Influence of dietary supplementation of Garden cress (Lepidium sativum L.) on histopathology and serum biochemistry in Diabetic Rats

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Abstract

The goal of this study was to determine histopathology some biochemical activities of two concentrations of Garden cress (Lepidium sativum L.) on normal and diabetic rats for 30 successive days. Forty mature rats of an average body weight of (150-180 gm) were divided into 4 groups 10 rats each. (G1) control, (G2) diabetic control group injected intraperitoneal by alloxan in a dose 150 mg/kg b.wt. (G3) diabetic rats fed on basal diet mixed with 1% dried Garden cress (Lepidium sativum L.), (G4) diabetic rats fed on basal diet mixed with 2% dried Garden cress (Lepidium sativum L.). At the end of the experiment, blood samples were collected to separate serum by centrifugation to determine some biochemical parameters, some oxidative markers, relative organs weights and histopathology of liver, kidney and pancreas were conducted at the end of the study. Rats were sacrificed for obtaining tissues samples (liver, Kidney and pancreases) for histopathological examination and organs were weighed to determine relative organs weight.

Results revealed that there were a significant decrease of serum levels of AST, ALT, ALP, cholesterol, triglyceride, urea and glucose as well as MDA than diabetic control groups at P<0.05. On the other hand result showed no effect on albumin, globulin and A/G ratio. While catalase enzyme activity showed a significant increase than control diabetic rats. The relative organs weight in (G3) and (G4) returned toward normal values. Conclusively, feeding of Garden cress at both concentrations for diabetic male had a beneficial practical tool to minimize the effect of diabetes without any adverse effect on metabolic parameters and organs weight of rats.

Keyword: Alloxan, Garden cress (Lepidium sativum L.), Histopathological examination, Biochemical parameters

Introduction

Diabetes mellitus (DM) is a metabolic disorder characterized by chronic hyperglycemia linked with total or partial deficiencies in insulin secretion or
function. It is one of the most frequent chronic diseases affecting millions of people globally leading to morbidity and mortality worldwide particularly in developing countries of Africa, Asia, and South America (Ping, et al 2010, and Cazzola and Estaro, 2014). Diabetes mellitus is considered an extended chronic metabolic disease that causes several other complications, such as cardiovascular diseases; fixed cost used for its treatment placed a huge burden on the economy and health systems worldwide (Prabhakar and Doble, 2011 and Lu, et al, 2012). Traditional medicines and plant-based systems continue to play an essential role in healthcare (Ranilla et al, 2010). Cinnamon and Garden cress seeds are members of a list containing 150 plants which are used in the treatment of diabetes mellitus (Eddouks et al, 2005)

Diabetes mellitus (DM) is characterized by absolute or relative deficiencies in insulin secretion and or insulin action associated with chronic hyperglycemia and disturbances of carbohydrate, lipid and protein metabolism. In addition, various biochemical disorders associated with vascular complications such as hyperlipidemia and oxidative stress frequently coexist with diabetes mellitus (DM) (Mosaad and Abd Allah, 2004).

Medicinal plants continue to provide valuable therapeutic agents, in both modern traditional and medicine system. The doubts about the efficacy and safety of the oral hypoglycemic agents have prompted a search for safer and more effective drugs in the treatment of DM (El-demerdash et al., 2005). In spite of the fact that insulin has become one of the most important therapeutic agents known to medicine researchers. It has been making efforts to find insulin substitutes for synthetic or plant sources for the treatment of diabetes. Many herbs have remained as an alternative to conventional therapy especially in poor areas where insulin is not readily available. (Sanchez et al., 1994).

Garden cress (Lepidium sativum) has been widely used to treat a number of ailments in traditional system of medicine. Lepidium sativum (L. sativum) is a fast-growing edible herb that belongs to the family Cruciferae (Brassicaceae; or Mustard family), and is being cultivated as culinary vegetable in North America, Europe, and all over Asia including India (Nadkarni, 1976). This is a popular herbal plant grown in many regions of Saudi Arabia, such as Hijaz, AL-Qaseem, and the Eastern Province and is called “Habel Rashaad” or “Thufa” (Ageel et al., 1987; Rahman et al., 2004). In Europe and America, the leaves are used in salad. In various countries of Africa, Lepidium sativum seeds are thought to be an effective medicinal remedy to cure respiratory disorders, like bronchitis and asthma (Kloos, 1976; Nadkarni, 1976). The edible whole seed is known to have health promoting properties hence; it was assumed that these seeds can serve as raw material for functional foods; its peppery tangy flavor and aroma (Snehal et al., 2012; Rehman et al., 2012). L. sativum plant is known to contain ascorbic acid. Seeds of L. sativum contain
carbohydrate, protein, fatty acid, riboflavin, niacin, flavonoids, isothiocynates glycoside, essential aromatic oils, and fatty oils (Nadakarni, 1976). Garden cress (Lepidium sativum L.) contains mucilage in its dry seed coat that has been isolated using dissimilar solvents and utilized by researchers as an excipient in a variety of pharmaceutical formulations for preferred functionality (Prajapati et al, 2014) it showed that Garden cress seed (L. sativum) have antiasthmatic (Paranjape and Mehta, 2006), bronchodilator (Mali et al., 2008), anti-inflammatory, analgesic, anticoagulant (Al-Yahya et al., 1994), antirheumatic (Ahsan et al., 1989), hypoglycemic (Patole, 1998), laxative, prokinetic (Rehman et al., 2011), antihypertensive (Maghrani et al., 2005) and diuretic (Patel et al., 2009) activities. It also has antihyperglycemic properties which help to control glucose level in diabetics (Behrouzian et al, 2014and Hassan et al, 2015). The seeds of L. sativum are aperient, diuretic, tonic, demulcent, carminative, galactagogue, and emmenagogue, are used to induce an abortion, and also possess antibacterial and antifungal properties (Bansal, et al 2012). The present study aimed to estimate the stimulation of the pancreas by the antidiabetic effect of 1 and 2% of garden cress seed (Lepidium sativum) and histopathological changes in male rats with alloxan induced diabetes.

Materials and Methods

Materials:

Plants:

Garden cress (Lepidium sativum) was obtained from Faculty of Agriculture farm. Plants were identified by Faculty of Pharmacy, Department of Pharmacognosy, Cairo University, Egypt. The plant was air dried then grinded and kept in a glass bottles till mixed with ration.

Chemicals:

Alloxan tetrahydrate pure (99%) was obtained from Sigma Company (Germany)

Experimental animals:

Forty apparently healthy male Albino rats of an average body weight 150-180g were obtained from the laboratory of animal colony, Helwan, Cairo, Egypt. Rats were fed on standard ration and water supply was given ad-libtum.

Preparation of diabetic rats:

Alloxan tetrahydrate pure (99%) was obtained from Sigma Company (Germany) was dissolved in sterile distilled water and injected to thirty rats intraperitoneally at a dose of 150 mg/Kg b wt according to (Desai and Bhide, 1985).

Experimental design:

Forty mature rats were divided into 4 equal groups. All groups were fed the experimental basal diet with or without the tested plant for 30 successive days as
follows:

Group (1): Control group was fed a basal diet.
Group (2): Diabetic rats were fed on basal diet.
Group (3): Diabetic rats were fed on basal diets mixed with *Lepidium sativum* in Concentration of 10g/kg ration (1%).
Group (4): Diabetic rats were fed on basal diets mixed with *Lepidium sativum* in concentration of 20 g/kg ration (2%).

**I - Sampling:**

**A- Blood samples:**

They were collected from each rat at the end of the experiment. Blood samples were taken from retro-orbital venous plexus into clean, sterile and labeled centrifuge tubes to separate serum to determine some biochemical parameters as follows:

Serum aspartate aminotransferase (AST) and serum alanine transferase (ALT) were determined according to Reitman and Frankel (1957) and alkaline phosphatase was measured were assayed (Malondialdehyde MDA) according to Ohkawa et al., (1979) and according to the method described by Tietz (1986). Total cholesterol (T.chol.) and triglycerides (Trigs) were estimated according to the method described by Watson (1960) and Whalerfeld (1974) respectively. Urea and glucose were measured spectrophotometrically according to the method described by Reises et al., (1965) and Trinder, (1969 ) respectively. Total protein (TP) and albumin (ALb) were determined according to method described by Weichselbaum (1946) and Doumas et al., (1971) respectively while globulin (glob) was calculated by subtracting albumin levels from total protein levels and A/G ratio was calculated mathematically. Activity of catalase enzyme was determined as reported by Aebi, (1974).

**B- Tissue sample:**

At the end of the experiment rats of all groups were weighed then sacrificed. Organs (liver, kidney, heart and spleen) were taken and weighed to calculate the relative organs weights (the organ/body weight per 100g ratio )

**II- Histopathological examination**

The histopathological samples (liver , kidney and pancreas) were fixed in 10% neutral buffered formalin. Fixed tissue samples were processed routinely by the paraffin embedding technique. Sections at 4 micron thickness were stained with Hematoxylin and Eosin (Bancroft and Gamble,2002)

**III- Statistical analysis:**

Parametric data were statistically analyzed by using ANOVA test and comparison between means were preformed using Duncan Multiple range test for comparative of
means using **SPSS version 14** (2006). Results were represented as (Mean + S.E.).

**Results and Discussion**

Diabetes mellitus (DM) is a chronic, systemic and metabolic disease manifested by hyperglycemia. It is characterized by alteration in the metabolism of carbohydrate, protein and lipids. The cumulative effects of these metabolic derangement lead to cell damage and circulatory changes. Other clinical consequences of diabetes include nephropathy, retinopathy and liver dysfunction (Wild et al., 2004). Herbal and natural products represents the most common forms of complementary and alternative medicine (Graham et al., 2005). They are readily available and can be obtained from supermarkets and pharmacies. As these products are usually used without medical prescription, they must be safe for human (Ernst, 2006). Numerous studies have reported the antioxidant properties of many natural products against many toxic materials (Shati and Alamri 2010). Antihyperglycemic effects of these plants were attributed to their ability to restore the function of pancreatic tissues by causing an increase in insulin output or inhibit the intestinal absorption of glucose or to the facilitation of metabolites in insulin dependent processes (Oliver – Bever, 1986). Most plants contain glucoside, alkaloids, terpenoids, flavonoids, and carotenoid which may be implicated as having antidiabetic and antioxidant effect (Loew and Kaszkin, 2002).

The present study was carried out to investigate the ameliorative effect of available herb as *Lepidium sativum* on some biochemical parameters, antioxidant markers and histopathology in normal and diabetic rats. The obtained results in (Table 1) showed that feeding rats on a ration mixed with *Lepidium sativum* 1% and 2% produced a significant decrease in AST, ALT and Alp levels at P<0.05. This finding are in agreement with (Datta et al. 2002) and (Safaa et al 2016) who studied the hypoglycemic and antioxidant effects of *Lepidium sativum* extracts in diabetic rats. They reported that all elevated above mentioned biochemical parameters are increased in the lowered doses, restoring them nearly to the normal levels of G1.

Moreover, Thnaian Althnaian, 2014; Chauhan et al.,2012& Al Hamedan, 2010 reported that the activities of AST and ALT remained comparable to that of control groups. The increase in ALT activity of rats fed high cholesterol diet comparing to control.

Data in (Table 1) indicate that diabetic rats showed a significant elevation in total cholesterol and triglyceride levels comparing with control group while rats fed *Lepidium sativum* showed significant reduction in total cholesterol and triglyceride concentrations compared to diabetic control group at P < 0.05. Similar observations on *Lepidium sativum* were reported with (Jelodar et al. 2007 and Khan and
Balishe 2001) and who found that Lepidium sativum on some biochemical parameters of decreased serum levels of total cholesterol and triglyceride of diabetic rats. Also, Thnaian Althnaia, (2014) reported that oral administration of Lepidium sativum or Their extracts at 5% and 10% lowered total cholesterol and triglyceride concentrations.

The effect of feeding 1% and 2% Lepidium sativum to normal and diabetic rats are recorded in (Table 1). Significant reduction in urea level (P < 0.05) in groups fed 1% Lepidium sativum than diabetic rats. These results are consistent with (Datta et al. 2002) and (Safaa et al. 2016) who recorded that Lepidium sativum are effective in reducing urea and uric acid levels and have the ability to decrease inflammation and oxidative stress, they are also rich in vitamins B complex, A and C.

Concerning glucose level (Table 1) it was reduced significantly in Lepidium sativum treated rats as compared to diabetic group rats at P < 0.05. This finding was in agreement with Khalid et al. (2013). This effect may be attributed to their phenolic content which are known to be involved in the scavenging of free radical mediated oxidative stress and consequently the diseases. Moreover the flavonoids content which are the active principles, it also possesses an inhibitory effect on the aldose reductase enzyme. This enzyme played a role in catalyzing the reduction of glucose to sorbitol which cannot diffuse out of cell membrane. (Bafeel and Ali, 2009 and Jain et al. 2009).

Data tabulated in (Table 2) indicated that feeding Lepidium sativum to diabetic rats at concentrations 1% & 2% for 30 successive days exhibited no marked alterations in the level of total protein, albumin, globulin and A/G. Our finding is in agreement with (Thnaian Althnaia, 2014; Al-Tae 2013 Kholif and El-shewy, 2004) who described that the administration of Lepidium sativum showed insignificant differences in serum total proteins, albumin, globulins in rats and rabbits fed on cholesterol diet.

However, the findings of our study disagreed with that of Bafeel and Ali (2009) who reported that the oral administration of LS in different concentrations to rats showed an increase in serum total protein, while albumin increase only at high concentration group.

Table) demonstrated the antioxidant parameters after feeding 1% & 2% Lepidium sativum to diabetic rats. The results revealed significant decrease MDA levels while catalase activity was significant increased in groups fed the tested plants compared to diabetic control rats at P<0.05. Our results consistent with Safaa et al. 2016 and Yae et al., (2010) who said that Lepidium sativum contains high level of phenolic content that cause scavenging of free radicals which is one of the major antioxidation mechanisms to inhibit the chain reaction of lipid peroxidation (Ghosh et al., 2015 and Dugoua et al., 2007) In addition, our results concerning the
elevation of MDA and reduction in antioxidants enzyme activity due to induction of diabetes and amelioration after L.sativum. Antioxidant compounds like phenolic acids, polyphenols, terpenoids and flavonoids scavenge the free radicals and thus inhibit the oxidative stress (Kintzios et al. 2010).

**Table (4):** illustrated the effect of feeding tested plant L.sativum in concentration 1% & 2% for 30 successive days on relative organ weight (kidney, liver and spleen) of normal and diabetic rats. The total body weight (g) in G2 showed a significant decrease as a result of induction of diabetes, whereas it increased with received Lepidium sativum. This result is consistent with that of (Beejmohun et al., 2014 and Safaa et al., 2016). In our study, weight of kidney, and liver in all groups showed a significant increase as a result of diabetes. Restoring the normal organs weight as a result of treating diabetic rats with cress seed is consistent with (Elgawish and Abdelrazek 2014).

The histology of the pancreas (**Figure 1**) reveals that the normal control rats had intact pancreatic islets and exocrine cells. However, alloxan-induced diabetic rats (diabetic control rats) showed depleted islet cells (DIL) and areas of cell necrosis (CN). Diabetic rats that had been treated with the 1% LS had small, preserved islet cells (PIL), which is an improvement from what occurred in the untreated alloxan-induced diabetic rats. Further improvements were observed in rats that had been treated with 2% of LS, such as more prominent islet cells and exocrine cells, which indicated an improvement in the architecture of the pancreas as the concentration of the Lepidium sativum increased.

The histology of the kidneys is shown in (**Figure 2**). The kidney of a normal rat has glomeruli (GL) and a compact-tissue appearance (**Figure 4a**), the tissue of untreated, alloxanised rats (positive control rats) showed the presence of dissociated cells (DC) due to cell necrosis and widespread infiltrations by inflammatory cells (IC) (**Figure 4b**). However, as the concentration of LS increased from 1% to 2%, noticeable improvements in the tissue architecture were evident, and more visible glomeruli and fewer inflammatory cells were observed (**Figure 4c–4d**).

The histology of the liver is shown in (**Figure 3**). (a) The histology of normal, control rats, showing normal arrangement of hepatocytes. (b) cell necrosis was evident in the untreated, alloxanised rats (positive-control rats). (c&d) livers of treated groups with LS. In these instances, the liver tissues were compact and healthy.

The histological studies showed altered pathological changes in the tissues of kidney, liver and pancreas as a result of diabetes in the positive control group similar (Ping et al., 2010 & Al-Malki and El Rabey, 2015) whereas treating the diabetic rats with cress seed restored the altered tissues nearly to the normal conditions. Ullah et al. (2012) stated that Garden cress and cinnamon significantly attenuated aminoglycosides-kidney toxicity by improving the urea, creatinine, uric acid, urinary...
protein levels, and histopathological alterations of the kidneys. In addition, our result is in agreement with that of Al-Malki and El Rabey (2015)

**Conclusion:**
It could be concluded that both cress seed (*Lepidium sativum*) seeds succeeded in controlling hyperglycemia in rats with alloxan induced diabetes. These seeds also ameliorated all biochemical parameters and kidney, liver and pancreas functions and tissues and restored them to the normal state. These effects may be due the antioxidant activity of both phenolic and flavonoids phytochemical constituents of these seeds.
Table (1): Effect feeding 1% and 2% of *Lepidium sativum* for 30 successive days on some biochemical parameters in normal and diabetic rats (n =10)

<table>
<thead>
<tr>
<th>Groups</th>
<th>AST (U/L) ±0.24</th>
<th>ALT (U/L) ±0.596</th>
<th>ALP. (U/L) ±0.618</th>
<th>T.Chol. (mg/dl) ±2.7</th>
<th>Trig. (mg/dl) ±0.62</th>
<th>Urea (mg/dl) ±0.046</th>
<th>Glucose (mg/dl) ±2.92</th>
</tr>
</thead>
<tbody>
<tr>
<td>control</td>
<td>25.26 ±0.24a</td>
<td>12.5 ±0.596a</td>
<td>40.38 ±0.618a</td>
<td>62.23 ±2.7a</td>
<td>76.56 ±0.62a</td>
<td>36.23 ±0.046a</td>
<td>104.7 ±2.92a</td>
</tr>
<tr>
<td>Diabetic control</td>
<td>49.4 ±0.192b</td>
<td>25.65 ±0.152b</td>
<td>80.49 ±1.57b</td>
<td>90.77 ±3.375b</td>
<td>166.4 ±1.5b</td>
<td>39.05 ±0.877b</td>
<td>211.13 ±3.6b</td>
</tr>
<tr>
<td>Diabetic rats Fed 1% L.S</td>
<td>24.31 ±0.2c</td>
<td>9.5 ±0.37c</td>
<td>34.28 ±0.29c</td>
<td>99.34 ±0.18c</td>
<td>55.4 ±2.2a</td>
<td>29.23 ±0.15c</td>
<td>110.32 ±0.17c</td>
</tr>
<tr>
<td>Diabetic rats fed 2% L.S</td>
<td>43.268 ±0.219d</td>
<td>15.4 ±0.416d</td>
<td>45.58 ±0.163d</td>
<td>87.6 ±2.55b</td>
<td>162.3 ±0.21c</td>
<td>36.212 ±0.189a</td>
<td>140.6 ±0.134d</td>
</tr>
</tbody>
</table>

Mean±SE. Means with different superscripts in the same column are significantly (P<0.05) different.
Table (2): Effect of feeding 1% and 2% of *Lepidium sativum* for 30 successive days on some protein profile in normal and diabetic rats (n = 10)

<table>
<thead>
<tr>
<th>Groups</th>
<th>T.P (g/dl)</th>
<th>Alb (g/dl)</th>
<th>Globulin (g/dl)</th>
<th>A/G</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>6.44 ± 0.13a</td>
<td>3.55 ± 0.098a</td>
<td>2.89 ± 0.125a</td>
<td>1.24±0.07a</td>
</tr>
<tr>
<td>Diabetic Control</td>
<td>6.5 ± 0.245a</td>
<td>4.79 ± 0.17b</td>
<td>1.7 ± 0.156b</td>
<td>2.96±0.32b</td>
</tr>
<tr>
<td>Diabetic rats Fed 1% L.S</td>
<td>6.21±0.04ab</td>
<td>3.9±0.11cd</td>
<td>1.99±0.08b</td>
<td>2.2±0.13bc</td>
</tr>
<tr>
<td>Diabetic rats fed 2% L.S</td>
<td>6.36 ± 0.175a</td>
<td>3.812 ± 0.134bc</td>
<td>1.92 ± 0.29b</td>
<td>1.7±0.2abcd</td>
</tr>
</tbody>
</table>

Mean±SE. Means with different superscripts in the same column are significantly (P<0.05) different.

Table (3): Effect of feeding 1% and 2% of *Lepidium sativum* for 30 successive days on some oxidative markers in normal and diabetic rats (n=10)

<table>
<thead>
<tr>
<th>Groups</th>
<th>MDA (nmol)</th>
<th>Catalase (mmol)</th>
</tr>
</thead>
<tbody>
<tr>
<td>control</td>
<td>7.83 ± 0.28a</td>
<td>196 ± 1a</td>
</tr>
<tr>
<td>Diabetic control</td>
<td>10.4 ± 0.37b</td>
<td>110.8 ± 3.2b</td>
</tr>
<tr>
<td>Diabetic rats fed 1% L.S</td>
<td>8.6 ± 0.31b</td>
<td>134 ± 4.84abc</td>
</tr>
<tr>
<td>Diabetic rats fed 2% L.S</td>
<td>8.8 ± 0.32b</td>
<td>124.8 ± 2.35c</td>
</tr>
</tbody>
</table>

Mean±SE. Means with different superscripts in the same column are significantly (P<0.05) different.
Table (4): Effect of feeding 1% and 2% of *Lepidium sativum* for 30 successive days on relative organs weight in normal and diabetic rats (n=10)

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Body weight (g)</th>
<th>Kidney (g)</th>
<th>Liver (g)</th>
<th>Spleen (g)</th>
</tr>
</thead>
<tbody>
<tr>
<td>control</td>
<td>206.6 ±3.06&lt;sup&gt;a&lt;/sup&gt;</td>
<td>0.71 ±0.007&lt;sup&gt;a&lt;/sup&gt;</td>
<td>2.74 ± 0.04&lt;sup&gt;a&lt;/sup&gt;</td>
<td>0.192 ±0.007&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>Diabetic control</td>
<td>195 ± 4.28&lt;sup&gt;b&lt;/sup&gt;</td>
<td>0.85 ±0.022&lt;sup&gt;b&lt;/sup&gt;</td>
<td>2.51 ±0.092&lt;sup&gt;a&lt;/sup&gt;</td>
<td>0.364 ±0.02&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>Diabetic rats fed 1% L.S</td>
<td>203.33±2.11&lt;sup&gt;a&lt;/sup&gt;</td>
<td>0.765±0.07&lt;sup&gt;c&lt;/sup&gt;</td>
<td>2.63±0.15&lt;sup&gt;a&lt;/sup&gt;</td>
<td>0.242±0.009&lt;sup&gt;c&lt;/sup&gt;</td>
</tr>
<tr>
<td>Diabetic rats fed 2% L.S</td>
<td>201.7±2.01&lt;sup&gt;ab&lt;/sup&gt;</td>
<td>0.792±0.021&lt;sup&gt;c&lt;/sup&gt;</td>
<td>2.76±0.16&lt;sup&gt;a&lt;/sup&gt;</td>
<td>0.255±0.017&lt;sup&gt;c&lt;/sup&gt;</td>
</tr>
</tbody>
</table>

Mean±SE , Means with different superscripts in the same column are significantly (P<0.05) different
Figure 1: (a) Photomicrography of pancreas of rat from the negative control group showing normal pancreatic islets and glands; (b) photomicrography of pancreas of rat from the positive control group shows severe pathological changes; (c&D) photomicrography of pancreas of rat from G3&G4 treated with 1%&2% *L. sativum* (H&E ×200).
Figure 2: (a) Photomicrography of a kidney of negative control group (G1) reveals normal histological structure; (b) photomicrography of a kidney of the positive control group with pathological changes; (c&D) photomicrography of a kidney of G3&G4 treated with *L. sativum* (1%&2%) shows nearly normal tissues; ((H&E ×200).
Figure 3 Histopathological image of diabetic rat liver and treated with two doses of Lipidium sativum (1% & 2%). (1a) Liver of control rats showing normal central vein and regular hepatic cords (arrow), HE bar = 40 μm. (1b) Liver of diabetic rats showing fatty degeneration (arrowhead) and fatty cysts (arrow). (1c) Liver of Lepidium sativum (LS) powder 1% diet treated rats showing mild vacuolar degeneration of hepatocytes (arrow). (1d) Liver of Lepidium sativum (LS) powder 2% diet treated rats showing the same for 1c, mild vacuolar degeneration of hepatocytes (arrow).

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