



Medicinal Plants with Anti-Inflammatory Activity

Sahil Bhadu Temkar¹, Harshala damdhar², Dr Rupali Tasgaonkar³

¹ Student Of B Pharm at Yadavrao Tasgaonkar Institute Of Pharmacy, Mumbai University , Bhivpuri Road, Karjat, Maharashtra, India.

² Prof. at Yadavrao Tasgaonkar Institute Of Pharmacy, Mumbai University , Bhivpuri Road , Karjat, Maharashtra, India.

³ Principal Of Yadavrao Tasgaonkar Institute Of Pharmacy, Mumbai University, Bhivpuri Road, Karjat, Maharashtra, India

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Corresponding Author:
Sahil Bhadu Temkar

Abstract:

Inflammation is a largely conserved physiological response that protects apkins from injury, infection, and chemical annoyances(12). Acute inflammation is salutary, promoting towel form and pathogen concurrence; still, habitual inflammation is a major contributor to conditions similar as rheumatoid arthritis, cardiovascular diseases, diabetes, neurodegenerative conditions, seditious bowel complaint, and certain cancers(19). Encyclopedically, habitual seditious conditions pose a significant health and profitable burden, with millions affected annually(5).

Conventional anti-inflammatory curatives, including NSAIDs, corticosteroids, and biologics, are effective but frequently beget adverse effects similar as gastrointestinal bleeding, nephrotoxicity, hepatotoxicity, and immunosuppression(3).

Medicinal plants have surfaced as effective alternatives due to their rich phytochemical diversity, including flavonoids, alkaloids, terpenoids, saponins, and phenolic acids(21). Bioactive composites similar as curcumin, boswellic acids, gingerols, withanolides, thymoquinone, and catechins modulate multiple seditious pathways, including NF- κ B, MAPK, JAK/ STAT, COX- 2, and pro-inflammatory cytokines like TNF- α , IL- 1 β , and IL- 6(14). Preclinical studies in vitro and in vivo confirm the anti-inflammatory eventuality of these composites, while clinical trials demonstrate safety and efficacy in colorful seditious diseases(2). This review provides a detailed account of medicinal plants with anti-inflammatory exertion, their molecular mechanisms, experimental and clinical evidence , and prospects for development into standardized phytopharmaceuticals(25)..

Keywords: Medicinal plants, Anti-inflammatory activity, Phytochemicals, Herb medicine, Natural antiinflammatory agents, Plant-based therapeutics

1. Introduction

Inflammation is a complex defense medium that involves vascular, cellular, and molecular factors to cover the body from dangerous stimulants(17).

Acute inflammation is rapid-fire and generally tone- limiting, characterized by greenishness, heat, swelling, pain, and loss of function. crucial cells involved include neutrophils, macrophages, mast cells, dendritic cells, and T lymphocytes(4).

habitual inflammation results from patient stimulants and leads to towel redoing, fibrosis, and organ dysfunction(9).

Epidemiological data reveal that habitual seditious conditions affect hundreds of millions worldwide, contributing mainly to morbidity, mortality, and healthcare costs(11).



For illustration, rheumatoid arthritis affects of the global population, while cardiovascular conditions linked to inflammation account for million deaths annually(1).

Synthetic anti-inflammatory medicines similar as NSAIDs inhibit COX enzymes to reduce prostaglandin conflation, while corticosteroids suppress multiple cytokine pathways(13).

Despite their efficacy, dragged use is associated with serious adverse goods, including gastrointestinal bleeding, hypertension, renal impairment, osteoporosis, and increased threat of infections(3).

Traditional drug systems, including Ayurveda, Unani, and Traditional Chinese Medicine, have long employed plants to treat seditious conditions(23).

These remedies are now validated by ultramodern pharmacology, which demonstrates that factory- deduced phytochemicals act on multiple molecular targets to reduce inflammation while minimizing adverse goods(8).

The multifaceted nature of phytochemicals makes medicinal plants an seductive source for developing new, safer anti-inflammatory medicines(22).



Mechanisms of Inflammation and Herbal Modulation

Inflammation is initiated by the recognition of pathogens or damage- associated molecular patterns(PAMPs and DAMPs) by pattern recognition receptors similar as risk- suchlike receptors(TLRs) on vulnerable cells(6).

This triggers the release of pro-inflammatory intercessors, including prostaglandins, leukotrienes, nitric oxide, reactive oxygen species (ROS), and cytokines similar as TNF- α , IL-1 β , and IL-6 (12). Recap factors like NF- κ B, AP-1, and STAT are latterly actuated, upregulating genes garbling seditious enzymes (COX-2, iNOS) and adhesion moles (18). habitual activation of these pathways can lead to towel damage, fibrosis, and carcinogenesis (16).

Medicinal plants modulate inflammation through multiple mechanisms Enzyme inhibition COX-1, COX-2, and 5-LOX are inhibited by composites similar as curcumin, boswellic acids, and gingerols (7).

Antioxidant activity Phytochemicals scavenge ROS and stabilize redox-sensitive recap factors (8).

Transcriptional regulation NF- κ B, AP-1, and MAPK signaling are suppressed, reducing pro-inflammatory gene expression (11). Cytokine modulation TNF- α , IL-1 β , and IL-6 are downregulated, while anti-inflammatory cytokines like IL-10 are upregulated (15).

Immune cell regulation Phytochemicals modulate macrophage polarization, T cell isolation, and neutrophil exertion (22). Epigenetic effects Some composites impact histone acetylation and miRNA expression, further controlling seditious gene expression (5).

The capability of medicinal plants to act on multiple targets underlines their efficacy in treating both acute and habitual inflammation (19).

2. Major Medicinal plants with Anti-Inflammatory activity

Curcuma longa (Turmeric)

Curcumin is the main polyphenol in turmeric, inhibiting COX-2, LOX, iNOS, and NF- κ B pathways (20). It reduces pro-inflammatory cytokines (TNF- α , IL-1 β , IL-6) and ROS. Preclinical studies demonstrate repression of paw edema, colitis, and arthritis in Animal models (7). Clinical trials report bettered pain scores and seditious labels in osteoarthritis, ulcerative colitis, and metabolic pattern (14). Bioavailability is enhanced by nanoparticle, liposome, or phospholipid phrasings (19).

Zingiber officinale (Ginger)

Gingerols and shogaols inhibit prostaglandin and leukotriene conflation and suppress NF- κ B activation (16). In vitro studies show reduced cytokine product in LPS-stimulated macrophages, while in vivo studies demonstrate dropped paw edema and oxidative stress (1).

Clinical trials indicate enhancement in osteoarthritis pain and systemic seditious labels (12).

Boswellia serrata (Indian Frankincense)

Boswellic acids inhibit 5-LOX and leukotriene conflation, reducing granuloma conformation and paw edema in experimental models (2).

Clinical trials in osteoarthritis and rheumatoid arthritis show bettered common mobility, pain reduction, and dropped CRP and ESR (10).

Withania somnifera (Ashwagandha)

Withanolides suppress NF- κ B and MAPK pathways, reducing TNF- α , IL-1 β , and IL-6 (25). Animal studies show reduced paw edema, lower vulnerable cell infiltration, and dropped oxidative stress (19). Clinical evidence demonstrates efficacy in arthritis, stress-related inflammation, and habitual fatigue pattern (5).

Nigella sativa(Black Seed)

Thymoquinone modulates NF- κ B, COX- 2, and iNOS(5). Preclinical studies show dropped paw edema, colitis, and systemic inflammation. Clinical trials report lowered CRP and seditious cytokines in rheumatoid arthritis, metabolic pattern, and asthma cases(9).

Moringa oleifera

Flavonoids and phenolic acids inhibit ROS, TNF- α , IL- 1 β , and IL- 6(23). Animal models demonstrate reduced carrageenan- convinced paw edema and granuloma conformation. Clinical studies indicate bettered antioxidant status and reduced systemic seditious labels(21).

Camellia sinensis(Green Tea)

EGCG inhibits NF- κ B, JAK/ STAT, and MAPK signaling(12). Preclinical studies show reduced cytokine product and edema, while clinical trials indicate bettered endothelial function and lower CRP, IL- 6, and TNF- α (18).

Azadirachta indica(Neem)

Nimbidin and azadirachtin inhibit histamine, serotonin, prostaglandin, and leukotriene pathways(15). Preclinical studies report reduced paw edema, granuloma conformation, and leukocyte infiltration(11).

3. Preclinical and Clinical evidence

Preclinical evidence

Animal models and cell culture studies have constantly demonstrated the anti-inflammatory eventuality of these medicinal plants (9).

Curcumin Suppresses paw edema in carrageenan- convinced models, reduces colitis in DSS- convinced models, inhibits cytokine stashing in LPS- stimulated macrophages(14).

Ginger Gingerols reduce TNF- α , IL- 6, and prostaglandin E2 situations in in vitro and in vivo studies, dwindling inflammation and oxidative stress(1).

Boswellia Boswellic acids reduce leukocyte infiltration, granuloma conformation, and seditious cytokines in arthritic rats(10).

Ashwagandha Withanolides drop edema, oxidative stress, and vulnerable cell infiltration, modulating both ingrain and adaptive impunity(25).

Nigella sativa Thymoquinone reduces systemic inflammation, oxidative stress, and towel damage in experimental colitis and arthritis(5).

Moringa Flavonoid-rich excerpts reduce paw edema, neutrophil infiltration, and seditious cytokines in rodent models(23).

Green Tea EGCG reduces NF- κ B activation, cytokine product, and oxidative stress in preclinical cardiovascular and arthritis models(12).

Neem Nimbidin inhibits prostaglandin and leukotriene conflation, reducing granuloma conformation and seditious infiltrates(15).

Clinical evidence

Human trials support the efficacy of these medicinal plants (19).

Curcumin 500 mg doubly daily for 8 – 12 weeks bettered pain scores and dropped CRP and ESR in osteoarthritis cases(20).

Boswellia 300 – 400 mg formalized excerpt bettered common mobility and reduced pain in rheumatoid arthritis(2).

ginger 1 – 2 g diurnal reduced arthritis pain, morning stiffness, and systemic seditious labels(16).

Ashwagandha 300 – 500 mg/ day formalized excerpt bettered seditious biomarkers and reduced stress- convinced inflammation(25).

Nigella sativa 1 – 2 g/ day seed greasepaint reduced TNF- α and CRP situations in cases with metabolic pattern and arthritis(5).

Moringa Supplementation bettered oxidative status and lowered TNF- α and IL- 6 in humanstudies(21).

Green Tea Daily consumption reduced CRP, IL- 6, and bettered endothelial function(18).

Neem Limited clinical studies showed reduced inflammation in gingivitis and skin conditions(15).

4. Future Perspectives and Challenges

Despite promising results, several challenges must be addressed for the clinical restatement of medicinal factory curatives(7)

Standardization Variability in phytochemical content due to factory species, terrain, civilization, and birth styles hampers reproducibility(12).

Bioavailability numerous active composites(curcumin, EGCG, boswellic acids) have low oral bioavailability. Nanoparticles, liposomes, phytosomes, andco-administration with adjuvants(like piperine) can ameliorate immersion(21).

Safety and Herb – Drug relations Careful evaluation is demanded to help adverse relations, especially in cases on conventional curatives(18).

Polyherbal phrasings Combining multiple plants may give synergistic goods, targeting multiple seditious pathways, but requires rigorous clinical confirmation(16).

Regulatory Hurdles Establishing guidelines for civilization, birth, quality control, and clinical testing is pivotal for nonsupervisory blessing(19).

Novel Technologies Molecular docking, metabolomics, and omics- grounded approaches can identify bioactive composites and prognosticate mechanisms of action(25).

Herbal Medicine acclimatizing herbal curatives grounded on patient genetics, microbiome, and complaint profile may enhance efficacy(5).

Future research should integrate traditional knowledge, ultramodern pharmacology, and advanced delivery systems to optimize the remedial eventuality ofanti-inflammatory medicinal plants (22).

5. Conclusion

Medicinal plants give safe and effective strategies for managing seditious diseases(14). crucial plants , including *Curcuma longa*, *Zingiber officinale*, *Boswellia serrata*, *Withania somnifera*, *Nigella sativa*, *Moringa oleifera*, *Camellia sinensis*, and *Azadirachta indica*, act through multiple molecular pathways, including NF- κ B, MAPK, JAK/ STAT, COX- 2, and cytokine regulation(7). Preclinical and clinical studies support their efficacy and safety, but challenges remain in standardization, bioavailability, clinical confirmation, and nonsupervisory blessing(23).

Integrating traditional knowledge with ultramodern pharmacology and advanced delivery technologies offers openings to develop new, safe, and effective phytopharmaceuticals(2).

The evidence summarize in this review highlights that both preclinical and clinical studies explosively support theanti-inflammatory efficacy of plants similar as *Curcuma longa*, *Zingiber officinale*, *Boswellia serrata*, *Withania somnifera*, *Nigella sativa*, *Moringa oleifera*, *Camellia sinensis*, and *Azadirachta indica*(19).

These plants demonstrate remedial benefits in diseases like arthritis, colitis, asthma, and metabolic inflammation with minimum adverse goods(18).

In conclusion, medicinal plants represent not just remnants of traditional mending but the foundation for coming-generationanti-inflammatory medicine development(17).

Through continued scientific confirmation, technological invention, and ethical commercialization, these botanicals can offer safer, more affordable, and sustainable druthersfor combating the global burden of inflammation- related conditions(15).

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