ISSN: 2322 - 0902 (P)
ISSN: 2322 - 0910 (O)

International Journal of Ayurveda and Pharma Research

Research Article

EXPLORING THE ANTI-INFLAMMATORY POTENTIAL OF *CYPERUS PANGOREI* RHIZOME EXTRACTS AN IN VITRO AND IN VIVO STUDY

Biswajit Samanta[1], S. K. Gupta[1], Amal K. Maji[2]*

[1]CMJ University, Department of Microbiology, Jorabat, Meghalaya.
*[2]Xplora Clinical Research Services Pvt. Ltd., Bangalore, Karnataka, India.

Article info

Article History:
Received: 21-02-2024
Accepted: 16-03-2024
Published: 04-04-2024

KEYWORDS:
Cyperus pangorei, Anti-inflammatory, Cytokines, TNF-α and IL-1β.

ABSTRACT

Background and aim: Inflammation is a pivotal process implicated in various physiological and pathological conditions, necessitating the exploration of alternative anti-inflammatory agents with minimal side effects. This study aimed to investigate the anti-inflammatory potential of the standardized ethyl acetate (EtAc) fraction derived from *Cyperus pangorei* rhizomes. **Methods:** The rhizomes of *C. pangorei* were collected, processed, and subjected to extraction and fractionation to obtain the EtAc fraction. RP-HPLC analysis was employed to standardized the EtAc fraction against standard quercetin, luteolin, and apigenin. *In vitro* studies utilized peritoneal macrophages isolated from male Swiss albino rats to assess NO production and cytokine levels (IL-1β, IL-6, TNF-α) upon treatment with the EtAc fraction. *In vivo* evaluation was conducted using a carrageenan-induced rat paw edema model. **Results:** RP-HPLC analysis revealed the presence of quercetin, luteolin, and apigenin in the EtAc fraction. *In vitro* studies demonstrated dose-dependent inhibition of LPS-induced NO production and suppression of inflammatory cytokines (IL-1β, IL-6, TNF-α) by the EtAc fraction. Furthermore, in the carrageenan-induced rat paw edema model, the EtAc fraction exhibited dose-dependent inhibition of paw edema. **Conclusion:** The findings of this study highlight the significant anti-inflammatory potential of *C. pangorei* rhizome extracts, particularly the EtAc fraction. The identified compounds, quercetin, luteolin, and apigenin, contribute to its anti-inflammatory activity by modulating key inflammatory mediators. These results support the potential therapeutic use of *C. pangorei* in managing inflammation-related disorders. Further research is warranted to elucidate the underlying mechanisms and evaluate the long-term efficacy and safety of *C. pangorei* extracts as anti-inflammatory agents.

INTRODUCTION

Inflammation plays a pivotal role in various physiological and pathological processes within the human body, ranging from the body's natural defense mechanism against infections to the development of chronic inflammatory diseases [1]. Dysregulation of the inflammatory response can lead to the overproduction of pro-inflammatory mediators, such as nitric oxide (NO), tumor necrosis factor-alpha (TNF-α), interleukin-1beta (IL-1β), and interleukin-6 (IL-6).

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https://doi.org/10.47070/ijapr.v12i3.3155

These cytokines, among others, contribute significantly to the progression and exacerbation of inflammatory disorders, including rheumatoid arthritis, inflammatory bowel disease, and various autoimmune conditions [2,3]. Nitric oxide, synthesized by inducible nitric oxide synthase (iNOS), serves as a potent signalling molecule involved in immune responses [4]. TNF-α, IL-1β, and IL-6, on the other hand, are pro-inflammatory cytokines that regulate the immune response and contribute to the perpetuation of inflammation in chronic conditions [5]. In the context of inflammation, macrophages play a crucial role in immune responses by engaging in activities like antigen presentation, phagocytosis, and immuno-modulation through the production of cytokines and growth factors [6]. The regulatory dynamics of the inflammatory process, encompassing macrophage activation and deactivation, respond to diverse signals,

including cytokines (e.g., IFN-γ and TNF-α), bacterial lipopolysaccharide, and extracellular matrix proteins. Nonetheless, an excessive production of defense molecules can lead to pronounced inflammation [7].

Several anti-inflammatory drugs, such as NSAIDs and corticosteroids, effectively alleviate inflammation but may pose side effects, including gastrointestinal issues and systemic complications [8,9]. Biologic agents offer targeted therapy for inflammatory diseases but carry potential risks like infections and rare adverse events [10]. Therefore, there is a pressing need for the development of alternative, effective, and safe drugs, or therapies to manage pain and inflammation.

One promising avenue for combating inflammation involves exploring the therapeutic potential of natural compounds derived from medicinal plants. Plants with medicinal properties, abundant in bioactive compounds, harbor phytochemicals such as polyphenols, flavonoids, and terpenoids, acknowledged for their ability to regulate diverse inflammatory pathways [11]. Studies have explored the anti-inflammatory potential of medicinal plants have investigated their ability to inhibit inflammatory cytokines, including NO, IL-1β, TNF-α, and IL-6, offering promising therapeutic avenues for inflammatory disorders [12].

Cyperus pangorei, a member of the Cyperaceae family, has gained attention for its potential therapeutic properties such as anti-inflammatory, antipyretic, antioxidant, anticancer and analgesic properties [13]. The rhizome of *C. pangorei* has been traditionally used in various medicinal purposes including digestive issues, diarrhea, dysentery, jaundice, fever, and pain, [14] and recent research suggests its anti-inflammatory properties [15,16]. This study aimed to assess the *in vitro* and *in vivo* anti-inflammatory potential of standardized ethyl acetate (EtAc) fraction of *C. pangorei* rhizome.

MATERIALS AND METHODS

Chemicals and Reagents

The materials utilized in this study were sourced from various providers. Dexamethasone (DEX), O-toluidine blue, lipopolysaccharide (LPS), 3-[4,5-dimethylthiazol-2-yl]-2,5 diphenyl tetrazolium bromide (MTT), indomethacin, and Griess reagent were acquired from Sigma-Aldrich, St. Louis, MO, USA. Fetal bovine serum (FBS), penicillin-streptomycin solution, and insulin-transferrin-selenium were procured from Himedia Laboratories Pvt. Ltd, Mumbai. Rat enzyme-linked immunosorbent assay (ELISA) kits for IL-1β, IL-6, and TNF-α were obtained from BD Biosciences, San Jose, CA, USA. Moreover, all analytical-grade solvents used were purchased from Merck Ltd, Mumbai, India.

Plant Materials and Extract Preparation

Rhizomes of *C. pangorei* were sourced from the Sabong region in West Bengal, India, authenticated at the Department of Microbiology, CMJ University, Meghalaya. The collected rhizome was air-dried, ground into a fine powder, and subjected to cold maceration extraction with 70% ethanol for 15 days at 25°C with intermittent shaking, yielded 53.68 g of extract (10.74%, w/w) after evaporation and lyophilization. Further solvent-solvent partitioning (n-hexane, chloroform, ethyl acetate and water) resulted in fractions, among which the EtAc fraction exhibited the highest antioxidant activity, total phenolic, and flavonoid contents, as determined by phytochemical analysis and preliminary TNF-α production assay using LPS-stimulated macrophages.

Experimental Animals

Male Swiss albino rats weighing between 75 to 90 grams were employed for the *in vivo* and *in vitro* anti-inflammatory study. These animals were kept in standard conditions, including a temperature of 25 ± 2°C, relative humidity of 55.6 ± 10%, and a 12-hour light/dark cycle. They had access to standard food and water ad libitum. The experimental procedures were subjected to approval by the Animal Ethics Committee of CMJ University, Meghalaya, ensuring compliance with ethical standards for Animal Care and Use.

RP-HPLC Analysis

In this study, a Waters RP-HPLC system (Milford, USA) with a 600-controller pump, UV-Vis detector, Rheodyne 7725i injector (20 µl loop), and Empower2 software was used. Chromatographic separation was achieved using a Luna C18 (2) 100 Å, 250 x 4.6 mm column with 5 mm particles (Phenomenex, Torrance, USA). The method of Chen et al. [17] was used for the standardization of EtAc fraction. The mobile phase included 0.1% formic acid, acetonitrile, and methanol, with a column temperature 30°C, flow rate 1 mL/min, and a total run time 45-min. Analysis was performed at 350 nm, with a 20 µL injection volume. Quantification of luteolin, apigenin, and quercetin in the EtAc fraction was based on their respective calibration curves, and identification relied on retention time (RT) comparison between standards and the EtAc fraction.

Isolation of Peritoneal macrophages

The peritoneal macrophages were harvested from male Swiss albino rats (75 - 90 g) based on the method described by Mukherjee et al. [18]. To collect the macrophages, 20 mL of ice-cold 0.1 M phosphate-buffered saline (PBS, pH 7.0) was intraperitoneally injected and gently massaged. Euthanasia was promptly administered through ether asphyxiation, and the peritoneal fluid was withdrawn, followed by centrifugation at 600 g for 10 minutes at 4°C. The cell pellet was then collected onto sterile petri dishes,

subjected to two washes with PBS, and re-suspended in RPMI 1640 medium supplemented with 10% FBS, 100 U/mL penicillin, and 100 µg/mL streptomycin. The petri dishes were incubated at 37°C under 5% CO2 for 2 hours. Non-adherent cells were removed by gentle PBS washing, and fresh medium was added. Cell viability, assessed using the MTT assay, indicated a viability exceeding 98% for the macrophage cells. The cells were allowed a 24-hour acclimation period before any treatments.

In Vitro Study

Nitric oxide (NO) production

Nitric oxide (NO) production was assessed to determine the impact of EtAc fraction on LPS-induced NO production by macrophages, following the methodology outlined by Mukherjee et al. [18]. In 96-well flat-bottomed tissue culture plates, macrophage cells (1.5 × 105 cells/well) were seeded. The cells were incubated at 37°C for 24 hours under 5% CO2, in the presence of LPS (1 µg/mL) along with varying concentrations of EtAc fraction (25, 50, and 100µg/mL) or DEX (1µg/mL). Macrophages cultured with or without LPS served as normal and negative controls, respectively. To determine NO production by macrophages, the Griess reaction was employed. This involved mixing 100µL of cell-free culture supernatant with 100µL of Griess reagent (0.1% naphthylethylenediamine dihydrochloride and 1% sulfanilamide in 2.5% H_3PO_4) in 96-well flat-bottomed plates. The plates were then incubated at room temperature for 10 minutes, and the absorbance of the reaction mixtures was measured at 540 nm using a microplate reader (Bio-RAD, USA). The quantities of nitrite (µM) in the samples were determined based on the standard curve of sodium nitrite.

Cytokine Assay

Cytokine levels were assessed by collecting cell-free culture supernatants following 24 hours of macrophage incubation (1.5 × 105 cells/well) with LPS (1 µg/mL) and varying concentrations of EtAc fraction (25, 50, and 100 µg/mL), or dexamethasone (1 µg/mL). Macrophage cells were cultured with or without LPS, serving as normal and negative controls, respectively. The concentrations of IL-1β, IL-6, and TNF-α in the cell-free supernatants were determined using ELISA kits, following the manufacturer's instructions. ELISA results were measured at 450 nm using a microplate reader (Bio-RAD, USA). The concentration of each cytokine was quantified based on the respective linear dose-response standard curve of recombinant IL-1β, IL-6, and TNF-α [19].

In Vivo Study

Carrageenan-induced rat paw edema

The experimental procedure closely followed established method [20]. Briefly, inflammation was induced in the right hind paw by subplantar injection of 20 mL carrageenan (1% w/v) in 0.9% saline. The EtAc fraction, formulated in 1% w/v gum acacia, was orally administered at doses of 50 mg/kg, 100 mg/kg, and 200 mg/kg, one hour prior to the carrageenan injection. A control group received the vehicle alone, while a standard group was treated with indomethacin (20 mg/kg, p.o.). Paw volumes of the injected and contralateral paws were measured at 1-, 3-, and 5-hours post-induction of inflammation using a plethysmometer. The results were expressed as the percentage reduction in volume compared to the control group at different time intervals. The percentage inhibition of edema volume between EtAc fraction treated and carrageenan alone treated groups were calculated as follows:

$$\text{Percentage Inhibition} = \frac{Vc - Vt}{Vc} \times 100$$

Where, Vc - Vt and Vc represented the mean increase in paw edema volume in control and drug treated groups.

Statistical analysis

Statistical analysis was conducted to assess the experiment results, and the findings are presented as mean ± SEM. The data were subjected to one-way ANOVA, followed by Dunnett's multiple comparison test, utilizing Graph Pad Prism 5.0 statistical analytical software.

RESULTS

RP-HPLC Analysis

The RP-HPLC chromatogram of the EtAc fraction displayed distinct peaks corresponding to quercetin, luteolin, and apigenin at retention times (RT) of 17.33±0.15, 18.97±0.17, and 32.13±0.13 min, respectively. Quantitative analysis from calibration curves revealed that the EtAc fraction contained 2.89 ± 0.51% quercetin, 1.89 ± 0.17% luteolin, and 3.85 ± 0.19% apigenin.

NO assay

The EtAc fraction was evaluated for the inhibition of LPS-stimulated NO production by macrophage cells. In the present study, LPS alone significantly ($P \leq 0.001$) induced higher amounts of NO production (87.29 ± 11.62 µM) by macrophages in compared to the control group (10.45 ± 5.072 µM). Treatment with EtAc fraction dose dependently inhibited LPS-induced NO production (Figure 1A). The levels of NO production by EtAc fraction at 100 µg/mL dose concentration were significantly reduced NO production (38.54 ± 5.83 µM, $P \leq 0.001$) in compared to the LPS treated group. The standard drug, DEX (1 µg/mL) was also found to be suppressed the LPS-stimulated NO production (20.84 ± 6.35 µM; $P \leq 0.001$) by macrophages.

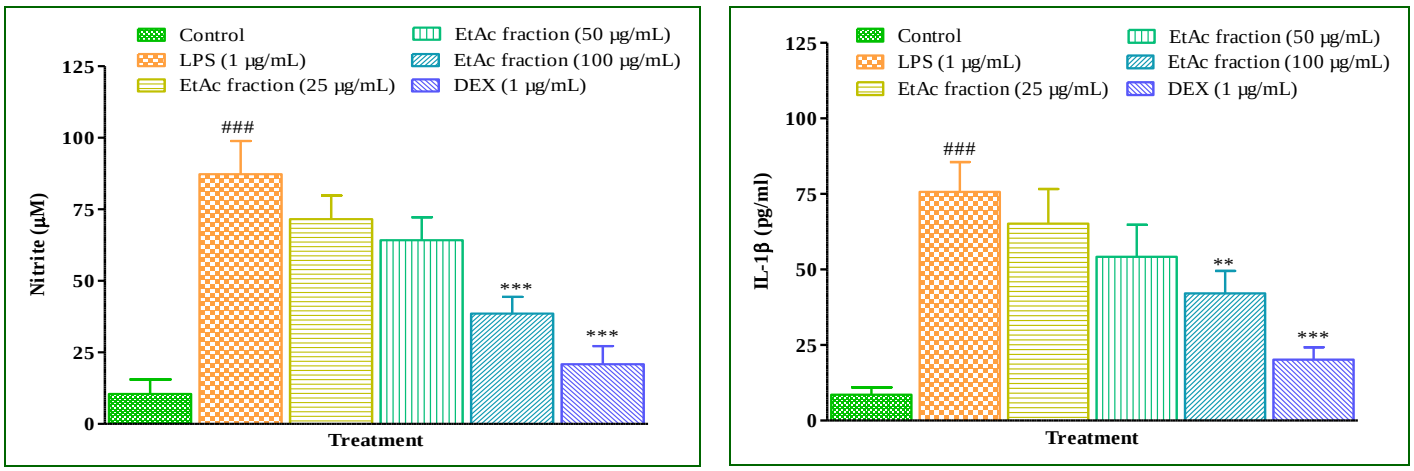

Figure 1: Effects of EtAc fraction on (A) NO production, and (B) IL-1β production in LPS-stimulated peritoneal macrophages.

Each bar represents mean ± SEM (n = 3). Statistical analysis was done through One-way ANOVA followed by Dunnett's multiple comparison test. Statistically significant differences compared as: Control Vs LPS (###P ≤ 0.001); LPS Vs EtAc fractions and DEX treated groups (***P ≤ 0.001 and (**P ≤ 0.01). (LPS = Lipopolysaccharide, and DEX = Dexamethasone).

IL-1β assay

Macrophage cells treated with LPS (1 µg/mL) markedly increased in the level of IL-1β, production in compared to the control group (75.67 ± 9.86 pg/mL and 8.58 ± 2.39 pg/mL, respectively, P ≤ 0.001). The EtAc fraction dose dependently suppressed the LPS-stimulated production of IL-1β (Figure 1B) by macrophages. The inhibition of IL-1β production by EtAc fraction at 100 µg/mL was found to be statistically significant (42.09 ± 7.41 pg/mL, P ≤ 0.01). DEX (1 µg/mL) was also significantly (P ≤ 0.001) suppressing the production IL-1β production by activated macrophages when compare with the control group (Figure 1B).

IL-6 assay

Macrophage cells treated with LPS (1 µg/mL) markedly increased in the level of IL-6, production in compared to the control group (2144.0 ± 61.49 pg/mL and 45.28 ± 7.09 pg/mL, respectively, P ≤ 0.001). The EtAc fraction dose dependently suppressed the LPS-stimulated production of IL-6 (Figure 2A) by macrophages. The inhibition of IL-6 production by EtAc fraction at dose 100 µg/mL was found to be statistically significant (1078.0 ± 195.8 pg/mL, P ≤ 0.01). DEX (1 µg/mL) was also significantly (P ≤ 0.001) suppressing the production IL-6 production by activated macrophages when compare with the control group (Figure 2A).

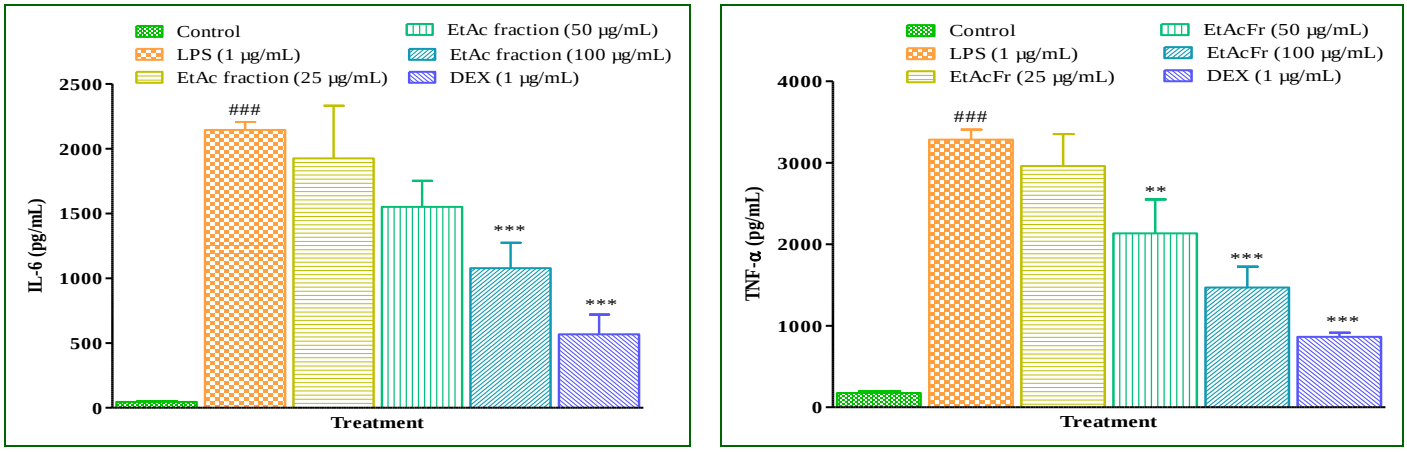

Figure 2: Effects of EtAc fraction on (A) IL-6 production, and (B) TNF-α production in LPS-stimulated peritoneal macrophages.

Each bar represents mean ± SEM (n = 3). Statistical analysis was done through One-way ANOVA followed by Dunnett's multiple comparison test. Statistically significant differences compared as: Control Vs LPS (###*P* ≤ 0.001); LPS Vs different EtAc fraction concentration and DEX treated groups (**P ≤ 0.01 and ***P ≤ 0.001). (LPS = Lipopolysaccharide; EtAc = Ethyl Acetate and DEX = Dexamethasone).

TNF-α Assay

Macrophage cells treated with LPS (1 µg/mL) significantly increased in the levels of TNF-α, production in compared to the control group (3285.0 ± 122.5 pg/mL and 175.5 ± 20.91 pg/mL, respectively, P ≤ 0.001). EtAc fraction dose dependently suppressed the LPS-stimulated production of TNF-α by macrophages (Figure 2B). The inhibition of TNF-α production by EtAc fraction at dose 50 µg/mL and 100 µg/mL was found to be statistically significant (P ≤ 0.01 and P ≤ 0.001, respectively). DEX (1 µg/mL) was significantly (P ≤ 0.001) suppressing the production TNF-α production by activated macrophages when compare with the control group.

Carrageenan-induced paw edema

The results of the carrageenan-induced anti-inflammatory activity are presented in the Table 1, indicating the percentage inhibition of edema volume at different time intervals (1 hr, 2 hr, and 5 hr) for each treatment group (n = 6). The control group (Vehicle) exhibited a substantial increase in edema volume over time, with values of 57.34%, 76.56%, and 98.76% at 1 hr, 2 hr, and 5 hr, respectively. Indomethacin (20 mg/kg), the standard anti-inflammatory drug, displayed a noticeable inhibitory effect, with values of 27.78%, 32.21%, and 54.16% at the corresponding time intervals.

The EtAc fraction treatment groups (50 mg/kg, 100 mg/kg, and 200 mg/kg) demonstrated varying degrees of inhibition. At 1 hr, EtAc fraction (50 mg/kg) showed a percentage inhibition of 16.39%, while EtAc fraction (100 mg/kg) and EtAc fraction (200 mg/kg) displayed higher inhibitions of 21.13% and 24.45%, respectively. The inhibitory effects diminished over time, with EtAc fraction (50 mg/kg) showing the least inhibition at 5 hr (11.08%), followed by EtAc fraction (100 mg/kg) and EtAc fraction (200 mg/kg) with values of 14.59% and 18.58%, respectively. These results suggest a dose-dependent response to the EtAc fraction treatment, with higher doses exhibiting more substantial inhibition of carrageenan-induced paw edema over the observed time intervals.

Table 1: Results of the carrageenan induced anti-inflammatory activity

Treatment	% inhibition in edema volume at different time intervals (n = 6)		
	1 hr	2 hr	5 hr
Control (Vehicle)	57.34 ± 1.80	76.56 ± 1.12	98.76 ± 0.96
Indomethacin (20 mg/kg)	27.78 ± 0.99***	32.21 ± 1.44***	54.16 ± 1.36
EtAc fraction (50 mg/kg)	16.39 ± 0.97***	14.08 ± 1.08***	11.08 ± 0.84
EtAc fraction (100 mg/kg)	21.13 ± 1.52***	19.63 ± 0.92***	14.59 ± 1.02
EtAc fraction (200 mg/kg)	24.45 ± 1.0***	20.34 ± 0.80***	18.58 ± 0.88

Note: Statistical analysis was done through One-way ANOVA followed by Dunnett's multiple comparison test. Statistical comparison Control vs Indomethacin and EtAc fraction treated group. ***P ≤ 0.001.

DISCUSSION

Inflammation, a complex physiological response against various harmful stimuli, including pathogens, damaged cells, or irritants [1]. It is crucial for the elimination of the initial causes of cell injury and the initiation of tissue repair, dysregulation or prolonged activation can lead to chronic inflammatory conditions, contributing to several diseases [1]. This study focused on exploring the anti-inflammatory potential of the standardized EtAc fraction of *C. pangorei* using both *in vitro* and *in vivo* models. Results from RP-HPLC analysis revealed the presence of quercetin, luteolin, and apigenin in the EtAc fraction, known for their anti-inflammatory activities [21,22].

The results of *in vitro* studies demonstrated the ability of the EtAc fraction to suppress the production of NO, IL-1β, IL-6, and TNF-α by macrophages stimulated with LPS. These findings are significant as excessive production of these inflammatory mediators is implicated in the pathogenesis of various inflammatory disorders [5,23]. The observed dose-dependent inhibition of NO, IL-1β, IL-6, and TNF-α production by the EtAc fraction suggests its potential as a therapeutic agent for combating inflammation.

The *in vivo* evaluation using the carrageenan-induced paw edema model further supported the anti-inflammatory efficacy of the EtAc fraction. Carrageenan-induced paw edema is a well-established experimental model utilized to investigate acute inflammation *in vivo*. This model mimics early stages of acute inflammation in humans, involving the release of key mediators like prostaglandins, histamine, and cytokines, resulting in increased vascular permeability and fluid accumulation in the paw [24]. Paw swelling serves as a quantitative measure of the inflammatory response, with inhibition of edema formation indicating potential anti-inflammatory properties of the tested compounds [25]. In this study, a significant reduction in paw edema were observed in a dose-dependent manner, with higher doses demonstrating more pronounced inhibitory effects. The observed anti-edematous activity of the EtAc fraction could be attributed to its ability to modulate inflammatory

pathways involved in the development of edema, such as the release of pro-inflammatory mediators and the recruitment of immune cells. Overall, the findings of this study highlight the promising anti-inflammatory potential of *C. pangorei* rhizome extracts, both *in vitro* and *in vivo*.

CONCLUSION

In conclusion, the comprehensive findings from this study underline the anti-inflammatory potential of *C. pangorei* rhizome extracts. The identification of quercetin, luteolin, and apigenin, coupled with the modulation of key inflammatory mediators, provides a foundation for further exploration of *C. pangorei* as a potential source for developing anti-inflammatory agents. Future studies elucidating the underlying molecular mechanisms and long-term effects of *C. pangorei* extracts are warranted to fully assess their therapeutic potential.

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Cite this article as:
Biswajit Samanta, S. K. Gupta, Amal K. Maji. Exploring the Anti-Inflammatory Potential of Cyperus pangorei Rhizome Extracts An In Vitro and In Vivo Study. International Journal of Ayurveda and Pharma Research. 2024;12(3):72-78.
https://doi.org/10.47070/ijapr.v12i3.3155
Source of support: Nil, Conflict of interest: None Declared

***Address for correspondence**
Amal K. Maji
Xplora Clinical Research Services Pvt. Ltd.,
Bangalore, Karnataka, India.
Email: majiamal99@gmail.com