



Original Article

Gastroprotective Effect of Aqueous Extract of Unripe *Musa Paradisiaca* (Banana) Fruit in Rats with Experimentally Induced Gastric Lesions

Zabir SM¹, Hasan MJ², Quadir R³, Haque S⁴, Sabiha K⁵, Ahasan MF⁶, Nila SH⁷, Chowdhury ASMS⁸

Abstract

Background: Unripe banana fruit extract is used to relieve stomach distress in some countries. A few studies in India, China, Sudan, Nigeria and South Africa revealed protection of gastric mucosa after exposed to aggressive agent following administration of unripe banana fruit extract in experimental animals. **Objective:** To determine the protective effect of aqueous extract of unripe *Musa paradisiaca* (banana) fruit against ethanol induced gastric lesion in rats. **Materials and Methods:** This was a prospective experimental study carried out in the department of Pharmacology, Dhaka Medical College, Dhaka within the period from January 2015 to June 2015. Aqueous extract of unripe banana fruit was prepared accordingly. Total 24 rats were randomly divided into 4 groups of 6 in each group. Group-A served as a control group and provided with distilled water (5 ml/kg/body weight) orally by gastric tube. Aqueous extract of *Musa paradisiaca* was administered orally as Group-B: 0.2 mg/kg/body weight/day, Group-C: 0.4 mg/kg/body weight/day and Group-D: 0.8 mg/kg/body weight/day for 7 days. After 7 days, 1 ml absolute ethanol (a known gastric lesion inducing agent) was orally administered to all groups by gastric tube. After 30 minutes of ethanol administration, all rats were sacrificed and dissected. After separating and opening stomachs, observed lesions were examined and measured with some morphological & histological parameters. Obtained data were subjected to analysis by Student's unpaired t-test. p -value < 0.05 was considered as statistically significant. **Results:** At the end of experiment, Group-A (control) showed a total of 33 gastric lesions. Group-B, Group-C and Group-D showed 22, 20 and 19 stomach lesions respectively. The difference between control group and experiment groups was statistically significant ($p < 0.01$). **Conclusion:** The unripe banana fruit extract has dose-dependent gastroprotective effect on rats. So, it may be effective in treating peptic ulcer disease.

Key words: Aqueous Extract, Gastroprotective, Gastric Lesion, *Musa paradisiaca*.

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Introduction

Peptic ulcer disease (PUD) affects four million people worldwide annually¹ and has an estimated lifetime prevalence of 5-10% in the general population². Although the global prevalence of PUD has dramatically decreased in the past decades³, the incidence of its complications has remained constant⁴. Higher peptic ulcer disease incidence has been found to be associated with male sex, smoking and chronic medical conditions^{5,6}. Peptic ulcer disease has also been found to be associated with increasing age⁷.

PUD mostly occurs in the duodenum and the stomach⁸. A peptic ulcer is the result of an imbalance

between the aggressive and defensive factors. On one hand, too much gastric acid and pepsin can damage the gastro-duodenal mucosa and cause ulcers; on the other hand, diminished mucosal protective factors may also predispose the cause ulcer. As many as 70-90% of such ulcers are associated with the *Helicobacter pylori*, a spiral-shaped bacterium that lives in the acidic environment of the stomach⁹. However, as the prevalence of *H. pylori* infection has declined in Western countries, Gastric Ulcer has become more commonly associated with the use of the nonsteroidal anti-inflammatory drugs (NSAIDs) and the acetylsalicylic acid (ASA)¹⁰⁻¹².

¹Sharif Mohammad Zabir, Assistant Professor, Dept. of Pharmacology, Kushtia Medical College, Kushtia, Bangladesh.

²Md. Jiaul Hasan, Associate Professor, Dept. of Pharmacology, M Abdur Rahim Medical College, Dinajpur, Bangladesh.

³Rukhsana Quadir, Assistant Professor, Dept. of Pharmacology, Dhaka Dental College, Dhaka, Bangladesh.

⁴Sumona Haque, Assistant Professor, Dept. of Pharmacology, Dhaka Dental College, Dhaka, Bangladesh.

⁵Kazi Sabiha, Assistant Professor, Dept. of Pharmacology, United Medical College, Dhaka, Bangladesh.

⁶Md. Faizul Ahasan, Assistant Professor, Dept. of Pharmacology, Ibrahim Medical College, Dhaka, Bangladesh.

⁷Sabrina Huda Nila, Assistant Professor, Dept. of Transfusion Medicine, Ad-din Women's Medical College, Dhaka, Bangladesh.

⁸Abu Saleh Md. Salauddin Chowdhury, Ex-Assistant Professor, Dept. of Pharmacology, Holy Family Red Crescent Medical College, Dhaka, Bangladesh.

Address of Correspondence: Dr. Sharif Mohammad Zabir, Assistant Professor, Department of Pharmacology, Kushtia Medical College, Kushtia, Bangladesh. Mobile: 01911406255. Email: smzabir@gmail.com

Helicobacter pylori predisposes to ulceration, mainly by gastric acid hypersecretion¹³. NSAIDs lead to peptic ulcer disease predominantly by compromising mucosal defenses. Other causes of peptic ulcer are smoking, steroids, alcohol consumption, psychological stress etc. Abdominal pain, classically epigastric with severity relating to meal is the main symptom of PUD^{14,15}. If untreated, Haematemesis (vomiting of blood), Melaena (passage of tarry, foul-smelling stool) and rarely, gastric, or duodenal perforation. The latter is extremely painful and requires immediate surgery¹⁶.

The current treatment of peptic ulcer is complicated and of high cost requiring minimum of two antibiotics in combination with a proton pump inhibitor, which often causes nausea, antibiotic resistance and other side effects^{14,15}. So, there is a need to search for cheap alternatives having antiulcer properties with less side effects. This is the basis of study for the development of new anti-ulcer agents. Some plant derived medicines have been significantly reported to possess potent antiulcer activity¹⁷.

Musa Paradisiaca is the most familiar of tropical fruits. From its origin in India/Malaysia, it spreads to the tropical world. *Musa Paradisiaca* is a monoecious herb. It grows 10-40 feet in height and has enormous broad green leaves which grow through hollow stem bearing flower and fruit. It occurs in all tropical areas native to Bangladesh, India and Myanmar. It is also distributed in New Guinea, America, Australia and tropical Africa¹⁸.

Banana fruits consist of carbohydrates, amino acids and other nutrients. The skin of the fruit is rich in cellulose (10%) and hemicellulose. The pulp protein is rich in arginine, aspartic acid, glutamic acid, methionine and tryptophan. The phytochemicals present in it may be associated with the wound healing (anti-ulcer) and mucosal healing properties¹⁹. The unripe fruit of *Musa paradisiaca* is cheap and easily available in our country. So, unripe *Musa paradisiaca* (Banana) fruit extract was chosen for the study. The aim of this study was to determine the protective effect of aqueous extract of unripe *Musa Paradisiaca* fruit on ethanol induced gastric lesion in rats.

Materials and Methods

The study was prospective experimental study carried out in the Department of Pharmacology, Dhaka Medical College, Dhaka from January 2015 to June 2015 on total 24 rats with unripe banana fruit extract with proper ethical approval from IERB. The collected unripe banana fruits were taxonomically identified and authenticated by Bangladesh National Herbarium, Mirpur, Dhaka (DACB Accession number - 41149)). Aqueous extract of unripe banana

fruits was prepared accordingly. A total of 24 rats were collected from ICDDR, Dhaka. They were of either sex, weighing about 150-200 gm. Rats were randomly divided into 4 groups of 6 in each group. Group-A served as control group that received distilled water 5 ml/kg/bodyweight orally daily for 7 days. Group-B, Group-C and Group-D received the extract at the doses of 0.2 ml/kg/bodyweight, 0.4 ml/kg/bodyweight and 0.8 ml/kg/bodyweight respectively orally daily for 7 days. At the end of 7 days, 1 ml absolute ethanol (a known gastric lesion inducing agent) was orally administered to all groups by gastric tube. After 30 minutes of ethanol administration, all rats were sacrificed and dissected. After separating & opening stomachs, observed lesions were examined and measured with the following morphological & histological parameters.

Morphological parameters:

1. Mean lesion number per rat in each group.
2. Mean lesion length and breadth in mm for each group.
3. Mean lesion area (length x breadth) in square mm for each group.
4. Mean lesion index (sum of length of all lesions in each stomach) in mm for each group.
5. Percentage inhibition of lesion by aqueous extract of *Musa paradisiaca*.

Histological parameters:

The degree of gastric damage was determined histologically by microscopy of gastric lesions. Gastric damage was graded histologically as 0° damage (Normal stomach), 1° damage, 1.5° damage, 2° damage and 3° damage.

Mean and standard deviation was calculated from the obtained Data. Student's Unpaired 't' test was performed to compare between Group A & Group B, between Group A & Group C and between Group A & Group D. p-value <0.05 was considered statistically significant. Then data were presented in the forms of tables and figures.

Results

This experimental study comprises 24 rats which were randomly divided into 4 groups (group A, B, C and D) of 6 in each group. At the end of the experiment, Group-A (control) showed total 33 stomach lesions. Group-B, Group-C and Group-D showed a total of 22, 20 and 19 stomach lesions respectively (figure-1). Table-I describes the mean lesion number, mean lesion length, mean lesion breadth, mean lesion area, mean lesion index and gastric damage was maximum in group A and tends to decrease in group B, C and D. Percentage inhibition of lesion was maximum in group D and minimum in group B and more in group D than group B and group C.

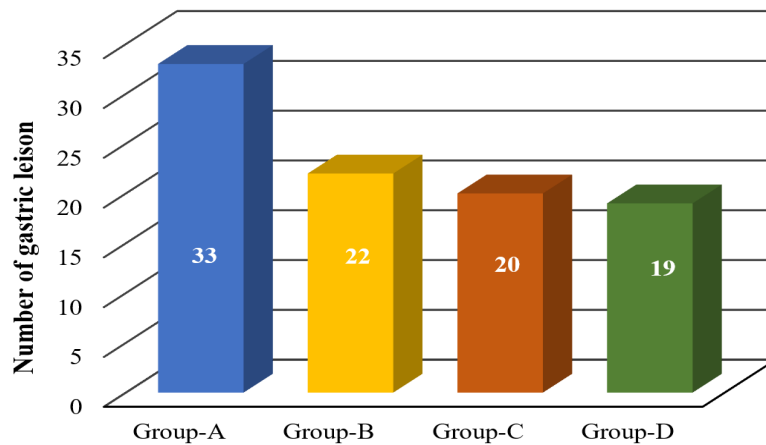


Figure-1: Bar diagrams showing the number of observed stomach lesions in four groups following administration of aqueous extract of unripe Musa Paradisiaca fruit

Table-I: Characteristics of gastric lesion among group A, B, C and D (n=24)

Variables	Group-A	Group-B	Group-C	Group-D
Mean lesions number	5.5 ± 1.04	3.66 ± 0.81	3.33 ± 1.21	3.16 ± 0.75
Mean lesion length	8.05 ± 2.54	2.93 ± 0.69	2.55 ± 0.77	2.38 ± 0.93
Mean lesion breadth	2.61 ± 0.51	0.65 ± 0.18	0.63 ± 0.21	0.60 ± 0.28
Mean lesion area	21.93 ± 9.97	1.87 ± 0.52	1.56 ± 0.84	1.51 ± 0.74
Mean lesion index	37.71 ± 7.47	9.17 ± 1.46	7.08 ± 1.45	6.16 ± 1.42
Degree of gastric damage	1-2	1-1.5	1-1.5	0-1.0
Percentage Inhibition of lesion	0	75.68	81.23	83.16

Table-II: Comparison of gastric lesion between Group A (Control) and Group B by t-test significance

Variables	Group-A	Group-B	p-value
Mean number of lesions (± SD)	5.5 ± 1.04	3.66 ± 0.81	< 0.05
Mean lesion length (± SD)	8.05 ± 2.54	2.93 ± 0.69	< 0.01
Mean lesion breadth (± SD)	2.61 ± 0.51	0.65 ± 0.18	< 0.001
Mean lesion area (± SD)	21.93 ± 9.97	1.87 ± 0.52	< 0.001
Mean lesion index (± SD)	37.71 ± 7.47	9.17 ± 1.46	< 0.001

Table-III: Comparison of gastric lesion between Group A (Control) and Group C by t-test significance

Variables	Group-A	Group-C	p-value
Mean number of lesions (± SD)	5.5 ± 1.04	3.33 ± 1.21	< 0.01
Mean lesion length (± SD)	8.05 ± 2.54	2.55 ± 0.77	< 0.001
Mean lesion breadth (± SD)	2.61 ± 0.51	0.60 ± 0.28	< 0.001
Mean lesion area (± SD)	21.93 ± 9.97	1.56 ± 0.84	< 0.001
Mean lesion index (± SD)	37.71 ± 7.47	7.08 ± 1.45	< 0.001

Table-IV: Comparison of gastric lesion between Group A (Control) and Group D by t-test significance

Variables	Group-A	Group-D	p-value
Mean number of lesions (± SD)	5.5 ± 1.04	3.16 ± 0.75	< 0.01
Mean lesion length (± SD)	8.05 ± 2.54	2.38 ± 0.93	< 0.001
Mean lesion breadth (± SD)	2.61 ± 0.51	0.65 ± 0.18	< 0.001
Mean lesion area (± SD)	21.93 ± 9.97	1.51 ± 0.74	< 0.001
Mean lesion index (± SD)	37.71 ± 7.47	6.61 ± 1.42	< 0.001

Student's Unpaired 't' test was performed to compare between Group A & Group B, between Group A & Group C and between Group A & Group D. Difference between Group A & Group B was significant as P value <0.05 (Table II). A statistically highly significant difference (P value <0.01) was observed between Group A & Group C and between Group A & Group D (Table III, IV).

Discussion

The present study has been undertaken to find out the gastroprotective effect of aqueous extract of *Musa paradisiaca* on ethanol induced gastric lesion in rats. For this study, 24 rats were taken and divided into 4 groups. Group A served as a control group that received distilled water 5 ml/kg BW orally daily for 7 days. Group B, Group C & Group D received the *Musa paradisiaca* fruit extract at the doses of 0.2 mg/Kg BW, 0.4 mg/Kg BW & 0.8 mg/Kg BW respectively orally daily for 7 days. At the end of 7 days, a single dose of 1 ml of absolute ethanol (5 ml/kg BW) was administered orally by gastric tube to induce gastric ulcer in each rat of each group. The dose and routes of administration was selected according to Koffuor GA, et al²⁰. Absolute ethanol penetrates the gastric mucosa very quickly, which explains why a period of 30 minutes was sufficient for developing gastric lesions in rats. Lui CF, et al²¹ in their study showed that oral administration of absolute ethanol (5.0 ml/kg) to fasted rats produced extensive necrosis of gastric mucosa and pretreatment with oral administration of propolis ethanol extract (PEE) could effectively and dose dependently prevent such necrosis.

To evaluate the gastroprotective effect of aqueous extract of *Musa paradisiaca*, some parameters of gastric damage such as number of lesions, lesion length, lesion breadth, lesion area, lesion index and percentage inhibition were measured at the end of the experiment. There were 33 stomach lesions in group A (control). Group B, Group C and Group D showed 22, 20 and 19 stomach lesions respectively following the administration of aqueous extract of unripe *Musa Paradisiaca* fruit in this study. Histological features of rat stomachs were also examined to confirm gastric damage and to determine extent. Aqueous extract of *Musa paradisiaca* 0.8 mg/kg BW > aqueous extract of *Musa paradisiaca* 0.4 mg/kg BW > aqueous extract of *Musa paradisiaca* 0.2 mg/kg BW reduced the mean lesion number, mean lesion length, mean lesion breadth, mean lesion area, mean lesion index and gastric damage caused by ethanol. Mean lesion number, mean lesion length, mean lesion breadth, mean lesion area, mean lesion index and gastric damage was maximum in group A and minimum in group D and more in group B than group C & group D. Percentage inhibition of lesion was maximum in group D and minimum in group B and more in group

D than group B & group C. It showed that the aqueous extract of *Musa paradisiaca* protect more in 0.8 mg/kg BW dose than 0.2 mg/kg BW dose and 0.4 mg/kg BW dose. This study result was in well agreement with the result of Koffuor GA, et al.²⁰ study. The highest percentage inhibition of lesion occurred in group-D (pretreated with aqueous extract of *Musa paradisiaca* in 0.8 mg/kg BW dose).

So, pretreated with aqueous extract of *Musa paradisiaca* before ethanol administration prevented the ethanol induced gastric changes and decreased lesion number, lesion length, lesion breadth, lesion area and lesion index. In this study, the aqueous extract of *Musa paradisiaca* was able to deliver satisfying dose-dependent gastroprotective effects in prevention of gastric mucosal lesion induced by absolute ethanol.

Conclusion

Unripe *Musa paradisiaca* (banana) fruit extract has dose dependent gastroprotective effect on rats. Further studies are required for better understanding the gastroprotective mechanism of aqueous extract of *Musa paradisiaca* by administration of the extract in experimental animals having induced gastric lesion of a large sample comparing with positive control receiving established antiulcer drugs. However, unripe *Musa paradisiaca* fruit or fruit extract may be used as a remedy for peptic ulcer disease after ascertaining its safety.

Conflict of interest

The authors declared that they have no conflict of interest.

References

1. Zelikson MS, Bronder CM, Johnson BL, Camunas JA, Smith DE, Rawlinson D, et al. Helicobacter pylori is not the predominant etiology for peptic ulcers requiring operation. *Am Surg.* 2011; 77 (8): 1054-60.
2. Lanas A, Chan FKL. Peptic ulcer disease. *Lancet.* 2017; 390 (10094): 613-24. doi: 10.1016/S0140-6736(16)32404-7.
3. Kavitt RT, Lipowska AM, Anyane-Yeboa A, Gralnek IM. Diagnosis and Treatment of Peptic Ulcer Disease. *Am J Med.* 2019; 132 (4): 447-56. doi: 10.1016/j.amjmed.2018.12.009.
4. Sung JJ, Kuipers EJ, El-Serag HB. Systematic review: the global incidence and prevalence of peptic ulcer disease. *Aliment Pharmacol Ther.* 2009; 29 (9): 938-46. doi: 10.1111/j.1365-2036.2009.03960.x.
5. Garrow D, Delegge MH. Risk factors for gastrointestinal ulcer disease in the US population. *Dig Dis Sci.* 2010; 55 (1): 66-72. doi: 10.1007/s10620-008-0708-x.
6. Lin KJ, Garcia Rodriguez LA, Hernandez-Diaz S. Systematic review of peptic ulcer

- disease incidence rates: do studies without validation provide reliable estimates? *Pharmacoepidemiol Drug Saf.* 2011; 20 (7): 718-28. doi: 10.1002/pds.2153.
7. Kang JY, Tinto A, Higham J, Majeed A. Peptic ulceration in general practice in England and Wales 1994-98: period prevalence and drug management. *Aliment Pharmacol Ther.* 2002; 16 (6): 1067-74. doi: 10.1046/j.1365-2036.2002.01261.x.
 8. Malik TF, Gnanapandithan K, Singh K. Peptic Ulcer Disease. [Updated 2023 Jun 5]. In: StatPearls. Treasure Island (FL): StatPearls Publishing; 2023. Available at: <https://www.ncbi.nlm.nih.gov/books/NBK534792>. [Accessed on March 15, 2015]
 9. Palmer KR, Penmen ID. Alimentary tract and pancreatic diseases. In: Walker BR, Colledge NR, Ralston SH, Penmen ID, Eds. *Davidson's Principles and Practice of Medicine*, 22nd ed. China: Elsevier; 2010. pp 872-5.
 10. Laine L. Nonsteroidal anti-inflammatory drug gastropathy. *Gastrointest Endosc Clin N Am.* 1996; 6 (3): 489-504.
 11. Yuan Y, Padol IT, Hunt RH. Peptic ulcer disease today. *Nat Clin Pract Gastroenterol Hepatol.* 2006; 3 (2): 80-9. doi:10.1038/ncpgas.thep0393.
 12. Ramakrishnan K, Salinas RC. Peptic ulcer disease. *Am Fam Physician.* 2007; 76 (7): 1005-12.
 13. Suerbaum S, Michetti P. *Helicobacter pylori* infection. *N Engl J Med.* 2002; 347 (15): 1175-86. doi: 10.1056/NEJMra020542.
 14. Lindsay J, Langmead L, Preston KL. Gastrointestinal disease. In: Kumar P, Clark M, Eds. *Kumar and Clark's Clinical Medicine*, 8th ed. Spain: Elsevier; 2012. pp 248-51.
 15. Beradi RR, Welage S. Peptic ulcer disease. In: Dipirob TJ, Talbert RL, Yeess G, Matzke G, Wells G, Posey M, Eds. *Pharmacotherapy: A Pathophysiologic approach*, 7th ed. Global: McGraw-Hill; 2008. pp 569-88.
 16. Milosavljevic T, Kostić-Milosavljević M, Jovanović I, Krstić M. Complications of peptic ulcer disease. *Dig Dis.* 2011; 29 (5): 491-3. doi: 10.1159/000331517.
 17. Imam MZ, Akter S. *Musa paradisiaca* L. *Musa sapientum* L. A phytochemical and pharmacological review. *J Appl Pharm Sci.* 2011; 1 (5): 14-20.
 18. Galani VJ. *Musa paradisiaca* Linn - A Comprehensive Review. *Sch Int J Tradit Complement Med.* 2019; 1 (1): 45-56. doi: 10.21276/sijctm.2019.2.4.1.
 19. Ragab M, Osman MF, Khalil ME, Gouda MS. BANANA (*Musa* sp.) Peels as a Source of Pectin and Some Food Nutrients. *J Agric Res Kafr El-Sheikh Univ.* 2016; 42 (4): 88-102.
 20. Koffuor GA, Ainoonson GK, Amponsah KI, Addotey JN, Asiamah EA, Akuffo SK, et al. Anti-ulcerant activity of an aqueous fruit extract of *Musa x paradisiaca* on acetic acid-induced gastric ulceration in ICR mice. *J Medical Biomed Sci.* 2013; 2 (2): 30-9.
 21. Liu CF, Lin CC, Lin MH, Lin YS, Lin SC. Cytoprotection by propolis ethanol extract of acute absolute ethanol-induced gastric mucosal lesions. *Am J Chin Med.* 2002; 30 (2-3): 245-54. doi: 10.1142/S0192415X02000387.

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