Antihyperlipidemic and antiobesity activity of ethanolic extract of *Benincasa hispida* fruits on hyperlipidemic rats

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ABSTRACT

Hyperlipidemia and obesity are the common and challenging health problems throughout the world. These are the conditions in which increased lipid levels in blood are the risk factors for atherosclerosis, coronary artery diseases and cerebral vascular diseases. In the present study the absolute ethanolic extract of *Benincasa hispida* was examined for its antihyperlipidemic effect and antiobesity activity in olive oil induced Wistar albino rats. Normal and olive oil induced hyperlipidemic rats were pretreated with single daily administration of 10ml/kg distill water, 20mg/kg simvasatin, 200mg/kg and 400mg/kg *Benincasa hispida* fruit extract orally, for 28 days for every 24 hours in each group (II, III, IV and V) except control. The activity was assessed by estimation of body weight, liver weight, food intake, serum lipid profile etc. The bio chemical parameters like Total cholesterol (TC), Triglycerides (TG), High density lipoprotein cholesterol (HDL-C), Low density lipoprotein cholesterol (LDL-C), Very low density lipoprotein cholesterol (VLDL-C), atherogenic index, Blood glucose levels and histopathology of liver were also assessed. The graded doses of *Benincasa hispida* extract significantly reduced the weight gain pattern of body and liver and causes dose related reduction in serum lipids, atherogenic index and blood glucose. The present study demonstrated that *Benincasa hispida* extract shows both antihyperlipidemic and antiobesity effects, which may be partly mediated by anorectic effect, probably through CNS mediation, with no effect on gastric emptying, and inhibition of denovo biosynthesis of cholesterol.

Key Words: Hyperlipidemia, *Benincasa hispida*, obesity, atherogenic index.

INTRODUCTION

Obesity is a common chronic disorder of carbohydrate and fat metabolism which results in accumulation of excessive fat deposition in adipose tissue and other internal organs such as liver, heart, skeletal muscle, and pancreatic islet etc [1]. Obesity remains a major global public health issue because of its increasing prevalence in all sex, age, ethnicity or race groups [2]. Hyperlipidemia is a major cause of atherosclerosis and atherosclerosis-associated conditions, such as coronary heart disease (CHD), ischemic cerebrovascular disease and peripheral vascular disease. Although the incidence of these atherosclerosis-related events has declined in the United States, these conditions still account for the majority of morbidity and mortality among middle-aged and older adults. The incidence and absolute number of annual events will likely increase over the next decade because of the epidemic of obesity and the aging of the U.S. Population. Dyslipidemias, including Hyperlipidemia (hypercholesterolemia) and low levels of high-density-lipoprotein cholesterol (HDLC), are major causes of increased atherogenic risk both genetic disorders and lifestyle (sedentary behavior and diets high in calories, saturated fat, and cholesterol) contribute to the dyslipidemias seen in developed countries around the world [3].

*Benincasa hispida* is wide-spreading, hairy, annual vine with branched tendrils reaching a length of 4-8 meters. Leaves are rounded or kidney-shaped, 10-20 cm diameter, 5-7 lobed, heart-shaped at the base. Flowers are large and yellow, with a densely hairy bell-shaped calyx tube. The five petals are spreading, 3-5 cm long. Fruit is ellipsoid or ovoid, 25-40 cm long, green, with a white and waxy bloom. The seeds are many, oblong, and compressed. Cultivated for the edible fruit distributed wildly. *Benincasa hispida* fruit is an

*Corresponding Author Address: Dr. Krishna Mohan Chinnala, Professor & Principal, Department of Pharmacology, St. John College of Pharmacy, Yellapur, Hasanparthy, Warangal, TELANGANA, INDIA-506371.
importantly a source of water-soluble polysaccharides. Hemicellullosic polysaccharides of Benincasa are chiefly composed of triterpenol acetates and triterpenols. It also contains the rich amounts of saponins, arginine, aspartic acid, glutamic acid, asparagine, cystine, L-leucine and guaridine. A new pentacyclictriterpene, isomultiflorenyl-acetate, has been isolated from plant [4]. *Benincasa hispida* commonly used as a general tonic, aphrodisiac, rejuvenative and also a brain tonic. It is useful in iron deficiency anemia, inhibits mental instability. Fruits are laxative, demulcent, cooling and diuretic. Juice of the fruit is antidote for mercury and alcohol poisoning [5]. Many scientific studies reported that various parts of the plant extract possess antiulcer activity, anticonvulsant properties, hepato protective activity, diuretic activity, antioxidant activity, anthelmintic activity and anti-inflammatory activity [6]. In the present research work we have studied the effects of ethanolic fruit extract for its antihyperlipidemic and antiobesity activity on hyperlipidemic Wistar rats.

**MATERIALS AND METHODS**

**Plant material:** The plant *Benincasa hispida* collected from local area of Warangal, Andhra Pradesh, India & authenticated by Prof. V.S.Raju. Senior Professor, Department of Botany and Plant Anatomy Research Center, Kakatiya University, Warangal and voucher specimen (SAM/O8/2012) is submitted at our institution for future reference.

**Preparation of plant extract:** After removing the outer skin and the seeds, the fruit of *Benincasa hispida* was mashed using an electric juicer to afford a soft mass. For the preparation of an ethanolic extract, 100ml of fresh juice was mixed with 500ml of ethanol and kept covered for seven days at room temperature with daily occasional stirring. The mixture was then filtered and the filtrate was heated (below 55°C) and clear serum was separated at 2500 rpm.

**Animals:** Experiments were performed in accordance with the Committee for the Purpose of Control and Supervision of Experimental Animals (CPCSEA). The experimental protocol in the study was approved by the Institutional Animals Ethics Committee (No: 004/IAEC/SUCOP/2012). Wistar albino rats of either sex weighing 180-200g were procured from the Sanzyme Pvt. Ltd. Hyderabad, Andhra Pradesh. Animals were housed at CPCSEA approved (Reg.No.1278/ac/09/CPCSEA) animal house of St. John College of Pharmacy, Warangal. The animals were kept in polypropylene cages (6 in each cage) under standard laboratory condition (12 hr light and 12 hr dark cycle) and had free access to commercial pellet diet with water *ad libitum*. The animal house temperature was maintained at 25±2°C with relative humidity at (50±15%). Ethical norms were strictly followed during all the experiments.

**Study design:**

- **Group-1:** Served as control received 0.9% acasia (5ml/kg/p.o).
- **Group-2:** The animals were pre-treated with 0.9% acasia 1 h before oral treatment with 5ml/kg of olive oil served as model control.
- **Group-3:** The animals were pre-treated with (20mg/kg/p.o) simvastatin, 1 h before oral treatment with (5mg/kg/p.o) of olive oil.
- **Group-4:** The animals were pre-treated with (200mg/kg/p.o) of EEBH in 0.9% acasia, respectively, 1 h before the olive oil treatment.
- **Group-5:** The animals were pre-treated with (400mg/kg/p.o) of EEBH in 0.9% acasia, respectively, 1h before the olive oil treatment.

The above mentioned treatment schedule was followed for the respective group of animals for 28 days. Daily all the animals were given olive oil 1hr before treatment with standard drug and Ethanolic extracts of *Benincasa hispida* [8]. The effects of these drugs on body weight, relative liver weight, food intake, serum lipids, atherogenic index (AI), blood glucose, histopathology of liver were investigated.

**BIOCHEMICAL STUDIES**

**Collection of blood samples:** On day 29 of experiment, the animal blood samples were collected in appendorff tubes from the retro-orbital plexus of rats by inserting a fine capillary gently in the inner angle of the eye [9]. The tubes containing the blood samples were allowed to stand for 30 min at 37°C and clear serum was separated at 2500 rpm for 10 min using micro centrifuge. The biochemical parameters were estimated using respective test kits.

**Estimation of Total Cholesterol by CHOD/PAP method:** Cholesterol esterase hydrolyses esterified cholesterol to free cholesterol. The free cholesterol is oxidised to form hydrogen peroxide which further reacts with phenol and 4-aminoantipyrine by the catalytic action of peroxidase to form a red coloured quinoneimine dye complex. Intensity of
the colour formed is directly proportional to the absorbance was measured at 500nm [10].

\[
\text{Calculation: } \text{Cholesterol concentration in mg\%} = \frac{\text{Abs of test}}{\text{Abs of std}} \times 200
\]

**Estimation of High density lipoprotein-cholesterol:** The estimation of HDL was performed as the method of McGowan et al [11].

**Step I:** Precipitation
- Serum -0.2 ml + HDL ppt reagent 0.3 ml

**Step II:** Colour development and the absorbance was measured at 500nm

\[
\text{HDL concentration in mg\%} = \left( \frac{\text{Abs of test}}{\text{Abs of std}} \right) \times 50
\]

**Estimation of Triglycerides by GPO/PAP method:** Lipoprotein lipase hydrolyses triglycerides to glycerol and free fatty acids. The glycerol formed with ATP in the presence of glycerol kinase forms glycerol 3 phosphate, which is oxidized by the enzyme glycerol phosphate oxidase to form hydrogen peroxide. The hydrogen peroxide further reacts with phenolic compound and 4 aminoantipyrine by the catalytic action of peroxidase to form a red coloured quinoneimine dye complex. Intensity of the colour formed is directly proportional to the amount of triglycerides present in the sample and measured the absorbance at 546nm [12].

\[
\text{Calculation: } \text{Triglyceride concentration in mg\%} = \left( \frac{\text{Abs of test}}{\text{Abs of std}} \right) \times 200
\]

**Estimation of very low density lipoproteins:** Very low density lipoproteins were estimated using the formula Ukwani et al [14]

\[
\text{VLDL} = \frac{\text{Total triglyceride}}{5}
\]

**Atherogenic index** [15] (Rekha Rajendhran et al.):
- It was calculated using formula

\[
\text{Atherogenic index} = \frac{\text{Total serum triglyceride}}{\text{total serum HDL-cholesterol}}
\]

**Percentage protection:** It was calculated using formula

\[
\text{Percentage protection} = \left( \frac{\text{Atherogenic index of control} - \text{Atherogenic index of treated groups}}{\text{Atherogenic index of control}} \right) \times 100
\]
Estimation of blood glucose by GOD/POD method: Glucose is oxidised to gluconic acid and hydrogen peroxide in the presence of glucose oxidase. Hydrogen peroxide further reacts with phenol and 4-aminoantipyrine by the catalytic action of peroxidase to form a red coloured quinoneimine dye complex. Intensity of the colour formed is directly proportional to the amount of glucose present in the sample and is measured as the absorbance at 505 nm.

\[
\text{Glucose} + \text{O}_2 + \text{H}_2\text{O} \xrightarrow{\text{glucose oxidase}} \text{Gluconate} + \text{H}_2\text{O}_2 \\
\text{H}_2\text{O}_2 + 4 \text{Aminoantipyrine} + \text{Phenol} \xrightarrow{\text{peroxidase}} \text{Red Quinoneimine dye} + \text{H}_2\text{O}
\]

Calculation:

\[
\frac{\text{Abs of test}}{\text{Abs of std}} \times 100
\]

Glucose concentration in mg% =

Estimation of total protein: Proteins bind with copper ions in the alkaline medium of biuret reagent and produce a purple coloured complex whose absorbance is proportional to the protein concentration and is measured as the absorbance of standard and test against blank on a colorimeter with yellow green filter or on a spectrophotometer at 546 nm.

Calculation:

\[
\frac{\text{Abs of test}}{\text{Abs of std}} \times \text{Std. Con.}
\]

Protein concentration in mg% =

Histopathology Studies: Rat livers were excised after humane sacrifice of the animals under anesthesia and fixed in 10% formalin. Fixed tissues were completely dehydrated in absolute ethanol and processed routinely for embedding in paraffin wax. From these 5μm sections were prepared and stained with haematoxylin–eosin dye. Stained slides were viewed using an optical photomicroscope at 100× magnification.

Statistical Analysis: Graph Pad Prism software, version 5.0 was used in the statistical analysis of experimental data. The statistical analysis was carried out using analysis of variance (ANOVA) followed by Bonferroni’s test. p values p<0.001, p<0.01, p<0.05 were considered as significant.

RESULTS

Effect on food intake: Group II animals fed with olive oil showed significant (p<0.0001) increase in daily food intake when compared with group I animals. Group III, IV & V animals exhibited a significant (p<0.01, p<0.0001) decrease in daily food intake as compared with group II animals (Table 1).

Effect on body weight: Group II animals fed with olive oil exhibited significant (p<0.0001) increase in body weight between day 1 and 29 as compared to group I animals. Group III, IV & V animals exhibited a significant (p<0.0001) decrease in body weight when compared with group II animals. The EEBH extract at two dose levels resulted in dose dependent decrease of body weight (Table 2).

Lipid profile

Total cholesterol: Group II animals fed with olive oil showed significant (p<0.001) increase in total cholesterol when compared with group I animals. Group III, IV & V animals exhibited a significant (p<0.01, p<0.0001) decrease in total cholesterol when compared with group II animals (Table 3).

HDL levels: Group II animals fed with olive oil showed significant (p<0.0001) reduction in HDL cholesterol when compared with Group I animals. The group III, IV & V exhibited a significant (p<0.001, p<0.05, p<0.01) increase in HDL level when compared to group II animals (Table 3).

Triglycerides: Group II animals showed significant (p<0.0001) increase in triglycerides when compared with group I animals. Group III exhibited (p<0.0001) Group IV exhibited (p<0.01) decrease in triglyceride when compared group II animals. Group V caused significant (p<0.0001) decrease in triglycerides when compared with group II animals (Table 3).

LDL level: Group II animals when compared with group I animals shows significant (p< 0.0001) increase in LDL levels. Group III, Group IV and Group V exhibited significant (p< 0.0001) decrease in LDL level when compared with Group II animals (Table 3).
**VLDL level:** Group II animals when compared with group I animals exhibited significant (p<0.0001) increase in VLDL level. Group III, Group IV and group V when compared with group II animals exhibited significant (p<0.01, p<0.0001) decrease in VLDL levels (Table 3).

**Atherogenic index and percentage protection:** There was a decrease in atherogenic index in treated groups. The percentage protection for group III (63.14) group IV (38.46) group V (46.16) animals (Table 4).

**Blood glucose:** The blood glucose levels in group II animals were significantly (p<0.01) increased when compared with group I animals group III, group IV and group V exhibited significant decrease (p<0.0001, p<0.05, p<0.01) in blood glucose level when compared with group II animals (Table 5).

**Total protein levels:** The total protein levels of group II animals were increased significantly (p<0.01) as compared with group I. Group III, IV &V animals exhibited a significant (p<0.05, p>0.01) decrease when compared with group II animals (Table 5).

**DISCUSSION**

It has been noted that there is a causal relationship between increased plasma lipid levels and the development of atherosclerotic diseases. Development of atherosclerotic disease is a complicated process involving accumulation of lipid containing particles in the walls of coronary arteries & other major arteries within the body. A high fat diet causes cholesterol levels to increase in susceptible people, which leads to obesity [16].

Olive oil induces hyperlipidemia and obesity by increasing the intestinal absorption of dietary lipids the anti hyperlipidemic and anti obesity drug interfering with this process and cause regulation of lipid levels in the body. Thus, the considerable reductions in the weight gain pattern and serum lipids by EEBH treatment are indicative of the weight losing and antihyperlipidemic property of the extract. Certain phytoconstituents, particularly saponins, have been reported to mediate their antihyperlipidemic and hypocholesterolaemic actions by inhibiting or delaying intestinal lipid absorption via a resin-like action and inhibiting pancreatic lipase activity [17], and by enhancing enterohepatic excretion of cholesterol in the bile acid [18 & 19].

Olive oil contains a high concentration of monounsaturated fatty acids. On the other hand, butter fat, beef fat, and palm oil contain a high proportion of saturated fatty acids. Sucrose and fructose have a greater effect in raising blood lipids, particularly triacylglycerols, than do other carbohydrates. It is clear, however, that one of the mechanisms involved is the up-regulation of LDL receptors by poly-and monounsaturated as compared with saturated fatty acids, causing an increase in the catabolic rate of LDL, the main atherogenic lipoprotein. In addition, saturated fatty acids cause the formation of smaller VLDL particles that contain relatively more cholesterol, and they are utilized by extra hepatic tissues at a slower rate than are larger particles—tendencies that may be regarded as atherogenic. EEBH were effective in decreasing daily food intake in olive oil induced rats, indicating that it possess hypolipidaemic property.

It has been known that high levels of cholesterol in the blood can raise a person’s risk of developing heart disease. Cholesterol is transported in the blood stream by carrier proteins known as lipoproteins. Low-density lipoproteins (LDL’s) tend to deposit cholesterol-laden “plaques” in artery walls, narrowing the opening through which blood flows and increasing the risk of heart disease. This is why LDL cholesterol has been dubbed the “bad” cholesterol. High-density lipoprotein (HDL) cholesterol is known as the “good” cholesterol because HDL carries cholesterol to the liver, where it is broken down and removed from the blood before it can wind up on artery walls. High blood levels of triglycerides, the body’s storage form of fat and a primary source of energy, are also associated with a greater risk of heart disease, at least in some people [20]. The size of TRL is determined by the amount of TG that are incorporated into nascent particles from the bulk stored in the intestine (chylomicrons) or the liver (VLDL) or by the rate of hydrolysis by LPL in plasma. In the process of VLDL assembly in the liver, a small amount of TG becomes associated with a single Apo B molecule. In a second stage, the bulk of VLDL-TG is incorporated into the particle from a pool of hepatic TG. The well-known TG reducing effect of n-3 fatty acids has been attributed to a lower TG secretion in VLDL from the liver and not to a lower secretion of Apo B [21]. EEBH has shown more antihyperlipidemic and antihypercholesterolemic activity.

**CONCLUSION**

The present pharmacological investigation revealed that olive oil induced significant increase in body weight (272.47gm), food intake (129.70gm), serum levels of glucose (135.12mg/dl), protein (12.237gm/dl), total cholesterol (152.81mg/dl),...
LDL cholesterol (44.71mg/dl), VLDL cholesterol (51.157) and Triglycerides (255.86mg/dl). Treatment with EEBH resulted in reduction of body weight in Olive oil indicating that the extracts possess weight reducing property. EEBH were effective in decreasing daily food intake in olive oil induced rats, indicating that it possess hypolipidaemic property.

The EEBH at two dose levels showed significant reduction in serum levels of total cholesterol, LDL cholesterol VLDL cholesterol, triglycerides along with significant increase in serum HDL cholesterol levels in Olive oil induced rats. Ethanolic extract (400mg/kg/p.o) has shown more antihyperlipidemic and antihypercholesterolemic activity.

Considering the enhancement of cardio protective lipid HDL, it can be concluded that fruit of *Benincasa hispida* a potent cardio protective agent. Blood glucose levels were also significantly decreased in Ethanolic extract. Total protein levels also decreased significantly in Ethanolic extract of *Benincasa hispida* but effect was more observed at higher dose levels of 400mg/kg/p.o. From the Histopathological studies we found that with the treatment of *Benincasa hispida* significantly decrease fat globules in the liver cells.
**Table 1:** Effect of Ethanolic extract of *Benincasa hispida* on daily food intake

<table>
<thead>
<tr>
<th>Group</th>
<th>Treatment</th>
<th>Feed intake</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Day 1</td>
</tr>
<tr>
<td>1</td>
<td>I Control</td>
<td>123.76±0.23</td>
</tr>
<tr>
<td>2</td>
<td>II Olive oil induced (5ml/kg/p.o)</td>
<td>143.4±0.52***</td>
</tr>
<tr>
<td>3</td>
<td>III Simvastatin (20mg/kg/p.o)</td>
<td>108.85±0.37***</td>
</tr>
<tr>
<td>4</td>
<td>IV EEBH (200mg/kg/p.o)</td>
<td>125.40±0.35**</td>
</tr>
<tr>
<td>5</td>
<td>V EEBH (400mg/kg/p.o)</td>
<td>119.50±0.03***</td>
</tr>
</tbody>
</table>

Values are expressed as Mean ± SEM of animals. Comparisons were done between: Group I vs Group II and Group II vs Group III, IV, V  *p<0.05, **p<0.01, ***p<0.001, ns- Non Significant. Statistical significance test for comparisons were done by ANOVA, followed by Bonferroni’s test.

**Table 2:** Effect of Ethanolic extract of *Benincasa hispida* on weight gain

<table>
<thead>
<tr>
<th>S.NO</th>
<th>Groups</th>
<th>Treatment</th>
<th>Weight on day 1</th>
<th>Weight on day 29</th>
<th>Weight gain</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>I</td>
<td>Control</td>
<td>163.45±0.82</td>
<td>182.41±0.63</td>
<td>18.96±0.24</td>
</tr>
<tr>
<td>2</td>
<td>II</td>
<td>Olive oil induced (5ml/kg/p.o)</td>
<td>172.65±0.43</td>
<td>291.68±0.95</td>
<td>119.03±0.55***</td>
</tr>
<tr>
<td>3</td>
<td>III</td>
<td>Simvastatin (20mg/kg/p.o)</td>
<td>165.15±0.3</td>
<td>174.60±1.26</td>
<td>9.45±1.13***</td>
</tr>
<tr>
<td>4</td>
<td>IV</td>
<td>EEBH(200mg/kg/p.o)</td>
<td>168.76±0.22</td>
<td>186.16±0.70</td>
<td>17.73±0.75***</td>
</tr>
<tr>
<td>5</td>
<td>V</td>
<td>EEBH(400mg/kg/p.o)</td>
<td>167.16±0.24</td>
<td>181.08±3.02</td>
<td>14.08±0.42***</td>
</tr>
</tbody>
</table>

Values are expressed as Mean ± SEM of animals. Comparisons were done between: Group I vs Group II and Group II vs Group III, IV, V  *p<0.05, **p<0.01, ***p<0.001, ns- Non Significant. Statistical significance test for comparisons were done by ANOVA, followed by Bonferroni’s test.
**Table-3:** Effect of Ethanolic extract of *Benincasa hispida* on Lipid Profile

<table>
<thead>
<tr>
<th>Groups</th>
<th>Treatment</th>
<th>Cholesterol</th>
<th>TG</th>
<th>LDL-C</th>
<th>VLDL-C</th>
<th>HDL-C</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Control</td>
<td>80.260±1.249</td>
<td>164.290±2.183</td>
<td>23.082±0.291</td>
<td>32.832±0.422</td>
<td>48.928±0.863</td>
</tr>
<tr>
<td>II</td>
<td>Olive oil induced (5ml/kg/p.o)</td>
<td>152.817±0.66***</td>
<td>255.783±3.203***</td>
<td>44.710±0.520***</td>
<td>51.157±0.641***</td>
<td>32.235±0.607***</td>
</tr>
<tr>
<td>III</td>
<td>Simvastatin (20mg/kg/p.o)</td>
<td>99.595±1.125***</td>
<td>174.523±2.950***</td>
<td>23.040±0.395***</td>
<td>34.905±0.590***</td>
<td>59.325±0.994***</td>
</tr>
<tr>
<td>IV</td>
<td>EEBH(200mg/kg/p.o)</td>
<td>116.567±0.475**</td>
<td>189.800±1.911***</td>
<td>28.425±0.546***</td>
<td>38.97±0.245**</td>
<td>50.867±0.481*</td>
</tr>
<tr>
<td>V</td>
<td>EEBH(400mg/kg/p.o)</td>
<td>110.124±0.481***</td>
<td>182.48±0.643***</td>
<td>26.100±0.138***</td>
<td>36.72±0.248***</td>
<td>54.200±1.26**</td>
</tr>
</tbody>
</table>

Values are expressed as Mean ± SEM of animals. Comparisons were done between: Group I vs Group II and Group I vs Group III, IV, V *p<0.05, **p<0.01, ***p<0.001, ns- Non Significant. Statistical significance test for comparisons were done by ANOVA, followed by Bonferroni’s test.

**Table-4:** Atherogenic index and percentage protection of Ethanolic extract of *Benincasa hispida*

<table>
<thead>
<tr>
<th>S.NO</th>
<th>Group</th>
<th>Treatment</th>
<th>Atherogenic index</th>
<th>% protection</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>I</td>
<td>Control</td>
<td>3.34</td>
<td>-</td>
</tr>
<tr>
<td>2</td>
<td>II</td>
<td>Olive oil induced (5ml/kg/p.o)</td>
<td>7.95</td>
<td>-</td>
</tr>
<tr>
<td>3</td>
<td>III</td>
<td>Simvastatin (20mg/kg/p.o)</td>
<td>2.93</td>
<td>63.14</td>
</tr>
<tr>
<td>4</td>
<td>IV</td>
<td>EEBH(200mg/kg/p.o)</td>
<td>3.89</td>
<td>38.46</td>
</tr>
<tr>
<td>5</td>
<td>V</td>
<td>EEBH(400mg/kg/p.o)</td>
<td>3.41</td>
<td>46.16</td>
</tr>
</tbody>
</table>
### Table-5: Effect of Ethanolic extract of *Benincasa hispida* on blood glucose and total protein levels

<table>
<thead>
<tr>
<th>S.NO</th>
<th>Group</th>
<th>Treatment</th>
<th>Blood glucose (mg/dl)</th>
<th>Total protein (gm/dl)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>I</td>
<td>Control</td>
<td>76.41±0.925</td>
<td>5.425.±0.008</td>
</tr>
<tr>
<td>2</td>
<td>II</td>
<td>Olive oil induced (5ml/kg/p.o)</td>
<td>135.12±1.567**</td>
<td>12.237±0.057**</td>
</tr>
<tr>
<td>3</td>
<td>III</td>
<td>Simvastatin (20mg/kg/p.o)</td>
<td>86.77±1.749***</td>
<td>4.36±0.063**</td>
</tr>
<tr>
<td>4</td>
<td>IV</td>
<td>EEBH(200mg/kg/p.o)</td>
<td>102.367±1.29*</td>
<td>6.44±0.050*</td>
</tr>
<tr>
<td>5</td>
<td>V</td>
<td>EEBH(400mg/kg/p.o)</td>
<td>93.30±0.849**</td>
<td>5.045±0.042**</td>
</tr>
</tbody>
</table>

Values are expressed as Mean ± SEM of animals. Comparisons were done between: Group I vs Group II and Group II vs Group III, IV, V. *p<0.05, **p<0.01, ***p<0.0001, ns- Non Significant. Statistical significance test for comparisons were done by ANOVA, followed by Bonferroni’s test.

### References