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Gastric Acid Profile of Ethanol and Aqueous Unripe *Musa paradisiaca* Peels Extract and Fractions, Omeprazole and Cimetidine in Ethanol-induced Gastric Ulcer

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ABSTRACT

The parietal cells of the stomach produce gastric acid that is essential for the digestion of food to provide nutrient availability for organ and muscle use. Excessive gastric juice production can lead to lesions in the gastric mucosa, resulting in ulcers. The rate and concentration of acid secretion are influenced by factors such as food, medications, and chronic diseases. This research was carried out to assess the role gastric ulcer plays in acid production or secretion. The research further examined how *Musa paradisiaca* extracts and fractions influence gastric acid in ulcer conditions. Sixty rats were divided into twelve groups: Groups A and B served as the control groups administered 10 mL/kg of distilled water; Groups C – E were induced with ulcer and administered with 48.99, 97.98, and 146.97 mg/kg *Musa paradisiaca* ethanol extracts; Groups F – I were induced with ulcer and administered with 97.98 mg/kg *Musa paradisiaca* fractions; Group J received 0.29 mg/kg omeprazole; Group K was given 5.71 mg/kg cimetidine; and Group L received 97.98 mg/kg aqueous *Musa paradisiaca* extract. The administration was oral and lasted for 28 days. On the last day, the animals were sacrificed after being anaesthetized with ketamine hydrochloride intraperitoneally, the stomach excised, and contents of the stomach removed for titratable acidity analysis. The obtained data were statistically analysed. The results showed that the presence of gastric lesions, acid volume, concentration, secretion rate, and basal output were substantially reduced as the immune system regulated acid production to defend the mucosa and limit exposure to its harsh effects. In conclusion, the aqueous extract and fractions of *Musa paradisiaca* peels demonstrated more potency than omeprazole and cimetidine in regulating gastric acid production and secretion in gastric ulcer conditions and promoting healing.

Keywords: *Musa paradisiaca* peels, gastric ulcer, gastric acid secretion, stomach, cimetidine.

1. INTRODUCTION

Gastric acid, also known as hydrochloric acid or gastric juice is found in the stomach. This acid is responsible for the digestion of ingested food substances, killing microorganism to modulate gastrointestinal tract microbiome, facilitates absorption of essential nutrients such as iron, calcium, and vitamins. Gastric juice also aids protein digestion to enhance a healthy gastrointestinal system (Schubert, 2016). Hydrochloric acid is produced by the oxyntic cells, otherwise known as parietal cells, found in gastric glands at a pH level of 0.8 and concentration of 160 mM (Fujimori, 2020). The rate by which acid is secreted in the stomach is regulated by hormonal, neural, and paracrine mechanisms, which includes gastrin from G cells, histamine from enterochromaffin cells, and acetylcholine from fibres of vagal nerve. These agonists employ different receptor pathways to mobilise proton pumps H+/K+ ATPase located in the large canalicular membrane (Samuelson and Hinkel, 2003). Acid secretion is inhibited by somatostatin, which has a direct inhibitory effect on parietal cells to maintain acid level balance and prevent excessive secretion (Samuelson and Hinkel, 2003). Low levels of histamine from mast cells, gastrin, and acetylcholine release at different times results in weak stimulation of acid secretion. Whereas the concurrent release of histamine, gastrin, and acetylcholine intensely stimulates acid secretion. Report from Yak and Forte (2003) showed that certain pharmacological substances, antagonists, and medicinal herbs ingested into the body can alter gastric acid secretion.

The gastric mucosa is a smooth, reddish brown to pink colour layer that continues with the parietal peritoneum, having invaginations that forms the gastric pits (Pirie and McLaren, 2022). This gastric mucosa contains surface mucous cells and mucous neck cells with tight epithelial junctions, that secretes mucus and bicarbonates, forming a protective barrier to protect the stomach from the corrosive actions of gastric acid. Erosion of the gastric mucosa caused by increased acid secretion when the bacteria *Helicobacter pylori* enters the body from ingested contaminated food or other contributing factors such as tobacco smoking, Crohn's disease, non-steroidal anti-inflammatory disease, and Behcet's disease (Steinberg, 2002) can lead to hypochlorhydria, achlorhydria, Zollinger-Ellison syndrome and hyperacidity syndromes such as gastric ulcer, − Uncontrolled or fluctuated levels of acid secretion results in gastrointestinal infections. Reduced secretion of acid in the stomach called hypochlorhydria can lead to bacterial and yeast overgrowth.

Musa paradisiaca also known commonly as plantain is a monocotyledonous and herbaceous plant of the Musa family, widely known for its rich antioxidants, anti-inflammatory, anti-diabetic, and analgesic properties. Plantain has been adopted as a valuable therapeutic agent for the treatment of various chronic and acute diseases (Zafar, 2011) ranging from arthritis, bronchitis, asthma, cuts, and wounds. Enye (2013) discovered that unripe peels of *Musa paradisiaca* are rich in bioactive compounds. However, when it is administered alone in its aqueous extract form, or as a combined therapy with other natural remedies like cabbage, they can reduce excessive acid secretion and enhance the protective mechanism of gastric mucosal lining. The antioxidant properties of *Musa paradisiaca* contribute to its protective role against oxidative stress, as its phenolic compounds mitigate oxidative damage to promote a healthy gastric system. Mukhopadhyaya et al. (1987) observed that *Musa paradisiaca* enhances gastric mucosal resistance by increasing the incorporation of thymidine into gastric mucosal DNA, which helps maintain gastric volume and prevent mucosal damage that may otherwise lead to increase gastric secretions and altered gastric capacity. In addition, Mukhopadhyaya et al. (1987) further demonstrated that the anti-ulcerogenic properties of the peels such as flavonoids manages gastric volume potentially reducing inflammation and irritation in the gastric mucosa that arises due to ulceration. Having an accurate understanding of gastric acid profile, acid regulatory mechanisms, secretion patterns, and the pH of gastric acid can provide valuable insight into treating and managing gastrointestinal disorders, including peptic ulcers. This research, therefore assessed the mechanism of gastric acid production in altered physiologic states, under ulcer conditions, and examines the therapeutic significance of *Musa paradisiaca* in controlling gastric ulcers.

2. MATERIALS AND METHODS

Plant Materials and Drugs

Musa paradisiaca (plantain) peels were collected from a local plantation in Uyo, Akwa Ibom State, to ensure that the plantains were grown naturally without the use of chemical fertilizers. The plant was identified at the Pharmacology and Toxicology Department, Faculty of Pharmacy, University of Uyo, with herbarium number UUPH51(a). Omeprazole (Omefast-20 20 mg) and Cimetidine (Tagacure® 400 mg) drugs were obtained from Anointed Brand Pharmacy, Uyo, Nigeria.

Plant Preparation

Matured *Musa paradisiaca* peels were collected during the months of September to December, thoroughly washed under running tap water, rinsed in distilled water, sliced into thin pieces, air dried for two weeks to remove stickiness, ground into powder, sieved, and stored in airtight bottles for use. The dried thin peels were weighed with a triple-beam balance and found to be 1.106 kg. The powdered

samples were soaked in 60 % ethanol solvent, covered, and kept for 72 hours with intermittent shaking using the method of maceration extraction. The macerated samples were filtered, and the filtrate evaporated in a water bath at 40°-45° C to dry and arrive at the crude ethanol extract (Uzairu and Kano, 2021).

Experimental Design

Sixty Wistar rats weighing 150 – 240 g were obtained housed in the animal facility of Faculty of Pharmacy, University of Uyo, according to the standard laboratory conditions. The rats were fed with standard rat pellets, given free access to water *ad libitum*, and allowed to acclimatized for two weeks. The animals were divided into twelve groups with five rats in each group, as shown in Table 1. Group A was administered 10 mL/kg of distilled water to serve as standard control. Groups B – L were all induced with ulcer using 0.5 mL of 90 % ethanol. Group B was administered 10 ml/kg of distilled water serving as negative control. Groups C – E were administered 48.99, 97.98, and 146.97 mg/kg *Musa paradisiaca* ethanol extracts, respectively. Groups F – I were given 97.98 mg/kg of n-hexane, dichloromethane, ethyl acetate, and n-butanol fractions, respectively. Group J was administered 0.29 mg/kg Omeprazole and Group K received 5.71 mg/kg Cimetidine as positive control groups. Group L was administered 97.98 mg/kg aqueous *M. paradisiaca* extract. All the administrations were oral and lasted for twenty-eight days.

Table 1. Schedule of treatment of animals in control and test groups

Groups (n=12)	Treatment/ Dosage	Duration
A	10 ml/kg BW of distilled water for	28 days.
B	Ulcer-induced given 10 ml/kg BW of distilled water for	28 days.
C	Ulcer-induced given 48.99 mg/kg BW of *MP* crude extract for	28 days.
D	Ulcer-induced given 97.98 mg/kg BW of *MP* crude extract for	28 days.
E	Ulcer-induced given 146.97 mg/kg BW of *MP* crude extract for	28 days.
F	Ulcer-induced given 97.98 mg/kg BW of n-hexane fraction for	28 days.
G	Ulcer-induced given 97.98 mg/kg BW of DCM fraction for	28 days.
H	Ulcer-induced given 97.98 mg/kg BW of Ethyl acetate fraction for	28 days.
I	Ulcer-induced given 97.98 mg/kg BW of n-Butanol fraction for	28 days.
J	Ulcer-induced given 0.29 mg/kg BW of omeprazole for	28 days.
K	Ulcer-induced given 5.71 mg/kg BW of cimetidine for	28 days.
L	Ulcer-induced given 97.98 mg/kg BW of aqueous extract for	28 days.

BW- Body weight. N – Number of rats. DCM - Dichloromethane MP – *Musa paradisiaca*

Method of ulcer induction

The animals were fasted for 24 hours before the experiment but given free access to water four hours before the induction (Ibrahim *et al.*, 2016; Simona *et al.*, 2019). Gastric ulcer was induced using 0.5 ml of 90 % ethanol per body weight, administered orally. The animals were kept for four hours before the commencement of treatment with the extracts, fractions, and drugs. To ascertain that ulceration has been established, macroscopic examination was carried out on twelve animals as a preliminary pilot study with a hand lens, and the presence of ulcer lesion was scored according to the standard method described by Nwafor et al. (2000).

Termination of Experiment / Sample Collection

On the final day of the experiment, the final body weights were taken. The animals were anaesthetised with ketamine hydrochloride 50 mg/kg body weight (Rosenbaum et al. 2024). The stomach was opened along the greater curvature, washed with normal saline to collect gastric contents, which were filtered into plain bottles for titratable acidity studies.

Titratable Acidity

The content of gastric organs was collected into universal bottles by adding 2 mL of normal saline then filtered using a filter paper. The content was titrated by measuring 1 mL of the content from each group using a 5 mL syringe into a conical flask (Morah and Peter,

2022). One part of the content was diluted using 20 mL of water as a blank. While the others were diluted using 0.1 M sodium hydroxide (NaOH) (Abdel-Gaber et al., 2021) until a bright purple or violet colouration was observed. Two drops of phenolphthalein were used as an indicator, allowing for an observable distinct pink colouration (Pradeep and Dave, 2013).

Statistical Analysis

The data obtained were subjected to statistical analysis of variance (ANOVA) with Graphpad Prism Version 7 (Graphpad Software Inc., California, USA). Statistical significance is considered at $p<0.05$ and the values were expressed in Mean ± Standard Error of Mean.

3. RESULTS

The results showed substantial increase in body weight (Table 2) among rats in Groups C (48.99 mg/kg) of ethanol extract, D (97.98 mg/kg) of ethanol extract, and E (146.97 mg/kg) of ethanol extract. In contrast, all other treatment groups recorded a reduction in body weight when compared with Group A (distilled water). The results from titratable acidity analysis revealed considerable increase in acid concentration in Groups C (48.99 mg/kg ethanol extract), D (97.98 mg/kg ethanol extract), E (146.97 mg/kg ethanol extract), G (97.98 mg/kg dichloromethane fraction), H (97.98 mg/kg ethyl acetate fraction), I (97.98 mg/kg n-butanol fraction). The other groups showed relatively stable levels when compared with group A (distilled water), as presented in Figure 1. Gastric juice volume was substantially reduced across all groups when compared with Group A, except in group E (146.97 mg/kg ethanol extract) which maintained a similar level (Table 3). Basal acid output was significantly higher in Group G (97.98 mg/kg dichloromethane fraction) than in all other groups, as presented in Table 4. The rate of acid secretion was substantially lower across all treatment groups compared with Group A (distilled water). However, Group F (97.98 mg/kg n-hexane fraction) exhibited a comparatively higher secretion rate than other treatment groups, as expressed in Table 5.

Table 2. Effect of omeprazole, cimetidine, ethanol, aqueous extract, and fractions of *Musa paradisiaca* on the body weight of the animals.

Groups	Before Administration (g)	After Administration (g)	Weight difference (g)
A	188.0 ± 6.97	197.0 ± 15.85	9.0 ± 8.88
B	160.6 ± 6.53	236.2 ± 7.76	75.6 ± 1.23
C	177.0 ± 6.03	243.2 ± 5.21 *a	66.2 ± 0.82
D	173.4 ± 17.41	240.0 ± 8.19 *a	66.6 ± 9.22
E	189.8 ± 11.68	205.2 ± 12.26	15.4 ± 0.58
F	170.6 ± 12.01	163.4 ± 9.06 ****bcd	-7.2 ± 2.95
G	180.8 ± 8.45	172.4 ± 6.55 ***b****cd	-8.4 ± 1.90
H	176.4 ± 7.31	161.6 ± 6.47 ****bcd*e	-14.8 ± 0.84
I	180.4 ± 3.22	170.6 ± 6.54 ***b****cd	-9.8 ± 3.32
J	153.6 ± 1.89	147.8 ± 1.83 ****bcd**e	-5.8 ± 0.06
K	173.8 ± 10.53	162.8 ± 10.73 ****bcd*e	-11 ± 0.20
L	164.4 ± 5.66	154.4 ± 4.11 *a****bcd**e	-10 ± 1.55
	P= 0.2175	P<0.0001	
	F= 1.371	F= 16.57	

Table 3. Effect of omeprazole, cimetidine, ethanol, aqueous extract, and fractions of *Musa paradisiaca* on gastric juice volume.

Groups	Gastric Juice Volume (mL)
A	2.800 ± 0.200
B	1.600 ± 0.245
C	1.400 ± 0.245 *a
D	2.200 ± 0.200

E	2.800 ± 0.200 *c
F	2.600 ± 0.245
G	2.400 ± 0.400
H	1.200 ± 0.200 **ae*f
I	2.600 ± 0.245 *h
J	1.600 ± 0.245
K	2.200 ± 0.374
L	1.400 ± 0.245 *ae
	P<0.0001
	F= 5.144

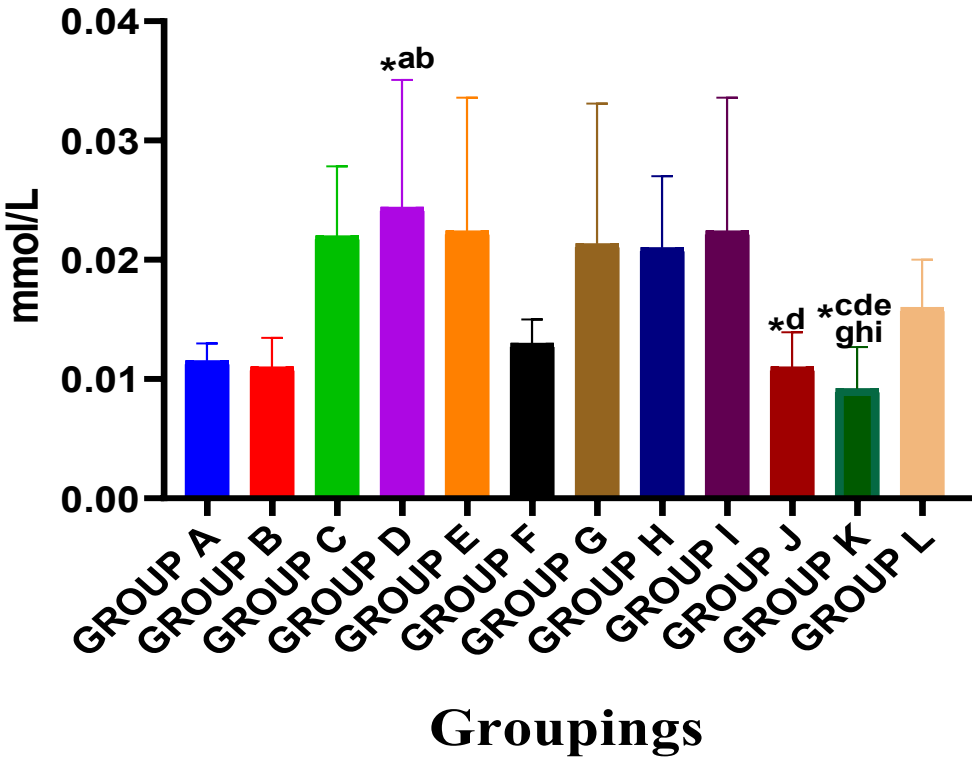

Figure 1. Effects of omeprazole, cimetidine, ethanol, aqueous extract, and fractions of *Musa paradisiaca* on acid concentration

Table 4: Effect of omeprazole, cimetidine, ethanol, aqueous extract, and fractions of *Musa paradisiaca* on basal acid output.

Groups	Basal Acid Output (mmol/h)
A	0.068± 0.033
B	0.016± 0.003*a
C	0.022± 0.006*a
D	0.062± 0.035
E	0.064± 0.034

F	0.032± 0.002*ade
G	0.090± 0.045
H	0.024± 0.006*ade**g
I	0.062± 0.034
J	0.016± 0.004*adei**g
K	0.016± 0.004*adei**g
L	0.020± 0.003*adei**g
	P= 0.3037
	F= 1.214

Table 5. Effect of omeprazole, cimetidine, ethanol, aqueous extract, and fractions of *Musa paradisiaca* on acid secretion rate.

Groups	Treatment	Acid Secretion Rate(mmol/h)
A	10 ml/kg distilled water	0.030 ± 0.003
B	Induced ulcer /10 ml/kg distilled water	0.016 ± 0.003*a
C	Induced ulcer / 48.99 mg/kg crude extract	0.028 ± 0.006
D	Induced ulcer / 97.98 mg/kg crude extract	0.026 ± 0.003
E	Induced ulcer / 146.97 mg/kg crude extract	0.028 ± 0.002
F	Induced ulcer / 97.98 mg/kg n-hexane fraction	0.032 ± 0.002
G	Induced ulcer / 97.98 mg/kg DCM fraction	0.020 ± 0.003
H	Induced ulcer / 97.98 mg/kg Ethyl acetate	0.024 ± 0.006
I	Induced ulcer / 97.98 mg/kg n-Butanol	0.026 ± 0.003
J	Induced ulcer / 0.29 mg/kg Omeprazole	0.016 ± 0.004*acef
K	Induced ulcer / 5.71 mg/kg of Cimetidine	0.016 ± 0.004*acef
L	Induced ulcer / 97.98 mg/kg aqueous fraction	0.020 ± 0.003
		P= 0.0145
		F= 2.493

4. DISCUSSION

The administration of ethanol can alter the rate of gastric acid secretions, inducing gastric lesions and wounds along the entire length of the gastric mucosa. This alteration affects pertinent structure of the body, such as the stomach, responsible for food digestion and nutrient absorption. In line with the results presented, it is said that the increase in gastric juice concentration in Groups C (48.99 mg/kg) of ethanol extract, D (97.98 mg/kg) of ethanol extract, and E (146.97 mg/kg) of ethanol extract increases body weight due to digestion which facilitates the breakdown of proteins, amino acids, calcium, and iron through its release of pepsin enzyme (Richardson et al., 1976) for nutrient absorption and bioavailability, and to protect the stomach mucosa against pathogens like *H. pylori* to reduce the rate of infection (Sascha and John, 2013). Thus, it is proven that an increase in acid concentration increases body weight as *Musa paradisiaca* extracts contain phytochemicals that can boost the digestion process, improve nutrient absorption, and stores glycogen as an energy source than omeprazole and cimetidine. For the fraction groups, it was observed that groups G given 97.98 mg/kg of dichloromethane fraction, H receiving 97.98 mg/kg of ethyl acetate fraction, and I given 97.98 mg/kg of n-butanol fraction showed increase in acid concentration compared to 97.98 mg/kg of n-hexane fraction respectively, but a decrease in body weight was observed across all the fraction groups. With these observations, it is interesting to conclude that, while the ethanol extracts of *Musa paradisiaca* promote weight gain, the fractions on the other hand, promote weight loss through lipolysis. To add to that, ulcer conditions are periods of excessive energy damage by the body to fight off the bacteria that has infiltrated the body, and to heal the wounds occurring in the gastric mucosa, caused by oxidative and metabolic stress. Thus, the meals taken in this period were utilised as energy source to suppress harmful lipid peroxidation, which damages the gastric membrane. In addition, the administration of *Musa paradisiaca* fractions

during this period, which contain flavonoids, tannins, alkaloids, and saponins reduced free radical-induced lipolysis of membrane phospholipids to maintain the integrity of the mucosal lining, prevent the excessive leakage of hydrogen ions, inhibit the release of enzymes that worsens ulcer, and stabilise the gastric mucosal barrier. These bioactive compounds also inhibited the release of histamine, gastrin, and cytokines for tissue preservation and healing to occur. *Musa paradisiaca* has been reported by Kumar et al. (2013) to reduce acid concentration in the stomach following gastric ulcer induction by influencing (decrease) the level of inflammatory cytokines such as tumour necrosis factor-α (TNF-α) and interleukins-1β (IL-1β) that had increased after ulcer induction to create a balance that makes the gastric environment conducive for healing. Groups J administered 0.29 mg/kg omeprazole and K receiving 5.71 mg/kg cimetidine showed no significant difference in acid concentration compared to control. Group L given 97.98 mg/kg of aqueous extract showed slight increase in acid concentration when compared to control group. A report highlighted that a reduction in acid concentration in groups treated with *Musa paradisiaca,* enhances gastric mucosal resistance by means of its anti-ulcerogenic properties, which modulates the inflammatory response, highlighting that these properties are a potential remedy for improving gastric ulcer (Mukhopadhyaya et al., 1987). Thus, it can be said that the n-hexane *Musa paradisiaca* fraction, omeprazole, and cimetidine possess therapeutic properties that are essential for ameliorating gastric disorders.

There was a considerable increase in the volume of gastric juice as observed in group E given 146.97 mg/kg of ethanol extract and group F given 97.98 mg/kg of n-hexane fraction, which showed that the presence of certain phytochemicals in the extract and fraction stimulate the parietal and gastric cells to produce gastric acid, and at the same time, enhance gastric mucosal resistance by incorporating thymidine into the mucosal cell DNA for cellular proliferation, to repair the gastric mucosal lining, and to strengthen mucosal integrity (Mukhopadhyaya *et al*., 1987). On the other hand, groups C given 48.99 mg/kg) of ethanol extract, D receiving 97.98 mg/kg of ethanol extract, G treated with 97.98 mg/kg of dichloromethane fraction, H given 97.98 mg/kg of ethyl acetate fraction, I administered 97.98 mg/kg of n-butanol fraction, J receiving 0.29 mg/kg of omeprazole, and L receiving 97.98 mg/kg of aqueous extract showed decrease in gastric juice volume, suggesting an inhibition of the parietal cells, a reduction in gastric acid production, and a protection of the gastric mucosal cells from hyperacidity. This decrease in volume of gastric juice indicates a shift in the balance of gastric secretions against gastric lesions and damage as the mucosal integrity has been altered. Mukhopadhyaya *et al*. (1987) also suggested that the decrease in volume of gastric juice can be linked to phytochemical properties in *Musa paradisiaca* that are anti-ulcerogenic, which helps in mitigating the actions caused by gastric ulcer, thus facilitating better nutrient absorption. Gastric function was regulated to restore balance in gastric acid secretion maintaining optimal homeostasis in group K given 5.71 mg/kg of cimetidine.

Group G given 97.98 mg/kg of dichloromethane fraction had significant increase in basal output than control group. The basal acid output was substantially lower in ulcer induced group, groups C given 48.99 mg/kg of ethanol extract, E receiving 97.98 mg/kg of n-hexane fraction, H given 97.98 mg/kg of ethyl acetate fraction, J treated with 0.29 mg/kg of omeprazole, K administered 5.71 mg/kg of cimetidine, and L receiving 97.98 mg/kg of aqueous extract. It is interesting to note that this substantial decrease demonstrated their ability to inhibit gastric cells from producing gastrin which decreases acid production and alleviates hyperacidity. Omeprazole does this by binding to the proton pump H^+/K^- ATPase enzyme located in the parietal or oxyntic cells, to block gastric acid production leading to a reduction in acid secretion and alleviate the symptoms of peptic ulcer to promote healing (Andersson *et al*., 1990). Cimetidine, on the other hand, alleviates hyperacidity by inhibiting histamine receptor found on parietal cells, which are responsible for stimulating acid secretion. As it blocks histamine receptors, the volume and concentration of gastric acid produced are reduced, and healing of ulcer lesions is promoted (Andersson *et al*., 1990). There were no contrasting differences between the control group and groups D given 97.98 mg/kg of ethanol extract, E receiving 146.97 mg/kg of ethanol extract, and I given 97.98 mg/kg of n-butanol fraction. It is worth noting that *Musa paradisiaca* extracts and its fractions have the potency to stimulate gastric cells to increase acid production, which aids digestion. Employing its flavonoids, which give gastroprotective effects, to protect the gastric mucosa. The administration of *Musa paradisiaca* extracts and fractions reactivate growth factors such as epidermal growth factor receptor (EGFR) and platelet endothelial cell adhesion molecule to promote ulcer healing and reduce the risk posed by high acid production (Alese *et al*., 2017).

The significant drastic decrease in control Group B (ulcer induced + distilled water) as compared to Group A (distilled water) indicates that the reduction in secretion of acid is due to the inflammation occurring in the gastric mucosa exposing the gastric lining to high acid secretions resulting in gastric wounds known as ulcers (John and Baron, 2001). This results in gastric atrophy, which is the reduction of the number of parietal cells responsible for acid secretion as a result of inflammation or infection caused by *H. pylori*, leading to a decrease in acid production and secretion to prevent further damage, as presence of ulcers disrupt the gastric protective mechanisms (John and Baron, 2001). It is therefore proven in this research that the decrease in acid secretion observed is the body's

defense mechanism response to inflammation to prevent further mucosal damage. The observable decrease in the rate of secretion across all groups, as explained by Sascha and John (2013) was linked to the administration of pharmaceutical drug or herbal remedies in pathological cases like gastric reflux disease and peptic ulcer decreases gastric acidity through the inhibition of gastric acid secretion and intraluminal neutralization of already secreted gastric acids. This action was achieved, by direct blocking of proton pump inhibitor H^+/K^+-ATPase effector or interfering with the neurohormonal signaling pathway leading to the secretion of gastric acid. This decrease also showed that *Musa paradisiaca* extract possesses gastroprotective properties that decrease acid production to protect the gastric mucosa from excessive acid damage and by doing so, heal and prevent chronic gastric ulcer. It also shows cytoprotective properties present in the extract to protect the gastric mucosal cells and glands from further damage.

5. CONCLUSION

In this study, gastric acid profile indicated that *Musa paradisiaca* peel extracts helped regulate acid concentration and basal acid output in ulcer-induced models. The results from titratable assay demonstrated in the treatment groups, a reduction in the rate of gastric acid secretion, an increase in the concentration of acid which enables digestion to occur effectively, while reducing the rate at which the gastric mucosa is exposed to gastric acid to reduce inflammation. While ethanol extracts promote weight gain through decrease in gastric acid volume but increase in concentration, enhancing digestion, glycogen deposits, and nutrient absorption; the fractions and aqueous extract enhance lipolysis through decrease acid secretion and increase in acid concentration by boosting the body's metabolic process and inhibiting ulcer causing enzymes and lipid peroxidation, to promote weight loss. In addition, *Musa paradisiaca* extracts, particularly the n-hexane, dichloromethane, and aqueous fractions offer promising alternatives or adjuncts to conventional antiulcer therapies. Their ability to modulate gastric acid secretion and enhance mucosal healing, underscores their potential in the clinical management of gastric ulcers.

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Authors' Contributions

The manuscript, design of the study, discussion and research was written by Mfonobong E Sampson, the data analysis and interpretation of result was curated by Kingsley A Okon. Eno-Obong I Bassey proofread, assisted in methodology development, supervised the research, reviewed, and edited the manuscript.

Ethical Approval

The study was approved by the Faculty of Basic Medical Sciences Research and Ethical Committee (UU_ FBMSREC_2024_005), regarding animal regulations followed in the Faculty of Basic Medical Sciences Animal House, University of Uyo, Nigeria. The Animal ethical guidelines are followed in the study for observation, identification & experimentation.

Informed Consent

Not applicable.

Conflicts of interests

The authors declare that there are no conflicts of interests.

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Data and materials availability

All data associated with this study are present in the paper.

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