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The Potential Role of *Daucus carota* Aqueous and Methanolic Extracts on Inflammation and Gastric Ulcers in Rats

Katia Wehbe, Mohamad Mroueh, and Costantine F. Daher

**Abstract**

*Daucus carota* (DC) is among commonly used plants in folk medicine in Lebanon and the region. The present investigation was undertaken to examine the effects of the aqueous and extracts of *Daucus carota* umbels against acute and chronic inflammation, gastric ulcer and antibacterial activity on rats. The effects of DC aqueous extract (DCAE) on glycemia, lipemia, hepatic, renal and pancreatic function were also examined. Results on acute inflammation showed that the aqueous and methanolic extracts (DCME) produced maximum anti-inflammatory activity at doses of 400 and 140 mg/kg body weight with 90.9 and 58.6 % inhibition, respectively. In chronic inflammation, the same doses showed maximum anti-inflammatory activity with 58 and 44.1 % inhibition, respectively. DCME showed significant protection against ethanol induced gastric ulcer with a curative ratio of 46.8 and 68.7%, respectively, at a dose of 250 mg/kg body weight. None of the extracts showed significant antibacterial activity. DCAE intake (250 mg/kg body weight) for one month period did not show adverse effects on lipemia, glycemia, hepatic and liver function except for a slight decrease in HDL cholesterol (p<0.05). In conclusion, both DCAE and DCME exhibited promising anti-inflammatory and anti-ulcerogenic potentials while showing no negative influence on liver, kidney and pancreas function.

**KEYWORDS:** *Daucus carota*, glycemia, inflammation, lipids, ulcer

**Author Notes:** This research project was sponsored by the Lebanese American University Research Council.
INTRODUCTION

Wild carrot, *Daucus carota* L. ssp. *carota* (Apiaceae), is a tall robust biannual spiny-fruited herb native to western Asia or the Near East. It is also found in the Mediterranean region, southwest Asia, tropical Africa, Australia, and North and South America (Reed, 1976). This plant is indigenous to Europe and is the precursor of the cultivated carrot (McGuffin et al., 1997). It is worth noting the confusion in the literature between the edible carrot, *Daucus carota* L. ssp. *sativus*, and the wild carrot, *Daucus carota* L. ssp. *carota*, which has inedible whitish tough root. Wild carrot is listed by the council of Europe as a natural source of food flavoring (Barnes et al., 2002). The plant is a part of the folk medicine in Lebanon where it is used to protect against gastric ulcer, diabetes, muscle and back pain and to enhance liver function and immune system.

Extensive studies have been conducted on *Daucus carota* L. ssp. *sativus*, while little was done on *Daucus carota* L. ssp. *carota*. The plant has been reported to possess antilithic, diuretic and carminative properties and has been traditionally used to treat urinary calculus, cystitis, gout and lithuria (Barnes, 1998; Thomas et al., 2001; WHO, 2004). In vivo studies showed conflicting results where significant (Prakash, 1984) and insignificant (Lal et al., 1986) antifertility activities were observed in rats. In European folk medicine, the volatile oil from wild carrot is used as an active urinary antiseptic and anti-inflammatory remedy for cystitis and prostatitis (Hoffmann 1990). It has been reported that rats fed on wild carrot exhibited protection against carbon tetrachloride-induced hepatotoxicity (Handa, 1986). Recent work in our lab showed that the aqueous extract of the wild carrot umbel possesses anticancer activity against human promyelocytic leukemia HL-60 cells (Diab-Assaf et al. 2007). Additionally, the oil extract was found to have potent anti-tumor promoting effects against chemically induced skin cancer in mice (Abou Zeinab et al. 2008). Data on clinical safety and toxicity of the wild carrot are still lacking. The present study was conducted to evaluate the potential pharmacological effects of the methanol and aqueous extracts of wild carrot in rats against inflammation (acute and chronic) and gastric ulcer, in addition to its effects on lipemia, glycemia and liver function. The potential antibacterial activity on some hospital isolates was also evaluated.

MATERIALS AND METHODS

Plant material collection and extraction procedure

Wild carrot, *Daucus carota* L. ssp. *Carota* L. was initially identified by Dr. Ahmad Houri, a Lebanese plant expert, and confirmed according to descriptions
found in “Medicinal Plants of the World” by van Wyk and Wink (2004). The plant was collected from diverse fields throughout Lebanon, during the summer (July to August). Top aerial parts (umbels) were air dried in shadow, soaked in methanol for 72 hours, and then the extract was filtered and evaporated to dryness by rotary evaporation. The aqueous extract was prepared by soaking the umbels in preboiled water with occasional stirring for 30 minutes. The extract was filtered and lyophilized. Both extracts were stored in a well-sealed dry box in the refrigerator until use.

Animals

Male Sprague-Dawley rats, maintained under a 12h photoperiod (0800 – 2000) at an environmental temperature of 20°C were used. All experimental protocols were conducted in compliance with the Guide for the Care and Use of Laboratory Animals (National Research Council of the United States 1985) and approved by the Lebanese American University Research Council.

Anti-inflammatory effect

(i) Carrageenan induced paw edema in rats

Animals were divided into eight groups of six animals each. In all groups acute inflammation was produced by a single subplantar injection of 0.02 ml of freshly prepared 1% carrageenan in normal saline in the right hind paw of rats (Ajith and Janardhanan, 2001). One group served as a control (non-treated), three groups received the DCAE (Daucus catota aqueous extract) at a concentration of 100, 200, or 400 mg/Kg BW (body weight), three groups received DCME (Daucus catota methanol extract) at a concentration of 75, 150, or 300 mg/Kg body weight, and the last group received diclofenac (10 mg/Kg BW) as a standard reference drug. The aqueous and methanolic extracts and diclofenac were administered intraperitoneally (i.p.) 30 min prior to carrageenan injection. The paw thickness was measured using vernier calipers before and 3h after carrageenan injection (Ajith and Janardhanan, 2001).

(ii) Formalin induced paw edema in rats

Animals were divided into eight groups of six animals each. In all groups, chronic inflammation was produced by a single subplantar injection of 0.02 ml of 2% formalin in the right hind paw (Ajith and Janardhanan, 2001). Thirty min prior to formalin injection, 3 groups received DCAE (i.p.) at a concentration of 100, 200, or 400 mg/Kg BW, 3 groups received DCME (i.p.) at a concentration of 75, 150,
or 300 mg/Kg BW, one group the standard reference drug diclofenac (10 mg/Kg BW, ip), and one group served as a control (non-treated). The administration of the extracts and diclofenac was continued once daily for 6 consecutive days. The paw thickness was measured using vernier calipers before and 6 days after formalin injection (Jose et al., 2004). The increase in paw thickness in both models was calculated using the formula:

\[ P_t - P_0 \]

Where \( P_t \) is the thickness of paw at time \( t \) (3 hours after carrageenan injection and 6 days after formalin injection) and \( P_0 \) is the paw thickness at 0 time. The percent inhibition was calculated using the formula:

\[ \frac{(C - T)}{C} \times 100 \]

Where \( C \) is the increase in paw thickness of the positive control and \( T \) is that of treatments.

**Anti-ulcerogenic effects**

The effects of DCAE and DCME on ethanol-induced gastric ulcer were conducted on male Sprague-Dawley rats according to the method described by Alkofahi and Atta (1999). Briefly, male Sprague-Dawley rats (250-300 g) were randomly assigned to 7 groups of 6 rats each. Forty-eight hours before use, animals were starved to ensure an empty stomach, and were kept in cages with raised floors of wide wire mesh to prevent coprophagy. To prevent excessive dehydration during starvation, all groups were supplied with sucrose 8% (w/v) in NaCl 0.2% (w/v) which was removed 1h before experimentation (Alkofahi and Atta, 1999; Gharzouli, 1999). Control group (non-treated) was given water (10 ml/kg BW) while the treated groups received respectively 200 and 400 mg/kg BW of DCAE, 150 and 300 mg/kg BW of DCME and 10 mg/kg BW of the reference drug Cimetidine (Xu et al., 1998). Doses were administrated orally with water (10 ml/kg BW) via a stainless steel intubation needle. Two doses were given on the first day at 9:00h and 17:00h; a third dose was given on the second day 1.5 h before induction of gastric ulceration. To induce gastric ulcer, the controls as well as the treated groups received by gastric gavage 10 ml/kg BW ethanol 50% (v/v) in water. Group I served as the reference group (no ulcer induction by ethanol) and received equivolmes of water instead. One hour after ethanol administration, all animals were sacrificed by an overdose of diethyl ether, stomachs were rapidly removed, opened along their greater curvature and rinsed under running tap water. Using illuminated stereomicroscope long lesions were counted and measured.
along their greater length. Petechial lesions (very small lesions) were also counted and each five were considered as 1 mm of ulcer. The average total length of long ulcers and petechial lesions in each group of rats represented the ulcer index (mm). The curative ratio was determined by the formula:

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

**Screenings for antibacterial activity**

DCME was tested for its ability to inhibit the activity of 11 hospital isolates from different patients by disc diffusion method. The bacterial strains used were *Enterobacter agglomerans* (Gram-), *Proteus mirabilis* (Gram-), *Salmonella* spp. (Gram-), *Citrobacter braakii* (Gram-), *Escherichia coli* (Gram-), *Klebsiella pneumoniae* (Gram-), *Pseudomonas aeruginosa* (Gram-), *Haemophilus parainfluenza* (Gram-), *Streptococcus pyogens* (S. pyogens) (Gram+), methicillin-resistant *Staphylococcus aureus* (MRSA), and methicillin-sensitive *Staphylococcus aureus* (MSSA) (Gram+). All the bacterial species tested were identified by API 20E except for *S. pyogens* (API Streptococcus) and MRSA and MSSA, which were identified by *Staphylococcus* coagulase test.

**Disc diffusion assay**

Sterile 5-mm diameter filter paper disks containing 400 µg of either the aqueous or methanolic extract were placed on the surface of Muller-Hinton agar inoculated with the appropriate bacteria tested except for *S. pyogens* and *Haemophilus parainfluenza* that were grown on blood agar and chocolate agar respectively. The inoculum size of each test strain was standardized by adjusting the optical density of the bacterial suspension to 0.08 (OD 620 = 0.08) at 620 nm that corresponds to $10^8$ bacteria / ml of suspension. All the plates were then incubated at 37°C for 24h. The antibacterial activity was evaluated by measuring the diameter of the inhibition zone. A specific reference drug was used for each type of bacteria.

**Minimal inhibitory concentration (MIC)**

The MIC values were studied for the microorganisms that were determined to be sensitive to the DC extract in the disc diffusion assay. The MIC determination was performed based on a micro-well dilution methods (Basri and Fan, 2005; Güllüce et al., 2004). The MIC was determined using the two-fold serial microdilution method with normal saline. The final concentration ranged from 20 mg/ml to 0.00976 mg/ml. The tested extracts were added to sterile NaCl into
microtiter plates before the diluted bacterial suspension was added (OD<sub>620</sub> = 0.08) and the background contributed by the extract color was determined. Maxipime (500 µg/ml) was used as a reference drug for the bacteria tested. The MIC values were taken as the lowest concentrations of the extracts that showed inhibition after 24h at 37ºC. The absorbance of the microtiter plates' wells was read at 620 nm (Basri and Fan, 2005) using an E-LizaMat 300 reader and the DGR ELISA regression program.

**Chronic intake effects of DCAE**

Male Sprague-Dawely rats (n=30) (Lebanese American University stock) were randomly divided into two groups comprising 15 animals each with an average weight of 250 g. The first group served as a control and received 6.5 g of food per 100 g BW. Food consisted of standard rat chow (19% protein, 9.6% fat, 4.3% fiber and 61% carbohydrate) to which 5% coconut oil was added. Coconut was used to make the diet more atherogenic (Daher et al., 2003). The second group, referred to as treatment group, received the same food as the control in addition to the DCAE in drinking water (300 mg/kg BW). Animals were maintained at an ambient temperature of 20 - 22°C and 12h photoperiod during the whole study period. All experimental protocols complied with the Guide for the Care and Use of Laboratory Animals (National Research Council of the United States 1985). After one month of extract intake, fasted animals (18h) were rapidly anesthetized using diethyl ether and a midline abdominal incision was made for about two-thirds of the length of the abdomen. About 8 ml of blood were withdrawn from the inferior vena cava and then divided into two tubes with and without Na<sub>2</sub>EDTA (1 mg/ml) to collect plasma and serum samples respectively after centrifugation (2000 g; 20 min; 4°C). Plasma samples, kept on ice, were immediately used for lipoprotein (d<1.063 g/ml) isolation by ultracentrifugation and determination of apo B concentration, while serum samples were used for the analyses of triacylglycerol, total cholesterol, high density lipoprotein cholesterol (HDL-C), low density lipoprotein cholesterol (LDL-C), aspartate aminotransferase (AST or SGOT), alanine aminotransferase (ALT or SGPT), alkaline phosphatase (ALP), lactate dehydrogenase (LDH), urea, creatinine, uric acid, calcium, phosphorus, glucose, insulin and amylase by using the appropriate kits (Dialab, Austria).

**Determination of total plasma apo B**

To minimize proteolytic degradation of apoB48 and apoB100 the following were added: 5 µl/ml plasma of aprotinin (Fluka, Switzerland), 2 mg/liter, and 5 µl/ml plasma of phenylmethylsulfonyl fluoride (PMSF), 5 mM in 2-propanol. Total apoB (apoB48 and apoB100) content in the plasma of fasted animal was
estimated in the lipoprotein fraction (d < 1.063 g/ml) that includes chylomicrons (CM), very low density lipoprotein cholesterol (VLDL-C), intermediate density lipoprotein cholesterol (IDL-C) and LDL-C. Briefly, 2 ml of plasma were put in a 10 ml polycarbonate ultracentrifuge tube (Sorvall, Kendro Laboratory Products) and 140 mg/ml of solid NaCl added to increase the density to 1.1 g/ml. The plasma sample was overlaid with 5 ml of NaCl solution (d = 1.063 g/ml) containing 0.01% (w/v) Na₂EDTA and 0.02% (w/v) NaN₃ (pH=7.4). The top 0.5 ml lipoprotein layer was collected after 48h of centrifugation at 28,000 rpm at 15°C (Sorvall RC 28S centrifuge; Supraspeed F-28/13 fixed angle rotor). Samples containing the lipoprotein fractions (d = 1.063 g/ml) were delipidated in a methanol-diethyl ether solvent system (Karpe et al., 1996). Protein material was dissolved in 0.15M sodium phosphate, 12.5% v/v glycerol, 2% w/v sodium dodecyl sulfate (SDS), 5% v/v mercaptoethanol, 0.001% w/v bromophenol blue, pH=6.8, at room temperature for 30 min, denatured at 90 °C for 4 min and centrifuged at 15680 g for 4 min. Samples were frozen at -20 °C, subjected to 4-20% SDS-PAGE within 3 days, and then analyzed for apoB concentration (Daher et al. 2003). All samples were run in duplicates.

**Statistical analyses**

Values are presented as means ± SEM. Normal distributions of the data were confirmed using the kolmogorov-Smirnov one-sample goodness of fit test. Unacceptable heteroscedasy was eliminated, where possible, by the logarithmic transformation of the data. Where data were normally distributed comparisons between groups were made using one-way analysis of variance with localization of differences being achieved with Duncan’s multiple range test. Where data were not normally distributed and could not be transformed to achieve normal distribution, the Kruskall-Wallis nonparametric analysis was used and differences located using the Mann-Whitney “U” test with appropriate adjustment to the critical value of p. Statistical significance was assumed at p< 0.05.

**RESULTS**

**Anti-inflammatory activity- aqueous extract**

The aqueous extract significantly inhibited the acute inflammation at 100, 200, and 400 mg/kg BW. Similar anti-inflammatory activity was observed in the chronic inflammation model study at all concentrations used (Table1 and Figure1).
Anti-inflammatory activity- methanol extract

DCME significantly inhibited the acute and chronic inflammation at concentrations of 70, 140, and 280 mg/kg BW. The inhibition observed was not dose dependant (Figure 2 and Table 1).

**Figure 1:** Effect of DCAE on carrageenan induced and formalin induced inflammation. Values denote mean increase in paw thickness ± SEM (n=6).

![Graph showing effect of DCAE on inflammation](image)

*Significant difference (p<0.05) with respect to control.

**Table 1:** Effect of DCAE and DCME on carrageenan induced and formalin induced inflammation. Values denote mean percentage inhibition of inflammation (n=6).

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Dose (mg/kg)</th>
<th>Carrageenan</th>
<th>Formalin</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diclofenac</td>
<td>10</td>
<td>79.2</td>
<td>42</td>
</tr>
<tr>
<td>DCAE</td>
<td>100</td>
<td>72.7</td>
<td>42</td>
</tr>
<tr>
<td>DCAE</td>
<td>200</td>
<td>81.9</td>
<td>48</td>
</tr>
<tr>
<td>DCAE</td>
<td>400</td>
<td>90.9</td>
<td>58</td>
</tr>
<tr>
<td>DCME</td>
<td>75</td>
<td>46</td>
<td>39.8</td>
</tr>
<tr>
<td>DCME</td>
<td>150</td>
<td>56.8</td>
<td>44.1</td>
</tr>
<tr>
<td>DCME</td>
<td>300</td>
<td>39.3</td>
<td>43.5</td>
</tr>
</tbody>
</table>
**Figure 2:** Effect of DCME on carrageenan induced and formalin induced inflammation. Values denote mean increase in paw thickness ± SEM (n=6).

*Significant difference (p<0.05) with respect to the control.

**Anti-ulcer activity**

Ethanol induced gastric damage was characterized by both long ulcers and petechial lesions. A significant protection against ethanol induced ulcer was observed with both DCAE and DCME. The best curative ratio was observed with the group that received the methanol extract when compared with the aqueous extract and the Cimetidine groups (Figure 3).

**Antibacterial activity**

The antibacterial activity of DCAE and DCME were evaluated against eleven human pathogenic bacterial species. Both extracts at a concentration of 20 mg/ml, showed no antibacterial activity against nine of the studied microorganisms, only a minimal inhibition zone was observed against *Staphylococcus aureus* meti $S$ and meti $R$ (data not shown). The minimal inhibitory concentrations (MIC) of the DCAE against *S. aureus* meti $S$ and meti $R$ were the same (20 mg/ml), whereas the MIC values of methanol extract were 10 mg/ml for both strains. Maxipime (Cefepime) was used as reference drug. The results of the MIC values revealed that *S. aureus* meti $S$ and meti $R$ showed moderate sensitivity towards the DC extracts.
Figure 3: Ulcer index (mm) and percentage curative ratio values in different experimental groups (n=6).

Lipid profile

Following one-month period of administering the DCAE, animals were sacrificed and blood samples were collected. Data presented in Table 2 showed the lipid profile of the experimental groups. The mean total cholesterol, TG and LDL-cholesterol concentrations were similar in both groups without significant changes. However, a significant decrease in serum HDL-cholesterol was observed with extract intake ($p=0.0143$). Calculation of the total cholesterol/HDL cholesterol, and LDL cholesterol/HDL cholesterol ratios showed no significant changes between the two groups. Determination of total plasma apoB concentration revealed no significant changes between the control and treated group.

Hepatic screening tests

Hepatic enzymes (SGOT, SGPT, ALP, and LDH) were tested in order to study the effect of DC aqueous extract on liver function. Table 3 summarizes the activities of these enzymes in the control and treated groups. Data showed no significant changes in liver enzyme concentrations in both the control and treated groups.
Table 2: Serum lipid concentration after one month supplementation of DCAE. Values denote mean ± SEM (n=15)

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Control Group</th>
<th>Treated Group</th>
</tr>
</thead>
<tbody>
<tr>
<td>Triacylglycerol</td>
<td>51.53 ± 4.63</td>
<td>61.73 ± 5.02</td>
</tr>
<tr>
<td>Total cholesterol</td>
<td>57.65 ± 2.04</td>
<td>54.07 ± 1.80</td>
</tr>
<tr>
<td>LDL-C</td>
<td>11.18 ± 0.98</td>
<td>10.60 ± 1.10</td>
</tr>
<tr>
<td>HDL-C</td>
<td>47.65 ± 1.40</td>
<td>42.93 ± 1.10 *</td>
</tr>
<tr>
<td>Total cholesterol/HDL-C</td>
<td>1.21 ± 0.15</td>
<td>1.26 ± 0.13</td>
</tr>
<tr>
<td>LDL-C/HDL-C</td>
<td>0.23 ± 0.02</td>
<td>0.25 ± 0.03</td>
</tr>
<tr>
<td>Total Apo B</td>
<td>102.0 ± 10.2</td>
<td>117.0 ± 13.0</td>
</tr>
</tbody>
</table>

* Significant difference (p<0.05) with respect to the control.

Table 3: Hepatic enzymes activities (U/L) in serum after one month supplementation with DCAE. Values denote mean ± SEM (n=15).

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Control Group</th>
<th>Treated group</th>
</tr>
</thead>
<tbody>
<tr>
<td>SGOT</td>
<td>122 ± 6.4</td>
<td>119 ± 2.9</td>
</tr>
<tr>
<td>SGPT</td>
<td>42.3 ± 1.6</td>
<td>41.6 ± 1.5</td>
</tr>
<tr>
<td>ALP</td>
<td>263 ± 10.3</td>
<td>260 ± 19.3</td>
</tr>
<tr>
<td>LDH</td>
<td>815 ± 168</td>
<td>605 ± 101</td>
</tr>
</tbody>
</table>

Renal screening tests

Urea, Creatinine, Uric acid, Calcium, and Phosphorus were tested in order to study the effect of the DCAE on renal function. Data, presented in Table 4, showed that the concentrations of creatinine, uric acid, calcium, and phosphorus were similar in both control and treated groups. However, a slight but significant increase in serum concentration of urea in the treated group was observed.

Table 4: Serum concentration (mg/dl) of urea, creatinine, uric acid, calcium and phosphorus after one month supplementation with DCAE. Values denote mean ± SEM (n=15).

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Control group</th>
<th>Treated group</th>
</tr>
</thead>
<tbody>
<tr>
<td>Urea</td>
<td>26.41 ± 0.70</td>
<td>28.87 ± 0.91 *</td>
</tr>
<tr>
<td>Creatinine</td>
<td>0.28 ± 0.01</td>
<td>0.30 ± 0.01</td>
</tr>
<tr>
<td>Uric acid</td>
<td>0.78 ± 0.07</td>
<td>0.79 ± 0.05</td>
</tr>
<tr>
<td>Calcium</td>
<td>10.27 ± 0.06</td>
<td>10.24 ± 0.08</td>
</tr>
<tr>
<td>Phosphorus</td>
<td>6.96 ± 0.22</td>
<td>6.78 ± 0.14</td>
</tr>
</tbody>
</table>
Glucose, insulin and amylase tests

The mean fasting serum concentrations of glucose, insulin and amylase as determined following DCAE supplementation, showed similar values in both control and treated groups. Data are shown in Table 5.

Table 5: Serum concentration of glucose (mg/dl), insulin (ng/ml) and amylase (U/L) in control and treated groups after one month supplementation with DCAE. Values denote mean ± SEM (n=15).

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Control group</th>
<th>Treated group</th>
</tr>
</thead>
<tbody>
<tr>
<td>Glucose</td>
<td>94.3 ± 3.21</td>
<td>96.3 ± 3.89</td>
</tr>
<tr>
<td>Insulin</td>
<td>0.51 ± 0.036</td>
<td>0.59 ± 0.052</td>
</tr>
<tr>
<td>Amylase</td>
<td>1456 ± 53</td>
<td>1363 ± 49</td>
</tr>
</tbody>
</table>

DISCUSSION

The effect of DCAE upon blood lipid profile was examined in the presence of an atherogenic diet administered to rats for a period of one month. Extract intake did not appear to have significant effect on blood lipid profile except for a 7.5% decrease in HDL-C cholesterol. In spite of the observed decrease in HDL-C, the ratio of LDL-C/HDL-C was not significantly affected. Also, the extract did not affect plasma total apo B concentration, indicating the absence of effect on the metabolism of VLDL-C and LDL-C. The aqueous extract of *Daucus carota* seems to maintain the structural integrity of the hepatocellular membrane since no significant changes in hepatic enzyme activities were observed in both control and treated groups. Although not significant, the group that received the extract showed relatively reduced liver enzyme activities, thereby supporting previous studies where *Daucus carota* exhibited hepatoprotective activity against CCL4 induced liver toxicity (Handa, 1986). The serum level of urea was significantly increased after one month supplementation of DCAE (<10% increase). Creatinine, a by-product of muscle energy metabolism, is usually a better marker of kidney function than urea. Since creatinine and other markers of kidney function like calcium, phosphorus, and uric acid (Cameron, 1996) were similar in both experimental and control groups, the observed increase in serum urea may be the result of dehydration rather than an impaired kidney function. This is supported by previous studies showing that *Daucus carota* possesses diuretic and antilithic actions (Hoffmann, 1990). While there is no available literature concerning effects of the plant extract on pancreatic function, the present study showed that DCAE has no effects on the endocrine and exocrine pancreatic function.
function, since serum levels of glucose and insulin and amylase activity were not affected.

The present results revealed that both DCAE and DCME possess significant anti-inflammatory activity against acute and chronic inflammation. The exhibited anti-inflammatory activity of the aqueous extract in both models was comparable to diclofenac. Several inflammatory mediators such as kinins, prostaglandins, and serotonin may account for the edema formation caused by subplantar formalin or carrageenan injection (Jose et al., 2004). The increased synthesis of prostaglandins could be due to increased release of arachidonic acid from the membrane phospholipids and/or the up-regulation of cyclooxygenase-2 enzyme. Preliminary phytochemical analysis of the plant indicated that it contains terpenes and flavonoids. Flavonoids, in general, have been recognized to possess anti-inflammatory activity by decreasing the release of inflammatory mediators, stabilizing cell membranes, and depressing cyclooxygenase activity (Martini et al., 2004, Damas et al, 1985; Ferrandiz and Alcaraz, 1991; Kim et al, 1998; Laughton et al, 1991; Yoshimoto et al, 1983). The results corroborate the use of *Daucus carota* in the Indian traditional medicine for the treatment of variety of ailments, including inflammation (Kirtikar and Basu, 1993).

Ethanol, when given intragastrically to rats, produces severe gastric hemorrhagic erosions. The genesis of ethanol-induced gastric lesions is multifactorial, including depletion of gastric wall mucus due to mucosal leukotriene release (Al-Harbi et al., 1997). DCAE and DCME showed significant reduction in ethanol induced gastric ulcers, with the latter exhibiting better curative ratio when compared with cimetidine and DCAE. This gastro-protective effect may be attributed to flavonoids and tannins present in the plant extracts. Several mechanisms have been proposed to explain the gastro-protective effect of flavonoids; these include increase of mucosal prostaglandin content (Alcaraz & Hoult, 1985) and decrease of histamine secretion from mast cells by inhibition of histidine decarboxylase (Bronner & Landry, 1985). In addition, flavonoids are reported to be free radical scavengers (Salvayre et al., 1982) thereby conferring an important role in preventing ulcerative and erosive lesions of the gastrointestinal tract. Tannins are also known to "tan" the outermost layer of the gastric mucosa and render it less permeable and more resistant to chemical and mechanical injury or irritation (Asuzu & Onu, 1990). It should be noted that substances such as flavonoids that possess both anti-inflammatory and anti-ulcer activity are of particular therapeutic importance as most of the anti-inflammatory drugs used in modern medicine are ulcerogenic.

The treatment of infectious diseases with current antimicrobial agents is faced with increased incidence of bacterial resistance (Finch, 1998; Kunin, 1993). Phytochemicals are still recognized as a potential source of antimicrobial agents (Evans et al., 2002). The present study showed weak inhibitory activity of DCME.
(20 mg/ml) against *Staphylococcus aureus* meti S and meti R (Gram-positive bacteria) and was ineffective when tested against Gram negative bacteria. *Daucus carota* is rich in flavonoids and tannins, both of which possess antimicrobial activity (Basri and Fan, 2005; Cushnie and Lamb, 2005). This may explain the traditional use of plant volatile oil as urinary antiseptic and for cystitis and prostatitis treatments (Hoffmann, 1990; Mills, 1994).

In conclusion, *Daucus carota* crude aqueous and methanol extracts appear to have promising beneficial effects against acute/chronic inflammations and gastric ulcers. Additionally, the plant showed no negative outcome on the functions of liver, kidney and pancreas and lipid profile except for a slight decrease in serum HDL-C level. Further investigations are needed to determine the active compound(s) responsible for gastroprotection and anti-inflammatory activities and to elucidate the mechanisms of action involved.

**REFERENCES**


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