

HERBAL DRUGS AS SAFE ANTI-INFLAMMATORY AGENTS

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Summary

Inflammation (Latin, *inflammare*, to set on fire) is part of the complex biological response of vascular tissues to harmful stimuli, such as pathogens, damaged cells, or irritants.^[1] Inflammation is a protective attempt by the organism to remove the injurious stimuli and to initiate the healing process. **Inflammation** can be classified as either *acute* or *chronic*. *Acute inflammation* is the initial response of the body to harmful stimuli and is achieved by the increased movement of plasma and leukocytes (especially granulocytes) from the blood into the injured tissues. There were 24 plants found to be effective in inflammation (eg. *Fennel Seed*, *Aloe Vera*, *Angel*) Present work was undertaken to revive anti-inflammatory potential of the plants.

Keywords: .Inflammation, Plasmin, Thrombin, *Fennel Seed*, *Aloe Vera*, *Angelica*

Introduction

Inflammation is protective mechanism of body. Without inflammation, wounds and infections would never heal. Similarly, progressive destruction of the tissue would compromise the survival of the organism. However, chronic inflammation can also lead to a host of diseases, such as hay fever, atherosclerosis, and rheumatoid arthritis. It is for that reason that inflammation is normally closely regulated by the body

Cause

- Burns
- Chemical irritants
- Frostbite
- Toxins
- Infection by pathogens
- Physical injury, blunt or penetrating
- Immune reactions due to hypersensitivity
- Ionizing radiation
- Foreign bodies, including splinters, dirt and debris

Cardinal signs



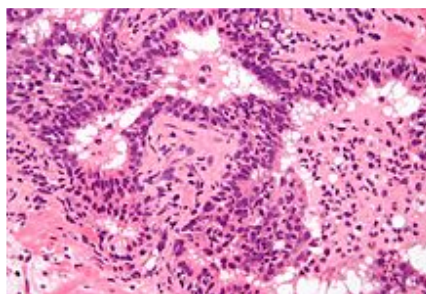
Infected ingrown toenail showing the characteristic redness and swelling associated with acute inflammation. Acute inflammation is a short-term process, usually appearing within a few minutes or hours and ceasing upon the removal of the injurious stimulus.^[4] It is characterized by five cardinal signs⁵

Types

	Acute	Chronic
Causative agent	Pathogens, injured tissues	Persistent acute inflammation due to non-degradable pathogens, persistent foreign bodies, or autoimmune reactions
Major cells involved	Neutrophils, mononuclear cells (monocytes, macrophages)	Mononuclear cells (monocytes, macrophages, lymphocytes, plasma cells), fibroblasts
Primary mediators	Vasoactive amines, eicosanoids	IFN- γ and other cytokines, growth factors, reactive oxygen species, hydrolytic enzymes
Onset	Immediate	Delayed

Duration	Few days	Up to many months, or years
Outcomes	Resolution, abscess formation, chronic inflammation	Tissue destruction, fibrosis

Process of acute inflammation



Micrograph showing acute inflammation of the prostate gland with the characteristic neutrophilic infiltrate. H&E stain.

The process of acute inflammation is initiated by cells already present in all tissues, mainly resident macrophages, dendritic cells, histiocytes, Kupffer cells and mastocytes. At the onset of an infection, burn, or other injuries, these cells undergo activation and release inflammatory mediators responsible for the clinical signs of inflammation. Vasodilation and its resulting increased blood flow causes the redness (*rubor*) and increased heat (*calor*). Increased permeability of the blood vessels results in an exudation (leakage) of plasma proteins and fluid into the tissue (edema), which manifests itself as swelling (*tumor*). Some of the released mediators such as bradykinin increase the sensitivity to pain (hyperalgesia, *dolor*). The mediator molecules also alter the blood vessels to permit the migration of leukocytes, mainly neutrophils, outside of the blood vessels (extravasation) into the tissue. The neutrophils migrate along a chemotactic gradient created by the local cells to reach the site of injury.^[4] The loss of function (*functio laesa*) is probably the result of a neurological reflex in response to pain.

Examples

Inflammation is usually indicated by adding the suffix "-itis", as shown below. However, some conditions such as asthma and pneumonia do not follow this convention. More examples re available at list of types of inflammation.



Acute dermatitis



Acute tonsillitis

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Sr. No.	Name Of Drugs	Family	Extractive Solvent	Uses
1.	Aloe Vera	<i>Aloaceae/ Liliaceae.</i>	Hexane, Petroleum Ether.	Anti-inflammatory, Purgative, Protective.
2.	Angelica	<i>Apiaceae.</i>	Chloroform.	Anti-inflammatory, Respiratory Ailment.
3.	Arnika	<i>Asteraceae</i>	Ethanol.	Anti-inflammatory, Rheumatism, Blindness, Spinary Paralysis.
4.	Arjuna	Combreta-ceae	Water, Ethanol	Anti-inflammatory Cardiotonic, Anti-dyssentric.
5.	Bilberry	Ericaceae.	Alcohol.	Anti-inflammatory, Anti-arthritic.
6.	Boswellia	Burseraceae	Methanol.	Anti-inflammatory, Anti-arthritic, Rheumatoid Arthritis.

7.	Clove	<i>Myrtaceae.</i>	Water.	Anti-inflammatory, Antiseptic, Dental Analgesic.
8.	Eucalyptus	<i>Myrtaceae.</i>	Ethyl Ether.	Anti-inflammatory. Bronchitis, Expectorant.
9.	Fenugreek	<i>Fabaceae.</i>	Acetone, Alcohol, Methylene Chloride.	Anti-inflammatory, Antiviral, Common Cold.
10.	Fennel Seed	<i>Umbelliferae.</i>	Alcohol.	Anti-inflammatory, Carminative, Expectorant.
12.	Guggul	<i>Burseraceae.</i>	Ethyl Acetate.	Anti-inflammatory, Anti-arthritic, Hypolipidemic.
13.	Liquorice/ Licorice.	<i>Fabaceae.</i>	Ethanol.	Anti-inflammatory, Expectorant, Peptic Ulcer.
14.	Mustard.	<i>Brassicaceae.</i>	Ethanol.	Anti-inflammatory, Counter Irritant, Rubifacient.
15.	Myrrh.	<i>Burseraceae.</i>	Alcohol.	Anti-inflammatory, Antiseptic, Astringent.

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Sr. No.	Name Of Drugs	Family	Extractive Solvent	Uses
17.	Pineapple	<i>Bromeliaceae.</i>	Chloroform, Benzene, Methylene Chloride.	Anti-inflammatory, Oedema.
18.	Rosemary	<i>Lamiaceae.</i>	Chloroform, Petroleum Ether, Hexane.	Anti-inflammatory, Carminative, Rubifacient, Flavour.
19.	<i>Stinging Nettle</i>	Urticaceae.	Ethanol.	Arthritis, Rheumatism, Rubifacient.
20.	<i>Seasame</i>	Pediliaceae.	Distilled Water.	Anti-inflammatory, Demulcent, Emollient, Laxative.
21.	<i>Turmeric</i>	Zingiberaceae.	Ethanol.	Anti-arthritic, Anti-inflammatory, Cosmetic.
23.	<i>Willow Bark</i>	Salicaceae.	Alcohol.	Anti-inflammatory, Rheumatism.
24.	<i>Yucca</i>	Agavaceae.	Ethyl Ether.	Anti-inflammatory, Rheumatism, Arthritis.

Conclusion

Inflammation is a protective attempt by the organism to remove the injurious stimuli and to initiate the healing process. Inflammation can be classified as either *acute* or *chronic*. By using herbal extracts of above herbs it may be minimized signs and symptoms of acute as well as chronic inflammation. Above drugs & there corresponding extracts were found to be effective against inflammation. NSAIDS are present antiinflammatory agents which are the drugs for all type of inflammation NSAIDS are going to cause renal failure, acute hepatotoxicity and GIT ulceration apendetitis hence innear feature it is sort of exploring the herbal and medicinal plant for all the types of inflammation this will reduce the risk and toxicity of NSAIDS and it can work effectively as an altenative safer antiinflammatory agents to mankind.

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References

1. Ferrero-Miliani L, Nielsen OH, Andersen PS, Girardin SE (February 2007). "Chronic inflammation: importance of NOD2 and NALP3 in interleukin-1beta generation". *Clin. Exp. Immunol.*
2. Stedman's Medical Dictionary, Twenty-fifth Edition, Williams & Wilkins, 1990.
3. Disturbance of function (functio laesa): the legendary fifth cardinal sign of inflammation, added by Galen to the four cardinal signs of Celsus. *Bull N Y Acad Med.* 1971 March; 47(3): 303–322
4. Cotran; Kumar, Collins (1998). *Robbins Pathologic Basis of Disease*. Philadelphia: W.B Saunders Company.
5. Parakrama Chandrasoma, Clive R. Taylor (ca. 2005). "Part A. *General Pathology*, Section II. *The Host Response to Injury*, Chapter 3. *The Acute Inflammatory Response*, sub-section **Cardinal Clinical Signs**". *Concise Pathology* (3rd edition (Computer file) ed.). New York, N.Y.: McGraw-Hill.
6. A Massage Therapist Guide to Pathology Ruth Werner (2009). *A massage Therapist Guide to Pathology* (4th ed.). Philadelphia, PA and Baltimore, MD.
7. Wolfgang H. Vogel, Andreas Berke (2009). "*Brief History of Vision and Ocular Medicine*". Kugler Publications. p.97.
8. Porth, Carol (2007). *Essentials of pahtophysiology: concepts of altered health states*. Hagerstown, MD: Lippincott Williams & Wilkins. pp. 270.
9. Dormandy, Thomas (2006). *The worst of evils: man's fight against pain*. New Haven, Conn: Yale University Press. pp. 22
10. Wiedermann U, et al. (1996). "Vitamin A deficiency increases inflammatory responses.". *Scand J Immunol.* **44** (6):

11. Coussens LM, Werb Z (2002). "Inflammation and cancer". *Nature* **420** ^ Eming, S.A., T. Krieg, and J.M. Davidson, Inflammation in wound repair: molecular and cellular mechanisms. *J Invest Dermatol*, 2007. 127(3): p. 514-25.
12. Ashcroft, G.S., et al., Mice lacking Smad3 show accelerated wound healing and an impaired local inflammatory response. *Nat Cell Biol*, 1999. 1(5): p. 260-6.
13. Ashcroft, G.S., Bidirectional regulation of macrophage function by TGF-beta. *Microbes Infect*, 1999. 1(15): p. 1275-82.
14. Werner, F., et al., Transforming growth factor-beta 1 inhibition of macrophage activation is mediated via Smad3. *J Biol Chem*, 2000. 275(47): p. 36653-8.
15. Sato, Y., T. Ohshima, and T. Kondo, Regulatory role of endogenous interleukin-10 in cutaneous inflammatory response of murine wound healing. *Biochem Biophys Res Commun*, 1999. 265(1): p. 194-9.
16. Serhan, C.N., Controlling the resolution of acute inflammation: a new genus of dual anti-inflammatory and proresolving mediators. *J Periodontol*, 2008. 79(8 Suppl): p. 1520-6.
17. Greenhalgh, D.G., The role of apoptosis in wound healing. *Int J Biochem Cell Biol*, 1998. 30(9): p. 1019-30.
18. Jiang, D., et al., Regulation of lung injury and repair by Toll-like receptors and hyaluronan. *Nat Med*, 2005. 11(11): p. 1173-9.
19. Teder, P., et al., Resolution of lung inflammation by CD44. *Science*, 2002. 296(5565): p. 155-8.
20. McQuibban, G.A., et al., Inflammation dampened by gelatinase A cleavage of monocyte chemoattractant protein-3. *Science*, 2000. 289(5482): p. 1202-6.
21. Serhan CN, Savill J (2005). "Resolution of inflammation: the beginning programs the end". *Nat. Immunol.*
22. Bastard J et al. (2000). "Elevated levels of interleukin 6 are reduced in serum and subcutaneous adipose tissue of obese women after weight loss". *J Clin Endocrinol Metab* **85** (9): 3338-42.