The low dose of drumsticks (*Moringa oleifera* L.) seed powder ameliorates blood cholesterol in hypercholesterolemic male rat

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**Abbreviations:** ALP, Serum alkaline phosphatase; ALT, Serum alanine aminotransferase; AST, Serum aspartate aminotransferase; Body wt., Body weight; BWG, Body weight gain; FER, Food efficiency ratio; GGT, Gamma-glutamyltransferase; G1, Negative control group; G2, Positive control group; G3, Hypercholesterolemic rats treated with 50 mg Moringa/kg body wt.; G4, Hypercholesterolemic rats treated with 100 mg Moringa/kg body wt.; HDLc, High-density lipoprotein cholesterol; LDH, Lactate dehydrogenase; LDLc, Low-density lipoprotein cholesterol; TC, Total cholesterol; TG, Triglyceride; VLDLC, Very low-density lipoproteins cholesterol

**Keywords:** Hypercholesterolemia, Liver, Moringa, Pathology, Triglyceride

Hypercholesterolemia is a major risk factor for coronary artery and heart diseases. Modification of lifestyle is the preferable form of treatment for most types of hyperlipidemia by following traditional and complementary herbal drugs¹,².

*Moringa oleifera* Lamarck (moringa or Drumstick-tree) of the family *Moringaceae*, is known in the developing world as a vegetable, a medicinal plant and a source of vegetable oil³. The aqueous and alcoholic extract of *Moringa oleifera* root-wood reduced the elevated urinary oxalate and lowered the deposition of stone forming constituents in the kidneys of calculogenic rats as a result of ethylene glycol treatment⁴. In developing countries, moringa is used to improve nutrition, boost food security, foster rural development support sustainable landcare, forage for livestock, a micronutrient liquid, a natural anthelmintic and possible adjuvant⁵,⁶. Vijay and Kumar⁷ reported that moringa increased wound healing of normal and dexamethasone suppressed wound healing in rats.

The crude leaves of *Moringa oleifera* Lam are used by Indians in herbal medicine as a hypocholesterolemic agent in obese patients and decreased the high-fat-diet-induced increases in serum, liver, and kidney cholesterol levels⁸. In addition, moringa seed extract has an ameliorative effect on liver fibrosis in rats by reducing of liver damage and symptoms of liver fibrosis, decreasing the CCl₄-induced elevation of serum aminotransferase activities and globulin level, reducing the elevated hepatic hydroxyproline content and myeloperoxidase activity⁹. Rajanandh *et al*.¹⁰ reported that moringa leaves could be prescribed as food appendage for coronary artery disease patients along with their regular medicines because they exhibit a hypolipidemic, antioxidant, anticoagulant, platelet anti-aggregatory and anti-inflammatory activity in experimental animals. Kumbhare¹¹ reported that the crude extract of moringa stem bark is a good...
scavenger for nitric oxide radicals and has a potential source of natural antioxidant.

Moringa leaf extract has high antioxidant activity because of its content of phenolics and flavonoids that have scavenging effect to the free radicals. It contains three classes of phytochemicals of medicinal benefits; glucosinolates such as glucomoringin, flavonoids such as quercetin and kaempferol, and phenolic acids such as chlorogenic acid. These three phytochemicals of moringa leaves and seeds possess antioxidant, hypoglycemic, hypotensive, antidysetropic, anticancer, and anti-inflammatory properties.

Asare et al. reported that moringa seed is used in the treatment of hypercholesterolemia and hyperglycemia, and also, as a nutritional supplementation. Its popularity use raises the question of possible toxicity at supra-supplementation levels. In spite of the medical and nutritional benefits of moringa, it was reported that it is genotoxic at supra-supplementation levels. Total phenolics, total flavonoids contents and antioxidant activity of Moringa oleifera leaf extract is increased by maceration and 70% ethanol extraction for high quality antioxidant raw material extract to be used in pharmaceutical and nutraceutical development.

This study aimed to test the efficiency of the seed powder of moringa in improving the lipid profile and protecting the heart of hypercholesterolemic male albino rats.

Materials and Methods

Plant materials

Moringa seeds were purchased from a local herbal medicine shop in Jeddah, Saudi Arabia and identified by botanists at King Abdulaziz Herbarium, Jeddah, Saudi Arabia. The seeds were washed, air dried, milled by the mixer and then mixed to the diet in a ratio of 50 mg/kg body weight and 100 mg/kg body weight.

Basal lipid-rich diet and cholesterol

The basal diet consisted of the following: 16% casein, 10% corn oil, 4% N.N cellulose, 4% salt mixture, 1% vitamin mixture, 0.2% choline chloride, 0.2% DL-methionine and 64.5% corn starch.

Animals and housing conditions

Twenty four male albino rats (Rattus norvegicus) of East China Origin weighing 180-200 g were obtained from Faculty of Pharmacy, King Abdulaziz University, Jeddah, Saudi Arabia. All the animal experiments were carried out under protocols approved by the Institutional Animal House of the University of King Abdulaziz at Jeddah, Saudi Arabia. Animals were housed six per polycarbonate cage. Cages, bedding, and glass water bottles (equipped with stainless steel sipper tubes) were replaced twice per week. The stainless steel feed containers were changed once per week.

Experiment design

The animals were fed a standard basal diet and kept under observation for 2 weeks before the start of the experiment to exclude any undercurrent infection. The test animals were divided randomly into four groups as follows: the first group is untreated control group fed basal lipid rich diet, the second group was fed 2% cholesterol (Sigma, USA) in the lipid rich diet to induce hypercholesterolemia and considered as the positive control group, the third group (G3) was fed 2% cholesterol and cotreated with 50 mg/kg body weight moringa seed powder for 8 weeks and the fourth group (G4) was fed 2% cholesterol and cotreated with 100 mg/kg body weight moringa seed powder for 8 weeks at libitum. The experiment was conducted for 8 weeks as an adequate period to induce hypercholesterolemia.

Preparation of the test diet

The basal fat-rich diet was milled using a mixer, the cholesterol amount was added to a ratio of 2%. The mixture was wetted with some water to make it hard paste, shaped to small balls, dried at 30°C and then kept in Fridge until use.

Moringa seeds were washed, air dried, milled by the mixer and then mixed to the 2% cholesterol diet powder (in a ratio of 50 mg and 100 mg/kg BW for feeding rats of G3 and G4, respectively). The mixture was wetted with some water to make it hard paste, shaped into small balls, dried at 30°C and then kept in Fridge until use.

Biochemical tests

At the end of the experiment, animals fasted 14-16 h after their last feeding, and blood samples were collected from the heart of Dimethyl-ether pre-anesthetized rats in plain tubes for biochemical analyses. Blood serum was obtained by centrifugation.
at 1000 rpm for 10 min at room temperature and then stored at −20°C until analysis was performed.

**Dissection**
At the end of the experiment and after collection of blood, anesthetize animals were sacrificed by cervical dislocation. The abdomen was dissected and the liver was rapidly excised. Part of the liver was saved in ice-cold for antioxidant enzymes and lipid peroxide estimation in tissue homogenate. A piece of the liver was saved in saline for histopathological investigations.

**Biochemical tests**

**Serum lipids**
Serum total cholesterol (S.TC), serum triglyceride (S.TG), serum high-density lipoprotein cholesterol was estimated colorimetrically using the Spinreact kit (Spain) according to the instruction of the supplier. The value of serum low-density lipoprotein cholesterol (S.LDLc) and serum very low-density lipoproteins cholesterol (VLDLc) was calculated as follows:

\[
S.LDL = S.TC - (HDL-C + S.TG/5)
\]

\[
S.VLDL = S.TC - (S.LDL + S.HDL)
\]

**Liver enzymes**
Serum alanine aminotransferase (ALT) was estimated colorimetrically using Human Kit (Germany), serum aspartate transaminase (AST) was estimated colorimetrically using Swemed diagnostics (India), serum alkaline phosphatase (ALP) was estimated colorimetrically using Human Kit (Germany) and serum Gamma-glutamyl transferase (GGT) was estimated colorimetrically using Abbott Diagnostics kit (USA). Estimation was done according to the instruction of the supplier.

**Estimation of lactate dehydrogenase**
Lactate dehydrogenase was spectrophotometrically assayed under ultraviolet using Human Kit (Germany) according to the instruction of the supplier.

**Estimation of creatine kinase-MB**
Creatine kinase-MB was estimated by enzyme immunoassay method using Oxis International Inc (USA) according to the instruction of the supplier.

**Quantification of bilirubin, total protein, globulins**
Total bilirubin, protein, and globulins were estimated colorimetrically using Human Kit (Germany) according to the instruction of the supplier.

**Estimation of albumin**
Albumin was estimated colorimetrically using Sigma-Aldrich (USA) according to the instruction of the supplier. Then the ratio of albumins: globulins was calculated.

**Biological evaluation**
Daily water consumption, food Intake, body weight gain (BWG), Percentage of Body weight gain (BWG%), Food efficiency ratio (FER) and Percentage of food efficiency ratio (FER%) were estimated.

**Histopathological investigations**
The liver was washed in sterile saline and fixed in 10% neutral formalin for histopathological studies. The tissue was then dehydrated in gradual ethanol (50-99%), cleared in xylene, and embedded in paraffin. Sections were prepared and then stained with hematoxylin and eosin (H&S) dye for microscopic investigation\(^{23}\). The stained sections were examined and photographed under a light microscope.

**Statistical analysis**
Values were analyzed using SPSS program to calculate the t-test and the mean ± SD and then analyzed using one-way analysis of variance (ANOVA) using Least Significance Difference (LSD) Test\(^{24}\).

**Results**

**Lipid profile**
Table 1 shows the effect of moringa seeds powder supplementation for 8 weeks on hypercholesterolemic male rats. The oral administration of 2% cholesterol to the rats of the group (2) for 8 weeks, significantly (at \(P < 0.001\)) increased serum total cholesterol, serum triglycerides, serum low-density lipoproteins and serum very low-density lipoproteins, and decreased serum high-density lipoproteins. The concurrent supplementation with 50 mg/kg body wt. of moringa to the hypercholesterolemic rats in G3, or 100 mg/kg body wt. in G4 significantly (at \(P < 0.001\)) ameliorated all lipid parameters by decreasing serum total cholesterol, serum triglycerides, serum low density lipoproteins and serum very low density lipoproteins, and decreasing serum high-density lipoproteins. The concurrent supplementation with 50 mg/kg body wt. of moringa to the hypercholesterolemic rats in G3, or 100 mg/kg body wt. in G4 significantly (at \(P < 0.001\)) ameliorated all lipid parameters by decreasing serum total cholesterol, serum triglycerides, serum low density lipoproteins and serum very low density lipoproteins, and increasing serum high density lipoproteins. The higher dose of moringa in G4 (100 mg/kg body wt.) was more efficient than the lower one in G3 in ameliorating lipid parameters.

**Liver enzymes**
In the positive control group (G2), liver enzymes (alanine aminotransferase, aspartate aminotransferase, alkaline phosphatase and Gamma-glutamyl transferase) were significantly (at \(P < 0.001\)) increased as a result
of cholesterol supplementation for 8 weeks as shown in Table 2. The concurrent supplementation of 50 mg/kg body wt. of moringa in G3 and 100 mg/kg body wt. of moringa in G4 significantly (at \( P < 0.001 \)) to the hypercholesterolemic male rats decreased all liver enzymes under study. The higher dose of moringa in G4 lowered the liver enzymes more than the lower one in G3.

**Lactate dehydrogenase**

In the positive control group (G2), lactate dehydrogenase enzyme activity was significantly (at \( P < 0.001 \)) increased as a result of cholesterol supplementation for 8 weeks as shown in Table 3. The concurrent supplementation of 50 mg/kg body wt. of moringa to the hypercholesterolemic male rats in G3 and 100 mg/kg body wt. of moringa in G4 for 8 weeks significantly (at \( P < 0.001 \)) decreased lactate dehydrogenase in the serum of hypercholesterolemic rats under study. The higher dose of moringa in G4 decreased lactate dehydrogenase more than the lower one in G3.

**Creatine Kinase-MB**

The serum creatine kinase-MB level was non significantly increased as a result of induced hypercholesterolemia in G2. Treating with the two

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Statistics</th>
<th>G1 −ve Control</th>
<th>G2 +ve Control</th>
<th>G3 50 mg moringa</th>
<th>G4 100 mg moringa</th>
</tr>
</thead>
<tbody>
<tr>
<td>S.TC mg%</td>
<td>Mean±SE LSD 0.05=6.076</td>
<td>132.67±2.83(^a)</td>
<td>292.83±1.42(^d)</td>
<td>216.50±2.01(^b)</td>
<td>181.17±1.24(^c)</td>
</tr>
<tr>
<td>S.T.G mg/dL</td>
<td>Mean±SE LSD 0.05=4.718</td>
<td>95.50±1.17(^a)</td>
<td>275.33±1.05(^d)</td>
<td>135.83±1.42(^b)</td>
<td>113.33±2.07(^c)</td>
</tr>
<tr>
<td>S.HDLc mg/dL</td>
<td>Mean±SE LSD 0.05=3.182</td>
<td>53.66±1.30(^a)</td>
<td>25.66±0.84(^d)</td>
<td>32.33±0.55(^b)</td>
<td>46.50±0.92(^c)</td>
</tr>
<tr>
<td>S.LDLc mg/dL</td>
<td>Mean±SE LSD 0.05=5.510</td>
<td>59.90±2.12(^a)</td>
<td>212.10±1.67(^c)</td>
<td>158.67±1.47(^b)</td>
<td>112.00±1.27(^d)</td>
</tr>
<tr>
<td>V.LDLc mg/dL</td>
<td>Mean±SE LSD 0.05=1.586</td>
<td>19.10±0.23(^a)</td>
<td>55.70±0.76(^c)</td>
<td>27.16±0.28(^b)</td>
<td>22.66±0.41(^c)</td>
</tr>
</tbody>
</table>

[Data are represented as mean ± SE. T-test values; *** \( P < 0.001 \). ANOVA analysis: within each row, means with different superscript (a, b, c or d) are significantly different at \( P < 0.05 \), whereas means, superscripts with the same letters mean that there is no significant difference at \( P < 0.05 \). LSD: Least significant difference].
doses of moringa in G3 and G4 nonsignificantly restored the creatine kinase-MB to its normal levels in G1 as shown in Table 3.

**Total bilirubin**

In Table 3, the level of bilirubin was not affected either by induced hypercholesterolemia in G2 or treating with the two doses of moringa in G3 and G4 as shown in Table 3.

**Serum proteins**

Total protein, albumin, globulin, and their A/G ratio were not affected with hypercholesterolemia as a result of cholesterol supplementation for 8 weeks in G2 or the concurrent administration of low dose of moringa in G3 or the higher dose in G4, as shown in Table 3.

**Biological evaluation**

Table 4 shows the effect of moringa seed powder supplementation for 8 weeks on biological evaluations in male rats. In G1, G2 and G3, the daily water consumption were nonsignificantly affected, whereas in G4 (fed on 100 mg/kg body wt. moringa seed powder), it was significantly (at $P<0.05$) increased compared with the negative control group. The daily food intake was not affected either by hypercholesterolemia or treating with moringa for 8 weeks. While the body weight gain in 8 weeks was high significantly (at $P<0.001$) increased in all groups compared with the initial weights. In addition, the percentage of BWG was significantly (at $P<0.05$) increased with induction of hypercholesterolemia in G2, G3 and G4 compared with the negative control in G1.

The food efficiency ratio and the percentage of food efficiency ratio were also high significantly (at $P<0.001$) increased as a result of hypercholesterolemia in G2, G3 and G4 compared with the negative control in G1.

**Histology of liver**

The histopathology of the liver is shown in Fig. 1. The hepatic tissues of the negative control group rats showing normal hepatic strands of cells, blood sinusoids and normal histological structure of hepatic lobule (Fig. 1A). The hepatic tissues of
hypercholesterolemic rats of the positive control group fed 2% cholesterol in the fat-rich diet for 8 weeks showing fatty hepatocytes and congestion of hepatic sinusoids, disrupted cells, disrupted hepatic strands, vacuolated cytoplasm, and necrosis (Fig. 1B). The hepatic tissues of hypercholesterolemic rats of the third group treated with 50 mg/kg body wt. moringa seeds powder for 8 weeks showing slight congestion of hepatic sinusoids and nearly restored normal appearance of the hepatic strands with well defined hepatic cords containing polyhedral hepatocytes and normal round nuclei (Fig. 1C). Fig. 1D shows hepatic tissues of hypercholesterolemic rats treated with 100 mg/kg body wt. moringa seeds powder for 8 weeks with restored normal hepatic appearance and normal hepatic strands.

**Discussion**

Dietary fiber is used more frequently in the treatment of diabetes, hypercholesterolemia, and diverticulitis through its effect on gastrointestinal function and reduction of low-density lipoprotein (LDL) cholesterol in hypercholesterolemic animals. The current study focused on the hypolipidemic activity of two doses of moringa seeds powder and the efficiency of these two doses in protecting the heart in male albino rats.

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**Table 4** — Effect of moringa seed powder supplementation on food intake (FI) body weight gain (BWG) and food efficiency ratio (FER) in hypercholesterolemic rats for 8 weeks.

<table>
<thead>
<tr>
<th>Biological evaluation parameters</th>
<th>Statistics</th>
<th>G1−ve Control</th>
<th>G2+ve Control</th>
<th>G3 50 mg moringa</th>
<th>G4 100 mg moringa</th>
</tr>
</thead>
<tbody>
<tr>
<td>Daily water consumption ml/day</td>
<td>Mean±SE</td>
<td>30.29±0.67a</td>
<td>30.55±0.63a</td>
<td>31.50±0.66a</td>
<td>32.66±0.57b</td>
</tr>
<tr>
<td></td>
<td>LSD 0.05=1.401</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>T-test</td>
<td>−0.32 NS</td>
<td>−1.36 NS</td>
<td>−3.45*</td>
<td></td>
</tr>
<tr>
<td>Daily food intake g/day</td>
<td>Mean±SE</td>
<td>17.07±0.19a</td>
<td>17.12±0.20a</td>
<td>17.20±0.21a</td>
<td>17.05±0.21a</td>
</tr>
<tr>
<td></td>
<td>LSD 0.05=0.290</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td></td>
<td>T-test</td>
<td>−0.40 NS</td>
<td>−0.46 NS</td>
<td>0.58 NS</td>
<td></td>
</tr>
<tr>
<td>BWG /8 week</td>
<td>Mean±SE</td>
<td>27.00±1.06a</td>
<td>32.83±1.85b</td>
<td>34.50±0.88c</td>
<td>33.50±3.47d</td>
</tr>
<tr>
<td></td>
<td>LSD 0.05=6.379</td>
<td></td>
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<tr>
<td></td>
<td>T-test</td>
<td>−2.66***</td>
<td>−4.83***</td>
<td>−5.98***</td>
<td></td>
</tr>
<tr>
<td>BWG %</td>
<td>Mean±SE</td>
<td>17.83±1.08a</td>
<td>20.66±6.50b</td>
<td>18.50±0.53d</td>
<td>17.70±2.04c</td>
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<tr>
<td></td>
<td>LSD 0.05=9.244</td>
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<tr>
<td></td>
<td>T-test</td>
<td>−2.97*</td>
<td>−2.78*</td>
<td>−3.20*</td>
<td></td>
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<tr>
<td>FER g/day</td>
<td>Mean±SE</td>
<td>0.026±0.00a</td>
<td>0.031±0.00b</td>
<td>0.033±0.00c</td>
<td>0.032±0.00d</td>
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<tr>
<td></td>
<td>LSD 0.05=0.005</td>
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<tr>
<td></td>
<td>T-test</td>
<td>−0.92***</td>
<td>−0.04***</td>
<td>−0.40***</td>
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<tr>
<td>FER %</td>
<td>Mean±SE</td>
<td>3.199±0.18a</td>
<td>4.80±0.87b</td>
<td>3.37±0.08c</td>
<td>3.27±0.33d</td>
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<td></td>
<td>LSD 0.05=0.617</td>
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<tr>
<td></td>
<td>T-test</td>
<td>−1.04***</td>
<td>−1.45***</td>
<td>−1.17***</td>
<td></td>
</tr>
</tbody>
</table>

[Data are represented as mean ± SE. T-test values; *** P <0.001. ANOVA analysis: within each row, means with different superscript (a, b, c or d) are significantly different at P <0.05, whereas means superscripts with the same letters mean that there is no significant difference at P <0.05. LSD: Least significant difference].

Fig. 1 — (A) Liver of rat from group 1 showing the normal histological structure of hepatic lobule, B; Liver of rat from group 2 showing fatty hepatocytes and congestion of hepatic sinusoids, C; Liver of rat from group 3 showing slight congestion of hepatic sinusoids and D; Liver of rat from group 4 showing slight dilatation and congestion of hepatic sinusoids (H & E × 400).
Feeding rats with 2% cholesterol in the diet for 8 weeks increased the serum total cholesterol and induced hypercholesterolemia in the positive control group\textsuperscript{21,27}. The concurrent oral administration of 50 and 100 mg/kg body wt. moringa to the hypercholesterolemic rats of G3 and G4, respectively for 8 weeks significantly improved the serum lipid profile parameters. It decreased the total cholesterol, triglycerides, low-density lipoproteins and very low-density lipoproteins and increased the useful form of cholesterol (high-density lipoproteins). This result is in concordant with the results of Kane and Malloy\textsuperscript{28} who reported that β-sitosterol of moringa is a sterol (similar in its structure to cholesterol, except for the substitution of an ethyl group at C24 of its side chain) is responsible for lowering plasma concentrations of LDL, so it may be a bioactive phytoconstituent of moringa. It is believed that hypercholesterolemia occurs in conjunction with other metabolic risk factors including glucose intolerance, obesity, diabetes, metabolic syndromes and oxidation of the lipid core of low-density lipoproteins that leads to a change in the lipoprotein conformation\textsuperscript{29}.

Furthermore, liver function parameters (serum alanine aminotransferase, serum aspartate transaminase, serum Gamma-glutamyltransferase and serum alkaline phosphatase) were also significantly increased under an induced hyperlipidemic condition in male rats. This result is consistent with that of Mahfouz and Kummerow\textsuperscript{30} and El Rabey et al.\textsuperscript{27}. The concurrent supplementation of the two doses of moringa to the hypercholesterolemic rats in G3 and G4 significantly ameliorated the liver enzymes under study and nearly restored them to the normal conditions. This result is consistent with the previous investigations\textsuperscript{8,9}.

The heart enzymes; lactate dehydrogenase and creatine kinase-MB were also significantly increased under an induced hyperlipidemic condition in the G2 rats. This result is consistent with that of Rathod et al.\textsuperscript{20}. Lactate dehydrogenase and creatine kinase-MB are released during heart tissue damage resulted from hypercholesterolemia. The concurrent supplementation of the two doses of moringa to the hypercholesterolemic rats in G3 and G4 significantly ameliorated the heart enzymes under study and nearly restored them to the normal conditions. This result is consistent with the previous investigations\textsuperscript{10}.

In addition, total bilirubin, total protein, albumins, globulins and albumins/globulins ratio were not affected with either hypercholesterolemia or concurrent supplementation of moringa, in the current study. On the other hand, water consumption and food intake were not affected by hypercholesterolemia or treating with moringa seeds powder, whereas body weight gain and food efficiency ratio were significantly increased by hypercholesterolemia and decreased by treatment with Moringa in for 8 weeks in G3 and G4.

The hepatic tissues were significantly affected in hypercholesterolemic rats of G2 as a result of the 2% cholesterol supplementation. They showed fatty hepatocytes and congestion of hepatic sinusoids, disrupted cells, disrupted hepatic strands, vacuolated cytoplasm, and necrosis. This result is consistent with other studies showed a correlation between hypercholesterolemia\textsuperscript{27,31-33}. The concurrent supplementation of moringa in G3 and G4 significantly improved the liver tissues and nearly restored them to their normal state.

It could be concluded that the two low doses of moringa under study succeeded in lowering hyperlipidemia induced by cholesterol feeding for 8 weeks in male rats by lowering TC, TG, LDLc, and VLDLC and increasing HDLc levels in the treated groups. Moringa treatment also improved liver enzymes, heart enzymes and nearly restored tissues of the liver to their normal structure. The high dose of moringa in G4 was more effective than the lower dose of G3.

Acknowledgement

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