Gastric antisecretory and cytoprotective effects of leaf extracts of *Amaranthus tricolor* Linn. in rats

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**Objective:** The present study was aimed to evaluate the antiulcer activity of leaf extracts of *Amaranthus tricolor* Linn. (Amaranthaceae) in rats.

**Methods:** The effects of *A. tricolor* leaves on gastric secretion and the effect of gastric cytoprotection were evaluated using five different models of gastric ulcers: acetic acid-induced, pylorus ligation-induced, ethanol-induced, indomethacin-induced and ischemia-reperfusion-induced gastric ulcers. The different extracts, namely, ethanolic extract (EAT), petroleum ether extract (PEAT), chloroform extract (CAT) and ethyl acetate extract (EAAT) of *A. tricolor* leaves were administered at a dose of 200 mg/kg per oral (p.o.).

**Results:** The acute oral toxicity study revealed that all the extracts were safe up to 2 000 mg/kg, p.o.; hence one-tenth of this dose was selected for evaluation of antiulcer activity. The EAT and EAAT (200 mg/kg, p.o.) showed gastric ulcer-healing effect in acetic acid-induced chronic gastric ulcers. The EAT and EAAT inhibited gastric secretion in pylorus-ligated rats and showed gastric cytoprotective effect in ethanol-induced and indomethacin-induced gastric ulcers, while PEAT and CAT showed no significant antiulcer effect.

**Conclusion:** The leaf extracts of *A. tricolor* are found to possess very good antiulcer property in the experimental animal models of gastric ulcers which is consistent with the literature report in folk medicine.

**Keywords:** *Amaranthus*; plant extracts; stomach ulcer; cytoprotection; antiulcer agents; rats

Peptic ulcer is an excoriated area of the gastric or duodenal mucosa caused by action of the gastric juice. It is a chronic and recurrent disease, and is the most predominant of the gastrointestinal diseases. Even though the etiology of gastric ulcer is still debated, it is accepted that ulcers are caused due to net imbalances of the mucosal offensive and defensive factors. Research advances have
offered new sights in the treatment and prevention of gastric or duodenal ulcerations by measures directed at strengthening the mucosal defense systems rather than by attenuating aggressive acid peptic factors responsible for the induction of ulcers.

In spite of the progress in conventional chemistry and pharmacology in producing effective drugs, the plant kingdom might provide a useful source of new antiulcer compounds for development as pharmaceutical entities or alternatively as simple dietary adjuncts to existing therapies[4]. Presently, plenty of strong evidence suggests that the consumption of fruits and vegetables can prevent many chronic non-communicable diseases[4]. Previous reports on the incidence of gastric ulcers in South Asian population indicated that the incidence may be lower due to the type of food consumed by people of this region. One of the foods that are speculated to protect against ulcers is leaves of *Amaranthus tricolor* [5].

*A. tricolor* (Amaranthaceae) is commonly known as “red amaranth” or “Joseph’s coat” cultivated throughout South-East Asia and many tropical countries mainly for its edible leaves[5]. *A. tricolor* is one of the traditional medicines used in many folk claims and the plant has been extensively used in Ayurveda and Siddha for treating menorrhagia, diarrhea, dysentery, haemorrhagic colitis, bowel hemorrhages, cough and bronchitis. It is also used externally as an emollient poultice or a mouth wash to treat ulcerated conditions of the throat and mouth[6]. Apart from this, the leaves of the plant have been reported to possess wide range of pharmacological activities, like antitumor effect[6], hepatoprotective activity[6], and inhibitory effect on cobra venom[7]. Betacyanins, the coloring pigments in *A. tricolor*, have been reported to possess antioxidant activity[8-11]. The leaves of *A. tricolor* have been used as external inflammations, as a diuretic, and as a treatment for bladder distress[11].

Phytochemical studies on *A. tricolor* succeeded in the isolation of the antioxidant betacyanins and heteropolysaccharides from the plant[12]. Similarly, Sarkar *et al*[13] revealed that the aequous extract inhibits the proliferation of a liver cancer cell line (HepG2), a breast cancer cell line (MCF-7), and a colon cancer cell line (CaCO-2). Furthermore, the whole plant of *A. spinosus*, a synonym of *A. tricolor* has been reported to possess antulcer activity[17]. However, none study was conducted scientifically to prove that leaves of *A. tricolor* possess any effect on gastric ulcers. Hence, the present study was undertaken to evaluate the protective effect of leaves of *A. tricolor* on experimental gastric ulcers in rats.

1 Materials and methods

1.1 Drugs and chemicals Ranitidine (Torrent Pharmaceuticals, Ahmedabad, India), misoprostol (Zydus Cadila, Ahmedabad, India) and the solvents used for extraction process and chemicals used for phytochemical analysis were of analytical grade and procured from local firms.

1.2 Collection and identification of the plant material

The leaves of *A. tricolor* were collected from local areas of Bangalore, India in January 2009. Botanical identification was done by Prof. Balakrishna Gowda, Gandhi Krishi Vignan Kendra (GKVK), Bangalore, India.

1.3 Extraction and preliminary phytochemical screening

The leaves of *A. tricolor* were shaded dried and reduced to coarse powders in a mechanical grinder and passed through sieve No. 40. The powdered materials obtained were then subjected to extraction by cold maceration using rectified spirit (90%) for 7 d. The extract was filtered and concentrated in rotary evaporator under reduced pressure to yield a thick green ethanolic extract (EAT). The crude extract obtained was thus partition-fractionated with 1 : 1 (v/v, volume ratio) of petroleum ether and ethanol (50%), and the mixture was shaken vigorously and kept for about 30 min to make the two layers separate. The upper layer consisted of petroleum ether and it was removed and concentrated in a rotary evaporator to obtain petroleum ether extract (PEAT). The same procedure was repeated with the bottom layer without PEAT part by using equivalent volume of chloroform and ethyl acetate.
to obtain chloroform extract (CAT) and ethyl acetate extract (EAAT), respectively. To determine the chemical constituents, the extracts obtained were thus subjected to phytochemical analysis[38].

1.4 Experimental animals Albinó Wistar rats weighing between 200 and 230 g were used. Institutional Animal Ethics Committee approved the experimental protocol. Rats were maintained under standard conditions in an animal house approved by Committee for the Purpose of Control and Supervision on Experiments on Animals.

1.5 Acute toxicity studies Acute oral toxicity was determined by using female, nulliparous and non-pregnant mice weighing 18 to 22 g. The animals were fasted for 3 h prior to the experiment. Up and down procedure OECD guideline no. 425 was adopted for toxicity studies[29]. Animals were administered with single dose of extract and observed for their mortality during 48-hour study period (short term) toxicity.

1.6 Antulcer activity

1.6.1 Acetic acid-induced chronic gastric ulcers The rats were fasted for 24 h prior to the experiment. Under light ether anesthesia, the abdomen was opened by midline incision below the xiphoid process and the stomach was exposed. Glacial acetic acid (0.05 mL) was applied onto the serosal surface using cylindrical mold (6 mm), which was allowed to remain there for 60 s. The acid solution was then removed by rinsing the mold with 0.9% saline to prevent possible damage to the surrounding tissues close to the point of application. The stomach was placed back carefully and the abdominal wall was closed by interrupted sutures. The rats were treated with different leaf extracts of A. tricolor (200 mg/kg, per oral (p.o.)) or ranitidine (50 mg/kg, p.o.) once daily for 10 d after induction of ulcer while the control group received 0.5% carboxy methyl cellulose (CMC) in water as vehicle (n = 6 in each group). Rats were sacrificed 6 h after the last dose; the stomach was removed and was cut open along the greater curvature[31]. The stomach samples were scanned using a computer scanner and the total mucosal area and total ulcerated area were measured using public domain image processing and analysis program developed at the National Institutes of Health, USA. The PC version of the program was downloaded free from Scion (Scion Image for Windows, Release Beta 4.0.2). The scale was set at 6.1 pixels per millimeter[21]. The ulcer index was determined using the following formula[25], where $X = \frac{\text{total ulcerated area}}{\text{total mucosal area}}$.

$$\text{Ulcer index} = \frac{10}{X}$$

The stomach samples were subsequently processed for histological examination. Three indices namely regenerated surface epithelial width, capillary density in the scar tissue and volume of collagen content were selected to reflect the rate and quality of ulcer healing[25].

1.6.2 Pylorus ligation-induced gastric ulcers The rats were fasted for 36 h before pylorus ligation. They received water ad libitum and were placed individually in cages to avoid coprophagy and cannibalism. Normal saline (1 mL for each rat, p.o.) was administered twice daily to all the rats. Under light ether anesthesia, the pyloric portion of the stomach was ligated. The different leaf extracts of A. tricolor (200 mg/kg, intraduodenally) or ranitidine (50 mg/kg, intraduodenally) were administered immediately after pylorus ligation (n = 6 in each group); 6 h after pylorus ligation, rats were sacrificed[21, 25]. The stomach was isolated and the content of the stomach was used for estimation of free and total acidity. The contents were centrifuged, filtered and subjected to titration for estimation of free and total acidity. 1 mL of centrifuged and filtered gastric secretion was titrated against 0.1 mol/L sodium hydroxide using Topfer’s reagent as indicator for determination of free acidity and 1% phenolphthalein as indicator for combined acidity. The sum of the two titrations was total acidity[27]. The ulcer index was determined using the same method as mentioned above.

1.6.3 Ethanol-induced gastric ulcers Albinó Wistar rats were fasted for 36 h before administration of ethanol (5 mL/kg, p.o.). Misoprostol (200 μg/kg, p.o.) or different leaf extracts of A. tricolor (200 mg/kg, p.o.) were administered 1 h before ethanol administration (n = 6 in each group)[37, 38]. The rats were sacrificed 1 h after ethanol administration and ulcer index was determined as mentioned above.

1.6.4 Indomethacin-induced gastric ulcers The ulcers were induced by administering indomethacin (20 mg/kg, p.o.) to the rats fasted for 36 h. Misoprostol (200 μg/kg, p.o.) or different leaf extracts of A. tricolor (200 mg/kg, p.o.) was administered 30 min before indomethacin administration (n = 6 in each group) and after 4 h, all rats were sacrificed, the stomachs were isolated and ulcer index was determined[39].

1.6.5 Ischemia-reperfusion-induced gastric ulcers Albinó rats were treated with ranitidine (50 mg/kg, p.o.) or different leaf extracts of A. tricolor (200 mg/kg, p.o.) for 2 d (n = 6 in each group). The rats were fasted for 24 h after last dose and were then anesthetized with ketamine (100 mg/kg, intramuscular injection (i.m.)) and xylazine (16 mg/kg, i.m.). Laparotomy was performed and the esophageal and pyloric ends of the stomach were clamped using bull dog clips. The celiac artery was then clamped for 30 min at a point 0.5 cm distal from the branch to aorta followed by reperfusion for 20 min[39]. The rats were then sacrificed and ulcer index was calculated.

1.7 Statistical analysis All the values were expressed...
as mean ± standard error of mean. Data of ulcer index were analyzed by non-parametric analysis of variance (ANOVA) while one-way ANOVA was used for statistical comparison of other results, followed by Dunnet-t comparison test. Differences between means were considered significantly different when P value was less than 0.05 using Graph-Pad Prism version 5.04 for Windows (GraphPad Software, San Diego, California, USA).

2 Results

2.1 Preliminary phytochemical screening The percentage yield of EAT, PEAT, CAT, and EAAT was found to be 15.1%, 6.9%, 5.4% and 4.1% (all in weight ratio), respectively. Preliminary phytochemical screening of the leaf extracts revealed the presence of steroids in PEAT and CAT; EAAT contained proteins, alkaloids, saponins, glycosides and flavonoids; EAT showed the presence of carbohydrates, proteins, saponins, flavonoids, tannins and glycosides.

2.2 Acute oral toxicity In acute oral toxicity study, no mortality was observed after treatment with the highest tested dose (2000 mg/kg, p.o.) of all the leaf extracts.

2.3 Antiulcer activity 2.3.1 Ulcer healing in acetic acid-induced chronic gastric ulcer model The EAT and EAAT groups showed a significant reduction in ulcer index when compared with the control (P<0.01). The EAT group was the most potent among all the extracts; it has shown 80% decrease in ulcer index when compared with the control. PEAT and CAT did not show any significant effect on ulcer index (Figure 1).

Sections of ulcerated area revealed that there was a significant increase in regenerated surface epithelial width after treatment with EAT, EAAT and ranitidine (P<0.01) while PEAT and CAT did not show any significant effect. A significant increase in capillary density in scar tissue was observed after treatment with EAT, EAAT and ranitidine compared with the control. The collagen content in the ulcerated tissue was significantly increased by EAT, EAAT and ranitidine, and maximum effect was observed in EAT. No significant difference in surface epithelium in scar tissue was observed in any of the treatment groups, including the ranitidine-treated group (Table 1).

![Figure 1 Ulcer index of acetic acid-induced chronic gastric ulcers in rats](image)

All the values are presented as mean±standard error of mean, n=6. * P<0.05, ** P<0.01, vs vehicle control group. CMC: carboxy methyl cellulose; EAT: ethanolic extract; PEAT: petroleum ether extract; CAT: chloroform extract; EAAT: ethyl acetate extract.

Table 1 Effects of Amaranthus tricolor L. on acetic acid-induced chronic gastric ulcers in rats

<table>
<thead>
<tr>
<th>Group</th>
<th>n</th>
<th>Regenerated glandular epithelium width (μm)</th>
<th>Capillary density in 196 mm²</th>
<th>Volume of collagen content (μmol)</th>
<th>Surface epithelium (μm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vehicle control</td>
<td>6</td>
<td>238±6</td>
<td>2.95±0.35</td>
<td>0.09±0.21</td>
<td>116.50±9.50</td>
</tr>
<tr>
<td>Ranitidine</td>
<td>6</td>
<td>315±3**</td>
<td>4.65±0.45**</td>
<td>0.15±0.02**</td>
<td>125.16±5.10</td>
</tr>
<tr>
<td>EAT (200 mg/kg, p.o.)</td>
<td>6</td>
<td>360±5**</td>
<td>5.90±0.19**</td>
<td>0.19±0.01**</td>
<td>120.33±8.70</td>
</tr>
<tr>
<td>PEAT (200 mg/kg, p.o.)</td>
<td>6</td>
<td>275±6</td>
<td>2.85±0.26</td>
<td>0.06±0.01</td>
<td>112.85±3.20</td>
</tr>
<tr>
<td>CAT (200 mg/kg, p.o.)</td>
<td>6</td>
<td>298±4</td>
<td>3.29±0.58</td>
<td>0.08±0.05</td>
<td>109.13±5.60</td>
</tr>
<tr>
<td>EAAT (200 mg/kg, p.o.)</td>
<td>6</td>
<td>385±3**</td>
<td>6.05±0.22**</td>
<td>0.17±0.07**</td>
<td>120.35±8.50</td>
</tr>
</tbody>
</table>

** P<0.01, vs vehicle control group. CMC: carboxy methyl cellulose; EAT: ethanolic extract; PEAT: petroleum ether extract; CAT: chloroform extract; EAAT: ethyl acetate extract; p.o.: per oral.
2.3.2 Pylorus ligation-induced gastric ulcer model

EAT, EAAT and ranitidine groups showed significant reduction in total acidity, free acidity and ulcer index when compared with the control (P<0.01). None of the treatments produced any significant effect on total hexoses and peptic content. EAT and EAAT produced a significant increase in mucin content (P<0.05). The ranitidine treatment produced a significant increase in total protein content (P<0.05), but no significant effect on mucin content (Table 2).

2.3.3 Ethanol-induced and indomethacin-induced gastric ulcer models EAT, EAAT and misoprostol groups showed a significant reduction (P<0.05 or P<0.01) in ulcer index in ethanol-induced and indomethacin-induced gastric ulcers when compared with the control (Table 3).

| Table 2 | Ulcer index, free acidity, total acidity, total hexoses, total proteins, mucin content and peptic activity in pylorus-ligated rats
<table>
<thead>
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</tr>
</thead>
<tbody>
<tr>
<td>Group</td>
<td>n</td>
<td>Ulcer index</td>
<td>Free acidity (mmol/h)</td>
<td>Total acidity (mmol/h)</td>
<td>Total hexoses (mg/mL)</td>
<td>Total proteins (mg/mL)</td>
<td>Mucin content (µg/g)</td>
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<tr>
<td>Vehicle control (0.5% CMC 10 mL/kg, i.d.)</td>
<td>6</td>
<td>0.21±0.23</td>
<td>0.67±0.46</td>
<td>1.08±0.47</td>
<td>5.20±0.29</td>
<td>55.15±5.10</td>
<td>6.85±0.36</td>
</tr>
<tr>
<td>Ranitidine (50 mg/kg, i.d.)</td>
<td>6</td>
<td>0.12±0.22**</td>
<td>0.31±0.08**</td>
<td>0.61±0.14**</td>
<td>5.80±0.42</td>
<td>85.20±9.13**</td>
<td>7.20±0.19</td>
</tr>
<tr>
<td>EAT (200 mg/kg, i.d.)</td>
<td>6</td>
<td>0.14±0.51**</td>
<td>0.26±0.16**</td>
<td>0.61±0.27**</td>
<td>6.40±0.13</td>
<td>69.40±2.70</td>
<td>8.65±0.05*</td>
</tr>
<tr>
<td>EAAT (200 mg/kg, i.d.)</td>
<td>6</td>
<td>0.16±0.18**</td>
<td>0.46±0.30**</td>
<td>0.93±0.16**</td>
<td>4.80±0.27</td>
<td>73.65±2.52</td>
<td>7.98±0.12*</td>
</tr>
</tbody>
</table>

* P<0.05, ** P<0.01, vs vehicle control group. CMC: carboxy methyl cellulose; EAT: ethanolic extract; EAAT: ethyl acetate extract; i.d.: intraduodenally.

| Table 3 | Ulcer index of ethanol- and indomethacin-induced gastric ulcers
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<tr>
<td>Group</td>
<td>n</td>
<td>Ethanol-induced</td>
<td>Indomethacin-induced</td>
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</tr>
<tr>
<td>Vehicle control (0.5% CMC 10 mL/kg, p.o.)</td>
<td>6</td>
<td>0.298±0.130</td>
<td>0.312±0.080</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Misoprostol (200 µg/kg, p.o.)</td>
<td>6</td>
<td>0.085±0.110**</td>
<td>0.091±0.180**</td>
<td></td>
<td></td>
</tr>
<tr>
<td>EAT (200 mg/kg, p.o.)</td>
<td>6</td>
<td>0.123±0.010**</td>
<td>0.108±0.160**</td>
<td></td>
<td></td>
</tr>
<tr>
<td>EAAT (200 mg/kg, p.o.)</td>
<td>6</td>
<td>0.165±0.010*</td>
<td>0.142±0.300**</td>
<td></td>
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</tr>
</tbody>
</table>

* P<0.05, ** P<0.01, vs vehicle control group. CMC: carboxy methyl cellulose; EAT: ethanolic extract; EAAT: ethyl acetate extract; p.o.: per oral.

2.3.4 Ischemia-reperfusion-induced gastric ulcer model The ischemia-reperfusion of the stomach produced severe gastric lesions in the glandular portion of the stomach. EAT, EAAT and ranitidine groups showed a significant reduction (P<0.05 or P<0.01) in ulcer index when compared with the control (Table 4).

| Table 4 | Ulcer index of ischemia-reperfusion-induced gastric damage
<table>
<thead>
<tr>
<th></th>
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</thead>
<tbody>
<tr>
<td>Treatment</td>
<td>n</td>
<td>Ulcer index</td>
</tr>
<tr>
<td>---</td>
<td>---</td>
<td>---</td>
</tr>
<tr>
<td>Vehicle control (0.5% CMC 10 mL/kg, p.o.)</td>
<td>6</td>
<td>0.249±0.030</td>
</tr>
<tr>
<td>Ranitidine (50 mg/kg, p.o.)</td>
<td>6</td>
<td>0.088±0.020**</td>
</tr>
<tr>
<td>EAT (200 mg/kg, p.o.)</td>
<td>6</td>
<td>0.113±0.010**</td>
</tr>
<tr>
<td>EAAT (200 mg/kg, p.o.)</td>
<td>6</td>
<td>0.176±0.010*</td>
</tr>
</tbody>
</table>

* P<0.05, ** P<0.01, vs vehicle control group. CMC: carboxy methyl cellulose; EAT: ethanolic extract; EAAT: ethyl acetate extract; p.o.: per oral.

3 Discussion

The etiology of peptic ulcer is unknown in most of the cases and generally it is believed that, it may be due to imbalance of the aggressive and defensive factors\[31\]. To regain the balance, different therapeutic agents including plant extracts are developed. The leaves of A. tricolor are one such herbal drug used in the present study to evaluate its antiulcerogenic potential. The extracts that showed ulcer healing effect in acetic acid-induced gastric ulcers were screened further to determine their effect on gastric cytoprotection and gastric secretion.

Application of glacial acetic acid (0.05 mL) onto the serosal surface of the stomach produces deep penetrating gastric ulcers identical to that of human peptic ulcer. Hence, this model is quite useful for the study of human ulcer and for evaluating pharmacological action of the agents used for the treatment of peptic ulcer\[32\]. EAT and EAAT increased the healing of ulcers as demonstrated by a decrease in ulcer index and an increase in regenerated surface epithelial width, capillary density and collagen content. While PEAT and CAT did not show any significant effect on ulcer index, regenerated surface epithelial width, capillary density and collagen content.

Pylorus ligation-induced ulcer was used to study the effect of extracts on gastric secretion. The ligation of the pyloric end of the stomach causes accumulation of gastric acid and causes ulcers in the stomach\[33\]. Agents that decrease gastric acid secretion and increase mucus secretion are effective in protecting the ulcers induced by this method. EAT and EAAT produced a reduction in gastric acid secretion and an increase in mucus content, proving their gastric antisecretory effect and gastric cytoprotective effect.
Ethanol-induced and indomethacin-induced gastric ulcers were employed to study the gastric cytoprotective effect of the extracts. Ethanol induces gastric lesions due to stasis of gastric blood flow that contributes to the development of the hemorrhage and necrotic aspects of tissue injury\textsuperscript{[34, 30]}. Indomethacin is known to produce erosions and ulcers in the stomach due to inhibition of cytoprotective prostaglandins\textsuperscript{[36]}. EA'T and EAAT were effective in both models and the gastric cytoprotective effect may be due to their direct action on the mucus secretion.

Gastrointestinal mucosa is one of the organs which are sensitive to ischemia. Ischemia followed by reperfusion causes erosion and ulceration in the gastric mucosa due to the formation of free radicals\textsuperscript{[37]}. EA'T and EAAT were effective in preventing stomach lesions caused by ischemia-reperfusion.

\textit{A. tricolor} contains number of flavonoids, \( \beta \)-cymenins, tannins, glycosides, saponins, steroids and many other chemical constituents\textsuperscript{[38, 39]}. The non-specific gastroprotective activity of the extracts may be due to the combined effect of the different phytoconstituents present. In previous studies the flavonoid compounds were proved to have anti-secretory and cytoprotective properties\textsuperscript{[40]}. The gastroprotective effect of \textit{A. tricolor} may be due to the presence of flavonoids\textsuperscript{[41, 42]}, polysaccharides, and other antioxidants\textsuperscript{[43]}. Apart from flavonoids, the leaves of the plant are also rich in saponins. The saponins have been shown to exhibit antiulcer properties through the formation of protective mucus on the gastric mucosa and by selectively inhibiting prostaglandin F\(_2\alpha\)\textsuperscript{[44, 45]}. The plant also contains tannins which act as an astringent. Tannins generally have vasoconstrictive and protein precipitating effects. Precipitation of protein at ulcer sites forms impervious protective pellicle which renders the lesion less permeable to toxic substances and more resistant to attack of proteolytic enzymes\textsuperscript{[46]}. These findings from previous studies defend the potent ulcer-healing effect of EA'T and EAAT. The antiulcer activity of \textit{A. tricolor} demonstrated in the present study provides support for the traditional use of this plant in the treatment of gastric ulcers. However, further investigations are required to identify its molecular mechanism and isolate the active components responsible for the antiulcer activity. Finally to conclude, the results of the present study suggest that consumption of the leaves of \textit{A. tricolor} may be beneficial in healing of ulcers in patients suffering from peptic ulcer disease.

4 Acknowledgements

The authors are thankful to Dr. Vinay Babu, Bioneed Preclinical Services Pvt Ltd., India for providing facilities to carry out the work. The authors are also thankful to Prof. Balakrishna Gowda, GKVK, Bangalore, India for identifying the plant and for his valuable suggestions during the work.

5 Competing interests

The authors declare that they have no competing interests.

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42 La Casa C, Villegas I, Alarcón de la Lastra C, Motilva V, Martin Calero MJ. Evidence for protective and antioxidant properties of rutin, a natural flavone, against...
目的：验证苋科植物雁来红（Amaranthus tricolor Linn.）叶的提取物对不同胃溃疡模型大鼠的抗溃疡作用。

方法：通过5种不同的大鼠胃溃疡模型（乙酸、幽门结扎、乙醇、消炎痛及缺血再灌注模型）证实雁来红叶对大鼠胃分泌功能的影响及胃细胞的保护作用。不同的雁来红叶的提取物（乙醇、石油醚、三氯甲烷及乙酸乙酯）以200 mg/kg的剂量给予大鼠服用以检测其功效。

结果：急性口服毒性实验结果显示各种提取物的口服安全剂量可达2 000 mg/kg，故选取该剂量的日分之一即200 mg/kg作为实验用剂量。乙醇提取物及乙酸乙酯提取物对乙酸所致大鼠慢性胃溃疡有治愈作用；对幽门结扎大鼠有抑制其胃分泌功能的作用；对乙醇及消炎痛所致胃溃疡大鼠有胃细胞保护作用。而石油醚及三氯甲烷提取物没有明显的抗大鼠胃溃疡的作用。

结论：本研究证明雁来红叶的提取物对实验性大鼠胃溃疡有很好的治疗作用，这一作用与文献所报道的该植物在民间医学中的应用相符。

关键词：苋属；植物提取物；胃溃疡；细胞保护；抗溃疡药物；大鼠