Original Article

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ANTIOXIDANT AND ANTIULCER ACTIVITY OF MUSA PARADISIACA IN RATS

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Abstract

The antiulcer potential of ethanolic extract of *Musa paradisiaca leaves* was evaluated by ethanol induced ulcer models. Effect of administration of ethanol extract of Musa paradisiacal leaves at dose of 100 mg/kg b.w. was given by oral route. Ethanol extract of *Musa paradisiaca* decreased gastric content, total acidity, ulcer index, and increase in pH of gastric pylorus ligation ulcer model. The anti-oxidant effect of the extract of Musa *paradisiaca* was also assessed in the gastric content of the ulcer induced as well as the *M.paradisiaca* treated rats. *M.paradisiaca* was found to be useful as a natural antiulcer as well as a gastric antioxidant in the treatment of experimental ulcer induced in albino rats.

Keywords: *Musa paradisiaca leaves,* Ulcer model, Anti-oxidant effect.

Introduction

Musa paradisiaca is one of the oldest and best known fruits of the world. It is delicious and seedless and is available in all seasons at a price which is within everybody's reach. It is a very hygienic fruit as it comes in a germ proof package. Its thick covering provides an excellent protection against bacteria and contamination. The mature fruits vary in sizes and may be greenish, yellow or reddish in color. Bananas are true sources of energy. A banana contains potassium, proteins, fibers, carbohydrates and an association of vitamins: A, B, B6, C and E; it is rich in calcium, magnesium, iron, zinc and folic acid. These facts being taken into consideration, the banana is one of the healthiest fruits. It also contains serotonin or the substance of happiness, having an anti-stress role. The plant is used in Siddha medicine for the treatment of ulcer. The present study was carried out to investigate the anti-oxidant and antiulcer activity of *Musa paradisiaca* leaf extract (**MPL**) extract in rats.[1-3]

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Gastric ulcer is a break in the tissue lining of the stomach. Most ulcers can be cured without complications; however, in some cases peptic ulcers can develop, such as in penetration, perforation, bleeding (hemorrhage), and obstruction. Ethanol and aspirin-induced gastric ulcer models have been widely used for the evaluation of gastro protective activity. Acute treatment with ethanol increases oxidative stress, DNA damage, xanthineoxidase activity and malondi aldehyde levels, and decreases the total glutathione content in gastric mucosal cells. Aspirin-induced ulcer is mediated through tissue damaging free radicals which are produced from the conversion of hydroperoxyl to hydroxy fatty acids, which leads to cell destruction. It has been found that oxygen-derived free radicals are implicated in the mechanism of acute and chronic ulceration in the gastric mucosa and scavenging these free radicals can play an appreciable role in healing the ulcer.[4,5]

Antiulcer Activity -Ulcer induced by ethanol

Wister rats of either sex (150–200 g) were properly housed in separate cages at controlled room temperature in a 12-h light dark cycle. They were fed with standard pellet diet and water *ad libitum*.

The rats were divided into four groups of six each. Group I (toxicant control) received absolute ethanol (1 ml/animal); Group II was treated with ranitidine (50 mg/kg); Groups III treated with Musa paradisiacal leaves extract(**MPL**),100 mg/kg.[6-9] The rats were fasted for 24 h and they received 1 ml of absolute ethanol orally. Standard and test drugs were administered orally 30 min before the ethanol dose. The animals were sacrificed after 1 h of ethanol administration, and their stomachs were excised and the gastric contents were aspirated. The contents were subjected to centrifugation at 1000 rpm for 10 min and then analyzed for pH (digital pH meter). The stomachs were washed with normal saline and kept in 10% formalin for the determination of ulcer index and histological studies. Ulcer index is determined. The lipid peroxides (LPOs) of stomach mucosa were determined indirectly by thiobarbituric acid reactive substances (TBARS) formation. Reduced glutathione concentration was read off a standard curve and expressed as μg GSH/g of wet tissue. The treated groups were compared with the toxicant group; results were expressed as a mean ± SD of six animals in each group. The results were analyzed statistically using one-way analysis of variance (ANOVA) followed by Dunnett's test.[10-12]

Table 01:
Effect of M.paradisiaca on the Gastric Secretion, Acidity and Lesions in ethanol induced ulcer in rats.

Group	Dose (mg.kg)	Gastric secretions at 6hr (Mean +SEM)		
		Vol. of Gastric Secretion	Titrable acidity	Ulcer index
Control	1% CMC (1ml/kg)	13.13±0.478	18.94±0.361	0.196±0.021
MPL	100 mg	10.56 ±0.478	12.728±0.478	0.124±0.033*
Ranitidine	50mg	1.33±0.247	1.295±0.519	0.059±0.014***

*P<0.05, **P<0.01, ***P<0.001

Result and Discussion

The **MPL** significantly reduced gastric pH of ulcer rats . In both the ulcer models, **MPL** (100 mg/kg) significantly reduced the lipid peroxidation levels when compared to untreated control.group. The results also show that MPL significantly increased the reduced glutathione levels. **MPL** at 100 mg/kg showed partial healing of ulcer with few inflammatory cells and showed the healed ulcer, normal mucosa, and no inflammatory cells.

Table 02: Antioxidant effect of M.paradisiaca. on ethanol induced ulcer models

Group	Dose (mg.kg)	In gastric contents	
		Lipid peroxide (MDA n moles/g)	Reduced glutathione (ug/g)
Control	1% CMC (1ml/kg)	46.22 ±0.51*	105.44±1.26*
MPL	100mg	25.14±0.20*	137.55±1.01*
Ranitidine	50mg	22±.450.36*	153.40±1.56*

*P<0.05, **P<0.01, ***P<0.001

The genesis of ethanol-induced gastric lesions is multifactorial with the depletion of gastric wall mucous content as one of the involved factors. It is also associated with significant production of free radicals, leading to an increased oxidative stress and damage to the cell and cell membrane. The present study reveals that MPL significantly reduced the ulcer index and increased the gastric pH of ethanol induced ulcer models. Lipid peroxidation is a free radical mediated process, which has been implicated in a variety of disease states. It involves the formation and propagation of free radicals, the uptake of oxygen and rearrangement of double bond in unsaturated lipids, which eventually result in destruction of membrane lipids. Ethanol significantly reduced the. Antioxidant enzymes such as glutathione present in oxygen handling cells, which are the first line of cellular defense against oxidative injury. They decrease the gastric mucosal damaging effect of absolute alcohol. There was a significant increase of glutathione activity in MPL pretreated rats. Hence, the antioxidant activity of MPL may be one of the important defensive factors involved in its antiulcer effect. (Table2)

Conclusion

To conclude, the ethanolic extract of *Musa paradisiacal* has been found to have antiulcerogenic effect, which could be related to its antioxidant potential. More

work is required for a clear understanding of the mechanism of action.

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