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Original Research Article

Study of anti-inflammatory activity of *Ficus racemosa linn* stem bark extract in albino rats

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ABSTRACT

Background: Inflammation is part of the complex biological response of vascular tissues to harmful stimuli. Though there are standard anti-inflammatory drugs like aspirin, diclofenac, etc., these are not free of side effects. This has led to an increase in demand for natural products with anti-inflammatory activity having less side effects. Hence the study was conducted to evaluate the anti-inflammatory activity of ethanolic extract of *Ficus racemose* (EEFR) in albino rats. **Methods:** Healthy albino rats of either sex were divided into 4 groups of 6 animals each. Group1-control, group 2-diclofenac sodium 2 mg/kg and group 3 and 4 EEFR (200 and 400 mg/kg respectively), anti-inflammatory activity was evaluated by Carrageenan induced paw oedema: formalin induced-peritonitis and cotton pellet induced granuloma model for in vivo activity and protein denaturation test for in vitro activity.

Results: EEFR exhibited significant *in vitro* (p<0.001) anti-inflammatory effect at the dose of 200 and 400 mg/kg. EEFR produced 61.37% inhibition at the dose of 400 mg/kg and diclofenac (standard drug) produced 62.95% of inhibition after 3 hours of drug treatment in carrageenan induced paw oedema. The exudate volume was decreased in formalin induced peritonitis by EEFR and diclofenac significantly (p<0.001). In cotton pellet induced granuloma EEFR (400 mg/kg) and diclofenac showed decreased formation of granuloma by 28.36% and 28.00% (p<0.001) respectively. **Conclusions:** EEFR has significant anti-inflammatory activity in both acute and chronic model in a dose dependant manner in comparison with standard drug.

Keywords: Anti-Inflammatory agents, Carrageenan, Diclofenac, Edema, Ficus, Granuloma, Peritonitis

INTRODUCTION

Inflammation is a protective attempt by the organism to remove the injurious stimuli and to initiate the healing process. However, inflammation is a stereotyped response, and therefore it is considered as a mechanism of innate immunity, as compared to adaptive immunity, which is specific for each pathogen.¹ Although it is a defence mechanism, the complex events and mediators involved in the inflammatory reaction can induce, maintain or aggravate many diseases.² Inflammatory diseases such as rheumatoid arthritis, inflammatory bowel disease, multiple sclerosis and other connective tissue diseases are a major cause of morbidity. Though there are standard drugs like Aspirin, indomethacin, phenylbutazone, etc. these drugs are not entirely free of side effects and have their own limitation.³

This has led to an increase in demand for natural products with anti-inflammatory activity having less side effects. Traditional and folk remedies have provided us with important drugs in the treatment of many diseases and are being increasingly subjected to scientific study. The family of anti-inflammatory drugs is no exception, to this. Salicylates had their origin in the 'willow bark' of folk medicine, paracetamol, cortisone, gold salts, and phenylbutazone made their way into clinical medicine serendipitously.³

Ficus racemosa commonly known as figs in English, Udumbar in Sanskrit, Gular in Hindi and Atti in Kannada is a widely cultivated plant all over India.

The stem bark is used to treat menorrhagia, leucorrhoea, gonorrhoea, urinary diseases, haemorrhage and skin diseases.⁴ Ethanol extract of stem bark inhibited COX-1 with IC50 value of 100 ng/ml proves that the drug is used in the treatment of inflammatory conditions.⁵

The bark is highly efficacious in threatened abortion and also recommended in urological disorders, diabetes, hiccough, leprosy, dysentery and piles.⁶ The literature survey reveals inadequate study on the anti-inflammatory activity of bark extract of *Ficus racemosa*. This prompted to investigate the anti-inflammatory activity of bark extract of *Ficus racemosa* in acute and chronic experimental models in albino rats.

METHODS

Animals

Laboratory bred albino rats of either sex, weighing 150-200 g were used for the study. The animals were maintained under standard laboratory conditions at 25°C, commercial pellet diet with water ad libitum and normal photo period (12-hours dark/12-hours light). Experimental protocol has been approved by the institutional animal ethics committee.

Drugs and chemicals

Diclofenac, carrageenan, formalin, ethanolic extract of *Ficus Racemosa* stem bark, ether, alcohol, gum acacia, distilled water.

Equipment

Digital Plethysmograph, oral gavage, syringes (5 and 2 ml), tuberculin syringe, dissection set, animal stand, bell jar, mortar and pestle, gloves and mask.

Extraction procedure of Ficus racemosa

Plant material

The dried stem bark of *Ficus racemosa* was procured from authentic suppliers from Bangalore, Karnataka and was identified by Mrs. Chandrika L., botanist, department of botany, PES university, Bangalore.

Procedure for extraction

About 1 kg shade dried stem bark powder of *Ficus* racemosa was subjected to extraction first with ethanol

(95%) in a Soxhlet apparatus (55C; 25-35 cycles) and then the dried bark was macerated with water for 3 days with distilled water containing chloroform (2.5 ml/1000 ml) to remove any impurities and the residue was concentrated by filtration to obtain the ethanolic extract of *Ficus racemosa* (EEFR). The yield of the extract was 9.5%.⁷

Establishment of dose of the extract

Based on the results obtained from earlier study by Prasanna Krishna V. where acute toxicity tests done in rats using OECD 423 guidelines have proven the LD50 of EEFR to be more than 2000mg/kg B. W., accordingly the dose of ethanolic extract of *Ficus racemosa* stem bark for anti-inflammatory activity was fixed to be 200 mg/kg and 400 mg/kg body weight.⁸

Inclusion criteria

Healthy male and female albino rats of 90-120 days old having weight ranges 150-200 gm were included in the study.

Exclusion criteria

Pregnant and diseased animals are excluded from the study.

In vitro anti-inflammatory activity

Protein denaturation was performed as described (Elias et al) with slight modifications. Test solution consisting of 1 ml of different concentrations of *Ficus racemosa* extract ranging from 200 and 400 mg/ml and standard diclofenac sodium 1 and 2 mg/ml was mixed with 1ml of egg albumin solution (1 mM) and incubated at $27\pm1^{\circ}$ C for 15 minutes. Denaturation was induced by keeping the reaction mixture at 70°C in a water bath for 10 minutes. After cooling the turbidity was measured spectrophotometrically at 660 nm. Percentage inhibition of denaturation was calculated from control where no drug was added. Each test was done in triplicate. ⁹

Percent inhibition=100 × [Vt/Vc-1]

Where, Vt=absorbance of test sample, Vc=absorbance of control.

Statistical analysis

Data is represented as mean \pm SD, which is statistically analysed by ANOVA test and p<0.001 considered to be significant.

In vivo anti-inflammatory activity

It is of *Ficus racemosa* was assessed by using adult albino rats of either sex weighing about 150-250 gm. The animals were maintained on standard pellet diet and water ad libitum. Overnight fasted albino rats were divided into four

groups of 6 animals each and were treated as follows. Group 1: Control, receive vehicle (gum acacia), group 2: Standard drug diclofenac sodium 0.2 mg/100 gm B. W, group 3: Test 1 EEFR low dose, 20 mg/100 gm B. W and group 4: Test 2 EEFR high dose, 40 mg/100 gm B. W.

Models used

Acute inflammation

Carrageenan induced paw oedema in rats and formalin induced peritonitis in rats.

Chronic inflammation

Cotton pellet induced granuloma in rats.

Carrageenan induced paw oedema in rats¹⁰

Inflammation was produced by sub plantar injection of 0.1 ml of freshly prepared 1% carrageenan in normal saline in right hind paw of the rat. Paw volume was measured by digital Plethysmometer at 0 and 3 hours after carrageenan injection. Animals were premedicated either with vehicle, *Ficus extract* or diclofenac orally one hour before carrageenan injection. Mean increase in paw volume was measured and percentage inhibition in standard and test group was calculated by using the below mentioned formula.

Percent oedema inhibition=(Vc-Vt)/Vcx100

Where, Vc-Mean oedema volume in the control group, Vt-Mean oedema volume in the test group.

Formalin induced-peritonitis in rats¹¹

The method of Teotino et al was adopted here to study the acute inflammatory reaction induced by formalin injected into the peritoneal cavity of the rats. All the rats were fed orally with the respective drug and one hour later peritonitis was induced by intra peritoneal administration of 1 ml of 1% formalin. After 4 hours the rats were sacrificed and the abdominal cavity was opened. The exudates were collected and measured by placing the animal on a glass funnel.

The anti-inflammatory activity of the drugs was calculated by the following formula,

Percent anti-exudate activity=100 (1-Vt/Vc)

Where, Vc= mean volume of exudates in the control group, Vt=mean volume of exudates in the test group.

After four hours peritoneal fluid which was collected and measured was subjected to peritoneal fluid analysis, which was processed by centrifugation at 2000 rpm for 10min and RBCs were destroyed. The total leukocyte count was determined using Neubauer chamber.

Cotton pellet induced granuloma in rats¹⁰

The method of Winter and Porter with slight modification was used to study chronic inflammation. 55 Four groups of six animals in each group were taken, anaesthetized with ether. The axillary skin was shaved and disinfected with 70% ethanol. An incision was made and by a blunt forceps subcutaneous tunnels were formed and a sterilized cotton pellet of 20 mg was placed in both axillae. The vehicle, test drug and standard drug were administered for 7 consecutive days starting from day of cotton implantation. At 8th day rats were anaesthetized again and the cotton pellet (along with granular tissue formed around) were removed surgically and freed from extraneous tissue. The pellets were dried in an incubator at 60°C until a constant weight was obtained. Mean increase in the weight of cotton pellet was considered as weight of granulation tissue.

Percentage inhibition of granulation formation in standard group and test group was calculated by:

Percentage inhibition=[Wc-Wd/Wc]x100

Wc=Difference in pellet weight of the control group, Wd=Difference in pellet weight of the drug treated group.

Statistical analysis

Statistical analysis of data was done using one-way analysis of variance (ANOVA) followed by post-Hoc Tukey test using the software namely SAS 9.2, SPSS 15.0, Stata 10.1, MedCalc 9.0.1, Systat 12.0 and R environment ver 2.11.1 and Microsoft word and excel have been used to generate graphs, tables etc. P value<0.05 was considered statistically significant.

RESULTS

Table 1 shows invitro anti-inflammatory activity of *Ficus racemosa* stem bark with reference to diclofenac sodium using protein denaturation test, EEFR (20 and 40 mg/100 gm B. W.) showed significant inhibition of denaturation of egg albumin in a concentration dependent manner.

The EEFR at concentration of 40 mg/100 gm B. W and diclofenac at concentration of 2 mg/ml showed maximum inhibition of protein denaturation of 71.32 and 73.52% respectively when compared with control, proving the anti-inflammatory activity of EEFR.

In Carrageenan induced rat paw oedema (Figure 1) inhibition method, Table 2 the extract produced dose dependent inhibition of oedema. At a dose of 20 and 40 mg/100 gm B. W. the extract showed 54.49 and 61.37% inhibition of paw oedema respectively (p<0.001) in comparison with the control group. The standard group

treated with diclofenac inhibited oedema by 62.95% (p<0.001).

Table 1: Protein denaturation test.

Group	Absorbance at 660 nm	Inhibition of denaturation (%)
Control	1.36±0.006	-
Diclofenac 1 mg/ml	0.72±0.007**	47.05
Diclofenac 2 mg/ml	0.36±0.007**	73.52
EEFR 20 mg/100 gm B. W.	1.04±0.17**	23.52
EEFR 40 mg/100 gm B. W.	0.39±0.005**	71.32

All values are expressed as mean \pm SD, n=3, **p<0.001. SD: standard deviation.

Table 2: Effect of EEFR on carrageenan induced ratpaw oedema.

Group	Baseline paw vol (ml)	Increase in paw volume (ml) after 3 hours	Edema inhibition (%)
Control	0.93 ± 0.05	1.56 ± 0.06	-
Diclofenac (0.2 mg/100 gm B.W.)	1.03±0.07	1.26±0.07**	62.95±2.7
EEFR- (20 mg/100 gm B. W.)	1.05±0.05	1.34±0.06**	54.49±6.5
EEFR- (40 mg/100 gm B.W.)	1.04±0.05	1.29±0.06**	61.37±1.6

All values are expressed as mean \pm SD, n=6, **p<0.001. SD: standard deviation.



Figure 1: Carrageenan induced paw oedema in rat.

Table 3 showing percentage inhibition of exudate volume in EEFR was maximum 51.71% (p<0.001) with high dose

(40 mg/100 gm B. W.) where as in standard group it was 63.33% (p<0.001).

Table 4, total leukocyte count in formalin induced peritonitis model shows that EEFR is effective in reducing peritonitis induced by formalin (Figure 2).

Table 5 showing cotton pellet induced granuloma in rats (Figure 3), EEFR significantly decreased the dry weight of the granuloma when compared to the control group. The percentage inhibition of extract was highest at the dose of 40 mg/100 gm B. W. 28% (p<0.001) which was comparable to that of diclofenac 28.36% (p<0.001).

Table 3: Peritoneal exudate volume in formalin induced peritonitis model.

Group	Peritoneal exudate vol (ml)	Inhibition (%)
Control	3.22±0.29	-
Diclofenac (0.2 mg/100 gm B.W.)	1.18±0.26**	63.33±8.25
Test 1-EEFR (20 mg/100 gm B. W.)	2.27±0.25**	29.38±7.80
Test 2-EEFR (40 mg/100 gm B. W.)	1.55±0.15**	51.71±4.71

All values are expressed as Mean \pm SD, n=6, **p<0.001. SD: standard deviation.

Table 4: Total leukocyte count in formalin induced peritonitis model.

Group	Total leukocyte count (cells/cu.mm)	Inhibition (%)
Control	4833.33±480.28	-
Diclofenac (0.2 mg/100 gm B.W.)	1316.67±163.30**	72.75±3.38
Test 1-EEFR (20 mg/100 gm B. W.)	1850.00±178.89**	61.72±3.70
Test 2-EEFR (40 mg/100 gm B. W.)	1375.00±175.36**	71.55±3.63

All values are expressed as mean \pm SD, n=6, **p<0.001. SD: standard deviation.



Figure 2: Formalin induced-peritonitis in rat.

Group	Initial weight of cotton pellet (mg)	Weight of cotton pellet after 7 days (mg)	Inhibition (%)
Control	40.00±0.0	88.96±7.61	-
Diclofenac (0.2 mg/100 gm B.W.)	40.00±0.0	63.73±4.20**	28.36±4.72
Test 1-EEFR (20 mg/100 gm B. W.)	40.00±0.0	82.47±4.37	7.29±4.91
Test 2-EEFR (40 mg/100 gm B. W.)	40.00±0.0	64.05±1.33**	28.00±1.48

Table 5: Cotton pellet-induced granuloma in rats.

All values are expressed as mean \pm SD, n=6, **p<0.001. SD: standard deviation.



Figure 3: Cotton pellet granuloma.

DISCUSSION

Inflammation is a reaction of the body against an aggressive agent, characterized by vasodilatation and access of fluid and cells to the target tissue.¹² Inflammatory mediators such as cytokines, histamine, serotonin, leukotrienes and prostaglandins, increase the vascular permeability and migration of leukocytes to inflamed tissue.¹³ In addition, different pathologic processes, such as cardiovascular and metabolic disorders, peptic ulcer and cancer are related to inflammation.¹⁴⁻¹⁸ Although nonsteroidal anti-inflammatory drugs are widely employed for controlling inflammatory disorders, adverse effects (e.g., gastric perforations, stomach ulcers and cardiovascular complications) are commonly observed, leading to increasing interest in research on the development of alternative tools that exhibit novel mechanisms of action from different origins.19-21

Hence, present study was done to screen acute and chronic anti-inflammatory activity of ethanolic extract of *Ficus racemosa* stem bark in both *in vitro* and *in vivo* models. There are studies showing anti-inflammatory activity of *Ficus racemosa* leaves, but not on stem bark.²²⁻²⁴

In this study anti-inflammatory activity of *Ficus racemosa* stem bark was studied with reference to diclofenac sodium using *in vitro* and *in vivo* anti-inflammatory models.

In protein denaturation test EEFR (200 and 400 mg/ml) showed significant inhibition of denaturation of egg albumin in a concentration dependent manner.

The EEFR at concentration of 400 mg/ml and diclofenac at concentration of 2 mg/ml showed maximum inhibition of protein denaturation of 71.32 and 73.52% respectively when compared with control, proving the anti-inflammatory activity of EEFR.

Denaturation of tissue proteins is one of the welldocumented causes of inflammatory and arthritic diseases. Production of auto antigens in certain arthritic diseases may be due to denaturation of proteins in vivo.^{25,26}Agents that can prevent protein denaturation therefore, can be used for anti-inflammatory drug development.

In Carrageenan induced rat paw oedema inhibition Method, the extract produced dose dependent inhibition of

oedema. At a dose of 200 and 400 mg/kg the extract showed 54.49 and 61.37% inhibition of paw oedema respectively (p<0.001) in comparison with the control group. The standard group treated with diclofenac inhibited oedema by 62.95% (p<0.001).

In a previous study by Sunil ethanol extract of *Ficus* racemosa stem bark at the maximum dose (500 mg/kg) showed comparatively significant (p<0.01) inhibition of paw volume in carrageenan and egg albumin induced paw oedema method to that of standard diclofenac sodium (100 mg/kg).²⁷ In another study by Mehtaa aqueous extract of *Ficus* racemosa stem bark at a dose of 20 mg and 40 mg/100 gm showed significant anti-inflammatory activity.²⁸

The development of carrageenan-induced edema has two phases; the early phase is attributed to the release of histamine, serotonin and kinins and the late phase is related to the release of prostaglandins and bradykinins.²⁹⁻³² TNF- α is also a mediator of carrageenan-induced inflammation and is able to enhance the further release of kinins and leukotrienes.³³

On the basis of the time of action of ethanolic extract of *Ficus racemosa* stem bark it is inferred that the inhibitory effect of carrageenan induced inflammation could be due to inhibition of the enzyme cyclo-oxygenase leading to inhibition of prostaglandin synthesis, similar to standard anti-inflammatory drug diclofenac.

Formalin when injected into peritoneal cavity irritates the peritoneum and bowel and induces acute inflammatory reaction producing aseptic peritonitis. According to a previous study, formalin at a low dose of 1.75% induces an oedema which mainly results from a neurogenic inflammation mediated by neuropeptides such as substance P. At higher dose of 5%, formalin induces oedema which mainly depends on the release of substance P, prostanoids, 5-hydroxytryptamine and histamine.³⁴

In our study percentage inhibition of exudate volume in EEFR was maximum 51.71% (p<0.001) with high dose (400 mg/kg) where as in standard group it was 63.33% (p<0.001). The above results show that EEFR is effective in reducing peritonitis induced by formalin. There are no studies documented till date on this activity of EEFR.

The cotton pellet granuloma model has been widely employed to assess the transudative, exudative and proliferative components of chronic inflammation. There are three phases in the inflammatory response in the cotton pellet induced granuloma.³⁵ In the first phase imbibition of fluid containing low protein takes place at the site of cotton pellet implantation. In the second phase after 2-3 days of pellet implantation, exudation of fluid containing the protein takes place. In the 3rd phase, i.e., the proliferative phase, appearance of collagen, mucopolysaccharide synthesis, and increase in the number of fibroblasts around the cotton pellets occurs.³⁶

The fluid absorbed by the pellet greatly influences the wet weight of the granuloma and the dry weight correlates well with the amount of granulomatous tissue formed.³⁷

The increase in dry weight of the granuloma measures the proliferative phase due to monocyte infiltration and fibroblast proliferation that take place in chronic inflammation.

In this study, ethanolic extract of *Ficus racemosa* significantly decreased the dry weight of the granuloma when compared to the control group. The percentage inhibition of extract was highest at the dose of 400 mg/kg, 28% (p<0.001) which was comparable to that of diclofenac 28.36% (p<0.001).

This anti-inflammatory action may be due to the ability of EEFR in reducing the number of fibroblasts and inhibiting the synthesis of collagen and mucopolysaccharide, which are natural proliferative agents of granulation tissue formation.

The above results show that ethanolic extract of *Ficus racemosa* stem bark possesses significant (p<0.001) antiinflammatory activity in both acute and chronic inflammatory models. Further studies are required to confirm the active compounds responsible for antiinflammatory activity.

CONCLUSION

The study shows that ethanolic extract of *Ficus racemosa* stem bark has significant anti-inflammatory activity and is comparable with standard anti-inflammatory drug diclofenac. Thus, ethanolic extract of *Ficus racemosa* stem bark shows a promise in the development of new anti-inflammatory drugs and further studies can be considered on isolation and fractionation of active components to determine the exact constituents that are responsible for these activities.

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