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Divergent Assessment of Anti-inflammatory Activity by Using *Salacia fruticosa*

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ABSTRACT

Salacia fruticosa is a medicinal tree used to treat several inflammatory disorders in the Indian traditional ayurvedic system of medicine. The aim of current study is to evaluate anti-inflammatory activity of the methanolic, aqeous extracts of the leaves of Salacia fruticosa support the pharmacological claims. The study was carried out by using rats. The methanol and aqeous extracts were precipitated using soxhlet extraction process. The inflammatory activity of the methanolic extract and the aqeous of the leaves of Salacia fruticosa were studied using egg albumin induced paw odema (in-vitro) and carrageenan induced paw odema (in-vivo), egg albunin induced paw odema (in-vivo) models. The methanolic and aqeous extract of the leaves of Salacia fruticosa at a doses of 25-800 microgram/ml (P.O) (oral administration) showed a dose dependent and significant inhibition of anti-inflammatory activity models. Indomethacin is used as a standard drug.

Keywords: *Salacia fruticosa*; Inflammatory activity; Indomethacin

INTRODUCTION

From olden days, traditional systems of medicine are of global importance. Even today in many developing countries, majority of population relies greatly on traditional practioners and medicinal plants to meet primary health care needs, despite of availability of modern medicines. These herbal phytomedicines have become popular for historical and cultural reasons. Currently many people in developed countries have begun to turn to alternative herbal medicines.

Scientific evaluation of medicinal herbs for their medicinal value and application is on rise. Safety and efficacy of data of many herbs, their extracts and active ingredients, and the preparations containing them was well established. However the market of herbal drugs is poorly regulated and assurance of safety, efficacy and quality of such herbal products has now become a key issue in industrialized and developing countries [1].

Important basic and clinical researches are being carried out on the therapeutic plants and their formulations, with the state-of-the-art methods in a number of Universities. There are some good examples. Many Indian medicinal plants provide a abundent source for antioxidants that prevent many diseased states. The antioxidant protection is observed at different levels. These plants also contain many important compounds like ingredients for functional foods. In the traditional system of medicine, most of the remedies were taken from plants and they were proved to be useful through the rational behind their use is not well established through systemic pharmacological and clinical studies except for some composite herbal drugs and plants.

The plant kingdom still holds lots of species of plants having substances of different medicinal value which are yet to be discovered in many number of plants are constantly being sheild for their possible pharmacological effects (Figure 1) [2].

Introduction to *Salacia fruticosa*

Figure 1. ***Salacia fruticosa*.**

Taxonomic Classification

Kingdom: Plantae,

Subkingdom: tracheobionta,

Division: Magnoliophyta,

Class: Magnoliatae,

Subclass: Rosidae,

Order: Celastrales,

Family: Hippocrateaceae,

Genus: Salacia,

Species: Fruticosa [3].

Vernacular Names

English: Common willow,

Malayalam: Ponkarandi, Ekanayakam,

Unani: Bedmushk, Bedsaada,

Ayrvedic: Saptachakra, Swarnmula [4].

Description

Salacia fruticosa grows from generally unbranched taproots and produces one to more than ten stems that are typically 5 to 40 cm tall but sometimes up to 70 cm tall. The stems can be tinted purplish, upright or lax, produced

flower heads that are held as taller than the foliage. The foliage is upright growing or horizontally orientated, with leaves having narrowly winged petioles or being unwinged. The stems can be glabrous or sparsely covered with very short hairs [5,6].

The 5-45 cm long and 1-10 cm wide leaves are oblanceolate, oblong in shape with bases gradually narrowing to the petiole. The leaf margins are regularly shallowly lobed and deeply lobed and often lacerate or toothed with sharp or dull teeth. The calyculi (the cup like bracts that hold the florets) is composed of 12 to 18 segments: each segment is reflexed and sometimes glaucous. The lanceolate shaped bractlets are in 2 series with the apices acuminate in shape. The 14 to 25 mm wide involucres are green to dark green orthey may be brownish green with dark gray or purplish tips. The florets number 40 to over 100 per head, having corollas that are yellow or orange-yellow in color. The fruits, which are called cypselae, they range in color from olive-green or olive-brown to straw-colored to grayish mostly they are oblanceoloid in shape and 2 to 3 mm long with slender beaks. They are mostly white to silver-white in color and almost around 6 mm wide. Plants mostly have maximum of 24 or 40 pairs of chromosomes but in some plants they have 16 or 32 chromosomes. Plants mostly have milky sap and the leaves are all basal where each flowering stem lacks bracts and has atleast one single flower head.

Distribution

Salacia fruticosa is distributed in South-West India, Peninsula, Ceylon, Java, Thailand southern Orissa and Phelippines. Within India, it is distributed in Karnataka (rare in semi evergreen forests of western Ghats of phanamthitta and Idukki districts [7].

Medicinal Uses and Indications

Traditionally *Salacia fruticosa* used as acrid, bitter, thermogenic, antiinflammatory, depurative, vulnary, livertonic, and stomachic, Analagesic [8].

Salacia fruticosa is useful in hemorrhoids, inflammation, leucorrhoea, leprosy, skin diseases, amenorrhoea, dysmenorrhoea, wounds, ulcers, hyperhydrosis, hepatopathy, dyspepsia, flatulence

Inflammation

Inflammation is one of the part of complex biological response of vascular tissues to harmful stimuli such as different pathogens, damaged cells, or irritants inflammation is a protective attempt by the organism to remove the injurious stimuli and to initiate the healing process. Inflammation is not a synonym of the Infection in most of the cases where inflammation is caused by infection. Although infection is generated by a microorganism and inflammation is the retaliation of the organism to the pathogen (Figure 2) [9].

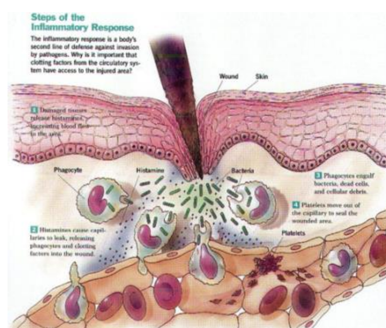

Figure 2. The inflammation response to the cells.

Without inflammation wounds and infections would never heal. In the same way a progressive destruction of the tissue would accomidate the survival of the organism. Although chronic inflammation will also lead to a host of diseases, such as hay fever, it is the reason that inflammation is closely synchronized by the body.

Inflammatory responses occur in three distinct phases, each apparently mediated by different mechanisms.

1. An acute transient phase characterized by the local vasodilatation and increased capillary permeability.
2. A delayed sub-acute phase, most predominantly characterized by infiltration of phagocytic cells.
3. Chronic proliferate phase is characterized by tissue degeneration and fibrosis.

Acute

First there are limited areas of redness and painful swelling, which may disappear in a day or two at most. Because of its short duration and because of the histology of tissue and cellular responses this is called acute inflammation. Example are insect bite, small abrasions, cuts, mild burns etc. [10].

Chronic

The outcome of the host reaction to minor injuries may be the continued evolution of the response, so that it lasts for many days or weeks and may spread to involve adjacent or distance tissues.

This type of response is considered as chronic because of the length of its duration. Acute inflammation characteristically shows large number of polymorphonuclear leukocytes, whereas chronic inflammation consists of lymphocytes, monocytes and plasma cells collectively called round cells. Naturally in many instances more than one factor may be responsible for the inflammatory responses (Table 1).

Table 1. Comparison between acute and chronic inflammation

	Acute	Chronic
Causative agent	Pathogens, injured tissues	Persistent acute inflammation due to non-degradable pathogens, persistent foreign bodies, or autoimmune reactions
Major cells involved	Neutrophils (primarily), eosinophils and basophils (response to helminth worms and parasites), mononuclear cells (monocytes, macrophages)	Mononuclear cells (monocytes, macrophages, lymphocytes, plasma cells), fibroblasts
Primary mediators	Vasoactive amines, eicosanoids	IFN-γ and other cytokines, growth factors, reactive oxygen species, hydrolytic enzymes
Onset	Immediate	Delayed
Duration	Few days	Up to many months, or years
Outcomes	Resolution, abscess formation, chronic inflammation	Tissue destruction, fibrosis, necrosis

Cyclooxygenase Inhibition

The main mechanism of action of NSAIDS is the inhibition of the enzymes processing cyclooxygenase activity, which are involved in the formation of prostaglandins and thrombaxanes from arachidonic acid contained in cellular membranes [11].

Phospholipase A2 (mostly cytoplasmic phospholipaseA2) activated in response to various stimuli, catalyze the release of arachdonic acid, the most abounded poly unsaturated fatty acid in the phospholipids component of the cell membranes.

The released arachdonic acid serves as the precursor for the synthesis of different eicosanoids, mediated through the cyclooxygenase and lipooxygenase. Prostaglandins are the main mediators involved in the inflammation (Figure 3).

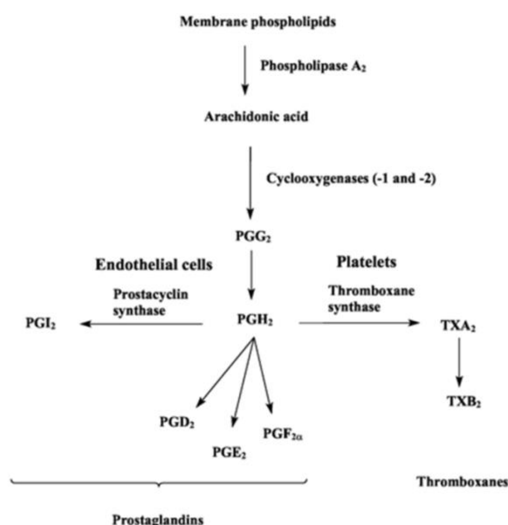

Figure 3. Cyclooxygenase inhibitor inflammation

RHEUMATOID ARTHRITIS

Rheumatoid arthritis (RA) is a chronic systemic inflammatory disorder that will affect many tissues and organs but majorly attacks synovial joints. This action produces a major inflammatory response of the synovium (synovitis) secondary to hyperplasia of synovial cells and excess synovial fluid and the development of pannus in the synovium. The pathology of the disease will often leads to the damage of articular cartilage and ankylosis of the joints. Rheumatoid arthritis will also produce a diffuse inflammation in lungs, pericardium, pleura and sclera, and also in the nodular lesions, most commonly in subcutaneous tissue. However the cause of rheumatoid arthritis is unknown autoimmunity will play a pivotal role in both the chronicity and progression, and it is considered as a systemic autoimmune disease [12,13].

Rheumatoid arthritis (RA) an autoimmune disease is one of the disorders prevalent all over the world, which cripples the activity of human beings without killing. Here the synoviol membrane of joints is inflamed and it goes under a series of histopathological changes, which is replaced by the fibrous tissue including development of an exudative synoviol fluid.

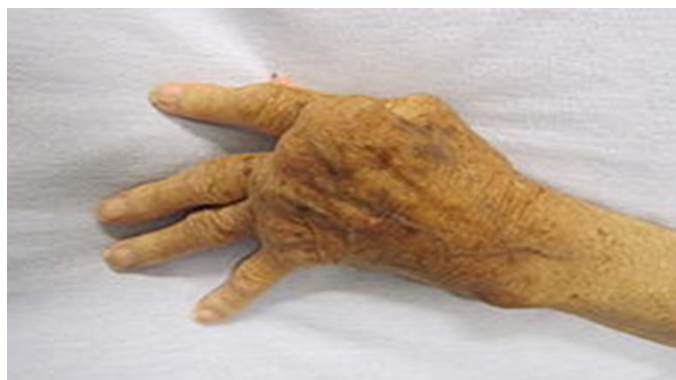

Figure 4. A hand affected by rheumatoid arthritis

RA is not one single disease entity but rather a syndrome described by a number of clinical criteria of signs and symptoms. There is a clear and well-documented sex difference for the suseptability to RA, which is 3-4 times more common in females than in males. This difference depends on both hormonal and genetic factors.

This tissue inflammation is reminiscent of delayed type hypersensitivity reaction occurring in response to soluble antigens or microorganisms. Rheuma inflammation could reflect persistent stimulation of T-cells bysynovial fluid. The exudative synovial fluid contains more polymorphonuclear cells than mononuclear cells. The mechanism by which the bone and the cartilage damage occurs has not been completely resolved, but the majority of damage is in the juxta position to the inflamed synovium or pannus that spreads to cover mainly the articular cartilage. This vascular granulation tissue is composed of proliferating fibroblasts, small blood vessels and a variable number of mononuclear cells. This produces a huge amount of degradation enzymes which includes collagenase and stromelyin that may ease tissue damage. The cytokinens IL-1, TNF-alfa play an important role by stimulating the cells of the pannus to produce collagenase and other proteases (Figure 4).

CLINICAL FEATURES

RA influence almost 1% of the adult Indian population and is the most common inflammatory joint disease which is seen in clinical practice. The disease follows a chronic course and in addition to morbidity results in a shortened life span.

Joint pain, stiffness and symmetrical swelling of a number of peripheral joints characterize RA. In the typical case the small joints of the fingers and toes are the first to be effected. As the disease headway it tries to spread to involve the wrists, elbows, shoulders, knees, ankles, subtalar and mid tarsal joints. Clinically synoviol inflammations cause swelling, tenderness and limitation of motion. Swelling results from accumulation of synoviol fluid, hypertrophy of synovium and thickening of the joint capsule. Motion is limited by pain.

Various Agents Used for the Treatment of RA

Non-steroidal anti-inflammatory agents: selective COX-2 inhibitors like celecoxib, etoricoxib and valdecoxib [14].

Extraction

Pharmaceutically extraction is the term used which involves in the separation of therapeutically active portions of plant or animal tissues from the inactive or inert components by using selective solvents in standard extraction procedures.

Collection and Authentication of Plant Material

The fresh leaves of *Salacia fruticosa* were collected from Thirupathi, Andhra pradesh, shade dried and ground powdered in a mechanical blender. The powdered is subjected to soxhlet extraction using methanol as solvent. A specimen of the plant is deposited in the herbarium of Vaageswari College of Pharmacy, Karimnagar identified and authenticated by Dr. K. Madhava Chetty, botanist of Sri Venkateswara university.

Preparation of Extracts

The dried and powdered leaves of *Salacia fruticosa* was extracted successively with of methanol and aqueous in soxhelt apparatus [15].

A greenish colored methanolic extract was obtained (5.6% with respect to dry powdered plant material), then, the same has been extracted with water to yield brownish green semisolid mass (yield 4.2% with respect to dry plant

material). Two extraction procedures were carried out until the solvent system becomes a colorless. All the extracts were collected and concentrated by evaporating the solvent completely. These extracts were dried and stirred in refrigerator for the future use of various chemical group identification and pharmacological evaluation.

PHARMACOLOGICAL INVESTIGATION

Experimental Animals

Animals were procured from Central animal house, Vaageswari College of Pharmacy ,Karimnagar (CPCSEA Reg. No: 1505/po/a/11). Male wistar albino rats weighing between 150 and 2000 gm were selected for the study. Female albino mice weighing between 25 gm and 30 gm were selected for the acute oral toxicity studies. The were maintained on 12 h/12 h light and dark cycle at ambient room temperature and relative humidity (50%). They were kept in propylene cages in a well-ventilated room under hygienic conditions throught the study. The animals were fed with commercial rst feed pellets and were given water ad libitum. Maintenance of animals was as per CPCSEA guidelines. All animals were carried out only after approval of IAEC [16,17].

Determination of LD50:

The herbal preparation *Salacia fruticosa* wall have been subjected to toxicity studies according to OECD guidelines and no death was found up to 3 g/kg body weight, so 10% and 20% of it i.e. 300 mg/kg and 600 mg/kg are taken for carrying pharmacological activities.

Acute Oral Toxicity Study (Acute Toxic Class Method)

A prilimenary pharmacological study was condected to assess the acute pharmacological effects and safety of the drug. Acute toxicity was conducted to determine the median lethal dose (LD_{50}) of the methanolic extracts of the leaves of *Salacia fruticosa*. The procedure was followed by OECD (organization for ethical and cooperative development) guidelines, 423(acute toxic class method). The acute toxic class method was step wise procedure with 3 animals of a single sex per step. Defined doses are used in this method (5, 50, 300, 2000 mg/kg body weight) (Table 2).

Table 2. Acute toxic class method

Group	Dose (mg/kg)	Number of animals used	Number of dead animals
1	5	3	0
2	50	3	0
3	300	3	0
4	2000	3	0

It was observed that the extract was not mortal for mice even at 2000 mg/kg dose. Hence one tenth $1/10^{th}$ of the dose (200 mg/kg) and one fifth $1/5^{th}$ of the dose (400 mg/kg) selected for further study.

STUDY OF INFLAMMATORY ACTIVITY

In-vitro Method

Egg albumin induced paw oedema [18].

Method

Test solutions (2 ml) containing different concentrations (25-800 µg/ml) of drug was mixed with 2 ml of egg albumin solution (1 mM) and incubated at 27 ± 1°C for 15 min. Denaturation was induced by keeping the reaction

mixture at 70°C in a water bath for 10 min. After cooling, the turbidity was measured at 660 nm. Percentage inhibition was calculated from control where no drug was added.

***In-vivo* Method**

1. Carrageenan Induced Paw Oedema

Material and Methods

Animals: Wistar albino rats weighing 180-200 gm of either sex were used in the study. Animals were procured from Laboratory Animal House of Vaageswari college of pharmacy Karimnagar, (CPCSEA Reg NO: 1505/po/a/11). All the animal experiments are strictly obeyed with the approval of institutional animal ethical committee. The animals are kept in polyacrylic cages and they are maintained under standard housing conditions of temperature (24-27°C) and humidity (60-65%) with 12 hours light-12 hours dark cycle. They were acclimatized for seven days. Food was provided in the form of dry pellets and water [19-23].

Equipment: Plethysmometer.

Method

All the animals received their respective doses of test drugs 1 h prior to the administration of the phlogistic agent. After 1 h, 0.1 ml of 1% solution of freshly prepared carrageenan in normal saline was injected into the plantar surface of the right hind paw of the rats. The paw volume was measured before and each hour afterwards for a period of 5 h using mercury displacement plethysmograph. The percent inhibition of edema is calculated for each different group with respect to its vehicle-treated control group**:**

$$\%\ \text{inhibition of edema} = \frac{\text{paw vol. of control- paw vol. of treated}}{\text{paw vol. of control}} * 100$$

Treatment protocol: The animals were numbered and later they are weighed and then they are divided into 6 groups with 6 animals in each as follows:

- Group I: Animals received carrageenan alone (0.1 ml of 1% solution.)
- Group II: Animals received carrageenan and 200 mg/kg body weight MESF (p.o-Per Oral)
- Group III: Animals received carrageenan and 400 mg/Kg body weight, MESF (p.o-Per Oral)
- Group IV: Animals received carrageenan and 200 mg/kg body weight, AESF (p.o-Per Oral)
- Group V: Animals received carrageenan and 400 mg/kg body weight, AESF (p.o.)
- Group VI: Animals received carrageenan and 10 mg/Kg body weight, diclofenac sodium (p.o-Per Oral)

2. Egg Albumin Induced Paw Oedema

Materials and Method

Animals: Wistar albino rats weighing 180-200 gm of either sex were used in the study. Animals were procured from Laboratory Animal House of Vaageswari college of pharmacy Karimnagar, (Reg. no.:/ac/CPCSEA).. All animal experiments strictly complied with the approval of institutional animal ethical committee. The animals are kept in polyacrylic cages andthey are maintained under standard housing conditions of temperature (24-27°C) and humidity (60-65%) with 12 hour light-12 hour dark cycle. They were acclimatized for seven days. Food was completely provided in the form of dry pellets and water.

Equipment: Plethysmometer.

Method

Acute inflammation was induced by the injection of 0.1 ml of fresh egg albumin into the sub plantar surface of the right hind paw of the rats. Test samples were administered 1 h prior to the phlogistic agent. Oedema was assessed for 5 h at 1 h intervals, an increase in paw volume of the albumin injected paw compared with the non-injected paw. The percent inhibition of edema as calculated for each group with respect to its vehicle-treated control group**:**

$$\% \text{ inhibition of edema} = \frac{\text{paw vol. of control- paw vol. of treated}}{\text{paw vol. of control}} * 100$$

Treatment protocol: The animals were numbered, weighed and then divided into 4 groups with 5 animals in each as follows:

- Group I: Animals received Egg albumin alone (0.1 ml of 1% solution)
- Group II: Animals received egg albumin and 200 mg/kg body weight MESF- Methanolic extract of *Salacia fruticosa* (p.o-Per Oral)
- Group III: Animals received egg albumin and 400 mg/Kg body weight, MESF - methanolic extract of *Salacia fruticosa* (p.o-Per Oral)
- Group IV: Animals received egg albumin and 200 mg/kg body weight, AESF- Aqueous extract of *Salacia fruticosa* (p.o-Per Oral)
- Group V: Animals received egg albumin and 400 mg/kg body weight AESF- Aqueous extract of *Salacia fruticosa* (p.o. -Per Oral)
- Group VI: Animals received egg albumin and 10 mg/Kg body weight, diclofenac sodium (p.o. -Per Oral)

3. Adjuvant Induced Arthritis in Rats

Arthritis was induced by injecting 0.05 ml of 0.5%w/v suspension of killed mycobacterium tuberculli in paraffin oil in to the right hind limb. Paw volume was measured till 12th day using mercury displacement plethysmograph. During treatment was started on day 13 and terminated on day 21. The difference in paw volume on day 3 and day 21 were considered, as oedema volume and % inhibition of oedema was determined [24,25].

Treatment protocol: Animals were numbered, weighed and divided into 5 groups with 6 animals in each group:

- Group I - Normal animals received normal saline.
- Group II - Arthritis induced animals received normal saline (10 ml/kg p.o-Per Oral)
- Group III - Arthritis induced animals treated with MESF- Methanolic extract of *Salacia fruticosa* at dose of 200 mg/kg in normal saline.
- Group IV- Arthritis induced animals treated with Aqueous extract of *Salacia fruticosa* (AESF) at a dose of 200 mg/kg in normal saline.
- Group V - Arthritis induced animals treated with indomethacin at a dose of 10 mg/kg in normal saline.

Acute Toxicity Study

Since no mortality was observed in the mice in acute toxicity study at 2000 mg/kh body weight, and based on the literature available, tha dose of methanolic extract and aquoes extract of *Salacia fruticosa* was fixed as 200 mg/kg and 400 mg/kg b.w., for further study.

RESULTS AND DISCUSSION

Anti-inflammatory Activity

Anti inflammatory activities are given in Table 3 and Figure 5.

Table 3. ***In-vitro*** **method Egg induced paw oedema**

	% Inhibition		
Concentration (µg/ml)	MESF	AESF	Indomethacin
25	4.75 ± 0.03	6.25 ± 0.01	27.91 ± 0.02
50	12.24 ± 0.01	12.55 ± 0.01	40.85 ± 0.02
100	22.58 ± 0.01	20.27 ± 0.01	57.50 ± 0.01
200	30.29 ± 0.01	27.29 ± 0.12	66.23 ± 0.02
400	42.66 ± 0.01	38.29 ± 0.09	79.39 ± 0.01
800	57.37 ± 0.01	54.03 ± 0.01	86.34 ± 0.01

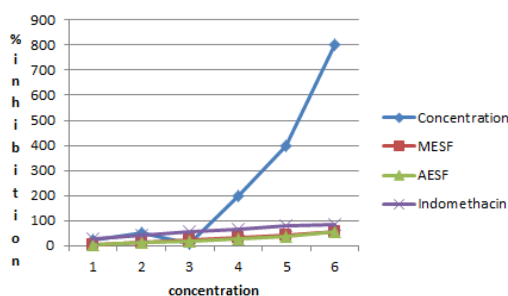

Figure 5. ***In-vitro*** **Anti-Inflammatory Activity**

In-vivo Method

Carrageenan Induced Paw Oedema is shown in Tables 4-6 and Figures 6-8.

Table 4. Carrageenan Induced Paw Oedema

Treatment	1 h	2 h	3 h	4 h	5 h
Control	0.20 ± 0.04	0.31 ± 0.06	0.34 ± 0.01	0.39 ± 0.01	0.41 ± 0.01
Diclofenac Sodium	0.19 ± 0.04	0.29 ± 0.08*	0.27 ± 0.01*	0.24 ± 0.08**	0.21 ± 0.08***
MESF	0.21 ± 0.01	0.30 ± 0.04	0.29 ± 0.09*	0.27 ± 0.08*	0.24 ± 0.08**
MESF	0.19 ± 0.02	0.29 ± 0.01*	0.28 ± 0.04*	0.26 ± 0.06**	0.22 ± 0.06***
AESF	0.21 ± 0.01	0.31 ± 0.04	0.30 ± 0.09	0.29 ± 0.08*	0.27 ± 0.08*
AESF	0.20 ± 0.01	0.30 ± 0.02	0.29 ± 0.04*	0.27 ± 0.01*	0.25 ± 0.01**

Values are mean ± SEM of 6 animals per group ; (n=6); $^{*}p < 0.05$, $^{**}p < 0.01$ $^{***}p<0.001$ vs. control (ANOVA) with Dunnet's t-test.

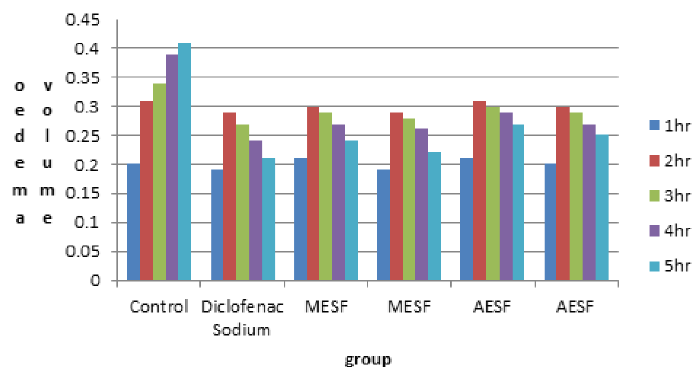

Figure 6. Effect of mesf and aesf on carrageenan induced paw oedema

Table 5. Egg albumin induced paw oedema

Treatment	1 h	2 h	3 h	4 h	5 h
Control	0.22 ± 0.01	0.38 ± 0.02	0.43 ± 0.02	0.44 ± 0.03	0.46 ± 0.03
Diclofenac sodium	0.22 ± 0.03	0.34 ± 0.01*	0.31 ± 0.04**	0.28 ± 0.01**	0.23 ± 0.02***
MESF	0.23 ± 0.01	0.36 ± 0.03*	0.34 ± 0.03*	0.31 ± 0.03**	0.28 ± 0.02**
MESF	0.22 ± 0.04	0.35 ± 0.04*	0.31 ± 0.04**	0.29 ± 0.03**	0.24 ± 0.03***
AESF	0.24 ± 0.05	0.37 ± 0.02	0.35 ± 0.03*	0.33 ± 0.02**	0.29 ± 0.01**
AESF	0.22 ± 0.01	0.36 ± 0.04*	0.32 ± 0.02*	0.30 ± 0.03**	0.25 ± 0.03 ***

Values are mean ± SEM of 6 animals per group; (n=6); *p<0.05, **p<0.01, ***p<0.001 vs. control (ANOVA) with Dunnet's t-test.

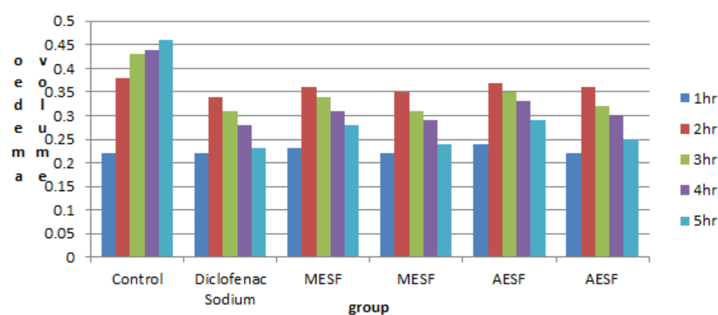

Figure 7. Effect of mesf and aesf on egg albumin induced paw oedema

Table 6. Adjuvant induced arthritis

Treatment	Initial	After day 3	After day 21
Control	0.95 ± 0.05	1.55 ± 0.05	1.50 ± 0.05
Indomethacin	0.95 ± 0.09	1.55 ± 0.06	1.11 ± 0.09***
MESF	0.96 ± 0.06	1.54 ± 0.07	1.16 ± 0.05**
MESF	0.96 ± 0.08	1.57 ± 0.08	1.14 ± 0.05***
AESF	0.95 ± 0.05	1.56 ± 0.08	1.17 ± 0.04**
AESF	0.96 ± 0.06*	1.59 ± 0.05	1.15 ± 0.06**

Values are mean ± SEM of 6 animals per group ; (n=6); *p<0.05, **p<<0.01, ***p<0.001 vs. control (ANOVA) with Dunnet's t-test.

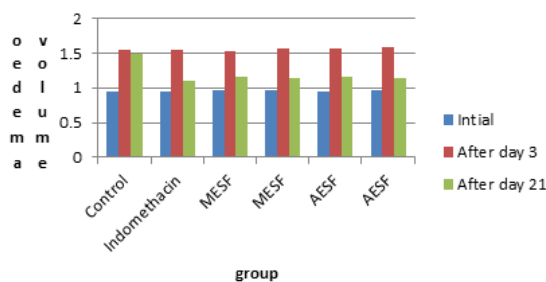

Figure 8. Graphical representation of group vs. volume of oedema

CONCLUSION

In *in-vitro* anti-inflammatory activity, both the extracts (methanolic and aqueous) showed inhibitory effect on albumin denaturation but less when compared with the standard drug indomethacin.

In *in-vivo* anti-inflammatory activity, it has been found that methanol and aqueous extracts have good activity can comparable with the standard drug diclofenac at the dose of 400 mg/kg body wt. of rat.

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