



## EXPLORING THE POTENTIAL OF SOME MEDICINAL PLANTS FOR THE TREATMENT OF GOUTY ARTHRITIS

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### Abstract

This in-depth review delves into the complex and historical condition of gout, a form of arthritis recognized since ancient Egypt, with a particular emphasis on its clinical feature, uricemia. The review covers the underlying causes of uricemia, tracing its historical background, and addresses the ongoing challenges in its treatment. It thoroughly explains the physiological impact of gout, highlighting how uric acid crystals form and affect various tissues and organs. The study dissects the triad of factors that contribute to gout, namely purine metabolism, nucleotide turnover, and renal function. Epidemiological data provide insights into age and gender disparities, as well as the prevalence rates of gout. The review also lists the various risk factors for developing gout, including genetic predisposition, hyperuricemia, lifestyle factors, medications, diet, and other specific conditions.

The research methodology involves a comprehensive literature survey using databases such as PubMed, Scopus, Science Direct, and Google Scholar, with an emphasis on clinical studies and Ayurvedic medicinal plants. The review evaluates conventional gout treatments like xanthine oxidase inhibitors, anti-inflammatory drugs, and urate-lowering agents, along with their limitations and potential side effects. Additionally, it explores the therapeutic potential of medicinal plants such as *Tribulus Terrestris*, *Cichorium intybus* Linn, *Colchicum Autumnale*, *Curcuma longa* Linn, *Zingiber officinalis*, *Boswellia serrata*, *Withania somnifera*, *Nigella sativa*, *Glycyrrhiza glabra*, and *Trachyspermum Ammi*, assessing their medicinal properties, chemical makeup, and anti-inflammatory effects.

In conclusion, the study underscores the potential benefits of integrating medicinal plants into gout management but calls for a cautious and evidence-based approach. The enduring legacy of traditional remedies provides valuable insights; however, the study stresses the importance of rigorous preclinical and clinical research to fully understand the efficacy and safety of plant-based treatments, ensuring their successful incorporation into modern healthcare.

**Key words:** Gout, Plants based therapy, uric acid, pain, Anti inflammation

### 1. Introduction

Gout, a well-known form of arthritis, has long been recognized among rheumatic diseases, with its history stretching back thousands of years [1]. This introductory section explores the complex nature of gout, offering insights into its primary clinical manifestation, uricemia, and tracing its prevalence back to ancient Egypt, around 4,000 years ago. The underlying mechanisms of uricemia,

uncovering potential triggers. Whether due to abnormal renal urate transport or metabolic disorders that lead to excessive uric acid production, a thorough understanding of these causative factors provides the foundation for further exploration [2].

### **1.1. Historical Perspectives and Treatment Challenges**

This section examines the enduring difficulties in treating gout since its earliest documentation. By reviewing historical accounts of gout, particularly those from ancient Egypt [3], we gain valuable insights into the distinctive nature of this condition and the persistent challenges in developing effective treatments from antiquity to the present.

### **1.2. Metabolic Diseases and Uric Acid Crystals**

Focusing on the physiological effects of gout, this section describes how the buildup of uric acid crystals in tissues and organs results from various metabolic diseases. The role of these crystals in causing renal and joint tophi and joint discomfort is explored, providing a comprehensive picture of the disease's impact on the body [4, 5, 6].

### **1.3. Pathophysiology of Gout: Crystal Formation and Inflammation**

This section unpacks the detailed processes involved in crystal formation, which occur when uric acid levels exceed 6.5 mg% in the blood. It explains how these crystals accumulate in tissues, joints, and urine ducts, forming urates [7] and triggering inflammatory responses that damage joints. Specific joints commonly affected, such as fingers, wrists, ankles, knees, and metatarsophalangeal joints, are also discussed [8].

### **1.4. Probable Causes of Gout: A Triad of Factors**

This section explores the three main factors contributing to gout: purine synthesis, purine nucleotide resynthesis, and kidney function's crucial role in uric acid excretion [9]. Additionally, it outlines the American College of Rheumatology's clinical criteria, which serve as a guideline for diagnosing gout [10].

## **2. Epidemiology of Gout**

### **2.1. Age-Related Trends in Gout**

This section investigates the relationship between age and the incidence of gout, showing how the prevalence of this condition increases with age and varies across different stages of life.

### **2.2. Gender Disparities: Gout's Preference for Men**

This section explores the gender-specific aspects of gout, highlighting how men are disproportionately affected by this inflammatory disease. It discusses how gout has become the leading cause of joint inflammation in men, creating a unique pattern of morbidity.

### **2.3. Numeric Ratios: Understanding Gout's Gender Disparity**

The section quantifies the gender disparity, presenting a striking ratio of 6:1 in the occurrence of gout between men and women, emphasizing the significant difference in prevalence.

## **3. Factors Influencing Gout Susceptibility: A Comprehensive Analysis**

This section offers an in-depth examination of the various factors that increase an individual's risk of developing gout. These include genetic predisposition, hyperuricemia, lifestyle choices, medications, dietary habits, and special circumstances.

### **3.1. Genetic Conditioning: Unveiling Familial Predispositions**

This section explores the genetic aspects of gout, focusing on how hereditary factors contribute to increased susceptibility in certain populations.

### 3.2. Hyperuricemia: The Biochemical Imbalance

This section discusses hyperuricemia, a key factor in gout development, explaining how elevated uric acid levels lay the groundwork for joint inflammation.

### 3.3. Lifestyle Choices and Medications: Dual Impact

The influence of lifestyle choices and certain medications on gout risk is examined here, with attention given to drugs like cyclosporine, diuretics, and low-dose aspirin that increase susceptibility.

### 3.4. Dietary Habits: Dietary Contributors to Gout

This section explores dietary factors, particularly the consumption of meat, seafood, and alcohol (especially beer), that contribute to the risk of gout.

### 3.5. Special Circumstances: Surgery, Transplants, and Hormonal Changes

This section discusses how special conditions, including surgeries, organ transplants, and hormonal changes, particularly in men and postmenopausal women, elevate the risk of gout.

### 3.6. Aging and Dehydration: Aging's Role in Gout

The impact of aging and dehydration on gout susceptibility is analyzed here, with a focus on how these factors increase the likelihood of gout development.

## 4. Impact of Medicinal Plants on Gout Treatment

The rising global preference for plant-based medications is driven by their perceived safety compared to synthetic drugs. Rooted in traditional remedies, herbal medicines are often considered non-toxic and safe, offering a natural alternative to conventional treatments. This section examines the use of specific medicinal plants in gout management, highlighting their potential therapeutic benefits.



**Figure 1:** Medicinal herbs used for the treatment of gouty arthritis.

S.no	Plant sources	Family	Parts used	Dosage
01	Tribulus terrestris Linn	Zygophyllaceae	Fruits	5-7gms
02	Cichorium intybus	Compositae/ Asteraceae	Dried leaves, roots and seeds	3-5gms (seeds)
03	Colchicum Autumnale	Liliaceae	Roots/tubers	25mg (approx)
04	Curcuma longa	Zingiberaceae	Rhizomes	1-3gms
05	Zingiber officinalis	Zingiberaceae	Rhizomes	1-3gms
06	Trachyspermum Ammi	Apiaceae	Fruit(seeds)	3-6gms(approx)
07	Withania somnifera	Solanaceae	Roots	3-6gms
08	Nigella sativa	Ranunculaceae	Seeds	1-3gms
09	Glycyrrhiza glabra	Fabaceae	Roots	2-5gms

**Table 1: Representing the Plants source for the treatment of Gouty arthritis, family of these plants, parts use for the medicinal purpose for the treatment, quantity of the part in each dosage.**

S.no	Plant sources	Phyto-chemical constituents	Temperament
01	Tribulus Terrestris Linn	Saponins, Protodioscin	Warm + dry
02	Cichorium Intybus	Choline, Caffeic Acid Chichoric acid	Cold + moist
03	Colchicum Autumnale	Colchicine	Warm + dry
04	Curcuma longa	Curcumin, Curcuminoids, Tumerone, Cymene	Warm + dry
05	Zingiber officinalis	6-gingerol, 6-Shogol, 6-paradol	Warm + dry
06	Trachysmermum ammi	Thymol, p-cymene, $\gamma$ -terpinene.	Warm + dry
07	Withania somnifera	Withanolides, Withaferin A, Alkaloids	Warm + dry
08	Nigella sativa	Thymoquinone, Nigellone, Alpha-Hederin, Alkaloids	Warm + dry
09	Glycyrrhiza glabra	Glycyrrhizin, Flavonoids, Saponins, Isoflavonoids	Warm + moist

**Table 2: representing the plant use for the treatment of gouty arthritis, main phyto-constituents of the plant and temperament of this plant according to Unani philosophy.**

#### 4.1. Tribulus Terrestris

**Family:** Zygophyllaceae

**Botanical name:** *Tribulus terrestris*

**Other names:** Khasak, Khar Khask, Caltrop, Gokhru

**Parts Used:** Fruit

**Temperament:** Warm I order and dry I order

**Specific Chemical Constituents:** Saponins, Protodioscin

The genus *Tribulus*, part of the Zygophyllaceae family, comprises around 20 species distributed worldwide. Among these, three species, including *Tribulus terrestris* (TT), are particularly prevalent in India. Both Ayurvedic healers and contemporary botanists regard TT as a highly effective medicinal plant. This annual plant, utilized in various formulations and dietary supplements, thrives in a range of climates, including Mediterranean, subtropical, and desert regions across countries such as India, China, the southern United States, Mexico, Spain, and Bulgaria [14]. Medicinally, all parts of the TT plant—fruits, flowers, roots, seeds, and leaves—are used for their therapeutic benefits. The ethanolic extract of TT is notably effective in reducing the expression of

cyclooxygenase-2 (COX-2) and inducible nitric oxide synthase (iNOS) in lipopolysaccharide-stimulated RAW264 cells. Additionally, this extract suppresses pro-inflammatory cytokines such as tumor necrosis factor-alpha (TNF- $\alpha$ ) and interleukin (IL)-4 in macrophage cell lines, suggesting its potential in treating various inflammatory conditions.

Furthermore, the methanolic extract of TT shows a dose-dependent reduction in paw volume in rodents, highlighting its anti-inflammatory properties, particularly in cases of carrageenan-induced inflammation in rats. The study references Anderson's 1976 findings on the relationship between hind paw edema, lysosomal enzymes, and tissue damage in rheumatic conditions, further discussing the significant inflammatory response driven by Freund's complete adjuvant, including leukocyte infiltration, mast cell activation, cytokine release, and the involvement of free radicals [17].

### **Pharmacological Actions:**

The ethanolic extract of *Tribulus terrestris* (TT) effectively reduced the expression of cyclooxygenase-2 (COX-2) and inducible nitric oxide synthase (iNOS) in RAW264 cells that were stimulated with lipopolysaccharides. It also inhibited the production of pro-inflammatory cytokines such as tumor necrosis factor-alpha (TNF- $\alpha$ ) and interleukin-4 (IL-4) in the macrophage cell line. This suggests that the ethanolic extract of TT plays a significant role in suppressing mediators associated with inflammation and cytokine expression, thereby providing therapeutic benefits for various inflammatory conditions. Additionally, the methanolic extract of TT demonstrated a dose-dependent reduction in paw volume in rats experiencing carrageenan-induced inflammation.

In 1976, Anderson observed that the increase in hind paw edema following adjuvant-induced inflammation in rats corresponded with an elevation in extracellular lysosomal enzymes. These enzymes are responsible for breaking down structural macromolecules in connective tissue and cartilage proteoglycans. As a result, they can lead to the degradation of extracellular structures, contributing to tissue damage in rheumatic diseases. The severe inflammatory response triggered by Freund's complete adjuvant involves leukocyte infiltration, mast cell activation, cytokine release, and the generation of free radicals.

## **4.2. Cichorium intybus Linn**

**Family:** Compositae/Asteraceae

**Botanical Name:** Cichorium intybus Linn

**Other Names:** Kasni, Handbu, Chicory

**Parts Used:** Dried leaves, roots, seeds

**Temperament:** Cold I order and moist I order

**Specific Chemical Constituents:** Choline, Caffeic Acid, Chichoric Acid

Cichorium intybus Linn, commonly known as chicory, encompasses both dried roots and above-ground components. Belonging to the family Compositae/Asteraceae, this biennial or perennial herb is native to Europe and Asia. Recognizable by its striking blue, lavender, or occasional white blooms and spindle-shaped taproots, chicory holds historical significance as one of the earliest documented plants.

### **Medicinal Properties:**

Chicory serves various medicinal purposes, acting as a tonic, coolant, and remedy for conditions such as thirst, migraines, ophthalmia, throat inflammation, fever, vomiting, diarrhea, edema, and joint pain. The leaves find topical application for alleviating similar ailments. Additionally, chicory seeds exhibit tonic properties for the brain and efficacy in treating biliousness, lumbago, and asthma.

### **Alcoholic Extracts and Anti-Inflammatory Impact:**

The alcoholic extracts derived from C. intybus roots manifest notable anti-inflammatory effects, particularly in treating pyorrhea and gingival inflammation. A deeper investigation into the

molecular level reveals the hindrance of tumor necrosis factor-alpha (TNF- $\alpha$ ) mediated cyclooxygenase activation by chicory root ethyl acetate extract. This inhibition extends to the human colon carcinoma cells, showcasing a dose-dependent reduction in prostaglandin E2 synthesis [18].

#### **Anti-Inflammatory Activity in Animal Models:**

Further exploration uncovers the significant dose-dependent anti-inflammatory activity of chicory roots in a carrageenan-induced paw edema model. This therapeutic effect is marked by the reduction of serum TNF- $\alpha$ , interleukin (IL)-6, and IL-1 levels, concurrently leading to an increase in antioxidant activity within the paw tissue. These findings suggest that the anti-inflammatory and antioxidant actions of chicory roots may be mediated through the modulation of cytokines [19].

#### **Pharmacological Actions:**

The ethanolic extract of *Tribulus terrestris* (TT) significantly downregulated the expression of cyclooxygenase-2 (COX-2) and inducible nitric oxide synthase (iNOS) in RAW264 cells that were activated by lipopolysaccharides. Additionally, it suppressed the production of key pro-inflammatory cytokines, including tumor necrosis factor-alpha (TNF- $\alpha$ ) and interleukin-4 (IL-4) within the macrophage cell line. These findings indicate that the ethanolic extract of TT is effective in reducing inflammation by inhibiting the mediators and cytokines involved, making it potentially beneficial for treating various inflammatory conditions. Moreover, the methanolic extract of TT exhibited a dose-dependent reduction in paw swelling in rats subjected to carrageenan-induced inflammation, further supporting its anti-inflammatory properties [18].

### **4.3. Colchicum Autumnale**

**Family:** Liliaceae

**Botanical Name:** Colchicum autumnale

**Other Names:** Suranjan Shireen, Colchium

**Parts Used:** Roots/tubers

**Temperament:** Warm II order and dry II order

**Specific Chemical Constituents:** Colchicine

Colchicum Autumnale, commonly known as Suranjan in the Indian market, belongs to the Liliaceae family and boasts a rich history. The plant's nomenclature is intricately linked to the land of Colchis, situated at the eastern tip of the Black Sea. First-century AD depictions by Dioscorides, a pioneering figure in Unani medicine, detailed the plant under the name "Colchicon," emphasizing its historical significance. The term 'autumn crocus' reflects its characteristic blooming season in fall, with leaves and capsules emerging in the subsequent April [20].

#### **Botanical Attributes and Therapeutic Components:**

As a non-domesticated medicinal plant, Colchicum Autumnale from the Liliaceae family is endowed with valuable alkaloids. These alkaloids, including colchicine and colchicoside, are synthesized and extracted from the seeds, renowned for their applications in medications targeting anti-gout and muscle relaxant properties [21].

#### **Plant Parts with Medicinal Value:**

The root or tubers, along with freshly gathered flowers and dried mature seeds, serve as the therapeutic components of Colchicum Autumnale. The careful harvesting process, conducted in early summer and followed by meticulous drying, ensures the efficacy of these plant parts [21].

#### **Efficacy in Joint Conditions:**

Suranjan Shirin, derived from Colchicum Autumnale, demonstrates significant efficacy in addressing three pivotal types of joint conditions: rheumatoid arthritis, osteoarthritis, and gouty arthritis. Its commendable anti-inflammatory and anti-arthritic actions rival the impact of potent



conventional inflammatory agents like Diclofenac sodium. Clinical evaluations substantiate the effectiveness of *Colchicum Autumnale* in treating gouty arthritis as a vital component of Gouticin and addressing rheumatoid arthritis as a key ingredient in Arthritin [22].

#### **Immunomodulatory Potential:**

An agglutinin extracted from *Colchicum Autumnale* tuber, known as *Colchicum Autumnale* agglutinin (CAA), exhibits intriguing immunomodulatory properties. While activating all murine T-lymphocytes, it selectively induces the proliferation of a subset of CD4<sup>+</sup> and CD8<sup>+</sup> mouse T-lymphocytes. These activated T-lymphocytes express heightened levels of activation markers, including CD69 and CD44, adding a layer of complexity to the plant's therapeutic mechanism [22].

#### **Pharmacological Actions:**

*Colchicum autumnale* (commonly known as Suranjan Shirin) has demonstrated significant efficacy in managing three major forms of joint pain: rheumatoid arthritis, osteoarthritis, and gouty arthritis. Its anti-inflammatory and anti-arthritic effects are robust across these conditions and are comparable to those of Diclofenac sodium, a well-established anti-inflammatory agent. Clinical evaluations have confirmed the effectiveness of *Colchicum autumnale* in treating gouty arthritis when included in formulations like Gouticin and in managing rheumatoid arthritis as a component of Arthritin. Furthermore, an agglutinin extracted from the tuber of *Colchicum autumnale*, known as *Colchicum autumnale* agglutinin (CAA), has been shown to activate all murine T-lymphocytes, although it does not trigger the proliferation of every activated cell. Instead, it selectively promotes the proliferation of a subset of CD4<sup>+</sup> and CD8<sup>+</sup> T-lymphocytes. These activated T-lymphocytes express elevated levels of activation markers, specifically CD69 and CD44, indicating a targeted immunomodulatory response. [21].

Colchicine binds to fibrillar protein tubulin to depolymerization of microtubules which decrease cell motility, precipitation of urate crystals in synovial fluids start inflammatory response by producing chemotactic factors to migration of granulocytes into joints colchicine prevent from this condition and relief from gout symptomatically.

#### **4.4. Curcuma longa Linn**

**Family:** Zingiberaceae

**Botanical Name:** *Curcuma longa* Linn

**Other Names:** Kurkum, haldi, turmeric

**Parts Used:** Rhizome

**Temperament:** Warm I order and dry I order

**Specific Chemical Constituents:** Curcumin, Curcuminoids, Tumerone, Cymene, Demethoxycurcumin

Turmeric (*Curcuma longa* L.) is a perennial herbaceous plant from the ginger family, Zingiberaceae. While it originates from tropical regions of South Asia, it is now extensively cultivated across tropical and subtropical areas worldwide. The distinctive deep orange-yellow turmeric powder is derived from the rhizomes of the plant, which are boiled and dried. Traditionally, turmeric has been widely used both as a spice and as a medicinal remedy, particularly in Asia. In Ayurvedic medicine, turmeric is primarily valued for its anti-inflammatory properties, while in traditional Chinese medicine, it serves as a stimulant, carminative, cordial, emmenagogue, astringent, detergent, and diuretic [24]. *Curcuma longa* is credited with various therapeutic benefits, including use as an anti-diabetic, hypolipidemic, anti-inflammatory, anti-diarrheal, hepatoprotective, anti-asthmatic, and anti-cancer agent. Additionally, it is widely applied in cosmetology [25].

The bioactive components of turmeric include curcuminoids, a class of flavonoids consisting of curcumin (diferuloylmethane), monodemethoxycurcumin, and bisdemethoxycurcumin, with curcumin comprising around 90% of the total curcuminoid content. Other components found in turmeric include sugars, proteins, and gums. Among these, curcumin is the most extensively

studied, representing 0.3-5.4% of raw turmeric. Turmeric also contains volatile oils such as tumerone, atlantone, and zingiberone, alongside its curcuminoids, sugars, proteins, and gums. Curcumin is a lipophilic polyphenol that remains stable in the acidic environment of the stomach, though it is insoluble in water [23].

Curcumin's phenolic structure contributes to its capability to neutralize oxygen-derived free radicals, including hydroxyl radicals, singlet oxygen, superoxide radicals, nitrogen dioxide, and nitric oxide (NO). Pharmacokinetic studies indicate that 40-85% of orally ingested curcumin passes through the gastrointestinal tract unchanged. Due to its limited absorption, curcumin is often combined with bromelain to enhance its absorption and boost its anti-inflammatory properties [23].

#### **Pharmacological Action:**

Curcumin has been found to inhibit a wide range of molecules that play key roles in inflammation, including phospholipase, lipoxygenase, COX-2, leukotrienes, thromboxane, prostaglandins, nitric oxide, collagenase, elastase, hyaluronidase, MCP-1, interferon-inducible protein, tumor necrosis factor (TNF), and interleukin-12. Notably, bisdemethylcurcumin (BDC), a derivative of curcumin, has been shown to be even more effective as an anti-inflammatory agent, evidenced by its superior ability to suppress TNF-induced NF- $\kappa$ B activation, its stronger anti-proliferative properties, and its enhanced capacity to induce reactive oxygen species (ROS) [25].

Curcumin also inhibits the enzyme cyclooxygenase-2 (COX-2), which is advantageous because COX-2 facilitates the activation of carcinogens and promotes the survival of cancerous cells by aiding in the formation of new blood vessels.

In cancerous cells, curcumin induces oxidative stress and increases ROS production, leading to apoptosis through interactions with DNA. Conversely, in normal cells, curcumin acts as a protective agent by preventing oxidative stress. It does so by conjugating with ROS, blocking their release in mitochondria, and inhibiting the activation of transcriptional factors. Literature suggests that this mechanism halts transcription, preventing abnormal cell growth, which allows normal cells to survive.

Given its potent anti-inflammatory effects, turmeric is highly effective in managing rheumatoid arthritis (RA). A recent study from Japan assessed the relationship between curcumin and interleukin-6 (IL-6), an inflammatory cytokine involved in the progression of RA. The study found that curcumin significantly reduced IL-6 levels, indicating that regular use of turmeric could be a valuable strategy for preventing the initial development of RA.

#### **4.5. Zingiber officinalis**

**Family:** Zingiberaceae

**Botanical Name:** Zingiber officinalis

**Other Names:** Ginger, Zinjbil, Adrak, sonth

**Parts Used:** Rhizome

**Temperament:** Warm III order and dry I order

**Specific Chemical Constituents:** 6-gingerol, 6-Shogaol, 6-paradol

Ginger, scientifically known as *Zingiber officinale*, is a member of the Zingiberaceae family and belongs to the genus *Zingiber* [27]. Native to tropical Asia, ginger is a perennial plant that thrives in tropical climates and is cultivated in regions such as Australia, Brazil, China, India, Jamaica, West Africa, and parts of the United States. The rhizome of ginger has a long history of use in traditional Chinese and Ayurvedic medicine, where it is valued for its antiemetic, antipyretic, and anti-inflammatory properties [28]. Ginger is commonly employed both as a spice and as an herbal medicine. The medicinal parts of the plant include its roots and rhizomes, which are utilized to treat various symptoms such as headaches, colds, nausea, and vomiting [27].

Ginger contains several bioactive compounds, with the most notable being gingerol, paradol, and shogaols [27].



**Pharmacological Action:**

Recent research has revealed that ginger contains bioactive compounds with significant antioxidant, anti-inflammatory, antimicrobial, and anti-cancer properties [27]. Moreover, an increasing number of studies suggest that ginger may be effective in managing various conditions, including neurodegenerative diseases, cardiovascular diseases, obesity, diabetes mellitus, chemotherapy-induced nausea and vomiting, and respiratory disorders [27].

Ginger has a longstanding reputation as an anti-inflammatory agent, with many of its components recognized for their anti-inflammatory effects. It has been shown to inhibit prostaglandin biosynthesis, disrupt the inflammatory cascade, and interact with the vanilloid nociceptor. Ginger's pharmacological properties are similar to those of non-steroidal anti-inflammatory drugs (NSAIDs) due to its ability to suppress prostaglandin synthesis by inhibiting both cyclooxygenase-1 and cyclooxygenase-2. Unlike NSAIDs, ginger also inhibits leukotriene biosynthesis by targeting 5-lipoxygenase. This dual inhibition of cyclooxygenase and 5-lipoxygenase suggests that ginger may offer a superior therapeutic profile with fewer side effects compared to traditional NSAIDs [28].

**4.6. Trachyspermum Ammi**

**Family:** Apiaceae

**Botanical Name:** *Trachyspermum ammi*

**Other Names:** Ajwain desi, bishop's weed

**Parts Used:** Fruit (seeds)

**Temperament:** Warm III order and dry III order

**Specific Chemical Constituents:** Thymol, p-cymene,  $\gamma$ -terpinene

*Trachyspermum ammi*, commonly known as ajwain, is native to Egypt and is also cultivated in countries such as Iraq, Iran, Afghanistan, Pakistan, and India. Within India, it is grown in regions including Madhya Pradesh, Uttar Pradesh, Gujarat, Rajasthan, Maharashtra, Bihar, and West Bengal. Belonging to the Apiaceae family, *Trachyspermum ammi* L. is a highly valued medicinal plant, with its seeds and roots holding significant therapeutic importance. The roots possess diuretic properties, while the seeds are renowned for their aphrodisiac effects. The seeds yield 2–4.4% of a brownish oil known as ajwain oil, with thymol as its primary component, which is widely used to treat gastrointestinal disorders, stimulate appetite, and alleviate bronchial issues. Additionally, ajwain oil exhibits fungicidal, antimicrobial, and anti-aggregatory properties, making it a versatile herbal remedy. Ajwain is traditionally used to treat various human and animal ailments. The seeds are recognized for their stimulant, antispasmodic, and carminative properties, making them an effective treatment for flatulence, atonic dyspepsia, and diarrhea [31].

Ajwain essential oil is primarily composed of thymol (87.75%) and carvacrol (11.17%), along with non-phenolic components like p-cymene (60.78%) and  $\gamma$ -terpinene (22.26%) [29].

An analysis of ajwain seeds reveals they contain fiber (11.9%), carbohydrates (38.6%), tannins, glycosides, moisture (8.9%), protein (15.4%), fat (18.1%), saponins, flavonoids, and mineral content (7.1%) that includes calcium, phosphorus, iron, and nicotinic acid. The seeds yield 2% to 4% brownish essential oil, primarily composed of thymol (35% to 60%). The non-thymol fraction includes compounds such as paracymene,  $\gamma$ -terpinene,  $\alpha$ - and  $\beta$ -pinenes, dipentene,  $\alpha$ -terpinene, and carvacrol. Additionally, small amounts of camphene, myrcene, and  $\alpha$ -3-carene have been detected in the plant. Alcoholic extracts of ajwain seeds also contain a highly hygroscopic saponin. From the natural products of *Trachyspermum ammi*, a yellow, crystalline flavonoid and a steroid-like substance have been isolated, alongside 6-O- $\beta$ -glucopyranosyloxythymol, glucoside, and 25% oleoresin containing 12% volatile oil, including thymol,  $\gamma$ -terpinene, paracymene, and  $\alpha$ - and  $\beta$ -pinene. The primary oil constituents of *Trachyspermum ammi* include carvone (46%), limonene (38%), and dillapiole (9%) [31].

**Pharmacological Action:**

The anti-inflammatory potential of the total alcoholic extracts (TAE) and total aqueous extract (TAQ) of the Ajwain seeds was determined. TAE and TAQ displayed significant ( $P < 0.001$ ) anti-

inflammatory activity in both the animal models. The weights of the adrenal organs were found to be significantly increased in TAE and TAQ treated animals. TAE and TAQ extracts from the ajwain seeds show significant anti-inflammatory potential [31].

#### 4.7. *Boswellia serrata*

**Family:** Burseraceae

**Botanical Name:** *Boswellia serrata*

**Other Names:** Indian frankincense, Salai guggul

**Parts Used:** Resin

**Temperament:** Warm II order and dry II order

**Specific Chemical Constituents:** Boswellic acids, Terpenoids

*Boswellia serrata* is a tree native to India, Africa, and the Middle East, valued for its resin, which has been used in traditional Ayurvedic medicine for centuries. The resin, known as Indian frankincense, contains boswellic acids, which are potent anti-inflammatory compounds. These acids inhibit the production of pro-inflammatory enzymes, including 5-lipoxygenase, which is implicated in the inflammatory process of gout. Studies have shown that *Boswellia serrata* extract significantly reduces inflammation and pain associated with gout and other forms of arthritis [32].

#### **Pharmacological Action:**

The anti-inflammatory effects of *Boswellia serrata* are primarily attributed to the inhibition of 5-lipoxygenase and other inflammatory mediators, such as leukotrienes. Clinical studies have demonstrated that boswellic acids can reduce the pain and swelling associated with gout by modulating the inflammatory process at the molecular level. Additionally, *Boswellia serrata* has been shown to protect against cartilage degradation, offering long-term benefits for gout sufferers [33].

#### 4.8. *Withania somnifera*

**Family:** Solanaceae

**Botanical Name:** *Withania somnifera*

**Other Names:** Ashwagandha, Winter cherry, Indian ginseng

**Parts Used:** Root

**Temperament:** Warm I order and dry I order

**Specific Chemical Constituents:** Withanolides, Alkaloids, Sitoindosides

*Withania somnifera*, commonly known as Ashwagandha, is a well-known herb in Ayurvedic medicine, revered for its adaptogenic and anti-inflammatory properties. The root contains bioactive compounds called withanolides, which have been shown to reduce inflammation and oxidative stress. Recent studies suggest that *Withania somnifera* can be effective in managing gout by lowering uric acid levels and inhibiting the inflammatory processes that lead to joint pain and swelling [34].

#### **Pharmacological Action:**

The anti-inflammatory action of *Withania somnifera* is mediated through the inhibition of pro-inflammatory cytokines and the suppression of oxidative stress markers. It also modulates the immune system, reducing the overactive inflammatory response typical in gout. Additionally, the herb's ability to enhance antioxidant defenses further contributes to its therapeutic potential in gout management [35].

#### 4.9. *Nigella sativa*

**Family:** Ranunculaceae

**Botanical Name:** *Nigella sativa*

**Other Names:** Black cumin, Kalonji, Habbat al-barakah

**Parts Used:** Seeds

**Temperament:** Warm II order and dry II order

**Specific Chemical Constituents:** Thymoquinone, Nigellone, Alpha-hederin

*Nigella sativa*, commonly known as black cumin or Kalonji, is a medicinal plant used extensively in Unani and traditional Islamic medicine. The seeds of *Nigella sativa* are rich in thymoquinone, an active compound known for its anti-inflammatory, antioxidant, and uric acid-lowering properties. Research has indicated that *Nigella sativa* can help reduce the symptoms of gout by lowering serum uric acid levels and inhibiting inflammatory pathways [36].

**Pharmacological Action:**

The anti-gout effects of *Nigella sativa* are primarily due to the action of thymoquinone, which inhibits the production of uric acid and reduces the activity of inflammatory cytokines such as TNF- $\alpha$  and IL-6. Additionally, the antioxidant properties of *Nigella sativa* help to neutralize free radicals, further reducing inflammation and preventing tissue damage in gout [37].

#### 4.10. *Glycyrrhiza glabra*

**Family:** Fabaceae

**Botanical Name:** *Glycyrrhiza glabra*

**Other Names:** Licorice, Mulethi, Sweet root

**Parts Used:** Root

**Temperament:** Warm II order and moist I order

**Specific Chemical Constituents:** Glycyrrhizin, Flavonoids, Isoflavonoids

*Glycyrrhiza glabra*, commonly known as licorice, has been used for centuries in traditional medicine for its anti-inflammatory, anti-viral, and immunomodulatory properties. The root of the plant contains glycyrrhizin, a saponin glycoside, which has been shown to reduce inflammation and lower uric acid levels. Licorice also acts as a natural cortisone, helping to manage the symptoms of gout by reducing pain and swelling [38].

**Pharmacological Action:**

The anti-inflammatory and uric acid-lowering effects of *Glycyrrhiza glabra* are attributed to glycyrrhizin, which inhibits the activity of enzymes involved in the production of uric acid and the inflammatory response. Additionally, the flavonoids present in licorice have strong antioxidant properties, which help in protecting tissues from oxidative damage caused by gout [39].

### 5. Conclusion

This paper provides an exhaustive exploration of medicinal plants as referenced in ancient literature, shedding light on traditional remedies employed for treating gout. The rich history of herbal interventions offers valuable insights into the roots of gout management practices, and the utilization of unprocessed extracts underscores the historical significance of these botanical solutions.

As we delve into the future of gout treatment, it becomes imperative to scrutinize not only the efficacy of these herbal remedies but also the specific phytochemical components responsible for their anti-gout potential. Rigorous preclinical and clinical investigations are crucial to unveil the full spectrum of these plant-derived medicines, ensuring their safety and efficacy in modern healthcare. Furthermore, the prospect of combination therapy presents a promising avenue. By harnessing the synergistic effects of various herbal components, we may formulate drugs that surpass the efficacy of individual remedies. This approach opens up possibilities for more successful and comprehensive gout management strategies. Despite the generally perceived safety of herbal pharmaceuticals, it is essential to acknowledge the inherent challenges associated with their usage. Safety concerns, stemming from factors like inherent toxicity, adulteration, contamination, heavy metals content, and interactions with other drugs and herbs, necessitate a vigilant approach. Rigorous quality control measures, standardized protocols, and continuous monitoring are imperative to mitigate potential risks and ensure the safety of herbal interventions in the complex landscape of gout treatment.

In conclusion, while the exploration of medicinal plants in gout management holds promise, a cautious and evidence-based approach is essential. The rich legacy of traditional remedies serves as an inspiration, but as we stride into modern scientific inquiry, a meticulous examination of safety and efficacy parameters will guide us towards harnessing the true potential of herbal interventions in the treatment of gout.

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