**Chinese Journal of Health Management**
***Volume 20 Issue 4, Year of Publication 2026, Page 1002-1018 | DoI-10.564220/1674-0815.2026.127***

ISSN: 1674-0815

**Chinese Journal of Health Management**

Chinese Medical Association


# Traditional Medicinal Plants as Natural Anti-Inflammatory Remedies: A Systematic Review

**Puja Kumari *[1], Chhatrapal[1], Shristi Gupta[1], Prerna Jaiswal, Rishikesh Gupta[3], Sanjay Jain[1]**

[1]Faculty of Pharmacy, Medicaps University, Indore, Madhya Pradesh
[2]Shri Nityanand College of Pharmacy, Dhar, Madhya Pradesh
[3]Institute of Pharmacy, Bundelkhand University, Jhansi, Uttar Pradesh

***Article Information***
Received: 13-03-2026
Revised: 08-04-2026
Accepted: 24-04-2026
Published: 25-05-2026

**Keywords**
*Anti-inflammatory, Phytochemicals, Medicinal Plants, Biological, Ingredients*

## ABSTRACT:
The intricate physiological vascular tissues exhibit specific responses when exposed to unfavorable stimuli, such as various illnesses, Cells that have been harmed or substances known as irritants, which induce an inflammatory response. Symptoms of the condition include redness, joint swelling, joint discomfort, joint stiffness, and impaired joint function. NSAIDs are now used to treat inflammation. Sadly, these procedures raise the possibility of blood clots causing heart attacks and strokes. Strong anti-inflammatory medicines made of natural components are already being developed as a consequence. Due to their chemical makeup, natural resources are a crucial source for the creation of novel medications. One of the most effective therapies for many diseases linked to inflammation is a natural substance derived from medicinal plants. The majority of the traditional anti-inflammatory drugs have negative side effects. The present study focuses on the gathering of information on potential phytochemicals derived numerous herbal plants have undergone rigorous testing in inflammatory models, demonstrating little adverse effects.

## INTRODUCTION:
Inflammation is a commonly seen defensive reaction Inflammatory responses are triggered as a result of tissue damage caused by physical trauma, exposure to hazardous substances, or the presence of microbiological organisms. The development of new pharmaceuticals depends heavily on medicinal plants. Humans may use medicinal plants to cure a variety of illnesses. Finding new, secure photochemical compound therapies is something that people all around the globe are interested in. A number of medicinal plants have received experimental validation. NSAIDs and other anti-inflammatory medications are used to alleviate pain and swelling brought on by inflammation.[1] The liver, gastrointestinal system, and other human biological systems may get damaged by NSAID usage over an extended period of time. [2] The ability to reduce inflammation is seen in many therapeutic plants. We looked at a few medicinal plants with potent anti-inflammatory qualities in this

review.[3] The signs of inflammation include swelling, discomfort, and redness. This enables professionals to handle these drugs with care and safety. In order to evaluate the quality of the research, this review lists the medicinal plants that may be found in Bangladesh that are said to have anti-inflammatory properties. The body reacts to an injury with a sophisticated system known as inflammation to promote healing and the return of normal function. Eleven plant species were chosen for this investigation based on their historical use as anti-inflammatory drugs in South African medicine [4]. It is related to a rise in capillary infiltration, vascular permeability, and leukocyte emigration. The five principal indicators of inflammation include elevated temperature (calor), increased blood flow resulting in redness (rubor), accumulation of fluid leading to swelling (tumor), discomfort or tenderness (dolor), and impaired functionality (functio laesa). According to how long the process takes to react to the detrimental cause, inflammation is distributed into three types: acute, which develops right away following an injury, a period of discomfort persists for a duration of many days.; chronic, which lasts for a few weeks; and subacute, which lasts for a few days. Inflammation is broadly categorized into two types:

### 1.1 Acute inflammation

Acute, or short-term, inflammation may be a symptom of a disease or injury. Numerous factors, such as mononuclear immune cells, The cellular constituents implicated in this biological process include macrophages, monocytes, and neutrophils, with fibroblast activation and proliferation, specifically in the context of angiogenesis, and fibrosis, exhibit interconnections with acute inflammation.

There are five main indications that are often used to assess the presence of acute inflammation:

**1. Pain:** The sensation described may either persist consistently or manifest upon contact with the affected area.
**2. Redness:** The increased blood flow to the capillaries in the area is responsible for this phenomenon.
**3. Loss of function:** including the inability to move a joint, breathe, smell, and other senses.
**4. Swelling:** If fluid accumulates, a condition known as edema may occur.
**5. Heat:** The afflicted region may become warm to the touch due to increased blood flow.

### 1.2 Chronic inflammation

The observed process is distinguished by the activation and subsequent proliferation of mononuclear immune cells, fibroblasts, angiogenesis, and fibrosis, along with macrophages, monocytes, neutrophils, and angiogenesis [6] Several variables that are shown in **Fig**ure 1 are associated with acute inflammation. The operational dynamics of the inflammatory response are contingent upon the relative abundance of pro-inflammatory and anti-inflammatory constituents. Different cytokines and hormonal effects are mediated by each of these antagonistic channels. TNF-, IL-1, IL-6, and TNF- induce inflammation whereas IL-10 and TGF- inhibit it. Depending on the situation, the differences may not always be the same. On the other hand, excessive or protracted inflammation develops when the systems that control homeostasis and the equilibrium between the two pathways are thrown off. Heat shock protein 70 (HSP70), TNF-, TNF-alpha, IFN-, interferon-gamma, IL-6, interleukin-6, IL-17, interleukin-17, CRP (C-reactive protein), and 4,interleukin-4.

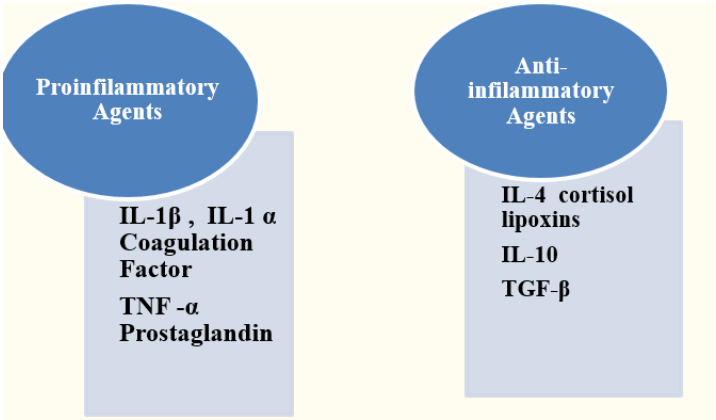


**Fig- 1 Pro-inflammatory agents and Anti-inflammatory**

**Chinese Journal of Health Management**
*Volume 20 Issue 4, Year of Publication 2026, Page 1002-1018 | DoI-10.564220/1674-0815.2026.127*

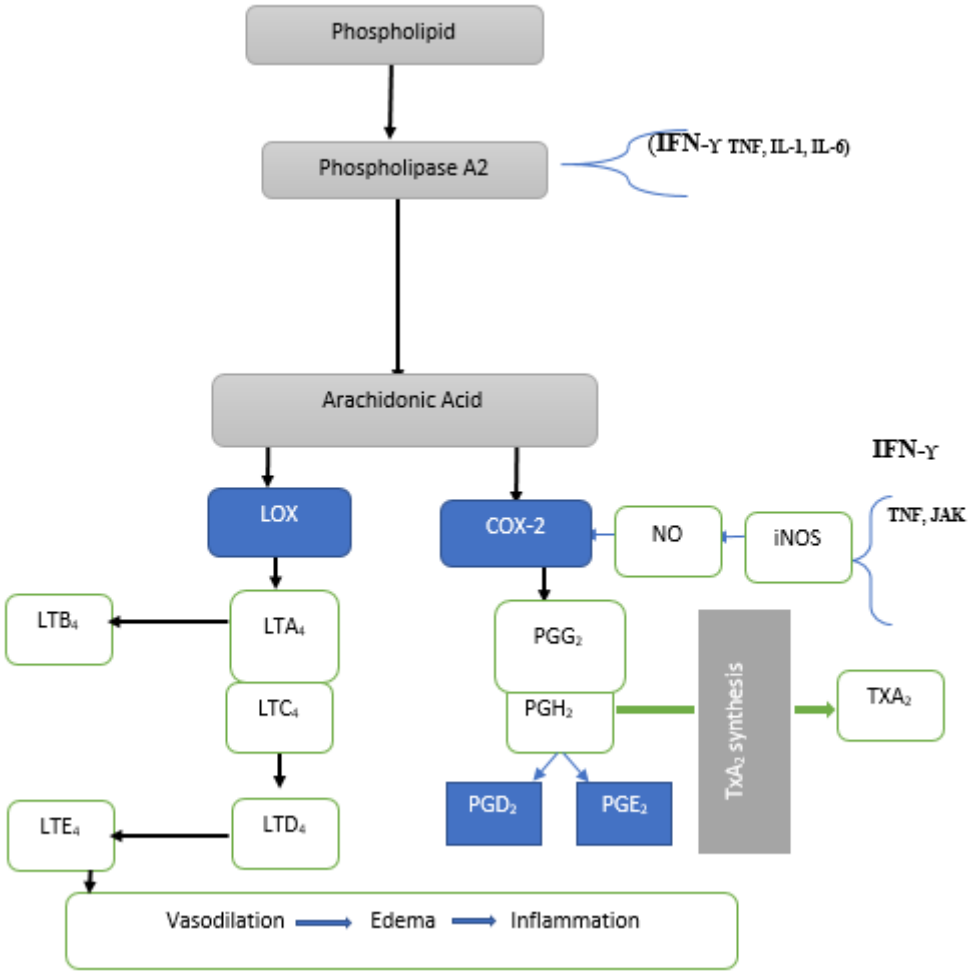


**Fig 2. Inflammation pathway. COX, cyclooxygenase; LOX, lipoxygenase; PG, prostaglandin; LT, leukotriene; TX, thromboxane; NO, nitric oxide; iNOS, inducible NO synthase; IFN, interferon; TNF, tumor necrosis factor; NF-κB, nuclear factor-κB; MAPK, mitogen activated protein kinase; JAK, janus kinase; IL, interleukin.**[8]

**1. *AchilleamillefoliumLinn.(Asteraceae)***

**Fig** -3 ***AchilleamillefoliumLinn*.[9]**

The perennial herb Achillea millefolium L., which is indigenous to Europe, The anti-inflammatory qualities of [subject] Traditional medicine has had a significant level of esteem within the discipline for a considerable duration. Historically, use of plant for the management of wounds and burns has been documented, inflammation, & dermatological irritation. According to study results, secondary metabolites such as isoprenoids & phenolics have shown a key role in manifestation anti-inflammatory characteristics in organisms. The use of extracts from A. Millefolium has been historically recognized in traditional medicine for its potential therapeutic effects on gastrointestinal and hepato-biliary disorders, as well as for its antiphlogistic properties. [9]

***2. Aconitum heterophyllum (Valeraneaceae)***

***3.***

**Fig-4** ***Aconitum heterophyllum (Valeraneaceae)*** **[10]**

A. Heterophyllum, often recognised as 'Ativisha' or 'Patis' in Ayurvedic medicine, botanical species. This treatment modality is used for the therapy of many pathological disorders impacting the neurological system, digestive system, pyrexia, and rheumatism. Ehanolic of A. Heterophyllum root comprises many chemical compounds such as alkaloids, glycosides, flavonoids, and sterols. Plants that possess chemical compounds belonging to certain chemical families have shown to have potent anti-inflammatory effects blocking prostaglandin pathways*.10].*

**3.** ***Adhatodavasica (Acanthaceae)***

**Fig-5** ***Adhatodavasica (Acanthaceae)*** **[11]**

Adhatodavasica L., often known as Adhatoda vasica, is a plant indigenous to the Acanthaceae family. Use of this plant as herbal remedy has been documented in several traditional medicinal practices throughout different regions. It has been historically employed for treating ailments such as colds, coughs, whooping cough, chronic bronchitis, asthma, as well as possessing sedative, expectorant, antispasmodic, anthelmintic, rheumatism, and rheumatic painful inflammatory swelling properties. Medicinal substance is administered several preparations, including fresh juice, decoction, infusion, and powder.

**4.** ***Bacopamonnieri Linn.(Scrophulariaceae)***

**Fig-6** ***Bacopamonnieri Linn.(Scrophulariaceae)*****[12]**

Bacopa monnieri, a glabrous succulent plant with creeping growth habit and distinct nodes, is often found in marshes and muddy coastal areas. Previously, it was used as a cognitive enhancer to boost memory, learning & attention. In the regions of India and Pakistan. Throughout history, the plant has been used for its potential as a cardiac tonic, digestive assistance, and as a means to improve respiratory function in cases of bronchoconstriction. The herb has anti-inflammation properties in a rat model of paw edema generated by carrageenan, resulting in a significant reduction of edema by 82% when compared to the effects of indomethacin. Furthermore, Bacopamonnieri exhibited a reduction in the activities of 5-lipoxygenase (5-LOX), 15-lipoxygenase (15-LOX), and cyclo-oxygenase-2 (COX-2). [12]

**5. *Cassia fistula L. (Caesalpiniaceae)***

**Fig-7 *Cassia fistula L. (Caesalpiniaceae)* [13]**

*Cassia fistula* tree widely distributed Indian timbers. The whole herb has medicinal properties that have the potential to be used in the management of dermatological ailments, inflammatory diseases, rheumatism, anorexia, and jaundice. The anti-inflammatory effect of Cassia fistula bark extracts has been shown to be considerable in both acute and chronic inflammation models in rats. Involvement of endogenous or exogenous reactive oxygen species (ROS) has suggested in the pathophysiology of several diseases, including as atherosclerosis, diabetes, cancer, arthritis, and the aging process.

**6. *Daphne pontica Linn. (Thymelaeaceae)***


**Fig**-8 ***Daphne pontica Linn. (Thymelaeaceae)*** [13]

The belief in the anti-cancer effects of Daphne species dates back to the second century AD. The roots of Daphne pontica have been identified as a source of flavonoid compounds, including daphnodorins, which has anticancer properties. Various kinds of Daphne have been used for the management of inflammatory conditions. Daphne pontica used for mitigation of rheumatic pain and inflammatory disorders. Findings indicate that extracts possess the ability to inhibit the production of PGE2 and IL-1ß.

**7. *Emblica officinalis (Euphorbiaceae)***

**Fig**-9 ***Emblica officinalis (Euphorbiaceae)***[14]

Emblica officinalis is geographically distributed over the subtropical and tropical areas of China, India, Indonesia & Malay Peninsula. The use of this substance in certain specific regions has been observed due to its anti-inflammatory and antipyretic properties. Recent research has shown anti-inflammatory properties in the aqueous portion of the methanol extract derived from plant leaves. The study was centered on the analysis of the impact of fractions on the synthesis of inflammatory mediators, namely leukotriene B4, platelet activating factor (PAF) & thromboxane, within a certain duration. [14]

**8.** ***Garcinia mangostana Linn. (Guttiferae)***

**Fig**-10 ***Garcinia mangostana Linn. (Guttiferae)***[15]

The use of the rinds of Garcinia mangostana fruit in traditional medicine has been seen for the purpose of treating injuries and skin infections. The hulls of the mangosteen fruit contain bioactive chemicals, namely alpha-mangostin and gamma-mangostin, which are notable xanthones. [15]

**9.** ***Lantana camara Linn. (Verbenaceae)***

**Fig- 11** ***Lantana camara Linn. (Verbenaceae)***[16]

Various species of Lantana possess aerial parts that are often used in traditional remedies for cancer and tumors. A herbal infusion consisting of leaves and blossoms was used as a remedy for fever, influenza, and gastrointestinal discomfort. Additional use of the plant include its antimalarial, antibacterial, and anti-diarrheal properties.

**10.** ***Lycopodium clavatum Linn. (Lycopodiaceae)***

**Fig- 12** ***Lantana camara Linn. (Verbenaceae)***[17]

Lycopodium clavatum, also known as club moss, has promising attributes that may contribute to its efficacy in wound healing. In a study conducted by Orhan et al., the authors investigated the potential anti-inflammatory effects of various extracts derived from the aerial parts of Lycopodium clavatum. The study employed a murine model in which capillary permeability was induced using acetic acid. Four distinct extracts were generated with petroleum ether, chloroform, ethyl acetate, methanol, and an alkaloidal fraction. The findings of the study revealed that just the chloroform extract and the alkaloidal fraction had a noteworthy anti-inflammatory impact, exceeding the effectiveness of Indomethacin.

**11.** ***Mangiferaindica Linn. ( Anacardiaceae)***

**Chinese Journal of Health Management**
*Volume 20 Issue 4, Year of Publication 2026, Page 1002-1018 | DoI-10.564220/1674-0815.2026.127*

**Fig- 13** ***Mangiferaindica Linn. ( Anacardiaceae)*** [18]

Mangifera indica, often referred to as mango, is a tropical and subtropical fruit-bearing tree from the Anacardiaceae family. The many constituents of this entity have been widely used in traditional medicine throughout various societies, including a wide range of medicinal uses. Mangifera indica has been recorded in traditional medicine as being used for several therapeutic applications. One of the methods used involves the application of a fluid extract or infusion derived from the bark to treat medical disorders such as monorrhagia, leucorrhoea, bleeding piles, and pulmonary haemorrhage. Calcined leaves of the idibs plant are used for the purpose of eliminating warts on the eyelids. The treatment of diabetes involves the use of dried powdered leaves.

12. ***Phyllanthuspolyphyllus Linn. (Euphorbiaceae)***

**Fig**-14 ***Phyllanthuspolyphyllus Linn. (Euphorbiaceae)***[19]

The aforementioned botanical specimen is a little shrub that is used in indigenous therapeutic customs due to its anti-inflammatory attributes throughout the tropical and subtropical territories of India and Sri Lanka. Four compounds that were extracted from the whole plant, comprising of one benzenoid and three arylnaphthalide lignans, have shown inhibitory properties on the generation of nitric oxide (NO) and cytokines, namely tumor necrosis factor (TNF-) and interleukin-12 (IL-12). The pro-inflammatory cytokines TNF-α and IL-12 Cytokines are generally recognized as crucial mediators that are generated during the first stages of acute and chronic inflammatory conditions, including allergies, rheumatoid arthritis, and septic shock. Chemical substances accountable using Phyllanthuspolyphyllus as an anti-inflammatory remedy in conventional medication. [19]

**13.** ***Ricinuscommunis Linn. (Euphorbiaceae)***

**Fig**-15 ***Phyllanthuspolyphyllus Linn. (Euphorbiaceae)***[20]

Ricinus communis Linn. Species is found in a broad range of tropical and subtropical areas around the globe. The primary objective of this study was to investigate the anti-inflammatory and free radical scavenging properties of

the methanolic extract derived from the root of Ricinus communis in Wistar albino rats, as conducted by llavarasan et al. The observed pharmacological effect in this investigation might potentially be ascribed to the existence of phytochemicals, including flavonoids, alkaloids, and tannins, present in the plant extract.[20]

**14. *Sesbaniasesban Linn. (Leguminosae)***

**Fig**- 16 ***Phyllanthuspolyphyllus Linn. (Euphorbiaceae)***[21]

The genus Sesbania esban has around 50 species, with the majority of them being annual plants. Africa has the highest level of spice variety, with a total of 33 distinct species. While there has been some attention given to annual species, modern studies have mostly concentrated on perennial species. Among the several perennial species, Sesbania sesban has shown considerable promise.The plant in question is a little, long-lasting tree characterized by its sturdy stems, vibrant yellow blossoms, and elongated seed pods.[21]

15**. *Sidacordifolia Linn. (Malvaceae)***

**Fig**-17 ***Sidacordifolia Linn. (Malvaceae)*** [22]

Sidacordifolia is classified as a perennial subshrub of the Malvaceae family, also known as the mallow family. The plant species in question has successfully established itself in several regions around the globe and Currently, it has been acknowledged that this particular plant species is regarded as an invasive weed in several regions, including Africa, Australia, the Hawaiian islands, New Guinea, and French Polynesia. Sidacordifolia is often used in traditional medicine for the management of oral mucosa infection, blenorrhea, asthmatic bronchitis, and nasal congestion. The compound in question has been the subject of scientific inquiry for its potential anti-inflammatory properties, its ability to inhibit cellular proliferation, and its potential to promote liver regeneration. [22]

16. ***Thespesiapopulnea (Malvaceae)***

**Fig**- 18 ***Thespesiapopulnea (Malvaceae)*** [23]

The oil derived from the leaves and bark of Thespesiapopulnea is used as a therapeutic intervention in the geographical areas of southern India and Sri Lanka. This substance is used in the medical field to address fracture wounds and is also utilised as an anti-inflammatory poultice for the purpose of managing ulcers and boils. The ethanolic extract obtained from Thespesia populnea has shown promising anti-inflammatory properties in both

acute and chronic conditions. The results of the phytochemical analysis revealed that the ethanolic extract derived from the bark of the plant had a diverse range of constituents, including alkaloids, carbohydrates, proteins, tannins, phenols, flavonoids, gums and mucilage, saponins, and terpenes.[ 23]

**17. *Curcuma longa***

**Fig-19 *Curcuma longa (Zingiberaceae)* [24]**

Curcuma longa, often identified as Turmeric, is a plant native to India. [22]. Curcumin, the primary secondary metabolite of C. longa, plant's anti-inflammatory qualities are significantly influenced by its participation in biological processes.[24].

**18. *Zingiber officinale***

**Fig-20 *Zingiber officinale* [23]**

The effects of orally administering Zingiber officinale extract have shown variability and inconsistency, contingent upon the dosage administered.[25] The single or double dose of ginger extract to mice has been shown to enhance the levels of tumor necrosis factor-α (TNF-α) in peritoneal cells. However, prolonged intake of the extract has seen to rise blood corticosterone levels and decrease pro-inflammatory indicators.[26].

**19. *Rosmarinus officinalis***

**Fig- 21 *Rosmarinus officinalis***

Reports suggest that Rosmarinus officinalis shows wound healing activity in mice [27]. The plant's anti-inflammatory action was shown to be linked to molecular disruption in the complementary system, particularly in the attachment of C3b [28].

**20. *Borago officinalis***

**Fig 22** ***Borago officinalis***

The plant under consideration serves as a significant reservoir of gamma linoleic acid (GLA), with a GLA content of 25%. This compound functions by increasing the amount of prostaglandin-E (PGE), so resulting in the augmentation of cyclic adenosine monophosphate (cAMP). Consequently, GLA may be regarded as a potent suppressor of TNF-α. The aforementioned mechanism elucidates the anti-inflammatory impact of borage oil in the context of rheumatoid arthritis (RA) [29].

**21.** ***Urticadioica***

**Fig. 23** ***Urticadioica***

The inhibitory ability of leaf extract from U. dioica has been shown in scientific investigations, specifically targeting the proinflammatory transcription factor NF-κB. It is noteworthy to mention that there has been seen an elevation in NF-κB levels within the synovial fluid of persons diagnosed with rheumatoid arthritis [30]. The present excerpt has shown potential anti-inflammatory effects in the context of allergic rhinitis via many mechanisms. These include the antagonism of the H1-receptor, reduction of PGD2 synthesis (a prostaglandin unique to allergies), and an inhibitory impact on mast cell tryptase. [31].

**22.** ***Oenotherabiennis***

**Fig. 24** ***Oenotherabiennis***

Oenotherabiennis is a plant species belonging to the Onagraceae family, which is indigenous to Central America [32].Gamma-linolenic acid (GLA), linear aliphatic alcohols (such as Tetracosanol), and the phenolic compound ferulic acid are active constituents found in evening primrose oil. Previous study has shown evidence of the preventive benefits of these components against proinflammatory markers[33]. The presence of sterols, including $\beta$-Sitosterol and Campesterol, in this oil has been shown to have a modulating effect on nitric oxide (NO), TNF-$\alpha$, IL-1$\beta$, and thromboxane B2 (TXB2). As a result, the expression of the COX-2 gene is suppressed. As a result, it can be seen that primrose oil has a more notable anti-inflammatory impact in comparison to borage oil. *[34].*

**23.** ***Harpagophytumprocumbens***

**Chinese Journal of Health Management**
*Volume 20 Issue 4, Year of Publication 2026, Page 1002-1018 | DoI-10.564220/1674-0815.2026.127*

**Fig. 24 *Harpagophytumprocumbens***

Previous research have provided confirmation on the anti-inflammatory benefits of harpagoside [35]. The suppressive effects of Devil's claw root extract on nitric oxide (NO) have been reported, inflammatory cytokines such as interleukin-6 (IL-6), interleukin-1 beta (IL-1$\beta$), and tumor necrosis factor-alpha (TNF-$\alpha$), Furthermore, it has been shown that this compound hinders the metabolic process of arachidonic acid and the subsequent synthesis of eicosanoids. These effects eventually lead to the suppression of cyclooxygenase-2 (COX-2) and the consequent decrease in inflammation. [36-38].

**24. *Boswelliaserrata.***

**Fig. 25 *Boswelliaserrata***

The material under consideration is an oleo gum resin obtained from the Boswellia tree, which is native to India [39].The modulation of inflammatory mediators, including TNF-$\alpha$, IL-1$\beta$, IL-6, IFN-$\gamma$, and PGE2, by the use of B. serrata extract has been shown in both in vivo and in vitro experiments [40, 41]. The primary constituent of this gum is boswellic acid, which has the ability to block C3 convertase and decrease the traditional route of the complement system [42 43]. Similarly, it has shown local anti-inflammatory properties as well as systemic benefits.

**Table of Medicinal used in Arthritis**

| S.N. | Phytochemicals | Plant source | Biological activities | References |
|---|---|---|---|---|
| 1 | Boswellic acid | Boswellia serrata Roxb. ex Coleb. Burseraceae | Showed anti-inflammatory and complement inhibitor activities in rats; down regulation of TNF-a and IL-1 | Kapil, 1994; Manjula et al., 2006 |
| 2 | Curcumin (diferuloyl methane) | Curcuma longa L. Zingiberaceae | Anti-rheumatoid activity in human clinical trials;Reduced inflammation in carrageenan induced edema, cotton pellet -granuloma and adjuvant-induced arthritis | Deoder et al., 1980; Shah Biren et al. 2006 |
| 3 | Emblica officinalis | Emblica officinalis Phyllanthus emblica L. | Antiadjuvant induced arthritis in rats. | Tripathyer al. 2010 |
| 4 | Bacopa monnieri | Bacopa monnieri (BM), a perennial creeping plant belonging to the family Scrophulariaceae | Inhibition of protein denaturation. etc | Volluri eral., 2011 |
| 5 | Athatoda vasica | Adhatoda vasica or Malabar nut. It belongs to the Acanthaceae family. | The use of a botanical specimen with medicinal properties has been documented throughout Asia, particularly in China, for the purpose of treating various ailments such as injuries, arthritis, and rheumatism. | Tahmida Shamsuddin et al. Mini Rev Med Chem. 2021. |
| 6 | Cimicifuga racemosa L. (Ranunculaceae) | Cimiracemate A | In vitro inhibition of TNF-a production by downregulation of ERK and NF-KB activities | Yang et al. (2009) |
| 7 | Mangifera indica Wall. | Mangiferin | In vivo inhibition of IgE production | Rivera et al. (2006) |

| | | | | |
|---|---|---|---|---|
| | (Anacardiaceae) | | | |
| 8 | Vitis vinifera L. (Vitaceae) | Resveratrol | In vitro/in vivo anti inflammatory effects | orallo 2006 |
| 9 | Withania somnitera | sitoindosides and withaferin A | A significant proportion of individuals, namely 76%, had amelioration in the signs and symptoms associated with rheumatoid arthritis (RA). | Kikshapathi and Kumari, 1999 |
| 10 | Alstonia scholarisa | Echitamine, tubotaiwine, echitamidine and picrinine | Reduction of total leucocyte migration along with COX, LOX and NO | 98Ahmad M et al Herb Med 2018 |
| 11 | Andrographis paniculata | Andrographolide and its derivatives. | Reduces PGE2 production. | Yan J et. al Cell Biol Toxicol 2012 |
| 12 | Black pepper | Piperene, sabinene, pinene, terpene, limonene, mercene, camphene. | Anti-inflammatory, carminative, anti-flatulent, pyridoxine, riboflavin, thiamin and niacin | Kumkum Agarwal drug and reserch 2014 |
| 13 | Lanata camara | L. camara oil | The aim of this research was to assess the anti-inflammatory and anti-hyperalgesic properties of the hydroethanolic extract derived from the aerial portions of L. canescens. | G.N. Silva et al.2005 |
| 14 | Achillea millefolium | The chemical composition of the substance consists of a volatile oil including azulene, caryophyllene, thujone, eucalyptol, α- and β-pinene, and borneol. | The medicinal applications of A. millefolium L. have been known for Anti, inflammation. | B.Gopalkrishnan 15 june 2017 |
| 15 | Zingiber officinale | Shogaols, paradols, and gingerols | Reduce the activity of several chemical substances that promote joint inflammation | Rownak jahan 2014 may 27 |

## CONCLUSION:

Inflammatory conditions have a significant influence on a vast proportion of the global population. Throughout history, flowers have had a substantial influence within the realm of human healing. Throughout history, a diverse array of plant species has been used as traditional treatments for the purpose of treating inflammatory disorders. A significant amount of these medications undergone scientific testing and verification, confirming their efficacy as anti-inflammatory agents. Numerous chemical families, including polyphenols, flavonoids, terpenoids, alkaloids, anthraquinones, lignans, polysaccharides, saponins, and peptides, are present, have been shown as exhibiting significant activity in the context of anti-inflammatory pharmaceuticals produced from natural origins. Certain compounds have been identified as inhibitors of phospholipase, while others have been said to have inhibitory effects on tumor necrosis factor (TNF) in various inflammatory circumstances. Moreover, biochemical studies have shown that flavonoids have the capacity to block both the cyclooxygenase and lipoxygenase pathways of arachidonic acid metabolism, depending on their unique chemical structures. It has been shown that alkaloids with a pyridine ring structure have significant anti-inflammatory effects. For instance, Berberine derived from Berberis recognized as a standard therapeutic agent for arthritis. Terpenoids have been shown to significantly impede the development of chronic joint inflammation. Terpenoids have potential to influence many pathways related to inflammation, which might arise from diverse etiological factors. Consequently, Conducting comprehensive research on the pharmacological mechanisms of these natural medications is of utmost importance, the objective is to ascertain the precise active ingredient accountable for the anti-inflammatory qualities shown by the subject, and thereafter devise suitable formulations that have the potential to successfully mitigate inflammatory disorders.

## REFERENCES:

1. V. Gyurkovska, K. Alipieva, A. Maciuk et al., “Anti-inflammatory activity of Devil’s claw *in vitro* systems and their active constituents,” *Food Chemistry*, vol. 125, no. 1, pp. 171–178, 2011.
2. M. Brzeski, R. Madhok, and H. A. Capell, “Evening primrose oil in patients with rheumatoid arthritis and side-effects of non-steroidal anti-inflammatory drugs,” *British Journal of Rheumatology*, vol. 30, no. 5, pp. 370–372, 1991.
3. K. L. Soeken, S. A. Miller, and E. Ernst, “Herbal medicines for the treatment of rheumatoid arthritis: a systematic review,” *Rheumatology*, vol. 42, no. 5, pp. 652–659, 2003.
4. Nadkarni AK. Indian Materia Medica.Popular Press Bldg. 2000.
5. Cardinali PD and Esquifino IA. Circardian disorganization in experimental arthritis.Neuro Signals. 2003;12:267-282.
6. M. C. Y. Heng, M. K. Song, J. Harker, and M. K. Heng, “Drug-induced suppression of phosphorylase kinase activity correlates with resolution of psoriasis as assessed by clinical, histological and immunohistochemical parameters,” *The British Journal of Dermatology*, vol. 143, no. 5, pp. 937–949, 2000.
7. Daniel SF. Therapeutic Administraion of a selective inhibitor of nitric oxide synthaseDoes not ameliorate the chonic inflammation and tissue damage associated with adjuvant-Induced arthritis in rats, J PharmacolExpt Ther.1998;32:714-721.

8. Zumora RA and Billar TR. Inducible nitric oxide synthase and inflammatory disease. Mol Med 2000;6:347-356.
9. Corbett JA. Interleukin-IB-induced formation of EPR-detectable iron-nitrosyl complexes inIslets of Langerhans. J BiolChem.1991;266:21351-21354.
10. Mederos M,. Effect of chronic nitric oxide synthesis inhibition on the inflammatory responseInduced by carrageenan in rats, Eur J Pharmacol.1995; 285:109.
11. Malizos KN. Do steroids, conventional non-steroidal anti-inflammatory drugs and selectiveCox-2 inhibitors adversely effect fracture healing. J Musculoskelet Neuronal Interact. 2009;9:44-52.
12. Trivedi P.C .Indian medicinal Plants, published by Avishkar publisher, distributor (Raj) 2009, pg .no7
13. Yadav R, Jain GC. Antifertility effect of aqueous extract of seeds of *Cassia fistula* in female rats. *Adv Contracept.* 1999;15:293–301. [PubMed] [Google Scholar]
14. Variya CB, Emblica officinalis (Amla): A review for its phytochemistry, ethnomedicinal uses and medicinal potentials with respect to molecular mechanisms, National Medicine of Library 2016 Sep;111:180-200
15. Obolskiy D, Pischel I, Siriwatanametanon N, Heinrich M. Garcinia mangostana L.: a phytochemical and pharmacological review, Phytother Res. 2009 Aug;23(8):1047-65.
16. Rajashekar Y, Ravindra KV, Bakthavatsalam N.J Food Sci Technol. 2014 Nov;51(11):3494-9. doi: 10.1007/s13197-012-0884-8. Epub 2012 Oct 30.PMID: 26396352
17. Orhan I, Terzioglu S, Sener B. Alpha-onocerin: an acetylcholinesterase inhibitor from Lycopodium clavatum Planta Med. 2003 Mar;69(3):265-7. doi: 10.1055/s-2003-38489.PMID: 12677532
18. Khan MN, Nizami SS, Khan MA, Ahmed Z. New saponins from *Mangifera Indica*. *J Nat Prod.* 1993;56:767–70. [PubMed] [Google Scholar]
19. Moreira J., Klein-Júnior L. C., Cechinel Filho V., de Campos Buzzi F. Anti-hyperalgesic activity of corilagin, a tannin isolated from *Phyllanthus niruri* L. (Euphorbiaceae) *Journal of Ethnopharmacology.* 2013;146(1):318–323. doi: 10.1016/j.jep.2012.12.052. [PubMed] [CrossRef] [Google Scholar]
20. Shukla B, Visen P, Patnaik G, Kapoor N, Dhawan B. Hepatoprotective effect of an active constituent isolated from the leaves of *Ricinus communis Linn*. *Drug Dev Res.* 1992;26(2):183–193. doi: 10.1002/ddr.430260207. [CrossRef] [Google Scholar]
21 Zerihun N and Getachew A ,Sesbaniasesban (L.) Merrill: Potential uses of an underutilized multipurpose tree in Ethiopia , African Journal of Plant Science, Vol 7(10), pp 468-475, October 2013 / DOI: 10.5897/AJPS2012.0716.
22. S. Montserrat-de La Paz, M. A. Fern´andez-Arche, M. ´Angel- Mart´ın, and M. D. Garc´ıa-Gim´enez, "Phytochemical characterization of potential nutraceutical ingredients from Evening Primrose oil (Oenotherabiennis L.)," Phytochemistry Letters, vol. 8, pp. 158–162, 2014.
23. M. R. Al-Sereiti, K. M. Abu-Amer, and P. Sen, "Pharmacology of rosemary (*Rosmarinus officinalis* Linn.) and its therapeutic potentials," *Indian Journal of Experimental Biology*, vol. 37, no. 2, pp. 124–130, 1999.
24. J. S. Jurenka, "Anti-inflammatory properties of curcumin, a major constituent of *Curcuma longa*: a review of preclinical and clinical research," *Alternative Medicine Review*, vol. 14, no. 2, pp. 141–153, 2009.
25. M. Brzeski, R. Madhok, and H. A. Capell, "Evening primrose oil in patients with rheumatoid arthritis and side-effects of non-steroidal anti-inflammatory drugs," *British Journal of Rheumatology*, vol. 30, no. 5, pp. 370–372, 1991.
26. H. Ueda, K. Ippoushi, and A. Takeuchi, "Repeated oral administration of a squeezed ginger (*Zingiberofficinale*) extract augmented the serum corticosterone level and had anti-inflammatory properties," *Bioscience, Biotechnology and Biochemistry*, vol. 74, no. 11, pp. 2248–2252, 2010.
27. M. A. Abu-Al-Basal, "Healing potential of *Rosmarinus officinalis* L. on full-thickness excision cutaneous wounds in alloxan-induced-diabetic BALB/c mice," *Journal of Ethnopharmacology*, vol. 131, no. 2, pp. 443–450, 2010.
28. A. Sahu, N. Rawal, and M. K. Pangburn, "Inhibition of complement by covalent attachment of rosmarinic acid to activated C3b," *Biochemical Pharmacology*, vol. 57, no. 12, pp. 1439–1446, 1999.
29. R. E. Kast, "Borage oil reduction of rheumatoid arthritis activity may be mediated by increased cAMP that suppresses tumor necrosis factor-alpha," *International Immunopharmacology*, vol. 1, no. 12, pp. 2197–2199, 2001.
30. K. Riehemann, B. Behnke, and K. Schulze-Osthoff, "Plant extracts from stinging nettle (Urticadioica), an antirheumatic remedy, inhibit the proinflammatory transcription factor NF-κB," FEBS Letters, vol. 442, no. 1, pp. 89–94, 1999.
31. B. Roschek Jr., R. C. Fink, M. McMichael, and R. S. Alberte, "Nettle extract (Urticadioica) affects key receptors and enzymes associated with allergic rhinitis," Phytotherapy Research, vol. 23, no. 7, pp. 920–926, 2009.
32. S. Montserrat-de la Paz, M. D. Garc´ıa-Gim´enez, M. ´Angel- Mart´ın, M. C. P´erez-Camino, and A. Fern´andez Arche, "Longchain fatty alcohols from evening primrose oil inhibit the inflammatory response in murine peritoneal macrophages," Journal of Ethnopharmacology, vol. 151, no. 1, pp. 131–136, 2014.
33. S. Montserrat-de La Paz, M. A. Fern´andez-Arche, M. ´Angel- Mart´ın, and M. D. Garc´ıa-Gim´enez, "Phytochemical characterization of potential nutraceutical ingredients from Evening Primrose oil (Oenotherabiennis L.)," Phytochemistry Letters, vol. 8, pp. 158–162, 2014.
34. S.Montserrat-de la Paz, ´ A. Fern´andez-Arche, M. ´Angel-Mart´ın, and M. D. Garc´ıa-Gim´enez, "The sterols isolated from Evening Primrose oil modulate the release of proinflammatory mediators," Phytomedicine, vol. 19, no. 12, pp. 1072–1076, 2012.
35. T. H.-W. Huang, V. H. Tran, R. K. Duke et al., "Harpagoside suppresses lipopolysaccharide-induced iNOS and COX-2 expression through inhibition of NF-κB activation," Journal of Ethnopharmacology, vol. 104, no. 1-2, pp. 149–155, 2006.
36. B. L. Fiebich, B. L. Fiebich, M. Heinrich, K. O. Hiller, and N. Kammerer, "Inhibition of TNF-$\alpha$ synthesis in LPS-stimulated primary human monocytes by Harpagophytum extract SteiHap 69," Phytomedicine, vol. 8, no. 1, pp. 28–30, 2001.
37. D. Loew, J. M¨ollerfeld, A. Schr¨odter, S. Puttkammer, and M. Kaszkin, "Investigations on the pharmacokinetic properties of Harpagophytum extracts and their effects on eicosanoid biosynthesis in vitro and ex vivo," Clinical Pharmacology & Therapeutics, vol. 69, no. 5, pp. 356–364, 2001.
38. Eichler C Koch (1970) Antiphlogistic, analgesic and spasmolytic effect of harpagoside, a glycoside from the root of Harpagophytum procumbens DC. *Arzneimittelforschung* 20 107–109 4392498
39. Krishnamurthy T, Shiva MP. Salai guggul (from *Boswellia serrata* Roxb.): Its exploitation and utilization. *Indian Forest.* 1977;103:466–74. [Google Scholar]

40. Leung AY, Foster S. 2nd ed. New York: John Wiley and Sons; 1996. Encyclopedia of common natural ngredients used in food, drugs and cosmetics; pp. 389–91. [Google Scholar]
41. Saudagar RB, Saokar S. Anti-inflammatory natural compounds from herbal and marine origin. Journal of Drug Delivery and Therapeutics. 2019 May 15;9(3):669-72.

42. .Lee JE, Kim IJ, Cho MS, Lee J. A Case of Rheumatoid Vasculitis Involving Hepatic Arteryin Early Rheumatoid Arthritis. J Korean Med Sci. 2017 Jul;32((7)):1207–10. [PMC free article] [PubMed] [Google Scholar]
43. Fox CQ, Ahmed SS. Physician Assistant's Clinical Review Cards. Philadelphia: F. A. Davis Company; 2002. pp. . pp. 138–139. [Google Scholar]
44. McInnes IB, Schett G. The pathogenesis of rheumatoid arthritis. N Engl J Med. 2011 Dec;365((23)):2205–19. [PubMed] [Google Scholar]
45. Chaudhari K, Rizvi S, Syed BA. Rheumatoid arthritis: current and future trends. Nat Rev Drug Discov. 2016 May;15((5)):305–6. [PubMed] [Google Scholar]
46. Picerno V, Ferro F, Adinolfi A, Valentini E, Tani C, Alunno A. One year in review: the pathogenesis of rheumatoid arthritis. Clin Exp Rheumatol. 2015 Jul-Aug;33((4)):551–8. [PubMed] [Google Scholar]
47. Alamanos Y, Voulgari PV, Drosos AA. Incidence and prevalence of rheumatoid arthritis, based on the 1987 American College of Rheumatology criteria: a systematic review. Semin Arthritis Rheum. 2006 Dec;36((3)):182–8. [PubMed] [Google Scholar].
48. Chopra A, Abdel-Nasser A. Epidemiology of rheumatic musculoskeletal disorders in the developing world. Best Pract Res Clin Rheumatol. 2008 Aug;22((4)):583–604. [PubMed] [Google Scholar]
49. McGonagle D, Hermann KG, Tan AL. Differentiation between osteoarthritis and psoriatic arthritis: implications for pathogenesis and treatment in the biologic therapy era. Rheumatology (Oxford) 2015 Jan;54((1)):29–38. [PMC free article] [PubMed] [Google Scholar]
50. Piyarulli D, Koolaee RM. A 22-Year-Old Female With Joint Pain. In: Piyarulli D, Koolaee RM, editors. Medicine Morning Report: Beyond the Pearls. Cambridge: Elsevier; 2016. pp. pp. 65–77. [Google Scholar]
51. Staheli LT. Lower extremity management. In: Staheli LT, Hall JG, Jaffe KM, Paholke DO, editors. Arthrogryposis: A Text Atlas. Cambridge: Cambridge University Press; 1998. pp. pp. 55–73. [Google Scholar]
52. Smolen JS, Aletaha D, Barton A, Burmester GR, Emery P, Firestein GS, et al. Rheumatoid arthritis. Nat Rev Dis Primers. 2018 Feb;4:18001. [PubMed] [Google Scholar]
53. Ong CK, Lirk P, Tan CH, Seymour RA. An evidence-based update on nonsteroidal anti- inflammatory drugs. Clin Med Res. 2007 Mar;5((1)):19–34. [PMC free article] [PubMed] [Google Scholar]
54. Combe B, Landewe R, Daien CI, Hua C, Aletaha D, Álvaro-Gracia JM, et al. 2016 updateof the EULAR recommendations for the management of early arthritis. Ann Rheum Dis. 2017 Jun;76((6)):948–59. [PubMed] [Google Scholar]
55. Liu D, Ahmet A, Ward L, Krishnamoorthy P,Mandelcorn ED, Leigh R, et al. A practical guide to the monitoring and management of the complications of systemic corticosteroid therapy. Allergy Asthma Clin Immunol. 2013 Aug;9((1)):30. [PMC free article] [PubMed] [Google
56. Whittle SL, Colebatch AN, Buchbinder R, Edwards CJ, Adams K, Englbrecht M, et al. Multinational evidence-based recommendations for pain management by pharmacotherapy in inflammatory arthritis: integrating systematic literature research and expert opinion of a broad panel of rheumatologists in the 3e Initiative. Rheumatology (Oxford) 2012 Aug;51((8)):1416–25.
57. Chitme RH, Patel PN. Antiarthritis activity of Aristolochia bracteata extract in experimental animals. Open Natl Pro J. 2009;2:6–15. [Google Scholar]
58. Tripathy S, Pradhan D, Anjana M. Anti-inflammatory and antiarthritic potential of Ammania baccifera Linn. Int J Pharm Bio Sci. 2010;1:1–7. [Google Scholar]
59. Chunxia C, Peng Z, Huifang P, Hanli R, Zehua H, Jizhou W. Extracts of Arisaema rhizomatum C.E.C. Fischer attenuate inflammatory response on collagen-induced arthritis in BALB/c mice. J Ethnopharmacol. 2011;133:573–82. [PubMed] [Google Scholar]
60. Babushetty V, Sultanpur MC. Evaluation of anti-arthritis activity of Asystasia dalzelliana leaves. Int J Pharma Biol Arch. 2012;3:377–82. [Google Scholar]
61. Kasper DL, Fauci AS, Longo DL, Braunwald E, Hauses SL, Jameson JL. 16th ed. II. United States of America: Mc-Graw Hill Companies; 2005. Harrison's Principle of Internal Medicine. [Google Scholar]
62. Ngoc DD, Catrina AI, Lundberg K, Harris HE, Ha NT, Anh PT, et al. Inhibition by Artocarpus tonkinensis of the development of collagen-induced arthritis in rats. Scand J Immunol. 2005;61:234–41. [PubMed] [Google Scholar]
63. Mazumder MP, Mondal A, Sasmal D, Arulmozhi S, Rathinavelusamy P. Evaluation of antiarthritic and immunomodulatory activity of Barleria lupulina. Asian Pac J Trop Biomed.
64. Rajkapoor B, Ravichandran V, Gobinath M, Anbu J, Harikrishnan N, Sumithra M, et al. Effect of Bauhinia variegate on complete freund's adjuvant induced arthritis in rats. J Pharmcol Toxicol. 2007;2:465–72. [Google Scholar]
65. Tandon V, Gupta RK. Histomorphological changes induced by Vitex negundo in albino rats. Indian J Pharmacol. 2004;36:176–7. [Google Scholar]
66. Vispute S, Khopade A. Glycyrrhiza glabra Linn.-—Klitaka‖: A review. Int J Pharm Bio Sci. 2011;2:42–51. [Google Scholar]
67. adami S, Moorkoth S, Suresh B. Caesalpinia sappan a medicinal and dye yielding plant. Nat Prod Rad. 2004;3:75–82. [Google Scholar]
68. Kalaria P, Gheewala P, Chakraborty M, Kamath J. A phytopharmacological review of Alstonia scholaris: A panoramic herbal medicine. IJRAP. 2012;3:367–371. [Google Scholar]
69. Sudha K, Mathanghi SK. Traditional underutilised green leafy vegetables and its curative properties. Int J Pharm. 2012;2:786–93. [Google Scholar]
70. Singh V, Patel H, Suvagiya V, Singh K. Some traditionally used anti-arthritic herbs a review. Int Res J Pharm. 2011;2:43–5. [Google Scholar]
71. Kiran D, Rohilla A, Rohilla S, Khan MU. Phyllanthus amarus: An ample therapeutic potential herb. Int J Res Ayur Pharm. 2011;2:1096–9. [Google Scholar]
72. Gohil K. J.; Patel J. A. A review on Bacopa monniera: Current research and future prospects. Int. J. Green Pharm. 2010, 4, 1–9. 10.4103/0973-8258.62156. [CrossRef] [Google Scholar]
73. Russo A.; Borrelli F. Bacopa monniera, a reputed nootropic plant: an overview. Phytomedicine. 2005, 12, 305–317. 10.1016/j.phymed.2003.12.008. [PubMed] [CrossRef] [Google Scholar]
74. Aguiar S.; Borowski T. Neuropharmacological review of the nootropic herb Bacopa monnieri. Rejuvenation res. 2013, 16, 313–326. 10.1089/rej.2013.1431. [PMC free article] [PubMed] [CrossRef] [Google Scholar]

75.Jeyasri R.; Muthuramalingam P.; Suba V.; Ramesh M.; Chen J. T. Bacopa monnieri and their bioactive compounds inferred multi-target treatment strategy forneurological diseases: a cheminformatics and system pharmacology approach. Biomolecules. 2020, 10, 536. 10.3390/biom10040536. [PMC free article] [PubMed] [CrossRef] [Google Scholar]
76. Brimson J. M.; Brimson S.; Prasanth M. I.; Thitilertdecha P.; Malar D. S.; Tencomnao T. The effectiveness of Bacopa monnieri (Linn.) Wettst. as a nootropic, neuroprotective, or antidepressant supplement: analysis of the available clinical data. Sci. rep. 2021, 11, 1–11.
77. Aggrey AA, Srivastava K, Ture S, Field DJ, Morrell CN. Platelet induction of the acute-phase response is protective in murine experimental cerebral malaria. *J Immunol.* 2013;190:4685–4691. [PMC free article] [PubMed] [Google Scholar]
78. Headland SE, Norling LV. The resolution of inflammation: Principles and challenges. *Semin Immunol.* 2015;27:149–160. [PubMed] [Google Scholar]
79. Reville K, Crean JK, Vivers S, Dransfield I, Godson C. Lipoxin A4 redistributes myosin IIA, Cdc42 in macrophages: implications for phagocytosis of apoptotic leukocytes. *Jof Immunol.* 2006;176:1878–1888. [PubMed] [Google Scholar]
80. Serhan CN, Savill J. Resolution of inflammation: the beginning programs the end. *Nat Immunol.* 2005;6:1191–1197. [PubMed] [Google Scholar]
81. Lintermans LL, Stegeman CA, Heeringa P, Abdulahad WH. T cells in vascular inflammatory diseases. *Front Immunol.* 2014;5:504. [PMC free article] [PubMed] [Google Scholar]
82. Sugimoto MA, Sousa LP, Pinho V, Perretti M, Teixeira MM. *Resolution of Inflammation: What Controls Its Onset? Front Immuno.* 2016 https://doi.org/10.3389/fimmu.2016.00.00160. [PMC free article] [PubMed]
83. He G, Karin M. NF-κB, STAT3 - key players in liver inflammation and cancer. *Cell Res.* 2011;21:159–168. [PMC free article] [PubMed] [Google Scholar]
84. Sofi F, Fabbri A, Casini A. *Mediterranean Diet.* Springer International publishing; 2016. Inflammation and Cardiovascular Disease and Protection by the Mediterranean Diet; pp. 89–96. [Google Scholar]
85. Khan N, Khymenets O, Urpísardà M, Tulipani S, Garciaaloy M, Monagas M, Moracubillos X, Llorach R, Andreslacueva C. Cocoa Polyphenols and Inflammatory Markers of Cardiovascular Disease. *Nutrients.* 2014;6:844–880. [PMC free article] [PubMed] [Google Scholar]
86. Lloydjones D, Adams RJ, Brown TM, Carnethon M, Dai S, Simone GD, Ferguson TB, Ford E, Furie K, Gillespie C. Heart Disease and Stroke Statistics—2010 Update A Report From the American Heart Association. *Circulation.* 2010;115:e25–e146. [Google Scholar]
87. Mathers CD, Loncar D. Projections of Global Mortality and Burden of Disease from 2002 to 2030. *Plos Med.* 2006;3:e442. [PMC free article] [PubMed] [Google Scholar]
88. Packard RRS, Peter L. Inflammation in atherosclerosis: from vascular biology to biomarker discovery and risk prediction. *Clin Chem.* 2008;54:24–38. [PubMed] [Google Scholar]
89. Libby P. History of Discovery: Inflammation in Atherosclerosis. *Arteriosclerosis Thrombosis Vascular Biology.* 2012;32:2045–2051. [PMC free article] [PubMed] [Google Scholar]
90. Libby P. Atherosclerosis in Inflammation. *Nature.* 2002;420:868–874. [PubMed] [Google Scholar]
91. Libby P, Okamoto Y, Rocha VZ, Folco E. Inflammation in atherosclerosis: transition from theory to practice. *Circulation J.* 2010;74:213–220. [PubMed] [Google Scholar]
92. Glezeva N, Baugh JA. Role of inflammation in the pathogenesis of heart failure with preserved ejection fraction and its potential as a therapeutic target. *Heart Fail Rev.* 2014;19:681–694. [PubMed] [Google Scholar]
93. Askevold ET, Gullestad L, Dahl CP, Yndestad A, Ueland T, Aukrust P. Interleukin-6 signaling, soluble glycoprotein 130, and inflammation in heart failure. *Curr Heart Fail Reports.* 2014;11:146–155. [PubMed] [Google Scholar]
94. Jennings RB, Murry CE, Steenbergen C, Jr, Reimer KA. Development of cell injury in sustained acute ischemia. *Circulation.* 1990;82:II2–12. [PubMed] [Google Scholar]
95. Pfeffer MA, Braunwald E. Ventricular remodeling after myocardial infarction. Experimental observations and clinical implications. *Circulation.* 1990;81:1161–1172. [PubMed] [Google Scholar]
96. Opie LH, Commerford PJ, Gersh BJ, Pfeffer MA. Controversies in ventricular remodelling. *Lancet.* 2006;367:356–367. [PubMed] [Google Scholar]
97. Frangogiannis NG. The immune system and cardiac repair. *Pharmacol Res.* 2008;58:88–111. [PMC free article] [PubMed] [Google Scholar]
98. Beg AA. Endogenous ligands of Toll-like receptors: implications for regulating inflammatory and immune responses. *Trends Immunol.* 2002;23:509–512. [PubMed] [Google Scholar]
99. Frantz S, Ertl G, Bauersachs J. Mechanisms of Disease: Toll-like receptors in cardiovascular disease. *Nat Clin Pract Card.* 2007;4:444–454. [PubMed] [Google Scholar]
100. Nijmeijer R, Lagrand WK, Visser CA, Meijer CJ, Niessen HW, Hack CE. CRP, a major culprit in complement-mediated tissue damage in acute myocardial infarction? *Int Immunopharmacol.* 2001;1:403–414. [PubMed] [Google Scholar]
101. Giordano FJ. Oxygen, oxidative stress, hypoxia, and heart failure. *J Clin Invest.* 2005;115:500–508. [PMC free article] [PubMed] [Google Scholar]
102. Griendling KK, Fitzgerald GA. Oxidative Stress and Cardiovascular Injury: Part I: Basic Mechanisms and In Vivo Monitoring of ROS. *Circulation.* 2003;108:1912–1916. [PubMed] [Google Scholar]
103. Hall G, Hasday JD, Rogers TB. Regulating the regulator: NF-κB signaling in heart. *J Mol Cell Cardiol.* 2006;41:580–591. [PubMed] [Google Scholar]
104. Gerard C, Rollins BJ. Chemokines and disease. *Nat Immunol.* 2001;2:108–115. [PubMed] [Google Scholar]
105. Moser B, Loetscher P. Lymphocyte traffic control by chemokines. *Nat Immunol.* 2001;2:123–128. [PubMed] [Google Scholar]
106. Kaur K, Dhingra S, Slezak J, Sharma AK, Bajaj A, Singal PK. Biology of TNFα and IL-10, and their imbalance in heart failure. *Heart Fail Rev.* 2009;14:113–123. [PubMed] [Google Scholar]
107. Frangogiannis NG. Targeting the transforming growth factor (TGF)-β cascade in the remodeling heart: Benefits and perils. *J Mol Cell Cardiol.* 2014;76:169–171. [PMC free article] [PubMed] [Google Scholar]
108. Booth GL, Kapral MK, Fung K, Tu JV. Relation between age and cardiovascular disease in men and women with diabetes compared with non-diabetic people: a population-based retrospective cohort study. *Lancet.* 2006;368:29–36. [PubMed] [Google Scholar]
109. Low Wang CC, Hess CN, Hiatt WR, Goldfine AB. Clinical Update: Cardiovascular Disease in Diabetes Mellitus: Atherosclerotic Cardiovascular Disease and Heart Failure in Type 2 Diabetes Mellitus - Mechanisms, Management, and Clinical Considerations. *Circulation.* 2016;133:2459–2502. [PMC free article] [PubMed] [Google Scholar]
110. Turner MD. The identification of TNFR5 as a therapeutic target in diabetes. *Taylor Francis.* 2017;21:349–351. [PubMed] [Google

Scholar]
111. Pradeep T, Haranath C. A Review on Diabetes Mellitus Type II. *Int J Pharma Res Rev.* 2014;3:23–29. [Google Scholar]
112. Larsen CM, Faulenbach M, Vaag A, Vølund A, Ehses JA, Seifert B, Mandrup-Poulsen T, Donath MY. Interleukin-1–receptor antagonist in type 2 diabetes mellitus. *New Engl J Med.* 2007;356:1517–1526. [PubMed] [Google Scholar]
113. Donath MY, Schumann DM, Faulenbach M, Ellingsgaard H, Perren A, Ehses JA. Islet inflammation in type 2 diabetes. *Diabetes care.* 2008;31:S161–S164. [PubMed] [Google Scholar]
114. Esser N, Legrand-Poels S, Piette J, Scheen AJ, Paquot N. Inflammation as a link between obesity, metabolic syndrome and type 2 diabetes. *Diabetes Res Clin Pr.* 2014;105:141–150. [PubMed] [Google Scholar]
115. Dinarello CA, Donath MY, Mandrup-Poulsen T. Role of IL-1β in type 2 diabetes. *Curr Opin Endocrinol.* 2010;17:314–321. [PubMed] [Google Scholar]
116. Donath MY, Shoelson SE. Type 2 diabetes as an inflammatory disease. *Nat Rev Immunol.* 2011;11:98–107. [PubMed] [Google Scholar]
117. Manohar M, Verma AK, Venkateshaiah SU, Sanders NL, Mishra A. Pathogenic mechanisms of pancreatitis. *World J Gastr Pharmacol Therapeut.* 2017;8:10–25. [PMC free article] [PubMed] [Google Scholar]
118. Yadav D, Lowenfels AB. The epidemiology of pancreatitis and pancreatic cancer. *Gastroenterology.* 2013;144:1252–1261. [PMC free article] [PubMed] [Google Scholar]
119. Zheng L, Xue J, Jaffee EM, Habtezion A. Role of immune cells and immune-based therapies in pancreatitis and pancreatic ductal adenocarcinoma. *Gastroenterology.* 2013;144:1230–1240. [PMC free article] [PubMed] [Google Scholar]
120. Zhang H, Neuhöfer P, Song L, Rabe B, Lesina M, Kurkowski MU, Treiber M, Wartmann T, Regnér S, Thorlacius H. IL-6 trans-signaling promotes pancreatitis-associated lung injury and lethality. *J Clin Invest.* 2013;123:1019–1031. [PMC free article] [PubMed] [Google Scholar]
121. Waddell N, Pajic M, Patch AM, Chang DK, Kassahn KS, Bailey P, Johns AL, Miller D, Nones K, Quek K. Whole genomes redefine the mutational landscape of pancreatic cancer. *Nature.* 2015;518:495–501. [PMC free article] [PubMed] [Google Scholar]
122. Ling S, Feng T, Jia K, Tian YU, Yan LI. Inflammation to cancer: The molecular biology in the pancreas (Review) *Oncol Lett.* 2014;7:1747–1754. [PMC free article] [PubMed] [Google Scholar]
123. Inflammation Whitcomb DC., Cancer V. Chronic pancreatitis and pancreatic cancer. *Am J Physiol-Gastr L.* 2004;287:G315–G319. [PubMed] [Google Scholar]
124. Li D, Xie K, Wolff R, Abbruzzese JL. Pancreatic cancer. *Lancet.* 2004;363:1049–1057. [PubMed] [Google Scholar]
125. Goggins M, Hruban RH, Kern SE. BRCA2 is inactivated late in the development of pancreatic intraepithelial neoplasia: evidence and implications. *Am J pathoL.* 2000;156:1767–1771. [PMC free article] [PubMed] [Google Scholar]
126. Shi Q, Le X Wang B, Abbruzzese JL, Xiong Q, He Y, Xie K. Regulation of vascular endothelial growth factor expression by acidosis in human cancer cells. *Oncogene.* 2001;20:3751–3756. [PubMed] [Google Scholar]
127. Xie K. Interleukin-8 and human cancer biology. *Cytokine Growth F R.* 2001;12:375–391. [PubMed] [Google Scholar]
128. Brenner C, Galluzzi L, Kepp O, Kroemer G. Decoding cell death signals in liver inflammation. *J Hepatol.* 2013;59:583–594. [PubMed] [Google Scholar]
129. Leitão HS, Doblas S, Garteiser P, D'Assignies G, Paradis V, Mouri F, Geraldes CF, Ronot M, Van Beers BE. Hepatic Fibrosis, Inflammation, and Steatosis: Influence on the MR Viscoelastic and Diffusion Parameters in Patients with Chronic Liver Disease. *Radiology.* 2016;283:98–1107. [PubMed] [Google Scholar]
130. Ramadori G, Moriconi F, Malik I, Dudas J. Physiology and pathophysiology of liver inflammation, damage and repair. *J Physiol Pharmacol.* 2008;59:107–117. [PubMed] [Google Scholar]
131. Pawlotsky JM. Pathophysiology of hepatitis C virus infection and related liver disease. *Trends in Microbiol.* 2004;12:96–102. [PubMed] [Google Scholar]
132. Dunn C, Brunetto M, Reynolds G, Christophides T, Kennedy PT, Lampertico P, Das A, Lopes AR, Borrow P, Williams K, Humphreys E, Afford S, Adams DH, et al. Cytokines induced during chronic hepatitis B virus infection promote a pathway for NK cell–mediated liver damage. *J Exp Med.* 2007;204:667–680. [PMC free article] [PubMed] [Google Scholar]
133. Gao B, Seki E, Brenner DA, Friedman S, Cohen JI, Nagy L, Szabo G, Zakhari S. Innate immunity in alcoholic liver disease. *Am J Physiol Gastrointest Liver Physiol.* 2011;300:516–525. [PMC free article] [PubMed] [Google Scholar]
134. Brenner DA, Seki E, Taura K, Kisseleva T, Deminicis S, Iwaisako K, Inokuchi S, Schnabl B, Oesterreicher CH, Yong HP. Non-alcoholic steatohepatitis-induced fibrosis: Toll-like receptors, reactive oxygen species and Jun N-terminal kinase. *Hepatol Res.* 2011;41:683–686. [PMC free article] [PubMed] [Google Scholar]
135. Maher JJ. DAMPs ramp up drug toxicity. *J Clin Invest.* 2009;119:246–249. [PMC free article] [PubMed] [Google Scholar]
136. Kubes P, Mehal WZ. Sterile Inflammation in the Liver. *Gastroenterology.* 2012;143:1158–1172. [PubMed] [Google Scholar]
137. Poltorak A, He X, Smirnova I, Liu MY, Huffel CV, Du X, Birdwell D, Alejos E, Silva M, Galanos C. Defective LPS Signaling in C3H/HeJ and C57BL/10ScCr Mice: Mutations in Tlr4 Gene. *science.* 1998;282:2085–2088. [PubMed] [Google Scholar]
138. Szabo G, Mandrekar P, Dolganiuc A. Innate immune response and hepatic inflammation. *Seminars in Liver Disease.* 2007;27:339–350. [PubMed] [Google Scholar]
139. Walford HH, Doherty TA. STAT6 and lung inflammation. *Jakstat.* 2013;2:e25301–e25311. [PMC free article] [PubMed] [Google Scholar]
140. Leitch AE, Duffin R, Haslett C, Rossi AG. Relevance of granulocyte apoptosis to resolution of inflammation at the respiratory mucosa. *Mucosal Immunol.* 2008;1:350–363. [PMC free article] [PubMed] [Google Scholar]
141. Brusselle G, Bracke K. Targeting immune pathways for therapy in asthma and chronic obstructive pulmonary disease. *Annals of the American Thoracic Society.* 2014;11:329–335. [PubMed] [Google Scholar]
142. Wong J, Magun BE, Wood LJ. Lung inflammation caused by inhaled toxicants: a review. *Int J of Copd.* 2016;11:1391–1401. [PMC free article] [PubMed] [Google Scholar]
143. Mroz RM, Noparlik J, Chyczewska E, Braszko JJ, Holownia A. Molecular basis of chronic inflammation in lung diseases: new therapeutic approach. *J Physiol Pharmacol.* 2007;58:453–460. [PubMed] [Google Scholar]
144. Kawayama T, Kinoshita T, Matsunaga K, Kobayashi A, Hayamizu T, Johnson M, Hoshino T. Responsiveness of blood and sputum inflammatory cells in Japanese COPD patients, non-COPD smoking controls, and non-COPD nonsmoking controls. *Int J Copd.* 2016;11:295–303. [PMC free article] [PubMed] [Google Scholar]
145. Ernandez T, Mayadas TN. The Changing Landscape of Renal Inflammation. *Trends Mol Med.* 2016;22:151–163. [PMC free article] [PubMed] [Google Scholar]
146. Poveda J, Sanz AB, Rayegomateos S, Ruizortega M, Carrasco S, Ortiz A, Sanchezniño MD. NFκBiz protein downregulation in acute kidney injury: Modulation of inflammation and survival in tubular cells. *BBA - Mol Basis Dis.* 2016;1862:635–646. [PubMed] [Google

Scholar]
147. Sanz A, Sanchez-Niño M, Ramos A, Moreno J, Santamaria B, Ruiz-Ortega M, Egido J, Ortiz A. NF-kappaB in renal inflammation. *J Am Soc Nephrol.* 2010;21:1254–1262. [PubMed] [Google Scholar]
148. Chawla LS, Kimmel PL. Acute kidney injury and chronic kidney disease: an integrated clinical syndrome. *Kidney Int.* 2012;82:516–524. [PubMed] [Google Scholar]
149. Sanchez MIP, Bercik P. Epidemiology and burden of chronic constipation. *Can J Gastroenterol.* 2011;25:11B–15B. [PMC free article] [PubMed] [Google Scholar]
150. Hunt R, Quigley E, Abbas Z, Eliakim A, Emmanuel A, Goh KL, Guarner F, Katelaris P, Smout A, Umar M. Coping with common gastrointestinal symptoms in the community: a global perspective on heartburn, constipation, bloating, and abdominal pain/discomfort May 2013. *J Clin Gastroenterol.* 2014;48:567–578. [PubMed] [Google Scholar]
151. Mcguckin MA, Eri R, Simms LA, Florin TH, Radford-Smith G. Intestinal barrier dysfunction in inflammatory bowel diseases. *Inflamm Bowel Dis.* 2009;15:100–113. [PubMed] [Google Scholar]
152. Cario E, Podolsky DK. Differential Alteration in Intestinal Epithelial Cell Expression of Toll-Like Receptor 3 (TLR3) and TLR4 in Inflammatory Bowel Disease. *Infection Immunity.* 2000;68:7010–7017. [PMC free article] [PubMed] [Google Scholar]
153. Strober W, Fuss I, Mannon P. The fundamental basis of inflammatory bowel disease. *J Clin Invest.* 2007;117:514–521. [PMC free article] [PubMed] [Google Scholar]
154. Fukata M, Arditi M. The role of pattern recognition receptors in intestinal inflammation. *Mucosal Immunol.* 2013;6:451–463. [PMC free article] [PubMed] [Google Scholar]
155. Thompson MR, Kaminski JJ, Kurt-Jones EA, Fitzgerald KA. Pattern Recognition Receptors and the Innate Immune Response to Viral Infection. *Viruses.* 2011;3:920–940. [PMC free article] [PubMed] [Google Scholar]
156. Goswami B, Rajappa M, Sharma M, Sharma A. Inflammation: its role and interplay in the development of cancer, with special focus on gynecological malignancies. *Int J Gynecol Cancer*. 2008;18:591–599. [PubMed] [Google Scholar]
157. Levi M, van der Poll T. Two-way interactions between inflammation and coagulation. *Trends Cardiovascular Med.* 2005;15:254–259. [PubMed] [Google Scholar]
158. Sampson MT, Kakkar AK. Coagulation proteases and human cancer. *Biochem Soc T.* 2002;30:201–207. [PubMed] [Google Scholar]
159. Nelson PT, Soma LA, Lavi E. Microglia in diseases of the central nervous system. *Annals Med.* 2002;34:491–500. [PubMed] [Google Scholar]
160. Vezzani A, Granata T. Brain Inflammation in Epilepsy: Experimental and Clinical Evidence. *Epilepsia.* 2005;46:1724–1743. [PubMed] [Google Scholar]
161. Block ML, Zecca L, Hong JS. Microglia-mediated neurotoxicity: uncovering the molecular mechanisms. *Nat Rev Neuro.* 2007;8:57–69. [PubMed] [Google Scholar]
162. Ekdahl CT, Claasen JH, Bonde S, Kokaia Z, Lindvall O. Inflammation is detrimental for neurogenesis in adult brain. *P Natl Acad of Sci USA.* 2003;100:13632–13637. [PMC free article] [PubMed] [Google Scholar]
163. Koltsakis GC, Stamatelos AM. Cytokines and acute neurodegeneration. *Nat Rev Neuro.* 2001;2:734–744. [Google Scholar]
164. Jankowsky JL, Patterson PH. The role of cytokines and growth factors in seizures and their sequelae. *Prog Neurobio.* 2001;63:125–149. [PubMed] [Google Scholar]