Hypouricemic and xanthine oxidase inhibitory activities of the fractions of *Coccinia grandis* L. Voigt

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**SUMMARY**

The present study was aimed at investigating the hypouricemic and xanthine oxidase inhibitory activities of the various fractions of the hydromethanolic extract of the leaves of *Coccinia grandis* L. Voigt (Cucurbitaceae). The leaves of this species was used in traditional medicinal system for the treatment of gout, rheumatism, jaundice, bronchitis, fever, skin eruptions, wounds, etc. The degree of xanthine oxidase inhibition was determined *in vitro* by measuring the increase in absorbance at 295 nm associated with uric acid formation. Among the fractions tested, the chloroform fraction exhibited highest potency (IC$_{50}$ 17.8 µg/ml). This was followed by the pet-ether (IC$_{50}$ 29.7 µg/ml), ethyl acetate (IC$_{50}$ 41.2 µg/ml) and residual (IC$_{50}$ 47 µg/ml) fractions. The IC$_{50}$ value of allopurinol was 6.1 µg/ml. In addition, the hypouricemic and hepatic xanthine oxidase (XO)/xanthine dehydrogenase (XDH) inhibitory activities of the fractions were examined *in vivo* using oxonate (280 mg/kg, i.p.) induced hyperuricemic mice. At a dose of 200 mg/kg orally for 7 days, the pet-ether, chloroform and ethyl acetate fractions produced a significant (P < 0.01) reduction in serum urate level and also inhibited hepatic XO/XDH activities when compared to hyperuricemic mice. These inhibitory effects were weaker than that observed for the standard drug, allopurinol (10 mg/kg, p.o.). Lineweaver-Burk analysis of the enzyme kinetics indicated that the mode of inhibition was of a mixed type. These results suggest that the use of *Coccinia grandis* leaves for the treatment of gout could be attributed to its XO inhibitory activity.

**Key words:** *Coccinia grandis*; Hyperuricemia; Xanthine oxidase; Xanthine dehydrogenase; Uric acid

**INTRODUCTION**

Xanthine oxidase (XO) is a highly versatile enzyme that is widely distributed among species (from bacteria to man) and among the various tissues of mammals. It is a member of the group of enzymes known as molybdenum-iron-sulfur-flavin hydroxylases. It catalyses the oxidation of hypoxanthine to xanthine and then to uric acid, the final reactions in the metabolism of purine bases (Fukunari *et al.*, 2004). The over-activity of this enzyme results in a condition, generally called as gout (Arromede *et al.*, 2002; Liote, 2003). Gout is one of the most common metabolic disorders affecting humans. It is characterized by marked hyperuricemia, leading to the deposition of monosodium urate crystals in the joints and kidney, resulting in gouty arthritis and uric acid nephrolithiasis (Kramer and Curhan, 2002). The deposition of needle shaped monosodium urate crystals in the synovial fluid of the major joints produces an extremely painful acute arthritis with repeated attacks of gout. The increased risk of hyperuricemia has been also linked with the development of hypertension, hyperlipidaemia,
cancer, diabetes and obesity (Emmerson, 1998; Lin et al., 2000). The treatment for gout is either increasing the excretion of uric acid or reducing the uric acid production (Emmerson, 1996). XO inhibitors are much useful, since they possess lesser side effects compared to uricosuric and anti-inflammatory agents. Allopurinol is the only clinically used XO inhibitor, which also suffers from many side effects such as hypersensitivity syndrome, Stevens-Johnson syndrome and renal toxicity (Burke et al., 2006). Thus, there is a need to develop compounds with XO inhibitory activity, which is devoid of the undesirable side effects of allopurinol. A potential source of such compounds can be obtained from medicinal plants (Theoduloz et al., 1991; Gonzalez et al., 1995; Kong et al., 2000). Many Indian medicinal plants have been used for the prevention and treatment of gout and related inflammatory disorders (Kirthikar and Basu, 1987), but they lack sufficient scientific evidence.

*Coccinia grandis* L. Voigt, commonly known as 'Ivy gourd' is a tropical plant belonging to the family Cucurbitaceae. It has been found in many countries in Asia and Africa. The leaves of this species have been used in the traditional medicine for the treatment of gout, rheumatism, jaundice, bronchitis, fever, skin eruptions, wounds, etc. (Wasantwisut and Viriyapanich, 2003). Literature suggests the use of the leaves and roots of this plant in the treatment of diabetes mellitus (Kirthikar and Pari, 2003). Our published results showed that the various fractions of the hydromethanolic extract of the leaves of *Coccinia grandis* possessed significant in vitro antioxidant activity (Umamaheswari and Chatterjee, 2008). The putative therapeutic effect of many traditional medicinal plants has been ascribed to the presence of phytochemical constituents with antioxidant activity. Flavonoids, triterpenoids and polyphenolic crude extracts have been reported to possess XO inhibitory activity (Iio et al., 1985; Chang et al., 1993). Preliminary phytochemical screening of the hydromethanolic extract of the leaves of *Coccinia grandis* in our laboratory revealed the presence of many phytochemical constituents such as triterpenoids, flavonoids, saponins, tannins and phenolics. The objective of the present study is to screen the various fractions of the hydromethanolic extract of the leaves of *Coccinia grandis* for its in vitro XO inhibitory activity and in vivo hypouricemic and XO/xanthine dehydrogenase (XDH) inhibitory activities using oxonate-induced hyperuricemic mice.

**MATERIALS AND METHODS**

**Plant material**

The plant material consists of dried powdered leaves of *Coccinia grandis* L. Voigt, belonging to the family Cucurbitaceae. The leaves were collected in and around Coimbatore district, Tamil Nadu, India during the month of August 2006 and were authenticated by Dr. G.V.S Murthy, Joint Director, Botanical Survey of India, Tamil Nadu Agricultural University Campus, Coimbatore (Ref No BSI/SC/5/23/06-07/Tech 1951).

**Preparation of the extract and fractionation**

The air-dried powdered leaves of *Coccinia grandis* (2 kg) was extracted with methanol-water (7 : 3) mixture using a mechanical shaker for 4 h. The resultant extract was concentrated under reduced pressure to yield a residue. The hydromethanolic extract was then extracted successively with equal volumes of petroleum-ether, chloroform and ethyl acetate. Each fraction was then concentrated under reduced pressure to obtain the pet-ether fraction (PEF) chloroform fraction (CF), ethyl acetate fraction (EAF) and residual fraction (RF).

**Drugs and chemicals**

XO (source: microorganisms), xanthine and allopurinol were obtained from Sisco Research Laboratories Pvt. Ltd., Mumbai, India. Oxonic acid potassium salt was obtained from Sigma-Aldrich, USA. Uric acid level was determined using commercial kits.
obtained from Agappe Diagnostics Pvt. Ltd., Kerala, India. All other drugs and chemicals used in the study were obtained commercially and were of analytical grade.

**In vitro XO inhibitory activity**

XO inhibitory activity was assayed spectrophotometrically under aerobic conditions using xanthine as the substrate (Owen and Johns, 1999). The assay mixture consisted of 1 ml of the fraction (5 - 100 µg/ml), 2.9 ml of phosphate buffer (pH 7.5) and 0.1 ml of XO enzyme solution (0.01 units/ml in phosphate buffer, pH 7.5), which was prepared immediately before use. After preincubation at 25°C for 15 min, the reaction was initiated by the addition of 2 ml of the substrate solution (150 µM xanthine in the same buffer). The assay mixture was incubated at 25°C for 30 min. The reaction was then stopped by the addition of 1 ml of 1N hydrochloric acid and the absorbance was measured at 290 nm using a UV spectrophotometer. Different concentrations of the fractions (5 - 100 µg/ml) were dissolved in dimethyl sulfoxide (DMSO) and the final concentration of DMSO was 5%, which did not affect the enzyme assay. Allopurinol (5 – 100 µg/ml), a known inhibitor of XO, was used as the positive control. One unit of XO is defined as the amount of enzyme required to produce 1 mmol of uric acid/min at 25°C. XO inhibitory activity was expressed as the percentage inhibition of XO in the above assay system calculated as,

\[
\% \text{ Inhibition} = \frac{(A - B) - (C - D)}{(A - B)} \times 100
\]

where, A is the activity of the enzyme without the fraction, B is the control of A without the fraction and enzyme, C and D are the activities of the fraction with and without XO respectively. The assay was done in triplicate and IC_{50} values were calculated from the percentage inhibition.

**Enzyme inhibition mechanism**

To determine the mode of inhibition by the most active chloroform fraction, Lineweaver- Burk plot analysis was performed. The enzyme kinetics was carried out in the absence and presence of the chloroform fraction with varying concentrations (10 - 150 mM) of xanthine as the substrate using the XO assay methodology. The mode of inhibition was compared with the positive control allopurinol. The Lineweaver-Burk transformed values were plotted to determine the mode of inhibition.

**In vivo hypouricemic activity**

Experimental model of hyperuricemia was induced by the uricase inhibitor potassium oxonate (Stavric et al., 1975). Mice were divided into seven groups consisting of six each. Group I received 0.5% carboxy methylcellulose (CMC) (10 ml/kg b.w., orally) and served as normal control. Group II received potassium oxonate (280 mg/kg b.w., i.p.) and served as normal control. Group II received potassium oxonate (280 mg/kg b.w., i.p.) and served as hyperuricemic control. Groups III to VI received the PEF, CF, EAF and RF of Coccinia grandis respectively at a dose of 200 mg/kg orally. Group VII received allopurinol (10 mg/kg, b.w., orally) and served as the positive control. The extracts and the reference drug were suspended in 0.5% CMC and administered orally for 7 days. On the 7th day, animals in the groups II to VII were injected intraperitoneally with potassium oxonate at a dose of 280 mg/kg 1 h before the final drug administration to increase the serum urate level (Wang et al., 2004). After one hour of the final drug administration, blood was collected by retro-orbital puncture under mild ether anesthesia. The blood was allowed to clot for approximately 1 h at room temperature and then centrifuged at 5000 g for 10 min to obtain the serum. The serum uric acid level was determined using standard kits.

**Assay of XDH/XO activities in mouse liver**

On the 7th day after blood collection, animals were killed by cervical dislocation and liver was excised, washed in cold 0.15 M KCl and homogenized (10% w/v) using 50 mM phosphate buffer, pH 7.5 (containing 0.5 mM EDTA). The homogenate was then centrifuged at 5,000 g for 10 min and the lipid
layer was carefully removed and the resulting supernatant was further centrifuged at 5,000 g for 10 min. The supernatant was used for the assay of XO and XDH enzyme activities (Hall et al., 1990; Kong et al., 2004). The reaction mixture contained 2 ml of 50 mM phosphate buffer, (pH 7.5), 0.5 ml of tissue homogenate and 1 ml of 1 mM potassium oxonate. For the assay of XDH activity, but not of XO activity, 200 µM NAD$^+$ was added as an electron acceptor to the above mixture. After preincubation for 15 min at 37ºC, the reaction was initiated by the addition of 1 ml of 150 µM xanthine (dissolved in phosphate buffer, pH 7.5). After 10 min, the reaction was stopped by adding 0.5 ml of 0.58 M HCl and the solution was centrifuged at 5,000 g for 5 min. The absorbance of the supernatant was measured at 290 nm against the blank (50 mM phosphate buffer, pH 7.5, containing 0.5 mM EDTA). XO/XDH activities were expressed as nanomole uric acid formed/min/mg protein. Each assay was performed in triplicate.

Protein determination
Protein concentration in the tissue homogenate was determined by the Lowry method using bovine serum albumin as the standard (Lowry et al., 1951).

Statistical analysis
All data were expressed as mean ± standard error of mean (S.E.M.) and statistical analysis was performed using one way analysis of variance (ANOVA) followed by Dunnett’s test. $P < 0.05$ were considered significant.

RESULTs

**In vitro XO inhibitory activity of the fractions of Coccinia grandis**
All the fractions of *Coccinia grandis* elicited a dose-dependent inhibition of XO enzyme activity. Inhibition of XO resulted in a decreased production of uric acid, which was measured spectrophotometrically. At a concentration of 100 µg/ml, highest activity was observed in the chloroform fraction (88.1 ± 0.2% inhibition). The percentage inhibition of the pet-ether fraction was 85.7 ± 0.8%, which was almost similar to the chloroform fraction. This was followed by the ethyl acetate (76.55 ± 0.75%) and residual (66.35 ±1.05 %) fractions. The concentration of the fractions at 50% of the XO inhibition (IC$_{50}$) was calculated. These results were compared with the standard drug allopurinol, which showed 92.25 ± 0.05% inhibition at 100 µg/ml concentration with IC$_{50}$ value 6.1 µg/ml (Table 1).

**Enzyme inhibition mechanism**
The inhibition mechanism of the chloroform fraction, which is a potent inhibitor of XO, was studied by kinetic analysis using double-reciprocal plotting. Lineweaver- Burk plots of reactions in the presence and absence of the chloroform fraction in a XO reaction mixture is shown in Fig. 1. The mode of inhibition was investigated and compared with the standard drug, allopurinol. The data indicates that the mode of XO inhibition is of mixed type

### Table 1. In vitro xanthine oxidase inhibitory activity of the fractions of *Coccinia grandis*

<table>
<thead>
<tr>
<th>Fraction</th>
<th>Yield of fraction (% w/w)</th>
<th>Percentage xanthine oxidase inhibition</th>
<th>IC$_{50}$ (µg/ml)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pet-ether</td>
<td>1.6</td>
<td>27.35 ± 0.94</td>
<td>37.35±0.85</td>
</tr>
<tr>
<td>Chloroform</td>
<td>1.8</td>
<td>31.35±0.65</td>
<td>44.29±0.99</td>
</tr>
<tr>
<td>Ethyl acetate</td>
<td>0.8</td>
<td>10.8±0.5</td>
<td>25.4±0.9</td>
</tr>
<tr>
<td>Residual</td>
<td>5.3</td>
<td>6.1±1.3</td>
<td>29.85±1.55</td>
</tr>
<tr>
<td>Allopurinol</td>
<td>-</td>
<td>44.91±1.88</td>
<td>66.91±0.88</td>
</tr>
</tbody>
</table>

Values are mean ± S.E.M. of three parallel measurements.
Hypouricemic and xanthine oxidase inhibitory activities of the fractions of Coccinia grandis L. Voigt

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Effect of the fractions of Coccinia grandis on urate level in hyperuricemic mice
Administration of potassium oxonate resulted in hyperuricemia as evidenced by a significant ($P < 0.01$) increase in the serum uric acid level (7.39 ± 0.42 mg/dl) when compared to the control group (3.72 ± 0.17 mg/dl). Pre-treatment with the pet-ether, chloroform and ethyl acetate fractions of Coccinia grandis for seven days reduced the serum urate levels significantly ($P < 0.01$) when compared with the hyperuricemic control. The reduction in urate level produced by the chloroform fraction (5.08 ± 0.20 mg/dl) was more potent among the fractions tested. Administration of the residual fraction did not produce any significant ($P > 0.05$) reduction in serum urate level. Allopurinol, at a dose of 10 mg/kg elicited a significant ($P < 0.01$) reduction in serum urate level (3.05 ± 0.13 mg/dl) compared to hyperuricemic control (Table 2).

Effect of the fractions of Coccinia grandis on serum urate level in normal mice
Animals were treated orally with the fractions at a dose of 200 mg/kg orally for 7 days to examine its effect on serum urate level in potassium oxonate untreated mice. Administration of the fractions to normal mice did not decrease the serum uric acid level and is almost similar to vehicle treated group. However, allopurinol significantly ($P < 0.01$) lowered the serum uric acid level in normal animals (Table 3). These results indicate that the fractions do not possess hypouricemic activity in normal mice.

Effect of the fractions of Coccinia grandis on XO/XDH activities in mouse liver
Animals treated with the pet-ether, chloroform and ethyl acetate fractions at a dose of 200 mg/kg b.w. for seven days produced a significant ($P < 0.01$)

Table 2. Effect of the fractions of Coccinia grandis on serum urate level in oxonate-induced hyperuricemic mice

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Dose (mg/kg)</th>
<th>Serum urate level (mg/dl)</th>
<th>% Inhibition</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.5% CMC</td>
<td>10 ml/kg</td>
<td>3.72 ± 0.17</td>
<td>-</td>
</tr>
<tr>
<td>Potassium oxonate</td>
<td>280</td>
<td>7.39 ± 0.42</td>
<td>-</td>
</tr>
<tr>
<td>Pet-ether fraction</td>
<td>200</td>
<td>5.55 ± 0.32$^b$</td>
<td>24.84</td>
</tr>
<tr>
<td>Chloroform fraction</td>
<td>200</td>
<td>5.08 ± 0.20$^b$</td>
<td>31.25</td>
</tr>
<tr>
<td>Ethyl acetate fraction</td>
<td>200</td>
<td>5.85 ± 0.21$^b$</td>
<td>20.83</td>
</tr>
<tr>
<td>Residual fraction</td>
<td>200</td>
<td>6.97 ± 0.27$^b$</td>
<td>5.68</td>
</tr>
<tr>
<td>Allopurinol</td>
<td>10</td>
<td>3.05 ± 0.13$^b$</td>
<td>58.64</td>
</tr>
</tbody>
</table>

All drugs were given orally and potassium oxonate injected intraperitoneally.

% Inhibition = 100 [(hyperuricemic control - experiment)/hyperuricemic control].

Values are mean ± S.E.M. N = 6 in each group. $^aP < 0.01$ vs normal control; $^bP < 0.01$, $^cP > 0.05$ vs hyperuricemic control.
inhibition towards XO (39.9%, 42.0% and 18.34%) and XDH (38.4%, 40.6% and 22.18%) respectively, when compared with the hyperuricemic control. The inhibition of the XO/XDH activities of the chloroform fraction was found to be the highest among the fractions tested. The inhibition of enzyme activities of the ethyl acetate fraction resulted similar to that of the pet-ether fraction. On the other hand, the action of the residual fraction on the inhibition of XO (9.37%) and XDH (65.93%) activities at a dose of 10 mg/kg, exhibiting more potent activity than the fractions (Table 3).

**Table 3.** Effect of the fractions of *Coccinia grandis* on serum urate level in normal mice

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Dose (mg/kg)</th>
<th>Serum urate level (mg/dl)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.5% CMC</td>
<td>10 ml/kg</td>
<td>3.72 ± 0.17</td>
</tr>
<tr>
<td>Pet-ether fraction</td>
<td>200</td>
<td>3.21 ± 0.14</td>
</tr>
<tr>
<td>Chloroform fraction</td>
<td>200</td>
<td>3.17 ± 0.11</td>
</tr>
<tr>
<td>Ethyl acetate fraction</td>
<td>200</td>
<td>3.95 ± 0.35</td>
</tr>
<tr>
<td>Residual fraction</td>
<td>200</td>
<td>3.76 ± 0.16</td>
</tr>
<tr>
<td>Allopurinol</td>
<td>10</td>
<td>2.82 ± 0.12</td>
</tr>
</tbody>
</table>

Values are mean ± S.E.M. N = 6 in each group. *P > 0.01 and **P < 0.01 vs normal control.

DISCUSSION

The enzyme XO catalyses the oxidation of hypoxanthine to xanthine and then to uric acid, which plays a crucial role in gout (Bowman and Rand, 1980). XO is an important source of oxygen-derived free radicals, which catalyzes the reduction of oxygen (during reperfusion phase), leading to the formation of superoxide anion radicals and hydrogen peroxide, as well as hydroxyl radicals (Halliwell and Gutteridge, 2001). It has been proposed as a central mechanism of oxidative injury in some situations like gout, ischemia, renal damage, hypertension, diabetes, etc. (Mazzali *et al.*, 2001; Berry and Hare, 2004; Nakagawa *et al.*, 2006). Gout is characterized by high level of uric acid in the blood, which can result from a number of genetic disorders that cause either over production or impaired excretion of uric acid. An acute attack of gout occurs as a result of inflammatory reactions to monosodium urate crystals that are deposited in the joint tissue (Rang *et al.*, 2001). Recent findings show that the occurrence of gout is increasing worldwide, possibly due to the changes in dietary habits like intake of high-purine foods viz., organ meats, yeast, beer and other alcoholic beverages (Lewis and Doisy, 1918). The main therapeutic approach for gout is the use of XO inhibitors such as allopurinol, which block the final step in the

**Table 4.** Effect of the fractions of *Coccinia grandis* on liver XO/XDH activities in oxonate-induced hyperuricemic mice

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Dose (mg/kg)</th>
<th>nanomole uric acid formed/min/mg protein</th>
<th>% Inhibition</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>XO</td>
<td>XDH</td>
</tr>
<tr>
<td>0.5% CMC</td>
<td>10 ml/kg</td>
<td>1.33 ± 0.07</td>
<td>1.51 ± 0.04</td>
</tr>
<tr>
<td>Potassium oxonate</td>
<td>280</td>
<td>3.38 ± 0.11</td>
<td>3.20 ± 0.12</td>
</tr>
<tr>
<td>Pet-ether fraction</td>
<td>200</td>
<td>2.03 ± 0.15</td>
<td>1.97 ± 0.12</td>
</tr>
<tr>
<td>Chloroform fraction</td>
<td>200</td>
<td>1.96 ± 0.09</td>
<td>1.91 ± 0.10</td>
</tr>
<tr>
<td>Ethyl acetate fraction</td>
<td>200</td>
<td>2.76 ± 0.05</td>
<td>2.49 ± 0.13</td>
</tr>
<tr>
<td>Residual fraction</td>
<td>200</td>
<td>2.92 ± 0.13</td>
<td>2.92 ± 0.18</td>
</tr>
<tr>
<td>Allopurinol</td>
<td>10</td>
<td>1.22 ± 0.08</td>
<td>1.08 ± 0.03</td>
</tr>
</tbody>
</table>

All drugs were given orally and potassium oxonate injected intraperitoneally. Values are mean ± S.E.M. N = 6 in each group. *P < 0.01 vs normal control; **P < 0.01, ***P > 0.05 vs hyperuricemic control.
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...synthesis of uric acid from purines. However, it has been observed that allopurinol induce side effects such as skin allergy, Steven-Johnson’s syndrome and kidney damage (Kumar et al., 1996; Wallach, 1998).

An alternative to allopurinol is the use of medicinal plants, which possess phytochemical constituents. We thus began our program to look for XO inhibitors of phytochemical origin from the various fractions of the hydromethanolic extract of the leaves of Coccinia grandis. The leaves of Coccinia grandis (Cucurbitaceae) are being frequently used in Indian traditional medicinal system for the treatment of gout and rheumatism.

All the fractions of Coccinia grandis inhibited XO in a concentration-dependent manner. The in vitro inhibition of XO by the chloroform fraction (IC$_{50}$ 17.87 µg) is moderate when compared to allopurinol (IC$_{50}$ 6.1 µg). However, at higher doses of the fraction, XO would be significantly inhibited. The in vivo experiments demonstrated that the fractions possess significant hypouricemic activity in oxonate-induced hyperuricemic mice. Potassium oxonate, an uricase inhibitor partially blocks the conversion of uric acid to allantoin and hence elevates the serum uric acid level in experimental animals. The pet-ether, chloroform and ethyl acetate fractions at a dose of 200 mg/kg restored the serum uric acid level of the hyperuricemic animals similar to the control group. The hypouricemic activity of the fractions could be explained, at least in part, by a lowering of XO/xanthine dehydrogenase activities in the drug-treated animals. It is interesting to note that the fractions only lower the serum urate levels of the hyperuricemic mice, but not of the normal mice. In contrast, allopurinol exerts its hypouricemic activity in both normal and hyperuricemic mice. This characteristic feature of the fractions could be considered as an advantage because excessive lowering of uric acid level in the circulation beyond that of the normal range might be counter-productive.

In addition, we have previously reported the in vitro antioxidant activity of the fractions of the leaves of Coccinia grandis and the presence of high phenolic and flavonoid content in the fractions has contributed directly to the antioxidant activity by neutralising the free radicals (Umamaheswari and Chatterjee, 2008). This antioxidant property is actually an added advantage for this species towards XO inhibition. Flavonoids are a group of polyphenolic compounds, which have been reported to possess XO inhibitory activity (Costantino et al., 1992). Hence, the antioxidant activity of the fractions may also contribute to its hypouricemic and XO inhibitory activities. Further investigations on the isolation and identification of active compounds present in the leaves are in progress to identify a potential chemical entity for clinical use in the prevention and treatment of gout and related inflammatory disorders.

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