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Original Research Article

Anti-inflammatory and analgesic effects of lipid extract from *Eryx* snakes in adjuvant-induced arthritis in rats

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ABSTRACT

Background: Rheumatoid arthritis (RA) is a chronic inflammatory disease in which long-term use of standard anti-inflammatory drugs is limited by incomplete efficacy and adverse effects, creating a need for safer alternative agents from natural sources. This study evaluated the prophylactic effect of a lipid extract obtained from Eryx snakes (LEES) in a rat model of adjuvant-induced arthritis (AIA).
Methods: Arthritis was induced in male rats by sub-plantar injection of Complete Freund's Adjuvant (CFA). Animals received daily oral administration of LEES (50 mg/kg), diclofenac (10 mg/kg), or indomethacin (4 mg/kg) for 13 days. The therapeutic effects were assessed by monitoring paw edema, local hyperthermia, and pain sensitivity (Hargreaves test) over 14 days.
Results: LEES administration significantly suppressed paw edema, achieving 45.6% inhibition by day 14, and effectively attenuated local hyperthermia. The extract also demonstrated significant analgesic activity by preserving thermal withdrawal latency and mitigated inflammation-associated body weight loss. Notably, the therapeutic efficacy of LEES was comparable to that of the reference NSAIDs, diclofenac and indomethacin.
Conclusions: The study confirms that LEES effectively mitigates clinical signs of chronic arthritis with an efficacy profile similar to diclofenac and indomethacin. Thus, LEES represents a viable natural candidate for the development of novel anti-inflammatory agents.

Keywords: Rheumatoid arthritis, Lipid extract of Eryx snakes, Adjuvant-induced arthritis, Anti-inflammatory activity

INTRODUCTION

Rheumatoid arthritis (RA) is a chronic immune-mediated inflammatory disease characterized by persistent synovitis, progressive joint damage, chronic pain, and systemic manifestations.[1,2] Despite substantial advances in understanding RA pathogenesis and the implementation of treat-to-target strategies, a clinically meaningful proportion of patients does not achieve sustained remission; difficult-to-treat RA remains a persistent challenge in routine practice.[3] Current pharmacotherapy includes disease-modifying antirheumatic drugs (DMARDs) alongside symptomatic agents such as non-steroidal anti-inflammatory drugs (NSAIDs) and glucocorticoids; however, long-term NSAID and steroid exposure is constrained by well-recognized adverse effects.[1,2] Therefore, identification of adjunct or alternative anti-inflammatory interventions

with improved benefit-risk profiles remains a relevant pharmacological objective.[4]

Natural products remain an important source of pharmacologically active molecules, including agents with immunomodulatory activity and potential to influence inflammation-resolution programs. Animal-derived remedies represent a long-standing component of traditional medicine (zootherapy), and snake-derived materials have been used in diverse ethnomedical systems as anti-inflammatory interventions.[5,6] In Uzbekistan and neighboring regions, the genus Eryx (Boidae) is documented as a traditional resource used for inflammatory, respiratory, and systemic conditions.[7,8]

Earlier work demonstrated anti-inflammatory activity of Eryx-derived preparations, including the aqueous snake autolysate (Eryxin) in acute inflammation models.[9,10] More recently, anti-inflammatory effects of the lipid extract from Eryx snakes (LEES) were reported in aseptic inflammation, supporting further pharmacological interest in this material.[11] Nevertheless, the effects of LEES in chronic, immune-driven inflammatory conditions that model key clinical features of RA have not been sufficiently characterized.

The complete Freund's adjuvant-induced arthritis (AIA) model reproduces inflammatory edema, local hyperthermia, and pain-related behavioral changes and is widely used for preclinical evaluation of anti-arthritic candidates.[17] Accordingly, the present study aimed to evaluate the prophylactic effects of LEES on the development and progression of CFA-induced chronic arthritis in rats, with comparative assessment against standard NSAID therapy.

METHODS

Animals

The study was conducted on mature outbred white male rats weighing 185–200 g. The animals were obtained from the breeding facility of the Department of Sanitary and Epidemiological Surveillance of the Main Medical Department under the Administration of the President of the Republic of Uzbekistan. Before the experiment, animals underwent a 14-day quarantine during which their general health, skin condition, and locomotor activity were assessed. The animals were housed in polycarbonate cages under standard vivarium conditions: 20–25°C, 50–60% humidity, 12/12-hour light/dark cycle, and access to standard chow and water ad libitum.

All experimental procedures were performed in accordance with the European Convention for the Protection of Vertebrate Animals Used for Experimental and Other Scientific Purposes (ETS No. 123) and approved by the Ethics Committee of the Ministry of Health of the Republic of Uzbekistan (Protocol No. 6, dated June 25, 2025).

Experimental design

Study period: The in vivo experiment was conducted from August 2025 to September 2025. Fifty rats were divided into five groups, ten animals each. Group 1 (healthy control) received a subcutaneous injection of 0.1 mL of paraffin oil into the plantar surface of the right hind paw. Groups 2–5 received a single subcutaneous injection of 0.1 mL of Complete Freund's Adjuvant (CFA) (heat killed *Mycobacterium butyricum* suspended in mineral oil, Chondrex Inc., USA, Cat.No:7009) into the same region to induce inflammation. One hour after CFA injection, animals in Groups 2–5 began daily oral treatment for 13 consecutive days with the following compounds: Group 2 (CFA control) received distilled water at a dose of 1.0 ml/100 g of body weight; Group 3 received lipid extract of *Eryx* snakes (LEES) at 50 mg/kg, dissolved in sunflower oil; Group 4 received diclofenac sodium at 10 mg/kg, dissolved in 0.9% saline; and Group 5 received indomethacin at 4 mg/kg, dissolved in 0.9% saline. All treatments were administered via intragastric gavage. The doses were selected based on previous studies and scaled to rat weight.[11]

The development of arthritis was monitored by changes in paw volume, local temperature, pain sensitivity.

Clinical assessment of AIA

Paw edema was measured using a digital plethysmometer (Ugo Basile Srl, Italy) before CFA injection and on days 3, 7, 10, and 14. The edema inhibition was calculated as:

$$\text{Edema inhibition}(\%) = \frac{V_{control} - V_{treated}}{V_{control}} \text{ x } 100$$

Skin temperature of the right hind paw was assessed using a non-contact infrared thermometer (model TF-600, Shenzhen Quik Zoom Technology Co., China) at the same time points. Fluctuations from baseline were recorded to reflect local inflammatory hyperthermia.

In addition, body weight of the rats was recorded on the same days of the experiment.

Nociceptive threshold was assessed using the plantar (Hargreaves) test with a radiant heat stimulus, recording paw withdrawal latency (s) (Ugo Basile, Italy). Tests were performed on days 0, 3, 7, 10, and 14 under acclimatized conditions, with a fixed cutoff to prevent tissue damage.

Statistical analysis

All experimental data were expressed as mean ± standard deviation (SD). Statistical comparisons between groups were performed using one-way analysis of variance (ANOVA), followed by Tukey's post hoc test to determine intergroup differences. When only two groups were compared, Student's t-test was applied. Statistical

significance was considered at $p < 0.05$. All analyses were conducted using GraphPad Prism.

RESULTS

Effect on paw edema

Following CFA injection, a significant progressive increase in paw volume was observed in the CFA control group ($p < 0.05$ compared to baseline). By day 14, paw volume in control rats had increased approximately fourfold relative to initial values, indicating active chronic inflammation development.

In contrast, prophylactic administration of LEES, diclofenac sodium, and indomethacin significantly suppressed the increase in paw volume across all observation periods. The results are summarized in Table 1.

Administration of LEES reduced paw edema by 35.9% on day 3, 38.5% on day 7, 42.7% on day 10, and 45.6% on day 14 compared to untreated controls. Notably, the anti-edematous effect of LEES did not differ significantly from that of the reference NSAIDs, diclofenac sodium and indomethacin.

The dynamics of paw volume changes are depicted in Figure 1.

These findings indicate that LEES possesses significant anti-inflammatory activity comparable to classical NSAIDs in a model of chronic immune inflammation, consistent with previous reports on anti-inflammatory effects of lipid extracts from animal sources.[6,11,12]

Table 1: Effect of LEES, diclofenac sodium, and indomethacin on paw volume in rats with adjuvant-induced arthritis (mean±SD, n=10).

Group	Day 0 (cm³)	Day 3 (cm³)	Day 7 (cm³)	Day 10 (cm³)	Day 14 (cm³)
CFA control	0.98±0.05	2.89±0.14*	3.11±0.09*	3.42±0.22*	3.99±0.22*
LEES 50 mg/kg	0.96±0.05	2.27±0.16*#	2.38±0.18*#	2.49±0.16*#	2.68±0.15*#
Diclofenac 10 mg/kg	0.84±0.05	2.05±0.08*#	2.21±0.07*#	2.31±0.06*#	2.39±0.06*#
Indomethacin 4 mg/kg	0.87±0.05	2.11±0.15*#	2.26±0.18*#	2.30±0.15*#	2.49±0.06*#

*- Reliable difference in relation to baseline, #- reliable difference in paw edema in relation to the control of the corresponding days of the study.

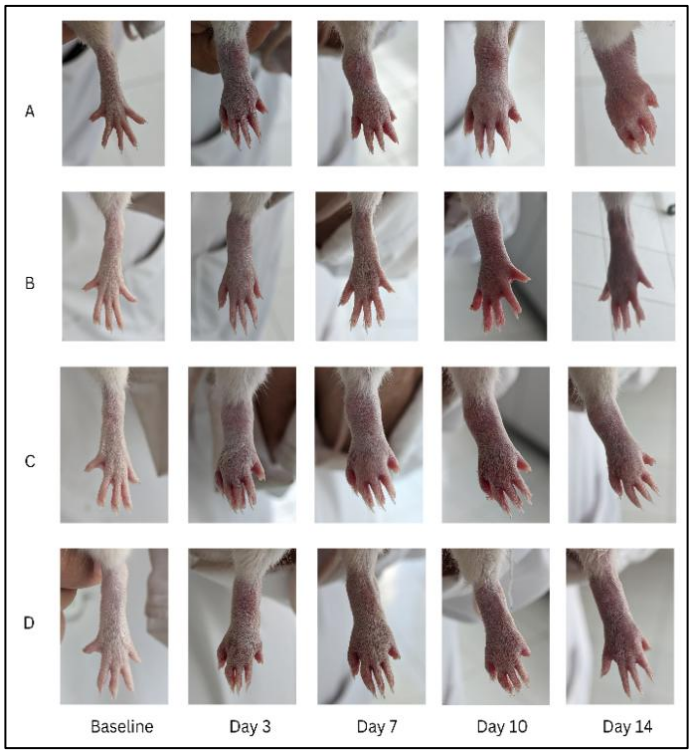

Figure 1: Visual assessment of inflammation in the right hind paw of rats during the course of adjuvant-induced arthritis. Representative photographs were taken on days 0 (baseline), 3, 7, 10, and 14. A: CFA control group (no treatment); B: LEES group (50 mg/kg); C: Diclofenac sodium group (10 mg/kg); D: indomethacin group (4 mg/kg). Marked paw swelling and erythema were observed in the CFA control group, while all treatment groups showed visibly reduced edema and erythema by day 14, with lees exhibiting effects comparable to NSAIDS.

Table 2: Skin temperature of the right hind paw in rats with adjuvant-induced arthritis (mean±SD, °c, n=10).

Group	Day 0	Day 3	Day 7	Day 10	Day 14
Healthy control	22.48 ± 0.11	22.33 ± 0.14	22.56 ± 0.13	22.28 ± 0.14	22.62 ± 0.15
CFAcontrol	22.21 ± 0.13	26.10 ± 0.18*	27.81 ± 0.16*	28.97 ± 0.19*	27.53 ± 0.17*
LEES	22.55 ± 0.14	24.63 ± 0.10*#	25.57 ± 0.13*#	26.55 ± 0.16*#	25.91 ± 0.11*#
Diclofenac	22.78 ± 0.16	24.02 ± 0.12*#	24.85 ± 0.16*#	25.55 ± 0.13*#	25.07 ± 0.16*#
Indomethacin	22.30 ± 0.11	24.18 ± 0.15*#	24.83 ± 0.14*#	25.33 ± 0.12*#	24.70 ± 0.17*#

*- Reliable difference with initial indicators of the corresponding groups of animals, #- reliable difference in skin temperature in relation to the CFA control of the corresponding days of the study.

Effect on local temperature

Subplantar injection of complete Freund's adjuvant (CFA) in rats caused a significant local temperature increase in the affected hind limb, reflecting ongoing inflammatory hyperemia. The maximum rise was recorded on day 10, with a 30.4% increase from baseline in the control group ($p<0.05$).

Prophylactic administration of LEES, diclofenac sodium, and indomethacin resulted in a significant attenuation of local hyperthermia across all time points (Table 2).

By day 10, the affected paw temperature in the control group reached 28.97°C, versus 22.28°C at baseline (+30.4%). LEES reduced this increase to 26.55°C (only +17.8%), a reduction comparable to diclofenac and indomethacin.

The attenuation of local hyperthermia by LEES indicates peripheral anti-inflammatory activity and is consistent with its anti-exudative effects in the carrageenan-induced paw edema model, as well as with the traditional use of snake-derived remedies for inflammatory conditions and the COX-2–inhibiting properties of structurally related marine lipid extracts.[6,11,13-15]

Effect on pain sensitivity

Pain sensitivity was assessed using the plantar (Hargreaves) test, where a decrease in withdrawal latency indicates heightened nociceptive response. In the CFA control group, a significant reduction in latency was observed starting from day 3, persisting throughout the study, consistent with chronic inflammatory pain development.

Prophylactic administration of LEES, diclofenac sodium, and indomethacin led to a significant increase in withdrawal latency compared to untreated rats, indicating analgesic/antinociceptive effects (Table 3).

In CFA control animals, withdrawal latency decreased by ~77% on day 3 and up to ~86% by day 10. LEES significantly attenuated this response, preserving up to 53.9% of the baseline latency by day 14, comparable to indomethacin and diclofenac.

Changes in body weight

Body weight was monitored to assess the general physiological status of animals during the 14-day experimental period. In the healthy control group, a steady gain in body mass was observed. In contrast, CFA-induced inflammation in the pathological control group resulted in significant weight loss, indicating systemic impact of chronic inflammation and associated catabolism.

Prophylactic administration of LEES, diclofenac sodium, and indomethacin effectively mitigated body weight loss, as shown in Table 4.

Table 3: Pain sensitivity threshold (paw withdrawal latency, sec) in rats with adjuvant-induced arthritis (mean±SD, n=10).

Group	Day 0	Day 3	Day 7	Day 10	Day 14
Healthy control	24.52±0.47	24.31±0.61	24.18±0.52	23.90±0.41	27.71±0.59
CFA control	24.81±0.29	5.62±0.27*	4.43±0.16*	3.51±0.30*	4.11±0.46*
LEES	24.95±0.69	7.87±0.58*#	9.47±0.68*#	11.03±0.63*#	13.38±1.01*#
Diclofenac	24.71±0.18	9.50±0.66*#	11.02±0.74*#	12.25±0.83*#	15.38±0.66*#
Indomethacin	24.02±0.79	9.12±0.65*#	10.43±0.86*#	11.56±0.72*#	14.17±0.17*#

*- Statistically significant difference compared to healthy control animals, # - significant difference compared to the CFA control of the corresponding study terms.

Table 4: Body weight changes in rats with adjuvant-induced arthritis under prophylactic treatment (mean±SD, g, n=10).

Group	Day 0	Day 7	Day 14
Healthy control	194.01±3.41	202.04±3.16	213.33±2.56*
CFA control	192.17±3.34	178.83±3.83*	174.50±4.25*
LEES	190.33±3.84	184.31±4.15	182.52±.38
Diclofenac	193.15±3.79	188.50±3.25	187.83±3.75
Indomethacin	195.50±2.79	190.03±2.13	187.72±1.91

* - Reliable difference with initial indicators of the corresponding groups of animals.

By day 14, the control group lost ~9.2% of initial body weight, reflecting systemic inflammation. In contrast, animals treated with LEES showed only ~4.1% loss, and those treated with NSAIDs showed similar mild reductions. Notably, body weight in the LEES group did not differ significantly from that of intact animals. These findings further confirm the systemic protective effects of LEES against CFA-induced catabolic stress, in line with its anti-inflammatory activity.

DISCUSSION

In the CFA-induced adjuvant arthritis model, prophylactic administration of the LEES produced consistent improvement across functional readouts of inflammatory burden and nociceptive sensitization, including paw volume, local temperature, plantar withdrawal latency, and preservation of body weight. These endpoints are commonly used in AIA/CFA paradigms and provide interpretable markers of disease activity and treatment response in rodents.[16,17,19,20] The magnitude of effect observed for LEES was comparable to reference NSAIDs (diclofenac and indomethacin) on the measured outcomes, indicating meaningful anti-inflammatory and antinociceptive activity in chronic immune inflammation.

The anti-edematous effect of LEES indicates suppression of the exudative component of chronic inflammation. In parallel, attenuation of local hyperthermia supports an effect on inflammatory hyperemia. Thermographic approaches provide a non-contact quantitative readout of inflammatory hyperemia and have been validated in experimental arthritis, with signal changes correlating with paw swelling and clinical status.[18] In the present study, temperature reductions paralleled improvements in edema and behavior, supporting internal consistency across endpoints.

The pattern of activity is coherent with ethnomedical evidence for snake-derived remedies and with previous pharmacological work on Eryx-derived preparations.[5-10] Furthermore, COX-2-related anti-inflammatory activity has been reported for structurally related marine lipid extracts, providing a plausible comparative framework for lipid-mediated effects on inflammatory pathways.[13-15]

Although the present study did not include target or mediator profiling, these lines of evidence justify further chemical characterization of LEES and fraction-guided testing to identify the components responsible for bioactivity.[11]

Analgesic-like activity was demonstrated by increased withdrawal latencies in the plantar (Hargreaves) test, a benchmark assay for thermal hyperalgesia with established methodology and reproducibility.[19,20] Reductions in thermal hyperalgesia are consistent with decreased peripheral inflammatory drive and/or mitigation of central sensitization accompanying chronic inflammatory pain. Reference NSAIDs served as appropriate pharmacological comparators given their established inhibition of cyclo-oxygenase (COX)-mediated prostanoid formation and robust efficacy in inflammatory pain models and clinical settings.[21]

At the same time, class-wide risks of NSAIDs (gastrointestinal, renal, and cardiovascular) motivate the exploration of adjuncts or alternatives with distinct mechanistic profiles and potentially improved safety margins.[1,2] This aligns with contemporary treat-to-target principles and current recommendations for RA management, in which sustained disease control is prioritized while minimizing toxicity and long-term harm.[22] The observation that LEES tracked within the efficacy band of NSAID controls on functional outcomes suggests potential utility as a natural-product candidate, either as a stand-alone option in milder settings or as an adjunct aimed at reducing NSAID exposure.

Finally, lipid-rich natural products may also engage resolution programs. Specialized pro-resolving mediators (SPMs), including resolvins, protectins, and maresins, actively terminate inflammation and can reduce pain without broadly compromising host defense, offering a complementary paradigm to COX inhibition.[23]

The present study is limited by a prophylactic dosing design, a 14-day follow-up, and the absence of blinded joint histopathology and systemic biomarkers. Subsequent studies should include therapeutic (post-onset) regimens, dose-response evaluation, histological confirmation of joint protection, and biomarker readouts to strengthen mechanistic interpretation and translational relevance.[17]

CONCLUSION

In CFA-induced chronic arthritis, LEES attenuated edema and local hyperthermia, reduced heat hyperalgesia, and mitigated inflammation-associated weight loss, yielding an efficacy profile comparable to standard NSAID controls on the measured functional outcomes. While mechanistic claims should remain conservative in the absence of biomarker and histological data, these results nonetheless support further studies aimed at more comprehensively characterizing the anti-inflammatory efficacy, mechanisms of action and safety profile of LEES in chronic inflammatory settings.

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