Hypolipidemic effect of triphala (*Terminalia chebula, Terminalia belerica and Emblica officinalis*) on female albino rats.

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Abstract

Hyperlipidemia, hyperlipoproteinemia or dyslipidemia is the presence of elevated or abnormal levels of lipids and / or lipoproteins in the blood. Lipid and lipoprotein abnormalities are extremely common in the general population and are regarded as a highly modifiable risk factor for cardiovascular disease due to the influence of cholesterol, one of the most clinically relevant lipid substances in atherosclerosis.

Aim of the work:
This study aimed to evaluate the possible treatment and protective effect of triphala on hyperlipidemic rats.

Material and methods:
Six groups (5rat/group) of female albino rats (*Rattus albinus*) were used. The 1st group used as control, in the 2nd group hyperlipidemia (25% fat & 2% cholesterol) was induced for 3 weeks only then sacrificed , the 3rd group was hyperlipidemic rats for 3 weeks then left for other 3 weeks without any additional treatment as a recovery period, the 4th group served as hyperlipidemic group for 3 weeks then treated with triphala for another 3 weeks (25 mg/100 gm b. wt.), the 5th group was hyperlipidemic (25% fat & 2% cholesterol) for 6 weeks and the 6th group served as hyperlipidemic rats for 6 weeks, and at the same time given triphala (25 mg/100 gm b. wt.) by oral administration.

Results:
The biochemical parameters showed highly significant increase in the body weight, serum glucose, ASAT, ALAT, GGT, LDH, total protein, albumin and total lipids in liver .Many histopathological and histochemical changes were detected in liver tissue of the hyperlipidemic rats. Meanwhile, the treatment with triphala ameliorated the biochemical parameters, histological and histochemical results.

Conclusion:
it is recommended to use triphala in diets for hyperlipidemic patients or those people who have hyperlipidemic family history.

Keywords:
Hyperlipidemia, Triphala, Lipid profile, Albino rats, Physiological parameters, Histopathological and histochemical changes.

Introduction

Hyperlipidemia is a heterogeneous disorder involving multiple etiologies. It is commonly characterized by an increased flux of free fatty acids (FFA), raised triglycerides, low-density lipoprotein (LDL)-cholesterol and apolipoprotein B (apo B) levels, and reduced plasma high-density lipoprotein (HDL)-cholesterol concentration, as a consequence of metabolic effects, or dietary and lifestyle habits (*Kolovou et al., 2005; Feng et al., 2011*).
The use of medicinal plants for health started from thousands of years and still a part of the medical practice in China, Egypt, India, and other developing countries. Modern pharmaceuticals still contain at least 25% of drugs derived from the plants (Thomas, 2000). Herbal medicines are highly in demand in developed as well as developing countries for primary health care because of their wide biological and medicinal activities, higher safety margins, and lower costs (Palav and D’mello, 2006; Chattopadhyay and Bhattacharyya, 2007). The hyperlipidemia-lowering effect of different plants has been well studied and various plants were shown to be helpful in lowering plasma lipid levels and encouraging safety profile. Many plants therefore are considered to be useful means to prevent disorders such as atherosclerosis (Choudhary et al., 2005).

One of the most important plants used as hyperlipidemia-lowering factor in the folk medicine in Egypt is triphala herb. Triphala, meaning "three fruits", is made from fruits of three trees that grow throughout India and the Middle East, including amalaki fruit (Emblica officinalis), bibhitaki fruit (Terminalia belerica), and haritaki fruit (Terminalia chebula).

According to Jagetia et al. (2004) triphala is used to promote appetite and digestion, increase the number of red blood cells and aid in removal of undesirable fat in the body, when dissolved in the mouth, Triphala is used to clear congestion and headaches. Other claimed benefits include helping to maintain normal blood sugar levels, as well as improvement in skin tone and colour. Triphala prevents aging, imparts immunity and improves mental faculties. It also helps to detoxify the liver and purify blood.

Sandhya et al. (2006) reported that triphala, an ancient herbal blend, is one of the most commonly used herbal remedies in the ayurvedic system of healing. Ayurvedic medicine originated in ancient India, has developed over thousands of years, and is one of the oldest systems of healing. Thus triphala is one of the longest-used herbal remedies in the world. Triphala is prescribed as the first line treatment of many ailments as Laxative, detoxifying agent and rejuvenator in Ayurveda. Its antidiabetic, antimutagenic, purgative and radio protective activities has been reported (Jagetia et al., 2002; Kaur et al., 2002; Sabu and Kuttan, 2002; Arora et al., 2003).

Material and methods

1-Experimental animals:
The present work was carried out on thirty mature female albino rats (150±20g). They were obtained from the Nile Company for Pharmaceutical and Chemical Industries. The experimental animals were randomly divided into six groups (5/group) and fed on rodent diet. The rats stayed for 3 weeks to adapt the place then the experimental steps were started.

2-Experimental design:
Six groups were used in this study each containing 5 female albino rats.

1- The 1st group: served as control (C).
2- The 2nd group: hyperlipidemic rats (25% fat & 2%cholesterol)3 weeks only then they were sacrificed (H3).
3- The 3rd group: served as hyperlipidemic rats for 3 weeks then left other 3 weeks without any additional treatment as a recovery period (R).
4- The 4th group: served as hyperlipidemic rats for 3 weeks then treated with triphala for 3 weeks (25 mg/100 gm b. wt.) (H3T).
5- The 5th group: included hyperlipidemic rats (25% fat & 2%cholesterol) for 6 weeks (H6).
6- The 6th group: served as hyperlipidemic rats for 6 weeks, and at the same time they were given triphala (25 mg/100 gm b. wt.) by oral administration(H6T).

Each rat was weighted at the beginning and the end of the experiment and percentage of body weight changes were calculated.
Preparation for measure total lipids in liver:
0.1gm of liver was placed in 1 ml of KOH (30%) and left to be digested in the incubator at 37ºC.

Collection of rat's serum:
At the end of the experiment, animals were decapitated and blood samples were collected from the retro-orbital plexus. The samples were collected in clean dry graduated centrifuge tubes and left for 20 minutes to clot, then centrifuged at 5000 rpm, for 15 minutes. Serum was separated and kept at -20ºC until analysis.

Serum glucose was estimated according to Trinder (1984). Aspartate aminotransferase (ASAT) was performed according to Bergmeyer (1978). Alanine aminotransferase (ALAT) was determined according to Breuer (1996). γ-Glutamyl transferase (γ-GT) was done according to Szasz and Persijn (1974). Serum LDH (Lactate dehydrogenase) concentration was done according to the kinetic ultraviolet method of Young (1990). Serum total protein was performed by the method of Tietz (1994). Serum albumin was done by the method of Doumas et al. (1971). Total lipids in liver was done by the method of Kaplan (1984).

The histological and histochemical preparation:
Fresh specimens of liver were taken from the control and experimental groups. The specimens were fixed in 10% neutral buffered formol and Carnoy’s fluid for the histological and histochemical studies. Sections were then cut at 5µ thickness and stained by haematoxylin and eosin stain according to the method of Drury & Wallington (1980), by periodic acid Schiff technique for demonstrating glycogen (Pearse, 1977), by mercuric bromophenol blue method for detecting total protein (Mazia et al., 1953), and by Mallory’s trichrome stain for demonstrating collagen fibers (Pearse, 1977).

Statistical analysis:
The data are expressed as means ± standard errors (SE). The (T) test was used to elucidate the differences between treated and control groups (Snedecor and Cochran, 1980). A difference was considered significant at p< 0.05 or p< 0.01.

Results
The percentage of body weight gain significantly increased (P < 0.01) in all treated groups. Concerning serum glucose level, the present data showed severe hyperglycemia (P < 0.01) in all treated groups. (Table 1). Results of the present study showed a highly significant increase (P < 0.01) in ASAT, ALAT, GGT and LDH activities in the treated groups when compared with control rats (Table 2). Also, highly significant increase (P < 0.01) was observed in serum total protein and albumin concentrations in all treated groups when compared with the control one during the experimental period (Table 3). Globulin concentration showed insignificant change in all treated groups (Table 3). All treated groups also showed insignificant change in albumin/globulin ratio (A/G ratio) except in the group that was fed hyperlipidemic diet for 6 weeks where it showed highly significant increase (P < 0.01), and also in the group that was treated with triphala for 6 weeks, where it showed a significant increase (P < 0.05) as compared with the control animals (Table 3).
Concerning liver total lipids highly significant increase (P < 0.01) was recorded in all treated groups except in the group that was fed hyperlipidemic diet for 3 weeks then was treated with triphala for another 3 weeks where it showed highly significant decrease (P < 0.01) when compared with the control group (Table 4).
Hyperlipidemia for 6 weeks elevated all the biochemical parameters, while feeding triphala for 3 weeks after stopping fat diets recorded the lowest measures of these parameters.

Figs. (1&2) show normal histological pattern of liver tissue of a control rat. Hyperlipidemic rats of groups H3 showed...
many dystrophic changes in the liver tissue. These changes included: highly distorted and ruptured endothelial lining of the blood vessels, increased lymphocytic infiltration in the portal area, haemolysed RBCs inside the blood vessels, degenerated and vacuolated hepatocytes (fig. 3). A slight amelioration was noticed in liver tissue of rats of the recovery group (fig. 4). Nearly normal hepatocytes were observed in liver tissue of group H3T. Well developed cords of hepatocytes surrounded the central vein and the portal area appeared well developed (fig. 5). Mild recovery was noticed in liver tissue of rats of group H6T (fig. 6).

Normal distribution of collagen fibers was observed in figs. (7&8). Increased collagen fibers were observed in liver tissue of groups H3 or H6, the recovery group and those treated with fats and triphala for 3 or 6 weeks (figs. 9, 10, 11 and 12). Normal distribution of total proteins in the hepatic tissue of a control rat was observed in figs. (13&14). Highly reduced total proteins was observed in liver tissue of group H3 (fig. 15), but the R group showed a mild decrease (fig. 16). Meanwhile, nearly normal total proteins were observed in hepatocytes of liver tissue of groups H3T or H6T (figs. 17 & 18).

Concerning all the previous biochemical parameters, histological and histopathological changes it was found that using triphala was better than relying only excluding dietary fats after hyperlipidemic diets without any additional treatment (recovery groups).

Table (1): Percentage of body weight change and Serum glucose level (mg/dl) in female albino rats after induction of hyperlipidemia and treating with triphala.

<table>
<thead>
<tr>
<th>Group Parameter</th>
<th>Cont-rol</th>
<th>3 weeks</th>
<th>6 weeks</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>Hyper L 3W</td>
<td>Hyper L 3W &amp; Recov 3W</td>
</tr>
<tr>
<td>Body weight change (%)</td>
<td>5.50 ± 0.65</td>
<td>12.92 ± 1.35</td>
<td>10.84 ± 0.98</td>
</tr>
<tr>
<td>Glucose (mg/dl)</td>
<td>Mean</td>
<td>Hyper L 3W</td>
<td>Hyper L 3W &amp; Recov 3W</td>
</tr>
<tr>
<td></td>
<td>64.8 ± 1.7</td>
<td>99.0 ± 2.4</td>
<td>76.2 ± 1.7</td>
</tr>
</tbody>
</table>

Hyper L = Hyperlipidemia  
Recov = recovery  
6W = 6 Weeks  
3W = 3 weeks  
N.s = non significant


Hypolipidemic effect…..

Table (2): Aspartate aminotransferase (ASAT), Alanine aminotransferase (ALAT) gamma glutamyl transferase (GGT) and Lactate dehydrogenase (LDH) activities in female albino rats after induction of hyperlipidemia and treating with triphala.

<table>
<thead>
<tr>
<th>Group Parameter</th>
<th>Control</th>
<th>3 weeks</th>
<th>6 weeks</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>Hyper L 3 W</td>
<td>Hyper L 6 W</td>
</tr>
<tr>
<td>ASAT (U/L)</td>
<td>32.5</td>
<td>89.8</td>
<td>87.6</td>
</tr>
<tr>
<td>± SE</td>
<td>0.8</td>
<td>0.96</td>
<td>2.99</td>
</tr>
<tr>
<td>P</td>
<td>&lt;0.01</td>
<td>&lt;0.01</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>% of change</td>
<td>-</td>
<td>176.3</td>
<td>169.5</td>
</tr>
<tr>
<td>ALAT (U/L)</td>
<td>23.2</td>
<td>73.4</td>
<td>53.0</td>
</tr>
<tr>
<td>± SE</td>
<td>1.4</td>
<td>1.8</td>
<td>1.1</td>
</tr>
<tr>
<td>P</td>
<td>&lt;0.01</td>
<td>&lt;0.01</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>% of change</td>
<td>-</td>
<td>216.3</td>
<td>128.4</td>
</tr>
<tr>
<td>GGT (U/L)</td>
<td>26.2</td>
<td>40.8</td>
<td>39.6</td>
</tr>
<tr>
<td>± SE</td>
<td>1.85</td>
<td>0.6</td>
<td>0.57</td>
</tr>
<tr>
<td>P</td>
<td>&lt;0.01</td>
<td>&lt;0.01</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>% of change</td>
<td>-</td>
<td>55.7</td>
<td>51.1</td>
</tr>
<tr>
<td>LDH (U/L)</td>
<td>177.8</td>
<td>363.0</td>
<td>244.0</td>
</tr>
<tr>
<td>± SE</td>
<td>2.8</td>
<td>1.9</td>
<td>2.26</td>
</tr>
<tr>
<td>P</td>
<td>&lt;0.01</td>
<td>&lt;0.01</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>% of change</td>
<td>-</td>
<td>104.1</td>
<td>37.2</td>
</tr>
</tbody>
</table>

Hyper L = Hyperlipidemia  Recov = recovery  3W = 3 weeks
6W = 6 Weeks             N.s = non significant

231
Table (3): Total protein, Albumin, Globulin concentrations and Albumin/Globulin ratio (A/G) ratio in female albino rats after induction of hyperlipidemia and treating with triphala.

<table>
<thead>
<tr>
<th>Group</th>
<th>Parameter</th>
<th>Control</th>
<th>3 weeks</th>
<th>Hyper L 3 W</th>
<th>Hyper L 3 W &amp; Recov 3 W</th>
<th>Hyper L 3 W then triphala 3 W</th>
<th>Hyper L 6 W</th>
<th>Hyper L &amp; triphala 6 W</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Total protein (g/dl)</td>
<td>Mean</td>
<td>6.7</td>
<td>8.46</td>
<td>8.0</td>
<td>7.8</td>
<td>9.12</td>
<td>8.3</td>
</tr>
<tr>
<td></td>
<td>± SE</td>
<td>0.127</td>
<td>0.17</td>
<td>0.145</td>
<td>0.114</td>
<td>0.065</td>
<td>0.057</td>
<td></td>
</tr>
<tr>
<td></td>
<td>P</td>
<td>&lt;0.01</td>
<td>&lt;0.01</td>
<td>&lt;0.01</td>
<td>&lt;0.01</td>
<td>&lt;0.01</td>
<td>&lt;0.01</td>
<td></td>
</tr>
<tr>
<td></td>
<td>% of change</td>
<td>26.2</td>
<td>19.4</td>
<td>16.4</td>
<td>36.1</td>
<td>23.8</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Albumin (g/dl)</td>
<td>Mean</td>
<td>4.32</td>
<td>5.7</td>
<td>5.4</td>
<td>5.1</td>
<td>6.5</td>
<td>5.8</td>
</tr>
<tr>
<td></td>
<td>± SE</td>
<td>0.143</td>
<td>0.065</td>
<td>0.103</td>
<td>0.108</td>
<td>0.096</td>
<td>0.074</td>
<td></td>
</tr>
<tr>
<td></td>
<td>P</td>
<td>&lt;0.01</td>
<td>&lt;0.01</td>
<td>&lt;0.01</td>
<td>&lt;0.01</td>
<td>&lt;0.01</td>
<td>&lt;0.01</td>
<td></td>
</tr>
<tr>
<td></td>
<td>% of change</td>
<td>31.9</td>
<td>25.0</td>
<td>18.0</td>
<td>50.4</td>
<td>34.2</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Globulin (g/dl)</td>
<td>Mean</td>
<td>2.38</td>
<td>2.74</td>
<td>2.56</td>
<td>2.7</td>
<td>2.6</td>
<td>2.52</td>
</tr>
<tr>
<td></td>
<td>± SE</td>
<td>0.163</td>
<td>0.160</td>
<td>0.216</td>
<td>0.086</td>
<td>0.061</td>
<td>0.119</td>
<td></td>
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<tr>
<td></td>
<td>P</td>
<td>N.S</td>
<td>N.S</td>
<td>N.S</td>
<td>N.S</td>
<td>N.S</td>
<td>N.S</td>
<td></td>
</tr>
<tr>
<td></td>
<td>% of change</td>
<td>15.1</td>
<td>7.5</td>
<td>13.4</td>
<td>9.2</td>
<td>5.88</td>
<td></td>
<td></td>
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<tr>
<td></td>
<td>A/G ratio</td>
<td>Mean</td>
<td>1.85</td>
<td>2.106</td>
<td>2.182</td>
<td>1.896</td>
<td>2.512</td>
<td>2.324</td>
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<tr>
<td></td>
<td>± SE</td>
<td>0.177</td>
<td>0.128</td>
<td>0.249</td>
<td>0.089</td>
<td>0.094</td>
<td>0.128</td>
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<tr>
<td></td>
<td>P</td>
<td>N.S</td>
<td>N.S</td>
<td>N.S</td>
<td>&lt;0.01</td>
<td>&lt;0.01</td>
<td>&lt;0.05</td>
<td></td>
</tr>
<tr>
<td></td>
<td>% of change</td>
<td>13.8</td>
<td>17.9</td>
<td>2.4</td>
<td>35.7</td>
<td>25.6</td>
<td></td>
<td></td>
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</table>

Hyper L = Hyperlipidemia  
Recover = recovery  
3W = 3 weeks  
6W = 6 Weeks  
N.s = non significant
Table (4): The level of liver total lipids in female albino rats after induction of hyperlipidemia and treating with triphala.

<table>
<thead>
<tr>
<th>Group</th>
<th>Parameter</th>
<th>Control</th>
<th>Hyper L 3 W</th>
<th>Hyper L 3 W &amp; Recov 3 W</th>
<th>Hyper L 3 W &amp; triphala 3 W</th>
<th>Hyper L 6 W</th>
<th>Hyper L &amp; triphala 6 W</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>4.52</td>
<td>5.64</td>
<td>5.34</td>
<td>3.84</td>
<td>7.46</td>
<td>6.58</td>
</tr>
<tr>
<td></td>
<td>± SE</td>
<td>0.21</td>
<td>0.13</td>
<td>0.125</td>
<td>0.044</td>
<td>0.16</td>
<td>0.065</td>
</tr>
<tr>
<td></td>
<td>P</td>
<td>&lt;0.01</td>
<td>&lt;0.01</td>
<td>&lt;0.01</td>
<td>&lt;0.01</td>
<td>&lt;0.01</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td></td>
<td>% of change</td>
<td>24.7</td>
<td>18.1</td>
<td>15.04</td>
<td>65.04</td>
<td>45.5</td>
<td></td>
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</tbody>
</table>

Hyper L = Hyperlipidemia    Recov = recovery    3W = 3 weeks
6W = 6 Weeks                N.s = non significant
Fig. (1&2): Showing photomicrographs of liver tissue of a control rat. 1- Showing the central vein (cv), sinusoidal spaces (s), kupffer cells (k) and hepatocytes (H).
2-The portal area contains a branch of the hepatic portal vein (hpv), bile duct (bd) and a branch of the hepatic artery (ha). (HX & E X100). Fig.(3): Showing photomicrographs of liver tissue of rats treated with fats (hyperlipidemia) for 3 weeks only. Numerous fatty cells, lymphocytic infiltration around the distorted central vein(7), lots of vacuolated hepatocytes(8) and many pyknotic nuclei (p). (HX & E X100).

Fig. (4): Showing photomicrographs of liver tissue of rats treated with fats for 3 weeks and left 3 weeks for recovery. Lymphocytic infiltration around the central vein (7), increased kupffer cells (k), fatty degeneration (F) with many vacuolated hepatocytes and haemolysed RBCs inside the central vein (HX & E X100). Fig.(5): Showing remarkable recovery in the liver tissue of a rat treated with fats for 3 weeks then 3 weeks with triphala. (HX & E X100). Fig.(6): Showing noticeable recovery in the liver tissue of a rat treated with fats and triphala for 6 weeks simultaneously. (HX & E X100).

Figs.(7&8): Showing normal distribution of collagen fibers in the liver tissue of a control rat. Notice thin collagen bundles supporting the central vein (cv), hepatocytes (H), sinusoidal spaces (s), hpv and walls of bile duct. (Mallory's trichrome stain X 100). Fig.(9): Showing increased collagen fibers in the liver tissue of a rat treated with fats (hyperlipidemia) for 3 weeks only. Collagen fibers increased around the hepatocytes, branches of the hepatic portal vein, hepatic artery, while collagen fibers decreased in the wall of the central vein (Mallory's trichrome stain X 100).
Fig.(10): Showing increased collagen fibers around most hepatocytes and sinusoidal spaces in the liver tissue of a rat treated with fats for 3 weeks and left 3 weeks for recovery (Mallory's trichrome stain X 100). Fig.(11): Highly increased collagen fibers in the central vein, hepatocytes and sinusoidal spaces in the liver tissue of a rat treated with fats for 3 weeks then 3 weeks with triphala (Mallory's trichrome stain X100). Fig.(12): Showing increased collagen bundles in the central area of liver tissue of a rat treated with fats and triphala for 6 weeks simultaneously (Mallory's trichrome stain X100). Figs.(13&14): Showing normal distribution of total proteins in the central and portal areas in the liver tissue of a control rat (Mercuric bromophenol blue X 100). Fig.(15): Showing highly reduced total proteins in the walls of the hepatic portal vein (hpv), and hepatocytes in the liver tissue of a rat treated with fats (hyperlipidemia) for 3 weeks only (Mercuric bromophenol blue X 100). Fig.(16): Showing a mild decrease in total proteins in the walls of the central vein of liver tissue of a rat treated with fats for 3 weeks and left 3 weeks for recovery (Mercuric bromophenol blue X 100). Fig.(17): Showing nearly normal distribution of total proteins in the central area of liver tissue of a rat treated with fats for 3 weeks then 3 weeks with triphala (Mercuric bromophenol blue X 100). Fig.(18): Showing total proteins distribution in the liver tissue of a rat treated with fats and triphala for 6 weeks simultaneously. Some hepatocytes were faintly stained, while, others were nearly normal (Mercuric bromophenol blue X100).
Discussion

Hyperlipidemia: is an elevation of lipids in the blood stream and these lipids include: fats, fatty acids, cholesterol, cholesterol esters, phospholipids, and triglycerides. Coronary heart disease (CHD) is caused by the narrowing of the artery that supplies nutrients and oxygen to the heart. The main reason for this narrowing is atherosclerosis. There is a relationship between the elevated plasma lipids and the development of atherosclerotic plaques (Jain et al., 2007).

Body weight:
Consumption of high fat diet led to obesity and overweight probably because it facilitated the development of a positive energy balance leading to an increase in visceral fat deposition, this lead to abdominal obesity in particular (Amin and Nagy, 2009). In the present investigation, triphala reduced the body weight gain. This may be due to the active components, hydroxyl-anthracene glucosides compounds, in triphala which improve gastrointestinal motility and influence colonic motility thereby reducing fluid absorption and facilitating weight loss (Amin and Nagy, 2009).

Glucose level:
Obesity is associated with decreased ability of the body to control blood glucose with normal levels of insulin (Kuczmarstta et al., 1994; Bloomgarden, 2004). Reduction in glucose level by triphala is probably mediated through enhanced secretion of insulin from the beta-cells of the pancreatic islets or through an extra pancreatic mechanism. Moreover, triphala may reduce inflammatory cytokine release during diabetes, which may be one of the causative agents for the insulin resistance (Rao and Nammi, 2006).

Liver and heart functions:
Injury to liver tissues due to hyperlipidemia alters their transport function and membrane permeability, leading to leakage of enzymes from the cells. Therefore, the marked release of ASAT, ALAT and GGT into the circulation indicates severe damage to hepatic tissue membranes (Ahn et al., 2007). Gallic acid (GA) present in triphala is reported to possess hepatoprotective and antioxidant activities. The quantification of GA can be used as an index in routine quality control of triphala and its different constituents (Kumagai et al., 2003).

In the present study triphala reduced the level of liver enzymes as compared with the hyperlipidemic groups. These results are in agreement with those obtained by Jadon et al. (2007), who showed that gallic acid, at 50 mg/kg body weight, could decrease plasma ASAT, ALAT and GGT activities elevated by acute hepatic damage.

Suchalatha and Shyamala (2004) reported that triphala extract treatment ameliorates the effect of lipid peroxide formation that is related to the activities of diagnostic myocardial marker enzymes. Improvement of cardiac muscle function and subsequent improved pumping activity of the heart seems to be the primary benefit of triphala (Singh et al., 1982).

Protein profile:
Increase in serum total protein and albumin in hyperlipidemic rats was observed and was explained by increased amino acids synthesis and greatly increased concentration of a variety of essential amino acids (Brosnan et al., 1984). Increase in protein synthesis which in turn may be due to increase in the amount and availability of mRNA, increase in translation factor and increase in ribosomal protein synthesis as a result of hyperlipidemia (Peavy et al., 1985).

Treating with triphala decreased total proteins and albumin, due to reducing amino acids synthesis and this led to reduction in ribosomal protein synthesis at the end of the cascade of events and returned to normal values as a result of antioxidant, antimutagenic and free radical scavenging activity of this plant (Wool et al., 1986).
Liver total lipids:
Hyperlipidemia is a genetic disorder of lipid metabolism associated with insulin resistance and abnormalities in fatty acid metabolism. It is characterized by an increase cardiovascular risk (Cavallo et al., 2005; Martijn et al., 2008). In the current investigation, triphala significantly turned back liver total lipids to the normal values. This may be due to inhibition of hepatic cholesterol biosynthesis that led to increased fecal bile acid excretion, and stimulation of receptor-mediated catabolism of LDL-cholesterol caused triphala’s lipid-lowering effects (Khanna et al., 1996). Our observations were in agreement with Kirby et al. (2004) who stated that significant decrease in the liver total lipids may be due to reduction in the absorption of cholesterol, and they found that oral administration of triphala reported to increase gastric emptying and this might be the reason for decrease in the cholesterol absorption. Triphala has antioxidant and hypolipidaemic activity and also has free radical scavenger activity. It also protect myocardial necrosis and reduces cholesterol-induced atherosclerosis (Ram et al., 2003).

The histopathological study:
Hyperlipidemia is known to enhance the risk of fatty liver disease (Festi et al., 2004) and carcinogenesis which is associated with hydroxyl radical formation (Tseng et al., 1996). Antolin et al. (2009) noticed a relation between obesity and different degrees of fibrosis and chronic liver diseases and they added that liver transplant patients show increased rates of obesity. Fatty liver is associated with overweight, hyperlipidemia, hyperglycemia, hyperuricemia and alcoholism in Taiwanese (Changchien et al., 2003). Rats treated with fats for 3 or 6 weeks showed many pathological changes in the liver tissue. These changes were more pronounced in liver tissue of group H6. These changes include: ruptured endothelial lining of the blood vessels, enlarged nuclei of the endothelial lining, increased lymphocytic infiltration in the portal area, haemolysed RBCs inside the blood vessels with degenerated and vacuolated hepatocytes and fatty cells. The hepatic portal areas lost their normal architecture and contained highly distorted blood vessels and bile canaliculi. In the present study rats of group R showed no detectable signs of improvement in the hepatic tissue. Well developed hepatocytes were detected in the liver tissue of rats of group H3T, but increased lymphocytic infiltration was detected in the portal area with moderate RBCs haemolysis inside the hpv. Detectable recovery was observed in the liver tissue of rats of group H6T, but some hepatocytes were still vacuolated with lymphocytic infiltration in the portal areas. Improvement in blood vessels architecture noted in liver of rats treated with fats and triphala in the present study was also observed by Jain et al. (2009) who noticed the cardio protective effect of this plant.

Results of the present study showed increased stain affinity of collagen fibers in liver of rats of groups H3, H6 and R, especially around the hepatocytes, walls of hepatic portal veins and the arterial walls with decreased stain affinity in the wall of the central vein. Enzan et al. (1995) attributed a similar finding to the activation of myofibroblast-like cells present normally within the hepatic and renal parenchyma. George et al. (2001) suggested that decreased synthesis of collagenolytic enzymes by the impaired hepatocytes might contribute to further accumulation of collagen. Liver of rats of group H3T & H6T showed increased collagen fibers in walls of blood vessels, hepatocytes and sinusoidal spaces. Hassan et al. (1988) reported that increased collagen fibers may lead to increased defensive reaction against toxic materials.

The histochemical study:
Highly reduced total proteins was detected in liver tissue of groups H3 & H6, but nearly normal protein content was realized in hepatocytes of liver tissue of group R, but a mild decrease was noted in walls of the blood vessels and bile canaliculi. Some what normal protein content was observed in liver tissue of groups H3T and H6T. Some vacuolated hepatocytes and fatty
cells were negatively stained with deeply stained RBCs inside the congested blood vessels. According to El Banhawy et al. (1986) decreased protein content in the liver tissue may be due to increased action of lytic enzymes. In 2007, Eid and Al Dossary stated that decreased protein content in liver tissue may be due to the drastic effect on rough endoplasmic reticulum (RER), mitochondria and Golgi apparatus and increased lysosomes in hepatocytes.

**Conclusion & recommendations:**
Results of the present study showed that triphala has hypolipidemic action specially when used with fat free diet for treating hyperlipidemia. So, we recommended to use it for treatment of hyperlipidemic patients.

**References:**


Hassan H, Ghaly E, El-Nashar A and Manggoud H (1988): Histochemical study on some organs of rats fed rape seed and
Hypolipidemic effect.....


تخفيف نسبة الدهون باستخدام نبات الهليلج في إناث الجرذان

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استهدفت هذه الدراسة إيضاح الدور الوقائي لنبات الهليلج ضد الأخطار الناتجة عن زيادة الدهون على بعض المعايير البيولوجية والتشخيصات الفيزيولوجية والعصبية، وتمت هذه الدراسات على المجموعات التالية:

1- المجموعة الأولى: استخدمت كمجموعة ضابط.
2- المجموعة الثانية: مجموعة الجرذان لمصابات الدهون (25% دهون و 2% كولستيرول) لمدة ثلاثة أسابيع فقط ثم تم ذبحها.
3- المجموعة الثالثة: مجموعة الجرذان المصابة بزيادة الدهون لمدة ثلاثة أسابيع ثم تركت ثلاثة أسابيع بدون أي علاج إضافي كفترة استشفاء.
4- المجموعة الرابعة: الجرذان المصابة بزيادة الدهون ثلاثة أسابيع ثم عولجت بالمستخلص المائي لنبات الهليلج (25 ملي جم / 100 جرام من وزن الجسم) لمدة ثلاثة أسابيع أخرى.
5- المجموعة الخامسة: المجموعة المصابة بالدهون لمدة ستة أسابيع (25% دهون و 2% كولستيرول).
6- المجموعة السادسة: مجموعة الجرذان المصابة بزيادة الدهون وفي نفس الوقت تم معالجتها بالمستخلص المائي لنبات الهليلج لمدة ستة أسابيع (25 ملي جم / 100 جرام من وزن الجسم).

ولد أوضحت نتائج هذا البحث أن معاملة الجرذان بالدهون لمدة ثلاثة أو ستة أسابيع أو حتى بعد التوقف عن تعاطي الدهون لمدة ثلاثة أسابيع له آثار سلبية عديدة تتمثل في زيادة معدل كلا من وزن الجسم، نسبة السكر في الدم، وظائف الكبد، والكلي، البروتينات الكلية، الألبومينوم، مجموعة الدهون الكلبية بالكبد، وصاحب كل ذلك وجود عدد كبير من التغيرات السسيجية والكيميائيه في كبد الجرذان البيضاء أما المعالجة بالمستخلص المائي لنبات الهليلج فقد أدت إلى ظهور تحسين ملحوظ في المعايير البيولوجية والعصبية والتشخيصات الفيزيولوجية والعصبية، ولذا يمكن استخدام نبات الهليلج في طعام مرضى زيادة الدهون أو لمهم تاريخ عائلي لمرض زيادة الدهون.