Hypolipidemic effect of coffee silver skin in rats fed a high-fat diet

Ayman Mohammed El-Anany, Rehab Farouk M. Ali

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A B S T R A C T

The present study was conducted to evaluate the hypolipidemic effects of coffee silver skin (CSS) supplementation in rats fed a high-fat diet (HFD). A total of 40 albino rats were used in the present study. The groups were as follows: Rats fed a normal diet, N group; high fat diet, HFD group; high fat diet +10% CSS, HFD 10; HFD + 15% CSS, HFD 15; HFD + 20% CSS, HFD 20; the diets were followed for 8 weeks. Blood samples were collected at the end of the experiment. At the time of sacrifice, the weights of heart, liver, kidneys, epididymal fat and retroperitoneal fat of the experimental rats with respect to body weight were recorded. The lipid parameter of the serum was recorded and liver and kidney function tests were conducted. Finally, a histopathological assay was performed on the liver and kidney tissues of the rats fed the tested diets. The weight gain of the rats fed a HFD supplemented with 10, 15 and 20% CSS was ∼1.05, 1.08 and 1.12 times lower than that of those rats fed HFD, respectively. The incorporation of CSS at a level of 20% reduced the increase in liver, kidney, epididymal fat and retroperitoneal fat weight by 17.84, 19.38, 47.23 and 18.00%, respectively, compared with HFD alone. HFD administration induced considerable increases in alanine aminotransferase, aspartate aminotransferase and alkaline phosphatase activities compared with the control group. The results also indicated that the HFD-fed rats exhibited increased levels of urea, uric acid and creatinine, by ∼26.38, 8.40 and 6.75%, respectively, compared with the control rats. With the exception of high-density lipoprotein cholesterol, all lipid fractions increased significantly in rats fed a HFD. The administration of a HFD induced marked pathological changes in the liver and kidneys of the rats. However, the incorporation of various levels of CSS in to a HFD reduced these changes. The results of the present study illustrate that the incorporation of CSS into HFDs reduces the hyperlipidemia effect of these diets.

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1. Introduction

Lipids serve a critical role as biomolecules. Among these molecules, cholesterol is an important component of the human cell membrane and a component of steroids and bile acids. Triglycerides are also important molecules and serve critical role in transferring the energy of food into body cells. However, the elevation of different forms of lipids in the bloodstream causes coronary heart disease [1].

Hyperlipidemia is a condition that is characterized by high levels of serum cholesterol, low-density lipoprotein cholesterol (LDL-c), very low-density lipoprotein cholesterol (VLDLc) and triglycerides [2]. These parameters are the diagnostic markers used to evaluate the risk of heart disease [3]. Cardiovascular disease is the most common cause of mortality in the USA. Every year, 500,000 Americans succumb to cardiovascular disease, and the cost of morbidity >200 billion USD annually [4]. Although the positive association between cardiovascular disease and a raised serum total cholesterol level is well known, the medical community has not yet endorsed this association as an area of prevention. Results from a 1996 study at a teaching hospital reported that only 30% of patients with cardiovascular disease and hyperlipidemia were being administered lipidlowering drugs by their cardiologists [5]. Approximately 52 million adults require significant dietary changes, and 12.7 mil-
Chemical composition and calorie content of coffee silver skin.

<table>
<thead>
<tr>
<th>Component</th>
<th>Coffee silver skin</th>
</tr>
</thead>
<tbody>
<tr>
<td>%</td>
<td>Energy (kcal/100 g)</td>
</tr>
<tr>
<td>Moisture</td>
<td>4.02</td>
</tr>
<tr>
<td>Protein</td>
<td>12.94</td>
</tr>
<tr>
<td>Crude fiber</td>
<td>64.13</td>
</tr>
<tr>
<td>Ash</td>
<td>6.96</td>
</tr>
<tr>
<td>Fat</td>
<td>1.98</td>
</tr>
<tr>
<td>Solublecarbohydrate</td>
<td>13.99</td>
</tr>
<tr>
<td>Total energy</td>
<td>125.54</td>
</tr>
</tbody>
</table>

2.1. Materials and methods

2.1.1. Animals and diet

A total of 40 male albino rats (Rattus norvegicus) of the Wistar strain, aged 810 weeks, with an average weight of 200,220 g were obtained from the Animal House at the Food Technology Research Institute, (Agricultural Research Center, Giza, Egypt). The rats were housed in polypropylene cages lined with husk in standard environmental conditions: Temperature 25 ± 2°C, relative humidity 55 ± 10% and a 12 h light/dark cycle. The animals were provided with feed and drinking water ad libitum. The animal experiments were conducted in accordance with the recommendations in the Guide for the Care and Use of Laboratory Animals published by the US National Institutes of Health (National Institutes of Health Publication no. 85-23, revised 1996) and was approved by the Institutional Animal Care and Research Advisory Committee of the Catholic University of Leuven (approval no. P154/2013). The experimental design and animal handling procedures were approved by the Ethics Committee of the Food Technology Research Institute of the Agricultural Research Center. Every effort was made to minimize the number of animals used (40 rats) and their suffering. Following one week to acclimatize to the American Institute of Nutrition (AIN)93 G diet [18], the rats were randomly divided into 5 groups (n = 8) for a period of 8 weeks (normal diet, N group; high fat diet, HFD group; high fat diet +10% CSS, HFD 10; high fat diet +15% CSS, HFD 15; high fat diet +20% CSS, HFD 20 group). Daily food intake and weekly body weight were recorded during the 8-week experimental period. The composition of the experimental diet was based on the AIN93 G diet, as presented in Table 2.

2.2. Sampling and chemical analysis

At the end of the 8-week experimental period, the rats were fasted for 12 h and anesthetized with inhaled isoflurane prior to sacrifice. Blood samples from each group of rats were collected, centrifuged at 1200 × g for 15 min at 10°C to obtain the serum, which was stored at 25°C until further analysis.

2.2.1. Biochemical analyses

The serum total lipid level was measured by the method of Frings and Dunn [19]. The concentrations of total cholesterol (T-Ch), high-density lipoprotein (HDL-Ch), low-density lipoprotein (LDL-Ch) and triglycerides (TG) were determined according to the procedure described by ElAnany and Ali [20]. The atherogenic index (AI) was calculated as previously described, using the following equation: Log [triglycerides (mg/dL)/HDL (mg/dL)] [21].

2.2.2. Relative organ weight of the experimental rats

At the time of sacrifice, the hearts, livers, and kidneys of the experimental rats were identified, removed, rinsed with physiological saline solution and dried carefully with tissue paper. The weights (g) of the organs, epididymal fat (E-fat) and retroperitoneal fat (R-fat) with respect to the body weight of the rats, were immediately recorded.

2.2.3. Liver and kidney function assay

The activities of alanine aminotransferase (ALT), aspartate aminotransferase (AST) as well as alkaline phosphatase (ALP) were measured according to the methods described by Bergmeyer and Harder [22], and Belfield and Goldberg [23], respectively. The urea and uric acid concentrations of the serum from the rats were deter-
mined as described by Fawcett and Scott [24], and Barham and Trinder [25], respectively.

2.3. Histopathological study

At the end of the nutritional experiments, following the sacrifice of the animals, the liver and kidneys were removed, stored in 10% neutral formalin and embedded in paraffin wax. The organs were sectioned (56 μm) and stained with hematoxylin and eosin, according to Culling [26]. The tissue sections were examined using an optical microscope, ×400 magnification, for histological evaluation.

2.4. Statistical analysis

The results are expressed as the mean ± standard deviation. Data were statistically analyzed in a randomized design in factorial arrangement, according to the procedure described by Gomez and Gomez [27]. The means of the treatments were compared by Fisher’s least significant difference test using Excel (Microsoft Office 2007; Microsoft Corporation, Redmond, WA, USA) and SPSS version 18.0 (SPSS, Inc., Chicago, IL, USA). Data are presented in the text and tables as the means of 5 determinations. P ≤ 0.05 was considered to indicate a statistically significant difference.

3. Results

3.1. Body weight and feed intake of the experimental rats

Table 3 presented the initial and final body weights, bodyweight gain and feed intake of the experimental rats. Significant differences (P ≤ 0.05) in weight gain and feed intake between the control and the HFD groups were observed (Table 3). The weight gain and food intake of the rats fed a HFD were significantly higher than those fed the control diet, with the highest weight gain (82.4 g; P ≤ 0.05) at the end of the experiment observed in the rats fed a HFD. The incorporation of various levels of CSS into the HFD caused significant (P ≤ 0.05) reductions in the weight gain of rats. These reductions significantly (P ≤ 0.05) increased as the incorporation levels increased. The weight gain of rats fed a diet supplemented with 10, 15 and 20% CSS was ~1.05, 1.08 and 1.12 times, respectively, lower than that of rats fed the HFD. The lowest gain in body weight was observed in the control group (P ≤ 0.05), followed by the HFD 20 and 15 groups. These decreases in weight gain might be due to the presence of the high content of fiber in CSS (Table 1).

The data presented in Table 3 indicate that no significant (P ≥ 0.05) differences in feed intake between the control group and the groups supplemented with CSS were observed. These findings have indicated that daily consumption of CSS is effective in preventing weight gain.

3.2. Relative weights of organs, E-fat and R-fat from rats administered experimental diets

Table 4 illustrated the changes in the relative weights of organs, E-fat, and R-fat from rats administered experimental diets. The liver, kidney, E-fat and R-fat weight significantly differed (P ≤ 0.05) between the groups. The rats of the control group exhibited the lowest (P ≤ 0.05) liver, kidney, E-fat and R-fat weights. The consumption of HFD for 8 weeks induced a significant increase (P ≤ 0.05) in the ratio of the liver, kidney, E-fat, and R-fat weights to body weight.

The administration of different levels of CSS significantly (P ≤ 0.05) reduced the liver, kidney, E-fat and R-fat weight compared with the HFD group (Table 4). In HFD10, the liver, kidney, epididymal fat and R-fat weight tended to be lower than HFD group, but not significantly so. HFD15 and HFD20 diets significantly (P ≤ 0.05) suppressed the increase in liver, kidney, epididymal fat and R-fat weight that was induced by HFD alone. The incorporation of CSS at the level of 20% lowered the increase in liver, kidney, E-fat and R-fat weight by 17.84, 19.38, 47.23 and 18.00%, respectively, compared with HFD alone.

As presented in Table 4, the data indicated that there were no significant (P ≥ 0.05) differences in heart weight among the five groups in the current study.

3.3. Effect of the experimental diets on some liver and kidney functions

The effect of experimental diets on the activities of ALT, AST, and ALP are shown in Table 5. ALT, AST, and ALP activities were higher in rats fed HFD than in the control, HFD10, HFD15 and HFD20 groups. HFD administration induced considerable increases in ALT, AST, and ALP activities, by 62.75, 80.91, and 137.80 IU/L, respectively, compared with the control group (26.17, 46.53 and 75.36 IU/L, respectively). Lower ALT, AST and ALP activities were observed

Table 3

<table>
<thead>
<tr>
<th>Group</th>
<th>Initial body weight (g)</th>
<th>Final body weight (g)</th>
<th>Weight gain (g)</th>
<th>Average food intake (g/day)</th>
</tr>
</thead>
<tbody>
<tr>
<td>N</td>
<td>208.5±15.1</td>
<td>279.8±18.0</td>
<td>71.3±3.32</td>
<td>14.7±0.75</td>
</tr>
<tr>
<td>HFD</td>
<td>204.7±11.3</td>
<td>287.1±16.8</td>
<td>82.4±2.2</td>
<td>17.6±0.95</td>
</tr>
<tr>
<td>HFD10</td>
<td>200.7±13.6</td>
<td>278.8±13.7</td>
<td>78.1±3.3</td>
<td>15.5±1.45</td>
</tr>
<tr>
<td>HFD15</td>
<td>220.5±18.2</td>
<td>296.5±14.5</td>
<td>76.0±2.8</td>
<td>16.2±1.13</td>
</tr>
<tr>
<td>HFD20</td>
<td>202.4±16.4</td>
<td>275.8±12.7</td>
<td>73.3±3.9</td>
<td>16.5±0.98</td>
</tr>
<tr>
<td>LSD at 0.05</td>
<td>27.5</td>
<td>5.74</td>
<td>1.96</td>
<td></td>
</tr>
</tbody>
</table>

Data are expressed as the mean ± standard deviation. Values followed by the same letter (‘a’–‘e’) are not significantly different (P ≤ 0.05). N, American Institute of Nutrition93 G diet; HFD, high-fat diet; CSS, coffee silver skin; HFD10, HFD supplemented with 10% CSS; HFD15, HFD supplemented with 15% CSS; HFD20, HFD supplemented with 20% CSS; LSD, least significant difference.

Table 4

<table>
<thead>
<tr>
<th>Weights</th>
<th>N</th>
<th>HFD</th>
<th>HFD10</th>
<th>HFD15</th>
<th>HFD20</th>
<th>LSD at 0.05</th>
</tr>
</thead>
<tbody>
<tr>
<td>Liver</td>
<td>3.02±0.10</td>
<td>4.09±0.14</td>
<td>3.92±0.09</td>
<td>3.42±0.68</td>
<td>3.36±0.23</td>
<td>0.61</td>
</tr>
<tr>
<td>Kidney</td>
<td>0.78±0.07</td>
<td>0.98±0.02</td>
<td>0.89±0.08</td>
<td>0.80±0.04</td>
<td>0.79±0.05</td>
<td>0.10</td>
</tr>
<tr>
<td>Heart</td>
<td>0.45±0.01</td>
<td>0.47±0.04</td>
<td>0.46±0.05</td>
<td>0.45±0.07</td>
<td>0.45±0.03</td>
<td>0.08</td>
</tr>
<tr>
<td>Retroperitoneal fat</td>
<td>1.05±0.09</td>
<td>2.71±0.21</td>
<td>2.57±0.10</td>
<td>2.19±0.14</td>
<td>1.43±0.10</td>
<td>0.25</td>
</tr>
<tr>
<td>Epididymal fat</td>
<td>0.92±0.06</td>
<td>2.11±0.07</td>
<td>2.01±0.04</td>
<td>1.84±0.09</td>
<td>1.73±0.12</td>
<td>0.15</td>
</tr>
</tbody>
</table>

Data are expressed as the mean ± standard deviation. Values followed by the same letter (‘+’–‘−’) are not significantly different (P ≤ 0.05). N, American Institute of Nutrition93 G diet; HFD, high-fat diet; CSS, coffee silver skin; HFD10, HFD supplemented with 10% CSS; HFD15, HFD supplemented with 15% CSS; HFD20, HFD supplemented with 20% CSS.
in rats fed a HFD supplemented with various levels of CSS, as compared with rats fed a HFD alone (Table 5). The administration of HFD incorporated with various levels of CSS induced significant (P < 0.05) reductions in the heightened activities of ALT, AST, and ALP stimulated by HFD alone. The decrease gradually rise as the level of CSS was increased. The HFD20 diet reduced the concentrations of ALT, AST and ALP by 49.89, 38.04 and 41.90%, respectively.

Table 5 demonstrated that the HFD-fed rats exhibited an increase in the concentrations of urea, creatinine, and creatinine by 26.38, 8.40, and 6.75%, respectively as compared with the control. However, administration of CSS to HFD rats reverted back near to normal concentrations of the measured kidney parameters (creatinine, urea, and uric acid).

This might be explained by the consumption of a HFD stimulating the extrarenal path of nitrogen secretion.

### 3.4. Serum lipid profile of rats administered experimental diets

The effect of CSS supplementation for 8 weeks on serum total lipids, triglycerides, T-Ch, LDL-Ch, and HDL-Ch concentrations in rats fed a HFD was shown in Table 6.

Excluding HDL-Ch, all lipid fractions increased significantly in rats fed a HFD as compared with those fed an AIN-93 G diet (P < 0.05). Total lipids were increased by 27.36%, triglycerides by 76.60%, total cholesterol increased by 116.67% and LDL-Ch increased by 237.25%, compared with the control group. In rats receiving HFD, the HDL-Ch concentration for rats fed with HFD decreased sharply by 41.97% as compared with the control group. The administration of various levels of CSS to rats fed a HFD significantly improved all lipid parameters (P < 0.05) by decreasing serum total lipids, T-Ch, serum triglycerides and serum LDLs, and increasing serum HDLs. The highest dose of CSS, 20%, has improved the lipid parameters more than the lowest dose (10%).

The administration of HFD supplemented with 20% CSS has resulted in significant (P < 0.05) reductions in total lipids, T-Ch, LDL-Ch, and HDL-Ch concentrations in rats fed a HFD compared with rats fed a HFD alone (Table 5). The administration of HFD supplemented with 10% CSS; HFD15, HFD supplemented with 15% CSS; HFD20, HFD supplemented with 20% CSS; ALT, alanine aminotransferase; AST, aspartate aminotransferase; ALP, alkaline phosphatase; IU/L, international units per liter; LSD, least significant difference.

Data are expressed as the mean ± standard deviation (n = 5). Values followed by the same letter (***) are not significantly different (P > 0.05). N, American Institute of Nutrition93 G diet; HFD, high-fat diet; CSS, coffee silver skin; HFD10, HFD supplemented with 10% CSS; HFD15, HFD supplemented with 15% CSS; HFD20, HFD supplemented with 20% CSS. ALT, alanine aminotransferase; AST, aspartate aminotransferase; ALP, alkaline phosphatase; IU/L, international units per liter; LSD, least significant difference.

### Table 6

Effect of CSS supplementation for 8 weeks on serum total lipids, triglycerides, T-Ch, LDL-Ch and HDL-Ch concentration in rats fed a HFD.

<table>
<thead>
<tr>
<th>Group</th>
<th>Total lipids (mg/dL)</th>
<th>Total Cholesterol (mg/dL)</th>
<th>LDL (mg/dL)</th>
<th>HDL (mg/dL)</th>
<th>TG (mg/dL)</th>
<th>Atherogenic index</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>285 ± 4.09</td>
<td>133.75 ± 2.13</td>
<td>57.85 ± 3.11</td>
<td>52.13 ± 1.06</td>
<td>96.51 ± 1.13</td>
<td>0.26 ± 0.005</td>
</tr>
<tr>
<td>HFD</td>
<td>363 ± 5.31</td>
<td>289.80 ± 1.91</td>
<td>195.10 ± 2.56</td>
<td>30.25 ± 0.91</td>
<td>170.44 ± 1.09</td>
<td>0.75 ± 0.011</td>
</tr>
<tr>
<td>HFD10</td>
<td>336 ± 3.80</td>
<td>239.67 ± 3.14</td>
<td>173.14 ± 3.19</td>
<td>31.50 ± 0.72</td>
<td>161.72 ± 2.90</td>
<td>0.68 ± 0.001</td>
</tr>
<tr>
<td>HFD15</td>
<td>330 ± 4.20</td>
<td>201.71 ± 4.16</td>
<td>156.10 ± 2.37</td>
<td>38.14 ± 1.16</td>
<td>138.10 ± 2.15</td>
<td>0.55 ± 0.005</td>
</tr>
<tr>
<td>HFD20</td>
<td>325 ± 3.12</td>
<td>182.78 ± 2.93</td>
<td>122.11 ± 3.11</td>
<td>42.30 ± 0.39</td>
<td>116.78 ± 1.43</td>
<td>0.44 ± 0.005</td>
</tr>
<tr>
<td>LSD at 0.05</td>
<td>7.58</td>
<td>12.25</td>
<td>4.72</td>
<td>1.62</td>
<td>3.4</td>
<td>0.02</td>
</tr>
</tbody>
</table>

Data are expressed as the mean ± standard deviation (n = 5). Values followed by the same letter (***) are not significantly different (P > 0.05). CSS, coffee silver skin; N, American Institute of Nutrition93 G diet; HFD, high-fat diet; HFD10, HFD supplemented with 10% CSS; HFD15, HFD supplemented with 15% CSS; HFD20, HFD supplemented with 20% CSS; Ch, cholesterol; T, total; LDL, low density lipoprotein; HDL, high density lipoprotein.
from rats administered only the basal control diet. The microscopic examination of the liver tissue revealed the presence of normal hepatic parenchyma, including normal hepatic cords, central veins and portal areas (Fig. 1).

Fig. 2 demonstrated the histological examination of liver tissues of rats administered a HFD. The microscopic examinations indicated that the changes in the liver of rats fed with HFD were similar to the earlier changes observed in alcoholic hepatic disease, microvesicular and macrovesicular steatosis and fatty liver. All sections indicated steatosis, and fat droplets were accumulated within hepatic parenchymal cells.

A lower incidence of steatosis was observed in the hypercholesterolemic rats fed a HFD supplemented with various levels of CSS. The liver sections of those rats fed a HFD supplemented with 10% CSS revealed little vacuolar degeneration of the hepatocytes, mild fatty degeneration of the hepatocytes and widely spread fat vacuoles in the hepatocytes (Fig. 3).

The incorporation of a higher level of CSS, 15%, appeared to protect against the hyperlipidemic diet. Rats fed a HFD supplemented...
Fig. 7. Cross section of kidney tissues of rats administered HFD. Hematoxylin and eosin staining; magnification, x400. HFD, high-fat diet.

Fig. 8. Cross section of kidney tissues of rats administered HFD10. Hematoxylin and eosin staining; magnification, x400. HFD10, high-fat diet supplemented with 10% coffee silver skin.

Fig. 9. Cross section of kidney tissues of rats administered HFD15. Hematoxylin and eosin staining; magnification, x400. HFD15, high-fat diet supplemented with 15% coffee silver skin.

Fig. 10. Cross section of kidney tissues of rats administered HFD20. Hematoxylin and eosin staining; magnification, x400. HFD20, high-fat diet supplemented with 20% coffee silver skin.

with 15% CSS exhibited mild fatty degeneration of the hepatocytes and a moderate degree of recovery, in which the foremost hepatocytes exhibited a mild degree of vacuolar degeneration (Fig. 4). After increasing the level of CSS incorporation to 20%, a positive effect against the HFD was observed. The livers of rats administered a HFD supplemented with 20% CSS demonstrated a high degree of recovery and revealed almost normal hepatic lobules (Fig. 5).

The microscopic examination of kidney tissue revealed normal cortical and medullary structures, including renal tubules and glomeruli (Fig. 6). Fig. 7 demonstrated the histological examination of the kidney tissue of rats administered a HFD. The kidney tissue was swollen, with minimal change disease (lipoid nephrosis). The examination of the kidney tissue of rats administered a HFD supplemented with 10% CSS for 8 weeks revealed cloudy swelling and some hydropic degeneration of the renal tubular epithelial cells. However, the features of the kidney tissue of the aforementioned rats was similar to the features of the renal tissue of rats fed the control diet (Fig. 8). The kidney tissue of rats fed a HFD supplemented with 15% or 20% CSS for 8 weeks exhibited restored tissue with architecture and glomeruli, and a structure and pattern with no histopathological changes, similar to the control rats (Figs. 9 and 10).

4. Discussion

Saturated fatty acids are known to be stored in adipose tissue rather than used as fuel while monounsaturated fatty acids and polyunsaturated fatty acids are less preferably stored [4,5]. Therefore, reports of HFD induced obesity in animals are affected by the fatty acid content of the HFD [28]. The higher intake of fiber has been associated with lower body weights, a reduced demand for insulin, improved stool bulk, and improved purgative properties [29]. Dietary fibers are associated with increased secretion of mucin facilitating lubrication, whereas lack of fiber has led to colonic disorders [30]. The weight of organs is one of the most critical types of evidence with respect to the impact of the tested compounds, as marked differences in organ weight between treated and untreated animals might occur in the absence of any morphological variations [31]. The assessment of organ weight changes with respect to body weight variations has led to the use of the relative weights of organs to evaluate the effects of treatments during toxicology experiments [31]. The accumulation of triglycerides in the parenchymal cells of the liver (steatosis) is a well-known effect of extreme obesity.
Dietary fibers usually delay stomach emptying, and to reduce the concentration of LDL-Ch in hypercholesterolemic patients with liver and kidney dysfunction induced by high fat-diet. In general, hyperlipidemia is of clinical concern due to the risk of chronic coronary artery disease. To diagnose hypoad hyperlipoproteinemia, lipoprotein pattern and plasma lipids must be analyzed [42]. A reduction in HDL concentrations in animals fed a high cholesterol diet may be due to the acceleration of apolipoprotein A1 [43]. Dietary cholesterol (0.5%–1.0%) may increase serum VLDL-c and total cholesterol levels. These changes in the liver of rats fed with a HFD were similar to the earlier changes observed in alcoholic hepatitis and fatty liver. The administration of a high-cholesterol diet has resulted in an elevated organ lipid profile, which has been confirmed by several previous studies [33]. The atherogenic value has gained significance in previous years as a marker of atherosclerosis [21]. Each 1% reduction in serum T-Ch was estimated to yield a 2–3% reduction in the risk of developing coronary heart disease [33]. Numerous hypotheses were proposed for the mechanisms underlying the lipid-lowering effect of soluble fibers. They include altering the absorption of bile acid, adjustment of lipid metabolism adjustment, and the influence of short chain fatty acids that resulted from the process of fiber fermentation, or lipoprotein or cholesterol metabolism [38,50], and changes in the concentration of insulin or other hormones [50].

Animal studies revealed that the administration of a HFD induced steatosis which is characterized by an excess accumulation of triglycerides within liver cells [51,52]. Liver steatosis is a well-known pathology in severely obese patients and is particularly associated with visceral adiposity and diabetes. The disease might progress in certain patients to steatohepatitis or cryptogenic cirrhosis, and the histological mechanism of non-alcoholic fatty liver disease remains unknown [52]. Hepatocytes that exhibited larger nuclei and an expanded eosinophilic cytoplasm are one of the signs of regeneration [33,38,45]. A previous study revealed that hyperlipidemia damages renal vascular endothelial cells and causes glomerular interstitial cell proliferation by increasing platelet aggregation and platelet-derived growth factor levels [53]. Glomerular interstitial cell proliferation increased the concentration of LDL-Ch and free radical generation [54]. HFD-induced obesity causes hyperlipidemia, exhibiting structural and functional damage, including an increased glomerular filtration rate, increased renal blood flow and renal hypertrophy, as demonstrated in animals and humans [55]. In conclusion, the consumption of a HFD has increased the weight gain and the level of liver, kidney, epididymal and retroperitoneal fat of rats. The administration of a HFD induced marked increase in the concentrations of total cholesterol, LDL, TG, and AL. However, the incorporation of various levels of CSS into the HFD reduced these changes. The changes in the liver of rats fed with HFD were similar to the earlier changes observed in alcoholic hepatic disease and fatty liver. The administration of CSS to rats supplemented with 20% CSS showed a high degree of recovery and revealed almost normal hepatic lobules. The results of the present study has illustrated that the incorporation of CSS into HFDs has reduced the hyperlipidemia effect of these diets.

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