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Antiulcer Activity of Herbal Tea Prepared from Unripe *Musa paradisiaca* Linn. in Rats

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Abstract

Musa paradisiaca (plantain) is a traditional medicinal plant used to treat various gastrointestinal disorders in humans. This study evaluated the anti-ulcer properties of herbal tea prepared from unripe *Musa paradisiaca* fruit using three ulcer models: indomethacin-, stress-, and ethanol-induced ulceration in rats. Herbal tea was prepared by steeping dried unripe fruits in warm water, and its safety profile was assessed using acute toxicity studies. Rats were administered herbal tea at doses of 100, 200, and 400 mg/kg for seven days. The positive control was omeprazole (20 mg/kg), and the negative control was distilled water (5 ml/kg). The results revealed that the extract exhibited dose-dependent inhibition of ulcer in all the models with curative ratios ranging from 80.77% to 100%, similar to that of omeprazole. The highest activity was observed in the ethanol-induced ulcer model, in which a 400 mg/kg dose of the herbal tea produced a 100% cure rate. In the stress-induced ulcer model, the protective effect reached a maximum of 95.23% at 400 mg/kg. Acute oral toxicity studies showed no mortality at doses up to 5000 mg/kg body weight, indicating low toxicity. These findings suggest that *Musa paradisiaca* herbal tea has significant anti-ulcer effects and low toxicity, making it a potential therapeutic agent for the management of peptic ulcers.

Keywords: *Musa paradisiaca*, herbal tea, anti-ulcer, peptic ulcer, antioxidant

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Introduction

Peptic ulcer disease (PUD) is one of the most common gastrointestinal disorders worldwide and greatly impairs the quality of life through recurrent epigastric pain, bleeding risk, and treatment burden (Sung *et al*., 2009; Gupta *et al*., 2023; Srivastav *et al*., 2023). It classically presents as ulcers in the stomach or duodenum, with duodenal ulcers being more closely associated with *Helicobacter pylori* infection and gastric acid exposure (Jin *et al*., 2024; Pan and Jiao, 2025). The pathogenesis reflects an imbalance between aggressive factors (gastric acid, pepsin, *Helicobacter pylori)*, and mucosal defenses. Nonsteroidal anti-inflammatory drugs, smoking, alcohol, and *Helicobacter pylori* are major risk factors for ulcer formation, and their pathogenesis (Kuna *et al*., 2019; Quan and Talley, 2002; Ardalani *et al*., 2020; Gupta *et al*., 2023;). Standard therapy relies on acid suppression using proton pump inhibitors (PPIs) and H2-receptor antagonists to heal ulcers and control symptoms (Chubineh and Birk, 2012; Kuna et al., 2019). Common adverse effects associated with these drugs include headache, dizziness, and gastrointestinal disturbances, and relapse rates are high after discontinuation of acid suppression if *H. pylori* is not eradicated or NSAID exposure persists (Quan and Talley, 2002; Sung *et al*., 2009; Kuna *et al*., 2019).

These limitations have stimulated interest in safer complementary strategies derived from medicinal plants with diverse actions that reinforce mucosal defenses in addition to moderating acid secretion (Kuna *et al*., 2019; Ajijolakewu *et al*., 2021). Among these candidates, *Musa paradisiaca* (plantain) stands out for its long-standing ethnomedicinal use in treating gastrointestinal disorders and its rich reservoir of bioactive constituents.

Plantains and related *Musa species* contain flavonoids, tannins, phenolic acids, alkaloids, and peptides, which collectively exhibit antioxidant, anti-inflammatory, cytoprotective, and antisecretory activities (Loganayaki *et al*., 2010; Akhtar *et al*., 2011; Ajijolakewu *et al*., 2021). These properties align fundamentally with contemporary models of ulcer pathogenesis, in which oxidative stress, proinflammatory cytokines, and disruption of the mucus-bicarbonate barrier synergize with aggressive factors such as acids, pepsin, NSAIDs, and *H. pylori* to injure the mucosa. Antioxidants and phenolics from *Musa spp*. can scavenge reactive oxygen species, preserve non-protein sulfhydryl pools, support prostaglandin-mediated cytoprotection, and modulate inflammatory signalling, thereby helping to restore the balance toward mucosal defense (Baskar *et al*., 2011; Sidahmed *et al*., 2016; Moawad *et al*., 2019).

In traditional rodent models, *Musa* preparations ranging from unripe fruit powder to methanol or aqueous extracts of the pulp and peel have consistently demonstrated potent gastroprotective effects. These preparations have been shown to reduce ulcer indices and lesion areas, improve gastric pH, decrease total acidity, and preserve epithelial architecture on histological examination. Additionally, *Musa* extracts enhance endogenous antioxidant defenses, particularly by increasing the activity of enzymes such as superoxide dismutase (SOD), catalase (CAT), and glutathione peroxidase (GPx), which collectively reduce lipid peroxidation and oxidative stress in gastric tissues (Motawi *et al*., 2008; Kadir *et al*., 2014; Gong *et al*., 2021; Sanpinit *et al*., 2021)

Early pharmacological studies on *Musa sapientum* var. *paradisiaca* powders and peel extracts reported significant anti-ulcerogenic and mucosal cytoprotective effects in both acute and chronic ulcer models in rats, aligning with the traditional use of plantain products for managing gastritis and peptic ulcers (Best *et al*., 1984; Ahmed *et al*., 2020; Ajijolakewu *et al*., 2021)

More recent studies have provided molecular insights into these effects, suggesting that *Musa paradisiaca* extracts act through multiple signaling pathways, including the modulation of the nuclear factor erythroid 2–related factor 2 (Nrf2) and nuclear factor-kappa B (NF-κB) pathways. These pathways are crucial for redox balance and inflammation control, respectively (Van Der Horst *et al.,* 2022)

The upregulation of Nrf2-dependent genes such as heme oxygenase-1 (HO-1) and NAD(P)H quinone oxidoreductase-1 (NQO1) indicates that *Musa* extracts strengthen antioxidant responses, while inhibition of NF-κB signaling reduces pro-inflammatory cytokines like TNF-α, IL-1β, and IL-6 pathways. These pathways are crucial for redox balance and inflammation control, respectively (Ghasemzadeh and Hosseinzadeh, 2023)

Such dual antioxidant and anti-inflammatory modulation are particularly valuable for gastric cytoprotection, as it not only limits tissue injury but also promotes mucosal healing and angiogenesis. Furthermore, metabolomic profiling has revealed that *Musa* species contain a rich polyphenolic matrix, including flavonoids (such as quercetin, catechin, and rutin), phenolic acids, saponins, and terpenoids, which collectively contribute to its cytoprotective efficacy (Akhtar *et al*., 2011). These bioactive compounds act synergistically to scavenge reactive oxygen species (ROS), stabilize cell membranes, and regulate nitric oxide (NO) bioavailability-an important mediator of gastric microcirculation and epithelial regeneration (Liu *et al*., 2023)

The polyphenolic complexity of *Musa paradisiaca* thus provides a broader therapeutic spectrum compared to single-target synthetic drugs, offering additive and possibly synergistic protection against diverse ulcerogenic insults such as ethanol, stress, and NSAID-induced gastric damage (Kinsey *et al*., 2011).

In addition to its cytoprotective and antioxidant effects, studies have pointed to the prebiotic potential of *Musa*

polysaccharides, which may support gastric and intestinal health by modulating gut microbiota composition. These non-digestible carbohydrates promote the growth of beneficial bacteria like *Lactobacillus* and *Bifidobacterium*, which enhance mucosal defense and suppress harmful pathogens (Singh *et al*., 2016; Bhatia *et al*., 2021). The integration of these findings suggests that *Musa paradisiaca* exerts a multifaceted protective role, combining direct mucosal defense with microbiota-mediated homeostasis a feature highly relevant to current approaches in gastrointestinal health management.

Overall, contemporary evidence supports the view that *Musa paradisiaca* functions as a multi-target gastroprotective agent. Through its antioxidant, anti-inflammatory, cytoprotective, and microbiota-stabilizing effects, it represents a promising natural therapeutic for the prevention and management of peptic ulcer disease and related gastrointestinal disorders. The growing body of molecular and clinical data positions *Musa paradisiaca* as more than a traditional remedy, it is now emerging as a scientifically validated phytopharmaceutical candidate for modern ulcer therapy.

Although translating these findings to human relevance is not complete for ulcer healing itself, clinical studies involving green bananas in children with persistent diarrhea indirectly support this (Rabbani *et al*., 2004). The improved outcomes of these studies suggest beneficial effects on the intestinal lining and inflammation. (Rabbani *et al*., 2004; Morsy and El-Moselhy, 2013). Additional translational cues originate from bioactive peptide fractions in green banana flour, which have demonstrable anti-inflammatory properties in *in vitro* and *ex vivo* models (Zielińska *et al.*, 2018). Collectively, this evidence positions *Musa paradisiaca* as both a functional food and a potential source of standardized phytopharmaceuticals for gastroprotection.

This study aimed to evaluate the anti-ulcer properties of herbal tea derived from the unripe fruit of *Musa paradisiaca* using three well-established ulcer models.

Materials and Methods

Plant Collection and Identification

Unripe *Musa paradisiaca* fruit (375 g) was purchased from Ogige Main Market, Nsukka, Enugu State, Nigeria, on April 16, 2024. The plant material was identified and authenticated by Mr. Felix Nwafor, a botanist at the Department of Pharmacognosy and Environmental Medicine, Faculty of Pharmaceutical Sciences, University of Nigeria, Nsukka, Enugu State, Nigeria. A voucher specimen (no. PCG/UNN/102) was deposited in the herbarium for reference.

Preparation of the Herbal Tea (HT)

The unripe fruits were peeled, washed with distilled water, and air-dried for 6 h. The samples were oven-dried for additional 1 h and subsequently milled into a fine powder using an industrial mill. Herbal tea was prepared by infusing a tea bag in 100 mL of hot water in a covered container for 10 min.

Experimental Animals

Seventy-five albino rats of both sexes (50-100 g) were obtained from the Animal House of the Department of Pharmacology and Toxicology, Faculty of Pharmaceutical Sciences, University of Nigeria, Nsukka. The animals were housed under standard laboratory conditions, acclimatized for one week prior to the experiment. They had food and water ad libitum. All experimental procedures and protocols were reviewed and approved by the Institutional Animal Ethics Committee of the Department of Pharmacology and Toxicology, University of Nigeria, Nsukka, in compliance with national and international guidelines for the care and use of laboratory animals.

Drugs and Chemicals

The following drugs and chemicals were used in this study: distilled water, normal saline, ether, omeprazole (McCoy Pharma Pvt. Ltd., India), indomethacin (Krishat Pharma Industries Ltd., Nigeria), absolute ethanol (analytical grade), and herbal tea prepared from *Musa paradisiaca*.

Acute Toxicity Test

Acute toxicity testing was conducted using 13 mice in two phases, following standard protocols (Lorke, 1983).

Phytochemical Analysis of Unripe Musa paradisiaca

Qualitative phytochemical screening and quantitative assays were conducted as previously outlined by Harborne (1998) and Trease and Evans (2002).

Anti-ulcer studies

Indomethacin-Induced Ulcer Model

Twenty-five albino rats were divided into five groups (n=5), each receiving a different treatment. The animals were fasted for 24 h before the experiment. Ulceration was induced by administering a single oral dose of 15 mg/kg of indomethacin (Jafari *et al*., 2022). The treatment groups were as follows:

Group I: 100 mg/kg HT, Group II: 200 mg/kg HT, Group III: 400 mg/kg HT, Group IV: 20 mg/kg omeprazole (positive control), Group V: 5 ml/kg distilled water (negative control). After seven days of treatment, the animals were sacrificed, and their stomachs were excised and opened along the greater curvature.

Determination of the mean ulcer index

Ulcerative index was measured briefly as reported by Divyapraba, *et al*., (2021), the stomach was opened and washed with running tap water, then was placed on a flat glass plate and observed under 10X magnification for ulcers (Brzozowski, 1996). Scoring of the ulcer will be made as follows: Normal stomach - (0) Red colouration - (0.5) Spot ulcer - (1) Hemorrhagic streak - (1.5) Ulcers - (2). The lesion score for each rat was calculated as the number of lesions in that rat multiplied by their respective severity factor. Mean ulcer sore for each animal will be expressed as ulcer index. The percentage of ulcer protection was determined as follows:

$$\% \text{ Protective} = \text{Control mean ulcer index} - \text{Test mean ulcer index} / \text{Control mean ulcer index} \times 100$$ (Inas *et al*., 2011)

Ethanol-Induced Ulcer Model

Albino rats (25) were divided into five groups (n=5), each receiving a different treatment. The animals were fasted for 24 h before the experiment. Ulceration was induced in rats by administering 5 ml/kg absolute ethanol (Karaboğa *et al*., 2018). The animals were grouped and treated as in the indomethacin model. After seven days of treatment, the animals were sacrificed, and the ulcer index and percentage protective ratio were calculated as previously described.

Stress-Induced Ulcer Model

Twenty-five albino rats were divided into five groups (n=5), each receiving a different treatment. The animals were fasted for 24 h before the experiment, and ulcer induction was achieved using a swimming stress model (Mostofa *et al*., 2017). The animals were grouped and treated as in the indomethacin model. After seven days of treatment, the animals were sacrificed, and the ulcer index and percentage protective ratio were calculated as previously described.

Statistical Analysis

The results are expressed as mean ± SEM. Statistical analysis was performed using one-way Analysis of Variance (ANOVA), followed by Dunnett's multiple comparison test using GraphPad Prism version 8.4. Statistical significance less than 0.05 ($P < 0.05$) was considered statistically significant.

Results

Acute Toxicity

The acute toxicity assessment indicated no mortality up to 5000 mg/kg. Nonetheless, manifestations of mild acute intoxication, including itching and depression, were observed within 24 hours post-treatment.

Phytochemical Analyses

Preliminary phytochemical screening revealed the presence of flavonoids, phenolics, tannins, alkaloids, saponins, steroids, and terpenoids. Cyanogenic compounds (HCN**)** were absent (Table 1). The quantitative phytochemical evaluation indicated that Phenolics recorded the highest concentration (1412.92 mg/100 g), Alkaloids (369.31 mg/100 g), terpenoids (309.09 mg/100 g), Flavonoids were the next most abundant at (297.27 mg/100 g), saponins (14.78 mg/100 g), and steroids (8.72 mg/100 g) (Table 2).

Table 1: Qualitative Phytochemical Analysis

Parameters	Results
Flavonoids	+
Phenolics	+
Tannins	+
Alkaloids	+
Saponins	+
Steroids	+
Terpenoids	+
HCN	–

Note: HCN = Cyanogenic compounds; - = Absent, + = Present.

Table 2: Quantitative Phytochemical Analysis

Secondary Metabolite	Concentration (mg/100 g)
Flavonoids	297.27 ± 6.10
Phenolics	1412.92 ± 10.14
Tannins	182.33 ± 14.37
Alkaloids	369.31 ± 17.80
Saponins	14.78 ± 2.95
Terpenoids	309.09 ± 10.29
Steroids	8.72 ± 32
Cyanogenic Compounds	NIL

Effect of the Herbal Tea on Indomethacin-Induced Ulcer Model

Herbal tea (HT) significantly ($P < 0.05$) reduced ulcers, as evidenced by a lower ulcer index than that in the negative control group. The activity of HT was comparable to that of omeprazole. Curative ratios of 100%, 100%, 80.77%, and 84.62% were achieved at 100, 200 and 400 mg/kg of HT respectively (Table 3).

Effect of the Herbal Tea on Ethanol-Induced Ulcer Model

A significant reduction in the ulcer index (UI) occurred at all doses of HT ($P < 0.05$). with corresponding curative

ratios of 95.6%, 98.6%, and 100% for the 100, 200, and 400 mg/kg, respectively, (Table 4).

Effect of the Herbal Tea on Stress-Induced Ulcer Model

A significant ($P < 0.05$) dose-dependent reduction in the ulcer index (UI) was evident in this model with corresponding percent ulcer curative ratios of 92.06%, 94.44%, and 95.23% for the 100, 200, and 400 mg/kg doses, respectively, (Table 5).

Table 3: Effect of the herbal tea on indomethacin-induced ulcer in rats

Treatment	Dose (mg/kg)	Ulcer index	% Curative ratio
HT	100	0.00*	100
HT	200	0.00*	100
HT	400	3.00±0.89*	80.77
Omeprazole	20	2.40±0.36*	84.62
Distilled water	5 ml/kg	15.60±3.52	-

HT- Herbal tea * $P < 0.05$

Table 4: Effect of the herbal tea on ethanol-induced ulcer in rats

Treatment	Dose (mg/kg)	Ulcer index	% ulcer curative
HT	100	0.600 ± 0.600	95.6
HT	200	0.200 ± 0.200	98.6
HT	400	0.000 ± 0.000	100
Omeprazole	20	0.000 ± 0.000	100
Distilled water	5 ml/kg	13.800 ± 7.324	-

HT – Herbal tea * $P < 0.05$

Table 5: Effect of the herbal tea on stress induced ulcer model in rats

Treatment	Dose (mg/kg)	Ulcer index	% ulcer curative
HT	100	1.000 ± 0.548	92.060
HT	200	0.700 ± 0.583	94.444
HT	400	0.600 ± 0.600	95.233
Omeprazole	20	2.600 ± 1.400	79.370
Distilled water	5 ml/kg	12.600 ± 2.581	-

HT – Herbal tea * $P < 0.05$

Discussion

The current study demonstrated the robust gastroprotective effects of *Musa paradisiaca* (plantain/banana) herbal tea in three complementary preclinical ulcer models: indomethacin-, ethanol-, and stress-induced ulcers. The alignment of outcomes noted in these models, each depicting different mechanistic routes to ulcer formation,

reinforces the claim that the observed gastroprotection is not model-specific but reflects a genuine cytoprotective effect.

Nonsteroidal anti-inflammatory drugs (NSAIDs), such as indomethacin, induce gastric ulcers primarily by inhibiting cyclooxygenase (COX) enzymes, which are essential for prostaglandin synthesis that maintains gastric mucosal defense (Suleyman *et al.,* 2010). Prostaglandins support mucus and bicarbonate secretion, preserve mucosal blood flow, and promote epithelial repair (Wilson, 1991; Højgaard *et al*., 1996). When COX is inhibited, these protective mechanisms are compromised, increasing susceptibility to acid-related injuries and ulceration (Devaraj *et al*., 2007; Motawi *et al*., 2008; Arab *et al*., 2015; Fazalda *et al*., 2018). In the present study, *M. paradisiaca* herbal tea provided gastroprotection comparable to that of omeprazole. This effect is likely attributable to the antioxidant activity that limits lipid peroxidation, a key contributor to gastric mucosal injury, and to the preservation or downstream support of prostaglandin-mediated cytoprotection and improved microcirculation (Luiz-Ferreira *et al.*, 2010; Falowo *et al*., 2021). These mechanisms align with prior evidence that plant-derived polyphenols and related phytochemicals can confer gastroprotection against oxidative and inflammatory stress (Fagundes *et al.*, 2021).

Ethanol induces ulcers by rapidly disrupting the gastric mucosal barrier, triggering reactive oxygen species (ROS) production, lipid peroxidation, and necrotic injury (Czekaj *et al*., 2017; Amin and Aziz, 2022). The protective effects of the tea in this model are likely due to its phenolic and flavonoids contents, which are well-known for their ROS-scavenging properties which contributes to gastroprotection (Ahmed *et al*., 2020). Furthermore, the membrane-stabilizing effects of tea, along with its ability to preserve the mucus layer and promote epithelial restitution, are essential mechanisms for preventing ulcer formation (Zhang *et al*., 2020). The gastroprotection suggests that *M. paradisiaca* herbal tea may have therapeutic potential in addressing oxidative damage and necrotic injury in the stomach.

Stress is another well-documented cause of gastric ulcers, primarily through the activation of neurohormonal pathways, including the hypothalamic-pituitary-adrenal (HPA) axis and sympathetic nervous system, which ultimately reduce gastric perfusion and increase inflammatory mediators (Shahzad *et al*., 2024). In the present study, *M. paradisiaca* tea provided consistent protection, indicating its potential as a stress-relieving agent. These protective effects are likely mediated by the attenuation of pro-inflammatory cytokines, antioxidant effects, and improved mucosal perfusion (Saji *et al*., 2020). These findings suggest that tea may help preserve barrier function and prevent stress-induced gastric injury by modulating oxidative stress and inflammatory pathways(Ahmed *et al*., 2020).

Phytochemical analysis of *M. paradisiaca* herbal tea revealed a diverse array of bioactive compounds, including phenolics, flavonoids, alkaloids, saponins, terpenoids, and steroids, which contribute to its gastroprotective properties. These compounds may act synergistically to provide multipathway protection. The antioxidant and anti-inflammatory properties of phenolic and flavonoid compounds are well documented, whereas alkaloids and saponins are known for their antimicrobial and mucus-enhancing properties (Nguyen *et al*., 2020; Falowo *et al*., 2021). Terpenoids and steroids are also involved in healing and analgesic effects (Ibrahim *et al*., 2018). Together, these bioactive molecules may likely contribute to the protection of the gastric mucosa from various injuries, (Khémiri and Bitri, 2019). The identification and standardization of these bioactive compounds could substantially improve their reproducibility and facilitate dose translation for therapeutic applications.

Safety is a crucial aspect of herbal remedies, particularly for long-term therapeutic use. In this study, acute oral administration of *M. paradisiaca* tea up to 5,000 mg/kg produced no mortality, and only transient discomfort was observed, suggesting a wide safety margin (Bernstein *et al*., 2020). The absence of cyanogenic glycosides further supports the safety profile of tea for medicinal use (Kale *et al*., 2019; Josiah *et al*., 2024). However, while the acute safety profile appears favorable, further studies are needed to assess subacute/subchronic toxicity, genotoxicity, and potential herb–drug interactions to ensure the tea's safety for long-term use.

Owing to its antioxidant and barrier-preserving properties, *Musa paradisiaca* tea presents a promising complementary strategy, particularly in situations involving NSAID exposure and stress. It can also serve as a candidate for the development of standardized functional foods or phytopharmaceuticals that enhance gastrointestinal health (Loganayaki *et al*., 2010; Ajijolakewu *et al*., 2021).

Although the findings of this study are promising, several limitations should be addressed in future studies. The mechanisms underlying the gastroprotective effects of the herbal tea remain unclear, as direct molecular validation, such as quantification of prostaglandin E2 (PGE2) and oxidative stress biomarkers, has not yet been performed. Future studies should validate these mechanisms using molecular assays (e.g., cytokine panels and oxidative stress markers such as MDA, SOD, CAT, and GSH) to clarify the mode of action of tea (Koc *et al*., 2018; Kim *et al*., 2020; Sanpinit *et al*., 2021). Additionally, the effects *of M. paradisiaca* tea on ulcer healing kinetics and relapse prevention remain unclear and warrant further investigation using longitudinal ulcer models (Fahmi *et al*., 2019; Sanpinit *et al.*, 2021).

Furthermore, to enhance reproducibility and facilitate the translation of findings into clinical practice, future research should focus on standardizing the preparation of herbal tea

to specific bioactive markers, such as total phenolics or particular flavonoids (Yang and Liu, 2012). Future directions also include bioassay-guided fractionation and the use of LC–MS/MS metabolomics to identify and standardize lead bioactive compounds, followed by clinical trials to establish the efficacy of tea in human subjects (He *et al*., 2021; Sarikurkcu *et al*., 2020). Mechanistic assays, such as the measurement of oxidative stress markers, cytokines, and barrier markers, are crucial for understanding the therapeutic potential of *M. paradisiaca* banana heart tea (Falowo *et al*., 2021).

Conclusion

In conclusion, *Musa paradisiaca* herbal tea demonstrates effective gastroprotection likely through antioxidant and anti-inflammatory mechanisms. Its safety and potential as a complementary therapy for ulcers merit further clinical investigation.

Declaration of conflicting interest

The authors declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

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