulate blood flow, vascular permeability, mucus secretion, and platelet function. Elevated levels of prostaglandins have been found in the synovial fluid of patients with rheumatoid arthritis and osteoarthritis, highlighting their role in chronic joint inflammation. [5]

1.4.2 Cytokines

Cytokines are signalling proteins released by a variety of immune and structural cells, including macrophages, neutrophils, fibroblasts, lymphocytes, and endothelial cells. For example, interleukin-1 (IL-1) is produced by multiple cell types and triggers a cascade of immune responses such as cytokine release (IL-2, IL-8, TNF-α), B- and T-cell chemo taxis, histamine release, and proliferation of fibroblasts and epithelial cells. Cytokines are central to the initiation and progression of inflammation. [5, 6]

1.4.3 Vasoactive Mediators

Vasoactive compounds like histamine, serotonin, and bradykinin are involved in regulating blood vessel tone and permeability during inflammation.

- Histamine, released from basophils, supports the acute inflammatory response.
- Serotonin, synthesized via tryptophan decarboxylation and stored in platelets and mast cells, acts through four receptor types to mediate inflammation.
- Bradykinin, produced via the kinin-kallikrein system, interacts with B1 and B2 receptors to induce pain, vascular leakage, and prostaglandin synthesis. [5, 6]

1.4.4 Free Radicals

Free radicals, particularly nitric oxide (NO), are produced by various cells such as neutrophils, macrophages, endothelial cells, smooth muscle cells, and neurons. NO plays a significant role in inflammation by promoting vasodilation, mucosal secretions, and increased vascular permeability. It is regulated by inducible nitric oxide synthase (iNOS) and is often up regulated in response to cytokines like TNF-α and IL-1β. In the respiratory system, NO contributes to mucin overproduction and may influence bone resorption in conditions such as chronic otitis media (COM). The harmful effects of NO are often due to reactive nitrogen species, which are formed when NO reacts with reactive oxygen species. [6]

2. NSAIDs in the Management of Inflammation

The treatment of inflammatory conditions typically involves a multimodal approach**,** including exercise, pharmacological interventions, stress-reduction techniques**,** and in severe cases, surgical procedures like joint replacement. Among pharmacological options, drugs such as hydroxychloroquine, corticosteroids, biologics, cyclophosphamide, and non-steroidal anti-inflammatory drugs (NSAIDs) are commonly used to reduce inflammation, alleviate joint pain, and potentially slow disease progression**.** [7]

NSAIDs represent the most widely prescribed class of anti-inflammatory medications. Despite their widespread use and effectiveness, they are associated with a high risk of adverse effects.[8] Reports suggest that up to 90% of anti-inflammatory drugs can cause toxic reactions, side effects, or complications, which may complicate treatment regimens. [1]

Commonly reported side effects of NSAIDs and corticosteroids include gastrointestinal disturbances**,** peptic ulcers**,** cardiovascular risks**,** immune suppression, and metabolic complications such as hyperglycaemia**.** Long-term use can also lead to osteoporosis**,** cushingoid features**,** hirsutism, and hypersensitivity reactions. Therefore, while NSAIDs remain a cornerstone in the pharmacological management of inflammation, their use requires careful risk assessment and monitoring. [1, 2]

3. Risks Associated with Long-Term Use of Conventional Anti-Inflammatory Drugs

Prolonged or repeated use of conventional anti-inflammatory agents is often associated with various adverse effects, ranging from gastrointestinal irritation to more severe organ toxicity. The following summarizes common side effects linked to specific drugs:

- **Aspirin (Acetylsalicylic acid):** May cause gastrointestinal irritation and increase the risk of bleeding.
- **Diclofenac sodium:** Often leads to upper abdominal pain and can impair kidney function.
- **Ibuprofen:** Associated with stomach lining erosion and related digestive issues.
- **Nimesulide:** Linked to liver toxicity, occasionally presenting with a feeling of fullness.

- **Rofecoxib:** Noted for increasing the risk of cardiovascular complications, particularly heart attacks.
- **Mephenamic acid:** May cause bleeding disorders, diarrhoea, and rare blood-related conditions like haemolytic anaemia.
- **Phenylbutazone:** Known for causing gastrointestinal discomfort and ulcers.
- **Corticosteroids:** Can suppress the immune system, potentially reactivating latent infections such as tuberculosis. [1]

There are various medications available to manage and reduce inflammation, with steroids, immunosuppressant, and NSAIDs being among the most commonly used. However, many of these drugs come with potential side effects. The goal in treatment is to use the smallest effective dose that provides maximum benefit while minimizing adverse effects, which is the standard practice. To improve the pharmacological response and reduce side effects, integrating herbal anti-inflammatory compounds into conventional drug therapies could be beneficial. Given the growing interest in herbal medicine, it is important to further explore and validate these natural remedies. Although complementary, alternative, and traditional medicines serve as key sources of natural treatment approaches, modern science must first validate their efficacy and safety before they can be widely adopted in clinical practice. [9]

4. Natural Anti-Inflammatory Agents (Herbs)

Herbal medicine plays a significant role in complementary and alternative therapies. Medicinal plants have been used for centuries to treat and prevent inflammatory conditions. Due to the increasing use of natural remedies and phytonutrients, also known as nutraceuticals, there has been a global rise in the consumption of these products. According to the World Health Organization (WHO), 75% of people rely on both conventional and plant-based natural medicines for their daily healthcare needs. Herbal remedies are gaining popularity due to their lower risk of side effects compared to synthetic drugs. Many inflammatory diseases have been effectively treated with medicinal plants, which contain active chemical compounds responsible for their therapeutic effects. These plant-derived chemicals—such as terpenoids, glycosides, carotenoids, alkaloids, flavonoids, saponins, and tannins—are known for their anti-inflammatory properties. Some of these plants have been used since ancient times, and others are integral to traditional Chinese and Ayurvedic medicine. [8, 10]

Table 1. Some Anti-Inflammatory Plants

Scientific Name	Family	Part Used (Type of Extract)	C.C.	Medicinal Activity	Ref.
Curcuma longa (Turmeric)	Zingiberaceae	Rhizome (Dichloro-methane extract)	Curcumin	Inhibits COX-2, NF-κB, 5-LOX, PGE-2, and proteoglycans.	8, 11, 12, 13, 14

Cinnamomm cassia (Chinese Cinnamon)	Lauraceae	Bark (Ethanolic extract)	Cinnamaldehyde, Cinnamic acid	Suppresses TNF-α, IL-6, and IL-1β, and inhibits NF-kB activation, reducing nitric oxide production.	13, 15
Boswellia serrata (Indian Olibanum)	Burseraceae	Gum resin (Hydro-alcoholic extract)	AKBA, Boswellic acid	Restricts 5-LOX production, inhibits inflammatory cytokines such as IL-2, IL-1, IL-4, IL-6, IFN-γ, and down regulates TNF-α via NF-κB inhibition.	8, 13, 14
Piper longum (Black Pepper)	Piperaceae	Fruit (Oil or Ethanolic extract)	Piperine, Piperlongumin, β-Sitosterol	Suppresses TNF-β, TNF-α, IL-23, IL-6, and T-cell proliferation.	13, 16
Camellia sinensis (Green Tea)	Theaceae	Leaves (Ethanolic extract)	Quercetin, Catechins, Epigallocatechin-3-gallate, Kaempferol	Inhibits inflammatory cytokines, prevents NF-kB activation, and reduces PGE-2 and COX-2 expression.	8, 13
Azadirachta indica (Neem)	Meliaceae	Leaves, Root, Fruit, Seed, Oil (Methanolic, Hydro-alcoholic extracts)	Margosine, Nimbin, Azadirachtin	Reduces nitric oxide levels and leukocyte counts.	8, 12, 17, 18, 19
Glycyrrhiza glabra (Licorice)	Fabaceae	Roots & Leaves (Ethanol or Hydro-ethanolic extract)	Glycyrrhizin, Glycyrrhetinic acid	Inhibits COX-2, inflammatory cytokines, and nitric oxide production, suppressing IL-1β and IL-18 expression.	8, 11, 12, 13, 20

Ginseng radix (Ginseng)	Araliaceae	Rhizome	Ginsenosides	Prevents COX-2-induced inflammatory enzymes and TNF-α-mediated NF-kB transcription, inhibiting NF-κB pathways.	14, 20
Urtica dioica (Nettle)	Urticaceae	Leaf	Esculetin, Scopoletin, Rutin	Reduces the activity of pro-inflammatory transcription factor NF-κB.	21, 22
Rosmarinus officinalis (Rosemary)	Lamiaceae	Essential Oil, Aerial parts (Alcoholic or Ethanolic extract)	Carnosol, Rosmarinic acid, Caffeic acid	Decreases COX-2 and iNOS expression.	8, 14, 21
Adhatoda vasica (Vasaka)	Acanthaceae	Leaves (Alcoholic extract)	Vasicine	Inhibits 5-LOX activity.	8, 18, 23, 24
Emblica officinalis (Amla)	Euphorbiaceae	Fruit (Hydro-alcoholic extract)	Ascorbic acid, Gallic acid	Inhibits COX-2 activity and reduces pro-inflammatory cytokine expression.	18, 25, 26
Bacopa monnieri (Brahmi)	Scrophulariaceae	Whole plant (Ethanolic extract)	Bacosides	Inhibits 5-LOX and COX-2 activities.	18, 27
Mangifera indica (Mango)	Anacardiaceae	Bark (Aqueous), Roots (Ethanolic extract)	Magniferin, Gallic acid	Inhibits prostaglandin synthesis and suppresses NF-κB pathways.	18, 21, 28
Withania somnifera (Ashwagandha)	Solanaceae	Root, leaf (Aqueous extract)	Withaferin-A, Withanolides	Modulates cytokines and blocks the MAPK/NF-κB pathways.	11, 19, 20

Ocimum sanctum (Tulsi)	Lamiaceae	Whole plant (Water or Aqueous extraction)	Linolenic acid, Eugenol	Inhibits lipoxygenase and cyclooxygenase pathways.	11, 20
Zingiber officinalis (Ginger)	Zingiberaceae	Rhizome (Aqueous extraction)	Gingerol, Shogaol	Reduces COX-2 activity.	11, 13, 20
Borago officinalis (Star flower)	Boraginaceae	Seed (Aqueous, Ethanolic, Methanolic extracts)	Gamma-linolenic acid	Inhibits prostaglandin synthesis and suppresses cyclooxygenase enzymes.	23
Allium sativum (Garlic)	Amaryllidaceae	Bulb	Allicin	Suppresses pro-inflammatory cytokines (IL-1α, TNF-α, IL-6, IL-8) in response to LPS stimulation.	20, 29
Paeonia lactiflora (Peony)	Paeoniaceae	Root (Hydro-ethanolic extract)	Paeoniflorin, Albiflorin, Paeonin	Inhibits pro-inflammatory cytokines.	20, 30
Ocimum basilicum (Basil)	Lamiaceae	Leaves (Hydro-distillation or Ethanolic extract)	1,8-Cineole, Linoleic acid, Eugenol	Suppresses synthesis of TNF-α, IL-6, and IL-1β cytokines.	20, 31
Salvia miltiorrhiza (Red Sage)	Lamiaceae	Rhizome (Aqueous extract)	Caffeic acid derivatives, Salvianolic acid (A and B)	Inhibits MAPK and NF-κB pathways, and reduces COX-2 and iNOS production.	20, 30, 32
Berberis vulgaris (Barberry)	Berberidaceae	Whole plant (Methanol extract)	Berberine	Reduces inflammatory cytokines.	20, 33
Lawsonia inermis (Henna)	Lythraceae	Bark, leaves (Alcoholic-methanolic, ethanolic, Hydro-	Lawsochylin, Kaempferol, Luteolin	Reduces LPS-induced nitrate generation in RAW 264.7 cells.	19, 34

		ethanolic extracts)			
Aloe barbadensis (Aloe Vera)	Liliaceae	Leaves (Petroleum ether, Ethanol extract)	Aloe emodin	Suppresses pro-inflammatory cytokines (IL-1β, TNF-α), inhibits inducible nitric oxide and PGE2 production.	13, 18, 35
Garcinia mangostana (Mangosteen)	Guttiferae	Fruit (Methanolic extract)	Xanthones (Mangostanin, Mangostanol)	Inhibits NO and COX-2 activity, halts MAPK and NF-κB activation.	10, 21, 36
Moringa oleifera (Drumstick)	Moringaceae	Leaves, Seeds, Root, Flowers, Bark (Ethanolic, Hydro-alcoholic extract)	Kaempferol, Quercetin	Inhibits LOX and COX activities.	8, 18, 37
Tribulus terrestris (Gokshura)	Zygophyllaceae	Root, fruit (Ethanolic extract)	Tyramines, Protodioscin	Reduces NO synthase, COX-2 expression, and inhibits IL-6 and TNF-α production.	13, 38
Commiphora wightii (Guggulu)	Burseraceae	Oleo-gum resin (Hydro-methanolic or Methanolic extract)	Guggulsterone, Guggulipid	Inhibits NO, PGE2 synthesis, and COX-2 activity, suppresses NF-κB expression.	13, 39
Punica granatum (Pomegranate)	Lythraceae	Peel (Hydro alcoholic extract)	Punicalagin, Ellagic acid, Granatin B, Punicic acid	Inhibits inflammatory mediators, suppresses prostaglandin production.	13, 40
Nigella sativa (Black Cumin)	Ranunculaceae	Seeds (Oil, ethanolic extract)	Thymoquinone	Inhibits IL-1β, TNF-α, COX-1,	13, 41, 42

				COX-2, and 5-LOX pathways.	
Achillea millefolium (Yarrow)	Asteraceae	Flower (Ethanolic extract)	Salicylic acid, Choline, Luteolin	Suppresses iNOS production and inhibits inflammatory proteases like HNE, MMP-2, and MMP-9.	43
Allium cepa (Onion)	Liliaceae	Bulbs (Aqueous or Methanolic extract)	Allicin, Quercetin	Inhibits synthesis of IL-1β, IL-1α, IL-4, IL-6, and TNF-α, and modulates lymphocyte activity. Reduces LOX and COX activity.	43, 44
Althaea officinalis (Marshmallow)	Malvaceae	Root, leaf, flower (Ethanolic or Aqueous extract)	Caffeic acid, Quercetin, Kaempferol	Prevents release of IL-6 and TNF-α cytokines.	43, 45, 46
Calendula officinalis (Marigold)	Asteraceae	Flower (Methanolic, Ethanolic, Hydro-alcoholic extract)	Palmitoyl esters of Faradiol, Triterpenoid	Inhibits NO production, reduces prostaglandins by blocking actions of IL-1β, TNF-α, IL-6, IFN-γ, COX-2, and 5-LOX activity.	43, 47, 48
Juglans regia L. (Walnut)	Juglandaceae	Leaves (Aqueous & ethanolic extract)	Protocatechuic acid, Gallic acid, Caffeic acid, Apigenin, Luteolin, Campesterol, Ursolic acid, 3-α-corosolic acid	Reduces prostaglandin and COX-2 levels and down regulates inflammatory cytokines	43, 49
Hypericum perforatum	Hypericaceae	Flowers (Lipophilic,	Hyperforin, Hypericin,	Inhibits inflammatory	43, 50

(St. John's Wort)		ethanolic, hydro-alcoholic extracts)	Quercetin, Chlorogenic acid, Amentoflavone, I3,II8-biapigenin	enzymes like iNOS, COX-2; suppresses IL-6 and PGE2; inhibits NF-κB signaling	
Matricaria chamomilla L. (Chamomile)	Asteraceae	Flowers (Aqueous extract)	Bisabolol, Sesquiterpenes	Suppresses NO and PGE2 synthesis; down regulates IL-10	43, 47
Mentha piperita L. (Peppermint)	Lamiaceae	Leaves (Essential oil & ethanolic extract)	Menthol, Menthofuran, 1,8-cineole, Luteolin, Luteolin-7-O-glucoside	Inhibits NO and PGE2 production and blocks pro-inflammatory cytokines (IL-6, TNF-α)	43, 51
Salix alba L. (White Willow)	Salicaceae	Bark (Methanolic and Aqueous extracts)	Salicin, Salicylic acid, Ferulic acid	Reduces levels of prostaglandins, histamine, serotonin, and bradykinin	43, 52
Mimosa pudica (Sensitive plant)	Mimosaceae	Leaves and roots (Methanol, Ethanol, Aqueous extract)	L-mimosine, Crocin, Jasmonic acid, Caffeic acid, Gallic acid, Ethyl gallate	Inhibits TNF-α activity, prostaglandin release, and COX enzyme expression	53, 54, 55
Artemisia vulgaris (Mugwort)	Asteraceae	Leaves (Methanolic extract)	6,7-Dimethoxycoumarin	Suppresses NO, TNF-α, and COX-2; inhibits LOX pathway	20, 56
Lonicera japonica (Honeysuckle)	Caprifoliaceae	Leaves & Flowers (Ethanolic & aqueous extract)	Linalool, Iridoids, Flavonoids, Chlorogenic acid, Luteolin	Inhibits COX-1/COX-2 and MAPK/NF-κB pathways; reduces IL-6, IL-8, TNF-α	20, 57, 58
Cassia occidentalis (Usaya ki Fali)	Caesalpiniaceae	Leaves and seeds (Ethanolic extract)	Aloe-emodin, Apigenin, Quinine, Methoxy-naphthalene	Suppresses IL-1β and TNF-α production	59, 60

Jasminum sambac Linn.	Oleaceae	Roots and Leaves (Ethanolic extract)	Jasminoids A-D, Salicylic acid, β-sitosterol, Iso-quercetin, Linalool	Inhibits cyclooxygenase (COX) activity	58, 61
Persicaria chinensis	Polygonaceae	Leaves (Methanolic extract)	Caffeic acid, Quercetin, Kaempferol	Reduces TNF-α, IL-6, and NO in response to LPS stimulation	58, 62
Solanum melongena (Brinjal)	Solanaceae	Leaves (Aqueous extract)	Ascorbic acid, Alanine, Arginine, Caffeic acid	Inhibits both COX and LOX inflammatory pathways	58, 63
Acacia catechu	Mimosaceae	Bark and heartwood (Aqueous extract)	Catechins, Tannins, Quercetin, Flavocoxid, Catechuic acid	Controls NO production and suppresses pro-inflammatory cytokines (TNF-α, IL-6, IL-1β)	12, 64, 65
Caesalpinia crista (Fever Nut)	Caesalpiniaceae	Seeds, Roots, Leaves, Bark (Ethanolic & Aqueous extract)	Caesalpinins, Oleic, Linoleic, Palmitic, and Stearic acids	Inhibits 5-LOX enzyme involved in inflammation	12, 66
Ginkgo biloba (Maidenhair Tree)	Ginkgoaceae	Leaves and Seeds (Ethanol & Ethyl acetate extracts)	Ginkgolide B, Ginkgetin, Bilobetin, Isoginkgetin	Inhibits NF-κB signaling and reduces inflammatory mediators such as NO, IL-6, TNF-α, COX-2	30, 67
Andrographis paniculata (Kalmegh)	Acanthaceae	Leaves (Alcoholic extract)	Andrographolide	Suppresses the production of pro-inflammatory cytokines, hinders leukocyte infiltration, and deactivates macrophages by modulating the	30, 68

				MAPK signaling pathway.	
Tripterygium wilfordii Hook. f (Thunder God Vine)	Celastraceae	Root	Triptolide, Celastrol	Inhibits NF-κB activity by blocking IκKα/β and preventing IκBα degradation. Regulates COX-2, TNF-α.	30, 69
Centella asiatica (Gotu Kola)	Apiaceae	Leaves (Methanolic extract)	Asiaticoside, Sapogenin, Asiatic acid, Madecassoside, Madecassic acid	Down regulates inflammatory mediators including PGE_2, IL-1β, IL-6, and TNF-α. Inhibits NF-κB and MAPKs while enhancing PPAR-γ expression.	68, 70
Magnolia liliflora Desr (Woody Orchid)	Magnoliaceae	Bark	Obovatol	Reduces NO synthesis and inhibits iNOS and COX-2 expression. Also interferes with NF-κB nuclear translocation and deactivates ERK and JNK signaling pathways.	68
Alstonia scholaris (Devil's Tree)	Apocynaceae	Leaves (Ethanol/ Methanol extracts)	Alstoprenyol, Picrinine, Scholaricine, Epischolaricin, Vallesamine, Perakine-N4-oxide, Vinorine-N4-oxide, Scholarisin I	Reduces levels of MDA, NO, and PGE_2. Inhibits COX-1, COX-2, and 5-LOX enzymes.	71, 72

Asparagus racemosus (Shatavari)	Liliaceae	Root and Leaves (Ethanolic extract)	Shatavarins (I–X), Asparagine, Arginine	Decreases myeloperoxidase activity and inhibits inflammatory cytokines including TNF-α, IL-6.	71
Cedrus deodara (Himalayan Cedar)	Pinaceae	Wood oil, Stem bark (Aqueous extract)	Methylacetophenone, Atantonl, Deodrin, Toxifolin	Suppresses the expression of COX-2, TNF-α, and NF-κB, thus reducing inflammation.	71, 73
Swertia chirayita (Chiretta)	Gentianaceae	Root, Stem (Ethanol and Aqueous extracts)	Amaroswerin, Amarogentin, Mangiferin, Swertiamarin, Sweroside, Xanthones (Isomangostin, Mangostin Triacetate)	Inhibits prostaglandin and bradykinin formation. Balances pro- and anti-inflammatory cytokines such as IL-1β, IL-10, IL-6, IFN-γ, and TNF-α.	71, 74
Vitex negundo (Nirgundi)	Verbenaceae	Leaves (Aqueous extract)	3,4,9-Trimethyl-7-propyldecanoic acid	Reduces histamine and prostaglandin synthesis, alleviates oxidative stress, and stabilizes cellular membranes in damaged tissues.	71, 75
Coriandrum sativum (Coriander)	Apiaceae	Seeds (Hydro-alcoholic extract)	γ-Linolenic acid	Inhibits inflammatory mediators such as cytokines and TNF-α.	12

5. Conclusion:

This review emphasizes the significant anti-inflammatory properties of various medicinal plants, which exert their effects through multiple mechanisms, such as modulating immune responses and inhibiting pro-inflammatory mediators. Analysis of numerous scientific studies confirms that phytochemicals from plant sources offer diverse biological activities to manage inflammation. These natural compounds represent a safer and more holistic alternative to synthetic anti-inflammatory drugs, which often carry adverse effects. The findings strongly support the continued exploration of herbal sources as potential candidates for the development of novel therapeutic agents. Overall, the role of medicinal plants in managing inflammation holds great promise and paves the way for future advancements in natural product-based drug discovery.

6. References:

1. Beg S, Swain S, Hasan H, Barkat MA, Hussain MS. Systematic review of herbals as potential anti-inflammatory agents: Recent advances, current clinical status and future perspectives. *Pharmacogn Rev*. 2011; 5(10):120–37.
2. Agarwal H, Nakara A, Shanmugam VK. Anti-inflammatory mechanism of various metal and metal oxide nanoparticles synthesized using plant extracts: A review. *Biomed Pharmacother*. 2019;109:2561–72.
3. Patil KR, Mahajan UB, Unger BS, Goyal SN, Belemkar S, Surana SJ, et al. Animal models of inflammation for screening of anti-inflammatory drugs: Implications for the discovery and development of phytopharmaceuticals. *Int J Mol Sci*. 2019;20(18):1–38.
4. Wei H, Kong S, Jayaraman J, Selvaraj D, Soundararajan P, Manivannan A. *Mentha arvensis* and *Mentha* × *piperita* – vital herbs with myriads of pharmaceutical benefits. *Horticulturae*. 2023;9:1–17.
5. Abdulkhaleq LA, Assi MA, Abdullah R, Zamri-Saad M, Taufiq-Yap YH, Hezmee MNM. The crucial roles of inflammatory mediators in inflammation: A review. *Vet World*. 2018;11(5):627–35.
6. Juhn SK, Jung M-K, Hoffman MD, Drew BR, Preciado DA, Sausen NJ, et al. The role of inflammatory mediators in the pathogenesis of otitis media and sequelae. *Clin Exp Otorhinolaryngol*. 2008;1(3):117–38.
7. Shaheen N, Azam A, Ganguly A, et al. Anti-inflammatory and analgesic activities of black cumin (BC, *Nigella sativa* L.) extracts in in vivo model systems. *Bull Natl Res Cent*. 2022;46:26. doi:10.1186/s42269-022-00708-0
8. Shingala Z, Chauhan B, Baraiya Z. A review on medicinal plants as a source of anti-inflammatory agents. *J Pharmacogn Phytochem*. 2021;10(6):364–71.
9. Ghasemian M, Owlia S, Owlia MB. Review of anti-inflammatory herbal medicines. *Adv Pharmacol Sci*. 2016;2016:1–11.
10. Gupta M, Singh N, Gulati M, Gupta R, Kalvatala SK, Kapoor B. Herbal bioactives in treatment of inflammation: An overview. *S Afr J Bot*. 2021;143:205–25.
11. Hah B, Seth AK. *Textbook of Pharmacognosy and Phytochemistry*. 1st ed. India: Elsevier; 2010.

12. Nunes CDR, Barreto Arantes M, Menezes de Faria Pereira S, Leandro da Cruz L, de Souza Passos M, Pereira de Moraes L, et al. Plants as sources of anti-inflammatory agents. *Molecules*. 2020;25(16):1–22.
13. Gandhi Y, Kumar R, Grewal J, Rawat H, Mishra SK, Kumar V, et al. Advances in anti-inflammatory medicinal plants and phytochemicals in the management of arthritis: A comprehensive review. *Food Chem Adv*. 2022;1:1–15.
14. Recio MC, Andujar I, Rios JL. Anti-inflammatory agents from plants: progress and potential. *Curr Med Chem*. 2012;19(14):2088–103.
15. Rao PV, Gan SH. Cinnamon: a multifaceted medicinal plant. *Evid Based Complement Alternat Med*. 2014;2014:1–12.
16. Kumar S, Malhotra S, Prasad AK, Van der Eycken EV, Bracke ME, Stetler-Stevenson WG, et al. Anti-inflammatory and antioxidant properties of *Piper* species: a perspective from screening to molecular mechanisms. *Curr Top Med Chem*. 2015;15(9):886–93.
17. Azab A, Nassar A, Azab AN. Anti-inflammatory activity of natural products. *Molecules*. 2016;21(10):1–19.
18. Kumar S, Bajwa BS, Singh K, Kalia AN. Anti-inflammatory activity of herbal plants: A review. *Int J Adv Pharm Biol Chem*. 2013;2(2):272–81.
19. Akhtar MA. Anti-inflammatory medicinal plants of Bangladesh—a pharmacological evaluation. *Front Pharmacol*. 2022;13:1–24.
20. Gonfa YH, Tessema FB, Bachheti A, Rai N, Tadesse MG, Singab AN, et al. Anti-inflammatory activity of phytochemicals from medicinal plants and their nanoparticles: A review. *Curr Res Biotechnol*. 2023;6:1–16.
21. Dhiman A, Singla C, Vishal, Kumar B. Natural medicinal resources for treating inflammatory conditions. *Int J Med Phar Sci*. 2021;11(9):1–10.
22. Semwal P, Rauf A, Olatunde A, Singh P, Zaky MY, Islam MM, et al. The medicinal chemistry of *Urtica dioica* L.: from preliminary evidence to clinical studies supporting its neuroprotective activity. *Nat Prod Bioprospect*. 2023;13(1):1–11.
23. Farag M, Pathan A, Aldoaij N. Literature review of 10 plant products for their anti-inflammatory, antioxidant, immunomodulatory, and antiviral properties. *NeuroPharmac J*. 2021;6(3):241–7.
24. Rudrapal M, Vallinayagam S, Aldosari S, Khan J, Albadrani H, Al-Shareeda A, et al. Valorization of *Adhatoda vasica* leaves: extraction, in vitro analyses and in silico approaches. *Front Nutr*. 2023;10:1–11.
25. Golechha M, Sarangal V, Ojha S, Bhatia J, Arya DS. Anti-inflammatory effect of *Emblica officinalis* in rodent models of acute and chronic inflammation: involvement of possible mechanisms. *Int J Inflamm*. 2014;2014:1–6.
26. Shrivastava S, Kaur J, Mehraj M, Feroz F, Chawla J, Kumari S. *Emblica officinalis* (Amla): a comprehensive review of the miracle berry. *Pharma Innov*. 2022;11(6):6–16.
27. Jeyasri R, Muthuramalingam P, Adarshan SK, Shin H, Ramesh M. Assessing the anti-inflammatory effects of *Bacopa*-derived bioactive compounds using network pharmacology and in vitro studies. *ACS Omega*. 2022;7(44):40344–54.
28. Kim H, Castellon-Chicas MJ, Arbizu S, Talcott ST, Drury NL, Smith S, et al. Mango (*Mangifera indica* L.) polyphenols: anti-inflammatory, intestinal microbial health benefits, and associated mechanisms of actions. *Molecules*. 2021;26(9):2732.

29. Savairam VD, Patil NA, Borate SK, Ghaisas MM, Shete RV. Allicin: a review of its important pharmacological activities. *Pharmacol Res Mod Chin Med*. 2023;8:1–13.
30. Wang YH, Zeng KW. Natural products as a crucial source of anti-inflammatory drugs: recent trends and advancements. *Tradit Med Res*. 2019;4(5):257–68.
31. Kamelnia E, Mohebbati R, Kamelnia R, El-Seedi HR, Boskabady MH. Anti-inflammatory, immunomodulatory and antioxidant effects of *Ocimum basilicum* L. and its main constituents: a review. *Iran J Basic Med Sci*. 2023;26(6):617–27.
32. Choi HG, Tran PT, Lee JH, et al. Anti-inflammatory activity of caffeic acid derivatives isolated from the roots of *Salvia miltiorrhiza* Bunge. *Arch Pharm Res*. 2018;41:64–70.
33. Kalmarzi RN, Naleini SN, Peluso I, et al. Anti-inflammatory and immunomodulatory effects of barberry (*Berberis vulgaris*) and its main compounds. *Oxid Med Cell Longev*. 2019;2019:1–10.
34. Moutawalli A, Benkhouili FZ, Doukkali A, Benzeid H, Zahidi A. The biological and pharmacologic actions of *Lawsonia inermis* L. *Phytomed Plus*. 2023;3(3):1–15.
35. Bałan BJ, Niemcewicz M, Kocik J, Jung L, Skopinska-Rozewska E, Skopinski P. Oral administration of *Aloe vera* gel, anti-microbial and anti-inflammatory herbal remedy, stimulates cell-mediated immunity and antibody production in a mouse model. *Cent Eur J Immunol*. 2014;39(2):125–30.
36. Abate M, Pagano C, Masullo M, Citro M, Pisanti S, Piacente S, et al. Mangostanin, a xanthone derived from *Garcinia mangostana* fruit, exerts protective and reparative effects on oxidative damage in human keratinocytes. *Pharmaceuticals (Basel)*. 2022;15(1):1–16.
37. Chiș A, Noubissi PA, Pop O-L, Mureșan CI, Fokam Tagne MA, Kamgang R, et al. Bioactive compounds in *Moringa oleifera*: mechanisms of action, focus on their anti-inflammatory properties. *Plants*. 2024;13(1):1–25.
38. Sudheendran N, Shajahan MA, Premlal S. Anti-inflammatory activity of root and fruit of Gokshura (*Tribulus terrestris* Linn.) in albino rats. *Int J Ayurveda Pharma Res*. 2017;5(7):1–4.
39. Sarup P, Bala S, Kamboj S. Pharmacology and phytochemistry of oleo-gum resin of *Commiphora wightii* (Guggulu). *Scientifica (Cairo)*. 2015;2015:1–14.
40. Parisi V, Santoro V, Donadio G, Bellone ML, Diretto G, Sandri C, et al. Comparative chemical analysis of eight *Punica granatum* L. peel cultivars and their antioxidant and anti-inflammatory activities. *Antioxidants*. 2022;11(11):2262.
41. Dwita LP, Yati K, Gantini SN. The anti-inflammatory activity of *Nigella sativa* balm sticks. *Sci Pharm*. 2019;87(1):3.
42. Bahrami M, Ghazavi A, Ganji A, Mosayebi G. Anti-inflammatory activity of *Silybum marianum* and *Nigella sativa* extracts on macrophages. *Rep Biochem Mol Biol*. 2021;10(2):288–301.
43. Radovanovic K, Gavaric N, Acimovic M. Anti-inflammatory properties of plants from Serbian traditional medicine. *Life (Basel)*. 2023;13(4):874.
44. Marefati N, Ghorani V, Shakeri F, Boskabady M, Kianian F, Rezaee R, et al. A review of anti-inflammatory, antioxidant, and immunomodulatory effects of *Allium cepa* and its main constituents. *Pharm Biol*. 2021;59(1):287–302.

45. Wang Z, Jiang X, Zhang L, Chen H. Protective effects of *Althaea officinalis* L. extract against N-diethylnitrosamine-induced hepatocellular carcinoma in male Wistar rats through antioxidative, anti-inflammatory, mitochondrial apoptosis and PI3K/Akt/mTOR signaling pathways. *Food Sci Nutr*. 2023;11(8):4756–72.
46. Bonaterra GA, Schmitt J, Schneider K, Schwarzbach H, Aziz-Kalbhenn H, Kelber O, et al. Phytohustil® and root extract of *Althaea officinalis* L. exert anti-inflammatory and anti-oxidative properties and improve the migratory capacity of endothelial cells in vitro. *Front Pharmacol*. 2022;13:948248.
47. Silva D, Ferreira MS, Sousa-Lobo JM, Cruz MT, Almeida IF. Anti-inflammatory activity of *Calendula officinalis* L. flower extract. *Cosmetics*. 2021;8(2):1–7.
48. Shahane K, Kshirsagar M, Tambe S, Jain D, Rout S, Ferreira MKM, et al. An updated review on the multifaceted therapeutic potential of *Calendula officinalis* L. *Pharmaceuticals (Basel)*. 2023;16(4):611.
49. Bhat AA, Shakeel A, Rafiq S, Farooq I, Malik AQ, Alghuthami ME, et al. *Juglans regia* Linn.: a natural repository of vital phytochemical and pharmacological compounds. *Life (Basel)*. 2023;13(2):380.
50. Klemow KM, Bartlow A, Crawford J, et al. Medical attributes of St. John's Wort (*Hypericum perforatum*). In: Benzie IFF, Wachtel-Galor S, editors. *Herbal Medicine: Biomolecular and Clinical Aspects*. 2nd ed. Boca Raton (FL): CRC Press/Taylor & Francis; 2011. Chapter 11.
51. Hudz N, Kobylinska L, Pokajewicz K, Horčinová Sedláčková V, Fedin R, Voloshyn M, et al. *Mentha piperita*: essential oil and extracts, their biological activities, and perspectives on the development of new medicinal and cosmetic products. *Molecules*. 2023;28(21):7444.
52. Shivatare RS, Kewatkar SM, Musale R, Lohakare P, Patil P, Choudhary D, et al. [Title not provided]. *Int J Pharm Sci Res*. 2021;12(6):3176–84.
53. Goli V, Bhaskar KV, Macharla SP, Bhaskar J, Suvarna Devi P, Ramchander T. Effects of anti-inflammatory activity of *Mimosa pudica*. *Asian J Pharm Res*. 2011;1(3):69–71.
54. Mistry S, Patidar R, Vyas V, Jena J, Dutt KR. Anti-inflammatory activity of *Mimosa pudica* Linn. (Mimosaceae) leaves: an ethnopharmacological study. *J Pharm Sci Res*. 2012;4(3):1789–91.
55. Adurosakina OE, Iweala EJ, Otikec JO, Dikec ED, Uchec ME, Owanta JI, et al. Ethnomedicinal uses, phytochemistry, pharmacological activities and toxicological effects of *Mimosa pudica* – a review. *Pharmacol Res Mod Chin Med*. 2023;7(4):1–18.
56. Siwan D, Nandave D, Nandave M. *Artemisia vulgaris* Linn: an updated review on its multiple biological activities. *Future J Pharm Sci*. 2022;8(47):1–14.
57. Tang X, Liu X, Zhong J, Fang R. Potential application of *Lonicera japonica* extracts in animal production: from the perspective of intestinal health. *Front Microbiol*. 2021;12:1–14.
58. Hsu HF, Hsiao PC, Kuo TC, Chiang ST, Chen SL, Chiou SJ, et al. Antioxidant and anti-inflammatory activities of *Lonicera japonica* Thunb. var. *sempervillosa* Hayata flower bud extracts prepared by water, ethanol and supercritical fluid extraction techniques. *Ind Crops Prod*. 2016;89:543–9.

59. Sami A, Usama M, Saeed MM, Akram M. Medicinal plants with non-steroidal anti-inflammatory-like activity. *Mediterr J Pharm Pharm Sci*. 2021;1(3):1–8.
60. Shyeed MA, Bashera MA, Sazal OS, Ali MM, Hossain MP, Mondol HSK, et al. Investigation of wound healing and anti-inflammatory activity of *Senna occidentalis* leaf extract, and in silico screening for both activities. *Pharm Sci Adv*. 2023;1:1–10.
61. Dhote V, Dangi U, Mandloi AS, Soni M, Shukla DN, Kawadkar M, et al. Preferential cyclooxygenase inhibition by *Jasminum sambac*: a possible relationship with potent anti-arthritic activity. *J Tradit Complement Med*. 2020;11(3):217–27.
62. Hossen MJ, Baek KS, Kim E, Yang WS, Jeong D, Kim J-H, et al. *Persicaria chinensis* methanolic extract targeting Src/Syk/NF-κB: in vivo and in vitro anti-inflammatory activities. *J Ethnopharmacol*. 2015;159:9–16.
63. Umamageswari MS, Maniyar YA. Evaluation of anti-inflammatory activity of aqueous extract of leaves of *Solanum melongena* Linn. in experimental animals. *J Clin Diagn Res*. 2015;9(1):FF01–3.
64. Adhikari B, Aryal B, Bhattarai BR. A comprehensive review on the chemical composition and pharmacological activities of *Acacia catechu* (L.f.) Willd. *J Chem*. 2021;2021:1–11.
65. Waseem U, Jafri SR, Khalid S, Qureshi F, Majeed N, Akif U. Anti-inflammatory activity of *Acacia catechu*-bark aqueous solution in aspirin-induced gastric ulcer in rodents. *Int J Community Med Public Health*. 2021;8(12):5649–54.
66. Chan EWC, Tangah J, Shigeyuki Baba S, Chan HT, Kainuma M, Inoue T. *Caesalpinia crista*: a coastal woody climber with promising therapeutic values. *J Appl Pharm Sci*. 2018;8(3):133–40.
67. Noor-E-Tabassum, Das R, Lami MS, Chakraborty AJ, Mitra S, Tallei TE, et al. *Ginkgo biloba*: a treasure of functional phytochemicals with multimedicinal applications. *Evid Based Complement Alternat Med*. 2022;2022:8288818.
68. Li X, Yuan W, Wu J, Zhen J, Sun Q, Yu M. Andrographolide, a natural anti-inflammatory agent: an update. *Front Pharmacol*. 2022;13:920435.
69. Tong X, Qiao Y, Yang Y, Liu H, Cao Z, Yang B, et al. Applications and mechanisms of *Tripterygium wilfordii* Hook. f. and its preparations in kidney diseases. *Front Pharmacol*. 2022;13:1–15.
70. Saha S, Guria T, Singha T, Maity TK. Evaluation of analgesic and anti-inflammatory activity of chloroform and methanol extracts of *Centella asiatica* Linn. *ISRN Pharmacol*. 2013;2013:1–6.
71. Rodríguez-Yoldi MJ. Anti-inflammatory and antioxidant properties of plant extracts. *Antioxidants (Basel)*. 2021;10(6):921.
72. Zhao M, Cai J, Yang Y, Xu J, Liu WY, Akihisa T, et al. Traditional uses, chemical composition and pharmacological activities of *Alstonia* R. Br. (Apocynaceae): a review. *Arab J Chem*. 2023;16:1–24.
73. Pathak H, Pathania S, Mehta S, Sharma R. *Cedrus deodara* (Roxb.): a review on the recent update on its pharmacological and phytochemical profile. *RPS Pharm Pharmacol Rep*. 2023;2(3):1–6.
74. Das SC, Bhadra S, Roy S, Saha KS, Islam MDS, Bachar SC. Analgesic and anti-inflammatory activities of ethanolic root extract of *Swertia chirata* (Gentianaceae). *J Adv Biotechnol Pharm Sci*. 2012;5(1):31–36.

75. Sivapalan S, Dharmalingam S, Ashokkumar V, Venkatesan V, Angappan M. Evaluation of the anti-inflammatory and antioxidant properties and isolation and characterization of a new bioactive compound, 3,4,9-trimethyl-7-propyldecanoic acid, from *Vitex negundo*. *J Ethnopharmacol*. 2024;319:117314.