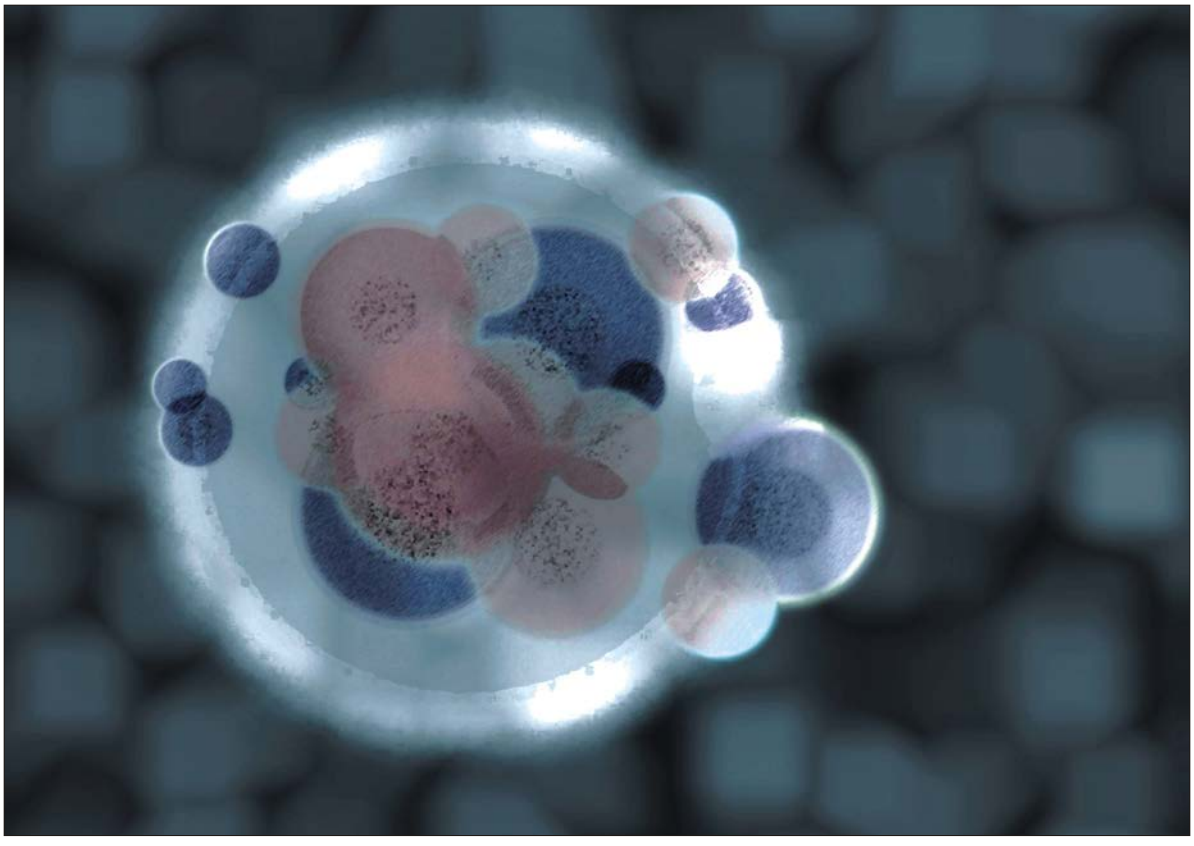

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Gastroprotective Effects of Aqueous Extract of *Adansonia digitata* Leaf on Ethanol-Induced Ulceration in Rats

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Abstract: Aqueous extract of *Adansonia digitata* inhibit ethanol-induced gastric ulceration in rats. Oral pretreatment with *Adansania digitata* (150-600 mg kg^{-1}) caused significant dose-dependent increase both in preventive ratio and percentage ulcer reduction. This effect might in part be due to its astringent, flavanoids and anti-oxidant properties earlier reported.

Key words: *Adansonia digitata*, ethanol, gastroprotective, ulceration

INTRODUCTION

Adansonia digitata is a largely tropical plant of African origin (Lucas, 1971), belonging to Bombaceae family. Various parts of the plant are used as food and popular folk remedy for ailments (Ramadan *et al*., 1994; Tal-Dia *et al*., 1974; El-Khalifa, 1999) in many African cultures. There are reports (Tal-Dia *et al*., 1974; El-Khalifa, 1999) that the plant is medicinally used for the treatment of various ailments including prophylactic, colic, fever, asthma, diarrhea, gastro enteric inflammation, ulcer amongst others.

By far the most profound claim is the one by herbalist in northern part of Nigeria, that when whole leaf soup preparation or aqueous suspension of the leaf is orally administered to apparently ulcer patients the, the pain is rapidly relief; presumably due to the ability of some components of *A. digitata* to stimulate or promote healing of gastric ulcerations in the body. The aim of the present study was therefore to investigate the effect of *A. digitata* on ethanol induced ulceration in rats. In view of the indiscriminate use of *A. digitata* and reported presence of (Arrigori and De Tullio, 2002) of high concentrations of flavonoids, vitamins A, C and E (antioxidants) the present investigation went further to determine the effects of ingestion by tissue histological examinations.

MATERIALS AND METHODS

Animals: Male Wister rats weighing between 180-200 g were obtained from National veterinary Research institute Vom plateau state, Nigeria. They were allowed to acclimatized for three weeks, fed with pellet feed (ECWA Nig Limited Jos) and water *ad- libitum* in our laboratory. Ethanol (1 mL/rat of 50% ethanol) was used to induce ulceration in the rats (Nwafor *et al*., 1996).

Plant material: The plant material used in this study was collected from Shekwari village, outskirt of Maiduguri metropolis in month of September 2005. It was identified and authenticated by Dr. S.S. Sanusi of Department of Botany University of Maiduguri. The leaves were air-dried and extracted according to the method of Mittal *et al*. (1981), Nwafor *et al*. (1996) and WHO (1992).

The dried leaf was pulverized using pestle and mortar. 200 g of powder was mixed with 1 L of distilled water in 2 L beaker and boiled for 11/2 h, then allowed to cool to 40°C and expressed. The expressed liquid was strained using what man qualitative fitter paper No. 1. The filtrated was collected in a beaker and evaporated at 40°C until the volume was reduced to 400 mL so that 1 mL of the extract represents 0.5 g of the dried weight. The dosage to be given is then calculated from the following formula (Karumi *et al*., 2003).

$$\text{Amount (mL)} = \frac{\text{Wt. of the rat (kg)} \times \text{Dosage to be given (mg kg}^{-1}\text{)}}{\text{Concentration of the stock solution}}$$

GASTRIC ULCERATION STUDY

During the studies, the rats were divided into 5 groups of 5 rats each. Food was withdrawn 24 h and

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Table 1: Ulcer scoring system criteria

Ulcer	Scoring system
0.0	Normal
0.5	Punctuated or pinpoint hemorrhagic ulcer
1.0	Two or more small hemorrhagic ulcers less than 3 mm in diameter
2.0	Ulcers greater than 3 mm in diameter
3.0	Several ulcers

Criteria Evbuoman and Bolarinwa (1990)

water 2 h before the commencement of the study. The route of administration is oral. The extract and ethanol were administrated through intragastric tube. Earlier, pilot test were carried out to establish the dose of ethanol that causes ulcer lesions after 4 h of oral administration (1 mL/rat of 50% ethanol). Group 1 was given 1 mL of normal saline. Group 2 was given 1 mL of ethanol, while groups 3-5 were pretreated with *Adansonia digitata* (150, 300 and 600 mg kg^{-1}) 1 h before administration of ulcerogen. Four hour later, the animals were sacrificed by stunning; the stomachs were removed and opened along the greater curvature. The tissues were fixed with 10% formal saline. Macroscopic and microscopic examination were performed on the stomach and scored for presence of lesions using Alpin and Ward (1967) method modified by Evbuoman and Bolarinwa (1990) (Table 1).

Ulcer Index (UI) of ethanol alone, ulcer index and preventive ratio of each of the groups pretreated with *Adansonia digitata* were calculated using the method of Zaidi and Mukerji (1958).

$$\text{Ulcer index (UI)} = \frac{\text{Ulcerated group} - \text{UI (Protectd group)}}{\text{UI (Ulcerated group)}} \times 100$$

$$\text{Degree of ulceration} = \frac{\text{Total ulcer score}}{\text{No. of animals ulceratrd}}$$

RESULTS AND DISCUSSION

The results of oral *Adansonia digitata* pretreatment on ethanol-induced gastric ulceration Fig. 1 and Table 2. There was a progressive decline of ulcer indices (0.5±0.02-0.10±0.03) in pretreated group (3-5) when compared with group 2 (1.5±0.05) ulcerogen. The preventive ratio of the extract also showed an ascending pattern (66.67, 73.33 and 93.33). There were significant difference in groups (3-5) when compared to group 2 ($p<0.05$ and $p<0.005$), respectively.

The histological examinations of the gastric mucosa were shown in Fig. 2-6. The *Adansonia digitata* treatment at dosages of 150-600 mg kg^{-1} body weight produced a progressive decline in ulcer lesion when compared to normal (Fig. 2).

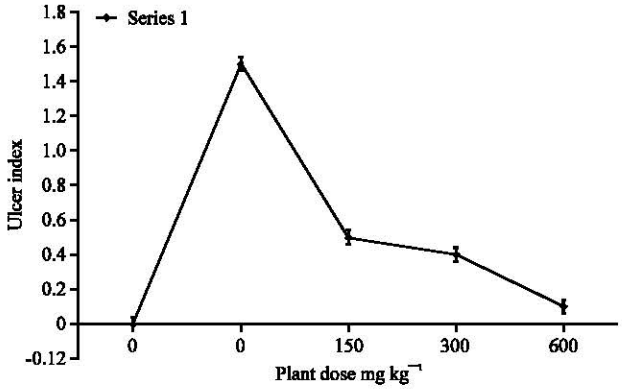

Fig. 1: Effect of Adansonia digitata on gastric ulceration in animals receiving ethanol (1 mL rat^{-1}) orally. Each data point represents mean±SEM of five animals

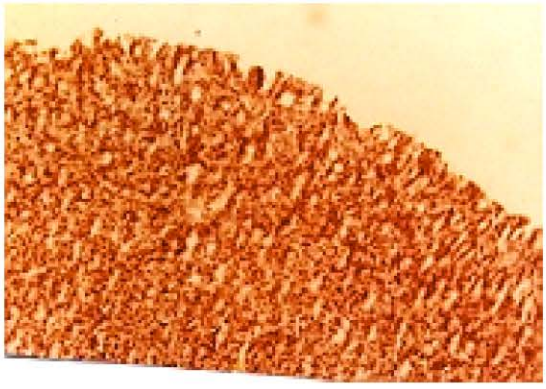

Fig. 2: The gastric mucosa control group gives normal saline only showing non-ulcerated mucosa (mg X 300) (NM)

Fig. 3: The gastric mucosa of ethanol induced (1 mL rat^{-1} of 50%) ulcerated group showing very severe ulcerated gastric mucosa (VSUM)

Table 2: The ulcer index preventive ratio and percentage reduction of ulceration obtained from rat orally pretreated with different concentration of extract 1 h prior to ethanol administration

Groups	Extract dose mg kg^{-1}	Ethanol mL/rat	Ulcer index	Preventive ratio	Ulcer reduction (%)
1	0.0	0.0	0.0	0.00	0.00
2	0.0	1.0	1.5±0.05	0.00	0.00
3	150.0	1.0	0.5±0.02	66.67	66.67*
4	300.0	1.0	0.4±0.01	73.33	73.33**
5	600.0	1.0	0.1±0.03	93.33	93.33**

Significant or not significant relative to group 2 (*p<0.005; **p>0.005) n=5

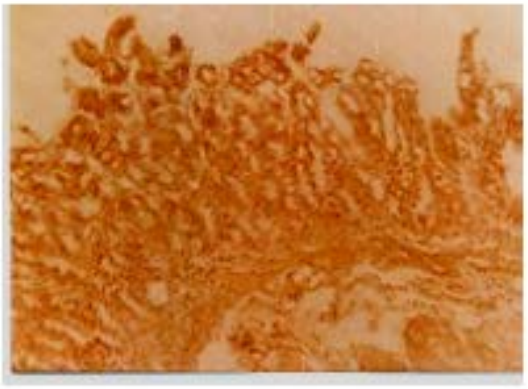

Fig. 4: The gastric mucosa of *Adansonia digitata* pretreated group (150 mg kg^{-1}) showing mild ulcerated mucosa (MUM)

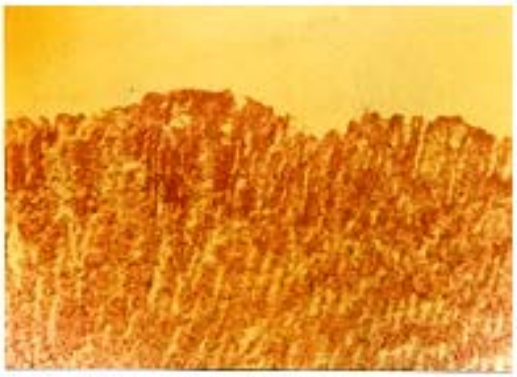

Fig. 5: The gastric mucosa of *Adansonia digitata* pretreated group (300 mg kg^{-1}) ulcerated group showing very mild ulcerated mucosa (VMM)

The result of this study showed that the oral administration of aqueous extract of *Adansonia digitata* reduced ethanol-induced ulceration in rats. The reduction was dose dependent. The mechanism by which the extract produced these effects seems unclear. However, an elucidation of the pathogenetic mechanisms of peptic ulceration will help throw more light on mechanism involved.

Fig. 6: The gastric mucosa of *Adansonia digitata* pretreated group (600 mg kg^{-1}) showing insignificant ulcerated mucosa (IUM)

Ethanol is an established ulcerogen especially in an empty stomach (Hirokawa *et al*., 1998). The ulcerogenicity of ethanol is due to intracellular oxidative stress, producing mitochondrial permeability, transition and mitochondrial depolarization which result to the death of cells in the gastric mucosa (Hirokawa *et al*., 1998; Hernandez *et al*., 2000). This is because of its congestive inflammation and tissue injury. It has been known that the protective function of flavanoids and anti-oxidant (vit A, E and C) present in the plant may be important (Penissi and Piezzi, 1999). This view is supported by the fact that, gastric mucosa is known to have certain anti-oxidant activity thereby reducing mucosal damage mediated by free radicals (Penissi and Piezzi, 1999), which in turn attack the cell membrane causing a lipid derived free radicals such as conjugated diene and lipid hydroperoxides. These free radicals are extremely reactive product that causes oxidative damages such as gastric ulcer (Bagchi *et al*., 1998).

The aqueous extract of *A. digitata* reduced ethanol-induced ulceration in rats enterally. The reduction was dose-dependent. The preventive ratios of *A. digitata* on ethanol-induced ulceration especially in higher doses were markedly high. Although the précised mechanism of action of *A. digitata* is not clear it can be proposed that mucosal protection induced by *A. digitata* leaves in the study may be partly due to its high content of flavanoids and anti oxidant (Arrigori and De Tullio, 2002) which are well known compound that prevent and combat the formation of free radicals. *A. digitata* is also used as an astringent (Wood, 1969). Being an astringent it may have also precipitated microproteins on the site of ulcer thereby forming an impervious protective pellicle over the lining to prevent absorption of toxic substance and resist the attack of proteolytic enzymes (Nwafor *et al*., 1996).

In conclusion, the gastro protective potential of the extract against ethanol-induced ulceration in rats might in part be due to its astringent properties or its anti-oxidant effect. However, further work on its effect on gastric acid secretion is advocated.

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