

Research Article



Effects of Aqueous and Methanol Extracts of *Portulaca oleracea* on Gastric Acid Secretion in Male Wistar Rats

Oyededeji K. Olusina^{1*}, Bolarinwa A. Folashade²

¹Department of Physiology, College of Medicine and Health Sciences, Afe Babalola University, Ado-Ekiti, Nigeria.

²Department of Physiology, College of Medicine, University of Ibadan, Nigeria.

*Corresponding author's E-mail: sinaoyededeji@yahoo.com

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ABSTRACT

Portulaca oleracea is a fleshy annual herb which is distributed throughout the temperate and tropical areas of the world. This plant is used in traditional medicine as a gastric sedative. Air-dried specimen of *Portulaca oleracea* was cold-extracted in distilled water and 70 % methanol respectively for 72 hours and concentrated using steam bath. The resulting aqueous and methanol extracts (AEPO and MEPO) were then subjected to gastric acid secretory study in male rats. Fifty – five male rats (150 – 200 g) were randomly divided into AEPO (25, 50, 75 mg/kg), MEPO (25, 50, 70 mg/kg) and histamine (1 mg/kg) treated groups. The combination of extracts with histamine (1 mg/kg) and cimetidine (100 mg/kg) groups were used to investigate the probable mechanism of action. The experimental animals also served as the control. Gastric acid secretory response was determined by titration method. Data were analyzed using t-test at $p < 0.05$. The results showed that treatment of rats with all the doses of AEPO and MEPO caused significant ($p < 0.05$) reductions in gastric acid secretion. The submaximal doses of AEPO (50 mg/kg) and MEPO (50 mg/kg) significantly ($p < 0.05$) inhibited histamine-induced gastric acid secretion. Cimetidine seemed to potentiate or augment the extracts (AEPO and MEPO) inhibition of gastric acid secretions. The results suggest that the extracts (AEPO and MEPO) gastric acid anti-secretory effects could be mediated via H_2 – receptors.

Keywords: *Portulaca oleracea*, Gastric acid secretion, Histamine, Cimetidine, Rats.

INTRODUCTION

Global economic depression as a result of fall in the price of crude oil in the international market with the attendant depreciation in currency values of most African countries relative to the U.S. Dollars coupled with the expensiveness and toxic side effects of most synthetic drugs; reversal to the use of the cheaper and more efficacious herbal remedies cannot be overemphasized by Africans, most of whom are bedeviled by poverty.

Portulaca oleracea belongs to the family of Portulacaceae. It is commonly called Purslane in English language, “Babbajibi” in Hausa language and “Esan omode” or “Papasan” in Yoruba language. It is a fleshy annual herb, much-branched and attaining 30 cm long¹.

It is used medicinally in Ghana for heart-palpitations². The plant is used as a diuretic in Nigeria³. A tisane of the plant is drunk in Trinidad as a vermifuge⁴. At some areas near Benin City (Nigeria), the plant, along with other ingredients is taken as an aid to the development of the fetus⁵. In Ivory Coast, the plant mixed with grains-of-paradise (*Aframomum melegueta* K Schum, *Zingiberaceae*) and karite butter, furnishes an ointment applied to areas of costal pains⁶. Further use as an anodyne to pain is found in Liberia as a gastric sedative⁷ and in Gabon where a decoction is used in lotion on the forehead for headache⁸.

Pharmacologically, it has been reported that aqueous and methanol extracts of *Portulaca oleracea* have contractile

effects on isolated intestinal smooth muscle in *in-vitro* preparations⁹. It has also been reported that aqueous and methanol extracts of *Portulaca oleracea* have some toxic and beneficial effects on the blood chemistry in male Wistar rats¹⁰.

The extracts of *Portulaca oleracea* have been reported to have protective effects on hypoxic nerve tissue¹¹, anti-inflammatory effects¹² and wound-healing activity^{13,14} also reported the skeletal muscle relaxant effect of the plant.

Since this plant has been reported to be used in Liberia traditional medicine as a gastric sedative⁷, this study therefore aims to scientifically authenticate the veracity of this claim by investigating the effects of aqueous and methanol extracts of this plant on gastric acid secretion in male Wistar rats.

MATERIALS AND METHODS

Experimental Animals

Adult male rats weighing between 150 g and 200 g bred in the Pre-Clinical Animal House of the College of Medicine and Health Sciences, Afe Babalola University, Ado-Ekiti were used. Female rats were not used because gastric acid secretion is known to vary with the phases of estrus cycle¹⁵. They were housed under standard laboratory conditions and had free access to feed and water. They were acclimatized for two weeks to laboratory conditions before the commencement of the experiments. All experiments were carried out in



compliance with the recommendations of Helsinki's declaration on guiding principles on care and use of animals.

Plant Material

Fresh specimens of *Portulaca oleraceae* were collected from the Botanical Garden of the Forestry Research Institute of Nigeria (FRIN), Jericho, Ibadan, where it was identified and assigned a voucher specimen number FHI 108334.

Preparation of the Extracts

Large quantity (2 kg) of the fresh specimens of *Portulaca oleracea* were washed free of soil and debris, and the roots were separated from the leaves and stems. The leaves and stems were air-dried for six weeks and then pulverized using laboratory mortar and pestle and was later divided into two samples A and B.

(i) Aqueous Extract of *Portulaca oleracea* (AEPO)

Weighted Portion (400.0 g) of sample A was macerated and extracted with distilled water (1:2 wt/vol) for 72 hours at room temperature (26–28 °C). The resulting solution was then filtered using a wire-gauze and a sieve with tiny pores (0.25 mm). The distilled water was later evaporated using steam-bath to give a percentage yield of 11.8 % of the starting material. The dried material was reconstituted in distilled water to make up test solutions of known concentrations.

(ii) Methanol Extract of *Portulaca oleracea* (MEPO)

Weighted portion (350.0 g) of sample B was macerated and extracted with 70 % methanol (1:2 wt/vol) for 72 hours at room temperature (26–28 °C). The resulting solution was then filtered using a wire-gauze and a sieve with tiny pores (0.25 mm). The 70 % methanol was later evaporated using steam-bath to give a percentage yield of 10.2 % of the starting material. The dried material was reconstituted in distilled water to make up test solutions of known concentrations.

Ten grams of AEPO and MEPO were dissolved in 100 ml of distilled water to give a concentration of 0.1 g/ml. The dosages of AEPO and MEPO administered in these studies were in accordance with those reported by ¹⁶.

Drugs and Chemicals

The following drugs and chemicals were used: sodium hydroxide (Sigma), Phenolphthalein (BDH), Histamine dihydrochloride (BDH, England), Cimetidine (Greenfield Pharm. Ltd), Sodium thiopental (Abbott Laboratories).

Animal Preparation

The rats were anaesthetized with thiopental (50 mg/kg) and body temperature was maintained at 38°C by an adjustable electric bulb directed to the animals. Each rat was tied face up on a rat dissecting board. The neck region was opened slightly with an incision followed by blunt dissection so that blood vessels and the vagus

nerves were not transected (cut). The trachea was exposed and cannulated with a size – 4 polythene trachea cannula and exteriorized to ensure free normal breathing throughout the period of the experiment. In no instance did any of the rats show any signs of pain during the operation and the experiment.

The abdomen was opened through a ventral laparotomy and the pylorus was ligated. A soft catheter (diameter 1.5 mm) was inserted into the stomach through an incision in the distal esophagus and held in place by ligature. The exterior (outside) end of the catheter was attached to a two-way valve into which an administering syringe was fitted.

Experimental Design

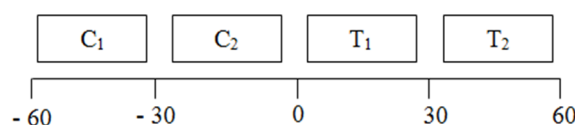


Figure 1: Schematic representation of the experimental design.

C₁ and C₂ = 2.0 ml of distilled water each (Control).

T₁ and T₂ = Different doses of the treatment extract dissolved in 2.0 ml of distilled water.

The experimental design is summarized in Figure 1. The rats were starved for 24 hours but had free access to water before the experiment. Before the administration of control solvent (2.0 ml distilled water) through the intraesophageal implanted intragastric soft catheter at the beginning of the experiment, the stomach was rinsed with distilled water and emptied by aspiration via the attached syringe. Before perfusing the stomachs with the control and test meals, the pH of the control and test meals were adjusted to 5.5 by the addition of HCl or NaOH in appropriate amount. To investigate the kinetics of gastric acid output in response to the treatment/test meals after ending perfusion, the two control periods (C₁ and C₂ in Figure 1) were followed by two treatment or test meal periods. Gastric acid output in response to the controls and various treatments (extracts and drugs) was determined in 30 minutes intervals, vis-à-vis, during each experiment, two control meals (2.0 ml of distilled water each at pH 5.5) and two treatment/test meals (different doses of the treatment/test meals dissolved in 2.0 ml of distilled water each at pH 5.5) were instilled into the stomach at 30 minutes intervals. After each 30 minutes period, the remaining gastric contents were emptied and the volume was measured and centrifuged at 3,000 rpm for 10 minutes (in case of turbidity) and the supernatant was used in titration; thereafter, the next meal was instilled. In this study, the experimental animals also served as the control, vis-à-vis, the first two meals served as controls (C₁ and C₂ in Figure 1) and the remaining two meals served as the test/treatment meals (T₁ and T₂ in Figure 1). The acid concentrations of the four reaspirates of the 30 minutes intervals were determined by titration

of aliquots (1.0 ml each) of the perfusate to pH 7.0 with 0.01 M NaOH using phenolphthalein as the indicator, and the gastric acid output was calculated.

Animal Grouping and Treatments

Fifty-five male rats weighing between 150–200 g were randomly divided into eleven groups (n = 5). Groups I – III were intragastrically given AEPO (25 – 75 mg/kg), groups IV – VI were intragastrically given MEPO (25-75 mg/kg), group VII was intraperitoneally given histamine (1.0 mg/kg) which is an H_2 - blocker.

In another set or series of experiments to investigate the probable mechanism of action, groups VIII and IX animals were respectively intragastrically pretreated with submaximal doses of AEPO (50 mg/kg) and MEPO (50 mg/kg) 30 minutes prior to intraperitoneal treatment with histamine (1.0 mg/kg). Groups X and XI animals were respectively intragastrically pretreated with submaximal doses of AEPO (50 mg/kg) and MEPO (50 mg/kg) in combination with intraperitoneal administration of cimetidine (100 mg/kg) (H_2 – receptor blocker) 30 minutes prior to intraperitoneal treatment with histamine (1.0 mg/kg).

Calculation

To calculate the 1 hour gastric acid output for each of the control and treatment periods, the average of the two values obtained during the 1 hour control and treatment periods were computed.

Statistical Analysis

The mean and standard error of men (S. E. M.) were calculated for all values. Comparison between the control and experimental group was done using the t-test. Differences were considered statistically significant at $p < 0.05$.

RESULTS

Treatment of rats with all the doses of AEPO (25 mg/kg, 50 mg/kg, 75 mg/kg) and MEPO (25 mg/kg, 50 mg/kg, 75 mg/kg) caused significant ($p < 0.05$) reductions in gastric acid output relative to their respective controls (Figures 1 and 2). Pretreatment of rats with submaximal doses of AEPO (50 mg/kg) and MEPO (50 mg/kg) prior to treatment with histamine (1.0 mg/kg) produced significant ($p < 0.05$) reductions in gastric acid output relative to their respective controls (Figure 3). However, the percentage reduction in gastric output produced by co-administration of histamine with 50 mg/kg AEPO (29.1 %) is lower than that of co-administration of histamine with 50 mg/kg MEPO (36.2%). Pretreatment of rats with submaximal doses of AEPO (50 mg/kg) and MEPO (50 mg/kg) and cimetidine (100 mg/kg) prior to treatment with histamine (1.0 mg/kg) caused significant ($p < 0.05$) reductions in gastric acid output relative to their respective controls (Figure 4). However, the percentage reduction in gastric output produced by co-administration of AEPO (50 mg/kg), cimetidine and histamine (7.5 %) is

lower than that of co-administration of MEPO (50 mg/kg), cimetidine and histamine (10.6 %).

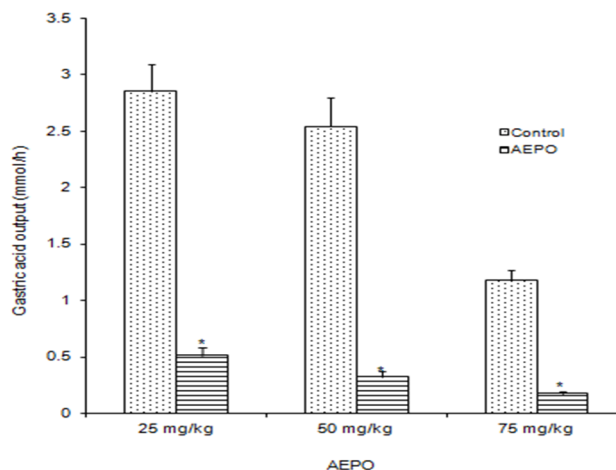


Figure 1: Effect of AEPO on gastric acid output in rats (n=5, * $p < 0.05$).

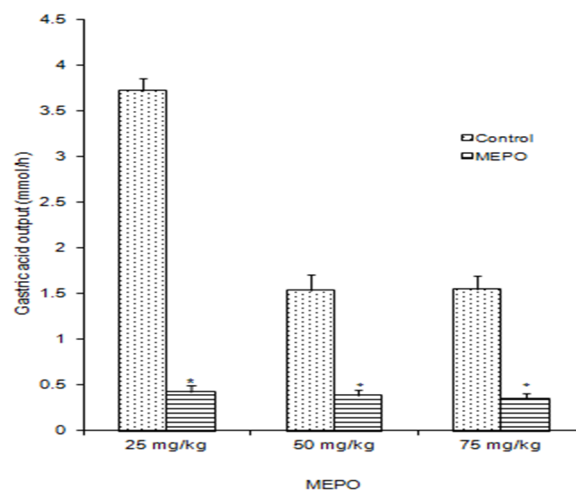


Figure 2: Effect of MEPO on gastric acid output in rats (n=5, * $p < 0.05$).

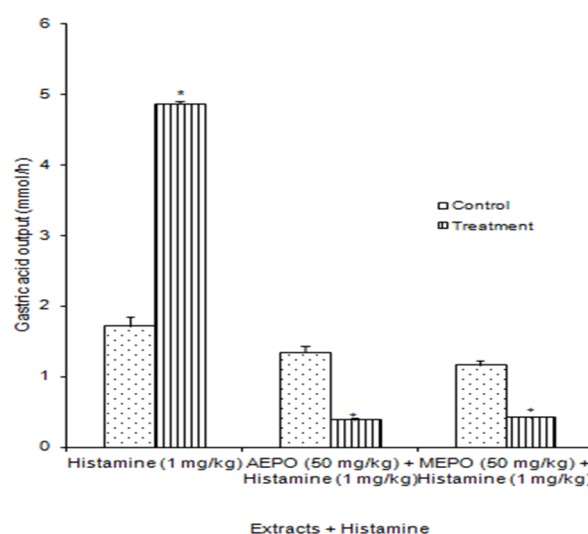


Figure 3: Effects of histamine and co-administration of extracts with histamine on gastric acid output in rats (n=5, * $p < 0.05$).

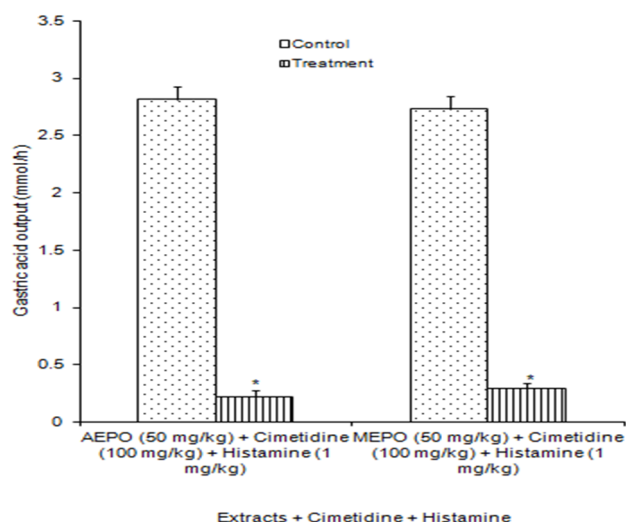


Figure 4: Effects of co-administration of cimetidine, extracts and histamine on gastric acid output in rats (n=5, *p<0.05).

DISCUSSION

The results obtained in this study have shown that the extracts (AEPO and MEPO) significantly decrease gastric acid output in rats at various treatment doses, these reductions in gastric acid output by the extracts indicate inhibition of the secretory process. The decrease in gastric acid output induced by the extracts may be due to back-diffusion of hydrogen ions into the gastric mucosa or by inhibiting the secretion of gastric acid. Submaximal doses of the two extracts (AEPO and MEPO) and cimetidine significantly reduced gastric acid secretion induced by histamine with AEPO showing higher anti-secretory activity than MEPO, and the two extracts seem to potentiate or augment the gastric acid inhibitory action of cimetidine (H_2 – receptor blocker). This suggests that AEPO and MEPO have inhibitory effects on gastric acid secretions which seem to mimic the inhibitory action of cimetidine (H_2 -receptor blocker) on gastric secretion.

The extracts may act by blocking H_2 – receptors leading to inhibition of histamine release whose stimulatory action on gastric acid secretion had been established¹⁷. It is established that inhibition of histamine through H_2 – receptors inhibit intracellular adenylate cyclase, Na^+ - K^+ ATPase and inhibition of proton pump of parietal cells that eventually reduce the gastric acid secretion^{18,19}. Cimetidine, which is a well – known H_2 – receptor antagonist, inhibits the activation of adenylyl cyclase, thus, blocking the formation of cyclic AMP that is necessary for HCl production²⁰.

However, there is the possibility of the involvement of other receptors which are yet to be investigated. Similar results were reported by²¹ in *Eremomastax speciosa* extract treated rats.

It can be concluded that *Portulaca oleracea* probably has gastric acid secretion inhibitory effect which provides scientific basis to the folkloric claim of the use of the plant as a gastric sedative⁷. Its gastric acid anti-secretory

effect could be mediated via H_2 – receptor. The active principles that were responsible for these gastric acid anti-secretory effects of this plant could be the three compounds that we had earlier isolated from this plant namely: ergosterol, lupone and tetracyclic steroid²²⁻²⁴.

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