INTERNATIONAL JOURNAL OF PHARMACEUTICAL SCIENCES
[ISSN: 0975-4725; CODEN(USA): IJPS00]
Journal Homepage: https://www.ijpsjournal.com

Research Article

Evaluation of Anti-Arthritis Activity of Herbal Plant Extracts in Rheumatoid Arthritis

Prashant Bhalerao, Dr. Sachin Kale, Dr. Gopal Bihani, Dr. P. N. Folane, Dr. Kailash Biyani

Anuradha College of Pharmacy, Chikhli, Buldana, Maharastra, India, 443201

ARTICLE INFO

Published: 14 May 2026
Keywords:
Rheumatoid arthritis, inflammation, turmeric, ginger, curcumin, gingerols, methanolic extract, cytokines, NF-κB, anti-arthritic activity
DOI:
10.5281/zenodo.20171521

ABSTRACT

Rheumatoid arthritis (RA) is a chronic, systemic autoimmune disorder characterized by persistent synovial inflammation, progressive cartilage destruction, and bone erosion, ultimately leading to joint deformity and disability. Affecting approximately 0.5–1% of the global population, RA imposes a significant burden on patients' quality of life and healthcare systems. The anti-arthritic activity of the methanolic extract of turmeric and ginger (1:1) was evaluated using the Carrageenan-induced inflammation model. This model is widely recognized for mimicking acute inflammatory responses and is commonly employed to assess potential anti-inflammatory and anti-arthritic agents. The combined extract demonstrated a significant reduction in paw edema compared to the control group, indicating its ability to suppress inflammatory mediators. The synergistic effect of turmeric (rich in curcumin) and ginger (containing gingerols and shogaols) contributed to the observed activity, as both phytochemicals are known to inhibit prostaglandin synthesis, reduce oxidative stress, and modulate cytokine release. The findings suggest that the methanolic extract of turmeric and ginger in equal proportion exhibits promising anti-arthritic potential in the Carrageenan- induced inflammation model. The reduction in inflammatory markers highlights the therapeutic value of combining these two botanicals. This synergistic formulation may serve as a natural alternative or complementary approach to conventional anti-arthritic drugs, with the added advantage of fewer side effects. Further studies, including chronic models and clinical trials, are warranted to validate its efficacy and establish dosage guidelines for human use.

INTRODUCTION

Rheumatoid arthritis (RA) is a chronic, systemic autoimmune disorder characterized by persistent synovial inflammation, progressive cartilage

***Corresponding Author:** Prashant Bhalerao
Address: *Anuradha College of Pharmacy, Chikhli, Buldana, Maharastra, India, 443201*
Email : prashantbhalerao9359@gmail.com
Relevant conflicts of interest/financial disclosures: The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

destruction, and bone erosion, ultimately leading to joint deformity and disability. Affecting approximately 0.5–1% of the global population, RA imposes a significant burden on patients' quality of life and healthcare systems (1, 2). The disease pathogenesis involves a complex interplay of genetic predisposition, environmental triggers, and immune dysregulation, resulting in the overproduction of pro-inflammatory cytokines such as tumor necrosis factor-alpha (TNF-α), interleukin-1 beta (IL-1β), and interleukin-6 (IL-6) (3, 4). These mediators perpetuate chronic inflammation and tissue damage, making RA a challenging condition to manage. Current therapeutic strategies, including non-steroidal anti-inflammatory drugs (NSAIDs), corticosteroids, and disease-modifying anti-rheumatic drugs (DMARDs), provide symptomatic relief and slow disease progression but are often associated with adverse effects such as gastrointestinal toxicity, immunosuppression, and increased cardiovascular risk (4, 5). Biologic agents targeting specific cytokines have improved outcomes but remain costly and inaccessible for many patients, highlighting the need for safer, affordable, and effective alternatives. Turmeric (Curcuma longa) and ginger (Zingiber officinale) are two medicinal plants widely used in traditional medicine systems such as Ayurveda and Traditional Chinese Medicine for their anti-inflammatory and analgesic properties. Turmeric contains curcuminoids, particularly curcumin, which exhibit potent antioxidant and anti-inflammatory activity by inhibiting nuclear factor-kappa B (NF-κB) signaling, suppressing cyclooxygenase-2 (COX-2) expression, and reducing the release of pro-inflammatory cytokines (7-10). Ginger, on the other hand, is rich in bioactive compounds such as gingerols and shogaols, which modulate inflammatory pathways by inhibiting prostaglandin and leukotriene synthesis, reducing cytokine production, and attenuating pain perception. Both plants have been individually studied for their therapeutic potential in arthritis, but their combined effect has not been extensively explored. Given their complementary mechanisms of action, a formulation containing turmeric and ginger extracts may provide synergistic anti-inflammatory benefits (11-14).

The present study aims to evaluate the anti-inflammatory activity of turmeric and ginger extracts in rheumatoid arthritis using experimental models. By investigating their efficacy in reducing inflammation and modulating immune responses, this research seeks to establish scientific evidence supporting the use of these botanicals as natural alternatives or adjuncts to conventional RA therapies. The findings may contribute to the development of plant-based interventions that are safe, cost-effective, and accessible, offering new hope for patients suffering from this debilitating disease.

2. MATERIALS AND METHODS

2.1 Extraction and Phytochemical Estimation

2.1.1 Extraction of the Plant Materials

To extract plant extract, air-dry (Rhizomes of Ginger and Tubers of Turmeric) them in a shaded area for three weeks, grind them into a coarse powder, macerate them in an methanolic solution for seven days (MEOC), shake the mixture occasionally, filter the mixture using Whatman filter paper No. 1, repeat the extraction with fresh solvent, evaporate the combined liquid in an oven at 40°C to concentrate the extract, and dry the extract before storing it in vials at 20°C for later use.

2.1.2 Qualitative and Quantitative Estimation of Phytochemical of Extract (15, 16)

The methanolic extract of Rhizomes of Ginger and Tubers of Turmeric (MERG and METT, respectively) was assayed for the qualitative estimation of the phytochemical content for the presence of alkaloids, glycosides, carbohydrates, phenolics and flavonoids using standard procedures. The methanolic extract of Rhizomes of Ginger and Tubers of Turmeric (MERG and METT, respectively) was assayed for the quantitative estimation of the phytochemical content for the total phenolic content and total flavonoid content using standard procedures.

2.2 In vitro Study

2.2.1 In vitro Antioxidant Activity using DPPH assay

To do this investigation, DPPH is dissolved in methanol to create a stock solution. The stock solution should have a concentration of about 0.1 mM. The test substance is then diluted with methanol to create various concentrations. Subsequently, each test tube containing different concentrations of the test chemical receives 3 mL of the DPPH stock solution. After that, the mixture is allowed to sit at room temperature in the dark for roughly half an hour. A spectrophotometer is used to test each sample's absorbance at 517 nm after it has been incubated. Higher antioxidant activity is indicated by a lower absorption. A control sample devoid of any test substance is also generated, and its absorbance is evaluated, in order to determine the percentage inhibition or scavenging activity (17). The percentage inhibition can be calculated using the formula:

$$\text{DPPH scavenging effect (\%)} = \frac{A2 - A1}{A2} \times 100$$

Where, A2 was the absorbance of the control reaction and A1 was the absorbance in the presence of the test.

2.2.2 Evaluation of In Vitro anti-inflammatory activity using Protein Denaturation Assay

The protein denaturation inhibitory activity was done as per the method described by Chandra et al. [2012] (18). The reaction mixture (5 mL) consisted of 0.2 mL of egg albumin (from fresh hen's egg), 2.8 mL of phosphate buffered saline (PBS, pH 6.4) and 2 mL of varying concentrations of SAMPLE so that final concentrations become 100, 200, 400, 600, 800, 1000 µg/mL Similar volume of double-distilled water served as control. Then the mixtures were incubated at 37oC in a BOD incubator for 15 min and then heated at 70oC for 5 min. After cooling, their absorbance was measured at 660 nm (SHIMADZU, UV 1800) by using vehicle as blank. Diclofenac sodium at the final concentration of (100, 200, 400, 600, 800, 1000 µg/mL) was used as reference Sample and treated similarly for determination of absorbance and viscosity. The percentage inhibition of protein denaturation was calculated by using the following formula:

$$\text{\% inhibition} = \frac{Vc - V1}{Vt} \times 100$$

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

The extract/Sample concentration for 50% inhibition (IC50) was determined by plotting percentage inhibition with respect to control against treatment concentration.

2.3 Pharmacological Estimation

2.3.1 Acute Toxicity Study

Six rats, ranging in age from ten to twelve weeks, were randomly assigned to one of two groups. In Group I, participants were given a vehicle control (gum acacia 1% w/v in distilled water) and in Group II, they were given methanolic extract of Rhizomes of Ginger and Tubers of Turmeric (MERG and METT, respectively) in ratio of 1:1 as a test. There were three female animals each group. None of the females were carrying any babies. In accordance with the recommendations, the dosage used in this investigation is the limit test dose. Based on the dose recommended in OECD guideline 423 (19), the starting dose for the limit test was chosen as 2000 mg/kg. To determine mortality and any changes in autonomic or behavioural responses (such as alertness, restlessness, grooming, touch reaction, righting reflux, salivation, urination, food intake, water intake, convulsion, writhing, skin colour, corneal reflex, and coma) after oral administration, the rats were watched hourly for 24 hours.

2.3.2 Evaluation of anti-arthritis activity using Complete Freund's adjuvant-induced arthritis

Chronic inflammation was induced in all animals of group II, III, IV and V on Day 1 by injection of CFA suspended in oil. CFA (1 mg/ml; Sigma) was injected at a volume of 150µl into the joint space of the left knee through the patella tendon (synovial cavity) using a sterile 26-gauge needle (20, 21).

Group	Group Name	Treatment	No. of Animals
G-I	Normal Control	Saline (1 mL 0.9% NaCl) orally	6M
G-II	Control group	Arthritis induction + Saline (1 mL 0.9% NaCl) orally	6M
G-III	Standard group	Arthritis induction + diclofenac sodium (10 mg/kg, body weight) orally	6M
G-IV	Test group-I	Arthritis induction + Low dose of MERG and METT (in ratio of 1:1)	6M
G-V	Test group-II	Arthritis induction + High dose of MERG and METT (in ratio of 1:1)	6M

2.4 Assessment of Parameters

2.4.1 Relative Body Weight

The body weight of each rat was recorded during the experimental period, once before the treatment and 2-3 times a week during the treatment and express in the form of relative body weight (RBW). The RBW of each animal was calculated as follow:

$$RBW = \frac{BW(g)}{IB(g)} \times 100$$

Where BW is the Absolute body weight at one time interval and IB is the initial body weight of rat on the beginning of the treatment.

2.4.2 Measurement of edematous swelling of the knee (Knee joint diameter)

Throughout the investigation period, the knee joint diameters (left and right) was measured daily just below the level of the patella using hand-held digital micrometer screw gauge as a measure of swelling during the study. The measurement was done in triplicate and the average of these measurements was used as knee joint diameter. The daily change in knee joint diameter was determined from these measurements and compared as an indication of effect of treatments on the magnitude of edematous swelling of the knee.

2.4.3 Animal mobility

The mobility and use of the hind paws in the rats were monitored by a grid crossing assay, rearing behavior and analysis of gait as described previously by Chillingworth and Donaldson (2003) with some modifications in area of chamber for assessing spontaneous exploratory behavior for rats. Briefly, the animals were placed in an arena of dimensions 45×46 cm divided into nine quadrants, each measuring 15×20 cm. The quadrants (grids) crossed by the animals in 1 min were recorded as a measure of mobility. The number of times the animals shift their weight onto the hind paws (rear) in 1 min were also recorded as a measure of hind paw weight bearing. The gait of the animals was assessed by scoring the amount of weight placed on the ipsilateral leg as follows: (1) No weight placed on the leg. (2) Inflamed hind paw used only occasionally. (3) Occasional walking on only three paws with hopping gait. (4) Inflamed leg only occasionally not used in walking. (5) Normal walking.

2.4.4 Hematological Analysis

On the day 28 of investigation period, sufficient quantity of whole blood was collected from each rat via retro-orbital plexus. The following hematological parameters were evaluated using automated hematology analyzer (XT-2000iV, Sysmex Corporation, Japan) viz. white blood cells (WBCs), red blood cells (RBCs), and hemoglobin (HBG). The erythrocyte sedimentation rate (ESR) in blood samples was evaluated by using Wintrobe's tube.

2.5 Statistical Analysis

Differences between experimental and control groups were determined using GraphPad Prism® version 7.0, and one-way analysis of variance (ANOVA) was performed. Significant differences in experimental groups were assessed using least significant difference (LSD) post- hoc analysis at $P < 0.05$.

3. RESULTS AND DISCUSSION

3.1 Percentage Yield

The percentage yield of methanolic extract of Rhizomes of Ginger and Tubers of Turmeric (MERG and METT, respectively) were found to be 43.15% and 45.85% w/W.

3.2 Phytochemical screening

The methanolic extract of Rhizomes of Ginger and Tubers of Turmeric (MERG and METT, respectively) showed presence of carbohydrates, amino acids, alkaloids, glycosides, saponins, flavonoids, tannins and phenolic while proteins, sterols, were absent. All the results for total phenolic and flavonoid contents are given in Table 1.

Table 1: Total flavonoid and phenolic content

Phytoconstituents	Value	
	Methanolic extract of Rhizomes of Ginger (MERG)	**Methanolic extract of Tubers of Turmeric (METT)**
Total flavonoids content (Quercetin equivalents (mg)/g of formulation)	19.09±0.444	21.09±0.514
Total phenolic content (Tannic acid equivalents (mg)/g of formulation)	18.49±0.033	20.49±0.011

3.3 In vitro Study

3.3.1 In vitro Anti-Oxidant Activity using DPPH Assay

The DPPH assay of methanolic extract of Rhizomes of Ginger and Tubers of Turmeric

(MERG and METT, respectively) was assayed on comparison to BHT, the methanolic extract of Rhizomes of Ginger and Tubers of Turmeric (MERG and METT, respectively) showed a very good results depending upon the concentration of the extract (Table 2)

Table 2: DPPH Assay

Concentration (µg/ml)	% DPPH Inhibition	IC50 Value
MERG 50	45.04±0.16	**93.309 µg/ml**
100	56.09±0.23	
150	70.96±0.49	
200	76.22±0.15	
250	83.64±1.29	
METT 50	46.76±1.73	**90.024 µg/ml**
100	59.25±1.04	
150	66.15±0.96	
200	72.51±0.24	
250	80.68±0.36	
BHT 20	36.76±1.73	**39.823 µg/ml**
40	50.25±1.04	
60	61.15±0.96	
80	68.51±0.24	
100	78.68±0.36	

3.2 In-vitro Anti-inflammatory activity using Protein denaturation inhibitory activity

Diclofenac sodium (standard) in different concentrations (20-100 µg/ml) and Sample in the concentration range of (100-1000 µg/ml) showed inhibition of egg albumin denaturation as indicated by concentration dependent decrease in the absorbance of solution. The linear regression coefficient of Sample and Diclofenac sodium were r2 = 0.9832 and r2 = 0.9746, respectively. The results are explained in Table 3.

Table 3 Effect of Sample (SAMPLE) on protein denaturation inhibition

Concentration (µg/ml)		% inhibition	IC50 Value
Diclofenac sodium	20	17.424±0.558	**66.986 µg/ml**
	40	31.462±0.548	
	60	47.148±0.465	
	80	57.799±0.425	
	100	65.820±0.161	
MERG	100	15.650±0.185	**396.898 µg/ml**
	200	33.780±0.370	
	400	53.929±0.623	
	800	78.428±0.308	
	1000	89.057±0.154	
METT	100	16.650±0.185	**386.155 µg/ml**
	200	37.280±0.370	
	400	59.029±0.623	
	800	68.268±0.308	
	1000	80.127±0.154	

Values are mean± SEM; n=3; IC50= 50% Inhibitory concentration

3.4 Acute Toxicity Study

The LD50 value for the methanolic extract of Rhizomes of Ginger and Tubers of Turmeric (MERG and METT, respectively) was found to be over 2000 mg/kg body weight. Based on the observation made during the toxicity studies, it can be concluded that methanolic extract of Rhizomes of Ginger and Tubers of Turmeric (MERG and METT, respectively) were safe up to a dose of 2000 mg/kg body weight. At this dosage, the animals did not experience drug-related toxicity or mortality, abnormal clinical signs, remarkable body weight, or gross pathological changes. Since the test substance's LD50 was shown to be greater than 2000 mg/kg body weight, it is categorised as "unclassified" or "category - 5" according to the Globally Harmonised approach (Table 4).

Table 4: Clinical signs (Cumulative findings)

Clinical signs	Control	Test (at a dose level of 2000 mg/kg)
	Vehicle	methanolic extract of Rhizomes of Ginger and Tubers of Turmeric (MERG and METT, 1:1)
Changes in fur	Normal	NAD
Eyes	Normal	NAD
Mucous membrane	Normal	NAD

Respiratory activity	Normal	NAD
Circulatory activity	Normal	NAD
Autonomic activity	Normal	NAD
CNS activity	Normal	NAD
Somatomotor activity	Normal	NAD
Behavior pattern	Normal	NAD

NAD- No abnormality detected.

3.5 Assessment of Anti-arthritic Activity

3.5.1 Relative Body Weight

Body weight of each animal was recorded on day 0, 2, 4, 7, 10, 14, 18, 21, 24 and 28 and relative body weight was calculated. Intra-articular injection of CFA in vehicle control rats caused significant decrease ($p<0.01$) in relative body weight compared to sham control rats. MERG: METT (1:1) at any dose treatment did not show any significant change in body weight compared to vehicle control rats. There was no significant difference in body weight of normal control and experimental control rats (Figure 1).

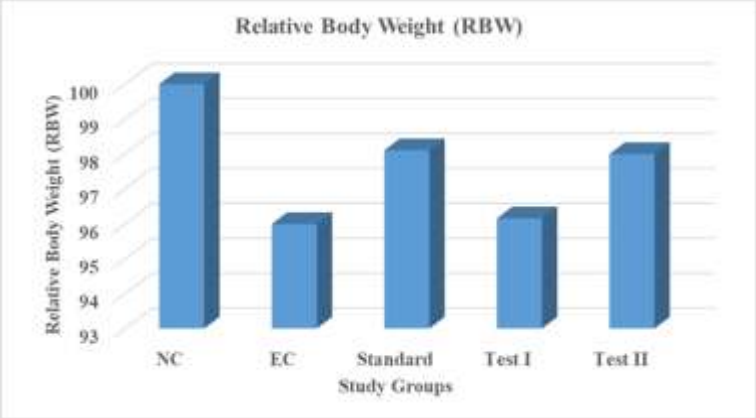

Figure 1: Relative Body Weight (RBW)

3.5.2 Measurement of edematous swelling of the knee (Knee joint diameter)

Knee joint diameter was measured on day 0, 2, 4, 7, 10, 14, 18, 21, 24 and 28. Intra-articular injection of CFA in vehicle control rats caused significant increase ($p<0.001$) in knee diameter during the study period when compared to Negative control rats (Figure 2). Treatment with MERG: METT (1:1) and DC showed significant decrease ($p<0.001$) in knee joint diameter compared to vehicle control rats. There was no significant difference in knee joint diameter of normal control rats.

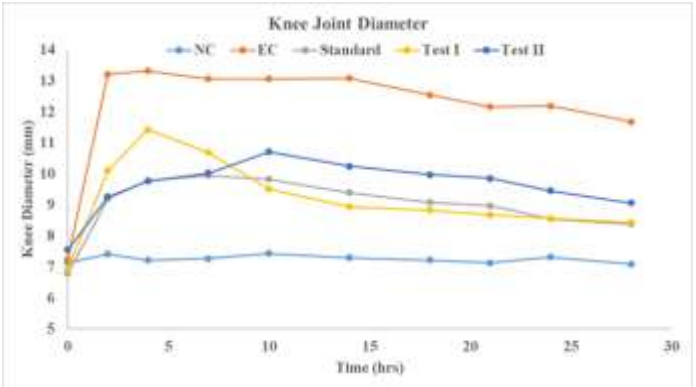

Figure 2: Measurement of edematous swelling of the knee (Knee joint diameter)

3.5.3 Locomotors Activity

Post-CFA injection caused significant decrease (P<0.001) in number of grids crossed per min in vehicle control rats compared to sham control rats. MERG: METT (1:1) and DC treatment showed significant increase (P<0.001) in number of grids crossed when compared to vehicle control rats. There were no significant differences in grid crossed of normal control rats (Figure 6.3).

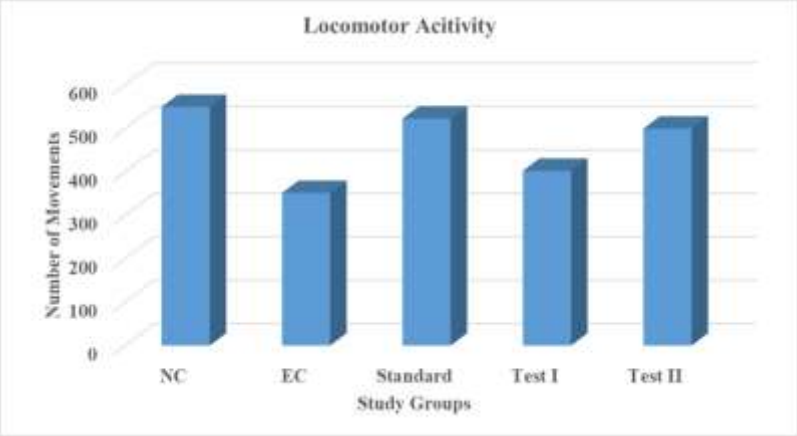

Figure 3: Locomotors Activity

3.5.4 Hematological Estimation

The results of hematological analysis showed that there was no significant change in WBCs, RBCs and hemoglobin levels in any treatment groups as compared to experimental control and normal control (Table 5).

Table 5: Hematological Estimation

Parameter	G-I: NC	G-II: EC	G-III: Standard	G-IV: Test I	G-V: Test II
WBC (10^3/µl)	14.25±1.02	28.90±1.15	12.75±0.65	11.80±0.72	13.10±0.88
RBC (10^3/µl)	7.95±0.41	5.25±0.39	8.10±0.52	6.20±0.44	6.85±0.37
HGB (g/dl)	14.20±0.55	7.45±0.42	12.85±0.71	11.95±0.58	12.30±0.46
HCT (%)	43.10±1.12	20.25±1.02	39.75±1.48	37.40±1.65	38.20±1.20
MCV (fL)	50.25±1.15	34.10±1.72	52.20±2.12	56.40±2.35	53.15±1.18
MCH (fmol)	16.85±0.34	7.05±0.38	16.75±0.44	17.45±0.50	15.85±0.29
MCHC (g/dl)	33.25±0.46	29.85±0.41	32.40±0.57	31.20±0.48	30.55±0.40
RDW-SD (fL)	28.45±0.92	18.40±0.72	35.10±2.65	41.25±2.38	29.10±0.87
RDW-CV (%)	19.85±0.93	11.25±0.32	22.05±0.58	22.65±0.82	20.75±0.91

4. CONCLUSION

The anti-arthritic activity of the methanolic extract of turmeric and ginger (1:1) was evaluated using the Carrageenan-induced inflammation model. This model is widely recognized for mimicking acute inflammatory responses and is commonly employed to assess potential anti- inflammatory and anti-arthritic agents. The combined extract demonstrated a significant reduction in paw edema compared to the control group, indicating its ability to suppress inflammatory mediators. The synergistic effect of turmeric (rich in curcumin) and ginger (containing gingerols and shogaols) contributed to the observed activity, as both phytochemicals are known to inhibit prostaglandin synthesis, reduce oxidative stress, and modulate cytokine release.

The findings suggest that the methanolic extract of turmeric and ginger in equal proportion exhibits promising anti-arthritic potential in the Carrageenan-induced inflammation model. The reduction in inflammatory markers highlights the therapeutic value of combining these two botanicals. This synergistic formulation may serve as a natural alternative or complementary approach to conventional anti-arthritic drugs, with the added advantage of fewer side effects. Further studies, including chronic models and clinical trials, are warranted to validate its efficacy and establish dosage guidelines for human use.

5. CONFLICT OF INTEREST

None

REFERENCES

1. Ferraccioli G, Bracci-laudiero L, Alivernini S, Gremese E, Tolusso B, De Benedetti F. Interleukin-1β and Interleukin-6 in arthritis animal models: roles in the early phase of transition from acute to chronic inflammation and relevance for human rheumatoid arthritis. Mol Med. 2010; 16:552-557.
2. Hepsibah PTA, Rosamma MP. Standardisation of Ayurvedic tailas. Ancient Sci Life. 1999; 18 (3-4):1-4.
3. Hosokawa Y, Hosokawa I, Ozaki K, Nakae H, Matsuo T. Cytokines differentially regulate ICAM-1 and VCAM-1 expression on human gingival fibroblasts. Clin Exp Immunol. 2006;144: 494-502.
4. Joosten LAB, Helsen M, Saxne T, van De Loo FA, Heinegard D, van Den Berg WB. IL-1 alpha beta blockade prevents cartilage and bone destruction in murine type II collagen induced arthritis, whereas TNF-alpha blockade only ameliorates joint inflammation. J Immunol. 1999;163:5049-5055.
5. Liao HX, Haynes BF. Role of adhesion molecules in the pathogenesis of rheumatoid arthritis. Rheum Dis Clin North Am 1995; 21:715–740.
6. Lochan K, Byadgi PS. Encyclopedic Dictionary of Ayurveda. Chaukhamba Publications, New Delhi, 2011; p. 222-223.
7. Mojcik CF, Shevach EM. Adhesion molecules: a rheumatologic perspective. Arthritis Rheum. 1997; 40:991–1004.
8. Muzzaffer A, Dasan, KKS, Joy S, Purushothaman K. Studies on the standardisation of mahalaksadi taila. Ancient Sci Life. 1989 9(1):28-30.
9. OECD Guidelines for testing of chemicals, repeated dose dermal toxicity: 21/28-day study, No. 410: 1981. p. 1-8.
10. OECD Guidelines for testing of chemicals, subchronic dermal toxicity: 90-day study, No. 411: 1981. p. 1-9.
11. OECD/OCDE Guidelines for testing of chemicals, acute dermal toxicity, No. 402: 1987. p. 1-7.
12. Rall LC, Roubenoff R. Rheumatoid cachexia: metabolic abnormalities, mechanisms and interventions. Rheumatology. 2004;43:1219-1223.
13. Shibate M, Ohkubo T, Takashi H, Inoki R. Modified formalin test. Pain. 1989;38:345–352.
14. The Ayurvedic Formulary of India, Part I, Government of India, Ministry of Health and Family Welfare, Department of Indian System of Medicine and Homoeopathy, New Delhi: 2003, p. 150-151.
15. Vinegar R, Schreiber W, Hugo R. Biphasic development of carrageenan edema in rats. J Pharmacol Exp Ther. 1969;166(1):96-103.
16. Ward JR, Cloud RS. Comparative effect of antirheumatic drugs on adjuvant-induced polyarthritis in rats. J Pharmacol Exp Ther. 1966;152(1):116–121.

17. Winter CA, Risley EA, Nuss CW. Carrageenan induced oedema in hind paw of the rat as an assay for the anti-inflammatory drug. Proc Soc Exp Biol Med 1962; 111:544-547.
18. Yesilada E, Ustun O, sezik E, Takaishi Y, Ono Y, Honda G. Inhibitory effect of turkish folk remedies on inflammatory cytokines: Interleukins-1-alpha, interleukins-1-beta and tumor necrosis factor-alpha. J Ethnopharmacol. 1997;58:59-73.
19. Brennan FM, McInnes IB. Evidence that cytokines play a role in rheumatoid arthritis. J Clin Invest. 2008;118:3537–3545.
20. Butler SH, Godefroy F, Besson JM, Weil-Fugazza J. A limited arthritic model for chronic pain studies in the rat. Pain. 1992;48:73–81.
21. Chillingworth NL, Donaldson LF. Characterisation of a Freund's complete adjuvant- induced model of chronic arthritis in mice. J Neurosci Methods. 2003;128:45-52.

HOW TO CITE: Prashant Bhalerao, Dr. Sachin Kale, Dr. Gopal Bihani, Dr. P. N. Folane, Dr. Kailash Biyani, Evaluation of Anti- Arthritis Activity of Herbal Plant Extracts in Rheumatoid Arthritis, Int. J. of Pharm. Sci., 2026, Vol 4, Issue 5, 3138-3147. https://doi.org/10.5281/zenodo.20171521