Gastroprotective Effects of Aqueous and Methanol Extracts of *Portulaca oleracea* on Ethanol-Induced Gastric Ulcer in Male Wistar Rats

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**ABSTRACT**

*Portulaca oleracea* is a much branch reddish – stemmed herb that succeed better in moist or dry environments that are bright and sunny. This plant is used in folk (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. This study investigates the effects of the resulting Aqueous and Methanol Extracts of *Portulaca oleracea* (AEPO, MEPO) on ethanol – induced gastric ulceration in male rats. Forty male rats (150 – 200 g) were randomly divided into eight groups of five animals each and pretreated with distilled water (10 ml/kg), ranitidine (150 mg/kg), AEPO (50, 100, 150 mg/kg) and MEPO (50, 100, 150 mg/kg). Thirty minutes after pretreatments, ulceration was induced in all the animals by ethanol (80 %). The ulcers were measure using a graticule attached to a dissecting microscope. The ulcer index and curative ratio were calculated for all the groups. All the treatment doses of the extracts (AEPO and MEPO) and ranitidine suppressed the ulceration induced by ethanol. The results have provided scientific basis to the use of this plant in folk medicine as a gastric sedative.

**Keywords:** *Portulaca oleracea*, Gastric ulceration, Ranitidine, Ulcer index, 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 cheaper and more efficacious herbal remedies cannot be overemphasized by Africans, most of whom are bedeviled by poverty¹.

*Portulaca oleracea* belongs to the family of Portulacaceae. It is commonly called Purslane in English language, “Babajibji” in Hausa language and “Esan omode” or “Papasan” in Yoruba language. It is a fleshy annual herb, much-branched and attaining 30 cm long².

It is used medicinally in Ghana for heart-palpitations³. The plant is used as a diuretic in Nigeria⁴. A tisane of the plant is drunk in Trinidad as a vermifuge⁵. At some areas near Benin City (Nigeria), the plant, along with other ingredients is taken as an aid to the development of the fetus⁶. In Ivory Coast, the plant mixed with grains-of-aradise (*Aframomum melegueta* K Schum, Zingiberales) and karite butter, furnishes an ointment applied to areas of costal pains⁷. Further use as an anodyne to pain is found in Liberia as a gastric sedative⁸ and in Gabon where a decoction is used in lotion on the forehead for headache⁹.

Pharmacologically, it has been reported that aqueous and methanol extracts of *Portulaca oleracea* have contractile effects on isolated intestinal smooth muscle in *in-vitro* preparations¹⁰. It has also been reported that aqueous and methanol extracts of *Portulaca oleracea* have some toxic and beneficial effects on the blood chemistry of albino rats¹¹. The extracts of *Portulaca oleracea* have been reported to have protective effects on hypoxic nerve tissue¹², anti-inflammatory effects¹³ and wound-healing activity¹⁴,¹⁵ 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 sedative⁶, this study therefore aims to investigate the gastroprotective effects of the aqueous and methanol extracts of this plant on ethanol-induced gastric ulcer 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. 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.

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Plant Material

Fresh specimens of *Portulaca oleracea* 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.

**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.

**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

Animal Grouping and Treatments

Forty male rats weighing between 150–200 g were randomly divided into eight groups, with each group consisting of five animals. All the animals were fasted for 24 hours before the commencement of the experiment to ensure empty stomachs, but were given water *ad libitum*.

Group I which served as the negative control group was orally given 10 ml/kg of distilled water, group II was orally given ranitidine (150 mg/kg) (a standard reference drug) as the positive control group, groups III – V were orally given AEPO (50 – 150 mg/kg), while groups VI – VIII were orally given MEPO (50 – 150 mg/kg). Thirty minutes after their pretreatments, all the animals were orally given 80 % ethanol (5 ml/kg) to induce gastric ulceration. One hour after the ethanol administration, all the animals were sacrificed by an overdose of diethyl ether and the stomachs were rapidly removed, opened along their greater curvatures and gently rinsed under a running water from a wash bottle and the lesions in the glandular part of the stomach were measured with a eyepiece graticule attached (inserted) to the eyepiece of a dissecting microscope (stereomicroscope). The eyepiece graticule was earlier calibrated using a stage micrometer so that it can be used to take measurements of specimens under the stereomicroscope.

Long lesions were counted and measured along their greater length. Petechial lesions were also counted. Each five petechial lesions were taken as 1mm of ulcer 17. The sum of the total length of long ulcers and petechial lesions in each group of rats was divided by its number to calculate the ulcer index (mm). The macroscopic curative ratio was determined by the formula:

\[
\text{Curative ratio} = \frac{(\text{Control ulcer index}) - (\text{test ulcer index}) \times 100}{\text{(Control ulcer index)}}
\]

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Ulcer Index (Macroscopic)</th>
<th>Curative ratio (%) (Macroscopic)</th>
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<tbody>
<tr>
<td>Control (10 ml/kg)</td>
<td>62.02 ± 2.63</td>
<td>-</td>
</tr>
<tr>
<td>Ranitidine (150 mg/kg)</td>
<td>25.01 ± 1.89&lt;sup&gt;†&lt;/sup&gt;</td>
<td>59.67</td>
</tr>
<tr>
<td>AEPO (50 mg/kg)</td>
<td>36.25 ± 1.64&lt;sup&gt;†&lt;/sup&gt;</td>
<td>41.55</td>
</tr>
<tr>
<td>AEPO (100 mg/kg)</td>
<td>1.07 ± 0.79&lt;sup&gt;†&lt;/sup&gt;</td>
<td>98.27</td>
</tr>
<tr>
<td>AEPO (150 mg/kg)</td>
<td>0.33 ± 0.18&lt;sup&gt;†&lt;/sup&gt;</td>
<td>99.47</td>
</tr>
<tr>
<td>MEPO (50 mg/kg)</td>
<td>38.12 ± 2.53&lt;sup&gt;†&lt;/sup&gt;</td>
<td>38.54</td>
</tr>
<tr>
<td>MEPO (100 mg/kg)</td>
<td>26.70 ± 1.94&lt;sup&gt;†&lt;/sup&gt;</td>
<td>56.95</td>
</tr>
<tr>
<td>MEPO (150 mg/kg)</td>
<td>0.00 ± 0.00&lt;sup&gt;†&lt;/sup&gt;</td>
<td>100.00</td>
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</table>

*Abbreviations: AEPO = Aqueous Extract of Portulaca oleracea; MEPO = Methanol Extract of Portulaca oleracea; † = Significantly different from the control group at 0.05 level of significance.*
Statistical Analysis
The mean and standard error of mean (S.E.M) were calculated for all values. Comparison between the control and experimental groups was done using one - way analysis of variance (ANOVA) with Duncan’s Multiple Range Test.

Differences were considered statistically significant at p<0.05.

RESULTS
The effects of varying doses of AEPO, MEPO and ranitidine on ulcer index and curative ratio are shown in Table 1.

On gross examination, animals pretreated with AEPO, MEPO and ranitidine showed very mild gastric lesions and sometimes no lesion at all compared to the negative control with marked gastric mucosal lesions characterized by long hemorrhagic bands with petechial lesions (Plates 1, 2, and 3).

Morphometric evaluation to assess the extent of ulceration showed that pretreatment of animals with all the treatment doses of AEPO and MEPO suppressed the ulceration induced by ethanol (80 %) as evidenced by the significant (p<0.05) dose – dependent reductions in ulcer indices relative to the negative control.

Also, animals pretreated with ranitidine significantly (p<0.05) suppressed the ulceration induced by ethanol (80 %) compared to the control.

All the treatment doses of AEPO and MEPO (as well as ranitidine) significantly (p<0.05) increased the curative ratio in a dose – dependent manner compared to the negative control. Cytoprotection were higher in animals pretreated with AEPO (100 mg/kg, 150 mg/kg) and MEPO (150 mg/kg) than ranitidine.

DISCUSSION
Gastric ulcers are due to inequality between aggressive (acid-pepsin secretion, Helicobacter pylori, bile, increased free radicals, and decreased antioxidants) and defensive factors (mucus, bicarbonate secretion, prostaglandins, blood flow and the process of restitution, and regeneration after cellular injury) of the gastric mucosa.

Different therapeutic agents including plant extracts are used to inhibit the gastric acid secretion or to boost the mucosal defense mechanism by increasing mucus production, stabilizing the surface epithelial cells or interfering with the PGs synthesis.

Ethanol is considered one of the agents that induce more intense gastric ulcers because it promotes serious disturbances in the gastric mucosa. Ethanol induced model is used to screen drugs for cytoprotection while indomethacin-induced ulcer model shows both cytoprotection and gastric acid secretion.

The results have shown that the extracts (AEPO and MEPO) and ranitidine suppressed the ulceration induced by ethanol. The anti-ulcerogenic effects of these extracts could be due to their gastric acid anti-secretory effects as we had earlier reported.
The anti-ulcerogenic effects of these extracts could so be due to their abilities to increase gastric mucosal blood flow and mucus production in the gastric lumen, increase in endogenous glutathione and prostaglandin levels and decrease of ischemia, gastric vascular permeability, acid “back diffusion,” histamine release, efflux of sodium and potassium, influx of calcium, generation of free radicals and production of leukotrienes; since it has been reported that ethanol induces ulcers by the reduction of gastric mucosal blood flow and mucus production in the gastric lumen, a decrease in endogenous glutathione and prostaglandin levels and an increase of ischemia, gastric vascular permeability, acid “back diffusion”, histamine release, efflux of sodium and potassium, influx of calcium, generation of free radicals and production of leukotrienes. Similar results were reported by in Argyreia speciosa extract treated rats.

It can be concluded that Portulaca oleracea probably has gastric anti-ulcerogenic effect which could be due to its gastric acid anti-secretory effect, which provides scientific basis to the folkloric claim of the use of the plant as a gastric sedative. The active principles that are responsible for the gastric anti-ulcerogenic effect of this plant could be from the three compounds that we had earlier isolated from this plant namely: ergosterol, lupone and tetracyclic steroid constituent of Virola surinamensis which could be due to its ulcerogenic effect which could be due to its gastric ulcerogenic effect, which provides scientific basis to the folkloric claim of the use of the plant as a gastric sedative. These results support the recommendation of the folk healers to treat acidic peptic ulcers with aqueous extracts of P. oleracea. Further detailed studies are needed to identify the active principles, mechanism of action and the necessary toxicological studies to allow the plant to be safely used in the treatment of these conditions.

REFERENCES


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