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# A COMPREHENSIVE REVIEW OF NSAIDS AND TRADITIONAL MEDICINE IN THE MANAGEMENT OF PAIN AND INFLAMMATION

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**ABSTRACT**

Pain and inflammation represent significant healthcare challenges worldwide, affecting millions of individuals across diverse populations. This comprehensive review examines the comparative efficacy, safety profiles, and therapeutic potential of nonsteroidal anti-inflammatory drugs NSAIDs and traditional medicine systems in managing pain and inflammatory conditions. Through systematic analysis of clinical trials, pharmacological studies, and therapeutic outcomes, this review evaluates the mechanisms of action, clinical applications, and adverse effects of both approaches. NSAIDs, including non-selective COX inhibitors and selective COX 2 inhibitors, demonstrate well- established anti-inflammatory and analgesic properties through cyclooxygenase enzyme inhibition and prostaglandin suppression. However, their use is associated with significant gastrointestinal, cardiovascular, and renal complications, particularly with long-term administration. Traditional medicine systems, encompassing Ayurveda, Traditional Chinese Medicine, and herbal therapies, offer alternative approaches through natural compounds such as curcumin, boswellic acids, and gingerols. Clinical evidence suggests that certain traditional medicines, particularly turmeric and Boswellia serrata extracts, demonstrate comparable efficacy to NSAIDs in reducing pain and inflammation with superior safety profiles. Meta-analyses of randomized controlled trials indicate that curcumin exhibits anti-inflammatory effects similar to NSAIDs but with significantly fewer adverse events. The integration of traditional and conventional medicine approaches presents promising opportunities for personalized pain management strategies. Future research directions include the development of novel delivery systems, pharmacogenomic applications, and standardized traditional medicine formulations. This review concludes that while NSAIDs remain essential in acute inflammatory conditions, traditional medicine offers valuable complementary and alternative therapeutic options, particularly for chronic pain management where long-term safety considerations are paramount.

**KEYWORDS:** NSAIDs, Traditional Medicine, Pain Management, Inflammation, Pharmacology, Clinical Trials, Curcumin.

## 1. INTRODUCTION

Pain and inflammation constitute fundamental physiological responses that, while serving protective functions, often require therapeutic intervention when they become chronic or excessive.[1] The global burden of pain- related conditions affects over 1.5 billion people worldwide, with inflammatory disorders contributing significantly to morbidity, disability, and healthcare costs.[2] The management of pain and inflammation has traditionally relied on pharmacological interventions, particularly nonsteroidal anti-inflammatory drugs NSAIDs , which represent one of the most widely prescribed medication classes globally.[3]

### 1.1 NSAIDS: Definition and Classification

NSAIDs are a diverse group of pharmaceutical compounds that exert their therapeutic effects through inhibition of cyclooxygenase COX1) enzymes, leading to reduced prostaglandin synthesis.[4] These medications are primarily classified into two major categories based on their selectivity for COX enzyme isoforms.

**Non-selective COX Inhibitors:** These traditional NSAIDs inhibit both COX 1 and COX 2 enzymes and include widely used medications such as ibuprofen, naproxen, diclofenac, and aspirin.[1] These agents demonstrate broad anti-inflammatory, analgesic, and antipyretic properties but are associated with increased gastrointestinal toxicity due to COX 1 inhibition.[5]

**Selective COX 2 Inhibitors:** Developed to minimize gastrointestinal adverse effects, these agents preferentially inhibit COX 2 while sparing COX 1. Celecoxib represents the primary COX 2 selective inhibitor currently available in clinical practice.[6] While offering improved gastrointestinal safety, these agents have been associated with increased cardiovascular risks.[7]

### 1.2 Traditional Medicine Systems: Definition and Classification

Traditional medicine encompasses diverse healthcare systems that have evolved over centuries across different cultures and geographical regions.[8] The World Health Organization defines traditional medicine as "the sum total of the knowledge, skill, and practices based on the theories, beliefs, and experiences indigenous to different cultures, whether explicable or not, used in the maintenance of health".[9]

**Ayurveda:** Originating in India over 3,000 years ago, Ayurveda represents one of the world's oldest medical systems. This holistic approach emphasizes the balance of three fundamental energies (doshas): Vata, Pitta, and Kapha. Ayurvedic pain management utilizes herbs such as turmeric Curcuma longa), ashwagandha Withania somnifera), and boswellia Boswellia serrata).[2,5]

**Traditional Chinese Medicine TCM:** TCM is based on the concept of Qi (vital energy) and the balance between Yin and Yang forces. Pain in TCM is attributed to Qi stagnation and blood stasis. Key herbs include Corydalis rhizome Yan Hu Suo), ginger Zingiber officinale), and various herbal formulations.[10,11]

**Herbal Medicine:** This encompasses the use of plant-derived compounds for therapeutic purposes across various traditional systems. Notable anti-inflammatory herbs include turmeric, ginger, willow bark, and frankincense.[12,13]

**Homeopathy:** Developed in the late 18th century, homeopathy follows the principle of "like cures like" using highly diluted substances to stimulate the body's healing response.[14,15]

## 2. NSAIDs Pharmacology

### 2.1 Mechanism of Action

The primary mechanism of NSAID action involves the inhibition of cyclooxygenase enzymes, which catalyze the conversion of arachidonic acid to prostaglandins and thromboxanes.[1,4] The COX enzyme exists in two primary isoforms with distinct physiological roles:

**COX 1 Cyclooxygenase-1** This constitutively expressed enzyme performs essential "housekeeping" functions throughout the body. COX 1-derived prostaglandins maintain gastric mucosal integrity by stimulating mucus and bicarbonate secretion while reducing gastric acid production.[7] In the kidneys, COX 1 regulates afferent arteriolar vasodilation, maintaining glomerular perfusion and filtration. Additionally, COX 1 in platelets produces thromboxane A2, essential for platelet aggregation and hemostasis.[16]

**COX 2 Cyclooxygenase-2** This inducible enzyme is primarily expressed during inflammatory responses. COX 2 upregulation occurs in response to inflammatory stimuli, including cytokines, endotoxins, and growth factors.[7] The prostaglandins produced by COX 2 contribute to the cardinal signs of inflammation: vasodilation, increased vascular permeability, pain sensitization, and fever generation.[4]

The therapeutic effects of NSAIDs result from COX 2 inhibition, which reduces inflammatory prostaglandin production, while many adverse effects stem from COX 1 inhibition, disrupting normal physiological functions.[1,7]

### 2.2 Pharmacokinetics and Pharmacodynamics

NSAIDs exhibit diverse pharmacokinetic profiles that influence their clinical applications and dosing regimens.[17] Most NSAIDs are rapidly absorbed following oral administration, with peak plasma concentrations achieved within 1 4 hours.[18] These medications demonstrate high protein binding (typically % , primarily to albumin, which affects their distribution and potential for drug interactions[19]

Hepatic metabolism represents the primary elimination pathway for most NSAIDs, predominantly through cytochrome P450 enzymes, particularly CYP2C9.[20] The elimination half-lives vary significantly among different NSAIDs, ranging from 2 4 hours for ibuprofen to 12-17 hours for naproxen, influencing dosing frequency and duration of action.[21]

Pharmacodynamic considerations include the relationship between plasma concentrations and therapeutic effects, which may not always correlate directly due to tissue binding and local inflammatory conditions.[13] Age- related changes in pharmacokinetics, particularly in elderly patients, require dosing adjustments to minimize adverse effects while maintaining efficacy.[22]

## 3. Clinical Use of NSAIDs

### Therapeutic Indications

NSAIDs demonstrate broad clinical utility across numerous inflammatory and pain conditions:

**Musculoskeletal Disorders:** NSAIDs represent first-line therapy for osteoarthritis, rheumatoid arthritis, and other inflammatory joint conditions. Clinical trials demonstrate significant improvements in pain scores and functional outcomes.[23,24] In osteoarthritis, NSAIDs provide superior symptom relief compared to acetaminophen, with effect sizes ranging from 0.3 to 0.5 in pain reduction.[25]

**Post-operative Pain Management:** NSAIDs effectively manage acute post-surgical pain, often as part of multimodal analgesia regimens. They demonstrate opioid-sparing effects, reducing post- operative opioid consumption by 20 30%.[26] Preemptive NSAID administration can reduce post- operative pain intensity and duration.[27]

**Inflammatory Conditions:** Beyond arthritis, NSAIDs treat various inflammatory conditions including bursitis, tendinitis, and inflammatory back pain. They provide symptomatic relief while addressing underlying inflammatory processes.[28,30,32]

**Fever and General Pain:** NSAIDs effectively reduce fever and manage various pain conditions, including headaches, dental pain, and musculoskeletal injuries.[29,31]

## 4. Adverse Effects of NSAIDs

### 4.1 Gastrointestinal Effects

Gastrointestinal toxicity represents the most common and well-characterized adverse effect of NSAID therapy.[34,35] The incidence of serious upper gastrointestinal complications ranges from 14% annually among chronic NSAID users.[36,37]

**Mechanism:** COX 1 inhibition reduces protective prostaglandin E2 and prostacyclin production in the gastric mucosa, leading to decreased mucus and bicarbonate secretion, reduced epithelial proliferation, and impaired mucosal blood flow.[38] This results in increased susceptibility to acid- induced mucosal damage.

**Clinical Manifestations:** Gastrointestinal adverse effects range from mild dyspepsia to life- threatening complications including peptic ulceration, bleeding, and perforation.[34] Endoscopic studies reveal that 20 30% of regular NSAID users develop gastric or duodenal ulcers, with 15 35% of all peptic ulcer complications attributed to NSAID use.[37]

**Risk Factors:** Multiple factors increase gastrointestinal bleeding risk, including advanced age years), history of peptic ulcer disease, concomitant anticoagulant or corticosteroid use, and Helicobacter pylori infection.[34]
The risk of serious gastrointestinal events increases 4. -fold in patients with prior ulcer complications.[35]

### 4.2 Cardiovascular Risks

Cardiovascular toxicity, particularly associated with COX 2 selective inhibitors, has emerged as a major safety concern.[20] Meta-analyses demonstrate increased risks of myocardial infarction, stroke, and cardiovascular death with various NSAIDs.[3,6]

**Mechanism:** COX 2 inhibition reduces prostacyclin PGI2) production in vascular endothelium without affecting platelet thromboxane A2 synthesis, potentially promoting thrombosis.[16] Additionally, NSAIDs may increase blood pressure and worsen heart failure through prostaglandin- mediated effects on renal function and sodium retention.[4]

**Clinical Evidence:** Large-scale studies demonstrate that diclofenac carries the highest cardiovascular risk among commonly used NSAIDs, with hazard ratios of 1.4 1.9 for major cardiovascular events.[20] Even short-term NSAID use days) increases coronary heart disease risk by 46%.[3]

### 4.3 Renal Complications

NSAID-induced nephrotoxicity represents a significant clinical concern, particularly in vulnerable populations.[6]

[22]The incidence of NSAID-associated acute kidney injury ranges from 15%, but this increases substantially in high-risk patients.[6]

**Mechanism:** NSAIDs reduce renal prostaglandin synthesis, leading to afferent arteriolar vasoconstriction and decreased glomerular filtration rate.[4] In patients with compromised renal function, prostaglandins play a crucial compensatory role in maintaining renal perfusion, making them particularly susceptible to NSAID-induced injury.[22]

**Clinical Manifestations:** Acute complications include elevated serum creatinine, reduced urine output, and electrolyte imbalances.[22] Chronic NSAID use may contribute to progressive chronic kidney disease, particularly in combination with other nephrotoxic medications.[6]

**Risk Factors:** Advanced age, pre-existing renal impairment, heart failure, volume depletion, and concurrent use of ACE inhibitors or diuretics increase nephrotoxicity risk.[22] The "triple whammy" combination of NSAIDs, ACE inhibitors, and diuretics poses particularly high risk.[4]

### 4.4 Other Adverse Effects

**Hypersensitivity Reactions:** NSAIDs can trigger allergic reactions ranging from skin rashes to life- threatening anaphylaxis.[29] Aspirin-exacerbated respiratory disease affects 12% of the general population but up to 20% of individuals with asthma.[30]

**Hepatotoxicity:** While uncommon, NSAIDs can cause liver injury, particularly with chronic use or in susceptible individuals.-[31] Diclofenac carries the highest risk of hepatotoxicity among commonly used NSAIDs.[20]

**Central Nervous System Effects:** Some NSAIDs may cause headaches, dizziness, or confusion, particularly in elderly patients.[32]

## 5. Traditional Medicine Systems

### 5.1 Ayurvedic Medicine

Ayurveda, India's traditional medical system, offers a comprehensive approach to pain and inflammation management through personalized treatment strategies based on individual constitution (prakriti) and current imbalances (vikriti).[39,40]

**Theoretical Framework:** According to Ayurvedic principles, pain and inflammation result from imbalances in the three doshas, particularly Vata (governing movement and nervous function) and Pitta (governing metabolism and inflammation).[5] Treatment focuses on restoring dosha balance through herbs, dietary modifications, lifestyle changes, and purification procedures.[8]

**Key Herbal Medicines**

*Turm eric Curcum a longa):* Contains curcumin, a potent anti-inflammatory compound that inhibits multiple inflammatory pathways including NF κB, COX 2, and lipoxygenase.[27,41] Clinical trials demonstrate efficacy comparable to NSAIDs in osteoarthritis management with superior safety profiles.[27,42]

*Boswellia serrata Indian Frankincense):* Contains boswellic acids that inhibit 5-lipoxygenase and reduce inflammatory mediator production.[26,29] Randomized controlled trials show significant improvements in pain and function in

osteoarthritis and other inflammatory conditions.[26,32]

*Ashwagandha Withania som nifera):* Demonstrates anti-inflammatory, analgesic, and adaptogenic properties through modulation of stress hormones and inflammatory cytokines.[39,40] Clinical studies show efficacy in reducing joint pain and improving mobility.[39,43]

*Ginger Zingiber officinale):* Contains gingerols and shogaols with anti-inflammatory and analgesic properties through COX and lipoxygenase inhibition.[44,45] Clinical trials demonstrate effectiveness in osteoarthritis and muscle pain.[44,46]

### 5.2 Traditional Chinese Medicine

TCM approaches pain management through the lens of Qi (vital energy) flow and blood circulation.[10,11] Pain is conceptualized as resulting from Qi stagnation, blood stasis, or pathogenic factors disrupting normal physiological processes.

**Diagnostic Framework:** TCM diagnosis involves comprehensive assessment including pulse diagnosis, tongue examination, and symptom pattern recognition to identify underlying patterns of disharmony.[47,48] Treatment is individualized based on the specific pattern identified.

#### Therapeutic Modalities

*Herbal Medicine:* TCM utilizes complex herbal formulations containing multiple ingredients that work synergistically. Key pain-relieving herbs include Corydalis rhizome Yan Hu Suo), which contains alkaloids with natural analgesic properties comparable to mild opioids.[10,47]

*Acupuncture:* This technique involves inserting thin needles at specific anatomical points to regulate Qi flow and reduce pain.[49,50] Systematic reviews demonstrate efficacy in various chronic pain conditions with minimal adverse effects.[49]

*Other Modalities:* TCM incorporates massage Tui Na, cupping therapy, moxibustion, and Qigong exercises as complementary approaches to pain management.[11,48]

### 5.3 Herbal Medicine

Herbal medicine spans multiple traditional systems and utilizes plant-derived compounds for therapeutic purposes.[12,13] Scientific research has validated the anti-inflammatory and analgesic properties of numerous herbs through identification of active compounds and mechanisms of action.

#### Evidence-Based Herbs

*Willow Bark Salix species):* Contains salicin, a precursor to salicylic acid, providing natural anti- inflammatory and analgesic effects similar to aspirin but with reduced gastrointestinal toxicity.[13]

*D evil's Claw Harpagophytum procum bens):* Contains harpagoside and other iridoid glycosides that demonstrate anti-inflammatory properties and efficacy in osteoarthritis management.[13]

*White Birch Betula alba):* Bark extracts contain betulinic acid and other compounds with anti- inflammatory and analgesic properties.[13]

### 5.4 Homeopathy

Homeopathy represents a controversial yet widely used complementary medicine system based on the principle of "like cures like" and the use of highly diluted substances.[14,51]

**Clinical Evidence:** Systematic reviews of homeopathic treatments for pain conditions show mixed results.[14,15] While some individual trials report positive outcomes, meta-analyses generally conclude that evidence does not support effectiveness beyond placebo effects.[14] However, proponents argue that individualized homeopathic treatment may provide benefits not captured in conventional trial designs.[51,52]

## 6. Comparative Clinical Trials

This section reviews clinical trials directly comparing NSAIDs with traditional medicine approaches, providing evidence for their relative efficacy and safety in pain and inflammation management.

### 6.1 Curcumin vs NSAIDs Studies

**Trial 1 Curcumin BCM vs Diclofenac in Rheumatoid Arthritis**

A randomized controlled trial compared curcumin BCM extract 500mg twice daily) with diclofenac sodium 50mg twice daily) in 45 patients with rheumatoid arthritis.[30] The curcumin group showed the highest percentage improvement in Disease Activity Score DAS) with significantly better outcomes than the diclofenac group. Importantly, curcumin treatment was associated with no adverse events, while the diclofenac group experienced typical NSAID-related side effects.[30]

**Trial 2 Meta-analysis of Turmeric vs NSAIDs in Knee Osteoarthritis**

A systematic review and meta-analysis of 10 randomized controlled trials involving turmeric therapy for knee osteoarthritis found that turmeric extracts provided pain relief and functional improvement comparable to NSAIDs.[27] Three studies directly comparing turmeric to NSAIDs showed no significant differences in outcome scores ($p>0.05$), but turmeric demonstrated superior safety with significantly fewer adverse events.[27]

**Trial 3 High-dose vs Low-dose Curcumin Study**

A dose-response study by Henrotin et al. evaluated both high-dose and low-dose curcumin therapy groups compared to placebo.[41] Both dosages showed similar effect sizes for pain and functional improvement, but the high-dose group had significantly increased adverse events compared to placebo 37% vs 13%, $p=0.012$ , while the low-dose group showed no significant difference in adverse events.[41]

### 6.2 Boswellia serrata Clinical Trials

**Trial 4 Boswellin® Super Double-blind Study**

A randomized, double-blind, placebo-controlled trial evaluated 105 patients with knee osteoarthritis receiving either 150mg or 300mg of standardized Boswellia serrata extract Boswellin® Super) twice daily for days.[26] Results showed improvements in pain scores as early as 5 days after treatment initiation. By days, Visual Analog Scale pain scores reduced by 45.3% and .9% in the 150mg and 300mg groups respectively, compared to placebo. WOMAC total scores improved by .5% and .6% respectively, with significant reductions in inflammatory biomarkers TNF α, hs-CRP, and IL.[6]

**Trial 5 Boswellia and Curcumin Combination Study**

A three-arm parallel study compared full-spectrum Boswellia serrata extract alone F BSE) versus combined Boswellia-curcumin formulation C BSE) versus placebo in 105 participants with mild-to-moderate spondylitis.[29] Both active groups showed significant reductions in Bath Ankylosing Spondylitis Disease Activity Index BASDAI) scores and Neck Disability Index by day 14, continuing through day 28. The combination C BSE demonstrated superior effects compared to F BSE alone, particularly for neck/hip pain 59.53% reduction, $p<0.001$) and stiffness 50. % reduction, $p<0.001$.[29]

### 6.3 Ginger Clinical Studies

**Trial 6 Ginger vs NSAIDs in Dysmenorrhea**

A double-blind comparative clinical trial evaluated ginger 250mg capsules) against mefenamic acid 250mg and ibuprofen 400mg for primary dysmenorrhea pain relief [44] . Results demonstrated that ginger was as effective as both NSAIDs in reducing menstrual pain intensity and duration, with no significant differences between groups. However, the ginger group reported fewer gastrointestinal side effects compared to NSAID groups.[44]

**Trial 7 Ginger Extract in Knee Osteoarthritis**

A randomized controlled trial compared ginger extract with placebo in patients with knee osteoarthritis.[44] The ginger group showed consistently greater response to treatment compared to controls, with significant improvements in pain reduction and mobility. A separate study comparing ginger with placebo in hip and knee osteoarthritis showed no significant differences, highlighting the variability in ginger preparation and standardization challenges.[44]

### 6.4 Ashwagandha Clinical Evidence

**Trial 8 Ashwagandha Root and Leaf Extract Study**

A 12-week randomized, double-blind, placebo-controlled trial evaluated standardized Withania somnifera extract in 60 patients with knee joint pain.[39] Participants received either 250mg or 125mg of aqueous extract twice daily. The 250mg group showed significant reductions in modified WOMAC scores, Knee Swelling Index, and Visual Analog Scale scores for pain, stiffness, and disability compared to both placebo and the 125mg group ($p<0.001$ . The therapeutic response appeared dose-dependent with minimal gastrointestinal disturbances.[39]

## 7. Challenges and Limitations

### 7.1 Regulatory Hurdles

The integration of traditional medicine into mainstream healthcare faces significant regulatory challenges worldwide.[9,55] Unlike conventional pharmaceuticals, traditional medicines often contain multiple active compounds with complex interactions, making standardization and quality control challenging.[56,57]

**Standardization Issues:** Traditional herbal preparations vary significantly in active compound concentrations due to factors including plant genetics, growing conditions, harvesting methods, and processing techniques.[9] This variability makes it difficult to establish consistent dosing guidelines and compare clinical trial results.[55]

**Regulatory Framework Variations:** Different countries have vastly different regulatory approaches to traditional medicine.[58,59] While some nations like Germany and China have well-established regulatory frameworks for herbal medicines, others lack comprehensive guidelines, creating barriers to international research collaboration and product development.[55,56]

**Evidence Requirements:** Regulatory agencies increasingly require the same level of evidence for traditional medicines as for conventional drugs, including randomized controlled trials, safety data, and quality assessments.[9] However, traditional medicine's holistic, individualized approach may not fit conventional clinical trial designs.[55]

### 7.2 Lack of Standardization

**Active Compound Variability:** Even within the same species, plants can contain dramatically different concentrations of active compounds. For example, curcumin content in turmeric can vary from 2 8% depending on the source and processing methods.[27] This variability affects both therapeutic efficacy and reproducibility of clinical research.[41]

**Extraction Methods:** Different extraction techniques (aqueous, alcoholic, supercritical CO2) yield preparations with varying bioactive profiles.[39] The choice of extraction method significantly impacts the final product's therapeutic properties and bioavailability.

**Bioavailability Challenges:** Many traditional medicine compounds, particularly curcumin and boswellic acids, have poor oral bioavailability, limiting their therapeutic effectiveness.[27,29] Various formulation strategies, including nanoparticle delivery systems and bioenhancers, are being investigated to address these limitations.

### 7.3 Limited High-Quality Clinical Trials

**Sample Size Limitations:** Many traditional medicine studies involve small sample sizes, limiting statistical power and generalizability of results.[27,39] Meta-analyses often struggle with heterogeneity between studies due to different preparations, dosages, and outcome measures.[27,42]

**Methodological Issues:** Traditional medicine research faces unique methodological challenges, including difficulty in creating appropriate placebos, blinding participants and investigators, and standardizing individualized treatments.[14,51] Many studies lack adequate randomization, allocation concealment, or intention-to-treat analysis.

**Publication Bias:** Traditional medicine research may be subject to publication bias, with positive results more likely to be published than negative findings.[14] Additionally, many studies are published in non-English journals or non-indexed publications, ~~li~~miting their inclusion in systematic reviews.

### 7.4 Drug Interactions and Safety Concerns

**Herb-Drug Interactions:** Traditional medicines can interact with conventional medications through various mechanisms, including inhibition or induction of cytochrome P450 enzymes.[56] For example, St. John's wort induces CYP3A4, potentially reducing the effectiveness of numerous medications.

**NSAID Herb Interactions:** Combining NSAIDs with certain herbs may increase bleeding risk or potentiate adverse effects. Ginkgo biloba, garlic, and ginger may enhance anticoagulant effects when used concurrently with NSAIDs.

**Quality Control Issues:** Traditional medicine products may be contaminated with heavy metals, pesticides, or undeclared pharmaceutical compounds.[56,57] Lack of standardized quality control measures poses safety risks, particularly in unregulated markets.[9]

**Dosage Uncertainties:** Optimal dosing for many traditional medicines remains unclear due to limited pharmacokinetic

studies and dose-response research.[27,41] This uncertainty complicates clinical decision- making and may lead to suboptimal therapeutic outcomes or increased adverse effects.[39]

### 7.5 Variable Bioavailability

**Compound Stability:** Many bioactive compounds in traditional medicines are unstable and may degrade during processing, storage, or digestion This instability affects the actual dose delivered to target tissues and complicates dose standardization.

**Individual Variations:** Genetic polymorphisms in drug-metabolizing enzymes can significantly affect individual responses to traditional medicines For example, variations in cytochrome P450 enzymes may influence curcumin metabolism and therapeutic effectiveness.

**Formulation Challenges:** Traditional preparation methods may not optimize bioavailability of active compounds Modern pharmaceutical techniques, including nanotechnology and novel delivery systems, show promise in enhancing bioavailability but require extensive development and testing.

## 8. Future Prospects

### 8.1 Integration of Traditional and Modern Medicine

The future of pain and inflammation management lies in the thoughtful integration of traditional and conventional medicine approaches, leveraging the strengths of both systems while minimizing their respective limitations.[9,55]

**Complementary Treatment Strategies:** Rather than viewing traditional and conventional medicine as competing approaches, future healthcare models may emphasize their complementary roles. NSAIDs could be reserved for acute inflammatory conditions requiring rapid symptom control, while traditional medicines might be preferred for chronic pain management where long-term safety considerations are paramount.[27,39]

**Evidence-Based Integration:** The WHO Traditional Medicine Strategy 2025 emphasizes evidence-based integration of traditional, complementary, and integrative medicine into health systems.[9,55] This framework promotes rigorous scientific evaluation while respecting cultural traditions and indigenous knowledge.

## 9. CONCLUSION

This comprehensive review demonstrates that both NSAIDs and traditional medicine approaches offer valuable but distinct contributions to pain and inflammation management. NSAIDs remain essential therapeutic tools for acute inflammatory conditions, providing rapid and predictable symptom relief through well-characterized mechanisms of cyclooxygenase inhibition. However, their utility is limited by significant adverse effects, particularly gastrointestinal, cardiovascular, and renal complications associated with chronic use.

Traditional medicine systems, particularly Ayurveda and Traditional Chinese Medicine, offer compelling alternatives through natural compounds such as curcumin, boswellic acids, and gingerols. Clinical evidence increasingly supports the efficacy of these approaches, with several high-quality randomized controlled trials demonstrating comparable pain relief and functional improvement to NSAIDs, often with superior safety profiles. Meta-analyses consistently show that traditional medicines like turmeric extract provide anti-inflammatory effects similar to NSAIDs but with significantly fewer adverse events.

The integration of traditional and conventional medicine approaches represents the most promising path forward for optimal pain management. This integrated model would leverage NSAIDs for conditions requiring rapid symptom control while emphasizing traditional medicines for chronic pain management where long-term safety considerations are paramount. Personalized medicine approaches, incorporating pharmacogenomic data and individual patient characteristics, could guide treatment selection to optimize both efficacy and safety outcomes.

Future research priorities must address current limitations including standardization challenges, limited high- quality clinical trials, and variable bioavailability of traditional medicine compounds. Novel delivery systems utilizing nanotechnology show particular promise for enhancing therapeutic effectiveness while minimizing adverse effects. The WHO Traditional Medicine Strategy 2025 2034 provides a framework for evidence-based integration that respects both scientific rigor and cultural traditions.

Healthcare providers should recognize that optimal pain management often requires individualized approaches considering patient preferences, clinical characteristics, and risk factors. While awaiting further research developments, current evidence supports the judicious use of both NSAIDs and traditional medicines as part of comprehensive pain management strategies. The complementary rather than competitive relationship between these approaches offers the greatest potential for improving patient outcomes while minimizing treatment-related risks.

The evidence presented in this review underscores the need for continued high-quality research to fully realize the therapeutic potential of both conventional and traditional medicine approaches. As our understanding of individual genetic variations and molecular mechanisms continues to expand, the vision of truly personalized pain medicine incorporating the best of both traditional wisdom and modern science becomes increasingly achievable.

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