

“A Review on Anti-Inflammatory Activity of Medicinal Plant”

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ABSTRACT

Medicinal plants and their secondary metabolites are progressively used in the treatment of diseases as a complementary medicine. Inflammation is a pathologic condition that includes a wide range of diseases such as a rheumatic arthritis and immune mediated conditions ,diabetes ,cardiovascular diseases and etc. We introduced some herbs which their anti-inflammatory effects have been evaluated in clinical and experimental studies .Since the treatment of inflammation is not a one dimensional remedy, this review tries to reach a multidimensional therapeutic approach to inflammation with the help of herbal medicine along with modification in lifestyle. So there is need of scientific and evidence based screening of ayurvedic preparations using various animal models for their in vivo and in vitro studies by taking into considerations ayurvedic preparations will be the best options to cure the disease. Study reflects pathology of inflammation and various mechanisms of Anti-inflammatory activity of various ayurvedic herbal and synthetic allopathic medicines. review will help in selection of appropriate model for studying Anti-inflammatory activity .

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I. INTRODUCTION

Ayurvedic medicine is an ancient holistic healthcare system from India, focusing on balancing the mind, body and spirit through methods like herbal remedies, diet, exercise and meditation to achieve wellness and prevent illness .The term “Ayurveda” combines Ayu(life) and Veda(knowledge) ,reflecting its goal to provides holistic guidance for maintaining health and life . The aim of this review article is to compare and contrast the two system of medicine with special reference to their efficiency and limitation .

AYURVEDA

Ayurveda is a traditional system of medicine that has its roots in ancient India. It is a holistic approach to health and wellness that seeks to balance the body, mind, and spirit. Ayurvedic medicine is based on the belief that good health is the result of a delicate balance between the body, mind, and spirit. The word "Ayurveda" comes from two Sanskrit words, "ayus" meaning life and "veda" meaning knowledge. This system of medicine is based on the idea that the body has a natural ability to heal itself, and that the key to good health is to support this natural healing process. Ayurveda recognizes that each person is unique, and therefore each person's health needs are different. The system of medicine focuses on promoting health rather than treating disease, and places a strong emphasis on prevention. Ayurvedic practitioners use a variety of techniques to restore balance to the body, including dietary changes, herbal remedies, massage, and yoga. Overall, Ayurveda offers a holistic and personalized approach to health and wellness. Its emphasis on prevention, natural healing, and individualized care makes it a popular choice for those seeking alternative and complementary healthcare options.

ALLOPATHY

Allopathy, also known as modern or Western medicine, is a system of medicine that uses drugs, surgery, and other interventions to treat disease and promote health. It is based on the principles of evidence-based medicine, which means that medical decisions are made based on scientific research and clinical trials. The term "allopathy" was coined by Samuel Hahnemann, the founder of homeopathy, to distinguish modern medicine from his own system of medicine. However, the term is not commonly used by practitioners of modern medicine today. Allopathic medicine is focused on diagnosing and treating specific diseases and conditions using drugs and other interventions. It is based on the idea that diseases are caused by specific pathogens or abnormalities in the body, and that the goal of treatment is to eliminate or manage these causes.

Allopathic medicine uses a variety of tools and techniques to diagnose and treat disease, including diagnostic tests, medications, surgery, and other medical interventions. It also emphasizes preventive care, such as vaccinations and regular health screenings, to help individuals maintain good health and prevent the onset of disease. Overall, allopathic medicine is a highly structured and scientific approach to healthcare that relies on evident treatments and interventions to promote health and treat disease. It is widely used around the world and is often the first choice for individuals seeking medical care on urgent basis but Allopathic medicines have risk of potential hazardous effects on body like liver damage, kidney damage, risk of carcinogenicity, etc on their chronic use. Geriatric , paediatric patients and pregnant women are at high risk due to side effect of Allopathic medicines.

ANTI-INFLAMMATORY ACTIONS

Inflammation is a pathophysiological response of living tissue to injuries that leads to the local accumulation of plasmic fluid and blood cells. The complex event and mediators involved in the inflammatory reaction can induce, maintain or aggravate many disease. However, many studies have been continuing and inflammatory disease and the side effect of the currently available anti-inflammatory drug are the major problem during their clinical uses. The most commonly used drug for management of inflammatory condition are non-steroidal anti-inflammatory drug (NSAIDs), which have several adverse effect especially gastric irritation leading to formation of gastric ulcer. Therefore, nowadays the development of newer and more substantial anti-inflammatory drugs with lesser side effect is necessary. For this reason, in recent time, more interest is shown in alternative and natural drugs for treatment of various diseases, but there is lack of scientific evidence.

Sr No.	NAME OF DRUG	BIOLOGICAL SOURCE/FAMILY	PART USED	CHEMICAL CONSTITUENT	USES
1)	Nirgudi	Vitex Negundo FAMILY: Verbenaceae	Leaves, flower, Bark.	Alkaloids, tannins, saponins, phenolics, Flavonoids,	Anti-inflammatory activity
2)	Ajwain	Trachyspermum ammi FAMILY: Apiaceae	fruit, seeds	P-cymene (30.8%) Terpinene (23.2%) Pinene (1.7) Terpinene-4-ol (0.8%)	Infection, Cough, cold
3)	Akarkara	Anacyclus Pyrethrum FAMILY: Asteraceae	Roots,	Alkaloid, tannins, flavonoids Sterol, phenols, etc	Inflammation, Oral health
4)	Almond	Prunus amygdalus FAMILY: Rosaceae	Dried ripe seeds	Fixed oil, glycosides, amygdalin.	Skin care, hair care,
5)	Bel	Maemelos linn FAMILY: Rutaceae	Ripe fruits	Palmitic, stearic, oleic, linoleic and linolenic acid	Anti-oxidant, Anti-bacterial
6)	Bhringaraj	Eclipta alba FAMILY: Asteraceae	Roots, stem, leaves	Alkaloids, Flavonoids, Saponins, Sterol	Digestion, nervous system, asthma
7)	Coleus	Coleus scutellarioides FAMILY: Lamiaceae	Root	Alkaloids, flavonoids, Tannins, saponins, glycosides.	Heart disease, Respiratory disorder, cardiac disease
8)	Coriander	Coriandrum sativum linn FAMILY: Umbelliferae	Dried ripe fruits	Carbohydrates, proteins, vitamin, flavonoids	Anti-inflammatory, Anti-oxidant
9)	Datura	Datura metel FAMILY: Solanaceae	Seeds, flower	Hyscymilato, scopolin, umkalin, daryolone	Analgesic, anti-inflammatory
10)	Fennel	Foeniculum vulgare FAMILY: Apiaceae	fruit, seeds	Trans-anethole, 2-pentanone	Digestion, cough, vom-iting
11)	Ginger	Zingiber officinale FAMILY: Zingiberaceae	Rhizomes	Volatile oil, bisabolene, zingiberene, Zingiberol	Anti-microbial, Flavoring agent, Anti-inflammatory
12)	Gudhal	Hibiscus FAMILY: Malvaceae	Flower	Protein, carbohydrate, Thiamine, riboflavin	Anti-aging, hair care, pigmentation.
13)	Jamun	Syzygium cumini FAMILY: myrtaceae	Fruit	Anthocyanins, peonidin, Delphinidin acetyl-diglycoside	Diabetes, inflammation
14)	Kasturi bhindi	Abelmoschus moschatus FAMILY: Malvaceae	Flowers, seeds, Leaves	Fixed oil, volatile oil, linolenic acid Plasmalogen, beta-sitosterol, myricetin	Anti-pyretic, Fever, Nervous system.
15)	Lily	Lilium speciosus	Leaves, flower	Alkaloids saponins, sterol,	Skin care, anti-

		FAMILY:Liliaceae		Polysaccharides.brownii	Inflammation
16)	Neem	Azadirachta Indica FAMILY:Meliaceae	Leaves ,seeds	Azadirachtin,nimbolinin ,nimbdin,sodium nimbinat,salannin etc	Inflammation, fever,skin disorder
17)	Papaya	Carica papaya FAMILY: caricaceae	Fruit,seeds	Vlt.A,B,C,proteolytic enzyme,isoithiocynate,etc	GIT disorders ,sedative
18)	Patatao	Solanum tuberosum FAMILY: solanaceae	Starchy tuber	Saturated fatty acid,vit.C,protein ,carbohydrates	Skin-on or pelled,p-otato chips,food
19)	Rauwolfia	Rauwolfia serpentine FAMILY: Apocynaceae	Dried roots,rhizomes	Alkaloids respine,ajmalin ,serpentine, serpentinine	Anti-hyperte nsive,angi na pectoris
20)	Sandal food	Santalum album linn FAMILY: Santalaceae	Stem, roots	Volatile oil,alpha-santalola,beta-santalol	Disinfecatant ,perfumery,Anti-microbial
21)	Termeric	Curcuma longa FAMILY: zingiberaceae	Dried rhizomes	Curcumin ,volatile oil,resin	Anti-inflammtory, Colouring agent
22)	Vajradanti	Barleria prionitis FAMILY:Acanthaceae	whole plant	Alkaloids,flavonoids ,glycosides,phonolics,saponins	Anti-inflam Matory,anti-fertility
23)	Vinca	Catheranthus rosenus FAMILY: Apocynaceae	Leaves ,flower	Alkaloids,vincristine,vinblastin , Serpentine,azamalaine	Anti-cancer

ANIMAL MODELS FOR INFLAMMATION

- 1) Paw edema in rats (various modifications and various irritants)
- 2) UV-erythematic in guinea pig
- 3) Vascular permeability
- 4) Oxazolone-induced ear edema in mice
- 5) Croton-oil ear edema in rats and mice
- 6) Pleurisy tests
- 7) Granuloma pouch technique (various modifications and various irritants)

1)CARRAGEENAN INDUCED PAW EDEMA MODEL:

Carrageenan – induced paw edema is the most commonly used method in experimental pharmacology. Carrageenan is a sulphated polysaccharide obtained from seaweed (Rhodophyceae), and by causing the release of histamine, 5-HT, bradykinin and prostaglandins it produces inflammation and edema.

PROCEDURE:

- 1) Albino wistar rats weighing between 150-200 gms were divided into 5 groups of 6 rats in each group.
- 2) Three animals being housed in labelled cage each .
- 3) Animals were given a period of time to adjust to a new environment provided with food and water.
- 4) The test compound and standard drugs are administered by oral or intra-peritoneal route.
- 5) Thirty minutes later, the rats are challenged by a subcutaneous injection of 0.05 ml of 1% solution of carrageenan into the plantar side of the left hind paw.
- 6) The paw is marked with ink at the level of the lateral malleolus and immersed in mercury column of plethysmometer for measuring the paw volume.
- 7) The paw is measured immediately after the carrageenan injection and then at 2, 3, 4 and 6 hours. The peak effect of carrageenan usually occurs at 3 hours after the injection .

2) ULTRAVIOLET ERYTHEMA IN GUINEA PIGS:

UV augments blood flow and infiltration by blood leukocytes, such as macrophages and neutrophils into the skin, observed clinically as inflammation. Increased production of NO and prostaglandins contribute to these events radiation-induced lipid peroxidation increases production of prostaglandins (pg), including pge2, which in turn cause inflammation in the skin.

PROCEDURE:

- 1) Albino guinea pigs of both sexes with an average weight of 350g are used.
- 2) Four animals are used each for treatment and control group.

- 3) Eighteen hr prior testing, the animals are shaved on both the flanks and on the back. Then they are chemically depilated by a commercial depilation product or by a suspension of barium sulphide.
- 4) Twenty min later, the depilation paste and the fur are rinsed off in running warm water. On the next day the test compound is dissolved in the vehicle and half of the test compound is administered by gavage (at 10 ml/kg) 30 min before UV exposure.
- 5) Control animals are treated with the vehicle alone. The guinea pigs are placed in a leather cuff with a hole of 1.5×2.5 cm size punched in it, allowing the UV radiation to reach only this area. Then animals are exposed to UV radiation.
- 6) After 2 min of expose the remaining half of the test compound is administered. The erythema is scored 2 and 4 hr after exposure.

3) VASCULAR PERMEABILITY:

During inflammation, vascular permeability increases to allow plasma constituents such as antibodies and complement to access injured or infected tissues. The test is used to evaluate the inhibitory activity of drugs against increased vascular permeability which is induced by phlogistic substances. Mediators of inflammation, such as histamine, prostaglandins and leucotrienes are released following stimulation e.g. of mast cells. This leads to a dilation of arterioles and venules and to an increased vascular permeability. As a consequence, fluid and plasma proteins are extravasated and edemas are formed. The increase of permeability can be recognized by the infiltration of the injected sites of the skin with the vital dye Evan's blue.

PROCEDURE:

- 1) Albino Wistar are used each group containing 4 rats.
- 2) Control group will receive distilled water 1%w/v 1ml/100g by oral route and other group will receive test compound by oral route and standard group will receive diclofenac 10ml/kg by intra-peritoneal route.
- 3) After 1h of these administration rats are injected with 0.25ml of 0.6% v/v solution of acetic acid intraperitoneally.
- 4) Immediately, 10 ml/kg of 10%w/v Evans blue is injected intravenously via tail vein. After 30 min, the animals are anesthetized with ether anaesthesia and sacrificed.
- 5) The abdomen is cut open and exposed viscera. The animals are held by a flap of abdominal wall over a Petri dish. The peritoneal fluid (exudates) collected, filtered and made up the volume to 10 ml using normal saline solution and centrifuged at 3000 rpm for 15 min.
- 6) The absorbance (A) of the supernatant is measured at 590 nm using spectrophotometer.

4) OXAZOLONE-INDUCED EAR EDEMA IN MICE:

The oxazolone-induced ear edema in mice is a model of delayed contact hypersensitivity that permits the quantitative evaluation of the topical and systemic anti-inflammatory activity of a compound following topical administration. The oxazolone-repeated challenge increased the level of Th2 cytokines and decreased that of a Th1 cytokine in the lesioned skin. The Th2 cytokines, especially IL-4, play major roles in the development of dermatitis in the present mouse model.

PROCEDURE:

- 1) Each contains 12 mice, the same skin site of the right ear was sensitized by a single application of 10 µl (each 5 µl for inner and outer of ear) of 0.5% oxazolone in acetone 7 days before the first challenge (day 0), and 10 µl of 0.5% oxazolone in acetone was repeatedly applied to the sensitized right ear 3 times per week.
- 2) The only acetone will be applied to the right ear. The mice are challenged 8 days later again under anesthesia by applying 0.01 ml 2% oxazolone solution to the inside of the right ear (control) or 0.01 ml of oxazolone solution, in which the test compound or the standard is solved.
- 3) One group of animals are treated with the irritant alone or with the solution of the test compound. The left ear remains untreated.
- 4) The maximum of inflammation after 24 hour.
- 5) At this stage the animals are sacrificed under anaesthesia and a disc of 8 mm diameter is punched from both sides. The discs are immediately weighed on a balance. The weight difference is an indicator of the inflammatory edema.

5) CROTON-OIL EAR EDEMA IN RATS AND MICE:

Croton oil contains 12-o-tetradecanoylphorbol-13-acetate (TPA) and other phorbol esters as main irritant agents. TPA is able to activate protein kinase C (PKC), which activates other enzymatic cascades in turn, such as mitogen activated protein kinases (MAPK), and phospholipase A2 (PLA2), leading to release of platelet activation factor (PAF) and AA. This cascade of events stimulates vascular permeability, vasodilation, polymorphonuclear leukocytes migration, release of histamine and serotonin and

moderate synthesis of inflammatory eicosanoids by cyclooxygenase (COX) and 5-lipoxygenase (5-LOX) enzymes. COX and 5-LOX inhibitors, leukotriene B₄ (LTB₄) antagonists and corticosteroids show topical anti-inflammatory action in animal models of Croton oil or TPA induced skin inflammation.

PROCEDURE:

- 1) Six animals are used each for treatment and control group.
- 2) The total of 15 µl of an acetone solution containing 75 µgm of croton oil is applied to the inner surface of right ear of each mouse. Left ear remains untreated.
- 3) Control animals receive only the irritant while indomethacin (100 µg/ear) serves as reference.
- 4) Different dose levels of test compounds are applied to the inner surface of right ear of each mouse by dissolving them in inflammation inducing solution.
- 5) Animals are sacrificed by cervical dislocation after 6 hours and a plug (6 mm in diameter) is removed from both the treated and untreated ear.
- 6) The difference in weight between the two plugs is taken as measure of edematous response. Since tetradecanoyl phorbol acetate (TPA) is the chief ingredient of croton oil, purified TPA has also been used to induce ear edema in mice.

6) PLEURISY TESTS:

In experimental animals pleurisy can be induced by several irritants, such as histamine, bradykinin, prostaglandins, mast cell degranulators, dextran, enzymes, antigens, microbes, and nonspecific irritants, like turpentine and carrageenan. Carrageenan-induced pleurisy in rats is considered to be an excellent acute inflammatory model in which fluid extravasation, leukocyte migration and the various biochemical parameters involved in the inflammatory response can be measured easily in the exudates.

PROCEDURE:

- 1) The mouse pleurisy was induced by a single intrapleural injection of 0.1 ml of carrageenan (1%).
- 2) After 4 h the animals were killed with an overdose of ether, the thorax was opened and the pleural cavity was washed with 1.0 ml of sterile PBS, containing heparin (20 IU per ml).
- 3) Samples of the pleural lavage were collected for determination of exudation, myeloperoxidase, adenosine-deaminase activities, and nitric oxide levels, as well as for determination of total and differential leukocyte counts. Total leukocyte counts
- 4) were performed in a Neubauer chamber. The cytospin preparations of pleural wash were stained with May-Grunwald Giemsa for the differential count which was performed under an oil immersion objective.
- 5) The serum level of the Creative protein was also analysed.
- 6) In another set of experiment animals were treated 30 min before carrageenan with a solution of Evans blue dye (25 mg/kg, i.v.) in order to evaluate the degree of exudation in the pleural space.
- 7) A sample (500 µl) of the fluid leakage collected from the pleural cavity was stored in a freezer (-20 °C) to further determine the concentration of Evans blue dye.
- 8) To this end, on the day of the experiments, a batch of samples was thawed at room temperature and the amount of dye was estimated by colorimetry using an Elisa plate reader at 600 nm, by interpolation from a standard curve of Evans blue dye in the range of 0.0 to 50 µg/ml.

7) GRANULOMA POUCH TECHNIQUE:

It is one of the classical methods for measuring subacute inflammation in rats. Earlier croton oil, Freund adjuvant and lipo-polysaccharide were used in rats which led to granuloma formation. This replaced by carrageenan as a phlogistic agent for the induction of granuloma.^[24] With the introduction of an irritant substance into an s.c. air pocket, granulation tissue begins to proliferate and soon covers the whole inside of the pouch. This tissue consists of fibroblasts, endothelial cells and an infiltrate of macrophages and polymorphonuclear leukocytes.

PROCEDURE:

- 1) Male or female Sprague-Dawley rats with a body weight between 150 and 200 g are used.
- 2) Ten animals are taken for controls and for test groups. The back of the animals is shaved and disinfected.
- 3) With a very thin needle a pneumoderma is made in the middle of the dorsal skin by injection of 20 ml of air under ether anaesthesia.
- 4) Into the resulting oval airpouch 0.5 ml of a 1% solution of Croton oil in sesame oil is injected avoiding any leakage of air.
- 5) Forty-eight hours later the air is withdrawn from the pouch and 72 h later any resulting adhesions are broken. Instead of croton oil 1 ml of a 20% suspension of carrageenan in sesame oil can be used as irritant.

- 6) Starting with the formation of the pouch, the animals are treated every day either orally or subcutaneously with the test compound or the standard. For testing local activity, the test compound is injected directly into the air sac at the same time as the irritant.
- 7) On the 4th or the 5th day the animals are sacrificed under anaesthesia. The pouch is opened and the exudate is collected in glass cylinders.
- 8) Controls have an exudates volume between 6 and 12 ml, which is reduced dose dependent in the treated animals.

MODE OF ACTION OF ANTI-INFLAMMATORY ACTIVITY

Process of inflammation starts when allergen, chemical irritation, infection and injury occur in body, chemical mediators released from mast cells, platelets, neutrophils, macrophages and lymphocytes. These chemical mediators are also called pro-inflammatory factors which released when membrane phospholipid converted to arachidonic acid in the presence of phospholipase A. From arachidonic acid prostaglandins, leukotrienes and cytokines are released in the presence of cyclooxygenase and lipoxygenase. Prostaglandins cause capillary dilation and increases blood flow. Leukotrienes cause degranulation, promote adhesion of neutrophils to vascular endothelial cells and produce superoxide. Cytokines (IL-1, IL-2, IL-3, IL-4, IL-5, IL-6, IL-8, IL-10, IL-12, TNF) inhibit viral replication, inhibit cell proliferation, increase tissue destruction and lytic activity of natural killer cells.

Anti-inflammatory mechanism of medicinal plants

Medicinal plants anti-inflammatory actions encompass a variety of routes and components that assist regulate and modulate the inflammatory response. Here are some examples of common mechanisms:

Inhibition of lipoxygenase:

Lipoxygenase is used in production of leukotrienes from arachidonic acid which are pro-inflammatory mediators. Through different parts of plants by inhibiting lipoxygenase inflammation is inhibited.

Inhibition of Nitric Oxide:

Many plant's flavonoids inhibit the production of nitric oxides. Nitric oxides are free radicals that involved in formation of inflammation by cytokines activated macrophages. These are also called pro-inflammatory mediators.

Inhibition of cyclooxygenase:

Many herbal derived compounds inhibit the biosynthesis of prostaglandins by two types of enzymes cyclooxygenase 1 and cyclooxygenase 2 inhibitions. Prostaglandins are inflammatory mediators. Prostaglandins are of 4 types including PGE₂, PGI₂, PGD₂, and PGF₂.

Prostaglandins play role in inflammation by causing classical sign of inflammation which are pain, redness and swelling. Redness and edema is caused by increase blood flow, increasing permeability and vasodilation. Pain occurs due to the effect of prostaglandins on sensory neuron and central site.

Inhibition of phospholipase:

From membrane lipid phospholipase, arachidonic acid is released. Arachidonic acid is a precursor of eicosanoid from which prostaglandins are synthesized. The inhibition of phospholipase plays key role in treatment of inflammation.

Inhibition of pro-inflammatory cytokines:

Some parts of plant inhibit inflammation by inhibiting Pro-inflammatory cytokines. Pro-inflammatory cytokines are signaling molecules, released from immune cells predominantly from T-cells and macrophages. Pro-inflammatory cytokines cause inflammation as IL-1 β , IL-6: involve in regulation of apoptosis in cell and TNF- α : regulate process of apoptosis by effecting in different signals pathway.

Modulation of pro-inflammation expression gene:

Many plant flavonoids inhibit the modulation of gene expression for isomers of nitric oxides, cyclooxygenase and lipoxygenase that play role in production of prostaglandins, leukotrienes and other inflammatory mediators e.g. cytokines and chemokines by inhibition of protein kinases and transcription factors which are nuclear factor Kappa B, activator protein 1, signal transducers and binding proteins.

APPROACHES OF PHARMACEUTICAL DEVELOPMENT OF ANTI-INFLAMMATORY ACTIVITY

1)Biorational Approach:

The biorational approach uses the information derived from biological specimens that are used either in traditional medicines or have ecological functions in an organism/plant to develop drugs. This approach includes two search strategies:

- (a) Ethnobotanically/ethnopharmacologically directed strategy and
- (b) ecologically directed strategy

2)Ethnopharmacology Approach:

This is a field of study that derives therapeutic agents from plants and animals traditionally used by people to treat various diseases. Ethnopharmacology is considered a straightforward approach to drug discovery as it uses indigenous knowledge for the targeted isolation of drug leads. Different cultures have invented their own ways of healing systems, and they exist mainly in two forms—scholarly traditional medical system (selected examples are Indian Ayurvedic medicine, Chinese traditional medicine, and Bhutanese Sowa Rigpa medicine) and oral/folklore medical system (selected examples are Australian aboriginal medicine, Kenyan medicine). Both medical systems contain enormous ethnopharmacological and ethnobotanical information that is resourceful for drug discovery and development. Intuitively, the success rate of drug discovery using ethnopharmacological data is expected to be greater than with the random approach. This approach has resulted in isolating several bioactive compounds, including anti-inflammatory.

(b) Ecological Approach:

This search strategy is driven by the chemical interaction of plants or organisms with their environmental factors, facilitated by and is also called chemical ecology. The secondary metabolites produced by organisms in response to interaction with their surroundings have mainly defensive roles and most of these defensive metabolites are reported to have diverse bioactive chemical constituents. Thus, this knowledge is often used to select starting materials not associated with ethnopharmacological knowledge in drug discovery.

2)Chemorational Approach:

The chemorational approach in drug discovery is grounded on chemotaxonomic information, including cheminformatics of a botanical specimen and targeted phytochemical class assessments. In this approach, preliminary evaluation of chemical classes and examining families and genera known for their analogous compounds with prior biological activities are necessary to increase the chance of getting the targeted drug lead. Studies suggest that species belonging to the same genus or closely related genera tend to yield compounds with similar chemical structures.

3)Random Approach/Find-and-Grind Approach:

This approach involves collecting samples randomly and screening them for any biological hits. The selected NPs, following a random approach, are usually screened for the desired chemical classes, including flavonoids, terpenoids, phenols, steroids, and alkaloids, and their bioactivities, if any. This approach is often known as “find and grind”, which involves randomly selecting NPs without prior ethnopharmacological, ecological or chemical information. It primarily involves random high-throughput screening (HTS) of crude extracts for phytochemical and biological activity. Based on biological hits, the best samples are selected for the further isolation and characterisation of drug leads.

The success rate of this approach in generating drug leads is comparatively lower than it is for the other two approaches. For instance, the National Cancer Institute (NCI) in the United States randomly screened 35,000 plant species in search of anti-cancer agents and discovered only two drug leads, paclitaxel and camptothecin.

AYURVEDIC PREPARATION AND MARKETING PREPARATION

Ayurvedic Preparation:

1. Ingredient Selection: Specific herbs known for anti-inflammatory properties are chosen. Common examples include:

Guduchi (*Tinospora cordifolia*): Used to make Guduchi Ghana, an aqueous extract of the stem.

Triphala: A blend of three fruits, known for its antioxidant and anti-inflammatory effects.

Turmeric (*Curcuma longa*): A potent anti-inflammatory agent, often combined with black pepper to enhance absorption.

Guggul (*Commiphora mukul*): The oleo-resin fraction of Guggul has significant anti-inflammatory and anti-arthritic properties.

2. Extraction & Decoction:

Aqueous Extracts/Decoctions: Plant materials are often steeped in water to create decoctions or concentrated extracts.

Soxhlet Extraction: For some formulations, sequential extraction with solvents like hexane and ethanol is performed to isolate different compounds, as seen in the preparation of extracts for testing.

Specific Preparations:

Śīrīṣāvaleha: A preparation from Śīrīṣā (*Albizia lebbek*) using water or Kanji (sour gruel).

Ghana: A unique preparation from an aqueous extract of a plant, like Guduchi.

Marketed Preparation & Testing:

There is various marketed preparation in market containing synthetic or natural anti-inflammatory agent like polyherbal.

Formulation: Market-ready forms are developed from classical Ayurvedic preparations.

1) Anti-inflammatory Activity Evaluation:

In vitro Studies: Tests on cells (like RAW264.7) to measure inhibition of pro-inflammatory markers such as nitric oxide (NO) and cytokines. Juice: Cold-pressed juice from *Vitex negundo* leaves is marketed as an Ayurvedic tonic to alleviate joint pain, stiffness, and inflammation

In vivo Models: Animal models are used to assess anti-inflammatory effects:

2) Granuloma Formation: In some studies, the reduction in granuloma tissue formation in a subacute inflammation model is used to measure anti-inflammatory effects.

Standardization: The efficacy of market samples is often compared with classically prepared samples and known anti-inflammatory drugs like dexamethasone

II. CONCLUSION

In Ayurveda since Samhita kala various Ayurvedic preparations mentioned during Vedic period used in various diseases like fever, inflammation, wound healing, indigestion, hyperacidity, etc. For immediate relief from such ailments we move towards allopathic medicine, though they are having risk of potential hazardous effects on body like liver damage, kidney damage, risk of carcinogenicity, etc on their chronic use. Geriatric , paediatric patients and pregnant women are at high risk due to side effect of Allopathic medicines. Acute and chronic inflammation is becoming major problem in adults and geriatric patients due to wrong food habits , job profile and life style changes. So there is need of scientific and evidence based screening .Ayurvedic preparations using various animal models for their *in vivo* and *in vitro* studies. Taking into consideration frequency of carcinogenicity and organ failure cases due to available allopathic medicines, Ayurvedic preparation will be the best option to resolve this problem. This review will help regarding available ayurvedic plants used against various diseases and various animal model used for screening of Anti-inflammatory activity.

REFERENCE

- [1]. Acharya, J.T., & Shukla, V.J. (2015). Ayurvedic concepts on food and nutrition. *Ayu*, 36(3), 239–242. doi: 10.4103/0974 8520.182756
- [2]. Bhatt, N. (2017). Ayurveda and its comparison with modern medicine. *International Journal of Ayurvedic Medicine*, 8(2), 75 77. doi: 10.4103/ijny.ijoyppp_12_17
- [3]. Chaudhary, A., & Singh, N. (2016). A comparative study of Ayurveda and allopathy for lifestyle disorders. *International Journal of Ayurveda and Pharma Research*, 4(6), 1–5.
- [4]. Chopra, A., Doiphode, V. V. (2009). Ayurvedic medicine—Core concept, therapeutic principles, and current relevance. *Medical Clinics of North America*, 93(4), 1225-1246.
- [5]. Bagad A. S., Joseph J. A., Bhaskaran N., Agarwal A. Comparative evaluation of anti-inflammatory activity of curcuminoids, turmerones, and aqueous extract of *Curcuma longa*. *Advances in Pharmacological Sciences*, 2013, 2013: 7. doi: 10.1155/2013/805756805756
- [6]. Ghasemian M., Owlia M. B. A different look at pulsed glucocorticoid protookoðeç is high dose oral prednisolone really necessary just after initiation of pulse therapy? *Journal of Case Reports in Practice*, 2015, 302 1-3
- [7]. Ak Gupta, Gupta Neeraj Tandon, madhu sharma the quality standarde of Indian medicinal plant .vol-3
- [8]. Gerhard vogel H, Wolfganga HV, Bernward AS, Jurgen S, Gunter M and Wolfganga FV. Drug discovery and evaluation pharmacological assays. Berlin, Germany .2002.
- [9]. Kulkarni SK. Handbook of Experimental Pharmacology. Vallabh Prakashan: 2007.
- [10]. Olumayokun A, Olajide, Modupe MJ and Olubusayo A. Effects of the aqueous extract of *Bridelia ferruginea* stem bark on carageenan- induced oedema and granuloma tissue formation in rats and mice. *J Ethnopharmacol*. 66;1999: 113-7.
- [11]. Parmar NS and Prakashan S. Screening Methods in Pharmacology; Narosa Publishing house.2006.
- [12]. Penna SC, Medeiros MV, Aimbire FSC, Faria-Neto HCC, Sertie JAA and Lopes-Martins RAB. Anti-inflammatory effects of the hydalcoholic extract of *Zingiber officinale* rhizomes on rat paw and skin edema. *Phytomedicine*. 10; 2003:381–5.
- [13]. Warren JB, Loi RK and Coughlan ML. Involvement of nitric oxide synthase in the delayed vasodilator response to ultraviolet light irradiation of rat skin *in vivo*. *Br. J. Pharmacol*. 109; 1993: 802–806.
- [14]. Yashraj.Y, Mohanty.P.K, Kasture.S.B. Anti inflammatory activity of hydroalcoholic extract of *Quisqualis indica* Linn. flower in rats. *International Journal of Pharmacy & Life sciences*. 2011;2(8):977-81.

- [15]. Tadafumi.T, Toru.A, Kenji.O, Haruhiko.M. The effects of olopatadine hydrochloride on the number of scratching induced by repeated application of oxazolone in mice. *Eur J Pharmacol.* 2005;524:149-54.
- [16]. Rogerio.A.S, Romagna.C.Oliveira, Mariana.K.A.Araruna, Kleber.D.P.Menezes, Gerlania.O.Leite, Marta.R.Kerntopf, et al. Topical anti-inflammatory effect of *Caryocar coriaceum* Wittm. (Caryocaraceae) fruit pulp fixed oil on mice ear edema induced by different irritant agents. *J Ethnopharmacol.* 2011;136:504- 10.
- [17]. Gupta.SK. Drug screening methods. 2 ed. New Delhi: Jaypee; 2009. 8. Penna.S.C, Medeiros.M.V, Aimbire.F.S.C, Faria-Neto.H.C.C, Sertie.J.A.A, Lopes Martins.R.A.B. Anti-inflammatory effect of the hydalcoholic extract of *Zingiber officinale* rhizomes on rat paw and skin edema. *Phytomedicine.* 2003;10:381–5.
- [18]. Chedea VS, Jisaka M. Inhibition of soybean lipoxygenases-structural and for the lipoxygenaseisoenzymes family: recent activity models trends for enhancing the diversity and quality of soybean products. In *Tech.*, 2005; 6: 100-130.
- [19]. Krol W. Czuba ZP, Threadgill MD, Cunningham BD, Pietse G. Inhibition of nitric oxide (NO) production inmurine macrophages by flavones. *Biochem Pharmacol.*, 1995; 50: 1031-1035.
- [20]. Nworu HS. Akah PA. Anti-inflammatory medicinal plants and the molecular mechanisms underlying their activities. *Afr J Tradit Complement Altern Med.* 2015; 12:52-61
- [21]. Lee TP, Matteliano MI, Middleton F Effect of quercetin on human polymorphonuclear leukocyte lysosomal enzyme release and phospholipid metabolism. *Life Sci.*, 1982: 31(24); 2765-2774
- [22]. Dzobo, K. The role of natural products as sources of therapeutic agents for innovative drug discovery. *J. Compr. Pharm.* 2022, 408–422. [CrossRef]
- [23]. Nasim, N.; Sandeep, I.S.; Mohanty, S. Plant-derived natural products for drug discovery: Current approaches and prospects. *Nucleus* 2022,65, 399–411.
- [24]. Wangchuk, P.; Loukas, A. Techniques and Technologies for the Biodiscovery of Novel Small Molecule Drug Lead Compounds from Natural Products. In *Natural Products and Drug Discovery*; Elsevier: Amsterdam, The Netherlands, 2018; pp. 435–465.
- [25]. Anwar, N.; Teo, Y.K.; Tan, J.B.L. The Role of Plant Metabolites in Drug Discovery: Current Challenges and Future Perspectives. In *Natural Bio-active Compounds*; Springer:Berlin/Heidelberg, Germany, 2019; Volume 2, pp. 25–51.