Original Article

Evaluation Of Anti-Inflammatory And Anti-Arthritic Activities Of Semecarpus Anacardium In Experimental Animals

Parijatha Bandigari [1]*, Dr. Ashok Dongamanti [2]

[1,2] University college of Technology, Department of Chemistry, Osmania University, Hyderabad, Telangana, INDIA - 500088.

Corresponding Author:
PARIJATHA BANDIGARI Department of Pharmaceutical Chemistry University college of Technology Department of Chemistry Osmania University, Hyderabad Telangana, INDIA.
Email: parijatha.pharmacy@gmail.com
DOI: 10.47750/pnr.2023.14.02.152

Abstract

Background: Arthritis is a joint inflammation, either temporary or persistent and may manifest in a variety of ways, each requiring a tailored approach to treatment. The objective of this research was to examine the anti-inflammatory, anti-allodynic, and anti-arthritic properties of Semecarpus anacardium nuts in carrageenan-induced paw edema and adjuvant-induced arthritis.

Methodology: The anti-inflammatory effects of S. anacardium nut extracts were determined in a rat model of inflammation using carrageenan-induced paw edema. The anti-allodynic activity was studied using von Frey test. The animal's withdrawal of its paw was seen as a positive response when von Frey filament was placed on the plantar portion of the rat's hind paw from its base. Arthritis was induced in rats through inoculation with Freund's complete adjuvant (CFA). Erythrocyte sedimentation rate (ESR), platelet count, and red blood cell count are the haematological markers evaluated to study adjuvant-induced arthritis. For evaluating the antioxidant activity, total antioxidant content (TAC) and ferrous reducing antioxidant content (FRAC) were estimated.

Results: Treatment with natural extract of S. anacardium (NESA) low and high doses and the standard drug have shown a decrease in paw volume compared to the diseased group. In von frey test, from 30 min onwards all the treatment groups have shown significant increase in paw withdrawal threshold ($p < 0.001$) when compared to carrageenan control group. Induction of arthritis caused an increase in WBC, ESR and platelet count, and decrease in RBS count in AIA control group. S. anacardium improved all of these parameters. At the concentration of 150 µg/mL, the absorbance in TAC assay was in range of 1.164 ± 0.065 - 1.694 ± 0.039, while in FRAC assay, the absorbance was in range of 1.774 ± 0.046 - 2.992 ± 0.127.

Conclusion: This research established that S. anacardium's physiologically active components, including steroids, flavonoids, polysaccharides, phenols, and terpenes, are responsible for its anti-inflammatory effects and may be able to lessen FCA-induced arthritis.

Keywords: Arthritis, anti-inflammatory activity, allodynia, anti-arthritic activity, carrageenan-induced paw edema.

1. Introduction

Inflammation is a natural and required defensive response against potential pathogens, immunogenic interactions, heat, chemicals, ischemia, physical and biological agents. It's brought on by factors like direct contact with the elements, exposure to the sun, infection, and immunological responses. Inflammation is characterized by redness, heat, swelling, and pain. Asthma, arthritic pain, multiple

sclerosis, inflammatory bowel disease, and psoriasis are all autoimmune disorders that result from chronic inflammation (**Sharif et al., 2018**). The aging of the global population has led to an increase in the incidence and severity of many of such conditions. By definition, arthritis is joint inflammation, either temporary or persistent. Pain, stiffness, reduced motion range and joint abnormalities are just some of the symptoms that arthritis may cause (**Senthelal et al., 2022**). Arthritis may manifest in a variety of ways, each requiring a tailored approach to treatment. Identifying the kind of arthritis requires a thorough history and physical examination, with further testing and imaging occasionally being required for confirmation (**Bardin et al., 2017**). A number of joints may be affected by arthritis. Fewer than one percent of people worldwide have it, usually women in more developed nations. Deterioration of the joints is brought on by inflammation and may be slowed with therapy. Hip pain, decreased mobility, and even permanent damage may result from this degenerative condition (**Buckwalter et al., 2004**), which affects 9.6% of men and 18% of women over the age of 60.

Numerous studies have examined different S. anacardium components for their potential to reduce inflammation and alleviate arthritis symptoms. The objective of this research was to examine the anti-inflammatory, anti-aalodynic, and anti-arthritic properties of S. anacardium nuts in carrageenan-induced paw edema and adjuvant-induced arthritis.

2. Material and methods

2.1 Collection and extraction of drug

Dried nuts of the Semecarpus anacardium Linn. tree were purchased from vendors in Hyderabad, Telangana, India. The fruits' authenticity was confirmed by the Botany Department at Osmania University in Hyderabad. It is a natural method it requires no harmful chemicals or reagents and no metal based vessels for extraction process. It requires two pots and Dung cakes. Firstly proper place for extraction process was selected and cleaned .The selected area was dugged up to half meter then placed one pot inside the dugged area. The opening of the pot was kept up side then the surrounding area was filled with sand. In another pot Marking nut seeds were taken it was placed upside down on the dugged pot, in between these two pots wire mesh was placed .The gap between the two pots sealed with wet clay to prevent leakage of extract. Flame supplied to the seeds by using dung cakes. The flame passed to the seeds and melted inside the pot. The melted extract passed to the underground pot through the mesh. The process was continued for three hours. After three hours the flame was removed and kept it for next day morning. The crude extract which was present in the down pot was collected.

2.2 Animals

Male Wistar rats weighing about 150-200 g each were used for the study. They were fed with standard pellet diet and were supplied with water ad libitum, housed under 12h light/dark cycle, with controlled temperature (22-25^{0}C). Animals were acclimatized for atleast one week before the start of experiment. Care of the animals and experimental procedures were done according to the guidelines of Institutional Animal Ethics Committee (IAEC). This project was approved by IAEC with protocol number (xx

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2.2 Experimental Method (Carrageenan Induced Rat Paw Edema)

The anti-inflammatory effects of S. anacardium nut extracts was determined in a rat model of inflammation using carrageenan-induced paw edema.

30 male Wistar rats were separated into seven groups, each having six rats. Group 1 received saline as vehicle, Group 2 received carrageenan and vehicle, Group 3 received 100 mg/kg p.o. NESA, Group 4 received 200 mg/kg p.o. NESA, and Group 5 received 100 mg/kg p.o. diclofenac sodium as standard drug.

Initially, animals were given medications (vehicle, standard, and treatments) based on the groups described above. In order to cause edema, 0.1 mL of a 1% solution of carrageenan was administered

subcutaneously into the subplantar area of the right hind paw 1 hour after the aforementioned treatment. The volume of the paws increased as a result of the carrageenan injection, which was used to quantify the edema. We used a Plethysmometer to determine the volume of the paws at 3 and 5 hours following carrageenan treatment. This formula was used to determine the proportion of reduction of paw thickness:

$$\text{Inhibition of Paw Thickness (\%)} = 1 - \frac{V_t}{V_c} \times 100$$

Where, V_t represents the mean relative increase or decrease in paw volume in experimental groups and V_c represents the mean relative increase or decrease in paw volume in the control group (**Patil et al., 2011, and Karim et al., 2019**).

Diclofenac sodium 100 mg/kg, was employed as a standard.

2.3 Paw withdrawal threshold (von Frey Test)

The rats were tested for mechanical allodynia by placing them one at a time on an elevated labyrinth apparatus housed in an acrylic cage. The rats were left there for at least 15 minutes. Von Frey filament was placed on the plantar portion of the rat's hind paw from its base. The filament was pressed hard enough against the paw to cause a little bend, and it was held for a brief moment. The animal's withdrawal of its paw was seen as a positive response (**Sanklecha et al., 2017; Micheli et al., 2021**).
The standard medicine used was oxycodone, and it was given at a dosage of 150 micrograms per 0.1 milliliter per kilogram (μg/mL/kg) after being dissolved in 0.9% sodium chloride.

2.4 Adjuvant-Induced Arthritis (AIA) in Rats

30 male Wistar rats (Grouped as discussed before) were used for the induction of arthritis through inoculation with Freund's complete adjuvant (CFA). On day 0, after being anesthetized with a ketamine and xylazine combination (80:10 mg/kg, i.p.), rats received an injection of 0.1 ml CFA. Intradermal injection of 1 mg/mL of heat-inactivated M. tuberculosis in 85% paraffin oil and 15% mannide monooleate (Sigma Aldrich, St. Louis, MO, USA) was given near the base of the tail. The animals in the control group received injections of saline at the same volume. The animals were divided into the following groups: Control (no adjuvant, saline), AIA (adjuvant, no treatment), NESA (100) (adjuvant, 100 mg/kg NESA), NESA (200) (adjuvant, 200 mg/kg NESA, 0.1 mg/kg methotrexate (MTX, Sigma Aldrich, St. Louis, MO, USA) as standard drug, it is the most utilized antirheumatic drug and hence was used as a standard drug administered p.o. Once the initial injection was administered, treatment continued every day for 27 days (**Zhang et al., 2015**). We took blood samples from the retro-orbital plexus to analyze for hematological characteristics. Erythrocyte Sedimentation Rate (ESR), Platelet Count, and Red Blood Cell Count are the haematological markers evaluated (**Tatiya et al., 2017**).

2.5 In vitro Models

2.5.1 Determination of total antioxidant capacity (TAC)

Rahman et al., 2015 provided a technique for calculating TAC, which was used to analyze the samples. Principally, this test relies on the drugs/samples reducing Mo (VI) to Mo (V), resulting in the development of a green phosphate/Mo (V) complex in an acidic condition. 3ml of the combination of 0.6 M sulphuric acid, 28 mM sodium phosphate, and 1% ammonium molybdate was added to sample/standard solution ranging in concentration from 12.5 to 150 μg/mL. For 10 minutes, the test tubes holding the aforementioned combinations were heated to 95 degrees Celsius to facilitate the completion of the reaction. The sample's absorbance was assessed against a blank solution at 695 nm when the reaction mixture had cooled to normal temperature. Catechin served as a standard. A blank solution was created by adding the same amount of solvent (3 mL) to 3 mL of the reaction mixture that was used to dissolve the samples and the standard. The blank sample was likewise subjected to the same incubation conditions (ten minutes at 95°C, after which the absorbance was measured at 695 nm). Enhanced overall antioxidant capability is shown by a higher absorbance value. For every antioxidant experiment, standards and samples were employed throughout a concentration range of 12.5–150 μg/mL. The concentrations were

chosen by trial and error in order to find the range of values that would adequately reflect the expected variation in antioxidant activity when sample concentration was increased.

2.5.2 Ferrous reducing antioxidant capacity assay (FRAC)

Rahman et al., 2015 technique was used to determine the FRAC of the samples. Perl's Prussian blue's development at 700 nm may be used as a tracer of Fe^{2+}. The following were included in each test tube: 0.25 Ml of a 12.5–150 μg/mL standard/sample solution, 0.625 mL of a 0.2 M potassium buffer, and 0.625 mL of a 1% potassium ferricyanide solution. To finish off the process, the aforementioned combinations were heated to 500 degrees Celsius for 20 minutes. Afterwards, 0.625 mL of a 10% TCA solution was poured into the test tubes. After centrifuging the mentioned combination at 3000 rpm for 10 minutes, 1.8 mL of the supernatant was removed and combined with 1.8 mL of distilled water and 0.36 mL of 0.1% ferric chloride solution in separate test tubes. Absorbance was determined by comparing the sample to a blank reading taken with a spectrophotometer at 700 nm. Under the same conditions, incubation, and absorbance measurement at 700 nm were performed on the blank solution. If the absorption of the reaction mixture goes up, it means there is more reducing power in the system. We performed the experiment 3 times at each dose.

3. Results

3.1 Effect of NESA on Carrageenan-Induced Rat Paw Edema

Both at 3 hours and 5 hours significant increase in paw volume was observed in the carrageenan control group compared to the control group ($p < 0.001$), treatment with NESA low and high doses and the standard drug have shown a decrease in paw volume compared to the diseased group ($p < 0.001$) (Table 1).

Table 1: Effect of NESA on Carrageenan-Induced Rat Paw Edema

Treatment	Increase in paw edema (ml) and % inhibition	
	After 3 h	After 5 h
Normal control	0.24 ± 0.027	0.24 ± 0.024
Carrageenan control	0.94 ± 0.022$^{\alpha}$	0.85 ± 0.022 $^{\alpha}$
NESA (100)	0.56 ± 0.039^{a}	0.42 ± 0.032^{a}
NESA (200)	0.32 ± 0.025^{a}	0.29 ± 0.022^{a}
Standard	0.230 ± 0.029^{a}	0.21 ± 0.022^{a}

Values are expressed as mean ± SEM. $^{\alpha}p < 0.001$, compared to control group, $^{a}p < 0.001$, compared to carrageenan control group.

3.2 Effect of NESA on Paw withdrawal threshold (Von Frey Test)

The withdrawal threshold was significantly decreased in carrageenan control group when compared to control group, whereas this was signifcantly increased in NESA (100), NESA (200), and standard groups at 15 mins ($p < 0.01$, $p < 0.001$, and $p < 0.001$), from 30 min onwards all the treatment groups have shown significant increase in paw withdrawal threshold ($p < 0.001$) when compared to carrageenan control group (Table 2).

Table 2: Effect of NESA on Paw withdrawal threshold (Von Frey Test)

Treatment	Paw withdrawal threshold (g)				
	0 min	15 min	30 min	45 min	60 min
Normal control	0.66 ± 0.01	0.65 ± 0.01	0.67±0.02	0.69±0.23	0.66±0.02
Carrageenan control	0.28 ± 0.02	0.23±0.01$^{\alpha}$	0.26±0.02$^{\alpha}$	0.27±0.02$^{\alpha}$	0.26±0.02$^{\alpha}$
NESA (100)	0.28±0.01	0.36 ± 0.02^{a}	0.44±0.02^{a}	0.54±0.04^{a}	0.55±0.04^{a}
NESA (200)	0.30 ± 0.02	0.49 ± 0.03^{a}	0.63±0.02^{a}	0.66±0.04^{a}	0.67±0.04^{a}

Standard	0.28 ± 0.02	0.55 ± 0.03[a]	0.75±0.02[a]	0.79±0.03[a]	0.78±0.03[a]

Values are expressed as mean ± SEM. [α]$p < 0.001$, compared to the control group, [a]$p < 0.001$, [b]$p < 0.01$, compared to the carrageenan control group.

3.3 Effect of NESA on AIA in Rats

Induction of arthritis caused significant increase in paw volume in AIA control group from 6th day onwards ($p < 0.001$) compared to normal control group, at 6th day decrease in paw volume was observed in NESA (100), NESA (200), and standard group ($p < 0.05$, $p < 0.001$, and $p < 0.001$) compared to AIA control group. From 8th day onwards significant decrease in paw volume was observed in all the treatment groups ($p < 0.001$), compared with AIA control group (Table 3).

Table 3: Effect of NESA on AIA in Rats

Days after induction	Normal control	AIA control	NESA (100)	NESA (200)	Standard
2	0.12 ± 0.0087	0.14 ± 0.005	0.13 ± 0.007	0.12 ± 0.005	0.12 ± 0.0058
4	0.12 ± 0.0061	0.14 ± 0.006	0.12 ± 0.0079	0.11 ± 0.004	0.12 ± 0.0048
6	0.12 ± 0.0098	0.31 ± 0.024[α]	0.23 ± 0.019[c]	0.17 ± 0.0097[a]	0.13 ± 0.0084[a]
8	0.12 ± 0.093	0.36 ± 0.023[α]	0.25 ± 0.021[a]	0.19 ± 0.0076[a]	0.13 ± 0.01[a]
10	0.12 ± 0.0075	0.38 ± 0.018[α]	0.28 ± 0.017[a]	0.21 ± 0.018[a]	0.13 ± 0.012[a]
12	0.12 ± 0.012	0.39 ± 0.012[α]	0.3 ± 0.019[a]	0.23 ± 0.018[a]	0.14 ± 0.011[a]
14	0.13 ± 0.0095	0.43 ± 0.014[α]	0.31 ± 0.014[a]	0.24 ± 0.017[a]	0.17 ± 0.007[a]
16	0.14 ± 0.0061	0.47 ± 0.0085[α]	0.33 ± 0.014[a]	0.24 ± 0.018[a]	0.21 ± 0.013[a]
18	0.15 ± 0.0034	0.47 ± 0.011[α]	0.34 ± 0.016[a]	0.25 ± 0.017[a]	0.21 ± 0.012[a]
20	0.16 ± 0.015	0.56 ± 0.016[α]	0.35 ± 0.016[a]	0.26 ± 0.02[a]	0.23 ± 0.01[a]
22	0.16 ± 0.015	0.73 ± 0.024[α]	0.36 ± 0.016[a]	0.27 ± 0.019[a]	0.32 ± 0.027[a]
24	0.16 ± 0.015	1.0 ± 0.056[α]	0.39 ± 0.019[a]	0.31 ± 0.018[a]	0.37 ± 0.027[a]
26	0.16 ± 0.017	1.1 ± 0.059[α]	0.42 ± 0.021[a]	0.33 ± 0.024[a]	0.38 ± 0.025[a]
28	0.16 ± 0.017	1.1 ± 0.053[α]	0.42 ± 0.021[a]	0.33 ± 0.026[a]	0.4 ± 0.017[a]

Values are expressed as mean ± SEM. [α]$p < 0.001$, compared to control group, [a]$p < 0.001$, [c]$p < 0.05$ compared to AIA control group.

3.4 Hematological parameters

Induction of arthritis caused an increase in WBC count in AIA control group ($p < 0.001$) compared with normal control group. Treatment with NESA (100), NESA (200), and standard group resulted in decrease in WBC count ($p < 0.001$) compared to AIA control group (Table 4).

Induction of arthritis caused a decrease in RBC count in AIA control group ($p < 0.001$) compared with normal control group. Treatment with NESA (200) and standard group resulted in increase in RBC count ($p < 0.001$) compared with AIA control group, whereas no significance was observed in NESA (100) group (Table 4).

Arthritis induction resulted in an increased platelet count in AIA control group ($p < 0.001$) compared to control group. Treatment with NESA (100), NESA (200), and standard group resulted in decrease in platelet count compared to AIA control group ($p < 0.05$, $p < 0.01$, and $p < 0.001$).
Arthritis induction caused an increase in ESR in AIA control group ($p < 0.001$) compared to normal control group. Treatment with NESA (200) and standard drug resulted in a decrease in ESR compared to AIA control group ($p < 0.05$ and $p < 0.001$), whereas no significance was observed in NESA (100) group.

Table 4: Effect of NESA on haematological parameter of rats treated with Freund's complete adjuvant.

Group	WBC count (10^3/mm^3)	RBC (x 10^6/µL)	Platelet count (10^5/mm^3)	ESR (mm/hr)
Normal control	8.7 ± 0.39	6.1 ± 0.17	2.5 ± 0.18	3.8 ± 0.21
AIA control	13 ± 0.42$^{\alpha}$	4.3 ± 0.25$^{\alpha}$	3.5 ± 0.14$^{\alpha}$	5.6 ± 0.14$^{\alpha}$
NESA (100)	10 ± 0.50^{a}	4.9 ± 0.23	2.8 ± 0.13^{c}	5.0 ± 0.17
NESA (200)	8.6 ± 0.32^{a}	5.7 ± 0.15^{a}	2.6 ± 0.16^{b}	4.7 ± 0.23^{c}
Standard	9.7 ± 0.26^{a}	6.1 ± 0.19^{a}	2.5 ± 0.12^{a}	4.5 ± 0.076^{a}

Values are expressed as mean ± SEM. $^{\alpha}p < 0.001$, compared to control group, $^{a}p < 0.001$, $^{b}p < 0.01$, $^{c}p < 0.05$ compared to AIA control group.

3.5 Effect of NESA on TAC and FRAC

TAC

At the concentration of 100 µg/mL the absorbance of NESA was in range of 0.824 ± 0.098 - 1.298 ± 0.076, while at the concentration of 150 µg/mL the absorbance was in range of 1.164 ± 0.065 - 1.694 ± 0.039. A higher absorbance indicates increased reducting power, thus increasing the concentration of extract increased the total antioxidant activity.

FRAC

At the concentration of 100 µg/mL the absorbance of NESA was in range of 1.356 ± 0.042 - 2.365 ± 0.127, while at the concentration of 150 µg/mL the absorbance was in range of 1.774 ± 0.046 - 2.992 ± 0.127. A higher absorbance indicates increased reducting power, thus increasing the concentration of extract increased the total antioxidant activity (Table 5).

Table 5: Effect of NESA on TAC and FRAC

Drugs	**TAC**		**FRAC**	
	At 100 µg/ml	At 150 µg/ml	At 100 µg/ml	At 150 µg/ml
NESA (100)	0.824 ± 0.098	1.164 ± 0.065	1.356 ± 0.042	1.774 ± 0.046
NESA (200)	1.298 ± 0.076	1.694 ± 0.039	2.365 ± 0.127	2.992 ± 0.127
CA	1.99 ± 0.066	2.494 ± 0.024	-	-
AA	-	-	3.509 ± 0.075	3.325 ± 0.087

CA and AA represents ascorbic acid and catechin
[a] Each value is the average of three analyses ± standard deviation

4. Discussion

Rat hind paw edema caused by carrageenan is the best model for primary screening of antiinflammatory drugs. It was characterized as a two-stage process in which injected carrageenan caused edema in the rat paw (**Koriem et al., 2012**). Histamine and serotonin secretion is responsible for the first hour's observations; secretion of prostaglandin-like substance accounts for the second. On this basis, it is

possible to postulate that blocking the secretion of early mediators like histamine and serotonin is responsible for the inhibition of the first phase, while blocking cyclo-oxygenase is responsible for the hinderance to the second phase. This study's findings suggest that extracts of Semecarpus anacardium may function as a shield against the acute inflammation caused by carrageenan. The presence of flavonoids in the preparation explains the drug's (SA) considerable suppressive action, which in turn demonstrates the drug's robust anti-inflammatory efficacy (**Bandigari et al., 2021**). Furthermore, the extract also showed anti-allodynic effects in von frey test by attenuating the mechanical hyperalgesia.

S. anacardium, which has been shown to possess physiologically active components such as steroids, flavonoids, polysaccharides, phenols, and terpenes, has been shown to have anti-inflammatory effects, suggesting it may be able to mitigate the arthritic effects of FCA (**Bandigari et al., 2021**). The most widely used animal model for studying secondary inflammation is Freund's adjuvant arthritis is rats, while mice seem to be immune to developing arthritis (**Narendhirakannan et al., 2005**).

In the present research, adjuvant arthritis in rats was studied, and the arthritic rats displayed soft tissue swelling surrounding the ankle joints, probably attributable to edema of periarticular tissues including ligaments and joint capsules. It is likely that the adjuvant is responsible for the initial decrease in edema and soft tissue swelling at the deposition site, while the later occurrence of widespread arthritis and flare in the treated foot are immunological reactions. Animal models of inflammation have indicated a cascade of low molecular weight mediators (histamines, serotonin, and prostaglandins) (**McGEER et al., 2004**), leading to an initial vascular response characterized by vasodilation and greater vascular permeability. After many hours of these acute vascular alterations, a considerable number of polymorphonuclear leukocytes, mostly neutrophils, are accumulated in the tissues. Prostaglandins dramatically amplify exudates by boosting blood flow to the area and relaxing the smooth muscle cells that line the arteries (**Fosslien, 2005**). One of the hallmarks of anti-inflammatory effect is the reduction of paw edema in adjuvant-induced arthritic rats. It's possible that the drug's ability to minimize edema development is linked to its capacity to limit prostaglandin production.

Hematological parameter estimations showed that the S. anacardium extract reduced secondary inflammation in adjuvant-induced arthritic rats. Clinical observations and the analysis of the ESR are two of the most common methods used to diagnose rheumatoid arthritis and evaluate therapy efficacy. Arthritis has been linked to anemia because RBCs die off more quickly when they are exposed to inflammation (**Stenvinkel, 2001; Weiss et al., 2019**). Chronic inflammatory conditions, such as active rheumatoid arthritis, may manifest as mild to severe anemia (**Chen et al., 2020**). Rats with arthritis also had elevated platelet and ESR counts. Inflammation may be tracked using measurements of ESR and fibrinogen levels. In order to combat the infectious pathogens, the white blood cell (WBC) count rises in arthritic rats. WBCs Leukocyte increase is the hallmark of arthritis since they are the primary mediator of the disease. S. anacardium was shown to drastically reduce WBC count, suggesting that it inhibits their migration into the inflamed region.

The antioxidant capacity of the S. anacardium extract was determined by evaluating its ability to inhibit the formation of a green phosphate/Mo (V) complex under acidic condition, after the conversion of Mo (VI) to Mo (V) by the antioxidant chemicals. At 150 µg/mL, the extractives had a reducing ability of between 1.164 ± 0.065 and 1.694 ± 0.039 µm green phosphate/Mo (V). There was a direct correlation between the amount of polyphenols present and the level of antioxidant activity. The overall phenol content of a plant seems to have a strong correlation with its antioxidant potential, as shown by recent studies (**Oktay et al., 2003**). By giving an electron to the Fe^{3+}-ferricyanide complex, the produced extract's iron reduction capacity was calculated. At 150 µg/mL, the extract had a reducing ability of between 1.774 0.046 and 2.992 0.127 µm Fe (II)/g. According to the results of this analysis, higher phenolic levels were associated with greater ferrous reducing antioxidant ability. Results obtained by us are in agreement with those found in the literature (**Huang et al., 2005**).

Many of its constituents, including flavonoids (Selvam et al., 2004), phenols, sterols, and glycosides (**Bahir et al., 2013**), have been shown to be effective anti-inflammatory compounds at very low concentrations (**Singh et al., 2017; Ilanchezhian et al., 2011; Jain et al., 2013**). As a result, it is possible that the steroids, flavonoids, and phenols found in S. anacardium nut extract are responsible for its anti-inflammatory properties. In conclusion, we established that the S. anacardium extract reduced FCA-induced arthritic inflammation. This study's findings provide credence to the traditional use of this herb to treat inflammatory conditions in folk medicine.

5. Conclusion

This study's findings, taken as a whole, indicate that S. anacardium extract has anti-inflammatory efficacy against both the acute phase of inflammation and adjuvant arthritis, and it does so without causing any adverse effects.

Acknowledgements

One of the authors Dr. D. Ashok is thankful to UGC,New Delhi for the award of UGC-BSR faculty fellowship [Sanction No. F. 18- 1/2011(BSR)/2018]

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