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A REVIEW ON INFLAMMATION

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ABSTRACT –

The study covers the essential role of inflammation in many diseases, particularly autoimmune ailments, and highlights the mechanisms involved in the inflammatory process. Here are the important points that could build a good abstract: The function of inflammation in addition to infectious and non-infectious diseases like cancer, diabetes, and cardiovascular disorders, inflammation plays a crucial role in tissue damage in autoimmune diseases. Reactive oxygen species (ROS), such as superoxide (O2), hydrogen peroxide (H2O2), and hydroxyl (OH) radicals, are produced by inflammatory cells and are what define it. By oxidizing the membrane's lipids, these ROS increase the permeability of cell membranes. Effects of Inflammation: The inflammatory response causes capillary dilatation, which causes redness and pain; additionally, increased vascular permeability allows plasma to escape into surrounding tissues, resulting in edema. This underscores the dual nature of inflammation, where it serves both protective and damaging roles. Inflammatory processes are crucial in determining the fate of tissues after injuries. Key components of inflammation include edema formation, leukocyte infiltration, and granuloma formation.

Key Words - Reactive Oxygen Species (ROS), Oxidative Stress, Inflammation, Herbal plants.

1.1 INTRODUCTION: -

Inflammation is part of the intricate biological response of vascular tissues to harmful stimuli, including pathogens, damaged cells, or irritants (Ferrero-Miliani et al. 2007; Palladino et al. 2003). It is a crucial physiological response that occurs to help limit damage and support tissue repair (Nathan 2002). The processes involved in inflammation are vital in determining whether tissues survive or are destroyed following various injuries. Components of inflammation include edema formation, leukocyte infiltration, and granuloma formation (Mitchell & Cotran 2000). Inflammation significantly contributes to the damage associated with autoimmune diseases and plays a key role in numerous infectious and non-infectious diseases, such as cancer, diabetes, cardiovascular disease, rheumatoid arthritis, Alzheimer's, and arteriosclerosis (McGeer & McGeer 2002; Howes & Houghton 2003). Inflammation is a generic response, and therefore is considered a mechanism of initiate immunity, whereas adaptive immunity is specific to each pathogen AB Abbas, (AH Lichtman 2009 et al).
The five cardinal signs namely include swelling, redness, heat, pain, and altered function (Murugan et al. 2012).

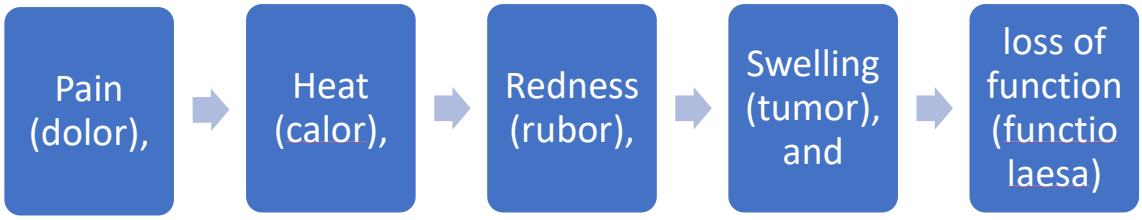

Figure No.1.1

Inflammatory cells produce wide variety of reactive oxygen species (ROS) which include superoxide (O2 •), hydrogen peroxide (H2O2) and hydroxyl (OH•) radicals which increase cell membrane permeability initiated by oxidation of lipids on the membrane

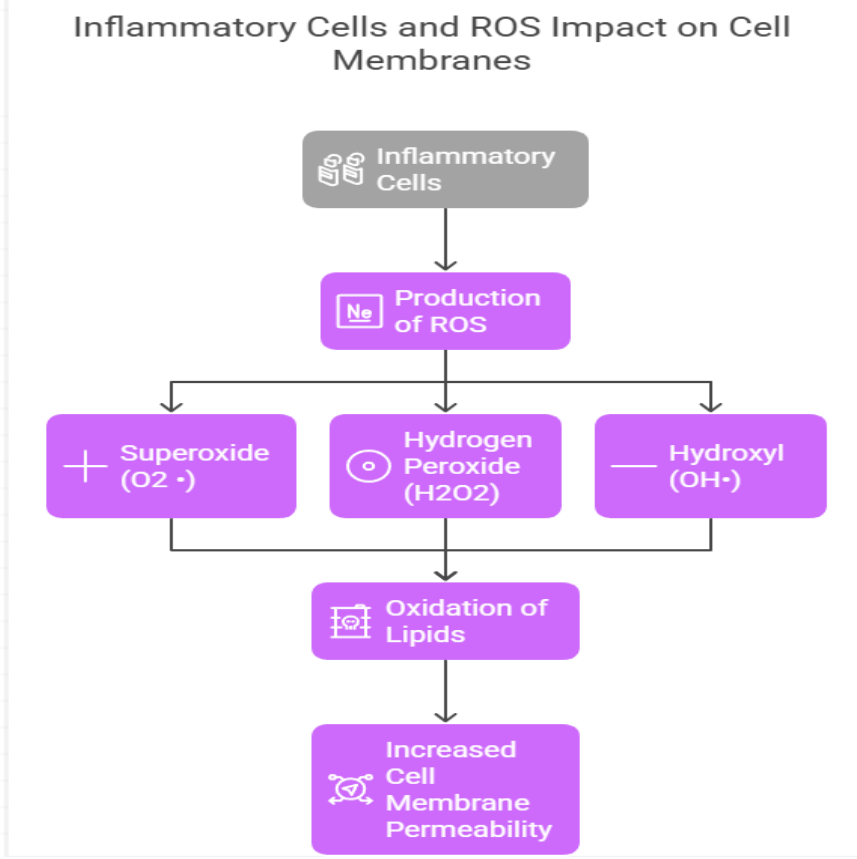

Figure No.1.2

These two results in capillary dilation causing redness and pain, and increased vascular permeability allows plasma to escape into the surrounding tissue giving rise to edema (Halliwell 1995).

Oxidative stress is associated with a variety of health issues, including cardiovascular diseases, diabetes, inflammation, degenerative diseases, cancer, anemia, and ischemia (Cai et al. 2004). Reactive oxygen species (ROS) refers to a group of oxygen-centered radicals, such as hydroxyl radicals and superoxide anions, along with non-radical derivatives like hydrogen peroxide and singlet oxygen. ROS primarily target polyunsaturated fatty acids, which are precursors to lipid peroxide formation and contribute to oxidative stress (Gutteridge 1994).

In humans, the over production of ROS can produce tissue injury. It has been implicated in the pathogenesis of cancer, alzheimer disease, inflammation, diabetes mellitus, atherosclerosis, rheumatoid arthritis aging and other diseases (Christen et al. 2000; Droge 2002).

Medicinal Plants can play a vital role in free radical mediated disorder. Antioxidants are compounds that protect cells against the damaging effects of reactive oxygen species. Medicinal plant which consists of numerous antioxidant compounds which prevent the damaging of cells. Plants are endowed with free radical scavenging molecules, such as vitamins, terpenoids, phenolic acids, lignins, stilbenes, tannins, flavonoids, quinones, coumarins, alkaloids, amines, betalains, and other metabolites, which are rich in antioxidant activity (Zheng & Wang 2001).

1.1.1 Types of inflammation

Acute inflammation and chronic inflammation

Acute inflammation is marked by heightened vascular permeability and the infiltration of cells, resulting in the development of edema due to the leakage of fluid and proteins, along with the gathering of leukocytes at the site of inflammation for a brief period (Posadas et al. 2004).

When the initial response is not enough to get rid of the pro-inflammatory substances, chronic inflammation results. It is brought on by the growth of proliferative cells, which have the potential to spread or form granulomas. It can also be brought on by an infection or antigen that persists, repeated tissue damage, or a malfunction in the body's natural anti-inflammatory defenses.

Oesophageal, gastric, hepatic, pancreatic, and colorectal cancers are among the gastrointestinal cancers for which chronic inflammation is a risk factor (Ramos-Nino 2013; Wang & Du Bois 2008).

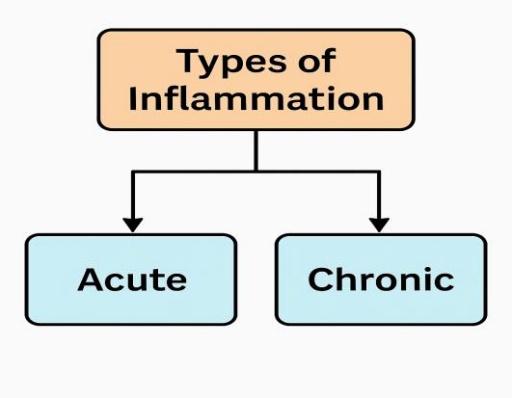

Figure No.1.3 Types of Inflammation

1.1.2. Problems associated with chronic inflammation

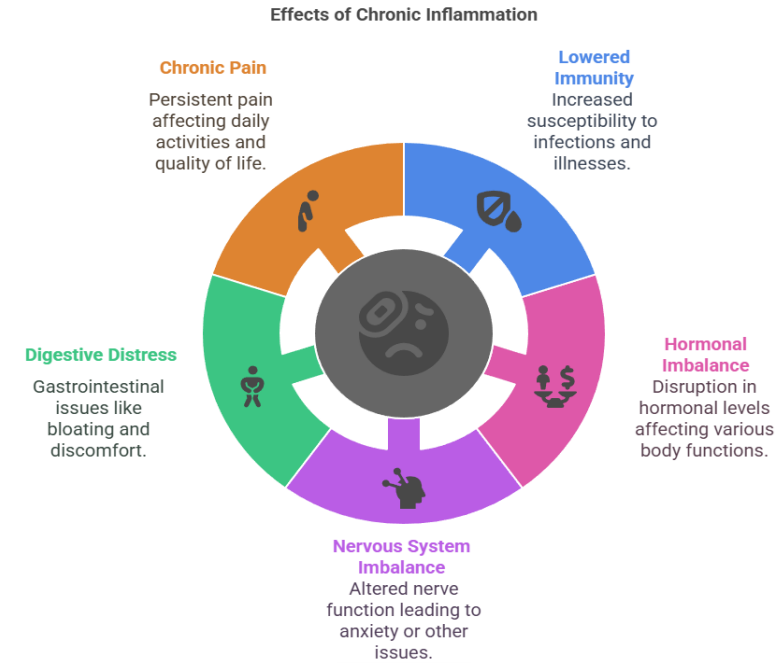

Figure No.1.4

1.1.3. Symptoms of inflammation Redness, swollen joint that is warm to touch, joint pain, joint stiffness, loss of joint function, fever, chills, fatigue/loss of energy, headaches, loss of appetite, muscle stiffness etc.

1.1.4. Causes of inflammation Pain is associated with actual or potential tissue inflammation. Inflammation, pain and fever are all associated with enhanced production of prostaglandins (Rang et al. 2003).

- Chemicals – food allergy, hay fever, handling or breathing harsh chemicals (cleaners, gasoline, etc.), air pollution and others can cause inflammation. Even cosmetics and toiletries.
- Infections also cause inflammation – from bacteria, virus, fungus, or yeast.
- Increased body fat can cause inflammation because fat cells produce inflammatory chemicals. This is especially serious in those who are obese.
- Intense physical activity, such as anaerobic exercise – high intensity training, weight lifting and competition – also causes significant inflammation.
- Nutritional imbalances can cause chronic inflammation.

- Arthritis is a general term that describes inflammation in joints. Some types of arthritis associated with inflammation.

Inflammatory diseases are diagnosed by careful evaluation of:

Complete medical history and physical examination, the location of painful joints, joint stiffness in the morning, evaluation of other symptoms like pain, fever, muscle stiffness etc., results of X-rays and other tests.

1.1.5. Mediators of inflammation

Inflammation is characterized by the release of proinflammatory cytokines such as tumor necrosis factor (TNF)-α, interleukin (IL)-1β, and IL- 6, as well as inflammatory mediators, including NO and prostaglandin E2 (PGE2), that are synthesized by inducible nitric oxide synthetase (iNOS) and cyclooxygenase-2 (COX-2) (Won 2006; Sautebin 2000). These mediators play critical roles in the development of acute and chronic inflammation. Inflammatory mediators are divided into two types. They are exogenous and endogenous mediators. Bacterial products and toxins can act as exogenous mediators of inflammation. Macrophages play a central role in managing many different immunopathological phenomena including the overproduction of proinflammatory cytokines and inflammatory mediators, generated by activated iNOS and COX-2 (Walsh 2003).

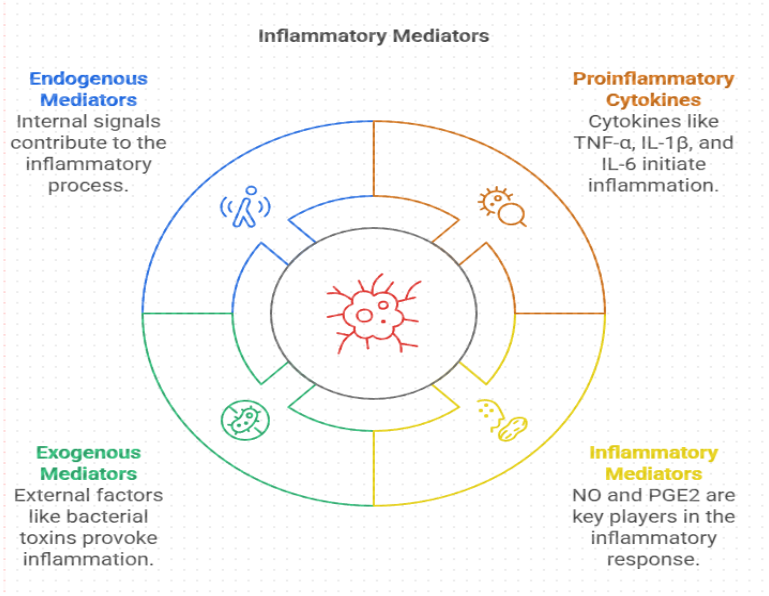

Figure No.1.5 Mediators of inflammation

1.1.6. Prostaglandins- Prostaglandins are found in most tissues and organs. They are produced by all nucleated cells except lymphocytes. Prostaglandins are lipid autacoids derived from arachidonic acid. They are generated from arachidonate by the action of cyclooxygenase (COX) isoenzymes. It is the key enzyme required for the conversion of arachidonic acid to prostaglandins explained in the Figure .1.1 (Ricciotti & Fitz Gerald 2011).

Prostaglandin endoperoxide synthase is commonly called cyclooxygenase (COX). Every prostaglandin contains 20-carbon atoms, including a 5-carbon ring (Smith & Marnett 1991). Prostaglandins are produced in the inflamed tissues, and treatment with NSAIDs inhibits the production of prostaglandins and down-regulates inflammation-related pathological symptoms such as pain and swelling (Morita 2002). During inflammation, COX-1 levels do not change, but COX-2 levels increase dramatically, and, as a result, prostaglandin production increases (Profita et al. 2003). The COX-2 gene is particularly responsive to mediators of inflammation (Rahman et al. 2006). COX-2 specific inhibitors are administered; prostaglandin production and subsequent inflammation are significantly reduced (Simmons et al. 2004). Therefore, COX- 2 specific inhibitors have been used to attenuate the symptoms of inflammation such as osteoarthritis, rheumatoid arthritis and musculoskeletal pain.

1.1.7 Role of cyclooxygenase in inflammation- Cyclooxygenase (COX), often referred to as prostaglandin endoperoxide synthase (PGHS), is the main enzyme that transforms arachidonic acid into prostaglandins. The two isoforms of COX are COX-1 and COX-2 (Pairet & Engelhardt 1996). Alter et al. (2001) claim that both COX-1 and COX-2 are constitutive and inducible enzymes involved in inflammation and cryoprotection.

According to Vane & Botting (1998), the enzyme that catalyzes the first two stages of the production of prostaglandins (PGs) from the substrate arachidonic acid (AA) is cyclooxygenase (COX) or prostaglandin H2 synthase (PGHS). They include the reduction of AA to the hydroxyl endoperoxide PGH2 after it has been oxidized to the hydroperoxyl endoperoxide PGG2. A variety of enzymes and nonenzymatic processes convert PGH2 into the major proteinoids PGE2, PGF2, PGD2, PGI2, and TXA2. It was discovered that all nonsteroidal anti-inflammatory drugs target the cyclooxygenase (COX) enzymes medications, or NSAIDs (Pouplana et al. 2002). In inflammatory cells, COX-2 is crucial for the production of PG (Aid & Bosetti 2011).

1.1.8 Inflammatory diseases Gautam & Jachak (2009) reported that inflammatory diseases include rheumatoid arthritis, atherosclerosis, alzheimer's, asthma, psoriasis, multiple sclerosis and inflammatory bowel diseases and many of these inflammatory diseases are becoming common throughout the world (Figure 1.6). Chronic inflammation is a root cause of many diseases of advanced age such as heart attacks, alzheimer's diseases, and cancer (Mueller 2006). Rheumatoid arthritis and osteoarthritis also associated with inflammation (Mythilypriya et al. 2008).

1.1.9 Standard drugs for inflammation and its side effects

Three major groups of drugs used in treatment of inflammatory diseases are

- Corticosteroids
- Disease-Modifying Anti-rheumatoid Drugs (DMARDS)
- Non-Steroidal Anti-Inflammatory Drugs (NSAIDs) such as Aspirin, Ibuprofen or naproxen.
- Many inflammatory illnesses can be effectively treated with these commonly used medications. The most common reason NSAIDs are prescribed is to relieve inflammation. By inhibiting the activity of COX, which is implicated in preventing the release of PGs, NSAIDs diminish inflammation-related fever and pain (Simmons et al. 2004). There are about 50 distinct NSAIDs on the market (Chiroli et al. 2003). These NSAIDs have been well-known for their three primary effects: analgesia (pain reduction), anti-inflammatory, and antipyretic (fever reduction). According to Kalgutkar et al. (2000), prolonged usage of NSAIDs results in bleeding, platelet malfunction, and stomach ulcers.

NSAIDs have three major types of action (Vane & Botting 1998):

- Anti-inflammatory action for treating several conditions including rheumatoid arthritis, osteoarthritis, musculoskeletal disorders and pericarditis.
- Analgesia for treating pain of mild to moderate intensity. Their maximum therapeutic efficiency is much lower than that of the opioids, but they do not cause dependence.
- Antipyretic action mediates by the release of endogenous pyrogen from monocytes and macrophages in the presence of infection or inflammation.

By inhibiting the COX enzymes and lowering prostaglandins throughout the body, nonsteroidal anti-inflammatory medications (NSAIDs) lower fever, discomfort, and inflammation. NSAIDs are frequently used to treat acute or chronic disease symptoms. Asthma, psoriasis, and arthritis are examples of chronic inflammatory conditions that have been treated with NSAIDs (Poddubnyy et al. 2009). Aspirin, indomethacin, ibuprofen, naproxen, diclofenac, and others are among the most often used medications. According to McGettigan and Henry (2013), diclofenac is the most widely used NSAID globally. The majority of COX inhibitors have therapeutic applications as anti-inflammatory, anti-pyretic, analgesic, and anticoagulant drugs. According to Masferrer & Kulkarni (1997), NSAIDs block the cyclooxygenase active site of COX isozymes, which in turn stops the COX action (Bancos et al. 2009). NSAIDs can be divided into different groups based on their chemical structure, pharmacokinetics and selectivity towards COX-1 or COX-2 (Cryer & Feldman 1998).

NSAIDs are divided into two major groups such as

- Cyclooxygenase (COX) -2-selective inhibitors (COXIBs)
- Non-selective NSAIDs.

The toxicity and recurrence of symptoms upon withdrawal are major drawbacks of the powerful anti-inflammatory synthetic medications now on the market (Chandran et al. 2011). According to Essex et al. (2013), prolonged use of NSAIDs damages the cardiovascular and renal systems and causes gastrointestinal ulcers. According to Donihi et al. (2006), prolonged usage of corticosteroids can result in a number of serious adverse effects, including osteoporosis, diabetes mellitus, insulin resistance, hyperglycemia, and anxiety. According to Bancos et al. (2009), NSAIDs can also impair immunological function, which can have detrimental effects on children, the elderly, and people with weakened immune systems. Two basic kinds of analgesic medications are used in analgesic therapy: opioids

and non-steroidal anti-inflammatory drugs (NSAIDs). Few NSAID drugs are listed.

- **Aspirin**

  Aspirin is the most commonly recommended and prescribed drug in the world because it acts as anti-inflammatory medicine (Schwab et al. 2007). Aspirin is effective in treating fever, pain, and inflammation in the body. Long term use of aspirin causes peptic ulcer and gastrointestinal disorders.

- **Diclofenac**

  Diclofenac, a nonsteroidal anti-inflammatory drug which is used for the treatment of mild to moderate pain, fever, and inflammation. Diclofenac reduces inflammation, swelling and arthritic pain. Long term uses of Diclofenac cause gastrointestinal disorders (Aydin et al. 2003) and cutaneous lesions by intramuscular injection (Suwalsky et al. 2009) and are hepatotoxicity (O'connor20003).

- **Coxibs**

  Coxibs are selectively to COX-2. It inhibits only the inflammatory prostaglandins, not the COX-1. Rofecoxib, lumiracoxib, celecoxib and etoricoxib are coxibs. Coxibs are associated with serious cardiovascular events (Pilotto et al. 2009) and hepatotoxicity (Alegria et al. 2002). Celecoxib produces fewer side effects of gastrointestinal complications (Mallen et al. 2011). Celecoxib (Celebrex) and rofecoxib (Vioxx) were removed from market because the long-term use of these drugs produces cardiovascular problems (Mukherjee 1997; Wood 2001).

- **Indomethacin**

Indomethacin reduces fever, pain and inflammation. Indomethacin works by reducing the production of prostaglandins. Indomethacin blocks the enzymes that make prostaglandins (cyclooxygenase 1 and 2) and thereby reduces the levels of prostaglandins. As a result, fever, pain and inflammation are reduced. Indomethacin is used in the treatment of disorders such as rheumatoid arthritis, and osteoarthritis. Long-time use of indomethacin causes gastrointestinal complications, including intestinal perforation (Cassady et al. 1989) bronchopulmonary dysplasia and respiratory distress syndrome (Eronen et al. 1994).

- **Ibuprofen**

  Ibuprofen is a commonly used NSAIDs derivative of propionic acid. Ibuprofen is used for the treatment of mild to moderate pain, inflammation and fever. Long term use of ibuprofen has been associated with hepatotoxicity (Manoukian & Carson 1996).

- **Nimesulide**

  Nimesulide is a selective COX-2 inhibitor, with only residual activity against COX-1 (Giuliano et al. 2001). The drug can cause liver damage, hepatocellular necrosis (Lucena et al. 2001).

PHYTOMEDICINE

1.2.1 About medicinal plants and Phyto drugs

Medicinal plants had a particular place in human life. Plant medicines are an important part of primary healthcare in many developing countries. Plants have been used by humans to treat or prevent illness. Plant-based natural products offer a lot of unrealized potential. For hundreds of years, people have utilized these natural plant ingredients as insecticides, flavorings, fragrances, colors, medications, antioxidants, and more (Sajeesh 2011). For thousands of years, people have been searching for novel drugs to treat a wide range of ailments. There is a continuous hunt for new plant-based drugs that could be used as agents. Many of these chemicals are used as medications in modern medicine.

Many plant-based drugs are effective in treating a variety of ailments at affordable prices and without unpleasant side effects. Worldwide, the use of plant-based medicines is now permitted. Approximately 47% of the most widely prescribed medications in the US contain natural ingredients or derivatives (Newman & Cragg 2007). Plants are capable of producing a wide variety of phytochemical compounds. According to Raskin et al. (2002), the pharmacological activity of natural chemicals originating from plants is yet unknown or undiscovered. Julie and Jurenka (2009) claim that consuming plant-based diets reduces the

Like hood of acquiring illnesses such inflammatory diseases, cancer, neurological conditions, cardiovascular disease, and inherited diseases.

India is entitled to one of the world's largest collections of medicinal plants, and Ayurveda, Unani, and Siddha all use extracts from these plants to treat illnesses (Sasikala et al. 2011). In India, herbal treatments are widely used. Because plant-based medications have fewer or no negative effects than synthetic ones, Indians utilize a lot of them to treat a variety of illnesses. According to Tiwari (2008), it is the leading producer of raw materials for crude medications or bioactive chemicals used in the development of pharmaceuticals, cosmetics, and other products.

Medicinal plants Because they are readily available, affordable, and may be used quickly for personal needs, medicinal plants have been employed as medicine (Mukherjii 2008). Both rich and developing nations have made extensive use of plant-based Phyto drugs in recent years. Many components of medicinal plants are used to make these Phyto drugs. Latex, gum, and resins are used to make some medications. More than 20,000 species have been classified by the World Health Organization in an effort to identify all medical plants used worldwide (Pandey 2008). Additional details regarding the use of plants or plant parts as medicine are provided (Saikia 2008).

The growing interest in using medicinal plants worldwide has led to a massive rise in enterprises based on these plants in recent years. The creation of novel medications from natural sources is still regarded as significant despite the numerous advancements in contemporary medicine. (Parakh and others, 2010). Plants are the source of around half of contemporary medications, which are used to treat a variety of illnesses (Noorwez 2008). Pharmacological characteristics of plant products include analgesic, antipyretic, and inflammatory effects (Shukla et al. 2010).

Due to poverty and limited access to contemporary medical care, the World Health Organization (WHO) estimates that between 65 and 80 percent of the world's population relies on medicinal plants and plant-based products for their basic medical needs (Calixto 2005). In many nations, traditional medicine derived from medicinal plants has always been important (Pandiarajan et al. 2011). Drugs are generally administered as ethanolic extract or water decoction in conventional medical systems. The majority of medicinal plant components have a variety of therapeutic uses and are utilized as raw medications (Mahesh & Sathish 2008). People use medicinal plants as medicine, such as natural raw powder, fresh plant pieces, or fresh juice.

1.2.2 Advantages of phytomedicine

There are a number advantages associated with using herbal medicines as opposed to pharmaceutical products.

- Reduced risk of side effects Many phytomedicines are very safer to use over a long period and only a few, develop fewer side effects.

- Lower cost: medicinal plants are easily available and very cheaper than the synthetic chemical medications. Herbs tend to be inexpensive compared to drugs. Widespread availability: Herbs are available without a prescription. You can grow some simple herbs, such as peppermint and chamomile, at home. In some remote parts of the world, herbs may be the only treatment available to the majority of people.

1.2.3 Standardization of herbal medicine

Due to the accessibility, safety, and affordability of these medicinal plants and Phyto drugs, health organizations worldwide promote the use of more of them for all types of treatments. Investigating the quality, safety, and effectiveness of plant therapies is crucial because many medicinal plants are now being used to make contemporary therapeutic medications. Parts of medicinal plants should be genuine and free of contaminants such as pesticides, heavy metals, microbes, radioactive elements, etc. (Kamboj 2000). For all plant medicines, standardization is crucial. In several resolutions, the WHO assembly has underlined the necessity of employing contemporary methods and appropriate standards to guarantee the quality control of medicinal plant products (Raina 2003; Ahmad et al. 2006). Some of the standardization tests for herbal medicines are listed below (Ritch 2000).

- Macro and microscopic examination: For identification of right variety and search of adulterants.
- Foreign organic matter: Removal of foreign organic matter other than source plant to get the drug in pure form.
- Ash values: It is the criteria to judge the identity and purity of crude drug – Total ash, sulfated ash, water soluble ash and acid insoluble ash etc.
- Moisture content: To check moisture content, which helps is in prevent degradation of product.
- Extractive values: These are indicating the approximate measure of chemical constituents of crude drug.
- Crude fiber: To determine excessive woody material criteria for judging purity.

- Qualitative chemical evaluation: It covers identification and characterization of crude drug with respect to phytochemicals constituent.
- Quantitative chemical evaluation: To estimate amount the major class of constituents.

- Toxicological studies: Pesticide residue, potentially toxic elements, and microbial count approach to minimize their effect in final product.

Medicinal plants used for anti-inflammatory activity

Many therapeutic plants have possessed anti-inflammatory property. Numerous researchers have studied a variety of medicinal plants, and they have used the models listed in **Table 1.1** to verify their anti-inflammatory properties. According to Deepa and Renuka Devi (2014), carrageenan, formaldehyde, serotonin, dextran, and histamine-induced paw edema are frequently employed to identify the acute phase of inflammation. Among the several models, the Carrageenan model is frequently used to assess the anti-inflammatory potential of medicinal plants. Cotton pellet and Freund's full adjuvant models are used to assess the chronic phase of inflammation.

Table 1.1 List of medicinal plants used for anti-inflammatory activity

S. No	Name of the plant	Part Used	Type of plant extract	Model used
1.	*Acacia modesta*	Leaves	Methanol	Carrageenan
2.	*Adenenthera pavonina*	Leaves	Ethanolic	Carrageenen Cotton pellet
3.	*Albizia lebbeck*	Bark	Petroleum ether Chloroform Ethanol	Carrageenan Dextran Cotton pellet Freund ‘s complete adjuvant
4.	*Alpinia galangal*	Root	Alcohol	Carrageenan Bradykinin Formaldehyde
5.	*Alstonia scholaris*	Leaves	Ethanol	Carrageenan
6.	*Andrographis paniculata*	stem	Chloroform	Carrageenan
7.	*Argyreia speciosa*	Root	Methanol	Carrageenan
8.	*Balanites aegyptiaca*	Aerial Parts	Petroleum ether Ethanol	Carrageenan
9.	*Bambusa vulgaris*	Leaves	Methanol	Carrageenen Cotton Pellet
10.	*Barleria cristata*	Leaves	Aqueous	Carrageenan
11.	*Bauhinia purpurea*	Stem	Ethanol	Carrageenan
12.	*Boswellia serrate*	oleo-gum- rein	Petroleum ether Aqueous	Carrageenan
13.	*Bowdichia virgilioides*	Bark Leaves	Aqueous	Carrageenan
14.	*Calotropis*	Latex	Aqueous	Carrageenan

	procera			
15.	*Camellia sinensis*	Leaves	Aqueous Aqueous	Carrageenan Arachidonic Acid Carrageenan Histamine Serotonin Prostaglandin
16.	*Carum copticum*	Seeds	Alcohol, Aqueous	Carrageenen, Cotton Pellet
17.	*cassia fistula*	Bark	Aqueous Methanol	Carrageenan
18.	*Chloranthus eretus*	Leaves	Methanol	Carrageenan Histamine Serotinin Cotton Pellet
19.	*Crotala-ria juncea*	Leaves	Ethanol	Freund 's complete adjuvant
20.	*Dillenia indica*	Leaves	Methanol	Carrageenan
21.	*Dregea volubilis*	Leaves	Methanol Petroleum ether	Carrageenan
22.	*Drimys angustifolia*	Leaves, Stem bark	Aqueous	Carrageenan
23.	*Ecbolium viride*	Root	Ethanol	Carrageenan Cotton pellet
24.	*Ficus amplissima*	Bark	Acetone	Carrageenan Histamine
25.	*Ficus bengalensis*		Petroleum ether Ethanol	Carrageenan
26.	*Foeniculum vulgare*	Fruit	Methanol	Carrageenan Arachidonic acid Formaldehyde
27.	*Gynadropsis pentaphylla*		Aqueous	Carrageenan
28.	*Holoptelea integrifolia*	Leaves	Aqueous	Carrageenan
29.	*Hygrophila spinosa*	Leaves	Petroleum ether Chloroform Ethanol Aqueous	Carrageenan
30.	*Hypericum Rumeliacum*	Aerial parts	Ethanol	Carrageenan
31.	*Kalanchoe crenata*	Leaves	Hexane Ethyl acetate, N- Butanol Aqueous	Carrageenen Histamine Serotonin Formalin
32.	*Lamiophlomis Rotate*	Aerial parts	Aqueous	Carrageenan
33.	*Lantana trifolia*	Leaves	Ethanol	Carrageenan Histamine Serotonin
34.	*Leonotis Nepetaefolia*	Leaves Stem Flowers	Hexane Ethylacetate, Methanol	TPA
35.	*Leucas aspera*	Aerial parts	Ethanol	Formalin
36.	*Lotus pedunculat*	Aerial parts	Ethanol	Carrageenan
37.	*Mallotus Philippinensis*	Stem wood	Ethanol	Formaldehyde Carrageenan
38.	*Malva parviflora*	Stem	Methanol	Carrageenan Histamine
39.	*Mitragyna parvifolia*	Leaves	Ethanol	Carrageenan
40.	*Monochoria vaginalis*	Roots, Leaves	Methanol	Carrageenan
41.	*Moringa oleifera*	Leaves	Aqueous	Carrageenan
42.	*Mortonia greggii*	Roots, Leaves	Acetone	Carrageenan TPA
43.	*Nothospondias staudtii*	Leaves	Aqueous Methanol Chloroform	Carrageenan
44.	*Onosma aucheranum*	Root	Chloroform	Carrageenan
45.	*Onosma isauricum*	Root	Chloroform	Carrageenan
46.	*Onosma sauricum*	Root	Ethanol	Carrageenan
47.	*Onosma sericeum*	Root	Ethanol	Carrageenan
48.	*Passiflora foetida*	Leaves	Ethanol	Carrageenan Histamine
49.	*Pedilanthus Tithymaloides*	Stem, Leaves	Tincture	Carrageenan

50.	*Petroselinum Crispum*	Leaves	Ethanol	Carrageenan Cotton pellet
51.	*Pimenta racemosa*	Leaves	Aqueous	Carrageenan TPA
52.	*Pinus densiflora*	Pollen	Ethanol	Carrageenan Formalin Arachidonic Acid
53.	*Piper sp.*	Leaves	Methanol	Carrageenan Cottonpellet Dextran
54.	*Piper longum*	Fruit oil	Methanol Aqueous	Carrageenan
55.	*Piper sarmentosum*	Leaves	Aqueous	Carrageenan
56.	*Plantago majo*	Seeds	Methanol	Carrageenan
57.	*Plumeria Acuminate*	Leaves	Methanol	Carrageenan Dextran Histamine Serotonin Cotton pellet
58.	*Polyalthia Longifolia*	Leaves	Toluene, Chloroform Acetone Methanol	Carrageenan
59.	*Punica granatum*	Flower	Petroleum ether dichloromethane methanol	Carrageenan
60.	*Phyllanthus amarus*	Whole plant	Methanol	Carrageenan Cotton pellet
61.	*Phyllanthus emblica*	Fruit	Aqueous	Carrageenan Cotton pellet
62.	*Rivea hypocrateriformis*	Leaves	Ethanol	Carrageenan
63.	*Rungia pectinata*	Leaves	Hydroalcoholic	Carrageenen
64.	*Rungia repens*	Leaves	Hydroalcoholic	Carrageenan
65.	*Rubia cardifolia*	Root Stem	Petroleum Ether Ethanol	Carrageenan Freund'sdjuvant Carrageenan
66.	*Sapindus trifoliatus*	Pericarp	Aqueous	Carrageenan Histamine Serotonin ArachidonicAcid TPA
67.	*Schima wallichii*	Bark	Ethanol	Carrageenan Dextran Cotton pellet
68.	*Sclerocarya birrea*	Stem bark	Aqueous Methanol	Carrageenan Histamine Serotonin Freund's Adjuvant
69.	*Semecarpus anacardium*	Nut	Milk	Carrageenan Freund's Adjuvant
70.	*Silybum marianum*	Leaves	Methanol	Carrageenan
71.	*Simmondsia chinensis*	Seed	Methanol	Carrageenan
72.	*Smilax china*	Whole Plant	Methanol	Carrageenan
73.	*Solanum nigrum*	Berries	Methanol	Carrageenan
74.	*Solanum trilobatum*	Root	Methanol	Carrageenan
75.	*Sonchus oleraceus*	Aerial parts	Ethanol	Carrageenan cotton pellet
76.	*Spilanthes acmella*	Aerial parts	Aqueous	Carrageenan
77.	*Tabernaemontana Catharinensis*	Bark	Ethanol	Carrageenan
78.	*Terminalia arjuna*	Bark Leaves	Methanol	Carrageenan carrageenan, histamine dextran
79.	*Thespesia populnea*	Bark	Methanol	Carrageenan Histamine Serotonin
80.	*Tephrosia purpurea*	Gum	Ethanol	Carrageenan Cotton pellet
81.	*Trachelospermum Jasminoides*	Leaves	Aqueous	Carrageenan
82.	*Trichodesma Indicum*	Root	Chloroform	Carrageenan Histamine Dextran Serotonin Cotton

				pellet
83.	*Ventilago*	stem bark	Methanol	Ethyl Phenylpropiolate
84.	*V.negundo*	Leaves Leaves Leaves Leaves	Aqueous Methanol Petroleum Ether	Carrageenan Carrageenan Carrageenan cotton pellet
85.	*Zingiber officinale*	Rhizome	Hydroalcoholic	Carrageenan Serotonin

Many researchers have isolated a greater number of phytocompounds from various medicinal plants and reported as anti-inflammatory agents. These phytocompounds inhibit the anti-inflammatory mediators. The main target was Cyclooxygenase (COX) enzyme (Menozzi et al. 2009; Niu et al. 2011). The target protein inhibited by various phytocompounds are given in the **Table 1.2** (Aggarwal et al. 2011).

Compounds	**Source**	**Common Name**
Boswellic acid	Boswellia serrata	Salai Guggul
Berberine	Berberis vulgaris	Barberry
Celastrol	Tripterygium wilfordii	Thunder God Vine
Cucurbitacin	Cayaponia tayuya	Tayuya
Curcumin	Curcuma longa	Turmeric
Eugenol	Syzygium aromaticum	Cloves
Guggulsterone	Commiphora mukul	Guggul
Genistein	Glycine max	Soybeans
Luteolin	Thymus vulgaris	Thyme
Morin	Chlorophora tinctoria	Fustic
Quercetin	Quercetin Allium cepa	Onions
Resveratrol	Vitis vinifera	Red Grapes
Rosmarinic acid	Rosmarinus officinalis	Rosemary
Silymarin	Silybum marianum	Milk Thistle
Tea polyphenols	Camellia sinensis	Black Tea
Ursolic acid	Ocimum sanctum	Holy Basil
Withanolides	Withania somnifera	Ashwagandha

(Courtesy Aggarwal et al. 2011)

Conclusion –

Role of Inflammation: Inflammation is a critical process that can determine the fate of tissues following injuries. It involves several components, including edema formation, leukocyte infiltration, and granuloma formation, which are essential for the healing process but can also lead to tissue destruction if uncontrolled Impact of Reactive Oxygen Species (ROS): Excess ROS production has been linked to tissue damage and has been implicated in the pathogenesis of several diseases, such as rheumatoid arthritis, diabetes, Alzheimer's disease, and cancer. This emphasizes the dual role of ROS as signaling molecules in normal physiological processes and as contributors to pathological conditions when produced in excess. Consequences of Oxidative Stress: The paper discusses how oxidative stress, which is caused by an imbalance between ROS production and antioxidant defenses, can cause significant cellular damage, underscoring the significance of redox homeostasis for cellular health and function. Clinical Implications: Knowledge of oxidative stress and inflammation pathways can help identify possible treatment targets for autoimmune disorders and other inflammatory ailments. This information is essential for creating plans to reduce tissue damage and enhance patient outcome

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