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Antiulcer Activity of Aqueous Peel Extract of Musa Sapientum in Albino Rats

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Abstract- ***The use of conventional antiulcer drugs is limited by their side effects and by the risk of relapse after treatment is stopped. This study investigated the antiulcer activity of the aqueous peel extract of Musa sapientum (banana) against ethanol induced gastric ulcers in albino rats. Ripe banana peels were dried, powdered, and extracted in distilled water; twenty-five Wistar albino rats of both sexes were assigned to five groups (n = 5) – normal control, ulcer (positive) control, standard control (ranitidine, 100 mg/kg), extract treated (100 mg/kg), and extract plus ethanol and treated for 14 days. Ulcer index, percentage protection, gastric free and total acidity, and pH of gastric juice were determined, and stomach tissue was examined histologically. Phytochemical screening showed that the peel extract contains phenols, flavonoids, and other secondary metabolites. The extract treated and extract plus ethanol groups showed marked reductions in ulcer index relative to the ulcer control, corresponding to 70.3% and 77.7% protection against ethanol induced ulceration respectively, and produced significant reductions ($p < 0.05$) in total gastric acidity, comparable to the standard drug ranitidine. Gastric juice pH was significantly higher (2.90 ± 0.21 and 3.04 ± 0.25) in the extract treated groups than in the ulcer control (1.83 ± 0.56). Histological examination confirmed reduced mucosal injury in the extract treated tissues relative to the ulcer control. These findings indicate that the aqueous peel extract of Musa sapientum protects the gastric mucosa against ethanol induced ulceration, most likely by strengthening mucosal defence and reducing gastric acidity, and support its potential as a low-cost, plant derived antiulcer agent.***

Keywords: Antiulcer activity; Musa sapientum; aqueous peel extract; ethanol induced ulcer; albino rats; gastroprotection.

I. INTRODUCTION

Descriptions of ulcer disease date back to antiquity. Hippocrates (c. 460–370 BC), regarded as the “Father of Medicine,” described ulceration while distinguishing carcinos and carcinoma as non-ulcer forming and ulcer forming tumours, and later Hippocratic writers described gastric ulcer and its management.[1] The physician Avicenna (980–1037 AD) studied the relationship between gastric pain and mealtimes, and the first clinical description of a gastric ulcer is attributed to the Italian physician Marcello Donati, who reported a case in 1586. Following the discovery of Helicobacter pylori in 1983, it became evident that most peptic ulcers previously attributed solely to acid hypersecretion were in fact associated with this organism; more than half of the world's population is thought to carry H. pylori, although only 5–10% of carriers go on to develop ulcer disease.[31]

An ulcer is a breach in the epithelial lining of an organ that disrupts the underlying tissue architecture and normal physiology.[46] Peptic ulcer disease (PUD) refers to ulceration of the gastric or duodenal mucosa associated with acid and pepsin activity, and affects an estimated 10% of the world's population.[13] Peptic ulceration occurs when the protective mucus layer of the stomach and duodenum is overwhelmed by corrosive digestive acid, disrupting the normal balance between aggressive (acid/pepsin) and protective (mucus/bicarbonate) factors.[10] The principal symptoms include abdominal pain, nausea, vomiting, loss of appetite and weight loss, with gastrointestinal bleeding in severe cases.[4, 41]

Current pharmacological management of peptic ulcer disease aims to reduce gastric acid secretion, neutralise existing acid, and protect the injured mucosa while it heals. However, the commonly used antacids, H2receptor antagonists and proton pump inhibitors are associated with adverse effects, tolerance, and a high incidence of relapse on

withdrawal, in addition to being costly.[10, 33] This has motivated interest in plant derived alternatives that are affordable, widely accessible and better tolerated, and that act mainly by strengthening mucosal defensive factors such as mucin and bicarbonate secretion and mucosal blood flow.[22]

Plants remain an important source of new therapeutic agents, and a large proportion of drugs in traditional and modern medicine are of plant origin.[58] Musa sapientum (banana), known as Ayaba in Hausa and Ogede in Yoruba, is a large perennial monocotyledonous herb reaching 2–8 m in height, propagated from subterranean corms.[30] Various parts of the plant including the unripe fruit, powder and peel have previously been reported to possess antiulcer activity, attributed largely to their mucosal protective rather than acid suppressing action.[9, 20, 45]

Peptic ulcer disease remains one of the most prevalent gastrointestinal disorders worldwide, with prevalence estimated at 30–40% in the United States, 80–90% in South America, and 70–90% in parts of Africa.[17, 47, 52, 61] Its prevalence rises with age, from about 20% among teenagers to 50–60% among individuals in their sixth and seventh decades of life, and is more common in developing countries. Existing therapeutic agents mainly create a physical barrier against acid and pepsin or enhance mucosal defence, but are commonly associated with adverse effects such as constipation, diarrhoea, urinary retention and dizziness, and may alter biochemical processes on chronic use.[7] There is therefore a continuing need for antiulcer agents that are effective, affordable and better tolerated. Because diverse plant derived chemical constituents have demonstrated antiulcer activity, the development of antiulcer agents from medicinal plants remains an attractive strategy. An indigenous, low-cost agent such as Musa sapientum peel extract could offer a safer and more accessible approach to the management of peptic ulcer disease, particularly in resource limited settings. The aim of this study was to determine the antiulcer activity of the aqueous peel extract of Musa sapientum in albino rats. The specific objectives were to:

i. Determine the percentage protection against ethanol induced ulcer in albino rats treated with the aqueous peel extract.
ii. Evaluate the antiulcer activity of Musa sapientum peel extract in an ethanol induced ulcer model in albino rats.
iii. Determine the effect of the aqueous peel extract on gastric secretion parameters (free acidity, total acidity and pH).

II. MATERIALS AND METHODS

Equipment

Equipment	Specification/Model	Manufacturer
Centrifuge	SP300	Optima, Germany
Weighing balance	800D	Shanghai Med. Instr. Ltd, China
Glassware	Pyrex	USA
pH meter	Electric B.	Brom Sci., England
Micropipette	Diapette	TECO Diagnostic, USA
Retort stand	—	Pharyx, England
Hot air oven	Electronic B.	Brom Sci., England
Syringes (5 ml)	—	Shandong Qiaopai Group Co. Ltd
Mortar and pestle	—	Pharyx, England
Hand gloves	—	TG Medical Sdn Bhd, Malaysia

Chemicals and Reagents

All chemicals and reagents used were of analytical grade, including ethanol, chloroform, sodium hydroxide, methyl orange, phenolphthalein and distilled water.

Experimental Animals

A total of twenty-five (25) healthy Wistar albino rats of both sexes, weighing 150–200 g, were obtained from the animal house of the Department of Biochemistry, Umaru Musa Yar'adua University, Katsina. The animals were housed in standard cages and acclimatised for two weeks prior to the experiment, with free access to standard growers' feed and water. All procedures were carried out in accordance with WHO guidelines for the use of experimental animals.[66]

Plant Material Collection

Healthy ripe banana fruits were obtained from the Central Market, Katsina, Nigeria. The plant was identified and authenticated by a taxonomist in the Botany Unit, Department of Biological Sciences, Umaru Musa Yar'adua University, Katsina.

Preparation of the Aqueous Peel Extract

Ripe banana peels were dried under shade and ground into powder using a mortar and pestle. Fifty grams (50 g) of the powdered peel was macerated in 500 ml of distilled water for 48 hours with intermittent shaking, and the mixture was then filtered through Whatman filter paper. The filtrate was evaporated to dryness in a hot air oven at 45°C, and the resulting extract was stored for subsequent use.

Experimental Design and Grouping

The twenty-five rats were randomly assigned to five groups of five animals each, as summarised in Table 1. Except for the normal control group, all animals received 70% ethanol orally to induce gastric ulceration. The aqueous peel extract (100 mg/kg) and the reference drug ranitidine (suspended in 1% methanol) were administered orally once daily for 14 days, as indicated below.

Group	Treatment	n
I – Normal control (NC)	Standard feed and water only; no treatment	5
II – Ulcer/positive control (PC)	70% ethanol (ulcer induction) + distilled water for 14 days	5
III – Standard control (SC)	70% ethanol (ulcer induction) + ranitidine (100 mg/kg) for 14 days	5
IV – Extract treated (BE)	70% ethanol (ulcer induction) + aqueous peel extract (100 mg/kg) for 14 days	5
V – Extract + ethanol (E+E)	Aqueous peel extract (100 mg/kg) before ulcer induction, then continued for 14 days after induction	5

After 14 days of treatment, animals were fasted for 24 hours and then anaesthetised in a chamber saturated with chloroform vapour. The stomach was excised, opened along the greater curvature, and the gastric juice collected into a labelled test tube. The gastric tissue was then rinsed gently with distilled water and examined for haemorrhagic lesions and ulceration prior to pH measurement, acidity determination and histological processing.

Measurement of Ulcer Score

Ulcer severity was scored according to the method of Almasaudi et al. (2016), as follows: normal coloration = 0; red coloration = 0.5; spot ulcers = 1.0; haemorrhagic streaks = 1.5; deep ulcers = 2.0; and perforation = 3.0.[6]

Measurement of Ulcer Index

The ulcer index (UI) was determined for each stomach as previously described.[62, 64] The mean length (mm) of all lesions was recorded, and the ulcer index calculated as:

$$UI = (UN + US + UP) \times 10^{-1}$$

where UN = mean number of ulcers per animal, US = mean severity score, and UP = percentage of animals with ulcers.

Measurement of Percentage Protection

Percentage protection was calculated using the method of Mahmood et al. (2011):[39]

$$\% \text{ Protection} = [(\text{Ulcer index of control} - \text{Ulcer index of test}) / \text{Ulcer index of control}] \times 100$$

Determination of Free and Total Acidity

Free and total acidity of the gastric juice were determined by the method of Ganguly (1969).[15] One millilitre of gastric juice was placed in a 100 ml conical flask, and 2–3 drops of methyl orange indicator were added; the mixture was titrated with 0.01 N NaOH until the colour changed from red to yellowish orange (free acidity endpoint), and the volume of alkali used was recorded. Titration was then continued after adding 2–3 drops of phenolphthalein until a persistent red pink colour reappeared (total acidity endpoint). Acidity was calculated as:

Acidity = (Volume of NaOH × Normality of NaOH × 100) / 100

Determination of pH

The pH of the gastric juice from each rat was measured using a calibrated pH meter.

Histopathological Evaluation

Excised stomachs were fixed in 10% formalin, processed, and embedded in paraffin wax. Sections were stained with haematoxylin and eosin and examined under a light microscope for histopathological changes, including congestion, haemorrhage, inflammation, cellular infiltration and ulceration.

Statistical Analysis

Data are expressed as mean ± standard error of the mean (SEM). Differences between groups were analysed by one-way analysis of variance (ANOVA); $p < 0.05$ was considered statistically significant.

III. RESULTS

Administration of 70% ethanol to the positive control group produced extensive haemorrhagic and necrotic lesions of the gastric mucosa. Oral administration of the aqueous peel extract of Musa sapientum (100 mg/kg) for 14 days markedly reduced ulcer formation in both extracts treated groups relative to the ulcer control (Table 1). The extract treated (BE) and extract plus ethanol (E+E) groups showed 70.3% and 77.7% protection against ethanol induced ulceration respectively, compared with 40.7% protection in the ranitidine treated standard control group.

Table 2 shows that the extract treated groups had significantly lower total gastric acidity ($p < 0.05$) and higher gastric juice pH than the ulcer control, with values comparable to those of the ranitidine treated group.

Table 1: Ulcer Index and Percentage Protection

Group	Dose (mg/kg)	Ulcer index	% Protection
NC	—	—	—
PC	—	5.40 ± 0.54	—
SC	100	3.10 ± 0.44*	40.7
BE	100	1.60 ± 0.82	70.3
E+E	100	1.20 ± 0.43**	77.7

Values are expressed as mean ± SEM (n = 5). *$p < 0.05$, **$p < 0.05$ vs positive control (one-way ANOVA). NC – normal control; PC – positive (ulcer) control; SC – standard control (ranitidine); BE – banana extract; E+E – extract plus ethanol.

Table 2: Effect of Musa sapientum Aqueous Peel Extract on Gastric Secretion

Group	Dose (mg/kg)	Free acidity	Total acidity	pH
NC	—	14.12 ± 10.86	39.23 ± 25.45	3.28 ± 0.64
PC	—	25.15 ± 12.04	71.73 ± 24.97	1.83 ± 0.56
SC	100	22.12 ± 11.06	62.20 ± 20.43*	2.86 ± 0.34*
BE	100	17.67 ± 6.44	46.37 ± 24.45	2.90 ± 0.21

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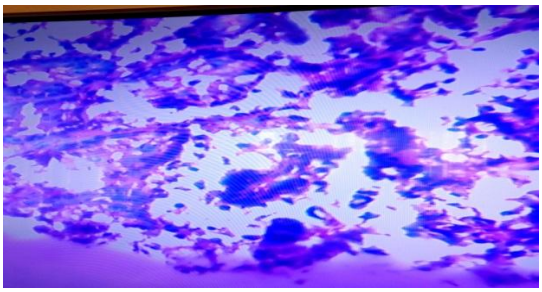
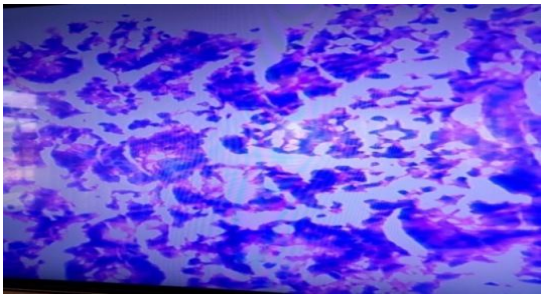
E+E	100	16.92 ± 8.71	43.45 ± 23.71**	3.04 ± 0.25

Values are expressed as mean ± SEM (n = 5). *p < 0.05, **p < 0.05 vs positive control (one-way ANOVA). NC – normal control; PC – positive (ulcer) control; SC – standard control (ranitidine); BE – banana extract; E+E – extract plus ethanol.

Histological examination (Plate 1) showed unremarkable gastric mucosa with intact lamina propria in the normal control group. The positive (ulcer) control, standard control and extract treated groups showed areas of fibrosis with necroinflammation and lymphoplasmacytic infiltration, while the extract plus ethanol group showed comparatively less severe histological changes.



(A)



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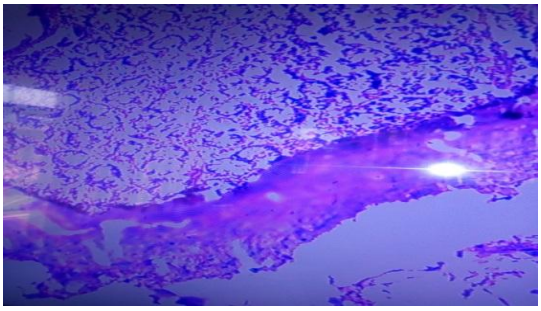

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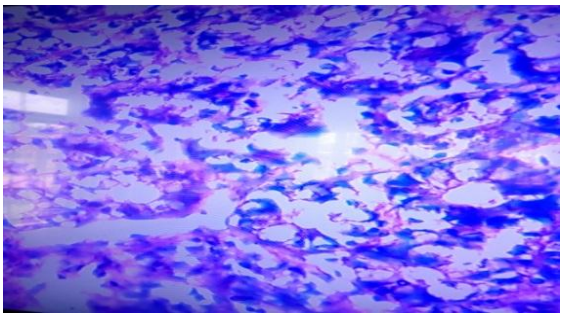

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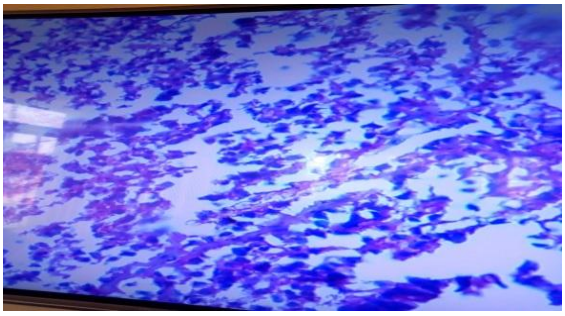

(E)

Plate 1: Histological appearance of gastric tissue in control and treated groups. (A) Normal control; (B) Ulcer (positive) control; (C) Standard control (ranitidine); (D) Extract plus ethanol group; (E) Extract administered group. Orange arrows indicate fibrosis; green arrows indicate inflammation.

IV. DISCUSSION

Oral administration of the aqueous peel extract of Musa sapientum (100 mg/kg) reduced ulcerogenic changes in the ethanol induced ulcer model over the 14day treatment period, with the most pronounced protection observed in the extract plus ethanol group. This protective effect is consistent with the mucosal protective, rather than purely acid suppressing, mechanism previously reported for banana derived preparations, possibly involving stimulation of prostaglandin biosynthesis.[45] Ethanol induced gastric ulceration is a well-established model for studying the pathogenesis of gastric mucosal injury, in which increased gastric HCl secretion damages the mucosal barrier and permits autodigestion of the underlying tissue.[40] Gastric mucosal and submucosal microcirculatory disturbances have also

been implicated as a major contributor to ethanol induced injury.[36] Most conventional antiulcer drugs – antacids, H2receptor antagonists such as ranitidine and famotidine, anticholinergics, and proton pump inhibitors – act principally by reducing acid secretion.[54]

Plant extracts, by contrast, are thought to exert antiulcer activity mainly by decreasing gastric acid secretion or increasing mucus production, thereby enhancing mucosal defence.[12] The antiulcer effect of banana has previously been attributed not to its serotonin (5HT) content but to its action on mucosal defensive factors: dried banana powder increased the total carbohydrate to protein ratio (TC:PR) of gastric mucus, an index of mucus secretion, and reversed the decrease in this ratio induced by ulcerogenic agents such as aspirin, indomethacin, phenylbutazone and prednisolone.[19, 45]

The pharmacological activity of medicinal plants generally reflects their phytochemical composition, and the gastroprotective action of Musa sapientum peel extract observed in this study is likely attributable to its content of alkaloids, flavonoids, saponins and tannins. This is consistent with reports that flavonoid rich plant extracts possess marked antiulcer activity,[44] and that saponins of the glycyrrhizin/carbenoxolone type protect the gastric mucosa by promoting mucus formation.[23] Ethanol induced ulceration is predominantly localised to the glandular region of the stomach, and has been linked to increased production of leukotrienes, mast cell secretory products and reactive oxygen species.[43] Prevention of ethanol induced depletion of gastric mucus, as observed with the peel extract in this study, is consistent with a protective mechanism involving increased prostaglandin[53] and sulfhydryl compound[60] activity in the gastric mucosa.

V. CONCLUSION AND RECOMMENDATIONS

Conclusion

The aqueous peel extract of Musa sapientum demonstrated antiulcerogenic activity against ethanol induced gastric ulceration in albino rats, evidenced by a reduced ulcer index, significant percentage protection, reduced gastric acidity, elevated gastric pH, and improved mucosal histology relative to the untreated ulcer control. These findings support the traditional use of Musa sapientum in the management of mucosal injury and suggest that its peel possesses anti-inflammatory and acid reducing properties comparable, in this model, to the standard antiulcer drug ranitidine.

Recommendations

1. Further studies should investigate the effect of Musa sapientum peel extract on cyclooxygenase1 (COX1) expression and prostaglandin synthesis, given the role of these pathways in mucosal blood flow, platelet aggregation and ulcer pathogenesis.
2. Longer-term studies are recommended to determine the effect of extended treatment duration on ulcer healing.
3. Further screening of other locally available plants for antiulcer activity is recommended, to expand the range of affordable, plant derived options for ulcer management.

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CONFLICT OF INTEREST

The author declares no conflict of interest.

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