A REVIEW ON ANTI-INFLAMMATORY HERBAL PLANT

GHUTE JYOTI RAOSAHEB¹, AMBEKAR PRIYANKA GANESH², NIKAM NIKITA DNYANESHWAR³

Nandkumar Shinde College of Pharmacy, Vajapur ,Dr. Babasaheb Ambedkar Technological Universit,y Lonere,Maharashtra, India.

ABSTRACT

Numerous nonsteroidal anti-inflammatory medications have been demonstrated to lessen pain and inflammation by preventing the isoform of cyclooxygenase enzyme from metabolising arachidonic acid, which lowers the generation of prostaglandin. Sadly, using nonsteroidal anti-inflammatory medications has a lot of adverse effects. However, there are medicinal herbs that have low to no adverse effects and anti-inflammatory therapeutic properties. The African continent is incredibly rich in medicinal plants, many of which have anti-inflammatory properties and have been successfully used in traditional medicine to treat inflammatory diseases. It's interesting to note that researchers have investigated several of these African medicinal plants and documented their biological and therapeutic functions. Sadly, not a single review article has mentioned any African medicinal herbs with anti-inflammatory qualities. The use of medicinal plants and their secondary metabolites as a form of supplementary medicine is increasing. Rheumatoid and immune-mediated diseases, diabetes, cardiovascular accidents, and many other diseases are all included in the broad category of pathologic disorders known as inflammation. We include several plants whose anti-inflammatory properties have been proven in both clinical and experimental research. Some of the therapeutic plants mentioned in this review are evening primrose, Devil's claw, Zingiberofficinale, and, Allium sativum, Curcuma longa, Elettariacardamomum, Piper nigrum L., Camellia L., Rosmarinusofficinalis, Cinnamout, etc.

KEYWORD: - *Herbal plant, Active Biological Compound, Anti-Inflammatory, Inflammation, Treatment, etc.*

INTRODUCTION:-

History and used of Herbal plants:-

Herbal medicine is the earliest type of healthcare ever used by humans. Man has been reliant on plants from the dawn of humanity. Since a very long time before recorded history, plants have been utilised as medicines. Herbal medicines, usually referred to as botanical medicines or phytomedicine, are made from complete plants as well as leaves, seeds, roots, berries, bark, flowers, and other plant parts. According to Ayurveda, more than a thousand herbs are employed in various treatments. Many diseases have been treated for a very long time using plants. In daily life, several plants are used as food, medicine, or spices. India is one of the world's top producers of medicinal plants and one of the biggest nations for the production of herbs. Herbal plants are an intriguing source of all-natural remedies for different medical issues. [1]

Inflammation:-Around 2,000 years ago, Celsius defined inflammation using four Latin words: rub or, tumour, dolour, and Caldor. The term "inflammation" is widely used to describe the protective reaction that the human body has to unpleasant stimuli [2]

Plants as natural anti-inflammatory agents:-Herbal medications function in a way that depends on an unspecified mechanism, as opposed to current allopathic treatments, which have single active components that target one particular pathway orchestral method numerous things make up a plant. Comprising several molecules that work in concert on Targets in the intricate biological pathway[3]Since ancient times, medicinal plants have provided a wide range of biologically active chemicals, which have been widely used as raw materials or For the treatment of many diseases as pure subst ances[4]Due to the negative effects and toxicity of allopathic medications, the usage of herbal remedies is growing in popularity. The use of medicinal herbs is Plays a crucial part in the

development of powerful Medicinal substances. Over 1.5 million people exist. Practitioners of the conventional medical system who Therapeutic use of plants in promotion, prevention, and Applications for cure[5] India may continue to play a significant role in the manufacture of due to its largest collection of medicinal plant in the word. Ether raw material use directly to make crude medicine or as the bioactive ingredient use to create cosmetic, drug and other products. [6]

Sr no.	Plant name	family
1.	Zingiber officinalae (Ginger)	Zingiberaceae
2.	Allium sativum(garlic)	Amaryllidaceae
3.	Curcuma longa(turmeric)	Zingiberaceae
4.	Piper nigrum(Black paper)	Piperaceae
5.	Asian Ginseng (Ginseng)	Araliaceae
6.	Azardirachta indica(Neem)	Meliaceae
7.	Emblica officinalis(Amla)	Euphorbiaceae
8.	Aloe barbadensis Miller (Aloe vera)	Asphodelaceae
9.	Camellia sinensis (Green tea)	Theaceae
10.	Adhatoda vasica Nees(Malabar nut)	Acanthaceae
11.	Astragalus membranaceus(Astragalus)	Fabaceae
12.	Calendula officinalis (Calendula)	Asteraceae
13.	Uncaria tomentosa(Cats claw)	Rubiaceae
14.	Matricaria chamomilla L (Chamomile)	Asteraceae
15.	Coriandrum sativum(Cilantro)	Apiaceae
16.	Echinacea purpurea (Echinacea)	Asteraceae
17.	Ocimum tenuiflorum (Holy basil)	Lamiaceae
18.	Piper methysticum(Kava kava)	Piperaceae
19.	Melissa officinalis. (Lemon Balm)	Lamiaceae
20.	Glycyrrhiza glabra(Licorice Root)	Fabaceae
21.	Citrus sinensis (Oregano)	Rutaceae
22.	Petroselinum crispum(Parsley)	Umbelliferae
23.	Passifloraceae (Passion flowers)	Passifloraceae
24.	Achillea millefolium(Common Yarrow)	Asteraceae
25.	Aconitum heterophyllum(Ativisha)	Valeraneaceae
26.	Adansonia digitata (Baobab tree)	Malvaceae
27.	Aegle marmelos(Bilwa)	Rutacae
28.	Annona squamos(Custard apple)	Annonaceae
29.	Baccharis incarum(Salt bush)	Astereae
30.	Bacopa Monnieri (Brahmi)	Scrophulariaceae
31.	Barleria prionitis(Porcupine flower)	Acanthaceae
32.	Bonafousia sananho(Lobo Sanango)	Apocyanaceae
33.	Boussingaultia Gracilis(Anredera Cordifolia	Bassellaceae
34.	Boswellia serrata(Indian Frankincense)	Burseraceae
35.	Bryophyllum Pinnatum(Cathedral bells)	Crassulaceae
36.	Bursera simaruba(Gumbo-limbo)	Burseraceae
37.	Caralluma Thberculata(Chongan)	Asclepiadaceae
38.	Cassia fistula(Golden shower tree)	Caesalpiniaceae
39.	Cassia obtusifolia (Sicklepod)	Leguminosae
40.	Citrus auranticum(Bitter Orenge)	Rutaceae

41.	Commiphora mukul(Guggul)	Burseraceae
42.	Cordia ulmifolia(Varronia linnaei)	Boraginaceae
43.	Daphne pontica(Twin flowered)	Thymelaeaceae
44.	Elephantophs scaber(Tutup bumi)	Compositae
45.	Erythrospermum monticoloum	Flacourtiaceae
46.	Garcinia mangostana(Mangosteen)	Guttiferae
47.	Hammada elegans(Ajram)	Chenopodiaceae
47.	Hedera rhombea(Japanese ivy)	Araliaceae
48.	Iberis amara(Wild candytuft)	Brassicaceae
49.	Kirkia acuminata(White seringa)	Simaroubaceae
50.	Lantana camera(Sage)	Verbenaceae

HERBAL ANTI- INFLAMMATORY PLANT EXAMPLE

1. ZINGIBER OFFICINALAE (GINGER):-



Chemistry of Ginger:-

Chemical makeup of zinger the gingerly were found to be the primary active ingredients in fresh ginger rhizomes, and [7] gingerol [5-hydroxy-1-(4-hydroxy-3-methoxy phenyl) decan-3-one is the most prevalent gingerol in the series (Table 1). The powdered rhizome is made up of 3-6% fat, 9% protein, 60–70% carbs, 3-8% crude fibre, 8% ash, and 2-3% volatile oil. Alpha-farmesene, camphene, beta-phellandrene, curcumene, cineole, geranial acetate, terphineol, terpenes, borneol, geraniol, limonene, and linalool are among the main mono- and sesquiter-pene components of volatile oil. Alpha-zingiberene, which makes up 30–70% of the oil, beta-sesShogaol, a dehydrated form of gingerol, is the most potent pungent component in dried ginger powder and can contribute up to[8,9]

10000

1.	6-gingerol (S)-5-hydroxy-1-(4-hydroxy-3- methoxyphenyl)-3-decanon
2.	8- gingerol (5S)-5-hydroxy-1-(4-hydroxy-3-
	methoxyphenyl) dodecan-3-one
	3. 10-gingerol (E)-1-(4-hydroxy-3-
3.	methoxyphenyl) dec-4-en-3-one
4.	6-shogaol (E)-1-(4-Hydroxy-3-
	methoxyphenyl) dec-4-en-3-one

Table no. 1 Active Anti- Inflammatory component of ginger

Mechanism of Action:-

Ginger reduces airway inflammation: - Ginger improves the Th1 response, which in turn reduces airway inflammation in mice, and it also reduces the Th2 responses that ovalbumin causes[10,11] also by decreasing the levels of IL4, IL5[12] Due to the modulation of calcium channel function, it can help lessen asthmatic symptoms by relaxing the smooth muscle in the airways.[13]

Ginger and kidney function:-Gentamicin-induced nephrotoxicity is avoided by gingerol fraction from Zingiberofficinale. It enhances renal functions, lessens nitrosative stress, and minimises lipid peroxidation. [14]Additionally, ginger extract reduces the damage to rats' kidneys brought on by chronic fructose ingestion by reducing the overexpression of pro-inflammatory cytokines in their kidneys. [15]

Ginger and Liver Function:-In a mouse model, dried ginger (Zingiberofficinale) reduces inflammation, improves pathological alterations, andDecreases IL6 and INF levels. It may also lessen liver PProTNF, IL-6, and other inflammatory reactionsCytokines via preventing NF-B activation [16]

Enhancing the symptoms of neurological degenerative diseases with ginger:-In animal models of dementia, 6-Shogaol, an active component of ginger, reduces neuro-inflammation and cognitive impairments. It thus has a significant impact on the im-Proof of Alzheimer's disease symptoms in patients with several neurological conditions. It enhances memory by blackening glial cell activity in animal models of dementia and Additionally by lowering memory corruption[17]The activity of NF-B is also decreased by ginger[18,19]iNOS, Cyclooxygenase 2, and (COX2)[20]It shields C57BL/6 mice and Hecate cells from inflammation brought on by ultraviolet B.[21]Ginger can prevent skin from becoming darker because it inhibits the production of melanin in B16F10 melanoma cells[22]

Ginger and Rheumatic Disorders:-Through its anti-inflammatory, antioxidant, and anti-serotonin properties, ginger offers preventive effects against joint inflammation, arthritis, and musculoskeletal ailments. It blocks the 5-Lipoxygenase and Cyclooxygenase-2 pathways. Ginger increases the production of T-helper-2 and anti-inflammatory cytokines like IL-4 and IL-10. [23, 24] reduces the amount of substance P released (mediator of inflammation and pain) [25] and lowers the levels of prostaglandins, IL1, IL6, IL6, TNF, and IL1. In one study, it was discovered that ginger is more potent than indomethacin at easing the pain brought on by inflammation and oxidative stress.[26]

1. ALLIUM SATIVUM(GARLIC):-



Chemistry of Garlic:-Approximately 33 sulphur compounds, including aliin, allicin, ajoene, allylpropyldisulfide, diallyltrisulfide, sallylcysteine, vinyldithiines, S-allylmercaptocystein, and others, are found in garlic, along with several enzymes, 17 amino acids, including arginine, and minerals like selenium, germanium, tellurium, and others (Newall et al., 1996)[27]

1.	Allicin[S-(2-propenyl)-2-propene-1-sulfinothioate]
2.	Alliin (S-allyl-L-cysteine sulfoxide)

Table no. 02 Active Anti – Inflammatory component of garlic

Machanism of Action:-The reduction of IL-12 synthesis and dysregulation of IL-10 in inflammatory bowel disease (IBD) inhibits IL-12 from attaching to its receptor on T and NK cells, hence inhibiting the generation of IFN-?[28]The gastroprotective effects of garlic have been investigated. In an animal model, AGE capsules were able to reverse indomethacin-induced OS in the gastric tissue by lowering TNF-? And malondialdehyde levels, decreasing myeloperoxidase activity, and raising total glutathione, superoxide dismutase, and catalase activities. [29]T and B lymphocytes have a demonstrated role in the pathophysiology of the stomach. Proteins, lipids, and DNA are harmed by OS. [29, 30]

Metabolism Syndrome:-Insulin resistance, hypertension, and other disorders are all part of the metabolic syndromeobesity in the abdomen, hyperlipidemia, and glucose intolerance. This syndrome typically comes before type 2 diabetes and atherosclerosis[31]Garlic's participation in several of these illnesses has been explored, and its effects on immune system factors connected to the proinlammatory state of metabolic syndrome include modulation of oxidative stress (OS), proapoptotic signal pathways, inlammatory mediators, and cellular activities.

Cardiovascular Disorders:-The most common cause of death worldwide, cardiovascular diseases (CVD) are still on the rise globally. Heart, brain, and blood vessel conditions collectively are referred to as CVD. [32] Additionally investigated as prospective techniques for predicting the risk of coronary events are plasma inlammation indicators. Acute-phase protein and high-sensitivity C-reactive protein (CRP) are examples of indicators of systemic inlammation. [33]In fructose-fed rats, VCAM-1 expression could be reduced by long-term treatment of aqueous garlic. Resulting with reduced vascular inflammation from garlic chemicals. [34, 35]

Obesity:-Obesity is connected to low-grade chronic inflammation characterised by aberrant cytokine production, increased acute-phase reactants, and other mediators in response to excessive nutrition in metabolic cells [36] an unresolved inflammatory response develops inside the tissue as a result of the activation of a network of inflammatory signalling pathways in the cell. This activation also results in the activation of specific immune cells[37]NK – cell [38]Micro- phases [39]Adipose tissue from obese people exhibits iniltration, which plays a role in the inflammatory process associated with obesity and raises the risk of developing insulin resistance.[39]

2. CURCUMA LONGA(TURMERIC):-



Chemistry of Turmeric:-Turmeric contains protein (6.3%), fat (5.1%), minerals (3.5%), carbohydrates (69.4%) and moisture (13.1%). Phenolic diketone, curcumin (diferuloylmethane) (34%) is responsible for the yellow colour, and comprises curcumin I (94%), curcumin II (6%) and curcumin III (0.3%). Other phenolic diketonesdemethoxycurcumin and bis-demethoxycurcumin have also been isolated from the rhizomes of Curcuma longa. Presence of tumerones (a and b), curdione, curzerenone, mono- and di-demethoxycurcumin have been reported in the rhizomes. The essential oil (5.8%) obtained by steam distillation of rhizomes has a-phellandrene (1%), sabinene (0.6%), cineol (1%), borneol (0.5%), zingiberene (25%) and sesquiterpines (53%)[40]The essential oils of leaves of C. Longa have been analyzed by GLC (Perkina Elmer auto-system fitted with capillary column carbowax 20 m of 50 m length flux ionization detector) and was reported to contain $\hat{1}$ -pinene, $\hat{1}^2$ -pinene, sabinene, myrcene, a-phellandrene, 1,8-cineole, p-cymene, C8-aldehyde, linalool, caryophyllene, geraniol and methyl heptanone[41]

1.	Curcumin (Curcumin I)	Diarylheptanoid
2.	Bisacurone	Bisabolane
3.	Bisacurone A.	Bisabolane
4.	Bisacurone	Bisabolane

	В.	
5.	Bisacurone C.	Bisabolane

Table 3.:- Anti- inflammatory Chemical constituents of turmeric

Ulcerative Colitis:-Diarrhoea was significantly improved, colonic architecture was improved, neutrophil infiltration was significantly reduced, and lipid peroxidation in colonic tissue was significantly reduced after administration of 50 mg/kg curcumin for 10 days prior to the induction of colitis with 1,4,6-trinitrobenzene sulphonic acidconsiderably reduced neutrophil infiltration and lipid peroxidation in colonic tissue, improved colonic architecture, and significantly reduced diarrhoea. Additionally, colonic mucosal nitric oxide and O2 radical levels were decreased, and NF-B activation was repressed, all of which are signs of decreased inflammation and a reduction in symptoms.[42]

Ocular Condition:-Untreated anterior uveitis can cause clouded vision and long-term damage to the eye's uveal tract, which includes the iris. Although the precise aetiology of anterior uveitis is unknown, it has been reported to be associated with eye injuries, other eye conditions, tuberculosis, rheumatoid arthritis, measles, or mumps. Typically, treatment aims to reduce inflammation[43]

Anti Arthritis:-Due to curcumin's anti-inflammatory and cartilage-protecting properties, it can reduce joint inflammation and relieve pain symptoms. Curcumin increased the chondroprotective transcriptional regulator Cbp/p300 interacting transactivator with ED-rich tail 2 and decreased the mRNA expression of MMPs 1.3 and 13, ADAMTS5, IL-1, and TNF- in primary cultured chondrocytes (CITED2)[44]In rat models of arthritis caused by lipopolysaccharide (LPS), collagen type II, and monoiodoacetic acid, curcumin decreases the production of inflammatory mediators like TNF-, IL-17, IL-1, transforming growth factor (TGF), and cyclooxygenase-2 (COX-2). It also lessens inflammation of the cartilage and synovium. [45, 46] by blocking the TLR4 pathway and its downstream NF-B signalling pathway, curcumin reduces inflammation. [46, 47]

Psoriasis:-Psoriasis is a chronic inflammatory skin disease. Psoriatic arthritis, cardiovascular disease, obesity, metabolic syndrome, liver disease, kidney disease, and depression are among the comorbidities that frequently accompany psoriasis. It is believed that their pathophysiology involves inflammation. [48]By working on MAPKs, AP-1, and NF-B pathways, cur cumin has anti-inflammatory, antioxidant, and immunomodulatory properties and can reduce the number of T cells, their ability to proliferate, and their ability to produce inflammatory factors. [49]

3. PIPER NIGRUM(Black paper) :-



Chemistry of Balck paper: - Piperine is found in black pepper. The chemical name for piperine is C17H19NO3.Piperine, whose content varies in different species of P. Nigrum, is the most significant possibility. For instance, the amount of piperine in long pepper can vary by up to 2%, whereas that in white and black pepper might vary by up to 5% to 10%. The presence of piperine, which improves the bioavailability of many medicines and nuitrients by blocking a number of metabolising enzymes, explains why it is so crucial for therapeutic use. As a result, it is crucial in controlling the dyslipidemia brought on by obesity. Black pepper's unripe fruit can be used to extract piperine. It is concentrated in black pepper at a rate of 6-9%. Beta-carotene, lauric acid, palmitic acid, and piperine are also included in it.

1) Piperine2E, 4E)-5-(benzo[d] [1,3]dioxol-5-yl)1-(piperidin-1-yl)penta-2,4-dien-1one

Table 4.:- Active Anti- Inflammatory component of Black Paper

Rheumatoid Arthritis:-Inflammatory immune cell infiltration into the synovial fluid, chronic proliferative synovitis and cartilage degradation are the hallmarks of rheumatoid arthritis. Because they generate large amounts of proinflammatory mediators such matrix metalloproteinases (MMPs), interleukin (IL)6, IL8, and prostaglandin E2, proliferative fibroblast-like synoviocytes (FLSs) play key roles in the spread of inflammation and the destruction of joints (PGE2)[50,51]

4. ASIAN GINSENG (Ginseng):-



Chemistry of Ginseng:-Many disorders linked to inflammation can be treated with panax ginseng. One of the main active panaxadiols isolated from Panax ginseng is ginsenoside Rb3 (GRb3), a natural substance with antiinflammatory and immunomodulatoryactivities. If GRb3 prevented LPS-induced inflammation in macrophages by inhibiting TLR4/NF-B/MAPK signalling. By decreasing the expression of iNOS and COX2, GRb3 reduced the generation of NO and PGE2. Furthermore, IL-1, IL-6, and TNF-a levels were reduced by GRb3. The restrictive effects of GRb3 on the NF-B/MAPK pathway and inflammatory mediators were partially reversed by overexpressing TLR4.

Offischoside (R01)
Compound (K)
Ginsenoside (Rg1)
Ginsenoside (Rg3)
Ginsenoside (Rh2)

Table No. 5 :- Anti- inflammatory Chemical constituents of Ginseng

Anti- inflammatory Activity:-There are two types of inflammatory reactions: acute inflammation and chronic inflammation. The duration of acute inflammation is seven days or one week, whereas the duration of chronic inflammation is four weeks. Th2 cells release IL-4, IL-6, IL-10, and transforming growth factor to counteract the cytokines generated by Th1 cells during inflammation (IL-2, interferon [IFN]-, TNF-, and others). While diverse situations with unbalanced Th1/Th2 responses lead to chronic inflammation, the mutual balance between Th1 and Th2 responses in inflammation quickly reduces acute inflammation conditions back to normal.[52]Ginseng has been shown to have anti-inflammatory properties in several in vivo, in vitro, and clinical trials. Due to the negative modulation of pro-inflammatory cytokine expressions (TNF-, IL-1, and IL-6) and enzyme expressions in M1-polarized macrophages and microglia, gensenosides Rb1, Rg1, Rg3, Re, Rd, Rh1, Rc, Rf, Rg5, Rg6, Rh3, Rk1, Ro, and Rz1 have been found to have anti-inflammatory effects.[53] The creation of NO and the expression of TNF-, iNOS, and COX-2 in lipopolysaccharide-activated macrophages were reported as an anti-inflammatory mechanism for P. Ginseng berry calyx extract (Pg-C-EE)[54]In contrast to ginsenosides Rc, which blocks cytokines produced by macrophages, ginsenosides Re and Rp1 can inhibit the NFB signalling pathway. According to clinical research, patients who took ginseng lived 38 % longer than those who did not take ginseng[52]

5. AZARDIRACHTAINDICA(Neem) :-



Chemistry of Neem :-Nimbolinin, nimbin, nimbidin, nimbidol, sodium nimbinate, gedunin, salannin, and quercetin are the other active ingredients, with azadirachtin being the most significant. N-hexacosanol, amino acids, ascorbic acid, 6-desacetylnimbinene, nimbandiol, nimbolide, 7-desacetyl-7-benzoylazadiradione, 7-desacetyl-7-benzoylgedunin, 17-hydroxyazadiradione, and nimbiol are all components found in leaves.[55]Polyphenolic flavonoids isolated from freshly harvested neem leaves include quercetin and β-sitosterol, which are known to have antimicrobial and antifungal activities[56]Its seeds contain beneficial substances including azadirachtin and gedunin.[57]

1)	Nimbidin
2)	Sodium nimbidate
3)	Garlic acid
4)	Polysaccharides
5)	Polysaccharides G2A,G3A

6)Table:- Anti- inflammatory constitute of Neem

Mechanism of action:-The chloroform extract of stem bark substantially reduces carrageenan-induced paw edoema and ear irritation in rats and mice. Children with inflammatory stomatitis can benefit from bark extract treatment. The anti-pyretic properties of neem oil are strong. In experimental animals, the plant has also been found to have analgesic effect that is mediated by opioid receptors. It has been reviewed how different extracts' anti-pyretic and anti-inflammatory properties work.[58]the parts of neutrophils and macrophages that are related to inflammation are suppressed by nimbidin, according to study findings[59]

6. EMBLICAOFFICINALIS(Amla):-



Chemistry of Amla:-One of the species that has received the most research is amla. Alkaloids, phenols, and polyphenols are reported to be present.[60]28% of the total tannins found in the plant are dispersed in the fruits. Emblicanin A and B are two hydrolyzable tannins found in the berry.[61]which both have antioxidant qualities; one when hydrolyzed yields gallic acid, ellagic acid, and glucose, whereas the other yields ellagic acid and

respectively, glucose. The berry also contains Phyllemblin.[62]Numerous compounds, including gallic acid, corilagin, furosin, and geraniin, were discovered through activity-directed fractionation.[63]

1)	Garlic acid
2)	Methanolic fruit extract

Table No. 7 Anti- inflammatory constitute Amla

Mechanism of Action:-In the current study, we looked into the impacts of Emblicaofficinalis extracts on carrageenan- and dextran-induced rat hind paw oedema. The plant's leaf methanol extract's water fraction was discovered to have anti-inflammatory properties. The same fraction's effects on the production of inflammatory mediators like leukotriene B4 (LTB4), platelet-activating factor (PAF), and thromboxane B2 (TXB2), as well as on LTB4- and N-formyl-L-methionyl-L-leucyl-L-phenylalanine (FMLP)-induced in-vitro migration of human polymorphonuclear leukocyte Human PMNs were blocked from migrating in relatively low quantities by the water part of the methanol extract. It did not prevent human PMNs from producing LTB4 or PAF, nor did it prevent human platelets from producing TXB2 during clotting, indicating that the anti-inflammatory activity shown in the rat paw model is not caused by these processes.

7. ALOE BARBADENSIS MILLER (Aloe vera) :-



Chemistry of Aloe Vera:-Gas chromatography and mass spectrometric (GCMS) and high performance thin layer chromatographic (HPTLC) analyses are used to identify the following substances: Up to 30% of anthroquinones, primarily C-glucosides. The mixture, sometimes known as aloin, contains barbloin. Free anthroquinones, emodin (glycosides), and isobarbaloin (e.g. aloe-emodin). Resins, alosin, and its aglyconealoesone (a chromene) are other components. Bradykinase, Sorbitol, Sorbic Acid, Potassium Sorbate, Aloin, Benzoic Acid, Vitamin B, Vitamin C Acids such as citric, salicylic, -sitosterol, and amino; Octadecanoic acid, C-glycosylchromone, D-mannitol, and Hexadecanoic acid.

1) Steroid	
2) Saponin	

Table No.8 :- Anti- inflammatory constitute of Aloe Vera

Mechanism of Action:-The cyclooxygenase pathway is blocked by aloe vera, which also lowers the amount of prostaglandin E2 produced from arachidonic acid. C-glucosylchromone, a brand-new anti-inflammatory substance, was just recently isolated from gel extracts.[64]The anti-inflammatory properties of aloe vera extracts and their possible inhibition of the arachidonic acid pathway by cyclooxygenase have been hypothesised.

8. CAMELLIA SINENSIS (Green tea):-



Chemistry of Green tea:-Green tea's active ingredient, epigallocatechin-3-gallate (EGCG), has been shown to have anti-inflammatory properties. According to earlier research, inflammatory cytokines and enzymes associated to inflammation are suppressed by green tea and EGCG at the gene and/or protein levels.[65]

|--|

TableNo. 9 .Anti- inflammatory constitute of Green tea

Mechanism of Action:-Inflammatory bowel illnesses (IBD) such as ulcerative colitis and Crohn's disease have been linked to inflammation, and research on animals and in test tubes has demonstrated that EGCG can help alleviate these symptoms.[66,6768]Moreover, green tea polyphenols seem to be helpful for inflammatory illnesses like osteoarthritis, rheumatoid arthritis, Alzheimer's disease, gum disorders, and even some cancers.[67,68]

9. ADHATODAVASICANEES(Malabar nut) :-



Chemistry of Malabar nut :- Alkaloids, tannins, saponins, phenolics, and flavonoids are only a few of the phytochemicals found in the leaves of Justiciaadhatoda. A quinazoline alkaloid called vasicine is the most significant. According to dry weight measurements, the herbage's vasicine yield ranges from 0.541 to 1.1%. It was Justiciaadhotada from which bromhexine, a serine protease inhibitor with mucolytic effects, was originally produced.

1.	Adhatodine
2.	Vasicine
3.	Vasicine acetate
4.	Anisotina
5.	Betaine
6.	Vasicinolone

Table No. 10 :- Anti- inflammatory constitute of Malabar nut

Mechanism of Action:-

Joint pain :- Many people deal with joint pain, which can significantly impact their way of life. One of the main causes of such pain is an elevated uric acid level. Vasaka powder benefits by lowering uric acid levels. Malabar Nut also effectively lessens gout-related aches and pains. Its anti-inflammatory qualities aid in reducing joint inflammation as well, Vasicinolone, 2- acetyl benzyl amine , Vasicine , these constitutes shows very strong anti- inflammatory Activity.[69]

CONCLUSION:-

One of the most significant elements of alternative therapies is herbal medicine. Numerous studies have demonstrated the effectiveness of various herbs in the reduction of inflammation. We discuss a few herbs whose anti-inflammatory properties have been examined in clinical and experimental studies; obviously, clinical data is more trustworthy than other types of data.

Many plant species have been used traditionally or as folk remedies for inflammatory illnesses. Scientific research has shown that several of them are effective anti-inflammatory drugs. Due to their complex combinations, the active components of the majority of plant extracts have not been fully explained, despite the diverse bioactivities of plant medicines against various diseases. Nonetheless, it has been noted that the primary chemical classes of anti-inflammatory substances from natural sources interact with a wide variety of substances, including polyphenils, flavonoids, terpenoids, alkaloids, anthraquinones, lignans, polysaccharides, saponins, and peptides.Natural herbs are more secure, efficient, and preferable solutions. With a similar mode of action to synthetic compounds, phytoconstituents are just as effective. The focus of future study should be on the molecular mechanisms behind the varied therapeutic uses of these herbal plants in treating a range of ailments. Current patents on painkillers and herbal plants have been covered, giving information on the situation right now and the potential for the future. The majority of chemicals created today come from herbal plants. It is evident from historical records and reviews and research articles that medicinal plants play a significant part in themedical system and have the potential to treat all diseases.

As there are so many plants that have been claimed to have anti-inflammatory properties that reviewing them all would be beyond the scope of this paper, we have only mentioned the herbs for which there is more support.[70,71]

REFERENCE:-

1.Abeloff MD, Armitage JO, Niederhuber JE, Kastan MB, McKenna WG. Abeloff's Clinical Oncology E-Book. Elsevier Health Sciences. 2008;p4

2.Lalrinzuali K, Vabeiryureilai M, Jagetia GC. Investigation of the anti-inflammatory and analgesic activities of ethanol extract of stem bark of SonapathaOroxylumindicum in vivo. International journal of inflammation. 2016:8247014.

3.Durmowicz AG and Stenmak KR. Mechanisms of structural remodeling in Chronic pulmonary, Hypertension. Pediatr Rev. 1999;20:91-101

4.Arif T, Bhosale JD, Kumar N, Mandal TK, Bendre RS, Lavekar GS and Dabur R.NaturalProducts-antifungal agents derived From plants. Journal of Asian Natural Products Research. 2009;7:621-638.

5.Dasilva EJ. Medicinal plants: a reemerging health aid, Electronic Journal of Biotechnology. 1999;2:57-70.

6. Tiwari S. Plants: a rich source of herbal Medicines. Journal of Natural Productsv2008;1:27-35

7.Schulick P. Ginger-common spice and wonder drug. Edn 2. Herbal Free Press Ltd. Brattleboro Vermont USA 1994; 111-125

8.Mustafa T, Srivastava KC, Jensen KB. Drug Development Report (9): Pharmacology of ginger, Zingiberofficinale. J Drug Dev 1993; 6(24).

9.Awang DVC. Ginger, CPJRPC July1992; 309.

10.Shieh Y-H, Huang H-M, Wang C-C, Lee C-C, Fan C-K, Lee Y-L. Zerumbone enhances the Th1 response and ameliorates Ovalbumin-induced Th2 responses and airway inflammation in Mice. International immunopharmacology. 2015; 24(2): 383-391. Doi: 10.1016/j.intimp.2014.12.027

11.Muhammad Khan A, Shahzad M, Raza Asim M, Imran M, Shabbir A. Zingiberofficinale ameliorates allergic asthma ViaSuppression of Th2-mediated immune response. Pharmaceutical Biology. 2014; 1-9

12. Ahui MLB, Champy P, Ramadan A, et al. Ginger prevents Th2-mediated immune responses in a mouse model of airway Inflammation. International immunopharmacology. 2008; 8(12): 1626-1632.

13.Townsend EA, Siviski ME, Zhang Y, Xu C, Hoonjan B, Emala CW. Effects of Ginger and its constituents on airway smooth Muscle relaxation and calcium regulation. American journal of Respiratory cell and molecular biology. 2013; 48(2): 157-163. Doi: 10.1165/rcmb.2012-02310C

14.Rodrigues FA, Prata MM, Oliveira IC, et al. Gingerol fraction from Zingiberofficinale protects against gentamicin-induced nephrotoxicity. Antimicrobial agents and chemotherapy.2014; 58(4): 1872-1878.

15.Yang M, Liu C, Jiang J, et al. Ginger extract diminishes Chronic fructose consumption-induced kidney injury through Suppression of renal overexpression of proinflammatorycyto-kines in rats. BMC complementary and alternative medicine. 2014; 14(1): 174. Doi: 10.1186/1472-6882-14-174

16.Li XH, McGrath KC, Nammi S, Heather AK, Roufogali BD. Attenuation of liver pro-inflammatory responses by Zingiberofficinale via inhibition of NF-kappa B activation in high-fat Diet-fed rats. Basic ClinPharmacolToxicol. 2012; 110(3): 238-244. doi: 10.1111/j.1742-7843.2011.00791.x

17.Moon M, Kim HG, Choi JG, et al. 6-Shogaol, an active Constituent of Ginger, attenuates neuroinflammation and cognitive deficits in animal models of dementia. Biochemical and Biophysical research communications. 2014; 449(1): 8-13. Doi:10.1016/j.bbrc.2014.04.121

18)Ho S-C, Chang K-S, Lin C-C. Anti-neuroinflammatory capacity of fresh Ginger is attributed mainly to 10-Gingerol. Food chemistry. 2013; 141(3): 3183-3191. doi: 10.1016/j.foodchem.2013.06.010

19) Jung HW, Yoon C-H, Park KM, Han HS, Park Y-K. Hexane Fraction of ZingiberisRhizomaCrudus extract inhibits the production of nitric oxide and proinflammatory cytokines in LPSstimulated BV2 microglial cells via the NF-kappaB pathway. Food and Chemical Toxicology. 2009; 47(6): 1190-1197. Doi:10.1016/j.fct.2009.02.012

20)Shim S, Kim S, Choi D-S, Kwon Y-B, Kwon J. Anti-inflammatory effects of [6]-shogaol: potential roles of HDAC inhibition and HSP70 induction. Food and chemical toxicology. 2011; 49(11): 2734-2740. doi: 10.1016/j.fct.2011.08.012

21). Guahk G-H, Ha SK, Jung H-S, et al. Zingiberofficinale protects HaCaT cells and C57BL/6 mice from ultraviolet B-induced Inflammation. Journal of medicinal food. 2010; 13(3): 673-680. Doi: 10.1089/jmf.2009.1239

22) Huang H-C, Chiu S-H, Chang T-M. Inhibitory effect of [6]-Gingerol on melanogenesis in B16F10 melanoma cells and a possible mechanism of action. Bioscience, biotechnology, and biochemistry. 2011; 75(6): 1067-1072. doi: 10.1271/bbb.100851

23)Srivastava K, Mustafa T. Ginger (Zingiberofficinale) and rheumatic disorders. Medical Hypotheses. 1989; 29(1): 25-28. doi: 10.1016/0306-9877(89)90162-X

24) Srivastava K, Mustafa T. Ginger (Zingiberofficinale) in rheumatism and musculoskeletal disorders. Medical hypotheses.1992; 39(4): 342-348. doi: 10.1016/0306-9877(92)90059-L

25) Stratz T, Müller W. The use of 5-HT3 receptor antagonists in Various rheumatic diseases-a clue to the mechanism of action of These agents in fibromyalgia? Scandinavian Journal of Rheumatology. 2000; 29(113): 66-71.

26) Muller W, Fiebich BL, Stratz T. New treatment options using 5-HT3 receptor antagonists in rheumatic diseases. Current Topics in medicinal chemistry. 2006; 6(18): 2035-2042. Doi:10.2174/156802606778522122

27)Newall C.A., Anderson L.A., Phillipson J.D. Pharmaceutical Press; London: 1996. Herbal medicines: a guide for health-care professionals, vol. Ix. P. 296.

28) G. Hodge, S. Hodge, and P. Han, "Allium sativum (garlic) suppresses leukocyte inlammatory cytokine production in vitro:potential therapeutic use in the treatment of inlammatorybowel disease," Cytometry, vol. 48, no. 4, pp. 209–215, 2002.

29)G. M. Badr and J. A. Al-Mulhim, "he protective efect of aged Garlic extract on nonsteroidal antiinlammatory drug-induced Gastric inlammations in male albino rats," Evidence-BasedComplementary and Alternative Medicine, vol. 2014, Article ID759642, 9 pages, 2014.

30) M. M. D'Elios, A. Amedei, and G. Del Prete, "Helicobacter Pylori antigen-speciic T-cell responses at gastric level in chronic Gastritis, peptic ulcer, gastric cancer and low-grade mucosaassociated lymphoid tissue (MALT) lymphoma," Microbes and Infection, vol. 5, no. 8, pp. 723–730, 200

31) P. Ernsberger, J. L. Johnson, T. Rosenthal, D. Mirelman, and R. J. Koletsky, "herapeutic actions of allylmercaptocaptopril And captopril in a rat model of metabolic syndrome," American Journal of Hypertension, vol. 20, no. 8, pp. 866–874, 2007.

32) L. H. Kuller, R. P. Tracy, J. Shaten, and E. N. Meilahn, "Relation of C-reactive protein and coronary heart disease in the MRFIT nested case-control study," American Journal of Epidemiology, vol. 144, no. 6, pp. 537–547, 1996

33) L. H. Kuller, R. P. Tracy, J. Shaten, and E. N. Meilahn, "Relation of C-reactive protein and coronary heart disease in the MRFIT nested case-control study," American Journal of Epidemiology, vol. 144, no. 6, pp. 537–547, 1996.

34) S. R. Kim, Y. R. Jung, H. J. An et al., "Anti-wrinkle and Anti-inlammatoryefects of active garlic components and the Inhibition of MMPs via NF-?B signaling," PLoS ONE, vol. 8, no.9, Article ID e73877, 2013.

35)M. A. Vazquez-Prieto, C. Rodriguez Lanzi, C. Lembo, C. R. Galmarini, and R. M. Miatello, "Garlic and onion attenuates vascular inlammation and oxidative stress in fructose-fed rats," Journal of Nutrition and Metabolism, vol. 2011, Article ID 475216, 7 pages, 2011.

36) G. S. Hotamisligil, "Inlammation and metabolic disorders," Nature, vol. 444, no. 7121, pp. 860–867, 2006

37) M. F. Gregor and G. S. Hotamisligil, "Inlammatory mechanisms in obesity," Annual Review of Immunology, vol. 29, pp.415–445, 2011

38) K. Ohmura, N. Ishimori, Y. Ohmura et al., "Natural killer T cells are involved in adipose tissues inlammation and glucose intolerance in diet-induced obese mice," Arteriosclerosis, hrombosis, and Vascular Biology, vol. 30, no. 2, pp. 193–199,2010.

39) C. N. Lumeng, S. M. Deyoung, J. L. Bodzin, and A. R. Saltiel, "Increased inlammatory properties of adipose tissue macrophages recruited during diet-induced obesity," Diabetes, vol. 56, no. 1, pp. 16–23, 2007.

40) Bernard GT, Esteban P, Christopher JS. Turmerones: Isolation from Turmeric and their Structure Determination. Chemical communications. 6; 1982: 363.

41) Scientific Correspondence. Major constituents in leaf essential oils of Curcuma longa L. And Curcuma aromaticaSalisb. Current Science. 83(11); 2002: 1312-1313.

42) Ukil A et al. Curcumin, the major component of food flavour turmeric, reduces mucosal injury in trinitrobenzenesulphonic acid induced colitis. British journal of pharmacology. 139; 2003:209-218.

43) Anterior uveitis. http://www.aoa.org/x4719.xml [Accessed March 15, 2009

44) Zhang Z, Leong DJ, Xu L, et al. Curcumin slows osteoarthritis progression and relieves osteoarthritisassociated pain symptoms in a post-traumatic osteoarthritis mouse model. Arthritis Res Ther. 2016;18(1):128. Doi: 10.1186/s13075-016-1025-y [PMC free article] [PubMed] [CrossRef] [Google Scholar] Research Misconduct Found [Ref list]

45) Kang C, Jung E, Hyeon H, Seon S, Lee D. Acid-activatable polymeric curcumin nanoparticles as therapeutic agents for osteoarthritis. Nanomedicine. 2020;23:102104. Doi: 10.1016/j.nano.2019.102104 [PubMed] [CrossRef] [Google Scholar] [Ref list]

46) Yan D, He B, Guo J, Li S, Wang J. Involvement of TLR4 in the protective effect of intra-articular administration of curcumin on rat experimental osteoarthritis. Acta Cir Bras. 2019;34(6):e201900604. doi: 10.1590/s0102-86502019006000004 [PMC free article] [PubMed] [CrossRef] [Google Scholar]

47) Lepetsos P, Papavassiliou KA, Papavassiliou AG. Redox and NF-kappaBsignaling in osteoarthritis. Free RadicBiol Med. 2019;132:90–100. Doi: 10.1016/j.freeradbiomed.2018.09.025 [PubMed] [CrossRef] [Google Scholar]

48) Krueger JG, Brunner PM. Interleukin-17 alters the biology of many cell types involved in the genesis of psoriasis, systemic inflammation and associated comorbidities. ExpDermatol. 2018;27(2):115–123. Doi: 10.1111/exd.13467 [PubMed] [CrossRef] [Google Scholar] [Ref list]

49) Skyvalidas D, Mavropoulos A, Tsiogkas S, et al. Curcumin mediates attenuation of pro-inflammatory interferon gamma and interleukin 17 cytokine responses in psoriatic disease, strengthening its role as a dietary immunosuppressant. Nutr Res. 2020;75:95–108. Doi: 10.1016/j.nutres.2020.01.005 [PubMed] [CrossRef] [Google Scholar] [Ref list]

50) Lee YA, Kim JY, Hong SJ, Lee SH, Yoo MC, Kim KS, Yang HI. Synovial proliferation differentially affects hypoxia in the joint cavities of rheumatoid arthritis and osteoarthritis patients. ClinRheumatol. 2007;26:2023–2029. doi: 10.1007/s10067-007-0605-2. [PubMed] [CrossRef] [Google Scholar]

51) Mor A, Abramson SB, Pillinger MH. The fibroblast-like synovial cell in rheumatoid arthritis: a key player in inflammation and joint destruction. ClinImmunol. 2005;115:118–128. doi: 10.1016/j.clim.2004.12.009. [PubMed] [CrossRef] [Google Scholar]

52) Park J and Cho JY; Anti-inflammatory effects of ginsenosides from Panax ginseng and their structural analogs; African Journal of Biotechnology; 2009; Vol. 8 (16); P 3682-3690.

53) Im D-S. Pro-Resolving Effect of Ginsenosides as an Anti-Inflammatory Mechanism of Panax ginseng. Biomolecules. 2020; 10(3):444.

54) Han S Y, Kim J, Kim E, Kim S H, Seo D, Kim J H, Shin S S, Cho J Y; AKT-targeted anti-inflammatory activity of Panax ginseng calyx ethanolic extract; Journal of Ginseng Research; 2018; 42(4); P-496-503.

55) Ali A. Textbook of Pharmacognosy. New Delhi, India: Publication and Information Directorate; 1993. [Google Scholar] [Ref list]

56) Kokate C., Purohit A. P., Gokhale S. B. Pharmacognosy. Maharashtra, India: NiraliPrakashan; 2010. [Google Scholar] [Ref list]

57) Govindachari T. R., Suresh G., Gopalakrishnan G., Banumathy B., Masilamani S. Identification of antifungal compounds from the seed oil of Azadirachtaindica . Phytoparasitica. 1998;26(2):109–116. Doi: 10.1007/bf02980677. [CrossRef] [Google Scholar] [Ref list]

58) Biswas K, Chattopadhyay I, Banerjee RK, Bandyopadhyay U. Biological activities and medicinal properties of neem (AzadirachtaIndica). CurrSci 2002;82 (11):1336-45.

59) G. Kaur, M. SarwarAlam, and M. Athar, "Nimbidin suppresses functions of macrophages and neutrophils: relevance to its anti-inflammatory mechanisms," Physiotherapy Research, vol. 18, no. 5, pp. 419–424, 2004.

60) Zhang LZ, Zhao WH, Guo YJ, Tu GZ, Lin S, Xin LG, Studies on Chemical constituents in fruits of Tibetan medicine PhyllanthusEmblica,ZhongguoZhong Yao ZaZhi, 28(10), 2003, 940-3.

61) Bhattacharya SK, Bhattacharya A, Sairam K, Ghosal S, Effect of bioactive tannoid principles of Emblicaofficinalis on ischemia-reperfusion induced oxidative stress in rat heart, Phytomedicine, 9(2), 2002, 171-4

62) Yi-Fei W, Ya-Fenga W, Xiao-Yana W,ZheaR,Chui-Wena Q, Yi-Chenga L, Kitazatoc K, Qing-Duan Q, Yan W, Li-Yun Z, Jin-Hua Z, Chong-Rene Y, Qinge L, Ying-June Z,Phyllaemblicin B inhibits Coxsackie virus B3 induced apoptosis and myocarditis, Antiviral Research, 84, 2009, 150-58.

63) Rehman H, Yasin KA, Choudhary MA, Khaliq N, Rahman A, ChoudharyMI, Malik S, Studies on the chemical constituents of Phyllanthusemblica, Natural Product Research, 21(9), 2007, 775-81

64) Hutter JA, Salmon M, Stavinoha WB, Satsangi N, Williams RF, Streeper RT, et al. Anti-inflammatory C-glucosylchromone from Aloe barbadensis. J Nat Prod. 1996;59:541–3. [PubMed] [Google Scholar] [Ref list]

65) https://www.healthline.com/nutrition/anti-inflammatory-herbs

66) Doering J., Begue B., Lentze M.J., Rieux-Laucat F., Goulet O., Schmitz J., Cerf-Bensussan N., Ruemmele F.M. Induction of T lymphocyte apoptosis by sulphasalazine in patients with Crohn's disease. Gut. 2004;53:1632–1638. Doi;10.1136/gut.2003.037911. - DOI - PMC – PubMed

67) Najafzadeh M., Reynolds P.D., Baumgartner A., Anderson D. Flavonoids inhibit the genotoxicity of hydrogen peroxide (H2O2) and of the food mutagen 2-amino-3-methylimadazo[4,5-f]-quinoline (IQ) in lymphocytes from patients with inflammatory bowel disease (IBD) Mutagenesis. 2009;24:405–411. doi: 10.1093/mutage/gep016. - DOI – PubMed

68)Niu J., Miao J., Tang Y., Nan Q., Liu Y., Yang G., Dong X., Huang Q., Xia S., Wang K., et al. Identification of Environmental Factors Associated with Inflammatory Bowel Disease in a Southwestern Highland Region of China: A Nested Case-Control Study. PLoS ONE. 2016;11:e0153524. Doi: 10.1371/journal.pone.0153524. – DOI – PMC – PubMed

69) https://www.lybrate.com/topic/vasaka-malabar-nut-benefits-and-side-effects

