



Research Article

Gastroprotective Effects of Aqueous Leaf Extract of *Launaea taraxacifolia* Against Indomethacin-Induced Gastric Ulcer in Wistar Rats

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ABSTRACT

Gastric ulcer is a common NSAID complication, and affordable plant-based gastroprotective agents remain a priority in low-resource settings. This study evaluated the aqueous leaf extract of *Launaea taraxacifolia*, a West African ethnomedicinal leafy vegetable, against indomethacin-induced gastric mucosal injury in female Wistar rats using prophylactic and curative dosing. Thirty-five rats were allotted into seven groups of five: negative and ulcer controls, an extract-protective group (7-day pre-treatment), a standard-drug (omeprazole) control, and three curative groups (low, medium, or high dose; 21 days). The extract gave a 9.3% yield, rich in flavonoids (11.04%) and cardiac glycosides (9.21%); acute toxicity testing showed no mortality or signs of toxicity up to 5000 mg/kg (LD₅₀ > 5000 mg/kg). Indomethacin raised serum tumour necrosis factor- α (TNF- α) from 22.65 to 104.72 pg/mL and interleukin-1 beta (IL-1 β) from 30.93 to 105.72 pg/mL, lowered catalase from 39.62 to 7.40 U/L and SOD from 122.50 to 49.98 U/L, and raised MDA from 2.52 to 10.25 nmol/mL (one-way ANOVA, $p < 0.0001$ for all five). Extract pre-treatment and omeprazole restored TNF- α , IL-1 β , catalase, and MDA to negative-control levels (Tukey-adjusted $p > 0.05$) and raised SOD above the ulcer control, though only omeprazole did not differ from the negative control for SOD; pre-treatment gave lower IL-1 β than omeprazole ($p = 0.037$). Histologically, only the untreated ulcer control showed an overt erosive lesion; the standard-drug and curatively-dosed groups showed a dose-dependent recovery gradient. These findings indicate a dose-dependent gastroprotective effect of *Launaea taraxacifolia* against indomethacin-induced gastric injury, with a biochemical profile broadly similar to omeprazole on the parameters tested.

Keywords: Gastric ulcer; Gastroprotection; Indomethacin; inflammation; *Launaea taraxacifolia*; Oxidative stress; Wistar rats

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INTRODUCTION

Gastric ulcer is a type of peptic ulcer disease characterised by localized loss of the gastric mucosa extending through the muscularis mucosae into the submucosa, arising largely from acid-pepsin-induced

injury to the stomach lining (Kumar *et al.*, 2020). It remains a significant clinical problem worldwide, driven substantially by the widespread use of non-steroidal anti-inflammatory drugs (NSAIDs), which injure the gastric mucosa primarily through inhibition

of cyclo-oxygenase-1: this reduces protective prostaglandin synthesis and mucosal blood flow while promoting mitochondrial dysfunction and neutrophil-driven generation of reactive oxygen species (Sohail *et al.*, 2023). This mechanistic pathway directly motivates the biochemical markers assessed in the present study: depletion of the antioxidant enzymes catalase and superoxide dismutase (SOD) and elevation of the lipid-peroxidation product malondialdehyde (MDA) reflect the oxidative arm of mucosal injury, while elevation of the pro-inflammatory cytokines tumour necrosis factor- α (TNF- α) and interleukin-1 beta (IL-1 β) reflects the inflammatory arm, together providing a biochemical signature of the two convergent injury pathways that a gastroprotective agent would be expected to attenuate. This burden is expected to persist given the rising prevalence of chronic pain conditions and the corresponding increase in long-term analgesic use across both developed and developing countries (Islam *et al.*, 2024).

Although conventional anti-ulcer drugs such as proton pump inhibitors are effective, their long-term use is associated with adverse effects including high cost, vitamin B12 and magnesium deficiency, increased susceptibility to enteric infections, and rebound acid hypersecretion on discontinuation (Maideen, 2023). These limitations are particularly pronounced in developing countries, including Nigeria, where access to sustained pharmacological treatment may be limited and reliance on traditional medicine remains high; a substantial proportion of the population in many developing countries has long been estimated to rely on traditional medicine, including medicinal plants, as a primary source of health care (World Health Organization, 2025), underscoring the general public-health rationale for affordable, effective, and locally available alternatives.

Launaea taraxacifolia is an herbaceous plant of the Asteraceae family, growing as an erect annual herb reaching one to two metres in height and commonly known as wild lettuce in English, Efo yanrin in Yoruba, and Namijin dayii or Noomen barewa in Hausa (Kew Royal Botanic Gardens, 2023). Beyond its use as a leafy vegetable, it is used in West African ethnomedicine for digestive disorders, inflammation, hypertension, and diabetes, and recent studies have documented antioxidant, antidiabetic, nephroprotective, and neuroprotective activity for its leaf extract, alongside a favourable acute toxicological profile (Diallo *et al.*, 2020). Despite this pharmacological plausibility and its traditional use for

digestive complaints, to the authors' knowledge, evidence for its specific gastroprotective and anti-ulcer potential, particularly for the aqueous leaf extract, against experimentally induced gastric ulceration remains limited.

This study therefore tested the hypothesis that the aqueous leaf extract of *Launaea taraxacifolia* protects against indomethacin-induced gastric mucosal injury in Wistar rats, evaluating its phytochemical composition, acute oral toxicity, and gastroprotective effect on serum biochemical markers of oxidative stress and inflammation and on histological indices of mucosal injury. The design distinguished two related but distinct questions: whether seven days of extract pre-treatment before ulcer induction confers prophylactic protection against mucosal injury, and, separately, whether daily extract administration for 21 days after ulcer induction promotes curative healing of an established lesion, with the aim of generating experimental evidence to support the rational use of the extract as a complementary or alternative gastroprotective agent.

MATERIALS AND METHODS

Study Design and Area

An experimental research design was adopted at the Department of Medical Laboratory Science, Faculty of Allied Health Sciences, College of Health Sciences, Kwara State University, Malete, Kwara State, Nigeria, over six weeks. The design involved induction of gastric ulcer in a controlled setting, followed by treatment with graded extract doses, and comparison with negative and ulcer controls and a standard reference drug, to establish a dose-dependent therapeutic relationship.

Experimental Animals and Sample Size

A total of 52 mature female Wistar rats (*Rattus norvegicus*) weighing 120–150 g, procured from the Animal House, University of Ilorin, Ilorin, Kwara State, Nigeria, were used: 5 rats for determination of the ulcerogenic dose of indomethacin, 12 rats for the acute toxicity study, and the remaining 35 allotted into seven groups of five for the main experimental phase. Rats were acclimatized to the laboratory environment for 7 days before experimentation began. Five animals per group was adopted for the main experimental phase, consistent with the reduction principle of animal research ethics and with the group sizes commonly used in comparable exploratory rodent gastroprotection and pharmacology studies.

Plant Material and Extraction

Launaea taraxacifolia leaves were collected from the Ilorin countryside of Kwara State, Nigeria, and authenticated at the Herbarium of the Department of Plant Biology, University of Ilorin (voucher number UILH/001/1872/2026). Fresh leaves were sorted to remove damaged material, rinsed to remove dust and debris, air-dried under shade at room temperature for two weeks to preserve heat-labile constituents, and pulverised into a fine powder. Extraction was by maceration of 100 g of the powder in 1.0 L of distilled water for 72 hours with periodic stirring; the mixture was filtered, and the filtrate concentrated to dryness using a rotary evaporator maintained at a moderate temperature. Percentage yield was calculated from the dry extract mass relative to the starting powder mass, and the dried extract was stored in an airtight container at a low temperature until use. For dosing, the required mass of dried extract for each group was reconstituted by dissolving it in 1 mL of distilled water immediately before oral administration by gavage.

Ethical Considerations

Ethical approval was obtained from the Ethical Review Committee, Kwara State University Centre for Research Development (KWASU/CR&D/REA/2026/0321) prior to any animal experimentation. All animals received humane care in accordance with the Principles of Laboratory Animal Care of the National Society for Medical Research, and the study conformed to the ARRIVE 2.0 guidelines (Percie du Sert *et al.*, 2020). Animals were housed in well-ventilated cages under standard laboratory conditions (temperature 24–28°C; relative humidity 50–65%) and a 12-hour light-dark cycle, with free access to standard rodent feed and water except during mandated fasting, and every effort was made to minimise pain and distress throughout the study. Animals were randomly allocated to groups using a random-number-based method. Inclusion criteria were healthy, mature female Wistar rats weighing 120–150 g, verified free of pre-existing health conditions by physical examination; rats showing illness or outside this weight range were excluded.

Phytochemical Screening and Quantification

Preliminary qualitative phytochemical screening of the aqueous leaf extract of *Launaea taraxacifolia* was carried out using standard reagent-based colour and precipitation tests following the protocols described by Harborne (1980). The extract was screened for alkaloids using Mayer's and Dragendorff's reagents, flavonoids using the Shinoda (magnesium-hydrochloric acid) test, phenolic compounds and tannins using the ferric chloride test, saponins using

the froth test, cardiac glycosides using the Keller-Killiani test, and steroids and terpenoids using the Liebermann-Burchard test. The relative abundance of each constituent was visually graded, based on the intensity of colour change or precipitate formation, as trace amount (+), abundant (++), or most abundant (+++). Quantitative estimation of each constituent was subsequently carried out in triplicate using standard spectrophotometric methods with the appropriate reference-standard calibration curve for each class, and gravimetric methods for alkaloids, saponins, cardiac glycosides, steroids, and terpenoids, with each constituent expressed as a percentage (% w/w) of the dry extract.

Determination of the Ulcerogenic Dose of Indomethacin

A pilot study was conducted to determine the ulcerogenic dose of indomethacin for the main experimental phase, using five rats in total, one animal per dose group: one animal served as a negative control and received distilled water only; and one animal each received a single oral dose of indomethacin at 30 mg/kg, 40 mg/kg, 50 mg/kg, and a repeated 50 mg/kg (the last to confirm the reproducibility of the ulcerogenic response at that dose before adopting it for the main study), after an 18–24-hour fast with free access to water. Animals were euthanised 8–10 hours after administration, and the stomach was opened along the greater curvature and examined macroscopically and, following fixation in 10% neutral buffered formalin, histologically, for ulcerative lesions. No ulceration was observed in the negative control or at 30 or 40 mg/kg, whereas both animals dosed at 50 mg/kg developed consistent, reproducible gastric ulceration, meeting the reproducibility criterion for selecting an ulcerogenic dose. On this basis, 50 mg/kg body weight was selected as the ulcerogenic dose for the main experimental phase.

Acute Toxicity Study

Acute oral toxicity was evaluated by Lorke's method (Lorke, 1983), in two sequential phases: in Phase I, groups of three rats were dosed at 10, 100, and 1000 mg/kg body weight; in Phase II, single rats were dosed at 1600, 2900, and 5000 mg/kg. Animals were observed for mortality and clinical signs of toxicity, including behavioural changes (tremors, lethargy, altered feeding pattern), changes in skin and fur appearance, salivation, diarrhoea, altered sleep pattern, convulsions, and respiratory changes, for the first 4 hours and thereafter daily for 14 days. No mortality or observable toxicity signs occurred at any dose tested, up to and including the 5000 mg/kg limit

dose; as no lethal dose was reached, the median lethal dose (LD50) is reported as greater than 5000 mg/kg, rather than as evidence that the extract is unconditionally non-toxic. This LD50 threshold guided selection of the low, medium, and high therapeutic doses (250, 500, and 1000 mg/kg, respectively) used in the main experimental phase.

Experimental Grouping and Ulcer Induction

The 35 remaining rats were allotted into seven groups of five: Group I (negative control) received distilled water only, without ulcer induction; Group II (ulcer control) received distilled water only, following ulcer induction; Group III (extract protective group) received the aqueous extract for 7 days prior to ulcer induction, to test a prophylactic, pre-exposure paradigm; Group IV (standard-drug control) received omeprazole (20 mg/kg body weight/day), reconstituted in distilled water and administered orally by gavage once daily for 21 days after ulcer induction, matching the treatment duration of the curative extract groups to allow a direct endpoint comparison; and Groups V, VI, and VII (low-, medium-, and high-dose curative groups) received the low, medium, or high extract dose, respectively, once daily by gavage for 21 days after ulcer induction, to test a curative, post-injury healing paradigm. Because the prophylactic and curative groups differ in both the timing of extract exposure relative to injury and in total exposure duration (7 days versus 21 days), the two paradigms address related but distinct questions, protection against injury versus healing of an established lesion, and are not treated as directly equivalent; the only direct statistical comparison drawn between an extract-treated group and the omeprazole group in this study is the prophylactic-versus-omeprazole comparison reported for interleukin-1 beta. Gastric ulcer was induced by a single oral dose of indomethacin (50 mg/kg) by gastric gavage, following an approximately 24-hour fast with free access to water.

Data Collection, Tissue Processing, and Histopathological Staining

A logbook recorded dosage administration and daily observations of animal health, behaviour, and body weight; body weight was monitored throughout the study for welfare-monitoring and dose-adjustment purposes. All 35 animals allotted to the main experimental phase completed the study, with no animal excluded after randomization and no animal meeting a predefined humane endpoint for weight loss during the study. At the end of treatment, animals were euthanised under deep anaesthesia and blood was collected by cardiac puncture for

biochemical analysis, and stomachs were excised, examined macroscopically, and processed for histological analysis. For histology, stomachs were preserved in 10% neutral buffered formalin, processed through routine dehydration, clearing, and paraffin embedding, and sectioned using a rotary microtome. Sections were stained with haematoxylin and eosin (H&E): deparaffinization in xylene and rehydration through descending grades of alcohol; staining with Harris haematoxylin for 10–15 minutes; differentiation in 1% acid alcohol; bluing in running tap water; counterstaining with eosin for 1–2 minutes; dehydration through ascending grades of alcohol; clearing in xylene; and mounting with DPX mountant (Awwioro, 2010). Sections were photomicrographed at $\times 40$ and $\times 100$ magnifications and assessed qualitatively by an observer blinded to treatment group; the photomicrographs presented in Figures 1 and 2 are representative of the histological pattern observed across the animals in each group.

Biochemical Analysis of Oxidative Stress and Inflammatory Markers

Blood samples were allowed to clot at room temperature and centrifuged at 3,000 rpm for 10 minutes to separate serum, which was stored at -20°C until assay. Serum was assayed for the oxidative stress markers malondialdehyde (MDA) and superoxide dismutase (SOD) and the antioxidant enzyme catalase (CAT), and for the inflammatory markers tumour necrosis factor-alpha (TNF- α) and interleukin-1 beta (IL-1 β) by enzyme-linked immunosorbent assay (ELISA), following the respective assay-kit manufacturers' protocols.

Statistical Analysis

Data were analyzed using SPSS (SPSS Inc., Chicago, IL, USA), version 23.0. Results were expressed as mean $\pm$ standard error of the mean (SEM). Group comparisons used one-way analysis of variance (ANOVA), followed by Tukey's Honestly Significant Difference (HSD) post-hoc test for pairwise multiple comparisons, with $p < 0.05$ considered statistically significant.

RESULTS

Aqueous extraction of *Launaea taraxacifolia* leaves gave a percentage yield of 9.3%, with the extract gummy in texture, brown in colour, and sweet-smelling. Qualitative screening confirmed alkaloids, flavonoids, phenols, saponins, tannins, cardiac glycosides, steroids, and terpenoids, with flavonoids most abundant (+++), followed by alkaloids, saponins, and cardiac glycosides (++ each), while phenols, tannins, steroids, and terpenoids occurred in trace

amounts. Quantitatively, flavonoids recorded the highest concentration (11.04%), followed by cardiac glycoside (9.21%), alkaloids (8.43%), saponins (7.65%), phenol (2.81%), tannins (1.81%), terpenoids (0.57%), and steroids (0.43%), indicating that the extract was particularly rich in flavonoids and alkaloids (Table 1).

No mortality and no observable signs of toxicity occurred at any of the six acute toxicity doses tested, up to and including the limit dose of 5000 mg/kg body weight (Table 2). As no lethal dose was reached, the oral median lethal dose (LD50) of the extract in Wistar rats is reported as greater than 5000 mg/kg; this indicates the absence of acute lethality or overt toxicity signs under the conditions tested, rather than establishing the extract as unconditionally non-toxic, and guided selection of the low, medium, and high doses (250, 500, and 1000 mg/kg, respectively) used for the main experimental phase. Ulcer induction (Group II) sharply worsened all five biochemical markers relative to the negative control: TNF- α rose from 22.65 to 104.72 pg/mL, IL-1 β from 30.93 to 105.72 pg/mL, and MDA from 2.52 to 10.25 nmol/mL, while catalase fell from 39.62 to 7.40 U/L and SOD from 122.50 to 49.98 U/L (one-way ANOVA, $p < 0.0001$ for all five parameters; Table 3). Extract pre-treatment (Group III) and omeprazole (Group IV) both returned TNF- α , IL-1 β , catalase, and MDA to levels not significantly different from the negative control (Tukey-adjusted $p > 0.05$ vs Group I in each case), and

both significantly raised SOD above the ulcer control, though only omeprazole's SOD value did not differ significantly from the negative control (Table 3). Among the curatively-dosed groups, every parameter improved progressively with increasing extract dose. Histological sections corroborated the biochemical findings closely (Figures 1 and 2). The untreated ulcer control (Group II) showed the only overt erosive lesion among the seven groups, with necrotic slough, loss of surface epithelium and glands, and dense inflammatory infiltrate in the lamina propria (Figure 1).

A direct pairwise comparison between the extract protective group and the omeprazole group was significant for this parameter (Group III vs Group IV, Tukey-adjusted $p = 0.037$), indicating significantly lower IL-1 β with prophylactic extract pre-treatment than with the standard drug.

The negative control and extract protective groups (Groups I and III) showed preserved mucosa, submucosa, and muscularis layers with no erosion, ulceration, or significant inflammation (Figure 1). The standard-drug and curatively-dosed groups (Groups IV-VII) showed a visible, dose-dependent gradient of mucosal recovery: preserved architecture in the standard-drug group, moderate-to-dense inflammatory infiltrate with submucosal congestion at the low dose, moderate infiltrate at the medium dose, and near-normal architecture with no significant inflammation at the high dose (Figure 2).

Table 1: Phytochemical Constituents of *Launaea taraxacifolia* Aqueous Leaf Extract

Phytochemical constituent	Qualitative	Quantitative (%)
Alkaloids	++	8.43
Flavonoids	+++	11.04
Phenols	+	2.81
Saponins	++	7.65
Tannins	+	1.81
Cardiac glycosides	++	9.21
Steroids	+	0.43
Terpenoids	+	0.57

+ = trace amount; ++ = abundant; +++ = most abundant

Table 2: Acute Oral Toxicity of Aqueous Leaf Extract of *Launaea taraxacifolia* in Wistar Rats

Phase	Dose (mg/kg)	Number of animals	Mortality
I	10	3	0/3
I	100	3	0/3
I	1000	3	0/3
II	1600	1	0/1
II	2900	1	0/1
II	5000	1	0/1

Table 3: Effect of Aqueous Leaf Extract of *Launaea taraxacifolia* on Serum Oxidative Stress and Inflammatory Marker Concentrations across Experimental Groups

Group	N	TNF- α (pg/mL)	p (I/II)	IL-1 β (pg/mL)	p (I/II)	Catalase (U/L)	p (I/II)	SOD (U/L)	p (I/II)	MDA (nmol/mL)	p (I/II)
I – Negative control	5	22.65 $\pm$ 10.73	—/< 0.0001	30.93 $\pm$ 12.59	—/< 0.0001	39.62 $\pm$ 6.48	—/< 0.0001	122.50 $\pm$ 39.58	—/< 0.0001	2.52 $\pm$ 1.07	—/< 0.0001
II – Ulcer control	5	104.72 $\pm$ 12.45	< 0.0001/—	105.72 $\pm$ 16.32	<0.0001/—	7.40 $\pm$ 3.15	< 0.0001/—	49.98 $\pm$ 15.45	< 0.0001/—	10.25 $\pm$ 2.17	<0.0001/—
III – Extract protective	5	28.05 $\pm$ 10.78	0.9802/< 0.0001	26.82 $\pm$ 13.04	0.9967/<0.0001	35.15 $\pm$ 15.23	0.9285/< 0.0001	87.15 $\pm$ 6.37	0.0399/0.0275	3.12 $\pm$ 1.21	0.9786/<0.0001
IV – Omeprazole (standard drug)	5	41.20 $\pm$ 13.13	0.1195/< 0.0001	50.75 $\pm$ 6.71	0.1203/<0.0001	42.95 $\pm$ 3.11	0.9825/< 0.0001	90.42 $\pm$ 4.70	0.0755/0.0139	4.17 $\pm$ 0.67	0.2954/< 0.0001
V – Low dose (250 mg/kg)	5	81.15 $\pm$ 12.45	< 0.0001/0.0252	90.28 $\pm$ 12.72	<0.0001/0.3421	8.88 $\pm$ 1.43	< 0.0001/0.9998	65.28 $\pm$ 7.16	0.0004/0.7673	8.12 $\pm$ 0.98	<0.001/0.0926
VI – Medium dose (500 mg/kg)	5	64.83 $\pm$ 6.57	< 0.0001/0.0001	67.22 $\pm$ 7.07	0.0007/0.0004	13.33 $\pm$ 1.03	0.0001/0.7834	72.97 $\pm$ 3.31	0.0020/0.3436	6.43 $\pm$ 0.47	0.0004/0.0005
VII – High dose (1000 mg/kg)	5	50.35 $\pm$ 2.08	0.0063/< 0.0001	54.40 $\pm$ 4.54	0.0428/<0.0001	16.20 $\pm$ 2.55	0.0002/0.3801	82.88 $\pm$ 2.82	0.0166/0.0645	4.70 $\pm$ 0.49	0.0808/<0.0001

Data are expressed as Mean $\pm$ SD (n = 5 per group). Each “p (I/II)” column gives the exact Tukey HSD-adjusted p-value for the biomarker immediately to its left, versus Group I (negative control) / versus Group II (untreated ulcer control); — = a group's comparison with itself. One-way ANOVA: TNF- α F(6, 21) = 40.05; IL-1 β F(6, 21) = 34.23; catalase F(6, 21) = 27.02; SOD F(6, 21) = 9.40; MDA F(6, 21) = 30.15 (all p < 0.0001). TNF- α = tumour necrosis factor-alpha; IL-1 β = interleukin-1 beta; SOD = superoxide dismutase; MDA = malondialdehyde.

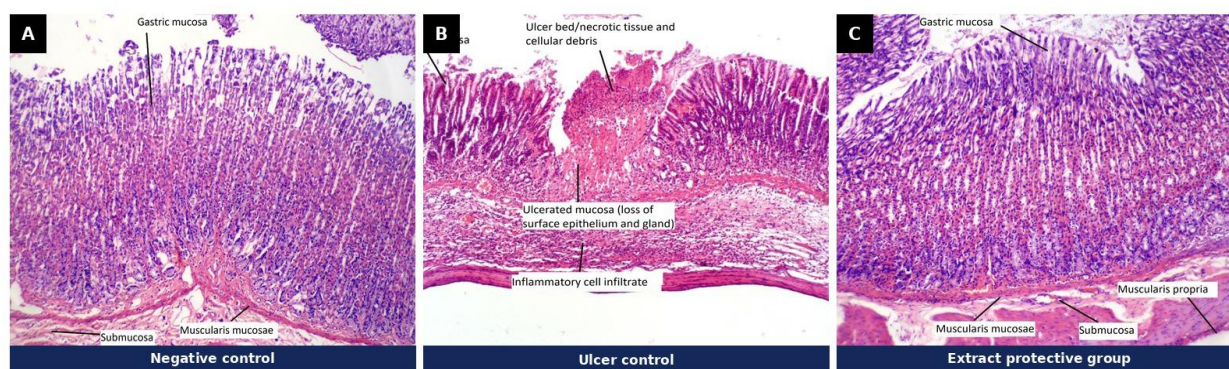


Figure 1: Photomicrographs of Negative Control, Ulcer Control, and Extract Protective Group (H&E, ×100)

H&E = haematoxylin and eosin. (A) Negative control: preserved gastric mucosa, submucosa, and muscularis mucosae; no erosion, ulceration, or significant inflammation. (B) Ulcer control: focal ulcer bed with necrotic slough, loss of surface epithelium and glands, and dense inflammatory infiltrate. (C) Extract protective group: preserved mucosa, submucosa, and muscularis propria; no erosion, ulceration, or significant inflammation.

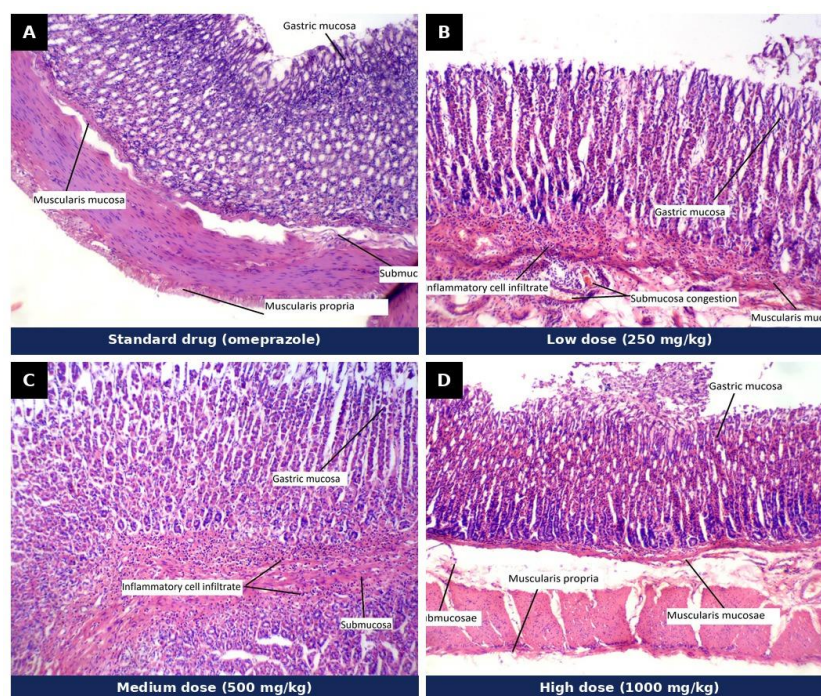


Figure 2: Photomicrographs of Standard Drug and Curatively-Dosed Groups (H&E, ×100)

H&E = haematoxylin and eosin. (A) Standard drug (omeprazole): preserved mucosa, submucosa, and muscularis propria; no erosion, ulceration, or significant inflammation. (B) Low dose (250 mg/kg): preserved mucosa with moderate-to-dense inflammatory infiltrate and submucosal congestion; no erosion or ulceration. (C) Medium dose (500 mg/kg): preserved mucosa with moderate inflammatory infiltrate; no erosion or ulceration. (D) High dose (1000 mg/kg): preserved mucosa, submucosa, and muscularis propria with near-normal architecture; no erosion, ulceration, or significant inflammation.

DISCUSSION

This study evaluated the therapeutic effect of the aqueous leaf extract of *Launaea taraxacifolia* against indomethacin-induced gastric ulcer in Wistar rats.

Indomethacin produced a marked rise in serum TNF- α , IL-1 β , and MDA, a marked fall in serum catalase and SOD activity, and an overt erosive lesion on histology, a pattern consistent with the established mechanism

by which indomethacin and other non-selective NSAIDs injure the gastric mucosa: inhibition of cyclooxygenase-1 depletes cytoprotective prostaglandins, while mitochondrial uncoupling and neutrophil infiltration generate reactive oxygen species that overwhelm mucosal antioxidant defence (Sohail *et al.*, 2023). This internal consistency indicates that the indomethacin-induced ulcer model was successfully reproduced, providing a reliable injury baseline against which the extract's effects can be judged. Treatment with the extract, whether given prophylactically before or curatively after ulcer induction, reversed this biochemical and histological pattern in a dose-dependent manner; because prophylaxis and curative healing are distinct biological processes assessed over different exposure durations.

Both TNF- α and IL-1 β were markedly suppressed by the extract relative to the untreated ulcer group, tracking dose in the curatively-treated groups in a manner consistent with other indomethacin-ulcer models in which gastroprotective phytochemicals suppressed the same cytokines, including normalisation of TNF- α , IL-1 β , IL-6, and NF- κ B by ferulic acid (Ermis *et al.*, 2023) and suppression of IL-1 β , IL-6, and TNF- α by carnosic acid alongside upregulation of the Nrf2/HO-1 pathway (Danisman *et al.*, 2023). Notably, IL-1 β in the extract-protective group was significantly lower than in the omeprazole group (Tukey-adjusted $p = 0.037$, Table 3), the only biochemical parameter for which a direct extract-versus-omeprazole comparison reached significance in this study; because omeprazole acts principally by suppressing gastric acid secretion rather than through a direct anti-inflammatory or antioxidant action (Maideen, 2023), this specific finding suggests that prophylactic extract administration may confer an additional layer of anti-inflammatory protection beyond that provided by acid suppression alone, plausibly related to the antioxidant and anti-inflammatory activity documented for flavonoids such as those abundant in this extract (Abou Baker, 2022), though the mechanism was not directly tested here. The extract likewise produced a clear, dose-related restoration of catalase and SOD activity and a corresponding reduction in MDA in the curatively-treated groups; catalase, the parameter most severely depleted in the untreated ulcer group, was substantially restored by prophylactic and standard-drug treatment, whereas curative dosing produced only a smaller, though still dose-dependent, rise. This coordinated pattern across catalase, SOD, and MDA parallels findings for other antioxidant-rich agents

evaluated against indomethacin-induced ulceration, including *Muscari neglectum* (Soydan *et al.*, 2025) and carnosic acid (Danisman *et al.*, 2023), and is consistent with the antioxidant activity already documented for *Launaea taraxacifolia* leaf extract in diabetic (Gbadamosi *et al.*, 2020), nephropathic (Afolabi *et al.*, 2026), and neurotoxic (Umoren *et al.*, 2025) rat models.

A recurring pattern across most biochemical parameters was the stronger performance of prophylactic relative to curative extract administration: the prophylactically treated group did not differ significantly from the negative control for TNF- α , IL-1 β , catalase, and MDA, with only its SOD activity remaining modestly, though significantly, below the negative control ($p = 0.040$) despite being significantly higher than the untreated ulcer control ($p = 0.028$); the curatively treated groups, by contrast, required the highest dose tested to approach the same benchmark for several parameters. Because the prophylactic and curative regimens differ in both timing and total exposure duration, this comparison is presented as a descriptive observation about the two paradigms rather than a formal statistical test of prophylaxis against curative healing, and should be interpreted with this design difference in mind; it is nonetheless of translational interest given the traditional use of *Launaea taraxacifolia* as a regularly consumed leafy vegetable in West African diets (Diallo *et al.*, 2020). Across the curatively-treated groups, every parameter improved progressively with dose; this monotonic dose-response pattern, consistent across five biologically distinct parameters, is an encouraging trend, though with five animals per group it should be interpreted with appropriate statistical caution rather than treated as definitive confirmation of a true dose-response relationship. The histopathological findings corroborated the biochemical results closely, in keeping with the established practice of using histological grading to complement biochemical indices of gastric mucosal injury (Avwioro, 2010); the standard-drug group performed at least as well as, and was frequently statistically indistinguishable from, the extract-protective group on the biochemical parameters where both were compared against the negative and ulcer controls, despite the two acting through different mechanisms. This pattern is broadly consistent with reports for other gastroprotective agents evaluated against indomethacin-induced ulceration, including near-complete restoration of mucosal architecture reported for the aqueous stem bark extract of *Ficus platyphylla* at its highest tested

dose (Ugwah-Oguejiofor *et al.*, 2026), and with reviews of gastroprotective medicinal plants from the wider West and North African region (Cherrada *et al.*, 2024). These findings are compatible with the mucosal-barrier and free-radical theories of gastric ulceration, in which indomethacin shifts the balance toward the aggressive pole through cyclo-oxygenase-1 inhibition and prostaglandin depletion, and the extract, given prophylactically or curatively, at least partly restores the defensive component. We propose, as a hypothesis for future mechanistic work rather than a demonstrated finding, that the extract's phytochemical constituents scavenge reactive oxygen species and support endogenous antioxidant enzyme activity, interrupting the cascade of lipid peroxidation and inflammatory amplification that follows indomethacin-induced mitochondrial dysfunction and neutrophil infiltration (Ermis *et al.*, 2023; Danisman *et al.*, 2023); no mechanistic endpoint specific to this pathway was directly measured in the present study, and this proposed mechanism should accordingly be read as a plausible hypothesis rather than a demonstrated one. Similarly, the biochemical profile of the extract is described here as broadly similar to that of omeprazole where both did not differ significantly from the negative or ulcer control; except for interleukin-1 beta, where a direct pairwise comparison was performed and reached significance, this is not equivalent to a formal demonstration that the extract and omeprazole are statistically indistinguishable from each other.

CONCLUSION

In this preliminary study, the aqueous leaf extract of *Launaea taraxacifolia* was associated with a dose-dependent reduction in indomethacin-induced serum biochemical and gastric histological injury in Wistar rats, evidenced by lower pro-inflammatory cytokine (TNF- α , IL-1 β) and MDA concentrations, higher catalase and SOD activity, and preserved mucosal architecture on histology, relative to the untreated ulcer control. Both prophylactic pre-treatment and curative post-induction dosing were associated with improvement, with prophylactic administration showing the more complete biochemical normalisation on the parameters tested. These findings are consistent with, and provide preliminary experimental support for, the traditional ethnomedicinal use of *Launaea taraxacifolia* for digestive and inflammatory conditions in West African folk medicine, and identify it as a candidate for further pharmacological development.

Authors' Contributions

Balogun, M.O. developed the concept, methodology and led the study as principal author. Fowotade, A.A. supervised the research. Ogunwale, K.A.T. and Garuba, W.O. carried out the biochemical analyses. Mustapha, A. carried out plant preparation and phytochemical analysis. Akanbiola, O.I. carried out tissue processing and staining. Hassan, S.O. performed the statistical analysis, writing and review. All authors read and approved the final manuscript.

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Data Availability: Data supporting the findings of this study are available from the corresponding author upon reasonable request.

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