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Original Research Article

ASSESSMENT OF METHANOL LEAF EXTRACT OF *CHROMOLAENA ODORATA* FOR PROTECTION AGAINST DICLOFENAC-INDUCED GASTRIC ULCER IN RATS

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ABSTRACT

The leaf (juice, infusion and decoction) of *Chromolaena odorata* is used in Nigerian traditional medicine to promote healing of wounds. This study investigated its gastroprotective potential against diclofenac-induced ulcer in albino rats. About 500 g of dried and pulverized leaves of *C. odorata* was extracted with 90% hydromethanol (2.5 L) by cold maceration. The extract was tested at the doses of 100, 200 and 400 mg/kg against diclofenac-induced gastric ulcer in albino rats. The same doses of the extract were also used on gastric ulcer induced by combining diclofenac administration and pyloric ligation of albino rats. The results showed significant reduction of ulcer lesions in the extract-treated rats, when compared with the control. However, treatment with the extract did not cause any significant difference in the volume of gastric content and stomach acidity of the treated rats when compared with the control. The gastroprotective activity of the extract in this study was mild, ranging from 2.5 –11.5% and compared less favourably to that of omeprazole (43.4 – 47.5%), which was used as the standard drug.

Keywords: *Chromolaena odorata*, diclofenac, gastroprotection, ulcer

INTRODUCTION

Among the most common pathologies requiring pharmacological treatment is gastrointestinal injury induced by the non-steroidal anti-inflammatory drugs (NSAIDs) (Onoja *et al.*, 2021). Such injury manifests mainly as ulceration of the gastric mucosa, but in severe cases it is complicated by bleeding and perforations. Diclofenac is a popular member of the NSAIDs and the drug is widely used clinically for alleviating both acute and chronic pain. However, gastrointestinal insult due to diclofenac is a serious clinical challenge, causing a heavy burden on the global health care system (Ramirez-Alcantara *et al.*, 2009). Studies have reported ultrastructural gastric surface epithelial damage, endoscopic gastroduodenal subepithelial haemorrhages and erosions, intestinal ulceration, progressive increase in epithelial permeability and marked increase in the number of enteric Gram-negative bacteria following administration of diclofenac (Reuter *et al.*, 1997; Hawkins & Hanks, 2000; Aycan *et al.*, 2018). A highly acidic gastric environment appears to favour the migration of non-ionized lipophilic diclofenac molecules into the epithelial cells. At

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the surface, they are dissociated into ions, thereby trapping hydrogen ions and inducing mucosal damage (Khan & Khan, 2013).

In order to ameliorate the gastrointestinal effects of diclofenac, the drug is usually administered simultaneously with an antiulcer agent, especially in patients with history of peptic ulcer and /or reflux esophagitis, and when prolonged therapy with diclofenac is needed. Orthodox medicine no doubt, plays an invaluable role in general health care delivery, however, adverse effects, cost of medications and resistance to conventional drugs have propelled a growing interest in alternative medicine. Furthermore, the use of herbal preparations for the treatment of various disease conditions justifies the study of this plant in the management of gastric ulcer (Zabidi *et al*., 2012, Balan *et al*., 2014, Malfertheiner *et al*., 2017).

The plant *Chromolaena odorata* (*C. odorata*; family: *Asteraceae*) is popular herbal remedy in treating wounds, burns and skin infections (Phan *et al*., 2000). Fresh leaves of the plant are squeezed and the juice applied on wounds to facilitate healing (Vital & Windell, 2009), while infusion of the leaf is taken orally to relieve heart burn and stomach ache (Phan *et al*., 2000). In addition, decoction of the leaf is used as a cough remedy and as an ingredient with lemon grass and guava leaves for the treatment of malaria (Vaisakh & Pandey, 2011). Although *C. odorata* has not been well established in promoting cytoprotection towards gastric mucosa, its ability to stimulate expression of essential proteins such as endogenous prostaglandins as well as increase proliferation of epithelial cells suggest its potential to enhance the healing process of gastric mucosa (Nur Janah *et al*., 2006). This study seeks to investigate the gastroprotective potential of methanol leaf extract *C. odorata* in a model of diclofenac-induced gastric ulcer in albino rats.

MATERIALS AND METHODS

PLANT COLLECTION AND EXTRACTION

Fresh leaves of *C. aodorata* were collected from Umudike in Abia State, Nigeria. The plant was identified by the Department of Botany, Michael Okpara University of Agriculture, Umudike (MOUAU). A voucher specimen with identification number: MOUAU/VPP/2023/09 is deposited in the herbarium of the Department of Veterinary Physiology and Pharmacology, MOUAU. The leaves were air-dried and reduced to course powder using an electric blender, 500 g of the coarse powder was extracted with 2.5 L of 90% hydromethanol, by cold maceration method. The extract was oven-dried (40 °C) after concentration in a rotary evaporator, and stored as methanol extract of *C. odorata* (MECO), at 4 °C until time of use (Madubuike *et al*., 2012).

PRELIMINARY PHYTOCHEMICAL ANALYSIS OF MECO

A qualitative phytochemical analysis of the extract was done by following the methods of Harbourne (1991) and Trease & Evans (1996).

ANIMALS

Nine-week old Wistar rats weighing 124.5 ± 4.21 g were procured from the Experimental Animal House of the Department of Veterinary Physiology and Pharmacology, MOUAU. They were housed in stainless steel rat cages and fed *ad libitum* with standard pelleted rat chow (Vital Finisher®).

All the experimental protocols complied with OECD guidelines for handling experimental animals and received the approval of the Research Ethical Committee of the College of Veterinary Medicine, MOUAU (Approval No. MOUAU/CVM/REC/202307).

ACUTE TOXICITY TEST

An oral acute toxicity testing of MECO was carried out following the method of Anaga *et al*., (2012). Thirty-five mature rats of either sexes were randomly assigned to 7 groups of 5 rats each and fasted for 12 hours. Group 1 served as control and received 5 ml/kg of distilled water, while the remaining 6 groups were treated with varying doses (500-5000 mg/kg) of MECO. The rats were observed for 24 hours for signs of acute toxicity and death.

EFFECT OF TREATMENT WITH MECO ON DICLOFENAC-INDUCED GASTRIC ULCER IN RATS

The effect of treatment with varying doses of MECO on diclofenac-induced gastric ulcer in rats was determined following the method of Cioli *et al*, (1979), as contained in Onyeike & Madubuike (2024). Thirty mature Wistar rats were randomly assigned into 5groups (n = 6), fasted for 36 hours and treated as follows: Group 1 served as control and received 5 ml/kg of distilled water; Group 2 received the standard anti-ulcer drug, omeprazole (20 mg/kg), while Groups 3-5 were dosed with 100, 200 and 400 mg/kg of MECO, respectively. All the treatments were administered by gastric gavage. One hour later, all the rats were dosed orally with 100 mg/kg of diclofenac. Six hours post diclofenac administration; the rats were sacrificed by cervical dislocation. The stomachs were removed, dissected along the greater curvature and washed with distilled water. Ulcer lesions were counted with the aid of magnifying glass (x 10 magnification), and scored on the basis of intensity as follows; 0 = no ulcer, 1= Spot ulcer, 2= short haemorrhagic streak, 3= Long haemorrhagic streak, 4 = widespread lined injury, 5 = Perforations. The ulcer index and the percentage ulcer inhibition were calculated as follows:

Ulcer index (U_I) = (U_N + U_S + U_P) X 1/10

Where, U_N = average number of ulcer; U_S = average severity score; U_P = percentage of animal with ulcer.

The percentage ulcer inhibition (PUI) was calculated using the formula: PUI = UI (control) – UI (treated) / UI (control) x 100.

PRELIMINARY PHYTOCHEMICAL ANALYSIS

The qualitative phytochemical analysis of MECO showed the presence of flavonoids, tannins, saponins, carbohydrates and glycosides in the extract.

TABLE I: ANTI-ULCER ACTIVITY OF MECO ON DICLOFENAC-INDUCED GASTRIC ULCER IN RATS

Treatment Group	Number of Ulcer	Ulcer score	Ulcer index	% Protection
Distilled Water-5 ml/kg	8.00 ± 0.44^{a}	12.67 ± 0.49^{a}	12.07	-
Omeprazole-20 mg/kg	3.50 ± 0.81^{c}	4.83 ± 1.19^{c}	6.83	43.4
MECO-100 mg/kg	7.00 ± 0.36^{ab}	10.67 ± 0.61^{a}	11.77	2.5
MECO-200 mg/kg	7.00 ± 0.25^{ab}	7.67 ± 0.33^{b}	11.47	5.0
MECO-400 mg/kg	5.16 ± 0.98^{bc}	7.83 ± 1.30^{b}	11.30	6.4

Note: Different superscript letters along column show significant differences at $p < 0.05$

PYLORIC LIGATION/DETERMINATION OF SECRETORY PARAMETERS.

The method of Shay *et al* (1945), as presented in Onoja *et al*. (2018) was adopted for this investigation, with slight modifications. Thirty mature Wistar rats were fasted for 36 h and assigned into 5 groups of 6 rats per group. Rats in Group 1 (control) were given 5 ml/kg of distilled water; Group 2 received the standard anti-ulcer drug, omeprazole (20 mg/kg), while Groups 3-5 were dosed with 100, 200 and 400 mg/kg of MECO, respectively. All the treatments were administered orally. One hour later, the rats were orally dosed with 100 mg/kg of diclofenac and 1 h later, they were anesthetized via ketamine induction. A laparotomy was carried out on each rat and its pyloric sphincter was loosely ligated to avoid occluding the blood vessels. The abdominal walls were carefully closed using interrupted sutures. Six hours post-operation, the rats were euthanized using chloroform and their stomach exteriorized. The entire stomach content of each rat was collected in a calibrated tube and centrifuged at 2000 r.p.m. for 10 minutes. The supernatant was titrated against 0.1 M NaOH to determine acidity.

STATISTICAL ANALYSIS

Data generated from the study were presented in tables as means ± standard error of the mean (S.E.M.). They were subjected to one-way analysis of variance (ANOVA), using SPSS version 20. The variant means were separated by post-hoc least significant difference (LSD) and $p < 0.05$ was regarded significant.

RESULTS

PLANT EXTRACTION

The extraction process yielded 7.4% (w/w). The extract was pasty, dark greenish and has a pleasant aroma.

ACUTE TOXICITY TEST

The oral acute toxicity testing of MECO did not record death or sign of acute toxicity at the doses (500-5000 mg/kg) tested.

EFFECT OF TREATMENT WITH MECO ON DICLOFENAC-INDUCED ULCER IN RATS

The extract at the doses tested did not cause any significant difference in the number of ulcers recorded in the groups treated with 100 and 200 mg/kg of MECO, when compared with the control. However, 200 and 400 mg/kg of the extract significantly reduced the ulcer severity score from 12.67 ± 0.49 in the control to 7.67 ± 0.33 and 7.83 ± 1.30, respectively in the treated rats. Treatment with omeprazole (reference drug) caused significant decrease in both the number of ulcers and the ulcer severity score, when compared with the control and the extract-treated groups, giving a percentage protection of 43.4 against 2.5, 5.0 and 6.4% protection exhibited by 100, 200 and 200 mg/kg of the extract, respectively (Table I).

EFFECT OF TREATMENT WITH MECO ON DICLOFENAC-INDUCED ULCER IN PYLORIC LIGATED RATS

The extract at all the doses tested caused significant decrease in both the number of ulcers and the ulcer severity scores in the treated rats, when compared with the control. However, the effects of 20 mg/kg of omeprazole (reference drug) with respect to both parameters were significantly ($p < 0.05$) higher than those of the extract. The percentage protection by omeprazole was 47.5, against 4.7, 8.9 and 11.5% evoked by 100, 200 and 400 mg/kg of the extract, respectively (Table II).

TABLE II: EFFECT OF TREATMENT WITH MECO ON DICLOFENAC-INDUCED ULCER IN PYLORIC LIGATED RATS

Treatment group	Number of Ulcer	Ulcer score	Ulcer index	% Protection
5 ml/kg Distilled Water	11.42 ± 0.72^{a}	15.42 ± 1.25^{a}	12.64	-
Omeprazole-20 mg/kg	2.71 ± 0.56^{d}	3.57 ± 0.81^{d}	6.63	47.5
MECO-100 mg/kg	9.42 ± 0.57^{b}	11.00 ± 0.87^{b}	12.04	4.7
MECO-200 mg/kg	6.42 ± 0.20^{c}	8.71 ± 0.86^{bc}	11.51	8.9
MECO-400 mg/kg	5.57 ± 0.48^{c}	8.00 ± 0.84^{c}	11.34	11.5

Note: Different superscript letters along column show significant differences at $p < 0.05$

With respect to the ulcer-related secretory parameters, there was no significant difference in the volume of gastric content, pH, total acidity and free acidity of the extract-treated groups, when compared with the control. However, there was significant reduction of the total acidity and free acidity from 2.32 ± 0.26 and 0.44 ± 0.08 in the control to 1.16 ± 0.33 and 0.25 ± 0.04, respectively in the omeprazole-treated group (Table III).

Mechanism of anti-ulcer drugs include: neutralization of gastric acid, histamine (H_2) antagonism and inhibition of H/K ATPase (the proton pump), which is the final step in the acid secretory pathway (Rangs *et al*., 2006). Other agents function by enhancing production of endogenous cytoprotective substances such as; mucus, bicarbonate and prostaglandins (Harvey & Champe, 2009; Onoja *et al*., 2018).

TABLE III: DICLOFENAC-INDUCED ULCER SECRETORY PARAMETERS OF PYLORIC LIGATED RATS PRETREATED WITH MECO

Treatment Group	Volume of gastric content (ml)	pH	Total acidity	Free acidity
Distilled Water-5 ml/kg	1.68 ± 0.23^{ab}	2.20 ± 0.20	2.32 ± 0.26^{a}	0.44 ± 0.08^{b}
Omeprazole-20 mg/kg	1.34 ± 0.22^{b}	3.00 ± 0.44	1.16 ± 0.33^{b}	0.25 ± 0.04^{c}
MECO-100 mg/kg	1.78 ± 0.08^{ab}	2.40 ± 0.24	2.54 ± 0.15^{a}	0.60 ± 0.07^{ab}
MECO-200 mg/kg	1.70 ± 0.13^{ab}	2.60 ± 0.24	2.80 ± 0.14^{a}	0.64 ± 0.09^{ab}
MECO-400 mg/kg	1.90 ± 0.09^{a}	2.60 ± 0.24	2.74 ± 0.15^{a}	0.38 ± 0.11^{bc}

Note: Different superscript letters along column show significant differences at $p < 0.05$

DISCUSSION

The absence of mortality or acute toxicity signs in the oral acute toxicity test, even at the highest dose of 5000 mg/kg implies that the LD_{50} of MECO is greater than 5000 mg/kg. The extract can thus be described as practically non-toxic (Anaga *et al*., 2012; Uzoma & Madubuike 2024).

Diclofenac-induced ulcer in rodents is a standard model for screening potential anti-ulcer drugs. Being a non-steroidal anti-inflammatory drug, diclofenac causes gastrointestinal ulceration via inhibition of cyclooxygenase, thereby by limiting prostaglandin synthesis. Decreased prostaglandin levels leads to impairment of mucus production and bicarbonate secretion, thus exposing the gastric mucosa to direct action of gastric acid (Trabadel *et al*., 2008).

In this study diclofenac successfully induced ulceration of the rats' gastric mucosa, evidenced by the observed ulcers lesions.

The mild anti-ulcer activity exhibited by MECO in this study could have resulted from any of these mechanisms, except inhibition of the proton pump, since the extract did not cause any significant change in the stomach acidity of the treated rats, when compared with the control. It has been suggested that the rich polysaccharides content of *C. odorata* acts as barrier against excessive pH changes and thus protects the gastric mucosa (Nur Jannah *et al*., 2006). In addition, the extract could have caused increased production of prostaglandins, since its capacity to stimulate the expression of essential proteins is documented in literature (Phan *et al*., 2000). Prostaglandin stimulates increased bicarbonate and mucus synthesis and enhance mucosal blood flow, hence, maintaining mucosal repair and integrity of the mucosa (Onoja *et al.*, 2018). Furthermore, *C. odorata* is known to increase proliferation of epithelial cells (Nur Jannah *et al*., 2006) and may be involved in the healing process of gastric mucosa.

The preliminary phytochemical analysis of MECO also reveal rich content of carbohydrates and glycosides, among other bioactive phyto-constituents which may have contributed to the observed gastroprotective activity of the extract.

The anti-ulcer activity of omeprazole which was used as reference drug in this study was significantly higher than that of MECO, throughout the study. Being a proton pump inhibitor, omeprazole was able to cause significant reduction of stomach acid in the treated rats.

CONCLUSION

The results of this study show mild gastroprotective effect of methanol leaf extract of *C. odorata* against diclofenac-induced gastric ulcer in rats. Fractionation of the crude extract could result in greater anti-ulcer activity and it is therefore recommended as a further study.

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CONFLICT OF INTEREST

The authors declare no conflict of interest regarding this work.

REFERENCES

Anaga, A.O., Asuzu, I.U., Shetty, S.N. & Anika, S.M. (2012). *Laboratory Manual of Pharmacology and Toxicology* (2nd ed.). Enugu, Nigeria: Fourth Dimension Publishing Co. Ltd.

Aycan, I.O., Eplek, O., Akkaya, B., Kirac, E., Tuzcu, H., Kaya, S., Coskunfirat, N. & Aslan, M. (2018). Diclofenac induced gastrointestinal and renal toxicity is alleviated by thymoquinone treatment. *Food and Chemical Toxicity*, 118, 795-804.

Balan, T., Mohd-Sani, M.H., Suppiah, V., Mohtarrudin, N., Suhaili, Z., Ahmad, Z. & Zakaria, Z.A. (2014). Antiulcer activity of *Muntingia calabura* leaves involves the modulation of endogenous nitric oxide and non-protein sulfhydryl compounds. *Pharmaceutical Biology*, 52(4), 410-418

Cioli, V., Putzolu, S., Rossi, V. & Corradino, C. (1979). The role of direct tissue contact in the production of gastrointestinal ulcers by anti-inflammatory drugs in rats. *Toxicology and Applied Pharmacology*, 50, 283-289.

Harbourne, J.B. (1991). *Phytochemical methods*. A guide to modern techniques of plant analysis (2nd ed.). London: Chapman and Hall.

Harvey, R.A. & Champe, P.C. (2009). *Lippincott's Pharmacology* (4th ed.). New Delhi: Wolters Kluwer Pvt. Ltd.

Hawkins, C. & Hanks, G.W. (2000). The gastrointestinal toxicity of non-steroidal anti-inflammatory drugs: a review of literature. *Journal of Pain Symptoms and Management*, 20, 140-151.

Khan M.I. & Khan M.R. (2013). Gastroprotective potential of *Dalbergiasissoo* Roxb stem bark against diclofenac-induced gastric damage in rats. *Osong Public Health and Research Perspectives*, 4(5), 271-277.

Madubuike, K.G., Ezeja, M.I. & Ezeigbo, I.I. (2012). Antidiarrhoeal activity of *Pterocarpus Santalinoides* leaf extract in mice. *Continental Journal of Animal and Veterinary Research*, **4**(1), 1-6.

Malfertheiner, P., Megraud, F., O'morain, C.A., Gisbert, J.P., Kuipers, E.J., Axon, A.T. & Hunt, R. (2017). Management of Helicobacter pylori infection – The Maastricht V/Florence consensus report. *Gut*, 66(1), 6-30.

Nur Jannah, M.H., Mohamaad, A.A, Sidik, K. & Salmah, I. (2006). Cytoprotective effects of honey in combination with aqueous and ethanol extracts from *Chromolaena odorata* L. (*Eupatorium odoratum* L.) in rats. *Journal of University of Malaya Medical Centre*, 9(1), 7-13.

Onoja, S.O., Chinagorom, O., Ikpa, C.B.C., Madubuike, K.G., Ezeigbo, I. I., Ijioma, S.N., Anaga, A.O. & Ezeja, M.I. (2018). Gastroprotective effects of methanol extract of *Eremomastax speciosa* leaf harvested in Southern part of Nigeria in rat. *The European Biotechnology Journal*, 2(4), 200-208.

Onoja, S.O., Ezeh, G.C., Udeh, N.E., Ikpa, C.B.C., Ugoji, D., Ukwueze, J.I., Madubuike, K.G., Aba, P.E. & Ezeja, M.I. (2021). Anti-ulcer property of methanol fraction of *Callichilia subsessilis* leaf extract in albino rats. *Notulae Scientia Biologicae*, 13(1), 10886.

Onyeike, K.M. & Madubuike, K.G. (2024). Comparison of the analgesic and ulcerogenic effects of five brands of diclofenac sold in Nigerian market. *Journal of Sustainable Veterinary and Allied Sciences*, 6(1), 34-38

Phan, T.T., Allen, J., Hughes, M.A., Cherry, G. & Wojnarowska, F. (2000). Up regulation of adhesion complex proteins and fibronectin by human keratinocytes treated with an aqueous extract from the leaves of *Chromolaena odorata*

(Eupolin). *European Journal of Dermatology*, 10(7), 522-225.

Ramirez-Alcantara, V., LoGuidice, A. & Boelsterli, U.A. (2009). Protection from diclofenac- induced small intestinal injury by the JNK inhibitor SP600125 in a mouse model of NSAID-associated enteropathy. *American Journal of Physiology of Gastrointestinal and Liver Physiology*, 297, 990-998.

Rang, H.P., Dale, M.M., Ritter, J.M., Flower, R.J. (2007). Rang and Dale's pharmacology (6th Ed.). Philadelphia: Churchill Livingstone/ Elsevier.

Reuter, B.K., Davies, N.M. & Wallace, J.I. (1997). Nonsteroidal anti-inflammatory drug enteropathy in rats: role of permeability, bacteria, and enterohepatic circulation. *Gastroenterology*, 112, 109-117.

Shay, H., Komarov, S., Fels, S., Meranze, D., Gruenstein, M. & Simplet, H.A. (1945). A simple method for the uniform production of gastric ulceration in rat. *Gastroenterology*, 5, 43-61.

Trabadel, C., Sanchez-Fidalgo, S., Mino, P., Berenguer, B., Quilez, A., de la Puerta, R. & Martin, M.J. (2008). Gastroprotective effects of *Piper carpunya* against diclofenac-induced gastric lesions in rats. *Pharmaceutical Biology*, 46, 829-837.

Trease, G.E. & Evans, W.C. (1996). *Pharmacognosy* (13th ed.). Bailliere Tindall.

Uzoma, C.C. & Madubuike, K.G. (2024). Toxicity studies of methanol leaf extract of *Momordica cissiodes* in Wistar rats. *Journal of Veterinary and Biomedical Sciences*, 6(1), 1-15.

Vaisakh, M.N. & Pandey, A. (2011). The invasive with healing properties: A review on *Chromolaena odorata*. *International Journal of Pharmaceutical Sciences and Research*, 3(1), 80-83.

Vital, P.G. & Windell, L.R. (2009). Antimicrobial activity and cytotoxicity of *Chromolaena odorata* (L.F.) King and Robbinson and Uncariaperrottetii (A. Rich) Merr. Extracts. *Journal of Medicinal Plants Research*, 3(7), 511-518.

Zabidi, Z., Zainulddin, W.W., Mamat, S.S., Din, S.S., Kamisan, F.H., Yaya, F. & Somchit, M.N. (2012). Antiulcer activity of methanol extract of *Melastomamala bataricum* leaves in rats. *Medical Principles and Practice*, 21(5), 501-503.