Hypolipidemic activity of a hydroalcoholic extract of *Cyperus scariosus* Linn. root in guinea pigs fed with a high cholesterol diet

Hiren M. Chawda 1, Divyesh R. Mandavia 2, Pravin H. Parmar 3, Seema N. Baxi 2, Chandrabhanu R. Tripathi 2*

1 Pacific Medical College & Hospital, Udaipur, Rajasthan, India; 2 Government Medical College, Bhavnagar-364001, Gujarat, India; 3 Analytical Chemistry Division, Department of Chemistry, Maharaja Krishnakumarsinhji Bhavnagar University, Bhavnagar 364002, India

Available online 20 Nov. 2014

**[ABSTRACT]**

Lipid-lowering and antioxidant activities of a hydroalcoholic extract of *Cyperus scariosus* Linn. root (HCS) were evaluated in guinea pigs fed with a high cholesterol diet. Serum lipid profile (total cholesterol, triglycerides, LDL-C, VLDL-C, and HDL-C), atherogenic indices and serum enzymes (ALT, AST, ALP, LDH, and CK-MB) were performed in each group at 0 days and at the end of 60 days. Histological study of liver and kidney was done in groups 1, 2, 5, 6 and 7. The total phenolic and flavonoid content in HCS and its antioxidant activity were evaluated by the DPPH assay. Both doses of HCS decreased serum lipid profile and atherogenic indices (*P* < 0.05). HCS has lipid lowering, immunosuppressive and antioxidant properties, and may have value in atherosclerosis prevention. The higher dose of HCS also reduced serum AST, ALP, and LDH levels and rosuvastatin increased AST and ALP levels (*P* < 0.05). Histology of the liver showed decreased lipid accumulation and improvement in hepatocytes in HCS-treated animals. The antioxidant activity of HCS may be responsible for its lipid lowering and cytoprotective action. HCS had significant lipid lowering and antioxidant activity, which; may be due to the phenolic compounds. HCS may be a safe and cost effective alternative to current statin therapy for patients with dyslipidaemia.

**[KEY WORDS]** Antioxidant activity; *Cyperus scariosus*; Dyslipidemia; Statin

**[CLC Number]** R965  **[Document code]** A  **[Article ID]** 2095-6975(2014)011-0819-08

**Introduction**

In India, 29% of deaths and 11% of all disability adjusted life years (DALYs) occur due to cardiovascular disease (CVD) in all ages (2005) [1-2]. CVD-related deaths accounted for the higher loss in potentially productive years of life lost (PPYLL) in middle-aged people of India than other countries. CVD could be a reason for the loss of 17.9 million PPYLL by 2030, in India [1, 3]. Metabolic syndrome is a group of interrelated metabolic abnormalities which includes insulin resistance, diabetes, obesity, elevated blood pressure, and dyslipidaemia. It plays an important role in increasing the risk of CVD associated morbidity and mortality [4]. The association between CVD and higher low-density lipoprotein cholesterol (LDL-C) is well-known and plays a major role in the development of CVD [5-6]. Dyslipidaemia induces oxidative stress in the liver, heart, and kidney by reactive oxygen species, which plays a significant role in the etiology of atherosclerosis and CVD [7-10].
Cyperus scariosus Linn. (Cyperaceae) is commonly known as “Nagarmotha” in Hindi, and is distributed in humid places in Bengal, Sundarban, Uttar Pradesh, and Eastern and Southern parts of India [10]. It is useful in a variety of diseases including diarrhea, epilepsy, gonorrhea, syphilis, and liver damage [12]. Plant products may be useful as the safest way to burn excess calories [13]. In India, the root of C. scariosus is used as a folk medicine for obesity and hyperlipidemia. So, the current study was planned to evaluate the hypolipidemic activity of C. scariosus root in normal and high fat diet fed guinea pigs, and also to know its implication in metabolic syndrome.

Materials and Methods

Animals

Forty-two guinea pigs weighing 520–860 g of both genders and female mice were used for the experiments. The animals were procured from the Central Animal House of Government Medical College, Bhavnagar, Gujarat, India.

Drugs and chemicals

Cholesterol powder (analytical grade): Purchased from High Purity Laboratory Chemicals Pvt. Ltd. (Mumbai). Rosuvastatin Calcium powder: Gift sample from Torrent Pharmaceuticals Ltd., Torrent research center, Ahmadabad, Gujarat (Batch no: ARD2110109). Hydroalcoholic extract of Cyperus scariosus Linn. root (HCS) was obtained from Kuber Impex Limited, Indore, India. Gallic acid, quercetin, and 2, 2'-diphenyl-1-picrylhydrazyl hydrate (DPPH) were procured from Sigma Life Science (Sigma-Aldrich), New Delhi, India. All other chemicals and solvents used were of analytical grade.

Acute toxicity study

Acute toxicity of HCS was determined according to the Organization for Economic Co-operation and Development (OECD) 423 guideline [14]. Female mice were given a single oral dose each of 5 mg to 2 000 mg·kg\(^{-1}\) body weight. Animals were observed for signs of toxicity, behaviour changes and mortality for 14 days.

Determination of total phenols and flavonoids

Total phenolic content was determined according to Folin-Ciocalteu method [15]. The absorbance was measured at 730 nm using a double beam UV-Visible Spectrophotometer. Gallic acid was used as a standard. The concentration of phenolic compounds was calculated according to the following equation-1 obtained from the standard gallic acid (5 to 50 µg) curve.

\[
\text{Absorbance} = 0.005 \times \text{GAE in } \mu\text{g} + 0.035 (R^2 = 0.935) \quad (1)
\]

Flavonoid content in the extract was determined by a colorimetric method [16]. The absorbance was read at 510 nm using UV spectrophotometer. Quercetin was used as a standard. The concentration of flavonoids was calculated according to the following equation-2 obtained from the standard quercetin (20 to 100 µg) curve.

\[
\text{Absorbance} = 0.001 \times \text{Quercetin in } \mu\text{g} + 0.012 (R^2 = 0.992) \quad (2)
\]

DPPH radical scavenging assay

In this assay, free radical scavenging activity of HCS was determined by measuring the bleaching of a purple-colored methanol solution of 2, 2’-diphenyl-1-picrylhydrazyl hydrate (DPPH). The absorbance was measured at 517 nm using a UV spectrophotometer. Butylatedhydroxytoluene (BHT) was used as the positive control, and methanol was used as negative [17]. The radical scavenging activity (RSA) was calculated as a percentage of DPPH discoloration using the equation-3.

\[
\% \text{ RSA} = \frac{(A_C - A_S)/A_C} \times 100 \quad (3)
\]

Where \(A_C\) and \(A_S\) are the absorbance of the control (containing all reagents, except the test compound) and test compound, respectively.

Methodology

The study was conducted at Department of Pharmacology, Government Medical College, Bhavnagar, Gujarat, after approval from the Institutional Animal Ethics Committee of the same institute. All the experiments were performed according to the Committee for the Purpose of Control and Supervision of Experiments on Animals (CPCSEA), New Delhi guidelines. Guinea pigs were housed in stainless steel cages and kept under normal light and temperature conditions (i.e. 12 h light/dark cycle; 24 ± 2 °C). Standard laboratory feed and water \textit{ad libitum} were provided throughout the experiments. After proper acclimatization for 15 days, they were divided into seven groups of six guinea pigs.

Groups of animals

Group 1: Normal diet plus distilled water (Vehicle control); Group 2: High fat diet plus distilled water (High fat control); Group 3: Normal diet plus a lower dose of HCS (125 mg·kg\(^{-1}\)); Group 4: Normal diet plus a higher dose of HCS (250 mg·kg\(^{-1}\)); Group 5: High fat diet plus a lower dose of HCS (125 mg·kg\(^{-1}\)); Group 6: High fat diet plus a higher dose of HCS (250 mg·kg\(^{-1}\)); Group 7: High fat diet plus rosuvastatin (1.5 mg·kg\(^{-1}\)).

All guinea pigs were fed diet according to the diet plan of their groups during the 60 days of the experimental period. Distilled water, rosuvastatin, and both doses of HCS were given from day 30 to day 60, as indicated for the above groups. All treatments were given orally by gavage feeding tube daily in the morning, in the fasting state to ensure maximum absorption.

Diet composition

Normal diet: In the morning: mixtures of cereals and pulses (60% wheat plus 25% bengal gram plus 15% peanuts), total 50 g/animal. In the evening: green leafy vegetables, 30 g/animal.

High fat diet: in the morning: cholesterol powder (500 mg·kg\(^{-1}\)) mixed with 10 g of wheat and bengal gram flour followed by 40 g of the above mixtures of the normal diet/animal. In the evening: green leafy vegetables, 30 g/animal.

After overnight fasting, a blood sample was collected at day and day 60 from the lateral saphenous vein of the hind paw of each animal. Blood samples were analyzed for the serum lipid profile, liver and cardiac enzymes in the Clinical Biochemistry Laboratory of the institute which is accredited by National Accreditation Board for Testing and Calibration Laboratories (NABL), New Delhi. Liver and kidney were obtained from each animal of groups 1, 2, 5, 6, and 7 for his-
histopathological analysis. Experimental animals of groups 3 and 4 were fed a normal diet, so they were not sacrificed according to advice from the ethics committee to reduce number of animals sacrificed after giving anaesthesia.

**Outcome measures**

**Serum lipid profile**

The serum levels of total cholesterol, triglycerides and high-density lipoprotein cholesterol (HDL-C) were analyzed. The Friedewald method was used to calculate LDL-C and very low-density lipoprotein cholesterol (VLDL-C) \[^{18}\]. The atherogenic indices were calculated from using equations 4, 5, and 6 \[^{19}\].


\[
\text{Cardiac Risk Ratio (CRR)} = \frac{\text{Total Cholesterol}}{\text{HDL-C}}
\]

Atherogenic Coefficient (AC) = \(\frac{\text{Total Cholesterol} - \text{HDL-C}}{\text{HDL-C}}\)

Atherogenic Index of Plasma (AIP) = \(\log(\frac{\text{Triglyceride}}{\text{HDL-C}})\)

Evaluation of Liver and Cardiac Enzymes

Alanine aminotransferase (ALT), aspartate aminotransferase (AST), and alkaline phosphatase (AP) levels in serum were analyzed to evaluate any functional abnormality of the liver. Cardiac function was assessed by measuring serum level of lactate dehydrogenase (LDH) and creatinine kinase MB (CK-MB).

**Effect of HCS on the weight of the animals**

The weight of each animal was recorded before and after sacrifice according to advice from the ethics committee to reduce number of animals. Experimetal animals of groups 3 vs high fat control, ANOVA followed by Tukey-Kramer multiple comparison test; **P < 0.05 vs baseline level in high fat control, paired t-test.

**Table 1** Effect of each treatment strategy on all atherogenic indices, HDL-C and VLDL-C in guinea pigs (\(\bar{x} \pm \text{SEM}, n = 6\))

<table>
<thead>
<tr>
<th>Treatment Groups</th>
<th>Time Period</th>
<th>HDL Cholesterol (/mg·dL(^{-1}))</th>
<th>VLDL Cholesterol (/mg·dL(^{-1}))</th>
<th>Cardiac Risk Ratio</th>
<th>Atherogenic Coefficient</th>
<th>Atherogenic Index of Plasma</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vehicle control</td>
<td>Base line</td>
<td>4.5 ± 0.6</td>
<td>18.2 ± 1.3</td>
<td>11 ± 0.9</td>
<td>10 ± 0.9</td>
<td>1.3 ± 0.04</td>
</tr>
<tr>
<td></td>
<td>60 Days</td>
<td>4.3 ± 0.4</td>
<td>17.7 ± 1.1</td>
<td>11 ± 0.9</td>
<td>7.3 ± 0.9</td>
<td>1.3 ± 0.04</td>
</tr>
<tr>
<td>High fat control</td>
<td>Base line</td>
<td>5.1 ± 0.7</td>
<td>16.8 ± 1.4</td>
<td>9.8 ± 1.6</td>
<td>8.8 ± 1.6</td>
<td>1.2 ± 0.1</td>
</tr>
<tr>
<td></td>
<td>60 Days</td>
<td>4.9 ± 0.6</td>
<td>21.5 ± 1.4(^{\ast})</td>
<td>13 ± 1.7(^{\ast})</td>
<td>12 ± 1.7(^{\ast})</td>
<td>1.3 ± 0.1</td>
</tr>
<tr>
<td>Normal diet plus</td>
<td>Base line</td>
<td>5.5 ± 0.3</td>
<td>16.7 ± 0.7</td>
<td>8.1 ± 0.8</td>
<td>7.1 ± 0.8</td>
<td>1.2 ± 0.02</td>
</tr>
<tr>
<td>HCS (125 mg·mL(^{-1}))</td>
<td>60 Days</td>
<td>5.1 ± 0.6</td>
<td>18.3 ± 1.4</td>
<td>9.6 ± 1.1</td>
<td>7.1 ± 0.7</td>
<td>1.3 ± 0.03</td>
</tr>
<tr>
<td>Normal diet plus</td>
<td>Base line</td>
<td>5.6 ± 0.4</td>
<td>19.4 ± 1.5</td>
<td>9.1 ± 0.8</td>
<td>8.1 ± 0.8</td>
<td>1.2 ± 0.1</td>
</tr>
<tr>
<td>HCS (250 mg·mL(^{-1}))</td>
<td>60 Days</td>
<td>5.5 ± 0.1</td>
<td>18.4 ± 2.2</td>
<td>12 ± 2.5</td>
<td>7.5 ± 0.8</td>
<td>1.3 ± 0.1</td>
</tr>
<tr>
<td>High fat diet plus</td>
<td>Base line</td>
<td>4.6 ± 0.7</td>
<td>25.9 ± 0.5</td>
<td>12 ± 2.3</td>
<td>11 ± 2.3</td>
<td>1.5 ± 0.1</td>
</tr>
<tr>
<td>HCS (125 mg·mL(^{-1}))</td>
<td>60 Days</td>
<td>9.3 ± 2.2</td>
<td>16.1 ± 1.3(^{\ast})</td>
<td>6.8 ± 0.7(^{\ast})</td>
<td>5.8 ± 0.7(^{\ast})</td>
<td>0.9 ± 0.1</td>
</tr>
<tr>
<td>High fat diet plus</td>
<td>Base line</td>
<td>2 ± 0.2</td>
<td>33.2 ± 2.5</td>
<td>30 ± 5.5</td>
<td>11 ± 5.5</td>
<td>1.5 ± 0.1</td>
</tr>
<tr>
<td>HCS (250 mg·mL(^{-1}))</td>
<td>60 Days</td>
<td>5.8 ± 0.8</td>
<td>14.5 ± 5.6(^{\ast})</td>
<td>8.5 ± 0.9(^{\ast})</td>
<td>6.6 ± 0.8(^{\ast})</td>
<td>1.1 ± 0.1</td>
</tr>
<tr>
<td>High fat diet plus</td>
<td>Base line</td>
<td>3.5 ± 0.2</td>
<td>16.6 ± 1.3</td>
<td>13 ± 0.9</td>
<td>12 ± 0.9</td>
<td>1.4 ± 0.1</td>
</tr>
<tr>
<td>rosuvastatin (1.5 mg·mL(^{-1}))</td>
<td>60 Days</td>
<td>11.6 ± 1.3(^{\ast})</td>
<td>15 ± 1.2(^{\ast})</td>
<td>4.1 ± 0.2(^{\ast})</td>
<td>3.1 ± 0.3(^{\ast})</td>
<td>0.8 ± 0.1</td>
</tr>
</tbody>
</table>

VLDL = Very low density lipoprotein; HDL = High density lipoprotein; HCS: Hydroalcoholic extract of *Cyperus scariosus* root; \(^{\ast}\) P < 0.05 vs high fat control, ANOVA followed by Tukey-Kramer Multiple comparison test; \(^{\ast}\) P < 0.05 vs baseline level in high fat control, paired t-test.
increase in the serum total cholesterol, serum triglyceride, LDL-C, VLDL-C level, and atherogenic indices at the end of 60 days as compared to the baseline lipid profile, in high fat control ($P < 0.05$).

There were no significant changes among lipid parameters in the normal diet-fed group treated with both doses of HCS. In the high fat diet-fed group treated with the lower dose of HCS, AST and ALP levels were significantly decreased in the high fat diet-fed group treated with the higher dose of HCS ($P < 0.05$). The rosuvastatin-treated group increased AST and ALP levels at the end of 60 days as compared to baseline ($P < 0.05$).

Normal histological structures of liver and kidney are shown in Figs. 4A and 5A, respectively. In the high fat control, histological study of liver showed diffuse areas and ballooning degeneration of hepatocytes (grade 3+, 4+), steatosis (up to grade 2.5), fatty changes (midzone and periportal) and congestion of central vein and hepatic sinusoids (Fig. 4.B) in all animals. Histological study of the kidney showed the normal structure (Fig. 5B). In the high fat diet-fed group treated with HCS, liver histology showed lesser degrees of ballooning degeneration (grade 1+ and grade 2+; Fig. 4C), no fatty changes and kidney examination of this group animals were
normal (Fig. 5C). In the rosuvastatin-treated group, no morphological changes in the liver and kidney were detected (Figs. 4D and 5D).

The increase in mean body weight of all groups at the end of 60 days is shown in Table 3 ($P < 0.05$). The extent of body weight gain was not statistically significant between the groups. The total phenolic content, flavonoid content and DPPH IC$_{50}$ of HCS roots are shown in Table 4.

**Discussion**

In the present research, the hypolipidemic activity of HCS in guinea pigs was examined because the lipoprotein metabolism of guinea pigs is closer to human. Several lines of evidence show that guinea pigs are admirable models to assess hypolipidemic activity and lipoprotein metabolism of drugs [20].

<table>
<thead>
<tr>
<th>Table 2</th>
<th>Effect of each treatment strategy on serum enzymes in guinea pigs ($\bar{x} \pm$ SEM, $n = 6$)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Treatment Groups</td>
<td>Time Period</td>
</tr>
<tr>
<td>Base line</td>
<td>60 Days</td>
</tr>
<tr>
<td>Vehicle control</td>
<td>Base line</td>
</tr>
<tr>
<td>High fat control</td>
<td>Base line</td>
</tr>
<tr>
<td>Normal diet plus HCS (125 mg·kg$^{-1}$)</td>
<td>Base line</td>
</tr>
<tr>
<td>Normal diet plus HCS (250 mg·kg$^{-1}$)</td>
<td>Base line</td>
</tr>
<tr>
<td>High fat diet plus HCS (125 mg·kg$^{-1}$)</td>
<td>Base line</td>
</tr>
<tr>
<td>High fat diet plus HCS (250 mg·kg$^{-1}$)</td>
<td>Base line</td>
</tr>
<tr>
<td>High fat diet plus rosuvastatin (1.5 mg·kg$^{-1}$)</td>
<td>Base line</td>
</tr>
</tbody>
</table>

ALT: Alanine aminotransferase, AST: Aspartate aminotransferase, AP: Akaline phosphatase, LDH: Lactate dehyrogenase, CK-MB: Creatine kinase-MB, HCS: Hydroalcoholic extract of *Cyperus scariosus* root, * < 0.05 vs high fat control, ANOVA followed by Tukey-Kramer multiple comparison test; **P < 0.05 vs baseline level, paired t-test.

![Fig. 4](image1.png) **Fig. 4** Histological appearances of liver of guinea pig (H&E 40×); (A) C, H, and S indicates normal structure of central vein, hepatocytes with round nucleus, and sinusoid, respectively; (B) Diffuse areas of ballooning degeneration and fatty changes (Grade 4+) (arrow) are shown in high fat diet-fed guinea pig; (C) Encircled area shows no fatty changes, only ballooning degeneration (Grade 2+) in high fat diet-fed guinea pig treated with HCS (250 mg·kg$^{-1}$); (D) No histological changes were seen in the high fat diet-fed guinea pig treated with rosuvastatin (1.5 mg·kg$^{-1}$)

![Fig. 5](image2.png) **Fig. 5** Histological appearances of kidney of guinea pig (H&E 40×); (A) G, T, and I indicate normal structure of glomeruli, tubule and interstitium, respectively; (B) No fatty changes were seen in high fat diet-fed guinea pig; (C) and (D) indicate normal histological appearance of kidney in high fat diet fed guinea pig treated with HCS (250 mg·kg$^{-1}$) and rosuvastatin (1.5 mg·kg$^{-1}$), respectively.
High fat diet plus HCS (125 mg·kg$^{-1}$)

611.6 ± 21.5

Similar types of changes were also observed in rats fed by normal diet-fed Wistar rats \[25\]. HCS has shown antioxidant effect in guinea pigs fed with normal diet (Table 1, Figs. 1–3). Rosuvastatin decreased the lipid profile in the fatty changes in guinea pigs, and is supported by previous studies \[21, 24\].

The data indicate that HCS treatment for 30 days was powerful enough to decrease both the serum and hepatic lipid profile, and provided liver protection from hypercholesterolemia.

### Table 3  Effect of each treatment strategy on weight of guinea pigs ($\bar{x} \pm$ SEM)

<table>
<thead>
<tr>
<th>Treatment Group</th>
<th>Weight of Animal in grams</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Baseline</td>
</tr>
<tr>
<td>Normal control</td>
<td>536.7 ± 6.1</td>
</tr>
<tr>
<td>High fat control</td>
<td>633.3 ± 54.8</td>
</tr>
<tr>
<td>Normal diet plus HCS (125 mg·kg$^{-1}$)</td>
<td>596.6 ± 31.7</td>
</tr>
<tr>
<td>Normal diet plus HCS (250 mg·kg$^{-1}$)</td>
<td>603.3 ± 35.7</td>
</tr>
<tr>
<td>High fat diet plus HCS (125 mg·kg$^{-1}$)</td>
<td>618.3 ± 20.7</td>
</tr>
<tr>
<td>High fat diet plus HCS (250 mg·kg$^{-1}$)</td>
<td>611.6 ± 21.5</td>
</tr>
<tr>
<td>High fat diet plus rosuvastatin (1.5 mg·kg$^{-1}$)</td>
<td>660 ± 32.8</td>
</tr>
</tbody>
</table>

HCS: Hydroalcoholic extract of Cyperus scariosus root

### Table 4  Total phenolic and flavonoid content in HCS and their DPPH scavenging activity (IC$_{50}$ in μg·mL$^{-1}$) ($\bar{x} \pm$ SEM)

<table>
<thead>
<tr>
<th>Extract</th>
<th>HCS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total phenolic content (mg·g$^{-1}$)</td>
<td>66.7 ± 23.1</td>
</tr>
<tr>
<td>Total flavonoid content (mg·g$^{-1}$)</td>
<td>73.3 ± 11.5</td>
</tr>
<tr>
<td>DPPH scavenging activity (IC$_{50}$ in μg·mL$^{-1}$)</td>
<td>7.9 ± 0.4’</td>
</tr>
</tbody>
</table>

HCS: Hydroalcoholic extract of Cyperus scariosus root; DPPH: 2, 2’-Diphenyl-1-picryl-hydrazyl hydrate; IC$_{50}$: Fifty percent inhibition of concentration; * DPPH scavenging activity (IC$_{50}$ in μg·mL$^{-1}$) of butylated hydroxytoluene (BHT): 70.57 ± 2.98

The results of the present study revealed that feeding with high fat diet for 60 days increased parameters of serum lipid profile, atherogenic indices, and induced histopathological changes in the liver (Table 1, Figs. 1–3 and 4B). Similar types of changes were also observed in rats fed by high fat diet \[7\]. High fat diet induces decreased numbers of the hepatic LDL-C receptors, and also decreases the rate of cholesterol removal. Liver synthesizes cholesterol ester-rich VLDL-C due to cholesterol rich diet supplementation \[21-23\]. The chosen study period of 60 days was sufficient to produce fatty changes in guinea pigs, and is supported by previous studies \[21, 24\].

The present study revealed that HCS has no lipid lowering effect in guinea pigs fed with normal diet (Table 1, Figs. 1–3). Rosuvastatin decreased the lipid profile in the normal diet-fed Wistar rats \[23\]. HCS has shown anti-hyperlipidemic effect, it may not alter normal lipid profile as compared to rosuvastatin.

It has been widely known that a rise of cholesterol level in blood can lead to atherosclerosis \[29\]. Foam cells and fatty streaks are trademarks of early atherosclerosis and their formation occurred on vessels by LDL-C oxidation \[28\]. In the present study, administration of HCS (125 and 250 mg·kg$^{-1}$) in guinea pigs reduces serum total cholesterol, serum triglyceride, LDL-C, VLDL-C, and atherogenic indices. HCS had not shown any dose dependent action on lipid profile in the present study (Table 1, Figs. 1–3). The chloroform fraction of C. scariosus inhibits IL-2 production by CD4$^+$ T cells \[28\]. IL-2, a growth factor for Th1 cytokines, decrease IL-2 secretion is responsible for decrease secretion of IFN-$\gamma$ by CD8$^+$ T cells \[29\]. IFN-$\gamma$ activates macrophage \[29\], its inhibition causing reduction in macrophage activity (phagocytosis) \[30-31\]. So, numbers of foam cells forming may decrease in blood vessels. Medicinal plants with phenolic compounds have antioxidant activity \[32\]. Phenolic compounds and flavonoids were present in the extract. It also has free radical scavenging activity which was measured by the DPPH assay (Table 4). HCS might be suppressing lipid peroxidation. HCS has lipid lowering (Table 1, Figs. 1–3), immunosuppressive \[27\], and antioxidant property (Table 4), so put in self value in atherosclerosis prevention.

Hypercholesterolemia endangers vital organs like the liver and heart. A high fat diet increases the retention of lipid in the liver, followed by hepatic steatosis and reduce hepatic functions \[7\]. This study results showed that a high fat diet suppressed hepatic and cardiac functions which were expressed as augmentation of serum levels of AST, ALT, AP, LDH, and CK-MB as compared to the normal diet fed group ($P < 0.05$; Table 2). HCS reduced the AST, ALP, and LDH levels as compared to high fat control ($P < 0.05$; Table 2).

The present study has a number of limitations. The molecular mechanism involved in lipid lowering activity of HCS is not known, and the phenolic compounds responsible for both actions have not been identified.

### Conclusions

In conclusion, HCS was able to decrease the high serum lipid profile in guinea pigs fed with a high fat diet for 60 days. The data indicate that HCS treatment for 30 days was powerful enough to decrease both the serum and hepatic lipid profile, and provided liver protection from hypercholesterolemia.
The phenolic compounds of HCS might be responsible for both the lipid-lowering and antioxidant actions. Further studies are recommended to understand their mechanism of action in different preclinical and clinical settings.

Acknowledgement

We would like to thank Dr. S. K. Mehta, Department of Botany and Dr. J. P. Mehta, Department of Chemistry of Maharaja Krishnakumarsinhji Bhavnagar University, Bhavnagar for plant authentication and providing guidance to evaluate the antioxidant activity of HCS, respectively. We would also like to thank Torrent Pharmaceuticals Ltd., Torrent research center, Ahmedabad for providing rosuvastatin calcium powder as a free gift sample.

References

[29] Wang J, Wakeham J, Harkness R, et al. Macrophages are a significant source of type 1 cytokines during mycobacterial in-


**Cite this article as:** Hiren M. Chawda, Divyesh R. Mandavia, Pravin H. Parmar, Seema N. Baxi, Chandrabhanu R. Tripathi. Hypolipidemic activity of a hydroalcoholic extract of *Cyperus scariosus* Linn. root in guinea pigs fed with a high cholesterol diet [J]. *Chinese Journal of Natural Medicines*, 2014, **12** (11): 819-826