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Review Article

## Anti-Inflammatory Potential of Medicinal Plants in the Management of Inflammatory Diseases: A Review of Mechanisms and Bioactive Compounds

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### Abstract

Inflammatory diseases, such as arthritis, asthma, and inflammatory bowel disease, present ongoing health challenges due to their chronic nature and the complexity of immune responses involved. While synthetic anti-inflammatory drugs are commonly used, they are often associated with adverse effects that drive a growing interest in natural therapeutic alternatives. Medicinal plants, recognized for their anti-inflammatory potential, offer promising avenues for safer treatment options. This review explores the current status and systematic strategies for discovering anti-inflammatory drugs from phytoconstituents found in medicinal plants. Bioactive compounds such as flavonoids, alkaloids, terpenoids, and polyphenols exhibit significant anti-inflammatory activity by interacting with key molecular pathways. These include the inhibition of pro-inflammatory cytokines, downregulation of COX and LOX enzymes, and reduction of oxidative stress. Despite their potential, challenges like bioavailability, lack of standardization pose obstacles to widespread clinical adoption. Recent advances in high-throughput screening, computational biology, and formulation technologies are paving the way for more targeted and efficient phytoconstituent-based drug discovery. This review highlights not only the therapeutic promise of phytochemicals but also emphasizes integrated research strategies necessary for their successful development into clinically approved anti-inflammatory agents.

**Keywords:** Natural therapeutics, Bioactive compounds, Inflammation management, Oxidative stress reduction.

### INTRODUCTION:

The Latin verb "inflammare," which means "to ignite" or "to set ablaze," is where the word "inflammation" originates, refers to a complex biological process involving the body's vascular tissue reacts to several adverse stimuli, including pathogens, pollens, irritants, and injured cells, by producing a variety of chemical mediators. It provides a protective reappearance that

encourages tissue healing. Inflammation can occasionally lead to extremely serious, chronic conditions like hay fever and rheumatoid arthritis, which can be fatal <sup>1,2</sup>. Therefore, appropriate representative action must be made to combat it. Acute and chronic infections are the two main categories of infections, to put it briefly. Table 1 lists the clinical alterations, etiology, mediators, and danger seen in acute and chronic infections.

**Table 1:** Categories of Inflammation <sup>3</sup>

Type of Inflammation	Causative agents	Important mediators	Major cells involved	Duration of occurrence
Acute Inflammation (Short lasting)	Pathogens, injured tissues	Monocytes, macrophages, neutrophils	Vasoactive amines, eicosanoids	Few days
Chronic Inflammation (Long lasting)	Non-degradable pathogens, persistent	Mononuclear cells (monocytes, macrophages,	Reactive oxygen species, hydrolytic	From months to years

	foreign bodies, or by autoimmune reactions	lymphocytes, plasma cells), fibroblasts	enzymes, cytokines, growth factors, IFN- $\gamma$	
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Leukocyte emigration, capillary infiltration, and enhanced vascular permeability are all linked to Acute Inflammation while, Chronic Inflammation is associated with neutrophils, monocytes, macrophages, fibroblast activation, proliferation, and fibrosis in addition to infiltration of mononuclear immune cells.

When tissue is injured, the body's protective response is Inflammation. Pathogens can arise from a variety of situations, including abrasions, chemical irritation, cell deformation or disruptions, and extremely high or low temperatures. The four cardinal indications of inflammation are usually redness, pain, warmth, and swelling. Exudation and function loss can also occasionally result from inflammation <sup>4</sup>. 5-hydroxytryptamine (5-HT), histamine tissue necrosis factor- $\alpha$ , IL-8 leukotrienes, prostacyclins, prostaglandins, lymphokines, and chemokines like interferon- $\alpha$  (IFN- $\alpha$ ), IL-1, and histamine are few of the potent substances that are involved in the inflammatory process <sup>5</sup>. To elicit a complimentary response in response to external stimuli, these mediators initiate a number of chemical pathways and events. If inflammation is not correctly treated with early first aid, a correct diagnosis, and medication therapy, they may even result in death. Asthma, rheumatoid arthritis, vasculitis, and glomerulonephritis are a few conditions where immune system mutations can be highly detrimental <sup>6,7</sup>. As a result, anti-inflammatory drug therapy needs to be sufficiently effective to reduce inflammation's severity. Up until now, a lot of synthetic drugs have been used to swiftly cure infections and diseases associated with them. Nevertheless, a number of clinical studies have demonstrated that these artificial substances are no longer safer. Approximately 90% of drugs used to treat inflammation, according to reports, include side effects, iatrogenic responses, and drug-related toxicities that make treatment more difficult <sup>8-10</sup>. As a result, a change from the use of pharmaceuticals to natural therapy has been noted in the field of immunological treatment. Table 2 provides a quick list of some illnesses caused by infiltration.

### THE INFLAMMATORY CASCADE:

Damage to tissue can affect endothelial cells, mast cells, platelets, and neutrophils, macrophage which lead to influx of  $Ca^{+2}$  into these cells during process of cell injury, there is lots of  $Ca^{+2}$  which moves inside the cytoplasm. This increases in intracellular  $Ca^{+2}$  level which activates many enzymes within cells and one of these enzymes activates phospholipase A2. Arachidonic acid is present in bound form to phospholipid membrane in each of these above cells of body. On activation of Phospholipase A2 enzyme arachidonic acid gets converted into free arachidonic acid. Once Arachidonic acid is in free form, it transforms quickly into bioactive mediators. It is also known as Eicosanoids (Prostaglandins, Thromboxin, Leukotrienes, Lipoxins.) Basically, Arachidonic acid is metabolized by two pathways i.e. cyclooxygenase pathway (COX) and 5-Lipoxygenase pathway (LOX) as shown in fig 1.

**Table 2:** Potentially harmful consequences of anti-inflammatory drugs <sup>3</sup>

Name of Drug	Drug related toxic effects
Aspirin	Gastric irritation, bleeding
Diclofenac sodium	Epigastric pain, kidney damage
Ibuprofen	Gastric erosion
Nimesulide	Fulminant hepatic failure
Rofecoxib	Cardiovascular risk (myocardial infarction)
Mephenamic acid	Diarrhoea, bleeding, hemolytic anaemia
Phenylbutazone	Epigastric distress, ulceration
Corticosteroids	Weakens host defence mechanism, tuberculosis

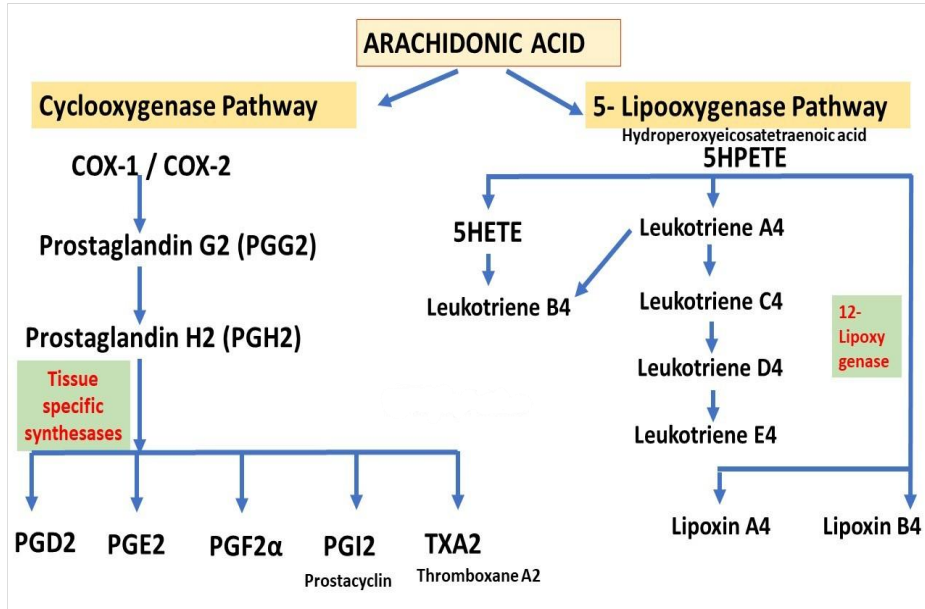


Figure 1: chemical mediators of inflammation

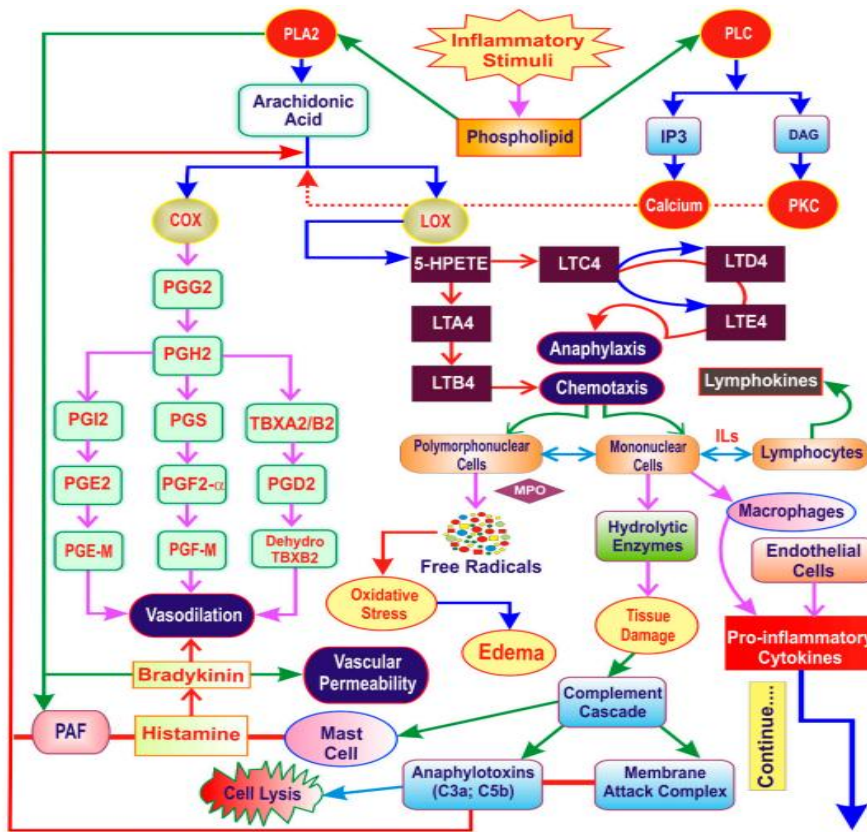


Figure 2: (A)

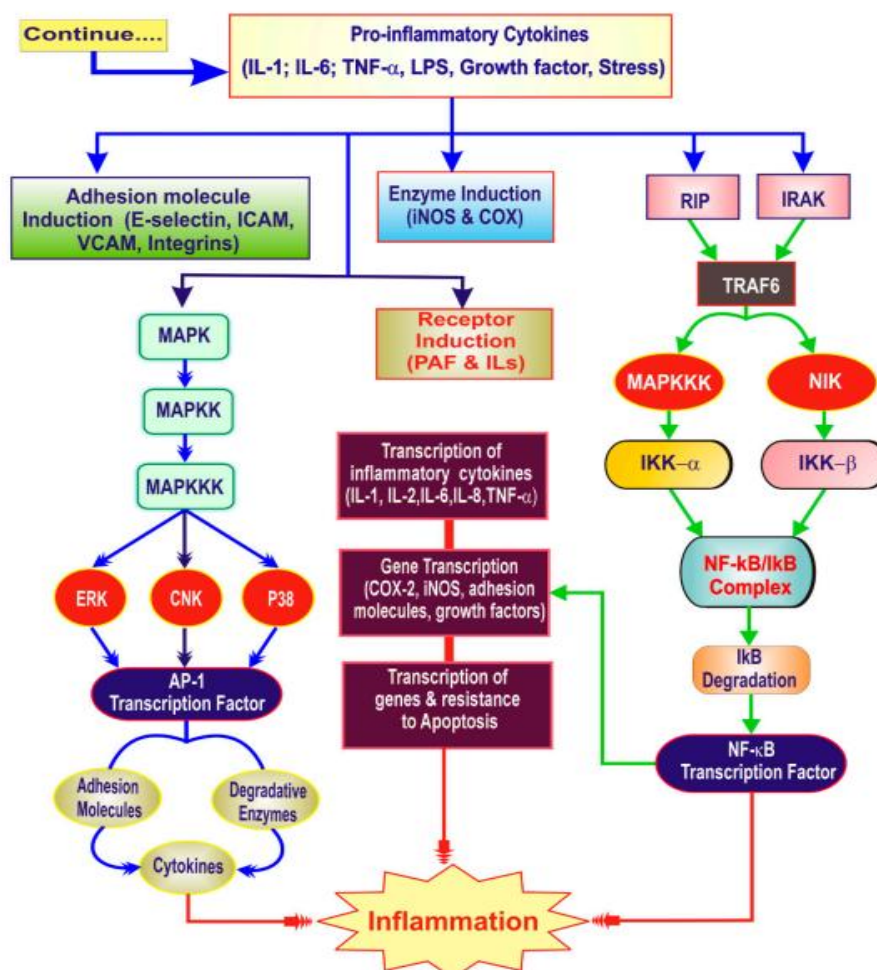


Figure 2B: Inflammatory Cascade <sup>11</sup>

## INFLAMMATORY BIOMARKERS:

**Lipid Derived Mediators:** Arachidonic acid (AA) is a vital component of all body cells and one of the main precursors of eicosanoid production. When several phospholipase enzymes, most notably phospholipase A2 (PLA2) are activated, AA is liberated from membrane phospholipid. AA is broken down into several oxygenated compounds known as eicosanoids via a variety of metabolic processes. The lipoxygenases (LOX) form leukotrienes (LTs) and lipoxins (LXs), while the cyclooxygenases (COX) form prostaglandins (PGs) and thromboxane (prostanoids). Epoxyeicosatrienoic acids (EETs), are another product of cytochrome P450 enzymes <sup>12</sup>. Numerous disorders are associated with the regulation of several inflammatory and homeostatic processes by eicosanoids<sup>13</sup>. Lipid-derived mediators have a well-established proinflammatory effect<sup>14</sup>. Vasodilatation, bronchoconstriction, mucus production, and vascular permeability are all linked to the prostaglandins. However, leukotrienes have been shown to be bronchoconstrictors, vasodilators, and vascular permeability stimulators as shown in fig 1. Nuclear activation and superoxide generation are linked to leukotriene B4 (LTB4). It also enhances the early gene transcription of other cytokines and raises the synthesis of interleukin-6 (IL-6)<sup>15, 16</sup>. Inflammatory and allergy processes rely heavily on leukotrienes (LTs), and 5-lipoxygenase (5-LOX) is essential for their formation. One effective method for treating dermatitis and

psoriasis is to block 5-LOX<sup>17</sup>. Numerous inflammatory cell types, such as neutrophils, eosinophils, macrophages, and endothelial cells, produce platelet-activating factor (PAF), which promotes chemotaxis, platelet activation, and bronchoconstriction<sup>15</sup>.

**Proinflammatory Cytokines:** The inflammatory and immunological processes are regulated by cytokines. Tumor necrosis factors, interleukins, colony stimulating factors, and interferons are components of the cytokine class. Cytokines modulate the expression of adhesion molecules as well as cell division, proliferation, death, immunoglobulin synthesis, and chemotaxis in target cells<sup>15</sup>. Activation of monocytes and macrophages results in the production of proinflammatory cytokines, including IL-1 $\beta$ , IL-6, and tumor necrosis factor- $\alpha$  (TNF- $\alpha$ ). TNF- $\alpha$  affects the pathogenesis of rheumatoid arthritis as well as the dissemination of cancer cells <sup>18</sup>. Inducing bone resorption, IL-1 $\beta$  stimulates lymphocytes<sup>19</sup>. TNF- $\alpha$  and IL-1 $\beta$  also bind to leukocytes in circulation and regulate the synthesis of adhesion molecules<sup>20</sup>. Furthermore, transcription is subsequently started by cytokines and intracellular signalling cascades <sup>21</sup>.

**Vasoactive Mediators:** Hepatocytes and mast cells store the majority of histamine, which is a widely generated and distributed proinflammatory mediator<sup>15,22</sup>. A rapid increase in permeability follows tissue damage due to histamine release. Endothelial cells are stimulated by

histamine to contract, which widens inter-endothelial junctions and allows protein and fluid passage<sup>23</sup>. Histamine induces oedema development and enhances gastric acid output in addition to increasing vascular permeability<sup>15</sup>. Higher concentrations of histamine result in leukocyte adhesion and endothelial cell oedema. Consequently, the primary mediator responsible for the early vascular alterations brought on by an inflammatory response is histamine<sup>24</sup>. Another vasoactive amine called serotonin is mostly located in the brain, intestinal, and platelet tissues. It increases vascular permeability and induces smooth muscle contraction<sup>23</sup>.

**Hydrolytic Enzymes:** Throughout the course of inflammation, proinflammatory cells that have been stimulated release a build-up of proteolytic enzymes. Elastin is the major elastic substance found in blood vessels, the lungs, and proteins such as immunoglobulins, collagen, and proteoglycans. Elastin hydrolysis and endothelial migration of activated proinflammatory mediators are caused by the release of human leukocyte elastase (HLE) from stimulated polymorphonuclear leukocytes (PMNL)<sup>15</sup>.

**Reactive Oxygen Species (ROS):** Numerous diseases have pathophysiological processes that are related to oxidative stress and inflammatory processes. They are essential components of cellular defence mechanisms. The inflammatory cells' production of ROS intensifies the consequences of oxidative stress<sup>25, 26</sup>. Proinflammatory gene expression can be induced and intracellular signalling pathways can be started by ROS<sup>27,28</sup>. The degradation of structures that are important to function

is brought on by an excess of radicals and peroxides, which also compromises the body's natural defences against oxidative stress and free radical scavengers<sup>15</sup>.

**Transcription Factors:** The main modulator of the immune system and the inflammatory response is nuclear factor-kappa beta (NF-κB). It controls the transcription of genes related to inflammatory pathways, cellular stress response, adhesion, proliferation, immune response, apoptosis, and tissue remodelling. The transcription of genes encoding COX-2, iNOS, growth factor receptors, immunological receptors, cell adhesion molecules, and inflammatory cytokines like IL-1β, IL-2, IL-6, IL-8, and TNF-α is regulated by NF-κB. Sulfasalazine, large doses of aspirin, and glucocorticoids all decrease NF-κB activation. Thus, for the pharmacological therapy of inflammatory diseases, NF-κB presents an interesting therapeutic target<sup>29</sup>.

**Complement System:** When the complement cascade is activated, the membrane collision complex and the anaphylatoxins C3a and C5a are produced<sup>19</sup>. Strong chemoattractant C5a increases oxidative stress as well as the synthesis, expulsion, and formation of leukotrienes, prostaglandins, and cytokines. Additionally, it encourages the acquisition of T lymphocytes, neutrophils, eosinophils, and monocytes, among other inflammatory cells<sup>30</sup>. Therefore, products that are activated by complement, such as C5a, have biological activities that are prominent and trigger the inflammatory cascade. Table 1 summarizes a number of inflammatory mediators.

**Table 3:** Various inflammation mediators and their respective pathophysiological roles<sup>11</sup>

Category	Mediator	Source Organ/Cells	Mechanism
Vasoactive amines	Histamine	Basophils, gastric cells, enterochromaffin cells, histaminergic nerve	Vasodilatation and increased vascular permeability
	Serotonin	Intestine, blood, spleen, nervous system	Vasodilatation and increased vascular permeability (less potent than histamine)
Arachidonic acid metabolites	Prostaglandins (PGs)	Formed by the metabolism of arachidonic acid by cyclooxygenases (COX)	Enhanced vascular permeability, fever, sensory nerve stimulation, and pain amplification. PGE2 causes production of oedema and erythema
	Leukotrienes (LTs)	Formed by the metabolism of arachidonic acid by lipoxygenase (LOX)	LTB4 stimulates neutrophil chemotaxis, enhanced neutrophil-endothelial interactions, neutrophil activation, degranulation and release of various inflammatory mediators, enzymes and free radicals
	Thromboxane (TX)	Granuloma tissues, macrophages, human synovial tissues, Thromboxane A2 (TXA2) is present in platelets and circulating leukocytes	Platelet aggregation, smooth muscle contraction
Platelet activating factor (PAF)	PAF	Liberated by macrophages, endothelial cells, platelets and	Initiates cardinal features of inflammation, expression of adhesion molecules, platelet aggregation, formation

			of leukotrienes, chemotaxis, sensitization of sensory nerves to pain
Kinins	Substance p (Sub-p)	Released from sensory nerves	Increased microvascular permeability, neutrophil accumulation, potentiates responses to bradykinin, serotonin, prostaglandin, and ATP
	Bradykinin	Plasma precursor protein kininogen produces bradykinin through kallikrein.	Increased vascular permeability, sensory nerve ending stimulation, inflammatory mediator release, activation of NF- $\kappa$ B, induction of cytokine gene expression
	Interleukins (ILs)	Produced by activated lymphocytes and macrophages	Up-regulation of adhesion molecule expression, stimulation of pro-inflammatory mediator release
Cytokines	Tumor Necrosis Factor- $\alpha$ and $\beta$ (TNF- $\alpha$ , $\beta$ )	Activated macrophages/monocytes, fibroblasts, mast cells, natural killer (NK) cells, T and B lymphocyte	Stimulation of PGE <sub>2</sub> , collagenase, IL-1 production, fever; induction of acute-phase reactant protein production, adhesion molecule up-regulation, cytokine induction, chemokine synthesis
	Transforming Growth Factor- $\beta$ (TGF- $\beta$ )	T cells, platelets, monocytes	Attraction of monocytes and other leukocytes to the site of injury, increased cell adhesion
	Interferons (IFN- $\alpha$ and $\beta$ )	IFN- $\alpha$ is produced by leukocytes and IFN- $\beta$ is produced by fibroblasts	Activation of macrophages and mononuclear phagocytes
Clotting system	Thrombin	Blood	Mobilization of p-selectin, the release of chemokines, adhesion molecule expression, induction of COX-2, production of PGs, PAF and nitric oxide
Complement system	Anaphylatoxins C3a and C5a	Complement proteins reside as inactive forms in plasma	Potentiate inflammation by binding to receptors on mast cells,
Miscellaneous	Nitric oxide (NO)	Leukocytes, endothelial cells, sensory nerve cells	Vasodilation and cytotoxicity
	Reactive Oxygen Species (ROS)	Phagocytic leukocytes like, neutrophils, monocytes, macrophages, eosinophils	Vascular leakage, chemotaxis, endothelial damage, oxidative stress, activation of transcription factors like nuclear transcription factor- $\kappa$ B (NF- $\kappa$ B)

### THE THERAPEUTIC POTENTIAL OF PHYTOCONSTITUENTS IN TARGETING INFLAMMATION:

Inflammation plays a major role in the pathophysiological mechanisms that cause the onset and progression of many diseases. In order to treat inflammatory illnesses, safer and more effective treatments can be found through systematic studies of phytoconstituents for their anti-inflammatory properties. For many years, medicinal plants have been utilized to treat inflammatory diseases. It is established that the phytoconstituents in these therapeutic plants are responsible for their anti-inflammatory benefits. The ability of phytoconstituents to impact multiple stages of pathophysiological processes accounting for their anti-inflammatory characteristics. The production of proinflammatory mediators, leukocyte migration, and complement cascade activation are examples of specific

components of the inflammatory pathways that the phytoconstituents may interact with to provide their anti-inflammatory properties. These pathways may be the main cellular targets<sup>31</sup>. In chronic inflammatory disorders, many biomolecules such as proinflammatory cytokines, matrix-degrading enzymes, and signalling pathway components are interesting candidates for therapeutic intervention. During an inflammatory phase, the primary objective of phytoconstituents is to control the expression of genes that generate chemicals that promote inflammation. Anti-inflammatory molecules may function through one or more of the many pathways<sup>32</sup>.

### THE CURRENT STATUS AND SYSTEMATIC STRATEGY FOR PHYTOCONSTITUENTS-BASED ANTI-INFLAMMATORY DRUG DISCOVERY:

The majority of pharmaceuticals on the market are natural compounds, and even synthetic or semi-synthetic medications have natural origins. Numerous scientific research conducted worldwide have shown the medicinal properties of plants used in many traditional systems. The scientific community has shown increased attention in establishing a connection between a plant's pharmacological activity and its phytoconstituents and botanical characteristics<sup>33</sup>. The discovery of novel drugs has been significantly aided by plant extracts and extracted phytoconstituents. Among the most complex tasks to accomplish in the drug discovery process and human medication development is identifying feasible, robust, and druggable lead candidates. Because screening hits are transformed into drug candidates, it involves expertise and experience<sup>34</sup>. It takes a lot of money, effort, and time to create new drugs. The typical time span between a novel lead's development and its clinical debut as a treatment agent is 12 years. Principal obstacles in the search for novel drugs are the decline in new drug approvals and the rising expense of development. The success rate of drug development remains unchanged despite the introduction of combinatorial chemistry which has rationalized the process. Drug research primarily focuses on finding novel chemical compounds with promising druggability properties<sup>35</sup>. Throughout ancient times, natural substances have served as the foundation for beneficial medicinal medicines. The discovery of novel treatments has allowed plants to continue helping humanity. There is evidence supporting the intriguing anti-inflammatory activities of a number of phytoconstituents, including flavonoids, triterpenoids, alkaloids, steroids, and phenols. In modern medicine, the primary sources of drug discovery seem to be the active ingredients derived from natural materials utilized as traditional remedies. Despite the progress in the field of allopathy, plants remain a potential source of therapeutic substances in both traditional and contemporary medical systems. As a result, efforts to isolate pure chemicals from their natural

sources and characterize those that are pharmacologically active are still ongoing<sup>36</sup>. Innovations in new technology and chemical variety have revolutionized the process of finding drugs from natural sources. Recent technological advancements have successfully addressed the inherent limitations traditionally associated with natural products. These developments have enabled the systematic investigation of natural compounds, reinforcing their significance as valuable lead structures in the field of drug discovery and development. The medicinal chemists can now synthesize the compounds via complete synthesis rather than isolating them from the plants thanks to the structural interpretation of phytoconstituents. Natural lead potency has increased and production costs have dropped as a consequence<sup>37</sup>.

#### NATURAL ANTI-INFLAMMATORY PROPERTIES OF PLANTS:

Herbal remedies operate by a symphonic method, in contrast to contemporary allopathic medications that include a single active component that targets a single channel. Many different compounds found in plants interact with certain elements within the complex biological system in an interdependent way<sup>38</sup>. Medicinal herbs have historically offered a wide range of biologically active substances that have been used extensively as pure substances or as ingredients for a variety of medical treatments. Because of the toxicity and negative effects of allopathic pharmaceuticals, the usage of herbal remedies is growing in popularity. Strong therapeutic medications are developed in large part from medicinal plants. More than 1.5 million physicians of traditional medicine employ medicinal plants in preventative, promotional, and therapeutic uses. With the greatest collection of medicinal plants in the world, India should continue to be a major producer of pharmaceutical and cosmetic raw materials as well as the bioactive ingredients utilized in their manufacture<sup>39</sup>.

**Table 4:** Anti-inflammatory attributes of traditional plant remedies.

Plants scientific Names	Common Name	Plant Part Used	Type of Extract	Marker Compound	Ref
<i>Aegle marmelos</i>	Bael	Roots, fruits	Aqueous, Ethyl acetate	Marmelosin	40
<i>Adhatoda vasica</i>	Malabar nut	leaves	Methanolic, Ethanolic	Vasicine	41
<i>Allium sativum</i>	Garlic	Leaves, Cloves	Garlic clove powder	Allin, Allicin	42
<i>Borago officinale</i>	Borage	Seed oil	Seed oil	Gamma- linoleic acid	43
<i>Bryophyllum pinnatum</i>	Goethe plant	Leaves	Ethanolic extract	Rutin, luteolin	44
<i>Boswellia serrata</i>	Salai guggul	Gum resin	Hydroalcoholic extract	Boswellic acid	45
<i>Camelia sinesis</i>	Green tea	Leaves	Ethanolic extract	Catechin, Epigallocatechin	46
<i>Capsicum annum</i>	Chilli	Fruit	Ethyl acetate extract	capsaicin	47
<i>Cinnamomum camphora</i>	Camphor	Leaves	Methanolic extract	Camphor, $\alpha$ -pinene, $\beta$ -pinene	48

<i>Cassia fistula</i>	Golden shower tree	flowers	Isolated rhein	Rhein	<b>49</b>
<i>Curcuma longa</i>	Turmeric	Rhizomes	Dichloromethane	Curcumin	<b>50</b>
<i>Commiphora mukul</i>	Guggul	Gum resin	Hydroalcoholic	Guggulsterone	<b>51</b>
<i>Elaeagnus angustifolia</i>	Russian olive, silverberry	Fruits	Methanolic extract	Catechin, Epicatechin	<b>52</b>
<i>Eucalyptus globulus</i>	Nilgiri	Oil from leaves	Oil	1,8-cineole	<b>53</b>
<i>Gaultheria procumbens</i>	American wintergreen	Leaves from oil	Hydroalcoholic extract	Quercetin, catechin	<b>54</b>
<i>Garcinia cambogia</i>	Malabar Tamarind	Fruit	Ethanollic extract	Hydrocitric acid	<b>55</b>
<i>Glycyrrhiza glabra</i>	Liquorice	Roots	Ethanollic	Glycyrrhizin	<b>56</b>
<i>Hibiscus tilliaceus</i>	Bhola	Leaves and bark	Aqueous, Methanollic	Tiliaceic acid	<b>57</b>
<i>Linum usitatissimum</i>	Flaxseed, Linseed	seeds	Oil from seeds	$\alpha$ -linolenic acid	<b>58</b>
<i>Madhuca longifolia</i>	Mahudo	Seeds, leaves	Oil from seed, aqueous extract of leaves	Oleic acid	<b>59</b>
<i>Mentha piperita</i>	Mint	Leaves	Ethanollic acid	Menthol	<b>60</b>
<i>Moringa oleifera</i>	Drumstick	Leaves, seeds, roots	Ethanollic, hydroalcoholic extract	Quercetin, Gallic acid, Rutin	<b>61</b>
<i>Ocimum sanctum</i>	Tulsi, Basil	Leaves	Essential oil from leaves	Ursolic acid, Eugenol	<b>62</b>
<i>Olea europaea</i>	Olive	Fruit	Methanollic extract	oleuropein	<b>63</b>
<i>Persea americana</i>	Avocado	Fruit, seeds	Lipid extracted from fruit and seeds	Palmitic acid, oleic acid, linoleic acid	<b>64</b>
<i>Pinus roxburghii</i>	Chir pine	Bark	Alcoholic extract	$\alpha$ -pinene, $\beta$ -pinene	<b>65</b>
<i>Pluchea indica</i>	Camphorweed	Leaves, roots	Ethanollic, chloroform extract	Quercetin	<b>66</b>
<i>Podophyllum emodi</i>	mayapple	Roots, rhizome	Isolated podophyllotoxin derivates	podophyllotoxin	<b>67</b>
<i>Piper nigrum</i>	Black pepper	Fruits	Hexane, ethanollic extract	Piperine	<b>68</b>
<i>Ribes nigrum</i>	Blackcurrant	Berries, buds, leaves	Ethanollic extract	Cyanidin-3-O-glucoside, Delphinidin-3-O-glucoside	<b>69</b>
<i>Rosa canina</i>	Dog rose	Rose hip	Hydroalcoholic extract	Linolic acid, $\alpha$ -linolic acid	<b>70</b>
<i>Rosmarinus officinalis</i>	Rosemary	Aerial parts essential oil	Ethanollic extract	Rosmarinic acid, caffeic acid, carnosol	<b>71</b>
<i>Salix alba</i>	willow	Bark	Ethanollic extract	Salicin	<b>72</b>
<i>Salvia officinale</i>	Sage	Oil from aerial parts	Chloroform extract	Borneol, camphor, caryophyllene, cineole	<b>73</b>
<i>Solanum xanthocarpum</i>	Kantakari	Fruit	Aqueous extract	Campesterol, chlorogenic acid	<b>74</b>

<i>Symphytum officinale</i>	Comfrey	Leaves, roots	Ethanollic extract	Allantoin	<b>75</b>
<i>Tinospora cordifolia</i>	Guduchi, giloy	Whole plant	Ethanollic, aqueous extract	B-sitosterol, stigmasterol	<b>76</b>
<i>Trigonella foenumgraecum</i>	Fenugreek	Seeds	Petroleum ether extract	Linolenic acid, galactomannan	<b>77</b>
<i>Vitex negundo</i>	Nirgundi	Leaves	Methanollic, petroleum ether extract	Caryophyllene epoxide	<b>78</b>
<i>Withania somnifera</i>	Ashwagandha	Roots	Aqueous extract	Withanolides	<b>79</b>
<i>Zingiber officinale</i>	Ginger	Rhizome	Essential oil	Gingerol, Shogaol	<b>80</b>

## CONCLUSION:

Medicinal plants offer a compelling alternative for managing inflammatory diseases due to their multi-targeted actions and relatively low risk of side effects compared to synthetic drugs. The diverse bioactive compounds in these plants can modulate several pathways involved in inflammation, making them promising complementary therapies for chronic inflammatory conditions. However, for these natural treatments to be fully integrated into mainstream healthcare, further research is essential to address challenges in standardization, bioavailability, and regulatory frameworks. Establishing consistent quality control and dosage guidelines will be crucial to harness the full therapeutic potential of medicinal plants, enabling their safe and effective use alongside conventional treatments in the management of inflammatory diseases.

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