Herbals as Source of Anti-inflammatory and Wound Healing Drugs : A Review

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Abstract

Inflammatory disease etiopathology is rising worldwide and caused due to processes such as tissue injury, cell death, cancer, ischemia and degeneration. The synthetic drugs used as anti-inflammatory agents are having deleterious side effects besides being costly and rarely available at all places. Natural product derived from medicinal plant for thousands of years are now yield their secrets and finding important role in development of modern medicines. Research and development of anti-inflammatory medicinal agents have proven beneficial. From effectiveness to low cost, ease of availability and less or no side effects, all aspects are proving beneficial. Exploration of novel methods of extraction and investigation of phytoconstituents from these medicinal plants are undergoing for better evaluation of active principles. Medicinal herbs are gaining importance for the prevention and treatment of inflammatory diseases. *In vitro* and *in vivo* evaluation of anti-inflammatory and wound healing potential of medicinal plants in experimental and clinical cases are being explored for proper prophylactic and therapeutic evaluation. Promising results have been obtained by the application of medicinal plants in wound healing and inflammatory diseases. Hence many patents have been granted in this field. However, evaluation at the molecular level of both phytoconstituents of medicinal plants and their physiological and pharmacological activities and mechanisms of action and their role as wound healing and anti-inflammatory agents in *in-vivo* studies need to be effectively explored before their application in clinical studies using advanced technologies and study designs.

Keywords: Antioxidant, anti-inflammatory, edema, inflammation, wound healing.

Introduction

Campos *et al*., 2014, the term inflammation is derived from Latin word - “inflamers” / “inflammatio” (to burn) is part of the complex biological response / defense of an organism (natural aspect of the immune system's response; sequence of events that occur in response to noxious stimuli, infection or trauma (Shih *et al*., 2007) against local injury, infections, acute / chronic inflammation (rheumatoid arthritis, asthma, colitis, hepatitis, cancer, cardiovascular and neurodegenerative disorders) / biological response to harmful stimuli. Azwanida *et al*., 2015, in Inflammation (body's severe reaction to any damage; ubiquitous process which induced homeostasis such as damage, exposure to contaminants / infection and also triggered by innate

immune system receptors), pain, redness, heat or warmth, and swelling are the four primary indicators of swelling and in injury arterioles in the local tissue dilate and results in increased blood flow to the affected area (redness). uncontrolled inflammatory response is the main cause of a vast continuum of disorders including allergies, cardiovascular dysfunctions, metabolic syndrome, cancer, and autoimmune diseases. Bauri *et al*., 2015, inflammatory diseases are major cause of morbidity in today's era of modern lifestyle across the globe (etiology or pathology are having an increase in incidence around the globe).

Inflammatory Process

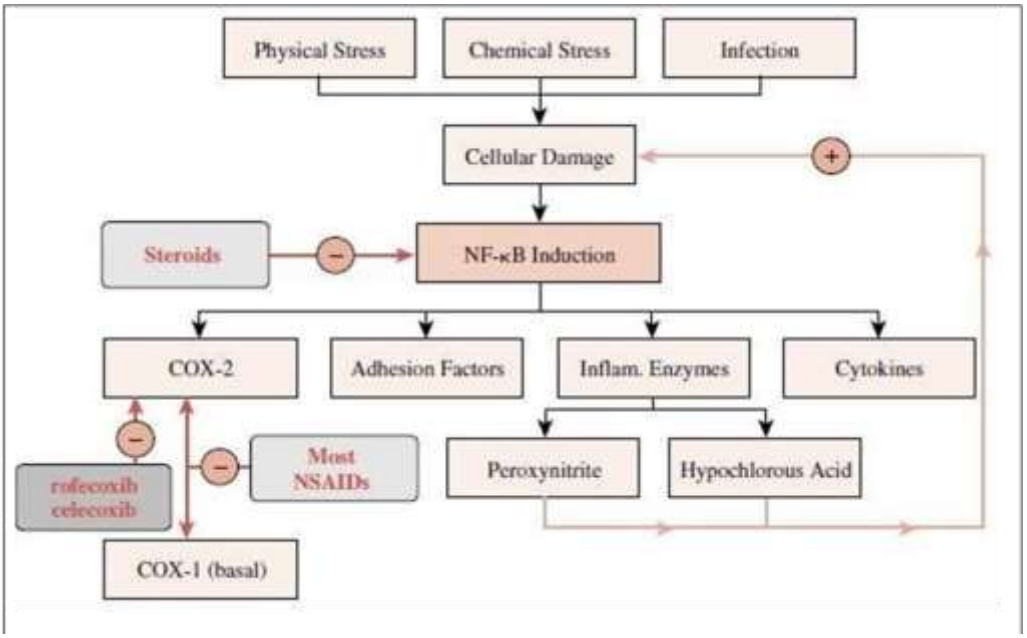

Fig. 1 : Overview of Inflammatory Processes.

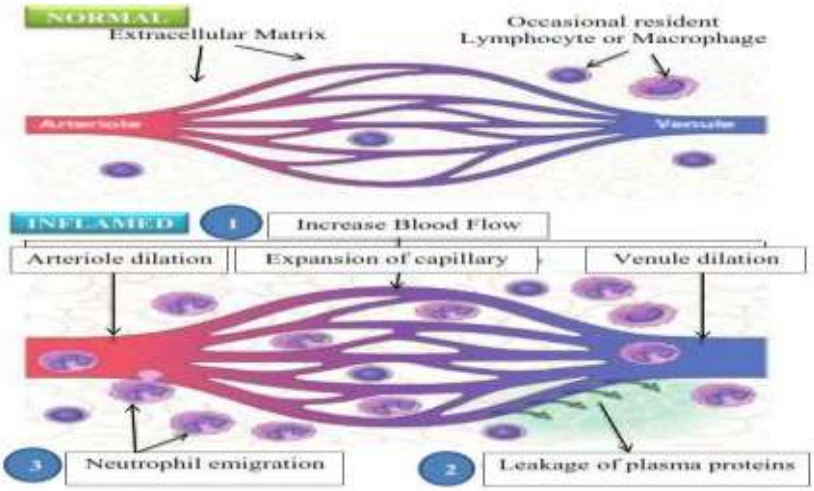

Fig. 2 : Inflammatory Process.

Types of Inflammation

Parks *et al*., 2004, there are two basic types of inflammation – acute and chronic. Acute inflammation is of short duration while chronic inflammation is long lasting. Pilotto *et al*., 2010, inflammation is either acute or chronic. Acute inflammation is an initial response of the body to harmful stimuli. Parhnam *et al*.,

2008, in chronic inflammation, the response resulting in damage to the body (out of proportion; rheumatoid arthritis, asthma, colitis, allergies, hepatitis, metabolic syndrome, autoimmune diseases cancer, cardiovascular dysfunctions and neurodegenerative disorders).

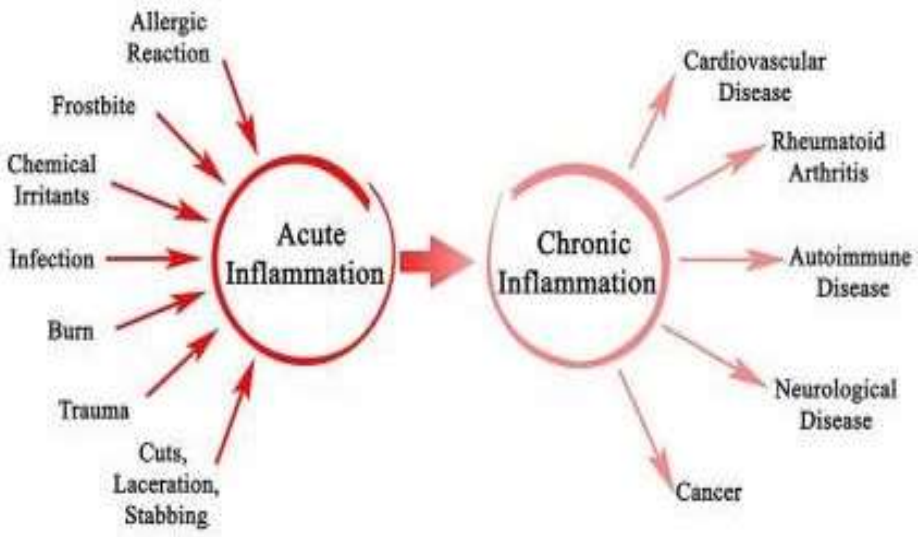

Fig. 3 : Types of Inflammation.

Cause and Consequences of Inflammation

Azwanida *et al*., 2015, Inflammation is sequence of events that occur in response to noxious stimuli, infection or trauma (disturb homeostasis) and severe cause of morbidity (etiology or pathology).

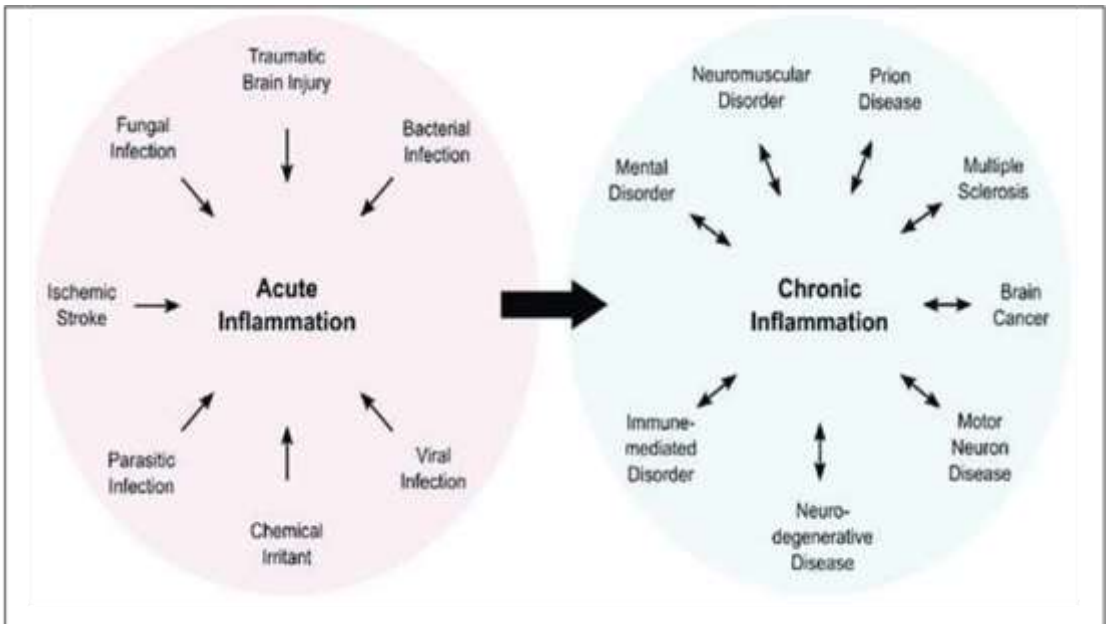

Fig. 4 : Cause and Consequences of Inflammation.

Causes of Inflammation

- Living organisms: ex. bacteria, viruses, fungi, protozoa & metazoa
- Chemicals: ex. Turpentine
- Mechanical & Thermal injuries: ex. burns, electricity, radiations
- Immune reaction: causes inflammation due to Ag-Ab reaction

Changes in inflammations

Blood Vessels Changes

(a) Momentary contraction of the B.V.

(b) Vessels dilation: causing more arterial blood i.e. hyperemia its mechanism: relaxation of arteriolar smooth muscle wall & precapillary sphincters.

Causes of the Dilation

(a) Axon reflex.

(b) Chemical mediators: are most important with a significant role, Increased permeability of venules & Capillaries: Its effect is leakage of plasma protiens, RBCs & WBCs.

Changes in Blood Stream

(a) Erythrocyte distribution

(b) Leukocytes margination (pavement); Its mechanisms either:-

1- WBCs get adhesive

2- Capillary. Wall gets sticky (its endothelium gets sticky)

Changes in Rate of Flow

(a) Accelerate of the rate: due to arteriolar dilation,

(b) Retardation due to : Fluid loss leads to increased blood viscosity, Leukocytes stickiness; Stasis: well established congestion that favors, molecular & cellular rescape i.e. exudation.

Leukocyticemigration

- Mechanism: ameboid movement

- Cause: chemotactic forces (Chemotaxis: process of attraction of leukocytes to certain area that has the chemotactics substances)
- Chemotactic substances are : Products from pathogenic bacteria; Substances from injured - cells ex. mechanical or thermal injuries; Chemicals ex. Turpentine; Serum complements ex. Lysates of neutrophilst.

Diapedesis of RBCs.

Serum exudation (Ismaeloudahamer *et al*., 2008)

- Large protein molecules is cells of inflammation (phagocytic cells);

Function of the exudates

- Toxic substances formed within the body especially bee-stings & snake-bite.
- Brings with it antibodies i.e. it brings immunity against specific infections.
- Brings leukocytes to the area for phagocytosis.
- Fibrinogen forms fibrin (support ameboid movement of leukocytes)
- Has mechanical action by washing the irritant.

Function of the Chemical Mediator

- Vasodilation of arteriol & venules.
- Increase permeability of venule& capillaries
- Induce smooth muscles stimulation; induce pain.
- Induce Emigration of lenkocytes.

Inflammatory cells

- Granulocytes Ex:-Nutrophils, Basophils, Eosinophils.
- Agranulocytes Ex: -Macrophage, plasma cells, lymphocyte, epithelial giant cell.

Neutrophils : The action of neutrophils is phagosytic, its cells in blood their phagocytic power is shown towardbacteria rather than to dead .them is killed by bacterial toxins. It will produce pus & this process call suppuration or purulent exudate.

Eosinophils : This cells present in the parasitic infection and hyper sensitivity due to release of eosinophils chemotactic factor from mast cells when intact with Ag+lge to mast cells & release of histamin.

Monocytes: It is phagocytes cells inside the blood & when reaches to the cells and tissue it will became macrophage cells or called histiocytes. The function of macrophage is phagocytes foreign body and removed (scavengers) the debris.

Epithelioid Cells: It's similar to macrophage and similar o epithelial cells close to each other with no different borders between it's a cytoplasm & have small neucleus. These cells are no phagocytic cells but release lysosomal enzyme.

Giant Cells : Form by fused the cytoplasm of the macrophages (2 – 3 or reach to 20). There are 2 types of giant cells:

1. Langhan's giant cells- Where their nuclei which be located at the periphery
2. Foreign body giant cells - In which their nuclei arranged through it (through its cytoplasm).

Signs of Inflammation

Parhnam *et al*., 2008, signs of inflammation include redness (local), swelling, pain, heat and loss of function (chemical mediators, including kinins, eicosanoids, complement proteins, histamine and monokines induce and regulate these manifestations).

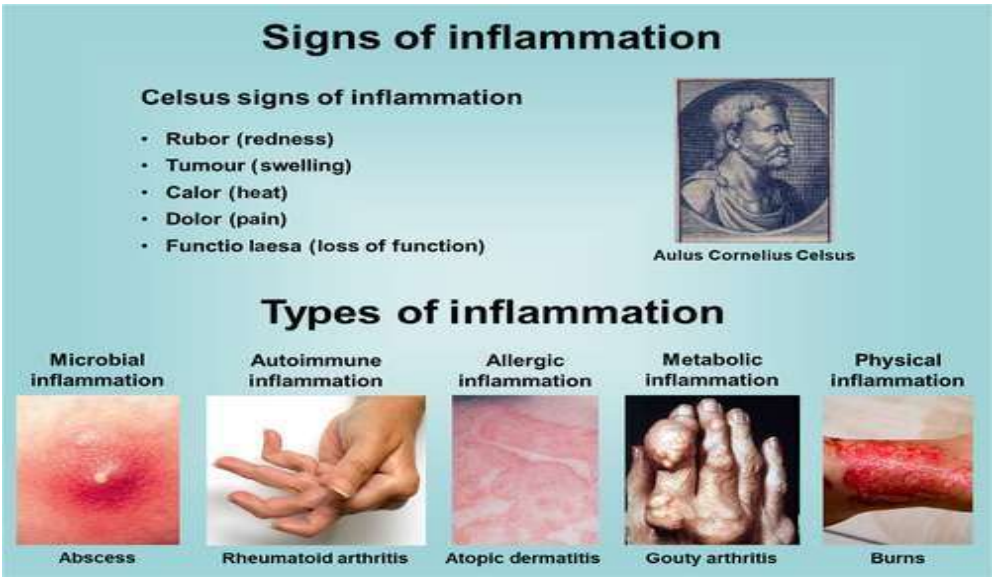

Fig. 5 : Cause and Consequences of Inflammation

Pathophysiology of Inflammation

Iwalewa *et al*., 2007, cyclooxygenase (COX) is a major enzyme involved in the production of prostacyclins, prostaglandins and thromboxanes which play a role in inflammation, pain and platelet aggregation.

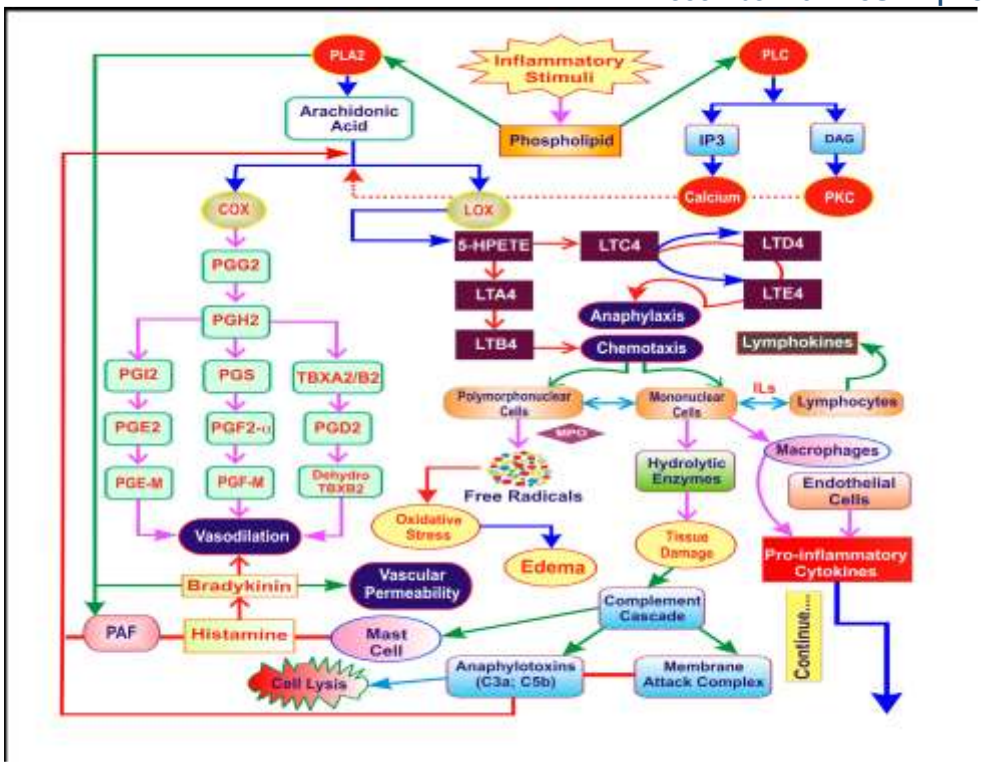

Fig. 1.6a: The inflammatory cascade A.

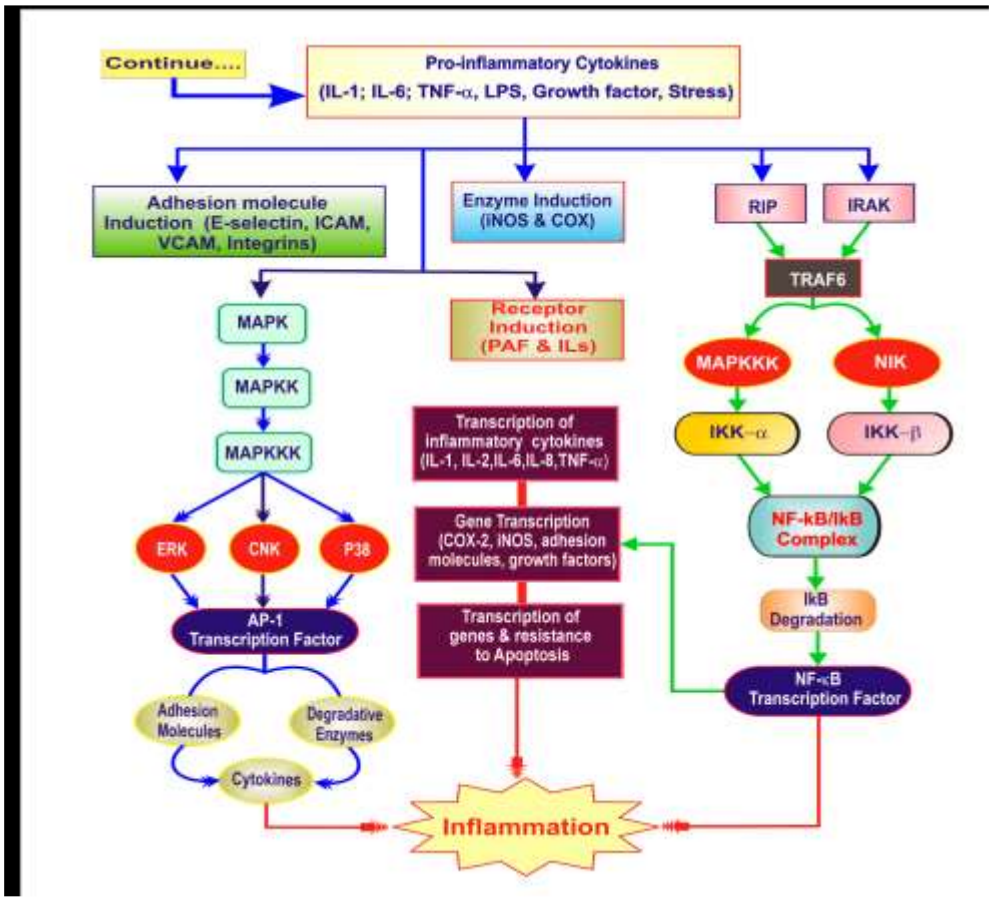

Fig. 1.6b: The inflammatory cascade B.

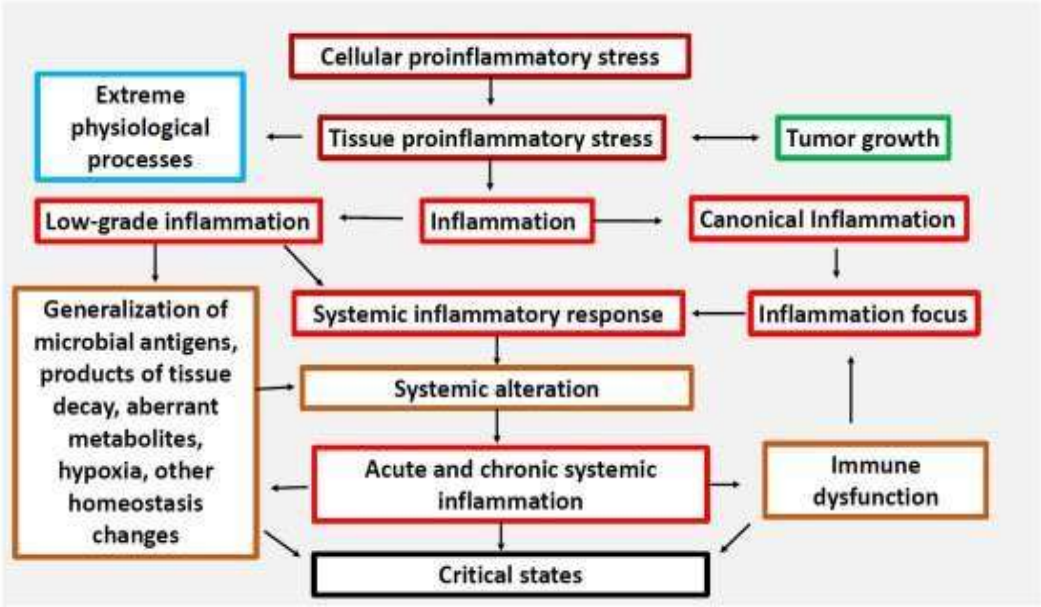

Fig. 7: Pathophysiology of Inflammation.

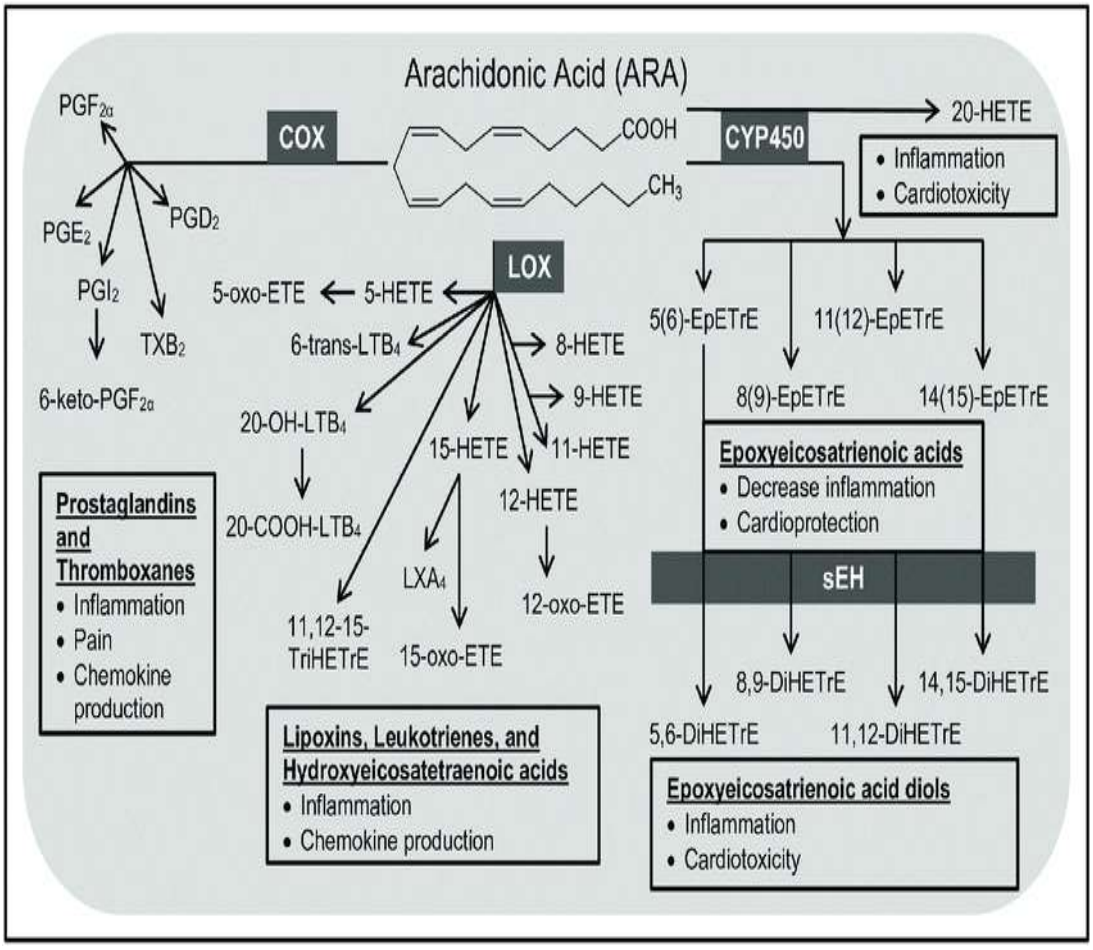

Fig.8: Pathophysiology of Inflammation and Arachidonic acid Pathway.

***In-vivo* Methods for the Evaluation of Anti-inflammatory Potential**

Table 1: Evaluation methods for anti-inflammatory potential of drugs.

S.No.	Test	Reference
1	Evaluation of analgesic and / or anti-inflammatory activity by formalin test	Hunskaar *et al*., 1986;
2	Carrageenan-induced peritonitis / Carrageenan-Induced Paw Edema	Batista *et al*., 2016
3	Oxazolone-Induced Ear Edema	
4	Croton oil-induced ear edema in mice / Croton Oil/TPA-Induced Ear Edema	Anuario *et al*., 2018;
5	Pleurisy / Pleurisy Tests	Da Silva *et al*., 2018;
6	Acetic Acid/ Compound 48/80-Induced Vascular Permeability / Acetic acid-induced writhing tests	Koster *et al*., 1959;
7	Histamine / 5-HT Induced Paw Edema	Al-Haboubi *et al*., 1983
8.	Bradykinin-induced Paw Edema	Katz *et al*., 1984;
9	Dextran-induced Edema	Coura *et al*., 2015
10	Lipopolysaccharide-Induced Paw Edema	Calil *et al*., 2014;
11	Arachidonic Acid-Induced Ear Edema	Moreno *et al*., 1993;

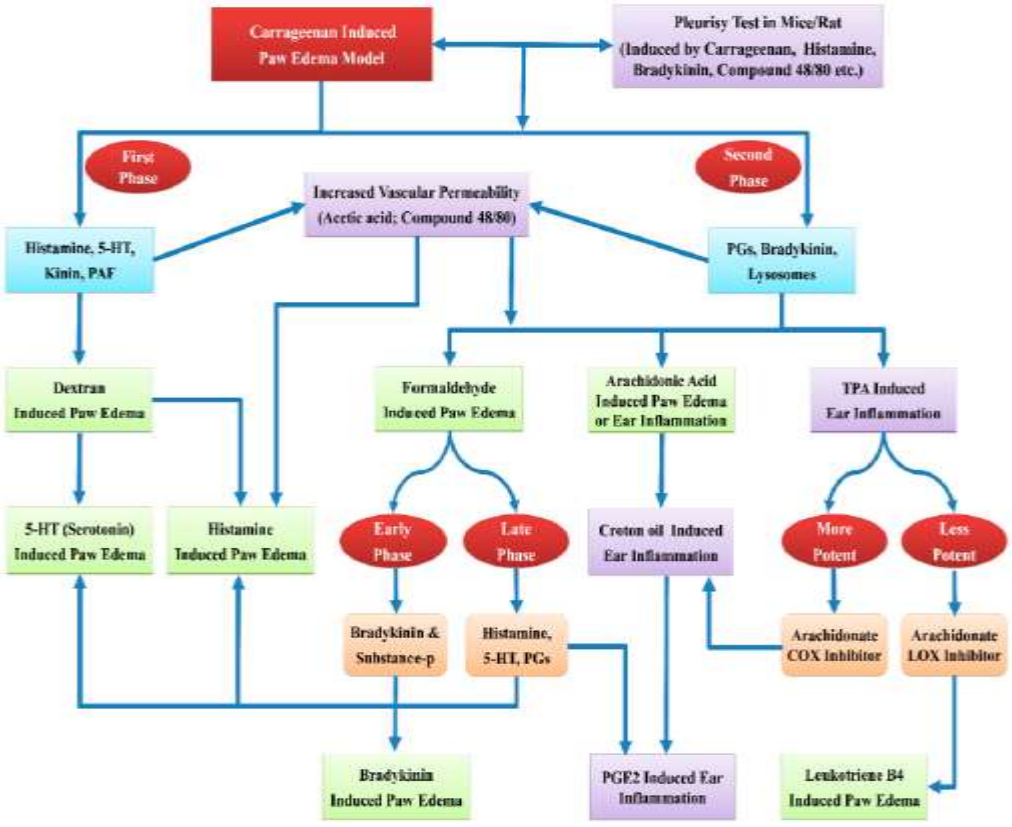

Fig. 9: Schematic evaluation of anti-inflammatory activity.

Anti-Inflammatory Agents

Non-steroidal anti-inflammatory drugs

Virshette *et al*., 2019, non-steroidal anti-inflammatory drugs (NSAIDs) are used to treat inflammation by inhibition of COX-1 and COX-2 which stop accumulation of prostaglandins and thromboxanes. Wallace

(2001), NSAIDs in prolonged therapy causes deleterious side effects such as gastric lesions, cardiovascular, renal and gastrointestinal damage. Percival (1999), disadvantage of NSAIDs is their toxicity and reappearance of symptoms after discontinuation. So, screening and development new anti-inflammatory drugs are the need of hour and efforts are made to find anti-inflammatory drugs from medicinal plants.

Anti-inflammatory Drugs

NSAIDs reduce mainly those components of the inflammatory and immune response in which prostaglandins, mainly derived from COX-2, play a significant part. These include:

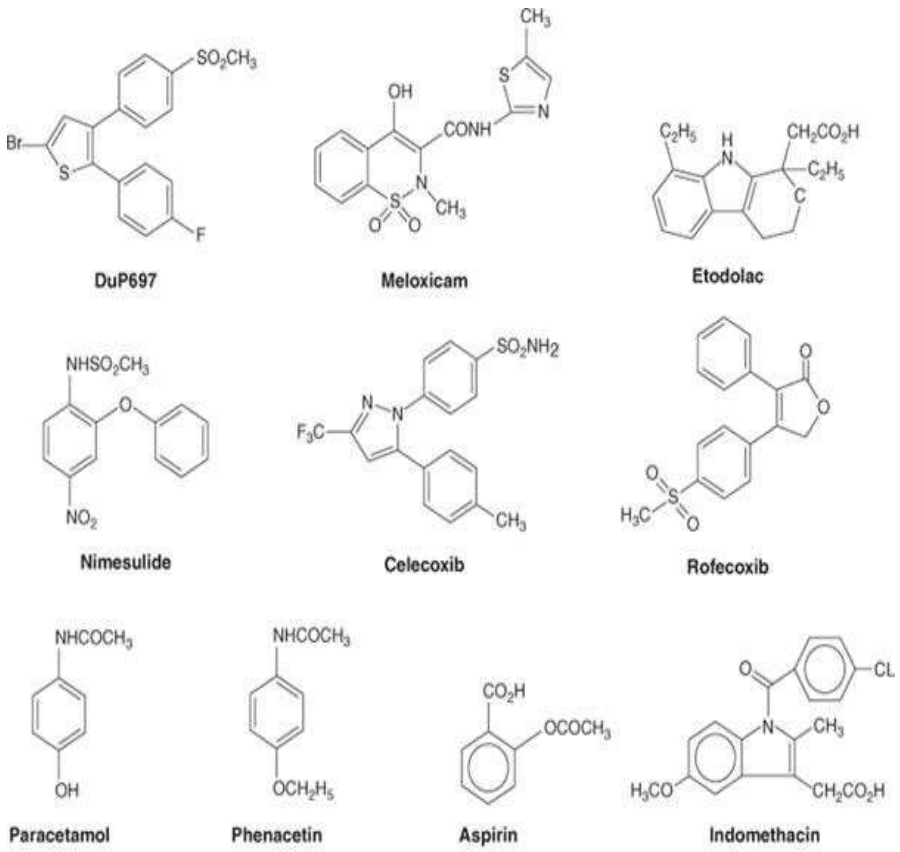

Fig. 10 : Non steroidal anti inflammatory drugs.

Medicinal Plants As Anti-inflammatory Agents (Mahesh *et al*., 2004)

Newman (2020), more than 80% of medicines have been developed from natural products (NP) obtained from natural source. It was found that major approved drugs are either natural products or derived from NPs or pharmacophore from a NP. Srinivasan *et al*., 2001, Medicinal plants have been a source of wide variety of biologically active compounds for many centuries and use extensively as crude material or as pure compounds for treating various disease conditions. Laloo and Hemalatha (2011), India with its biggest repository of medicinal plants in the world may maintain an important position in the production of raw materials either directly for crude drugs or as bioactive compounds in the formulation of pharmaceuticals and cosmetics etc.

Goel *et al*., 2020, Traditional herbs and their preparations for example were considered drugs in the Ayurvedic system of medicine; the "Sushruta Samhita" (an Ayurvedic classic) contains roughly 700 plants for the treatment of 1100 ailments. Numerous traditional medical systems (Chinese materiamedica, Greek, Arab, Egyptian and Mesopotamian) as well as folk medicine (ethnomedicine) provided a vast amount of information. Serturner's (1804) separation of morphine from opium marked the beginning of modern NP chemistry. Many such findings led to the discovery of bioactive isolated chemicals such as quinine (1820) from cinchona bark, strychnine (1818), cocaine (1859), tubocurarine (1935), penicillin and other bioactive isolated compounds.

Goel *et al*., 2020, Natural products (NPs) are substance found in the cosmos (animals, plants or microbes). NPs anti inflammatory activity have long been use as a traditional remedy for inflammatory conditions (low cost, easy available and less or no side effects). Alexandrina (2010), Medicinal plants play an important role in the development of potent therapeutic agents. There are over 1.5 million practitioners of traditional medicinal system using medicinal plants in preventive, promotional and curative applications.

Madhuri & Pandey (2009), mechanisms of action include modulation of detoxification enzymes, scavenging of oxidative agents, stimulation of the immune system, regulation of gene expression in cell proliferation and apoptosis, hormone metabolism, antibacterial and antiviral effects.

Soeken *et al*., 2003, NPs from steroids, flavonoids, alkaloids, polyphenols, glycosides, terpenoids, curcumins, GLA, phenolic linear aliphatic alcohols (e.g., tetracosanol), and diterpenes have anti-inflammatory properties. Vashishtha *et al*., 2014, Natural products with anti inflammatory activity have long been use as a folk remedy for inflammatory conditions such as fever, pain, migraine and arthritis.As the inflammatory basis of disease becomes clear, anti-inflammatory food and food products become of greater interest.

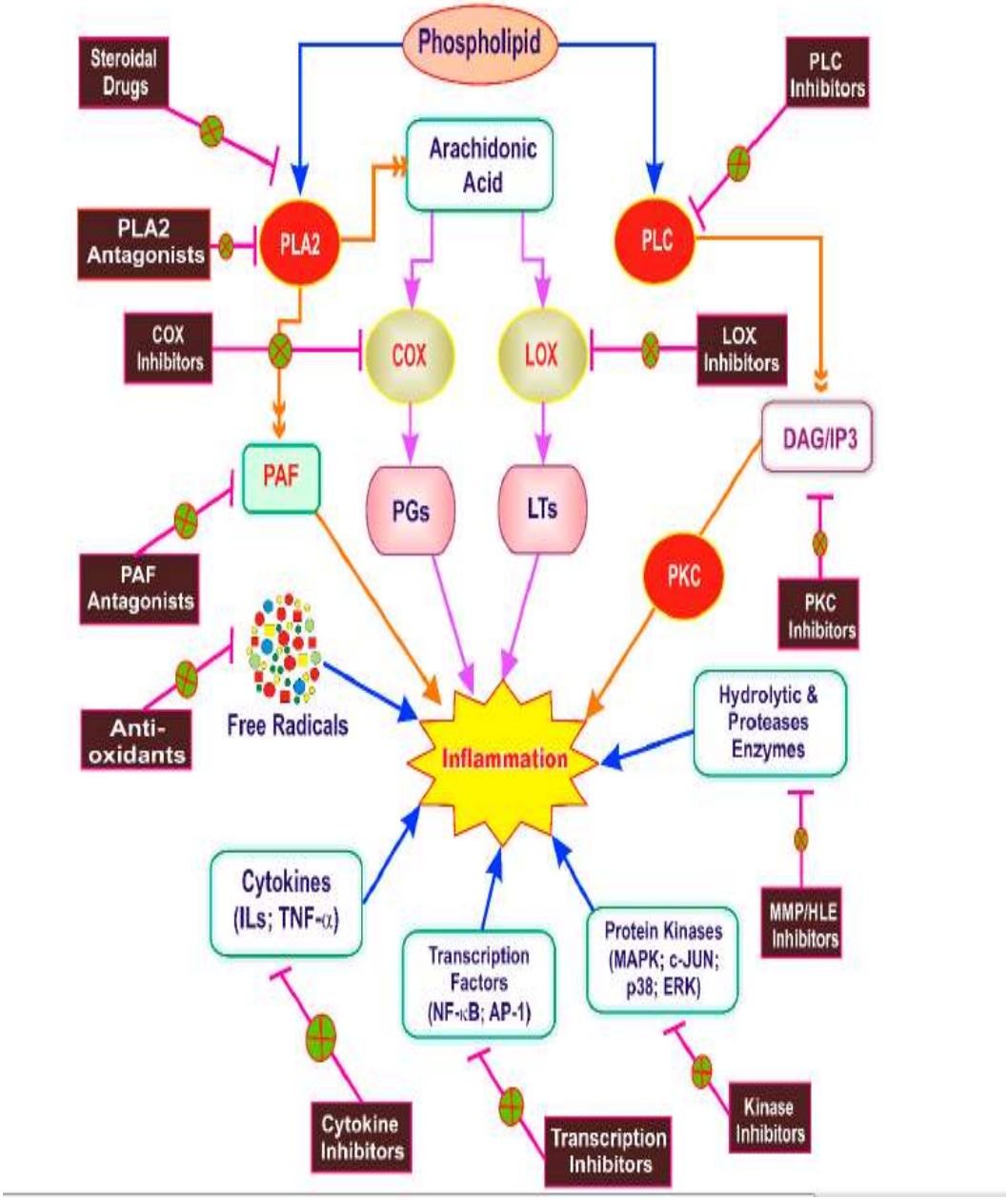

Fig. 11: Targets of phytoconstituents in inflammation.

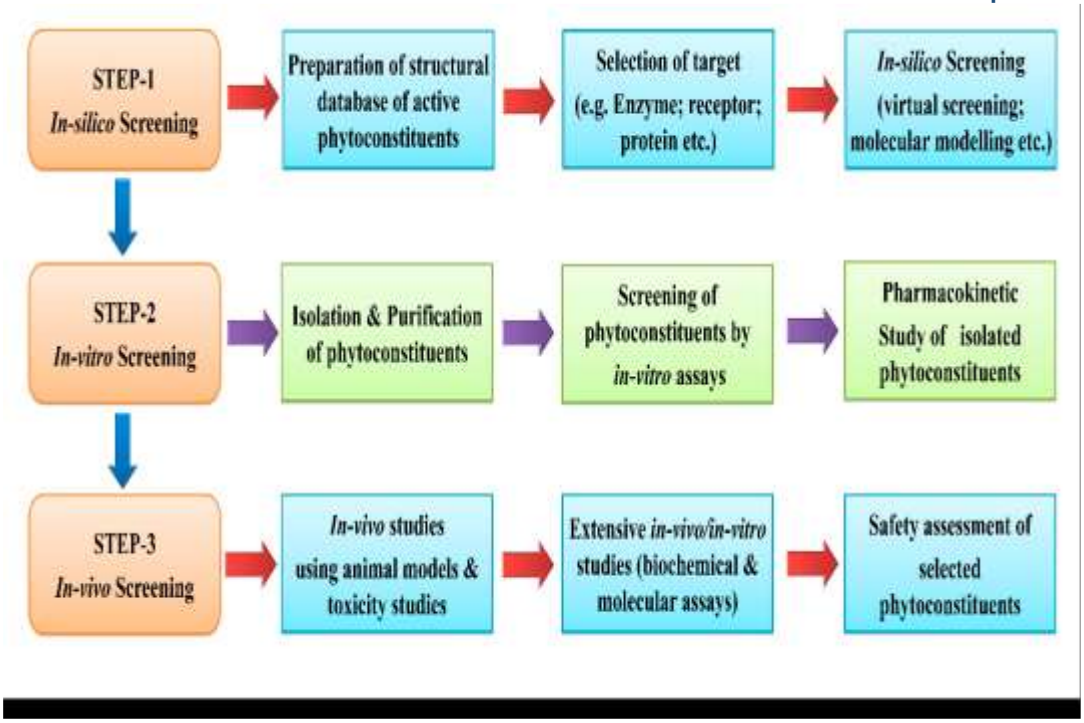

Fig. 12: Scheme for the preclinical evaluation of SPMs.

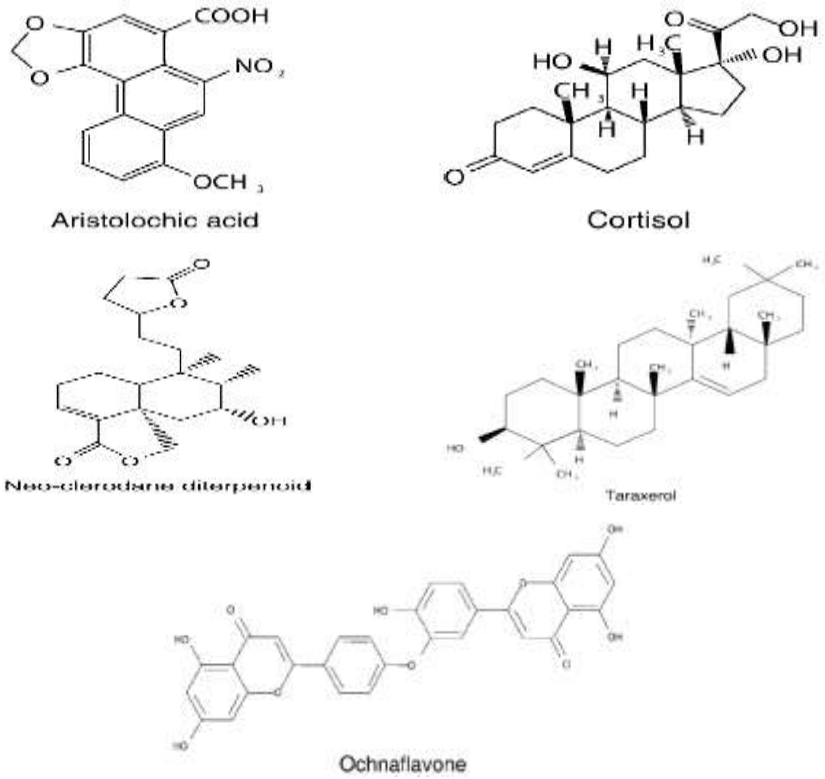

Fig. 13: Natural Products as Inhibitors of Phospholipase A2.

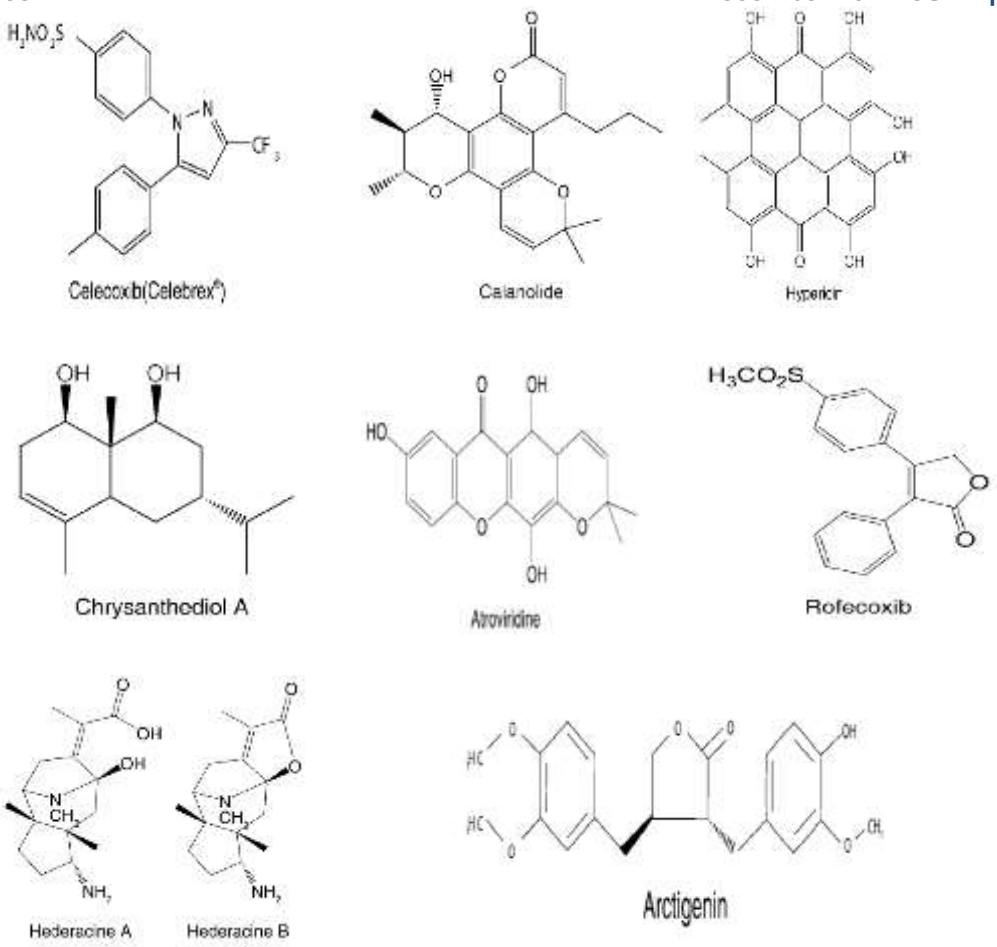

Fig. 14: Natural Products as Inhibitors of COX.

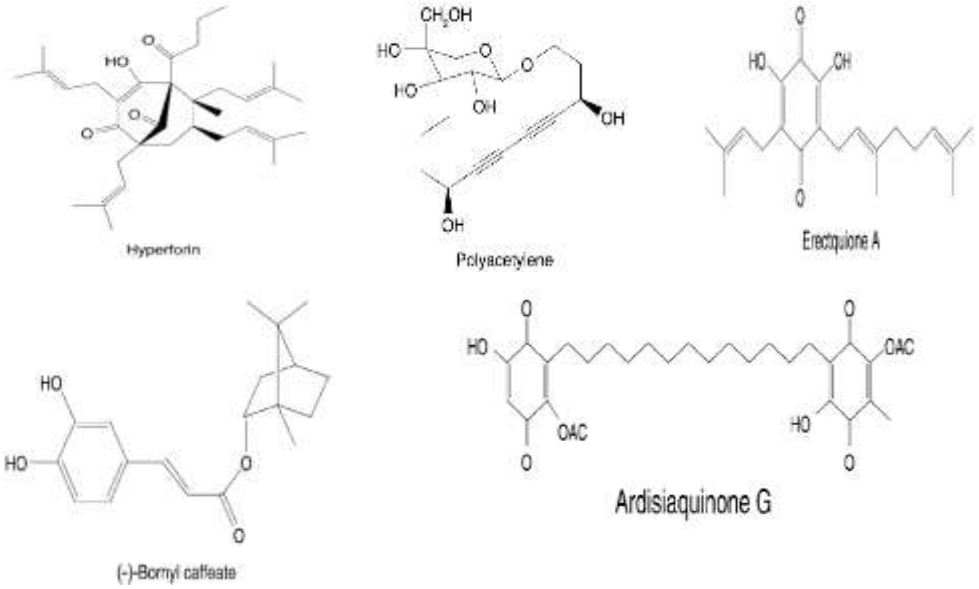

Fig. 15: Natural Products as Inhibitors of Lipoxygenases.

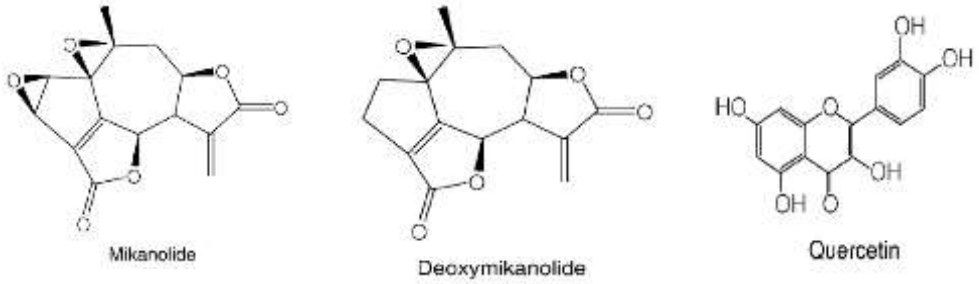

Fig.16: Natural Products as Inhibitors of Elastase.

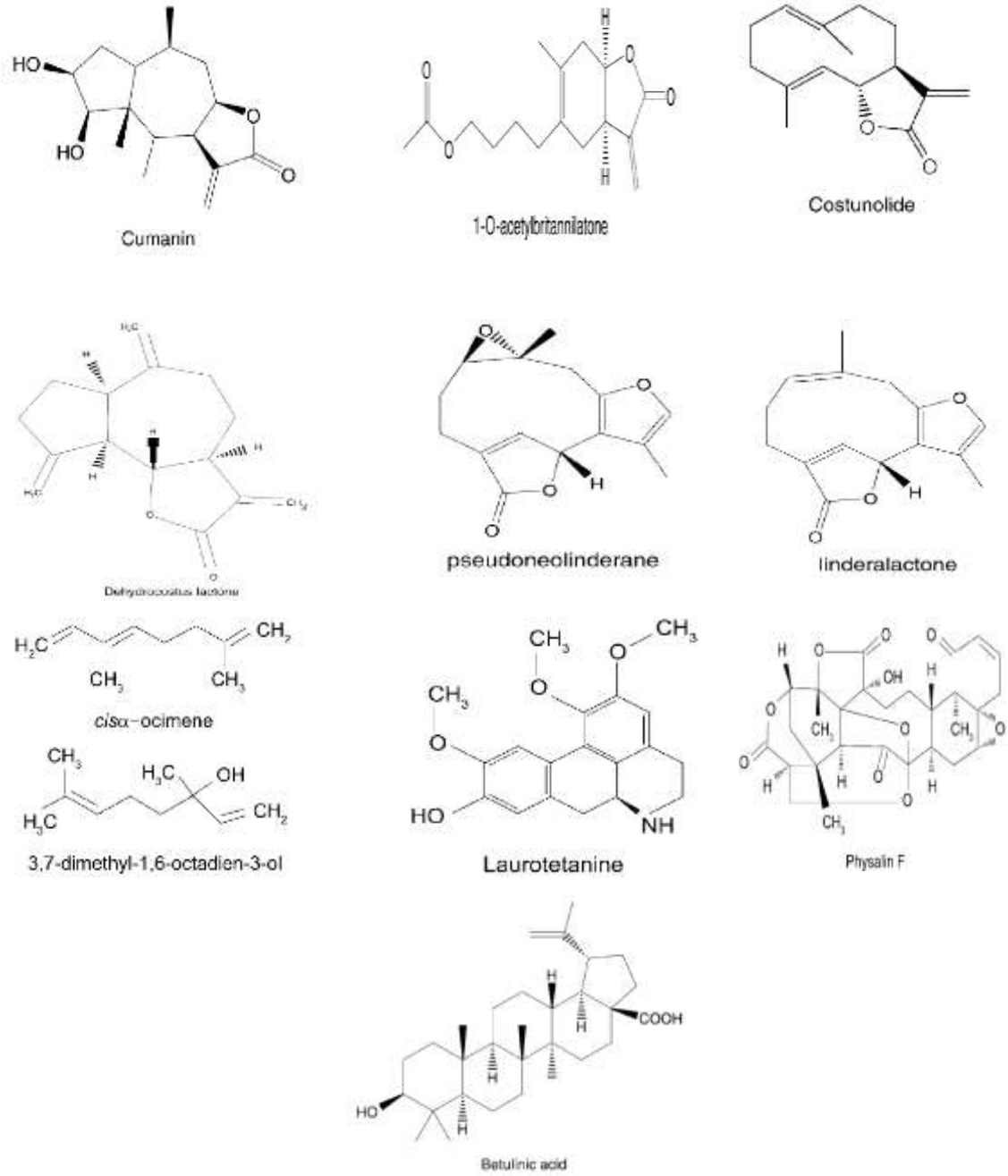

Fig. 17: Natural Products as Inhibitors of Nitric Oxide Synthetase.

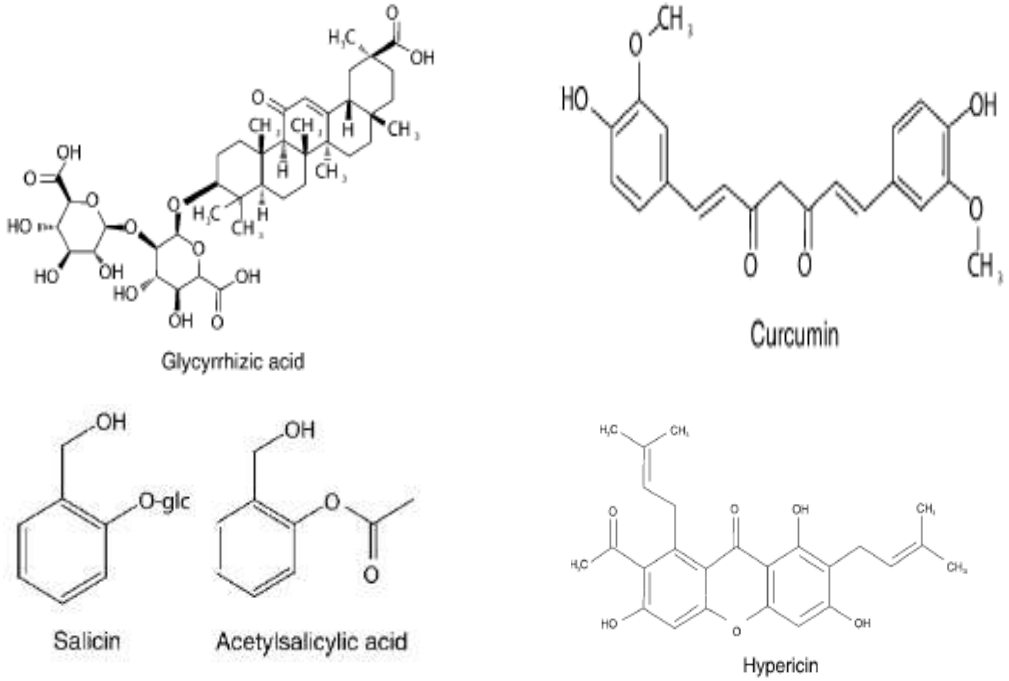

Fig. 18: Natural Products as Anti-inflammatory Agents.

Table 2: Plants which possess anti-inflammatory activity.

Plant & Family	Reference
Ananas comosus (L.) Merr.	Madisch *et al*., 2008
Boswellia serrata Roxb. (Burseraceae)	Gayathri *et al*., 2007;
Callophyllum inophyllum L. (*Clusiaceae*)	Etzel, 1996;
Camellia sinensis (L.) Kuntze (Theaceae)	Jurenka, 2009;
Centella asiatica (L.)	Deodhar *et al*., 1980
Curcuma longa L. (Zingiberaceae)	Nishiyama *et al*., 2005;
Harpagophytum procumbens	Gyurkovska *et al*., 2011;
Kalanchoe crenata Andr. (Crassulaceae)	Camargo *et al*., 2014;
Mangifera indica L. (Anacardiaceae)	Fiebich *et al*., 2001;
Olea europaea (Oleaceae)	Bogani *et al*., 2007;
Rosa canina (Rosaceae)	Orhan *et al*., 2007;
Rosmarinus officinalis. (Rosemary)	Sahu *et al*., 1999;
Uncaria tomentosa (Willd.) (Rubiaceae)	Aguilar *et al*., 2002;
Vaccinium myrtillus (bilberry)	Hoggard *et al*., 2013;
Zingiber officinale (Zingiberaceae)	Mahluji *et al*., 2013;

Survey reveals that species of 96 genera belonging to 56 families contain anti-inflammatory agents. These plants are as follows:

- *Acacia farnesiana* Linn. (Leguminosae).
- *Acanthopanax chiisanesis* Nakai (Araliaceae)
- *Achyrocline satureiodes* (Lam.) DC (Compositae)
- *Aconitum* spp. (Ranunculaceae)
- *Aegle marmelos* Correa (Rutaceae)
- *Aesculus hippocastanum* L. (Hippocastanceae)
- *Anacradium occidentale* Linn. (Anacardiaceae)
- *Angelica pubescens* Maxim. (Umbelliferae)
- *Antizoma angustifolia* (Burch.) Miers ex Harv. (Menispermaceae)
- *Azadirachta indica* A. Juss. (Meliaceae)
- *Baldwina angustifolia* (Compositae)
- *Boesenbergia pandurata* Roxb. Schltr. (Zingiberaceae)
- *Boswellia serrata* Roxb. ex Coleb. (Berseraceae)
- *Bougainvillea glabra* DC (Nyctaginaceae)
- *Bryophyllum pinnatum* Lam. (Crassulaceae)
- *Bumellia sartorum* Mart. (Sapotaceae)
- *Bupleurum* spp. (Umbelliferae)
- *Calophyllum inophyllum* Linn. (Guttiferae)
- *Cascora decussata* Schult. (Gentianaceae)
- *Catasetum barbatum* Lindl. (Orchdaceae)
- *Chrysanthemum indicum* Linn. (Compositae)
- *Cimmicifuga dahurica* Maxim. (Ranunculaceae)
- *Cocculus* spp. (Menispermaceae)
- *Coix lachryma –Jobi* L. (Graminae)
- *Commiphora mukul* Hook. Ex Stocks (Burseraceae)
- *Conzya Canadensis* (L.) Cronq. (Asteraceae)
- *Capaifera* spp. (Leguminosae)
- *Cordia obliqua* Wild. (Boraginaceae)
- *Cotinus coggyria* Scop. (Anacardiacae)
- *Croton lechleri* L. (Euphorbiaceae)
- *Cryptomeria japonica* D. Don. (Taxodiaceae)
- *Curcuma longa* Linn. (Zingeberaceae)
- *Cyperus rotundus* Linn. (Cyperaceae)
- *Dalbergia volubilis* Roxb. (Leguminosae)
- *Dismodium gangeticum* (Leguminosea)
- *Dianthus barbatus* C.V. (Caryophillacea)
- *Dioscorea mexicana (*Dioscoreaceae)

- *Dryopteris pacifica* Tagawa (Polypodiaceae)
- *Dysoxylum binectariferum* Hook. (Meliaceae)
- *Ecballium elaterium (*L) A. Rich.(Cucurbitacae)
- *Echinacea angustifolia* DC (Compositae)
- *Eisenia bicyclis (*Kjellman) Setchell
- *Embelia ribes* Burm. (Myrsinaceae)
- *Ephedra* spp.(Gnetaceae)
- *Eriobotrya japonica* Lindl.(Rosaceae)
- *Euphoria palustris* L. (Euphorbiaceae)
- *Garcinia mangostana* Linn. (Guttiferae)
- *Glycyrrhiza* spp.(Lehuminosae)
- *Haematoxylon campecchianum* (Linn.) (Leguminosae)
- *Hedychium spicatum* Ham. (Zingiberaceae)
- *Heimia salicifolia* and Otto (Lythraceae)
- *Hibiscus vitifolius* Linn. (Malvaceae)
- *Limonia crenulata* Roxb. (Rutaceae)
- *Madhuca longifolia* (L.) Macb. (Spotaceae)
- *Maesa chisia* D. Don (Myrisinaceae)
- *Magnolia salicifolia* Maxim. (Magnoliaceae)
- *Melilotus officinalis* Lam. (Leguminosae)
- *Menisremum dauricum* DC (Menispermaceae)
- *Mentha piperita* Linn. (Labiatae)
- *Mesua ferrea* Linn. (Guttiferae)
- *Myristica fragrans* Houtt. (Myristacaceae)
- *Ochrocarpus longifolius* Linn. (Guttiferae)
- *Panax japonica* C.A. Meyer (Arealiaceae)
- *Pedilanthus titymaloides* Poit. (Euphorbiaceae)
- *Peltophorum pterocarpum* Backer (Cesalpinaceae)
- *Physalis minima* Lin. (Solanaceae)
- *Phytolacca americana* Linn. (Phytolaccaceae)
- *Picrorrhiza kurroa* Royal ex – Benth (Scrophulariaceae)
- *Plagiorhegma dubium* Maxin. (Berberidaceae)
- *Platycodon grandifolium* A. DC (Campanulaceae)
- *Pluchea lanceolata* C. B. Clarke (Compositae)
- *Pluchea lanceolata* C. B. Clarke (Comositae)
- *Polygonum hydropiper* (Linn.) (Polygonaceae)
- *Potentilla erecta* Upenski ex Ledeb. (Rosaceae)

- *Psoralea coryfolia* Linn.(Leguminosae)
- *Pyrola rotundifolia* Linn. (Pyrolaceae)
- *Randia dumentorum* Lam. (Rubiaceae)
- *Rhamnua* spp (Rhamnaceae)
- *Rhus undulata* Jacq. (Anacardiaceae)
- *Schizogyme* spp (Compositae)
- *Scutellaria baicalensis* Georgi (Labiatae)
- *Sideritis* spp (Lamiaceae)
- *Solanum lycopersicum* Linn. (Solanaceae)
- *Solenostemma oleifolium* (Ascelpiadaceae)
- *Stephania* spp (Menispermaceae)
- *Terminalia ivorensis* (Cobretaceae)
- *Thalictrum sessile* Hayata (Ranunculaceae)
- *Triptergyium wilfordii* Hook. (Celastraceae)
- *Tylophora indica* (Burm. F.) Mirr. (Asclepiadaceae)
- *Usnea diffracta* Vain (Usneaceae)
- *Veratrum* spp (Liliaceae)
- *Vitex negundo* Linn. (Verbenaceae)
- *Withania somnifera* Dunal (Solanaceae)
- *Wrightia tinctoria* R. Br. (Apocyanaceae)

Table 3 : Anti-inflammatory activity of some medicinal plants.

Botanical Name / Family / Common Name	Parts Used	Constituents	Action / Uses
Acacia catechu (Mimosaceae) Katha	Bark, wood, flowering tops	Tannin, gum, catechuic acid	Anthelmintic, antipyretic, inflammation, bronchitis.
Allium sativum (Liliaceae) Lasun	Bulb, tuber, oil	Acrid volatile oil, starch.	Inflammation, anthelmintic, diuretic, antiseptic.
Azadirachta indica (Meliaceae) Neem	Leaf, root, seed, fruit, flower, oil.	Margosine, bitter oil, azadirachtin.	Lessening inflammation, anthelmintic, astringent, expectorant, stomachic.
Beta vulgaris (Chenopodiaceae) Beet	Root leaves	Betin.	Carminative, emmenagogue, Diuretic, inflammation.
Calotropics gigantea (Asclepiadaceae) Rhui	Root, flower, juice, bark	Glucosides, calotoxin, mudarine.	Purgative, anthelmintic, Expectorant, inflammation, Stomachic, leukoderma.
Cassia angustifolia (Caeasalpinaceae) Shona makhi	Pods, dried leaves.	Emodin, eatharitin, opleanic acid.	Laxative, purgative, skin diseases, inflammatory condition
Cinnamomum zeylanicum	Stem bark	Resin, linalon, tarmin	inflammation, stimulant, stomachic, diuretic.

(Lauraceae)		cinnamomum,	
Coriandrum sativum, Dhania (Umbelliferae)	Leaf, bark, mucilage of fruit	Tannin, catharin, albuminoids,	Carminative, anti-inflammation, jaundice, diuretic.
Cucurbita maxima (Cucurbitaceae)	Fruits seeds.	Organic acid, spinasterol	Diuretic, anthelmintic, in inflammation
Cuminum cyminum (Umelliferae) Jeera	Seed, fruit, Oil, flower.	Thymine, oil, thymol, gum.	Carminative, stomachic, inflammations, ulcer,
Curcuma longa (Zingiberaceae) Haldi	Tuber, rhizome.	curcumin, turmerol, curamone.	Inflammation, gastric disorder, scabies, leukoderma, swelling.
Cuscuta reflexa (Convolvulaceae)	Plant, seed, fruit, stem.	Cuscutine coumarin.	Inflammation, eye diseases, blood purifying.
Dalbergia sissoo (Fabaceae) Sisam	Roots, leaves,	Not reported	Anti-inflammatory, diuretic, bronchitis, leukoderma.
Emblica officinalis (Euphorbiaceae) Amla	bark, leaves, fruits.	Ellagic acid lupeol.	Astringent, antipyretic, inflammation, anaemia
Euphorbia hirta (Euphorbiaceae) Dudhi	Plant, roots, leaves	beta-amyrin, choline, inositol, linoleic-acid.	Anti-inflammatory, antioxidant, antitumor, antiseptic, antiviral, antiulcer, antiobesity.
Euphorbia tirucalli (Euphorbiceae)	Root, plant (milk, juice).	Beta-sitosterol, Ellagic-acid, ,	Anti-inflammatory, Anti - infertility, antitumor
Ficus benghalensis (Moraceae) Wad	roots, bark, seeds, leaves, latex.	Skin, fruits contain 10% tannin.	Anti-inflammatory, haemoptysis, dysentery, haemorrhages,
Ficus carica (Moraceae) Anjir	Fruit, root.	Ascorbic acid, Beta-carotene, Beta-Amyrin.	Carciogenic, Anticold, Insecticide, Antiasthmatic, Anti-inflammatory.
Ficus religiosa (Moraceae) Pipala	Bark, leaves, fruits, seeds	tannins, rubber and wax.	inflammation, diarrhoea, stomatitis, hemorrhages.
Foeniculum vulgare (Apiaceae) Shepu	Fruit, root, seeds, leaves.	Estragole, coumaric-acid,	antitumor, antioxidant, anti-inflammatory,
Gentiana kuroo (Gentianaceae) kadu	Rhizomes (roots)	Gentiopicrine and gentianic acid	Anti-inflammatory, haemorrhoids, leprosy, ulcers, fever.
Glycyrrhiza glabra (Papilionaceae) Jesthamadha	Roots, leaves.	Eugenol, Glycyrrhizin, Camphor,	Anti-inflammatory, antioxidant, antioxidant, Immunosuppressant.
Gymnema sylvesre (Asclepiadaceae)	Whole plant	gymnemic acid, tartaric acid,	Inflammation, bronchitis cardiotonic, laxative,
Hibiscus rosasinensis (Malvaceae) Jaswand	Buds, roots, leaves,	Quercetin, Ascorbic-acid.	anti-inflammatory, analgesic, cardiotonic.
Justicia gendarussa (Acanthaceae) Nilinirgundi	Roots, leaves.	Amino Beta-sitosterol.	Anti-inflammatory, thermogenic, bronchitis, ascites, cough.
Linum usitatissimum (Linaceae) Javas	Flowers, seeds, oil.	linoleic-acid, tocopherol,	Cardiotonic, antiulcer, local inflammation.
Maytenus emargiata,	Fruit, stem,	Tingenone,	Inflammation, ulcer, piles,

Yekaddi (Celastraceae)	bark, leaves, roots.	betulin, b-sitosteol.	burning, corneal opacity.
Momordica charantia, karela (Cucurbitaceae)	Whole plant	niacin, momordicoside	anti-inflammatory, emetic, antidiabetic, emmenagogue.
Nelumbo nucifera (Nymphaeceae) Kamal	Whole plant.	d-catechin, rutin, trigonelline	cardiotonic, inflammation, skin diseases, bronchitis,
Nicotiana tobacum (Solanaceae) Tamabaku	Leaves.	nicotinic-acid, nicotine, tocopherol	anti-inflammatory, laxative, trigonelline, mental
Nigella sativa (Ranunculaceae) Kalajira	Seeds.	carvone, methionine, stigmasterol	Anti-inflammatory, carminative, thermogenic, emmenagogue, anodyne.
Ocimum basilicum (Laminaceae) Tulsi	Whole plant	aspartic acid, apigenin, arginine.	Anti-inflammatory, cardiotonic, insecticidal, antipyretic.
Piper longum (Piperaceae) pimpli	Roots, fruits.	Piperogumine, piperine,	stomachic, antidiarrhoeal, Antidysenteric.
Plantago ovata (Plantaginaceae) Isabgola	Seeds	Oil, albumin, mucilage.	anti-inflammatory, expectorant, diuretic, anticholesterol.
Plumbago zeylanica (Plumbaginaceae)	Root, leaves, root, bark.	Plumbagin, elliptione,	Stomachic, astringent, anti-inflammatory.
Pterocarpus marsupium (Fabaceae)	Heart wood, leaves	Alkaloids, gum, essential	Anti-inflammatory, anthelmintic, constipating
Raphanus sativus (Brassicaseae) Mula	Roots, leaves, seeds.	aspartic acid, caffeic acid	antibacterial, laxative, anodyne, depurative.
Ricinus communis (Euphorbiaceae) Arandi	leaves, seeds, flowers, oil.	ricin, palmitin, sterine.	anthelmintic, diuretic, astringent, galactagogue, expectorant.
Rubia cordifolia (Rubiaceae) Manjesthta	Roots.	Starch, colouring matter	Anti-inflammatory, carminative, diuretic, galactopurifier.
Solanum nigrum (Solanaceae) Makoi	Whole plant.	Solenin, solasodine,	Anti-inflammatory, cardiotonic, emollient
Swertia chirayita (Gentianaceae) Chirayita	whole plant	resin, gum, resin, phosphate	Anti-inflammatory, antipyretic, thermogenic, antiperiodic.
Tamarindus indica (caesalpiniaceae) Chinch	Roots, leaves, Fruits	Tartaric, citric, malic, acetic,	Astringent, thermogenic, constipating, diuretic, stomachic
Taraxacum officinale (Asteraceae)	Whole plant	Latex contain taraxacerin.	Anti-inflammatory, stomachic, stimulant.
Tephrosia purpurea (Fabaceae) Unhali	Whole plant	Tephrosin, lupeol, rutin.	Anti-inflammatory, anthelmintic,
Terminalia arjuna (Combretaceae)	bark	Not reported	Anti-inflammatory, astringent, dysenteric.
Terminalia belirica (Combretaceae)	Bark, fruits / Beheda	Not reported.	Anti-inflammatory, thermogenic, astringent.
Tinospora cordifolia,	Stem	Alkaloids,	anti-inflammatory,

Giloi, Menispemaceae		starch.	antiemetic, expectorant.
Tribulus terrestris (Zygophyllaceae)	Whole plant	Diuretics.	Anti-inflammatory, laxative, appetiser, styptic, diuretic
Vitex negundo (Verbenaceae) Nirgundi	Whole plant	essential oil, resin, astringent.	Expectorant, anti-inflammatory, digestive, antipyretic,
Zingiber officinle (Zingiberaceae) Adrak	Rhizomes	cineol, shogaol zingiberene,	anodyne, expectorant, carminative, thermogenic.

Wound

Meenakshi *et al*., 2006, Wound is the type of the injury in which skin is torn, cut or puntured (an open wound), or where blunt force trauma causes a contusion (a close wound). It refers to the sharp injury which damages the dermis of the skin. Open Wound can be classified according to the object that causes the wound. Types of wounds are:

- Incision or incised wound caused by sharped edge object such as knife, razor.
- Lacerations: Irregular tear like wound caused by some blunt trauma
- Abrasions (Grazes): Superficial wounds in which the topmost layer of the skin (epidermis) is scrapped off.
- Puncture Wound: Caused by the object puncturing the skin such as needle, nail
- Penetration Wound: Caused by an object such as knife.
- Closed Wound: It includes the following types:
- Contusions (Bruises): Caused by a blunt force trauma that damage tissue in skin.
- Hematomas (Blood Tumour) : Caused by damage to the blood vessel that in turn causes the blood to collect under the skin.
- Crash Injury: Caused by extreme force applied over a long period of time

Fig. 19: Open Wound.

Fig. 20: A Laceration with the leg (Open Wound).

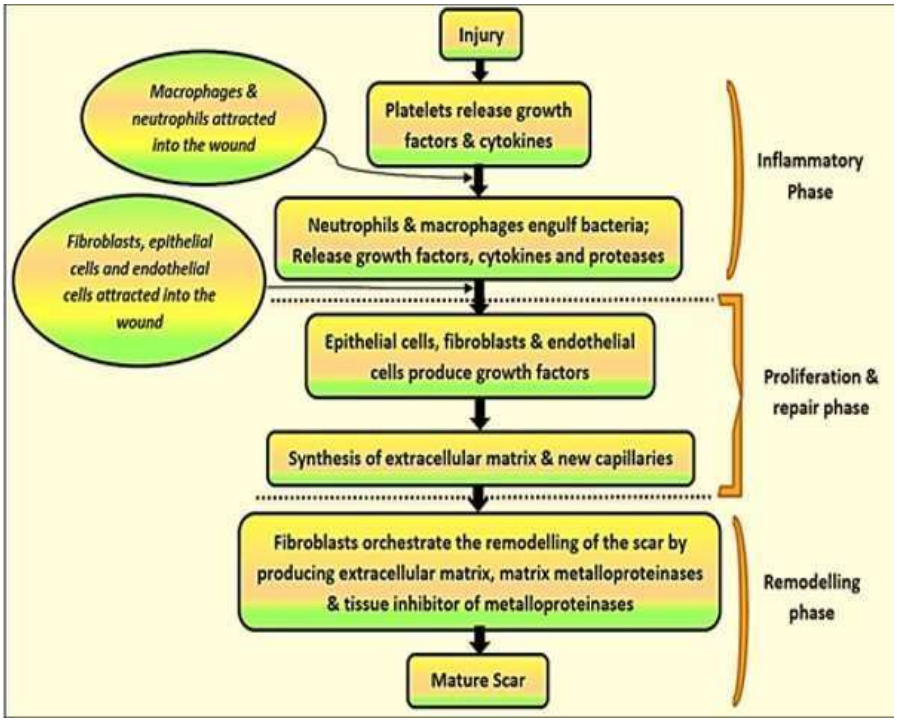

Fig. 21: Pathophysiology & Physiology of Wound Healing.

Wound Healing Process

i. Wound healing is a dynamic, complex, and delicate process that may be endangered at any point by improper or inadequate management.

ii. Hemostasis is the coagulation of blood leaking from a damaged, inflamed or dilated vessel. After hemostasis occurs, inflammation sets in.

iii. Inflammation appears as erythema and swelling due to vascular dilation and the inflow of plasma. Signs of inflammation should start to resolve 48 to 72 hours after the occurrence of a wound. Granulation, the third phase of healing, requires the presence of growth factors released by macrophages during the inflammatory phase.

iv. Wound contraction occurs in deep wounds as margins are pulled together by the contraction of specialized fibroblasts. This process facilitates epithelial proliferation, migration, and differentiation (re-epithelialization).

v. The last step of re-epithelialization is differentiation, restoring the protective outer layer of the skin. Closure of a wound does not mean that the healing process has been completed. The maturation phase, during which a wound gains tensile strength, may take several months. While superficial wounds heal by regenerating a perfect new epidermis, deeper wounds never achieve the former degree of dermal organization or strength.

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