INHIBITION OF GASTRIC SECRETION BY A WATER EXTRACT FROM
BACCHARIS TRIPTERA, MART

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Baccharis triptera Mart, is a widespread Compositae used in Brazilian folk medicine to treat
gastrointestinal disturbances, rheumatic disease, mild fever, diabetes and as an anti-helminthic.
Water extract of small branches of the plant (WE) administered to mice and rats (0.1 to 2 g/kg,
p.o.) did not alter spontaneous motor activity, sleeping time induced by barbiturates or the tail-
fllick response in mice. The extract decreased by 40% the number of writhings induced by 0.8%
acetic acid, i.p., but did not influence paw edema induced by carrageenan or dextran in rats. WE
(2 g/kg, p.o.) decreased the intestinal transit of charcoal in mice by 20%. Gastric secretion in
pylorus ligated rats was reduced after treatment with WE (1 and 2 g/kg, i.p. or intraduodenal) and
the gastric pH was raised. The extract (1 g/kg, p.o.) prevented gastric ulcers induced in rats by
immobilization at 4°C, but not those induced by indomethacin (10 mg/kg, s.c.). The results
indicate that WE may relieve gastrointestinal disorders by reducing acid secretion and gastro-
intestinal hiperactivity. Neither analgesic nor anti-inflammatory activities were detectable.

Key words: Baccharis triptera – gastric secretion – medicinal plant

Baccharis triptera (trimera) Mart, family Compositae, is a small plant widely distributed in
Brazil, popularly known as “Carqueja”, “Carqueja amargosa”, “Quina de Condaime” and “Vassoura”. Medicinal teas prepared with
the flowery plant are usually employed in folk medicine for gastrointestinal disturbances,
rheumatic disease, mild fever, diabetes and as an anti-helminthic (Coimbra, 1958; Pio Corrêa,
1926).

The present work aimed to study the pharmacological activities of the water extract of
B. triptera Mart mainly its reputed gastrointestinal, analgesic and anti-inflammatory
properties.

MATERIALS AND METHODS

The plant was collected in Brasília, DF, in a
plantation grown at EMBRAPA/Ministry of
Agriculture. Small branches of the plant were
extracted with water (5%, 73°C) for 30 min.

The extract (WE) was then concentrated and
freeze-dried. Biological tests were done using
rats (200-250 g body weight) and mice (30-
40 g) of either sex.

Pharmacological tests in vivo were: general
pharmacological screening (Malone, 1977);
sleeping time induced by sodium pentobarbital
(50 mg/kg, i.p.) in mice (Carlini & Burgos,
1979); analgesic activity was measured in mice
by the latency of response to immersion of
the tail in a water bath at 55°C (Janssen et al.,
1963), and writhing induced by 0.8% acetic
acid (0.1 ml/10 g, i.p.) in mice (Koster et al.,
1959); anti-inflammatory activity was tested by
the method of paw edema induced by either
1% carrageenan or 1% dextran (Winter et al.,
1962). The gastrointesstinal activity of WE was
evaluated by determination of: the gastro-
intestinal transit of charcoal 10% (Stickney
& Northup, 1959); the changes of gastric
secretion in pylorus ligated rats (volume, pH
and total acidity) (Visser et al., 1954; Domer,
1971); protection against acute gastric ulcer
induced by immobilization at 4°C (Paré,
1977) or by indomethacin (10 mg/kg, s.c.)
(Carlini, 1988) and by evaluation of the degree
gastrointestinal ulceration. The index of ulceration
was scored as follows: loss of mucosal foldings,

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mucosal discolouration, edema or hemorrhage (score 1 each); less than 10 petechiae (score 2), more than 10 petechiae (score 3); ulcers/cm² less than 1 mm (score number of ulcers x 2); ulcers more than 1 mm/cm² (score number x 3); perforated ulcers (score number x 4).

In vitro preparations of rat jejunum and vas deferens at 30°C were used to assay the extract either alone or in presence of acetylcholine or norepinephrine. Constitution of the nutritive solutions where as follows (mM): NaCl 135.0, KCl 5.0, MgCl₂ 1.0, CaCl₂ 1.8, NaHCO₃ 15.0, NaH₂PO₄ 1.0 and glucose 11.1 gassed with 95% O₂ - 5% CO₂ for jejunum preparations, and NaCl 64.0, KCl 81.7, NaH₂PO₄ 0.36, NaHCO₃ 15.0 and glucose 5.5 gassed with air for vas deferens preparations.

Results are given as means ± s.e. mean or s.d. when indicated. Differences between control and treated groups were determined by the Student “t” test at P < 0.05.

RESULTS

WE (0.01 to 2 g/kg, i.p.) in mice and rats induced writhings, ptosis and reduced spontaneous motor activity in a dose-dependent fashion. Oral administration was ineffective. The sleeping time (76.5 ± 4.2 min) was not significantly altered by previous administration (30 min) of WE 0.5 and 1 g/kg, p.o. (71.8 ± 4.4 min and 70.5 ± 4.2 min, respectively).

Similarly, the tail-flick latency in control mice (2.2 ± 0.1 s) did not differ from that obtained following treatment with WE (2 g/kg, p.o.), assessed 30 (2.1 ± 0.3 s) and 120 (2.1 ± 0.2 s) min after administration.

Writhings in control animals (62.8 ± 5.5 writhings/30 min) were reduced in mice pretreated with WE (1 and 2 g/kg, p.o.) by 30% and 40% of control. Administration of equal doses of WE to rats however, did not inhibit hind-paw edema induced by either carrageenan or dextran.

In control mice 91.1 ± 3.5% of the small bowel length was filled with charcoal after 45 min of gastric administration of the marker. Pretreatment (30 min) with WE (2 g/kg, p.o.) reduced the intestinal transit of charcoal by 20% of control.

In isolated jejunum preparations incubation of WE (0.5 – 8 mg/ml) caused relaxation of the smooth musculature, which was dependent on the dosis and on the previous tonus of the organ. WE (0.5 and 1 mg/ml) did not alter the maximal contractile response induced by acetylcholine (ACh), nor did it shift the dose-response curve to the agonist. In rat vas deferens preparations, WE (1, 5 and 10 mg/ml) reduced the maximal contraction induced by either ACh (40% to 60%) or noradrenaline (30% to 90%) without shifting the dose-response curves to either agonist.

Gastric secretion collected 4 h after pyloric ligation in rats was reduced by 93% after i.p. treatment and by 85% after intraduodenal administration of WE (1 g/kg). At the same time either treatment decreased total acid secretion by 94% and 49% and raised the gastric pH to 4.4 and 5.5, respectively. Ranitidine (50 mg/kg) used as a positive control also reduced the volume of gastric secretion and raised its pH (Table).

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Volume (ml)</th>
<th>pH</th>
<th>Gastric acidity (mEq/l)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>IP 4.1 ± 2.6</td>
<td>2.5 ± 1.3</td>
<td>49.4 ± 38.3</td>
</tr>
<tr>
<td></td>
<td>D 4.7 ± 1.7</td>
<td>2.0 ± 0.6</td>
<td>41.7 ± 21.4</td>
</tr>
<tr>
<td>WE (1 g/kg)</td>
<td>IP 0.3 ± 0.1</td>
<td>4.4 ± 1.6</td>
<td>2.8 ± 1.9</td>
</tr>
<tr>
<td></td>
<td>D 0.7 ± 0.1</td>
<td>5.5 ± 2.3</td>
<td>21.1 ± 20.9</td>
</tr>
<tr>
<td>Ranitidine (50</td>
<td>IP 1.9 ± 1.2</td>
<td>6.8 ± 0.7</td>
<td>7.7 ± 7.6</td>
</tr>
<tr>
<td>mg/kg)</td>
<td>D 2.3 ± 1.8</td>
<td>4.9 ± 1.9</td>
<td>11.1 ± 9.9</td>
</tr>
</tbody>
</table>

Secrption was collected during 4 h after pylorus ligation. Results are means ± s.d. of 5-6 animals. a: different from control (P < 0.05).

Pretreatment of rats with WE (1 and 2 g/kg, p.o.) reduced the ulceration index (by 76% and 84%) as well as, the number of ulcers (by 40% and 60%) induced by immobilization at 4°C for 2 h (Fig.). At equal doses however, WE was ineffective in protecting against gastric ulcers induced by indomethacin.
antispasmodic activity of the extract described above. Those data thus do not support the popular indication of *B. triptera* as an analgesic/anti-inflammatory agent.

On the other hand, the strong inhibition of gastric secretion produced by the plant extract is a good indication of the anti-acid, antidyspeptic and anti-ulcer activities referred popularly. The data so far obtained do not indicate however, which specific mechanism(s) is (are) responsible for those actions.

REFERENCES


**DISCUSSION**

The pharmacological effects of the water extract of *B. triptera* Mart on the intestinal transit and *in vitro* experiments justify its popular use as antidiarrhoeal and antispasmodic medicine. This antispasmodic activity is probably unrelated to an atropinic mechanism.

The extract did not inhibit the responses of mice on the tail-flick test, nor did it decrease the edema induced by either carrageenan or dextran in rats. Inhibition of writhing induced by acetic acid in mice may be due to the