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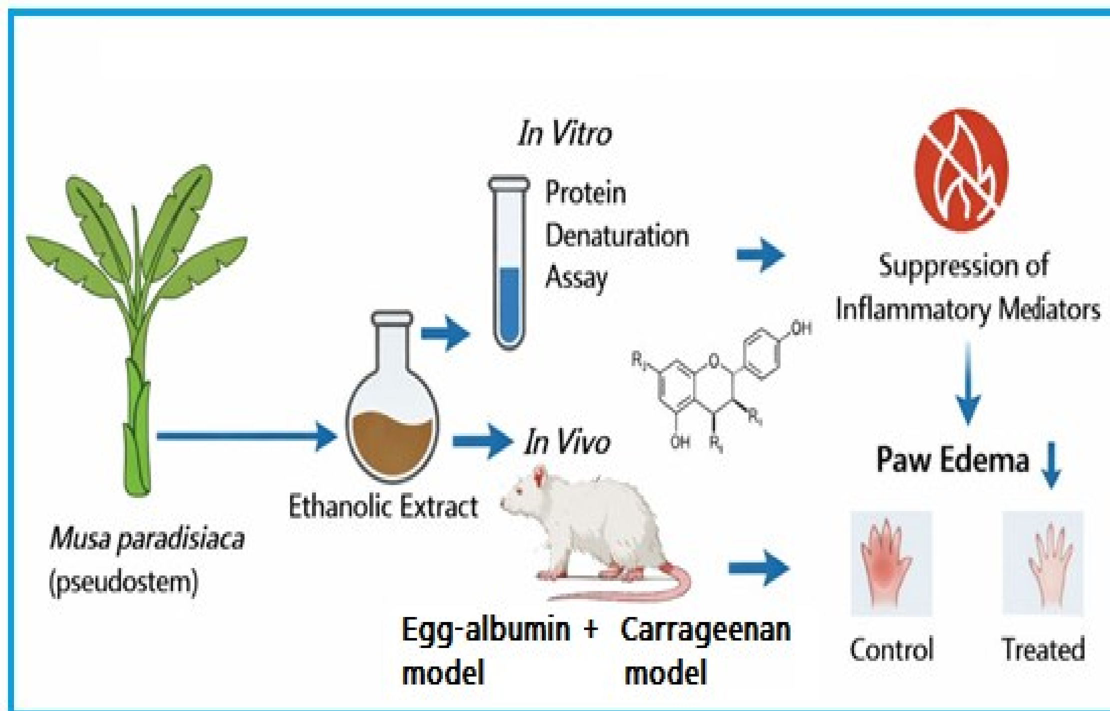

In vitro and *In vivo* Anti-Inflammatory Activities of Ethanolic Extract of *Musa Paradisiaca* L. (Plantain) Pseudo-Stem

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Abstract: *Inflammation is a protective biological response to tissue injury and infection; however, its dysregulation contributes to chronic diseases such as asthma, atherosclerosis, osteoarthritis, and cancer. The adverse effects associated with conventional non-steroidal anti-inflammatory drugs (NSAIDs) necessitate the search for safer alternatives from natural sources. This study evaluated the anti-inflammatory activity of the ethanolic extract of Musa paradisiaca pseudo-stem using in vitro and in vivo models. The pseudo-stem was air-dried, pulverized, and macerated in 95% ethanol for 72 h, followed by concentration under reduced pressure at 40 °C to obtain the crude extract. In vitro anti-inflammatory activity was assessed using the protein denaturation inhibition assay, while in vivo activity was evaluated using egg-albumin and carrageenan-induced paw edema models in Wistar rats. Data were expressed as mean ± SEM and analyzed using one-way ANOVA with Dunnett's post hoc test at $p < 0.05$. The extract exhibited concentration-dependent inhibition of protein denaturation, with percentage inhibitions of 47.6%, 52.3%, 55.8%, and 60.1% at 100, 200, 300, and 400 µg/mL, respectively, compared to aspirin (49.2%, 53.1%, 58.7%, and 63.9%). The IC_{50} values were 154 µg/mL for the extract and 116 µg/mL for aspirin. In the egg-albumin-induced model, the extract produced dose-dependent inhibition of paw edema, with a peak inhibition of 39.76% at 400 mg/kg, compared to 43.75% for diclofenac (50 mg/kg). In the carrageenan-induced model, the extract showed significant*

inhibition ranging from 23.00% to 42.37%, demonstrating activity in both early and late phases of inflammation. These findings indicate that the ethanolic extract of Musa paradisiaca pseudo-stem possesses significant anti-inflammatory activity, likely mediated through inhibition of protein denaturation and suppression of inflammatory mediators. The results provide scientific support for its traditional use and highlight its potential as a source of safer, plant-based anti-inflammatory agents.

Keywords: *Musa paradisiaca; pseudo-stem; anti-inflammatory activity; protein denaturation; paw edema.*

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1.0 Introduction

Inflammation is a complex biological response of body tissues to harmful stimuli such as pathogens, damaged cells, or irritants (Chen *et al*., 2018). It is beneficial in regulated amounts, but excess inflammation is responsible for chronic conditions, such as asthma, atherosclerosis, osteoarthritis and cancer (Medzhitov, 2021; Furman, *et al*., 2019). Phytochemicals have been reported to modulate inflammatory responses by regulating key mediators such as prostaglandins, histamine, serotonin, bradykinin, nitric oxide, and cytokines including tumour necrosis factor-α (TNF-α) and interleukin-1β (IL-1β). These mediators contribute to vascular changes, increased permeability, and leukocyte recruitment during inflammation (Gilroy & Kaplan, 2016). Phytochemicals such as flavonoids, terpenoids, alkaloids, and tannins exert anti-inflammatory effects through multiple mechanisms, including inhibition of pro-inflammatory enzymes (e.g., inducible nitric oxide synthase (iNOS) and cyclooxygenase-2 (COX-2)), suppression of signaling pathways such as Mitogen-Activated Protein Kinase (MAPK) and Nuclear Factor kappa B (NF-κB), reduction of oxidative stress, inhibition of protein denaturation, and stabilization of cellular membranes (Umapathy *et al*., 2010).

Conventional treatment for inflammatory conditions depends on the use of synthetic agents functioning as non-steroidal anti-inflammatory drugs (NSAIDS), corticosteroids, disease-modifying anti-rheumatic drugs (DMARDs), antihistamines, and antibiotics (Ghlichloo and Garriets, 2023; Benjamin, Goyal, and Lappin, 2024). These synthetic medicines are associated with various side effects on the body, for example, prolonged use of Non-Steroidal Anti-inflammatory Drugs (NSAIDs) is associated with gastrointestinal, renal, and cardiovascular side effects (Adams *et al*., 2011). These limitations have stimulated increased research interest in natural products as potential sources of safer and more effective anti-inflammatory agents. *Musa paradisiaca, commonly known as plantain, is a monocotyledonous herbaceous plant belonging to the Musaceae family.* It is abundant in the tropical and subtropical

countries of the world. Figure 1a shows *Musa paradisiaca* with mature fruit. The pseudo-stem of plantain (Figure 1b), though often regarded as agricultural waste, possesses several pharmacological properties. From literature, the juice of the plantain stem has been used as a diuretic to produce urine to flush out toxins from the body, and to dissolve urinary stones. It has also been reported to exhibit beneficial effects against conditions such as obesity, ulcers, hyperacidity, diabetes, and bleeding disorders. (Lavanya, Abi Beulah, and Vani, 2016). Other reports of the biological/pharmacological benefits of plantain pseudo stem include: antioxidant, antibacterial, antidiarrheal, hypocholesterolaemic and hepatoprotective properties (Hossain *et al*., 2011). However, most of these studies have focused on other parts of the plant such as the fruit and leaves, with comparatively limited investigation into the bioactive potential of the pseudo-stem, particularly with respect to its anti-inflammatory activity. Despite its widespread use in traditional medicine for the treatment of various ailments, there is limited scientific validation of the anti-inflammatory activity of *Musa paradisiaca* pseudo-stem, particularly using standardized *in vitro* and *in vivo* experimental models. In addition, the specific mechanisms underlying its effects remain inadequately characterized. Therefore, this study was designed to evaluate the anti-inflammatory activity of the ethanolic extract of *Musa paradisiaca* pseudo-stem using *in vitro* protein denaturation assays and *in vivo* egg-albumin and carrageenan-induced paw edema models.The *in vitro* activity was based on protein denaturation. The egg-albumin model indicates early-phase action, and is mediated by early-phase pro-inflammatory mediators such as bradykinin, histamine, serotonin (5-HT), early prostaglandins, mast-cell mediators and bradykinin (Dharmadeva *et al*., 2018).

The carrageenan model is a biphasic model which indicates both early and late phase activity. The early phase (0-2 h) is based on the same mediators in the egg–albumin model, including neuropeptides. The late action is mediated by mediators such as prostaglandins, cyclooxygenase-2 2 (COX-2) induction, cytokine-based edema and vascular factors, leucocyte infiltration, nitric oxide, tumour necrosis factor-α (TNF-α), interleukin-1β 1β (1L-1β), and reactive oxygen species (ROS) (Hamed *et al*., 2025). The findings from this study are expected to provide scientific evidence supporting the traditional use of plantain pseudo-stem and contribute to the development of novel, plant-based anti-inflammatory agents that are safer, more accessible, and cost-effective,

Fig. 1a Musa Paradisiaca (Plantain) Tree

Fig. 1b Musa Paradisiaca Pseudostem

2.0 Materials and Methods

2.1 Plant Material and Extract Preparation

Pseudo-stem of *Musa paradisiaca* (Figure 1(b)) was harvested from the botanical garden l to Alvan Ikoku Federal University of Education (AIFUE), Owerri, Imo State. It was authenticated by Dr. Evans Kemka, an expert in Plant Taxonomy at the Department of Biology, AIFUE, with voucher number: AIFUE-KEMKA-021-2025.The pseudo-stem was air-dried at room temperature and pulverized into a fine powder. A 500 g portion ق was macerated in 2.5 L of 95% ethanol for 72 h, after which it was filtered and concentrated using a rotary evaporator under reduced pressure at 40 °C to obtain the crude extract.

2.2 Chemicals and Reagents

The following analytical grade reagents were used: ethanol (96%, Sigma-Aldrich, USA), aspirin (Sigma-Aldrich, USA), diclofenac, disodium hydrogen phosphate (Na_2HPO_4), dipotassium hydrogen phosphate (K_2HPO_4), carrageenan (Sigma-Aldrich, USA), fresh egg albumin, and Tween 80 (polysorbate 80).

2.3 Experimental Animals

Adult Wistar rate of both sexes, 10 weeks old, weighing between 200-240 g, were obtained from the Department of Veterinary Medicine, Michael Okpara University of Agriculture, Umudike. They were kept under standard laboratory conditions of 25 °C, a light/dark cycle of 12 h, with access to water and feed. Ethical approval for the use of animals was obtained from the Research Ethics Committee of the College of Veterinary Medicine, Michael Okpara University of Agriculture, Umudike, Nigeria (Approval No.: MOUAU/CVM/REC/202608). A ll procedures were conducted in accordance with institutional guidelines for the care and use of laboratory animals.

2.4 Preparation of Extract and Drugs

The ethanolic extract of *Musa paradisiaca* and diclofenac were separately suspended in 1% v/v Tween 80 in normal saline prior to administration. Carrageenan and fresh egg albumin suspensions were freshly prepared in normal saline before use.

2.5 In vitro Anti-Inflammatory Activity

Albumin denaturation assays were conducted according to the method of Senadeera *et al.* (2021) , with modifications. Concentrations of the extract (100, 200, and 300 mg/mL) were prepared. Aspirin solutions were similarly prepared and used as the reference standard. Phosphate-buffered saline (PBS, pH 6.4) was prepared by dissolving 28.80 g of disodium hydrogen phosphate and 11.45 g of dipotassium hydrogen phosphate in 1000 mL of distilled water. For the preparation of the determined test samples, 2.8 mL of PBS, 0.2 mL egg albumin, and 0.2 mL plant extract was added. For the reference drug, test samples were prepared using 2.8 ml of PBS, 0.2 ml egg albumin, and 0.2 ml aspirin. A control sample was prepared using 2.8 ml of PBS, 2 ml of distilled water, and 0.2 ml of egg albumin. All prepared samples were incubated at 37 ºC in the water bath for 15-20 minutes using the laboratory shaking water bath, followed by heating at 70 ºC for five minutes. The samples were allowed to cool at room temperature for 10 minutes. Absorbances were taken at 660 nm against a blank, using the double-beam UV-Visible spectrophotometer. The whole test procedure was done in triplicate. % inhibition of protein denaturation was calculated using equation 1,

$$\%Inhibition = \frac{A_c - A_s}{A_c} \times \frac{100}{1} \quad (1)$$

where A_c and A_s are the absorbances of the control and sample, respectively. The IC_{50} values were calculated using dose–response nonlinear regression curves.

2.6 *In vivo* Anti-Inflammatory Activity

2.6.1 Egg albumin-induced paw edema

The modified method described by (Ezeja *et al.*, 2015) was adopted in this study. Briefly, 25 rats fasted of feed for 16 h were randomly divided into five groups (A-E) of 5 rats per

group. Access to water was withdrawn 2 h prior to the experiment and during the experiment. Group A (negative control) received 5 ml/kg of 1% v/v Tween 80 orally, group B (positive control) received 50 mg/kg of diclofenac orally, and groups C, D and E received 100, 200 and 400 mg/kg of extract, respectively, orally. One hour after treatment, paw oedema was induced by injecting 0.1 ml of egg-albumin into the sub-plantar surface of the hind right paw. Paw volume was measured using the water displacement method. The right paw volume was determined at 1, 2, 3 and 4 hours post-treatment. Increase in paw volume = right paw volume − left paw volume

$$\%inhibition = \frac{P(-ve)-P(s)}{P(-ve)} \times \frac{100}{1} \quad (2)$$

where $P(-ve)$ is paw volume of negative control, $P(s)$ is paw volume of test.

2.6.2 Carrageenan–induced paw edema

The modified method described by (Dumaro *et al*. (2016), was adopted in this experiment. Briefly, 25 rats fasted for 16 h were randomly divided into five groups (A-E) of 5 rats per group. Access to water was withdrawn 2 h prior to the experiment and during the experiment. Group A (negative control) received 5 ml/kg of 1% v/v Tween 80 orally, group B (positive control) received 50 mg/kg of diclofenac orally, and group C, D and E received 100, 200 and 400 mg/kg of extract respectively orally One hour after treatment, paw edema was induced by injecting 0.1 ml of egg-albumin into the sub-plantar surface of the hind right paw. The volume displacement method of water was used in paw volume measurement and the left hind paw served as the control. The right paw volume was determined at 1, 2, 3 and 4 hours post-treatment. The increase in paw volume = $right\ paw\ volume - left\ paw\ volume$. % inhibition was also calculated by the application of equation 2, given before.

2.7 Statistical Analysis

Results were expressed as mean ± standard error of mean (SEM). Statistical analysis was performed using one-way analysis of variance (ANOVA) followed by Dunnett's post hoc test. Differences were considered statistically significant at $p < 0.05$.

3.0 Results and Discussion

3.1 In vitro Anti-Inflammatory Activity

The *in vitro* anti-inflammatory activity of the ethanolic extract of *Musa paradisiaca* pseudo-stem was evaluated using the protein denaturation inhibition assay, with aspirin serving as the reference standard. The results (Table 1; Fig. 2) demonstrate that both the extract and aspirin inhibited protein denaturation in a concentration-dependent manner over the tested range (100–400 µg/mL).

At concentrations of 100, 200, 300, and 400 µg/mL, the extract exhibited percentage inhibitions of 47.6%, 52.3%, 55.8%, and 60.1%, respectively, whereas aspirin showed slightly higher inhibitions of 49.2%, 53.1%, 58.7%, and 63.9% at corresponding concentrations. The IC_{50} values further confirm this trend, with aspirin (116 µg/mL) demonstrating greater potency compared to the extract (154 µg/mL).

The observed inhibition of protein denaturation suggests that the extract possesses the ability to stabilize proteins against heat-induced denaturation, which is a well-recognised mechanism underlying anti-inflammatory activity. Protein denaturation is implicated in inflammatory conditions such as rheumatoid arthritis, where denatured proteins act as autoantigens, triggering immune responses. Therefore, the ability of the extract to inhibit protein denaturation indicates its potential to prevent inflammatory processes at the molecular level.

Although aspirin exhibited superior activity, the extract showed comparable inhibitory effects, particularly at higher concentrations, suggesting the presence of bioactive phytochemicals with anti-inflammatory

properties. The dose-dependent response observed indicates a direct correlation between extract concentration and anti-inflammatory efficacy, which may be attributed to increasing availability of active constituents such as flavonoids, tannins, and phenolic compounds.

Table 1. Percentage inhibition of protein inhibition of ethanolic extract of plantain pseudo-stem, and aspirin versus concentration

Conc. (µg/mL)	% Inhibition (Extract, IC_{50} = 154 µg/mL)	% Inhibition (Aspirin, IC_{50} = 116 µg/mL)
0	47.6	49.2
200	52.3	53.1
300	55.8	58.7
400	60.1	63.9

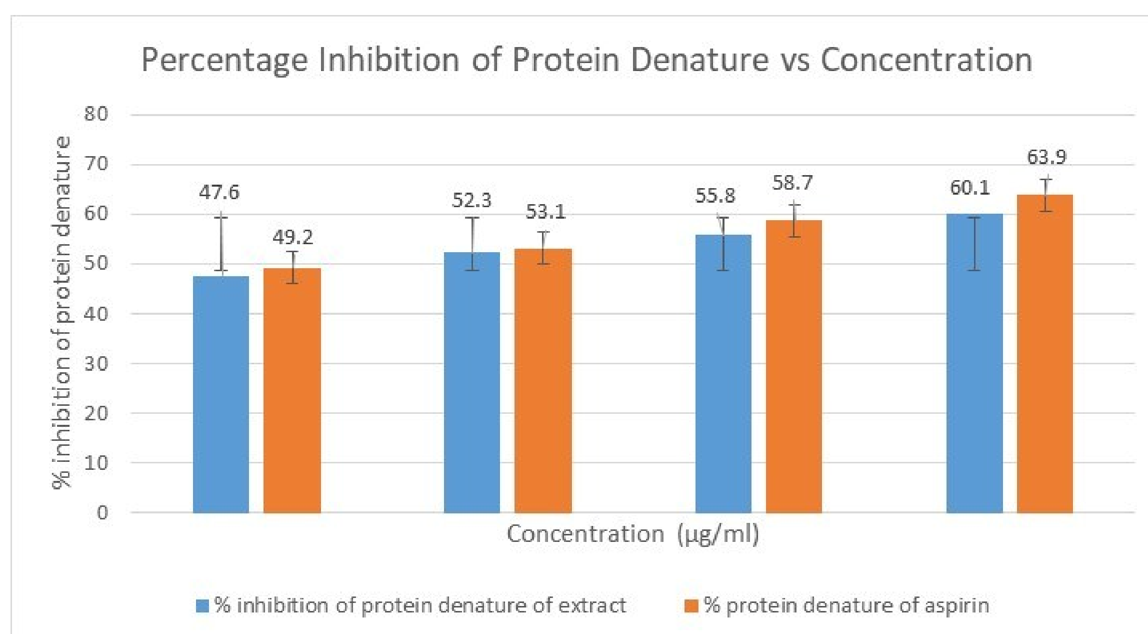

Fig. 2. Percentage inhibition of protein denature of extract and aspirin versus concentration

3.2 In vivo Anti-Inflammatory Activity

3.2.1 Egg Albumin-Induced Paw Edema

The anti-inflammatory effect of the extract was further evaluated using the egg albumin-induced paw edema model, which primarily reflects the early phase of acute inflammation mediated by histamine, serotonin, and bradykinin. The results (Table 2, Table 3; Figure 3) indicate that the extract significantly ($p < 0.05$) reduced paw edema in a dose-dependent manner.

At 100 mg/kg, the extract produced inhibition ranging from 19.54% to 36.25% across the 1–4 h observation period. At 200 mg/kg, inhibition ranged from 17.24% to 26.25%, while the highest dose (400 mg/kg) exhibited greater inhibition ranging from 29.59% to 39.76%. The standard drug, diclofenac (50 mg/kg), showed higher inhibition overall, reaching a maximum of 43.75% at 4 h.

Notably, at the 2 h time point, the extract at 400 mg/kg produced 35.63% inhibition, which exceeded that of diclofenac (28.74%), indicating a strong effect during the early phase of inflammation. This suggests that the extract may effectively inhibit early-phase inflammatory mediators such as histamine and serotonin. The reduction in paw edema

observed in this model indicates that the extract interferes with vascular permeability and fluid accumulation, which are hallmarks of acute inflammation. The dose-dependent inhibition further supports the presence of active phytoconstituents capable of modulating early inflammatory responses.

Table 2. *In-vivo* egg-albumin induced paw edema

Treatment	% paw-edema inhibition			
	1h	2h	3h	4h
50 mg/kg of Diclofenac	31.63	28.74	31.33	43.75
Extract 100 mg/kg	20.41	19.54	31.33	36.25
Extract 200 mg/kg	24.49	17.24	22.89	26.25
Extract 400 mg/kg	29.59	35.63	39.76	38.75

***p < 0.05 when compared to the negative control group**

Table 3. *In-Vivo* egg albumin-induced % paw-edema inhibition

Treatment	Negative Control	50 mg/kg of Diclofenac	Extract 100 mg/kg	Extract 200 mg/kg	Extract 400 mg/kg
1h	0.98 ± 0.03	0.67 ± 0.05 (31.63)*	0.78 ± 0.10 (20.41)*	0.74 ± 0.02 (24.49)*	0.69 ± 0.04 (29.59)*
2h	0.87 ± 0.03	0.62 ± 0.07 (28.74)*	0.70 ± 0.04 (19.54)*	0.72 ± 0.04 (17.24)	0.56 ± 0.06 (35.63)*
3h	0.83 ± 0.04	0.57 ± 0.03 (31.33)*	0.57 ± 0.04 (31.33)*	0.64 ± 0.02 (22.89)*	0.50 ± 0.05 (39.76)*
4h	0.80 ± 0.02	0.45 ± 0.03 (43.75)*	0.51 ± 0.07 (36.25)*	0.59 ± 0.06 (26.25)*	0.49 ± 0.05 (38.75)*

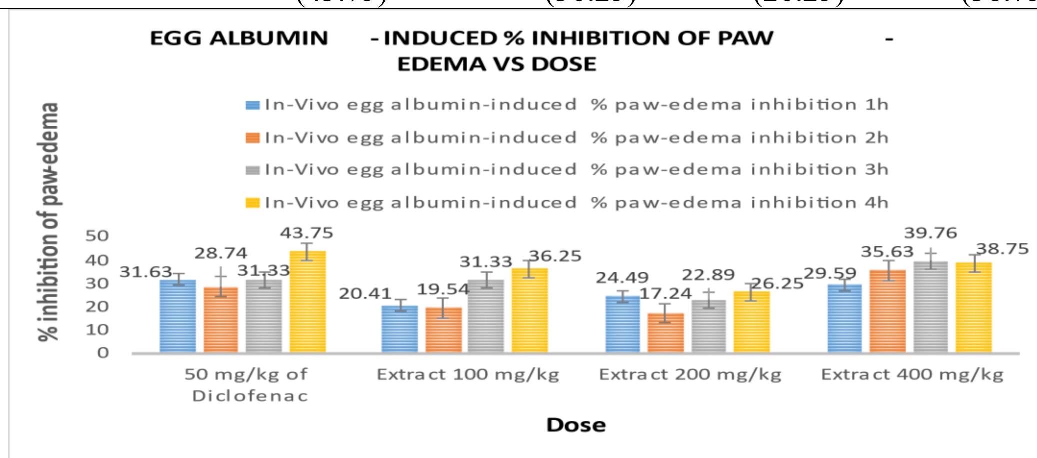

Figure 3. Bar chart showing *In-Vivo* egg-albumin induced -induced % paw-edema inhibition by *Musa paradisiaca* ethanolic extract

The extract showed a dose-dependent anti-inflammatory effect in the carrageenan model (Table 3). As displayed in figure 4, the extract gave 23-30.61% inhibition at 100 mg/kg, 20.41-41.18 % inhibition at 200 mg/kg, 25.88-42.37 % inhibition at 400 mg/kg, while Diclofenac at 50 mg/kg gave up to 36% across the time measured. The two models demonstrate that decreases in paw edema are

associated with higher doses of the plant extract.

Table 3. *In-Vivo* Carrageenan-induced paw edema (*p < 0.05 when compared to the negative control group)

	Mean increase in paw volume in ml ± SEM (% inhibition)			
	1h	**2h**	**3h**	**4h**
Negative control	0.49 ± 0.04	0.59 ± 0.01	0.85 ± 0.06	1.00 ± 0.08
Diclofenac 50 mg/kg	0.37 ± 0.05 (4.49)*	0.47 ± 0.06 (20.34)*	0.58 ± 0.05 (31.70)*	0.64 ± 0.07 (36.00)*
Extract 100 mg/kg	0.34 ± 0.03 (30.61)*	0.45 ± 0.03 (23.73)*	0.61 ± 0.03 (28.20)*	0.77 ± 0.03 (23.00)*
Extract 200 mg/kg	0.39 ± 0.03 (20.41)*	0.42 ± 0.06 (28.81)*	0.50 ± 0.05 (41.10)*	0.67 ± 0.05 (33.00)*
Extract 400 mg/kg	0.31 ± 0.02 (36.73)*	0.34 ± 0.02 (42.37)*	0.63 ± 0.09 (25.88)*	0.73 ± 0.05 (27.00)*

3.2.2 Carrageenan-Induced Paw Edema

The carrageenan-induced paw edema model was employed to evaluate both early and late phases of inflammation. The early phase (0–2 h) is mediated by histamine, serotonin, and bradykinin, while the late phase (3–4 h) involves prostaglandins, cytokines, and nitric oxide.

As presented in Table 4 and Figure 4, the extract exhibited significant ($p < 0.05$) anti-inflammatory activity across all doses and time points. At 100 mg/kg, inhibition ranged from 23.00% to 30.61%; at 200 mg/kg, from 20.41% to 41.10%; and at 400 mg/kg, from 25.88% to 42.37%. Diclofenac (50 mg/kg) showed inhibition up to 36.00%. During the early phase (1–2 h), the extract at 400 mg/kg demonstrated superior inhibition (36.73% and 42.37%) compared to diclofenac (4.49% and 20.34%), indicating strong suppression of early inflammatory mediators. In the late phase (3–4 h), the extract at 200 mg/kg produced a peak inhibition of 41.10% at 3 h, exceeding diclofenac (31.70%), suggesting effective inhibition of prostaglandin synthesis and other late-phase mediators. The biphasic inhibitory pattern observed indicates that the extract possesses a broad-spectrum anti-inflammatory effect, acting on both early and late phases of inflammation. This dual activity is particularly significant, as it suggests multiple mechanisms of action, including inhibition of cyclooxygenase pathways, cytokine production, and oxidative stress

3.3 Comparative Analysis and Mechanistic Insights

A comparative evaluation of the *in vitro* and *in vivo* results reveals that the ethanolic extract of *Musa paradisiaca* pseudo-stem exhibits consistent anti-inflammatory activity across different experimental models. Although the extract showed slightly lower potency than standard drugs *in vitro*, its performance *in vivo*, particularly at higher doses, was comparable and in some cases superior to diclofenac.

The enhanced activity observed *in vivo* may be attributed to synergistic interactions among phytochemicals present in the extract, which could modulate multiple inflammatory pathways simultaneously. The ability of the extract to inhibit protein denaturation, reduce paw edema, and suppress both early and late inflammatory mediators suggests a multi-target mechanism of action.

Table 4. *In-Vivo* carrageenan-induced % paw-edema inhibition

		Mean increase in paw volume in ml ± SEM (% inhibition)			
		1h	**2h**	**3h**	**4h**
Diclofenac mg/kg	50	0.37 (4.49)*	0.47 (20.34)*	0.58 (31.7)*	0.64 (36)*
Extract mg/kg	100	0.34 (30.61)*	0.45 (23.73)*	0.61 (28.2)*	0.77 (23)*
Extract mg/kg	200	0.39 (20.41)*	0.42 (28.81)*	0.50 (41.1)*	0.67 (33)*
Extract mg/kg	400	0.31 (36.73)*	0.34 (42.37)*	0.63 (25.88)*	0.73 (27)*

Table 5. Percentage inhibition of paw edema in carrageenan-induced inflammation in rats treated with *Musa paradisiaca* pseudo-stem extract and diclofenac over time

	In-Vivo carrageenan-induced % paw-edema inhibition			
	1h	**2h**	**3h**	**4h**
Diclofenac 50 mg/kg	4.49	20.34	31.70	36.00
Extract 100 mg/kg	30.61	23.73	28.20	23.00
Extract 200 mg/kg	20.41	28.81	41.10	33.00
Extract 400 mg/kg	36.73	42.37	25.88	27.00

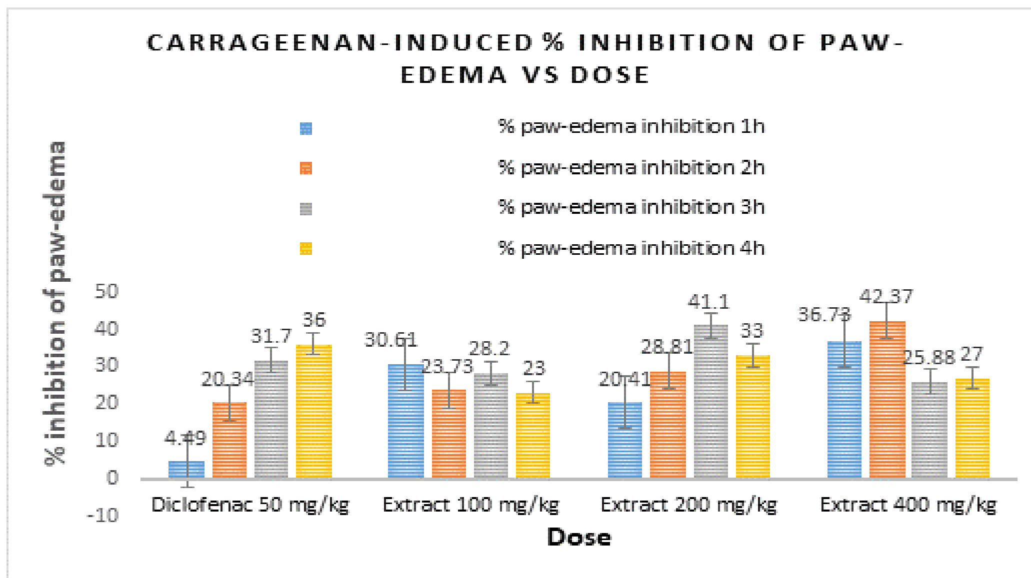

Figure 4. Bar chart showing *In-Vivo* carrageenan-induced % paw-edema inhibition by *Musa paradisiaca* ethanolic extract

The anti-inflammatory effects of the extract are likely mediated through stabilization of lysosomal membranes, which prevents the release of inflammatory enzymes. It may also act by inhibiting pro-inflammatory mediators such as prostaglandins and cytokines. In

addition, its antioxidant activity helps to reduce oxidative stress associated with inflammation, while the suppression of vascular permeability and leukocyte infiltration further contributes to its anti-inflammatory action.

These findings are consistent with previous reports on plant-derived anti-inflammatory agents, which attribute such activities to bioactive compounds including flavonoids, tannins, alkaloids, and phenolic acids.

3.4 Overall Interpretation

Overall, the ethanolic extract of *Musa paradisiaca* pseudo-stem demonstrated significant dose-dependent anti-inflammatory activity in both *in vitro* and *in vivo* models. While aspirin and diclofenac exhibited higher potency in some cases, the extract showed comparable efficacy, particularly at higher doses, and demonstrated the ability to act on multiple phases of inflammation.

These results provide scientific support for the traditional use of plantain pseudo-stem in the management of inflammatory conditions and highlight its potential as a source of bioactive compounds for the development of safer and cost-effective anti-inflammatory agents.

4.0 Conclusion

Results from the present study demonstrate that the ethanolic extract of *Musa paradisiaca* pseudo-stem possesses significant dose-dependent *in vitro* and *in vivo* anti-inflammatory activities. *In vitro*, the extract effectively inhibited protein denaturation, indicating membrane-stabilizing properties and potential anti-arthritic effects, although at slightly higher concentrations than aspirin.

The *in vivo* studies, using egg-albumin and carrageenan-induced paw edema models, further confirmed the anti-inflammatory activity of the extract across both early and late phases of inflammation. Notably, higher doses of the extract produced inhibitory effects comparable to, and in some instances exceeding, those of diclofenac. This suggests that the pseudo-stem contains bioactive phytochemicals capable of modulating multiple inflammatory pathways, including early-phase mediators such as histamine and serotonin, as well as late-phase mediators such as cytokines, prostaglandins, cyclooxygenase-2 (COX-2), and reactive oxygen species.

These findings provide scientific validation for the traditional use of plantain pseudo-stem in the management of inflammatory conditions and highlight its potential as a promising natural source of anti-inflammatory agents. Further studies are recommended to include the isolation and characterization of the bioactive compounds responsible for the observed anti-inflammatory activity, as well as comprehensive safety and toxicity evaluations. In addition, the development of standardized formulations for therapeutic application of *Musa paradisiaca* pseudo-stem is warranted.

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Declaration

Consent for publication

Not Applicable

Availability of data and materials

The publisher has the right to make the data public

Conflict of Interest

The authors declared no conflict of interest

Ethical Considerations

Not applicable

Competing interest

The authors report no conflict or competing interest

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Ethical Approval

Ethical approval for this research was granted by the Research Ethics Committee of the College of Veterinary Medicine, Michael Okpara University of Agriculture, Umudike, Abia State, Nigeria, with approval number MOUAU/CVM/REC/202608. The approval guaranteed that:

All procedures were carried out by trained personnel in appropriate laboratory facilities. The *in vitro* experiments were carried out in accordance with good laboratory practice under good conditions. The *in vivo* anti-inflammatory studies were conducted strictly following the principles of Replacement, Reduction, and Refinement (the 3Rs).

Adequate measures were taken to minimize pain, distress, and discomfort to the animals. The sourcing, collection, handling and use of plant in this research were conducted in a responsible manner and in compliance with international guidelines and regulations.

Authors' Contributions

RIU conceptualized the study, LCN edited the manuscript, CPN did the write-up, CM did literature search. All authors participated in conducting the experiment.