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A Review on Analgesic Properties of Herbal Extracts

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Abstract: Herbal extracts have long been investigated as sources of analgesic (pain-relieving) agents. This review summarizes preclinical mechanisms, clinical evidence, safety considerations, and research gaps for several widely studied analgesic herbs: curcumin (turmeric), eugenol (clove), ginger (Zingiber officinale), Boswellia serrata, and willow bark (Salix spp.). Overall, strong preclinical evidence supports anti-inflammatory and nociception-modulating effects for these botanicals; clinical trials show promising but heterogeneous results, with limitations including variable preparations/doses and small sample sizes. Future work needs larger, well-controlled clinical trials, standardized extracts, and pharmacokinetic / pharmacodynamic characterization.

Substances employed for relieving pain but not causing unconsciousness are analgesics. The term analgesic comes from ancient Greek roots: "an-" means without, while "-algos" signifies pain. Medications of analgesic nature influence different parts of the brain and spinal cord as well as other nerve tissues throughout the body. Different types of pain relievers include both artificial ones such as opioid medications derived from plants like Aloe vera, licorice root, ginger rhizome, cinnamon leaf, boswellia resin, willow bark (from Salix species), and various herbal extracts containing compounds known for their anti-inflammatory properties. Furthermore. The critique provides insights into various pain-relieving substances derived from nature.

Keywords: *Herbal Analgesic, Plant-Based Pain Relief, Medicinal Plants – Glycyrrhiza Glabra, Zingiber Officinale Analgesic, Eugenia Caryophyllata, Withania Somnifera, Matricaria Pubescens, Ocimum Sanctum, Boswellia Serrata, and Willow Bark.*

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I. INTRODUCTION

Pain is one of the most common symptoms prompting individuals to seek medical care. Conventional analgesics such as non-steroidal anti-inflammatory drugs (NSAIDs) and opioids are effective but are associated with adverse effects including gastrointestinal irritation, renal toxicity, cardiovascular risks, tolerance, and dependence during long-term use. These limitations have encouraged growing interest in herbal medicines as safer and culturally acceptable alternatives or adjuncts for pain management. Medicinal plants have been used for centuries in traditional systems such as Ayurveda, Traditional Chinese Medicine, and Unani medicine for the relief of pain and inflammation.

Herbal Analgesics typically exert their effects through multi-target mechanisms including inhibition of inflammatory mediators, modulation of nociceptive pathways, antioxidant activity, and interaction with ion channels and neurotransmitters. In recent decades, advances in phytochemistry and pharmacology have enabled the scientific validation of many traditional claims[1][2][3].

Pain is defined by the International Association for the Study of Pain (IASP) as an unpleasant feeling that involves both senses and emotions, and it is linked to real or possible harm to body tissues, or it is described as if there was harm.

Pain can be split into two types based on how long it lasts: acute and chronic. Chronic pain lasts longer than six months, while acute pain lasts less than six months.

Acute pain comes from damage to body tissues. It is short-term and the cause is often easy to find. Pain happens when there is inflammation, which is caused by tissue damage or damage to nerves. Nerve damage can happen because of surgery, cancer, infection, fractures, diabetes, or chemotherapy.

Chronic pain is long-lasting and often comes and goes. It is usually harder to treat than acute pain.

Nociceptors are pain receptors found outside the spinal cord, in the dorsal root ganglion. These sensory nerve endings look like small branches, as described by Theken KN in 2018.

Analgesics are medicines used to reduce pain without making someone lose consciousness.

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The word 'analgesic' comes from Greek, where 'an' means 'without' and 'algos' means 'pain'. These medicines work in both the central nervous system and the peripheral nervous system.

➢ *Injury → Inflammatory mediators →Nociceptor sensitization → Pain*

- *Herbal extract acts by:*

COX/LOX inhibition

↓

↓ Prostaglandins & cytokines

↓

Ion channel modulation

↓

Reduced pain transmission

↓

Analgesic effect

Prostaglandins are made by the COX-2 enzyme. This enzyme comes out from cells that are damaged and helps create a feeling of pain. It does this by connecting with certain receptors that are linked to G-proteins and also by raising the level of CAMP inside the cells. Today, people use non-steroidal anti-inflammatory drugs to manage pain. These drugs work quickly to relieve pain, but they have significant side effects, which is a big problem when using them (Theken KN, 2018).

These drugs can cause stomach issues, itching, blurred vision, dizziness, skin rashes, and harm to the liver. Non-steroidal anti-inflammatory drugs tend to be more costly. To reduce both their side effects and expenses, researchers are exploring natural medicines derived from herbs. Certain active ingredients in herbs that help reduce pain include volatile oils like monoterpenes and sesquiterpenes, coumarin, alkaloids, organic acids, glycoside steroids, limonenes, cineols, saponins, phenolic compounds such as thymol and carvacrol, flavonoids, and quercetin[4]. Herbs that contain flavonoids work by blocking the cyclooxigenase enzyme and tannins. The chemicals like iridoids and flavonoids found in the herb extracts are responsible for its pain-relieving effects. Cinnamon extracts contain a monoterpene called linalool, which affects pain receptors and helps reduce pain. Phenols such as eugenol stop calcium from entering cells, which reduces the feeling of pain. In ginger root, the active compound is gingerol, which strongly inhibits prostaglandins. It also works by lowering blood vessel permeability and reducing pain-related signals, making it the main pain-relieving component of ginger. The analgesic effects of ziziphora clinopodioiedes, which comes from the Lamiaceae family, come from its ability to block the production of acids and prostaglandins, and it also affects the arachidonic acid pathway and opioids[5].

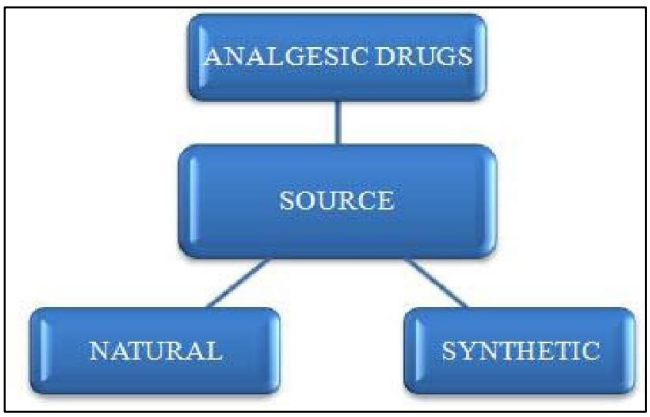

Fig 1 Source of Analgesic Drugs.

➢ *Synthetic Drugs:*

Many medicines are made in a lab and have pain-relieving effects, such as paracetamol, diclofenac, NSAIDs, ibuprofen, and COX-2 inhibitors.

➢ *Natural Analgesic Drugs:*

There are also many herbs that have pain-relieving properties and are taken from nature.

➢ *Opioid Analgesics:*

Opioid drugs are a type of narcotic that comes from opium. Opium is made from the dried latex of the opium poppy (Biological source: Papaver somniferum). These drugs work by affecting opioid receptors in the central nervous system and are often used for pain relief, especially in conditions like cancer where severe pain is present[6].

➢ *Objectives*

- *Pain Reduction:*

Alleviate pain without causing loss of consciousness, using substances derived from plants like ginger (Zingiber officinale), clove (Eugenia caryophyllata), or eucalyptus.

- *Anti-inflammatory Action:*

Many herbs contain compounds that reduce inflammation, a key source of pain, by inhibiting inflammatory mediators like prostaglandins.

- *Numbing/Soothing Effects:*

Provide topical relief through ingredients like camphor and menthol, creating a cooling or numbing sensation.

- *Central Nervous System (CNS) Modulation:*

Some extracts can affect CNS pathways, similar to opioids, to alter pain perception.

- *Holistic & Natural Approach:*

Offer an integrated, plant-based option for pain management, often alongside other lifestyle factors, as seen in Ayurvedic traditions.

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II. ANALGESIC HERBAL EXTRACTS

A. *Curcuma longa (Turmeric)*

Curcumin, the principal polyphenolic compound of Curcuma longa, has been extensively studied for its anti-inflammatory and analgesic properties. Preclinical studies demonstrate that curcumin inhibits COX-2, lipoxygenase, inducible nitric oxide synthase, and NF-κB, leading to reduced production of prostaglandins and pro-inflammatory cytokines[7][8][9].

Animal models of inflammatory and neuropathic pain have shown significant reductions in pain behaviors following curcumin administration. Clinically, curcumin has shown efficacy in reducing pain and improving function in osteoarthritis, rheumatoid arthritis, and postoperative pain. However, its poor oral bioavailability remains a major limitation, prompting the development of enhanced formulations such as curcumin-piperine combinations, nanoparticles, and phospholipid complexes[10][11][12].

➢ *Drug Profile:*

Fig 1 Curcuma longa (Turmeric)

- *Synonyms:*
  Haldi
- *Biological Source:*
  Dried Rhizome part of Curcuma Longa
- *Family:*
  Zingiberaceae
- *Colour:*
  Bright Yellow-Orange
- *Chemical Constituents:*
  Curcumin (Polyphenol).
- *Properties:*
  Potent anti-inflammatory, analgesic, antioxidant.
- *Mechanism:*

✓ Inhibits TNF-α, IL-6, NF-κB[13].
✓ Reduces inflammation-induced pain.

- *Uses:*

✓ Arthritic pain.
✓ Relieve muscle pain and support joint health.
✓ Post-exercise inflammation.
✓ Widely used as spice in cooking.
✓ Taken as a dietary supplement to support overall health and wellbeing.

- *Extract:*
  Hydro-alcoholic or standardized curcumin extract.

B. *Syzygium aromaticum (Clove)*

Clove contains eugenol as its major bioactive constituent. Eugenol exhibits local anesthetic, analgesic, and anti-inflammatory effects. It acts by blocking voltage-gated sodium channels, inhibiting prostaglandin synthesis, and reducing inflammatory mediator release[14][15][16].

Clove oil has been traditionally used in dentistry for toothache and pulpitis. Preclinical studies confirm its analgesic activity in thermal and chemical pain models. While topical and dental uses are well established, systemic clinical evidence is limited due to safety concerns at higher doses, including mucosal irritation and hepatotoxicity[17].

➢ *Drug Profile:*

Fig 2 Syzygium aromaticum (Clove)

- *Synonyms:*
  Laung
- *Biological Source:*
  Dried part of Syzygium aromaticum.
- *Active Constituent:*
  Eugenol
- *Family:*
  Myrtaceae
- *Color:*
  Reddish Brown

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- *Length:*
  About 13 to 19 mm.

- *Properties:*
  Analgesic, local anesthetic, anti-inflammatory, antimicrobial.

- *Mechanism:*

✓ Blocks voltage-gated sodium channels → numbing effect.
✓ Inhibits COX pathways → reduces prostaglandin synthesis[17].

- *Uses:*

✓ Toothache relief.
✓ Topical pain management.
✓ Used for sore.
✓ throat and digestive issues.
✓ Used widely as a spice, have a strong, aromatic flavor & fragrance.
✓ Oral pain.

- *Extracts:*
  Hydro-alcoholic or essential oil (steam distillation).

C. Zingiber Officinale (Ginger)

Ginger rhizomes contain gingerols and shogaols, which possess anti-inflammatory and analgesic properties. These compounds inhibit COX and LOX enzymes and reduce prostaglandin and leukotriene synthesis[18][19][20].

Animal studies show significant analgesic effects in models of inflammatory pain. Clinical trials suggest that ginger may reduce pain associated with osteoarthritis, dysmenorrhea, delayed-onset muscle soreness, and postoperative pain. However, results are variable, and the analgesic effect is generally moderate compared to standard NSAIDs[21][22].

Drug Profile:

Fig 3 Zingiber Officinale (Ginger)

- *Synonyms:*
  Adarak, Sunthi.

- *Biological Source:*
  Dried rhizome of Zingiber officinale.

- *Family:*
  Zingiberaceae.

- *Colour:*
  Deeper orange or brown.

- *Active Constituents:*
  Gingerols, shogaols.

- *Properties:*
  Anti-inflammatory, analgesic, antioxidants.

- *Mechanism:*

✓ Inhibits COX-2 and LOX enzymes → decreases inflammatory mediators.
✓ Reduces oxidative stress → lowers pain sensation.

- *Uses:*

✓ Muscular pain
✓ Headache
✓ Menstrual cramps
✓ Joint pain.
✓ Reducing nausea
✓ Aiding digestion
✓ Acts as an anti-inflammatory

- *Extract:*
  Ethanolic extract commonly used in tablet formulations.

D. Boswellia Serrata (Frankincense)

Boswellia serrata resin contains boswellic acids, which selectively inhibit 5-lipoxygenase, thereby reducing leukotriene-mediated inflammation. Preclinical studies demonstrate significant anti-inflammatory and analgesic effects[23][24].

Clinical studies indicate that Boswellia extracts may improve pain and joint function in osteoarthritis and inflammatory bowel disease-associated pain. Despite promising results, many studies suffer from small sample sizes and lack of standardization[25].

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➢ *Drug Profile:*

Fig 4 Boswellia Serrata (Frankincense)

- *Synonyms:*

Indian Frankincense, Indian Olibanum, Salai Guggal, Shallaki, Kundar, Loban.

- *Biological Source:*

Dried oleo-gum-resin exuded from incisions in the bark/trunk of the Boswellia serrata tree.

- *Family:*

Burseraceae

- *Colour:*

Milky-white or pale-yellow when fresh, but the dried gum resin tears can range from golden brown to dark brown or dark greenish-brown.

- *Active Constituents:*

Pentacyclic triterpene acids known as Boswellic acids (BAs), including;

✓ β-boswellic acid.
✓ Acetyl-β-boswellic acid.
✓ 11-keto-β-boswellic acid (KBA).
✓ 3-O-acetyl-11-keto-β-boswellic acid (AKBA), which is considered the most potent anti-inflammatory agent.
✓ Other constituents include essential oils (rich in monoterpenes like α-pinene), diterpenes, and polysaccharides.

- *Properties:*

Anti-inflammatory, anti-arthritic, analgesic (pain-relieving), anti-diarrheal, anti-asthmatic, and immunomodulatory properties.

- *Mechanism:*

The active boswellic acids primarily inhibit the enzyme 5-lipoxygenase (5-LOX), which is responsible for the synthesis of pro-inflammatory mediators called leukotrienes. They also suppress NF-κB activation and can inhibit matrix metalloproteinases (MMPs), which break down cartilage.

- *Uses:*

Used in traditional and modern medicine to treat inflammatory conditions such as:

✓ Osteoarthritis and rheumatoid arthritis.
✓ Inflammatory bowel diseases (Crohn's disease and ulcerative colitis).
✓ Asthma and bronchitis.
✓ Skin diseases like eczema and psoriasis.

- *Extract:*

The useful part is the oleo-gum-resin extracted from the tree. Extracts are often standardized to contain a specific percentage of total boswellic acids (e.g., 60% or 65%) or specifically AKBA (e.g., 30%).

E. Willow Bark (Salix alba)

Willow bark contains salicin, a natural precursor of acetylsalicylic acid. It exerts analgesic and anti-inflammatory effects through inhibition of prostaglandin synthesis[26][27].

Historically used for fever and pain, willow bark extracts have demonstrated efficacy in lower back pain and osteoarthritis in clinical trials. Variability in extract composition and dosing remains a challenge for consistent clinical outcomes.

➢ *Drug Profile:*

Fig 5 Willow Bark (Salix alba)

- *Synonyms:*

White willow bark, brittle willow, European willow, black willow, and Salix cortex.

- *Biological Source:*

Dried bark from the young branches or twigs of various Salix species.

- *Family:*

Salicaceae.

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- *Colour:*

The dried bark is typically Grey-brown and deeply fissured in older trees. The inner bark is often a pale, yellowish-brown colour.

- *Active Constituents:*

Phenolic glycoside Salicin. Other important compounds contributing its effect include Salicortin, flavonoids (quercetin, naringenin, rutin, epicatechin), tannins and various polyphenols and phenolic acids.

- *Properties:*

✓ Analgesic (Pain-relieving)
✓ Anti-inflammatory
✓ Anti-Pyretic (fever- reducing)
✓ Antioxidant
✓ Antiseptic/Antimicrobial
✓ Astringent (due to tannins).

- *Mechanism:*

✓ Salicylic acid inhibits COX enzyme → decreases prostaglandins synthesis.

- *Uses:*

✓ Relief of low back pain.
✓ Management of osteoarthritis and other rheumatic conditions.
✓ Alleviation of headaches and general muscle pain.
✓ Treatment of fever and symptoms of the common cold or flu.
✓ In cosmetic products, used for skin exfoliation, reducing oil, and anti-aging properties.

F. Withania somnifera (ashwagandha)

Withania somnifera (ashwagandha) has well-documented analgesic (pain- relieving) properties, supported by traditional use, animal studies and some human clinical trials, primarily attributed to active compounds like withanolides and withaferin A, which also provide anti-inflammatory and anti-arthritic benefits by targeting pain pathways[28][29][30].

Withania somnifera (Ashwagandha) is an adaptogenic herb used in Ayurveda for stress, anxiety, cognitive issues, inflammation, and general vitality, containing active compounds like withanolides that show neuroprotective, anti-inflammatory, immunomodulatory, and anti-cancer properties, acting as a general tonic to combat stress-related ailments and improve overall function, though traditional use varies and requires modern validation.

➢ *Drug Profile:*

Fig 6 Withania somnifera (ashwagandha)

- *Synonyms:*

Ashwagandha, Winter cherry, Indian Ginseng.

- *Biological Source:*

Dried roots and stem bases of the plant Withania somnifera (L.) Dunal, a small evergreen shrub.

- *Family:*

Solanaceae (Nightshade family).

- *Colour:*

Roots are typically whitish-brown to creamy white internally and yellowish externally.

- *Active Constituents:*

The primary active constituents are steroidal lactones known as withanolides (especially withaferin A and withanolide A and D) and various alkaloids (such as withanine and somniferine). Glycowithanolides and sitoindosides are also significant.

- *Properties:*

The plant is classified as an adaptogen, which helps the body cope with stress. It also possesses potent analgesic, anti-inflammatory, antioxidant, neuroprotective, immunomodulatory, anti-cancer, and cardioprotective properties.

- *Mechanism:*

Mechanisms include modulating inflammatory markers (e.g., inhibiting NF-κB and TNF-α), scavenging reactive oxygen species (ROS), enhancing GABAergic activity in the brain for anxiolytic effects, and inducing apoptosis in cancer cells[31].

- *Uses:*

✓ Arthritis Management.
✓ Nervous system modulation.
✓ Adaptogenic quality helps with stress-related pain.
✓ Inflammation reduction.

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Table 1 Summary Table of Analgesic Herbal Extracts

Sr. No.	Herbal Source	Major Active Constituents	Mechanism of Analgesic Action	Evidence Type
1.	Curcuma longa (Turmeric)	Curcumin	Inhibition of COX-2, NF-κB, cytokines; antioxidant activity.	Preclinical, RCTs, meta-analyses.
2.	Syzygium aromaticum (Clove)	Eugenol	Sodium channel blockade, COX inhibition, local anesthetic effect.	Preclinical, limited clinical.
3.	Zingiber officinale (Ginger)	Gingerols, Shogaols	COX/LOX inhibition, prostaglandin suppression	Preclinical, RCTs
4.	Boswellia serrata	Boswellic acids	5-LOX inhibition, anti- flammatory signaling	Preclinical, clinical trials
5.	Willow bark (Salix alba)	Salicin	Prostaglandin synthesis inhibition	Clinical trials, traditional use
6.	Withania somnifera	Withanolides	Anti-inflammatory, central modulation.	Preclinical, limited clinical.

III. PATHOPHYSIOLOGY OF PAIN AND TARGETS FOR HERBAL ANALGESICS

Pain may be classified as nociceptive, inflammatory, neuropathic, or involves the release of mediators such as prostaglandins, bradykinin, cytokines (TNF-α, IL-1β, IL-6), and reactive oxygen species, which sensitize nociceptors.

Many herbal analgesics act by:

- Inhibiting cyclooxygenase (COX) and lipoxygenase (LOX) pathways.
- Suppressing nuclear factor-κB (NF-κB) signaling.
- Modulating transient receptor potential (TRP) ion channels.
- Reducing oxidative stress.
- Interfering with central and peripheral neurotransmission.

Many herbal analgesics (e.g., curcumin, gingerols, eugenol, boswellic acids) exert analgesic effects by acting on inflammatory mediators, ion channels, oxidative pathways, and neural transmission at both peripheral and central levels.

A. Inhibition of Cyclooxygenase (COX) and Lipoxygenase (LOX) Pathways[33][34][35]

- Tissue injury activates phospholipase A_2, releasing arachidonic acid.
- COX enzymes (COX-1 & COX-2) convert arachidonic acid into prostaglandins (PGE_2).
- LOX converts it into leukotrienes.
- \- Prostaglandins and leukotrienes sensitize nociceptors, causing pain and inflammation.
- Herbal compounds inhibit COX and LOX, reducing inflammatory mediator synthesis.

- *Examples*

✓ Curcumin → COX-2 inhibition
✓ Gingerols → COX & LOX inhibition
✓ Eugenol → Prostaglandin suppression

B. Suppression of Nuclear Factor-κB (NF-κB) Signaling[36][37][38]

- NF-κB is a transcription factor activated during inflammation.
- It upregulates genes for:

- TNF-α
- IL-1β
- IL-6
- COX-2
- iNOS

✓ Herbal analgesics inhibit NF-κB activation, preventing cytokine production and chronic inflammation.

- *Result:*
  Reduced inflammatory pain and hyperalgesia.

C. Modulation of Transient Receptor Potential (TRP) Ion Channels[39][40][41]

- TRP channels are pain-sensing receptors on sensory neurons.
- Key channels:

- TRPV1 → heat, capsaicin, inflammatory pain.
- TRPA1 → chemical irritants.
- TRPM8 → cold sensation.

✓ Herbal compounds desensitize or block these channels, decreasing nociceptor activation.

- *Examples*

✓ Eugenol → TRPV1 modulation.
✓ Gingerols → TRPA1/TRPV1 inhibition.

D. Reduction of Oxidative Stress[42][43][44]

- Inflammation produces reactive oxygen species (ROS).
- ROS activate pain pathways and sensitize nociceptors.
- Herbal antioxidants:

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- Scavenge free radicals.
- Enhance antioxidant enzymes (SOD, catalase, glutathione).

✓ *Outcome:*
Reduced neuronal damage and inflammatory pain.

E. Interference with Central and Peripheral Neurotransmission[45][46][47]

➢ *Pain Signals are Transmitted Via Neurotransmitters:*

- Substance P
- Glutamate
- CGRP

➢ *Herbal Analgesics:*

- Reduce release of excitatory neurotransmitters.
- Enhance inhibitory pathways (GABAergic, serotonergic).
- Modulate opioid receptors (indirectly).

✓ *Effect:*
Reduced pain perception at spinal and supraspinal levels.

IV. MECHANISM

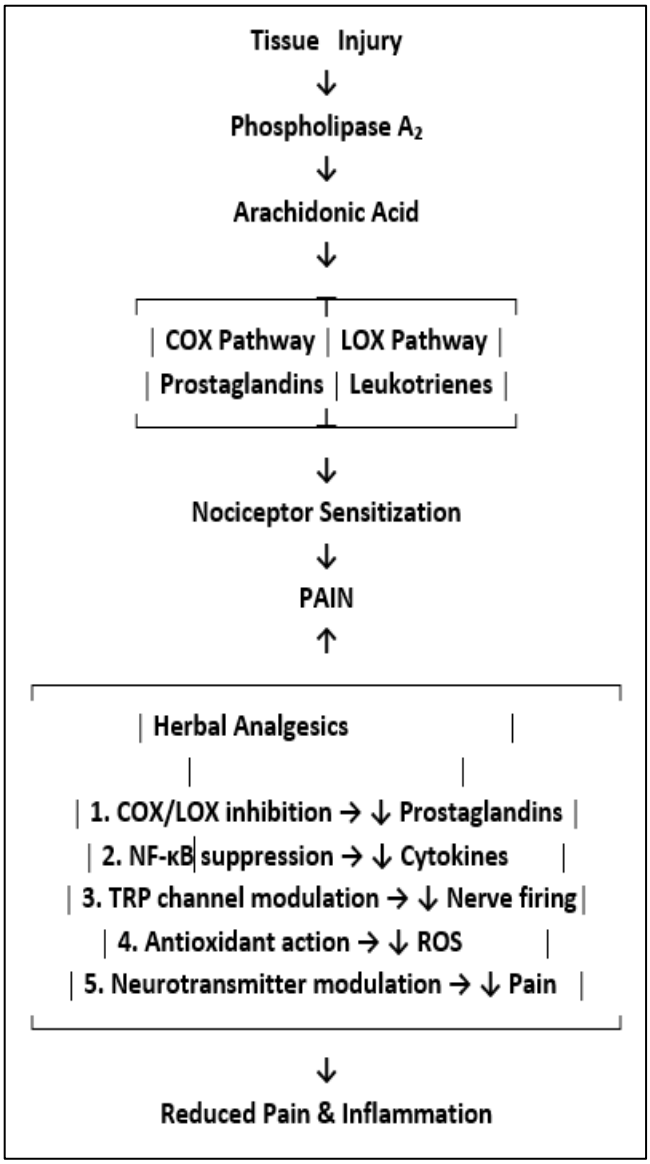

Fig 7 Mechanism

A. Step-Wise Mechanism of Analgesic Action of Herbal Extracts

➢ *Step 1: Tissue Injury or Inflammation Occurs*

- Injury, infection, or disease causes damage to tissues.
- This leads to the release of pain-producing substances such as:

✓ Prostaglandins
✓ Leukotrienes
✓ Bradykinin
✓ Histamine
✓ Cytokines (TNF-α, IL-1β, IL-6)

▪ These substances sensitize nociceptors (pain receptors).

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➢ *Step 2: Herbal Extract is Administered*

- Herbal extract enters the body (oral, buccal, topical, etc.).
- Active phytoconstituents (e.g., curcumin, eugenol, gingerols) are released.
- These compounds reach the site of inflammation and pain receptors.

➢ *Step 3: Inhibition of Inflammatory Enzymes*

- Herbal constituents inhibit enzymes responsible for pain mediator synthesis:

✓ Cyclooxygenase (COX-1 and COX-2)
✓ Lipoxygenase (LOX)

- This reduces formation of:

✓ Prostaglandins
✓ Leukotrienes

- *Example:*

✓ Curcumin, gingerols, boswellic acids

➢ *Step 4: Suppression of Pro-Inflammatory Cytokines*

- Herbal compounds block transcription factors such as NF-κB.
- This reduces release of:

✓ TNF-α
✓ IL-1β
✓ IL-6

- Inflammation decreases anHerbal extract enters the body (oral, buccal, topical, etc.).
- Active phytoconstituents (e.g., curcumin, eugenol, gingerols) are released.
- These compounds reach the site of inflammation and pain receptors.

➢ *Step 5: Reduction of Nociceptor Sensitization*

- Due to reduced inflammatory mediators:

✓ Pain receptors become less sensitive.
✓ Threshold for pain increases.

- Result:

✓ Reduced response to painful stimuli

➢ *Step 6: Modulation of Ion Channels*

- Certain herbal constituents act directly on nerve fibers:

✓ Block voltage-gated sodium channels
✓ Modulate TRP channels (TRPV1, TRPM8)

- Example:

✓ Eugenol (clove) → sodium channel blockade
✓ Capsaicin → TRPV1 desensitization

▪ Pain signal initiation is reduced.

➢ *Step 7: Antioxidant Action Reduces Oxidative Stress*

- Herbal extracts scavenge free radicals.
- Oxidative stress that worsens pain is reduced.

- *Example:*

✓ Curcumin, ginger, clove.

▪ Prevents chronic pain sensitization.

➢ *Step 8: Inhibition of Nitric Oxide and Substance P*

- Herbal constituents inhibit:
- Inducible nitric oxide synthase (iNOS)
- Release of substance P

✓ Reduced neurogenic inflammation and pain transmission.

➢ *Step 9: Central Nervous System Modulation*

- Herbal compounds act at spinal and brain levels:
- Enhance inhibitory neurotransmitters (GABA)
- Modulate serotonin and opioid receptors

✓ Brain perceives less pain.

➢ *Step 10: Overall Analgesic Effect*

- Combined actions lead to:

✓ Reduced inflammation
✓ Decreased pain signal transmission
✓ Lower pain perception

▪ Effective and sustained analgesic effect with fewer side effects.

V. CONCLUSION

Herbal extracts such as curcumin, clove, ginger, boswellia and willow bark possess significant analgesic potential supported by traditional use and modern scientific evidence. While preclinical data are robust, clinical evidence varies in strength and consistency. With improved standardization, formulation strategies, and rigorous clinical evalution , herbal analgesics may play an important role in future pain management strategies.

Herbal analgesics exert their effects through peripheral, central, biochemical, and molecular mechanisms. Their ability to modulate inflammation, nociceptor activity, oxidative stress, and neurotransmission makes them valuable

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alternatives or adjuncts to conventional analgesics. Understanding these mechanisms supports the rational development of herbal analgesic formulations, including mouth-dissolving tablets and other novel drug delivery systems.

Their multitarget action results in effective pain relief with improved safety compared to synthetic analgesics.

REFERENCES

[1]. Vane JR, Botting RM. Mechanism of action of NSAIDs. Am J Med. 1998;104(3A):2S-8S.
[2]. Ernst E. Herbal medicines in pain management. Pain. 2007;127(3):185-187.
[3]. Calixto JB. Efficacy, safety, quality control of herbal medicines. J Ethnopharmacol. 2000;72(1-2):1-16.
[4]. Theken KN, (2018); variability in Analgesic response to non-steroidal anti-inflammatory drugs; prostaglandins and other lipid mediators; 139; 63-70 .
[5]. Zafar A. (2010); A review on analgesic; from natural sources; International journal of pharmaceutical & Biological archive; 1(2); 95-100.
[6]. Ray R, Kattimani S and Sharma H.K. Opium Abuse abs Its management: Global Scenaro. Page no. 1-13.
[7]. Aggarwal BB, Harikumar KB. Potential therapeutic effects of curcumin: lessons learned from clinical trials. Int J Biochem Cell Biol. 2009; 41(1):40-59.
[8]. Basnet P, Skalko-Basnet N. Curcumin: An anti-inflammatory molecule from a curry spice. Molecules. 2011; 16(6):4567-4598.
[9]. Gupta SC, et al. Multitargeting by curcumin as revealed by molecular interaction studies. Nat Prod Rep. 2013; 30(4):544-559.
[10]. Daily JW, Yang M, Park S. Efficacy of turmeric extracts and curcumin for alleviating joint arthritis symptoms: A systematic review and meta-analysis. J Med Food. 2016; 19(8):717-729.
[11]. Hewlings SJ, Kalman DS. Curcumin: A review of its effects on human health. Foods. 2017; 6(10):92.
[12]. Sharma U, et al. Safety and efficacy of curcumin in inflammatory conditions. Phytother Res. 2017; 31(3):362-371.
[13]. Aggarwal BB, Harikumar KB. Potential therapeutic effects of curcumin: NF-κB inhibition. Int J Biochem Cell Biol. 2009; 41(1):40–59.
[14]. Nejad SM, Özgüneş H, Başaran N. Pharmacological and toxicological properties of eugenol. Avicenna J Phytomed. 2017; 7(4):337-349.
[15]. Pramod K, Ansari SH, Ali J. Eugenol: A natural compound with versatile pharmacological actions. Nat Prod Commun. 2010; 5(12):1999-2006.
[16]. Park SH, et al. Analgesic effects of eugenol through sodium channel blockade. Phytother Res. 2011; 25(3):345-349.
[17]. Daniel AN, et al. Anti-inflammatory and antinociceptive activities of eugenol essential oil. Pharm Biol. 2009;47(10):1007–1011.
[18]. Grzanna R, Lindmark L, Frondoza CG. Ginger—An herbal medicinal product with broad anti-inflammatory actions. J Med Food. 2005;8(2):125–132.
[19]. Chrubasik S, Roufogalis BD, Müller-Ladner U. Ginger and its analgesic potential. Phytother Res. 2005;19(9):760-766.
[20]. Rondanelli M, et al. Ginger and pain: A systematic review of clinical trials. Nutrients. 2020;12(2):356.
[21]. Altman RD, Marcussen KC. Effects of a ginger extract on knee pain in patients with osteoarthritis. Arthritis Rheum. 2001;44(11):2531-2538.
[22]. Black CD, O'Connor PJ. Acute effects of dietary ginger on muscle pain induced by eccentric exercise. J Pain. 2010;11(9):894-903.
[23]. Ammon HP. Boswellic acids and their role in chronic inflammatory diseases. Planta Med.2006; 72(12):1100-1116.
[24]. Sengupta K, et al. A double-blind, randomized, placebo-controlled study of Boswellia serrata extract in osteoarthritis of the knee. Int J Med Sci. 2008;5(3):133-141.
[25]. Kimmatkar N, et al. Efficacy and tolerability of Boswellia serrata extract in treatment of osteoarthritis.Phytomedicine. 2003;10(1):3-7.
[26]. Chrubasik S, et al. Effectiveness of willow bark extract for pain and inflammation. Phytother Res.2001;15(4):344-350.
[27]. Vlachojannis JE, et al. Willow bark extract for chronic low back pain: A randomized controlled trial. Am J Med.2000; 109(1):9-14.
[28]. Mishra, L. C., Singh, B. B., & Dagenais, S. (2000). Scientific basis for the therapeutic use of Withania somnifera. Alternative Medicine Review, 5(4), 334–346.
[29]. Kulkarni, S. K., & Dhir, A. (2008). Withania somnifera: An Indian ginseng. Progress in Neuro-Psychopharmacology & Biological Psychiatry, 32(5), 1093–1105.
[30]. Gupta, A., & Rana, A. C. (2007). Analgesic and anti-inflammatory activity of Withania somnifera. Pharmaceutical Biology, 45(6), 432–435.
[31]. Jayaprakasam, B., Zhang, Y., & Nair, M. G. (2003). Inhibition of cyclooxygenase-2 by withanolides from Withania somnifera. Journal of Natural Products, 66(8), 1068–1071.
[32]. Singh, G., Sharma, P. K., & Dudhe, R. (2010). Biological activities of Withania somnifera. International Journal of Pharmaceutical Sciences Review and Research, 3(2), 1–5.
[33]. Ammon HPT, Wahl MA. Pharmacology of Curcuma longa. Planta Med. 1991;57(1):1–7.
[34]. Srivastava KC, Mustafa T. Ginger (Zingiber officinale) inhibits COX and LOX pathways. Prostaglandins Leukot Essent Fatty Acids. 1992;46(2):117–124.
[35]. Pramod K, Ansari SH, Ali J. Eugenol: a natural compound with anti-inflammatory and analgesic activity. Pharmacogn Rev. 2010;4(7):1–6.

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[36]. Aggarwal BB, Harikumar KB. Potential therapeutic effects of curcumin: NF-κB inhibition. Int J Biochem Cell Biol. 2009;41(1):40–59.
[37]. Surh YJ. Anti-tumor promoting potential of selected spice ingredients through NF-κB inhibition. Food Chem Toxicol. 2002;40(8):1091–1097.
[38]. Ghosh S, Karin M. Missing pieces in the NF-κB puzzle. Cell. 2002;109(Suppl):S81–S96.
[39]. Yang BH, et al. Eugenol blocks TRPV1 channels and reduces pain sensation. Pain. 2003;103(1–2):43–52.
[40]. Dedov VN, et al. Gingerols modulate TRPA1 and TRPV1 ion channels. Br J Pharmacol. 2002;137(6):793–798.
[41]. Julius D. TRP channels and pain. Annu Rev Cell Dev Biol. 2013;29:355–384.
[42]. Menon VP, Sudheer AR. Antioxidant and anti-inflammatory properties of curcumin. Adv Exp Med Biol. 2007;595:105–125.
[43]. Reuter S, et al. Oxidative stress, inflammation, and cancer: curcumin as therapeutic agent. Free Radic Biol Med. 2010;49(11):1603–1616.
[44]. Ali BH, Blunden G. Pharmacological and toxicological properties of ginger. Food Chem Toxicol. 2003;41(11):1593–1600.
[45]. Millan MJ. The induction of pain: an integrative review. Prog Neurobiol. 1999;57(1):1–164.
[46]. Vriens J, Nilius B, Vennekens R. Herbal modulation of neuronal ion channels. Trends Pharmacol Sci. 2008;29(7):363–371.
[47]. Koster R, Anderson M, De Beer EJ. Acetic acid–induced writhing test for analgesics. Fed Proc. 1959;18:412–417.
[48]. Rainsford KD. Anti-inflammatory drugs in the treatment of pain. Pain Rev. 2000;7:213–245.
[49]. Rates SMK. Plants as source of drugs. Toxicon. 2001;39(5):603–613.
[50]. Abel SR. (1995); Tramadol: an alternative analgesic to traditional opioids and NSAIDs; Journal of Pharmaceutical Care in Pain & Symptom Control; 3(1); 5-29
[51]. Calixto JB, et al. Naturally occurring antinociceptive substances. Pharmacol Rev.2000; 52(3):319-370.
[52]. McGettigan P, Henry D. Cardiovascular risk with NSAIDs. BMJ. 2011;342:c7086.
[53]. Bannuru RR, et al. Comparative effectiveness of analgesics in osteoarthritis. Ann Intern Med. 2015;162(1):46-54.
[54]. Barnes J, Anderson LA, Phillipson JD. Herbal medicines: A guide for healthcare professionals. Pharm Press. 2013.
[55]. Calixto JB. Efficacy, safety, quality control of herbal medicines. J Ethnopharmacol. 2000;72(1-2):1-16.
[56]. Rates SMK. Plants as source of drugs. Toxicon. 2001;39(5):603-613.
[57]. WHO. WHO guidelines on safety monitoring of herbal medicines. Geneva: World Health Organization; 2004.
[58]. J Ethnopharmacol. 2000;72(1–2):1–16.
[59]. Ernst E. Herbal medicines in pain management.Pain. 2007;127(3):185–187.