Study the Effect of Some Natural Substances on Lowering Blood Cholesterol in Rats

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ABSTRACT

The present work was conducted to study the effect of soy lecithin capsules and sumac (*Rhus coriaria* L.) spices on the nutritional parameters of rats suffering from hypercholesterolemic. Thirty-six male rats weighting approximately 160 grams were divided into six groups, each group containing six rats. Group H1 fed on basal diet as a control negative group. Group H2 fed on diets containing 15% of beef tallow instead of the sunflower oil proportion and the other groups H4, H5 and H6 fed on the same diet used in group H2 and supplemented with different treatments (soy lecithin (10g/100g diet), sumac (4g/100g diet) and mixture of (5g soy lecithin and 2g sumac / 100gm diet) for groups H4, H5, H6, respectively. Group H3 fed on the same diet used in group H2 and supplemented with Pravachol (drug group, H3) (10mg / 100 g diet). After 8 weeks of rats feeding with treatments and hypercholesterolemic diet rats significantly decreased levels of glucose. The treatments also resulted in a significant improvement in lipid profile, liver function, and kidney function. However, a significantly increasment in the activities of catalase (CAT), glutathione peroxidase (GSH-Px) and glutathione (GSH) were observed in blood of hypercholesterolemic rats treated with soy lecithin and sumac powder. The treated groups showed a significant decreasment in thiobarbituric acid reactive substances (MDA) in serum. Since the study of induction of the redox enzymes is considered to be a reliable marker for evaluating the anti peroxidative efficiency of the soy lecithin and sumac. Treatment with soy lecithin and sumac powder reduces the histopathological, heart and liver abnormalities associated with hypercholesterolemic.

Keywords: Soy lecithin, Sumac, Hypercholesterolemic, Rats, Blood glucose, Cholesterol , Triglycerides, liver function, kidney function , CAT, GSH-Px, GSH, MDA .

Introduction

Cardiovascular disease (CVD) is the leading cause of morbidity and mortality worldwide and is increasing alarmingly in developing countries (Rehan *et al.,* 2016). Hypercholesterolemia (HC) is a major risk factor in the development of premature atherosclerosis (Riccioni and Sblendorio, 2012). Some studies have clearly shown that dietary factors such as continuous ingestion of high amounts of saturated fats and cholesterol are directly related to HC (Abdelhalim, 2010 and Sethi *et al.,* 2010). Moreover, high cholesterol levels along with generation of reactive oxygen species (ROS) play a key role in the development of coronary artery disease and atherosclerosis (Kamesh and Sumathi, 2012) since they generate oxidized low-density lipoprotein (oxLDL) (Ondrejovicová *et al.,* 2010). These atherogenic particles are involved in inflammatory processes and consequently in the progression of atherosclerotic lesions (Packard and Libby, 2008). To minimize the damaging effects of free radicals, enzymatic and non-enzymatic systems of defense are developed by the body such as superoxide dismutase (SOD) glutathione peroxidase (GSH-Px) and catalase (CAT), which act in concert to protect the organism from oxidative damage (Kamesh and Sumathi, 2012). Lecithin, (an important phospholipid) is found in the major organs in our body such as the heart, the liver, and the kidneys (Raj *et al.,* 2010). Lecithin, which is a component of most cells, will help in transport and responsible for overall health of the body. Though it is produced within our own bodies, we do not always consume enough of the nutrition needed to produce it in adequate amounts (Raj *et al.,* 2010). As a result, lecithin supplementation is necessary for overall health and prevention of many conditions and diseases. Lecithin is composed of phosphoric acid, choline, fatty acids, glycerol, glycolipids, triglycerides and phospholipids (e.g., phosphatidylcholine, phosphatidylethanolamine, and phosphatidylinositol) (Raj *et al.,* 2010). However, lecithin is sometimes used as a synonym for pure phosphatidylcholine, a phospholipid that is the major component of its phosphatide fraction. It may be isolated either from egg yolk or soyabeans. Few studies have been carried out exploring different biological activities of lecithin.

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Soya-derived lecithin have significant effects on lowering cholesterol and triglyceride, while increasing HDL levels in the blood (Raj *et al.,* 2010). Recent studies suggest that a lecithin enriched diet can modify the cholesterol homeostasis and lipoprotein metabolism. Lecithin diet modifies the cholesterol homeostasis in the liver, increasing the activity of 3-hydroxy-3 methylglutaryl coenzyme A (HMG-CoA) reductase and cholesterol 7 alpha-hydroxylase and decreasing the microsomal ACAT activity.

One of the most spectacular properties of lecithin is its ability to reduce the excess of LDL cholesterol. It also promotes the synthesis in the liver of great amount of HDL, the beneficial cholesterol. Bile acid secretion with high levels of cholesterol and phospholipids is encouraged by lecithin-rich diets when compared with diets without lecithin. Lecithin contains choline that is used in treatment of neural disorder. Lecithin is one of the nature elements (components) that have dispersing properties. That is why it can emulsify fat, avoiding its absorption (Mourad *et al.,* 2010, Sreedevi *et al.,* 2012 and Sarah and Luma, 2018).

Sumac (*Rhus coriaria* L., family Anacardiaceae) is one of the most popular spices in Mediterranean and Arabic countries, which is obtained by crushing the dried fruits. Sumac is used in traditional medicine for its antibacterial and antioxidant effect (Aliakbarlu *et al.,* 2013 ; Ali-Shtayeh *et al.,* 2013 and Kossah *et al.,* 2013), antifungal (Onkar *et al.,* 2011), anti-inflammatory (Panico *et al.,* 2009), DNA protective (Chakraborty *et al.,* 2009), hypoglycemic (Golzadeh *et al.,* 2012 and Anwer *et al.,* 2013), and hypolipidemic activities (Madihi *et al.,* 2013). Sumac is a rich source of tannins, phenolic compounds, oleic and linoleic acids, vitamins, minerals, anthocyanins and organic acids (Zargham and Zargham, 2008; Kossah *et al.,* 2009 and Kossah*,* 2010).

Liver function could be detected the state of liver. Uses of Liver Function Tests (LFTS) to check liver impairment, evaluate the development of diseases and monitor the impact of hepatotoxic drugs and necrosis in the liver of animals. Serum aminotransferases and alkaline phosphatase, bilirubin and albumin also the prothrombin time included in (LFTS) (Macfarlane *et al.,* 2000).

Creatine metabolism by muscles produces creatinine as a major waste product of this process. In kidneys, it's filtered by the glomerulus and excreted by the tubules. Besides free-creatinine appears in the blood serum (Stevens *et al.,* 2006), urea and uric acid are the principal waste products of protein catabolism. They synthesized in the liver from ammonia produced because of the deamination of amino acids. The rate of production is accelerated by a high protein diet or by increased endogenous catabolism due to starvation or tissue damage (Bequette and Sunny, 2005). Kidney function tests help to determine if the kidney is performing their task adequately.

The aim of this work is to examine the nutritional and protective effect of soy lecithin and sumac *(Rhuscoriaria* L.) on hypercholesterolemic rats.

Materials and Methods

Soy lecithin capsules [22% phospholipids (*Phosphatidylethanolamine*)], Pravachol sumac (*Rhus coriaria* L.) spices and Beef tallow were purchased from local market and pharmacy, spectrum Kits which used in biochemical assay, were bought from Egyptian market (El Fouad Lab supply company).

Animals: Thirty-six male Albino rats, average weight of 160 g $\pm$ 0.5 g. raised in the animal house of the Ophthalmology Research Institute, Giza, Egypt, were used in the present study. The rats were kept in normal healthy laboratory condition; temperature was adjusted at $25 \pm 2\ ^{\circ}$C and12-hour light – dark. Animals were adapted on free access of water, and fed for one week basal diet before the initiation standard of the experimental .The Composition of the basal diet: Casein, 21.7%, vegetable oil, 15%, corn starch, 58.1%; cholic acid 0.2%, salt mixture, 4% and vitamin mixture, 1% according to Zahra and Pooya, (2012).

Experimental design: The 36 rats were equally divided into 6 groups of six rats and were kept in wire cages in an equal room temperature of 25°C$\pm$ 2 with normal healthy conditions maintained. Food consumption was monitored, and the weekly weight gain was determined. The rats of (H1) we fed on basal diet and were considered a control negative group (normal control). The other five groups were given 15% of beef tallow instead of the sunflower oil proportion. Group H2 fed on diets containing 15% of beef tallow instead of the sunflower oil proportion. Four groups were administrated, Pravachol (drug group, H3)(10mg / 100 g diet), Soy lecithin (group, H4) (10g / 100 g diet), sumac powder (group,

H5) (4g / 100 g diet) and mixtures (5 gm soy lecithin and 2 gm sumac powder/ 100gm diet) (group, H6).

The examined parameters were chemical components, food consumption, body weight gain, relative organs weight, some biochemical tests and histopathological examination of heart and liver in normal and experimental rats. Their effects and constituents are recorded in tables.

Biochemical assay: when the 60 days was finished (the period of experimental), the blood samples were collected from the eye plexuses of the animals. Then they were put into a dry clean centrifuge glass tube without any coagulation to prepare serum. The samples were left for 15 minutes at normal temperature, after that the tubes were centrifuged for another 15 minutes at 3000 rmp. From then until the time of analysis the clean supernatant serum was kept frozen at -20 °C. Determination of serum glucose level was done according to Trinder, (1969) using spectrum kits. The determination the levels of Total cholesterol (TC) using methods according to Waston (1960), high density lipoprotein (HDL) (Assmann (1979), low density lipoprotein (LDL) (Wieland and Seidel (1983), VLDL- cholesterol (Wallach (1992), total lipid (TL) (Zollener and Kirsch (1962), triglycerides (TG) (Fossati and Prencipe (1982) and atherogenic index (Kikuchi *et al.,* 1998), respectively. Liver function: Alanine aminotransferase (ALT) and aspartate aminotransferase (AST) activities were using methods of (Bergmeyer and Harder, 1986). Alkaline phosphatase (ALP) activity was measured at 405 nm of paranitrophenol from para-nitrophenylphosphate of the formation as a substance using the method of (Varley *et al.,* 1980). Lactate Dehudrogenase (LDH) was measured using the method of Martinek, (1972). Kidney function: creatinine was measured using he method of Henry (1974), urea was measured using the method of Fawcett and Scott (1960) while uric acid was measured using the method of Caraway, (1955). The activity of lipid peroxidation level (Malondialdehyde, MDA) was determined in serum by the colorimetric method described by Meltzer *et al.* (1997).Glutathione peroxides (GSH-P$_X$),Glutathione reduced (GSH) and Catalase were measured calorimetrically in erythrocyte according to the method of Rotruck *et al.,* (1973), Ellman (1959) and Aebi (1984), respectively.

Histopathological examination: At the end of the experiment samples from hearts and livers of the rats of all groups were collected and fixed in a 10% neutral buffered formalin, the samples were dehydrated in alcohol, cleared in xylol and embedded in paraffin. 4µ thick Hematoxylene and eosin stained sections were prepared (Yoon *et al.,* 2001). These examinations were done by Prof. Dr. Abdelhameid Kukab professor of Histopathology in faculty of Veterinary Medicine at Cairo University.

Statistical analysis: The obtained results were subjected to statistical analysis using the standard analysis of variance as outlined by Snedecor and Cochran (1980).

Results and Discussion

Effect of soy lecithin, sumac powder and their mixture on intake of food and gain of body weight in hypercholesterolemic rats:

Effect of feeding on hypercholesterolemic diet for 60 successive days mixed with soy lecithin and sumac on body weight gain of rats and its results had been summarized in table (1).Data show that the initial body weights did not significantly differ among the groups and at the end of experiment, regardless of the diet variation, there was increased significantly differences among all the tested rat groups except in case of the (p. control) which was significantly decreased (18.85 %) in body weight gain comparing to non- hypercholesterolemic (N-control). Meanwhile, soy lecithin and sumac powders with hypercholesterolemic diet increased in body weight gain comparing to hypercholesterolemic diet (P-Control). On contrary, there were non- significant differences were found in heart, liver and kidney on their relative weight (%) of rats except that rats feed on hypercholesterolemic diet (H2 and H3) groups table (1).

Hypercholesterolemic diet for 60 successive days was increasing the weight gains and feed intake significantly this data agreement with Maha, (2015). The present results revealed that hypercholesterolemic diet significantly reduce food intake, compared to standard diet. These results may be attributed to higher caloric content of hypercholesterolemic diet compared to standard diet. High fat diet in the present study was responsible for increasing satiety and total calories. This result agreed

with Sheyla *et al.,* (2005) who explained that high fat diet induce hypercholesterolemic, leads to lower ingestion by the animals and cause malnutrition. The beneficial effect of antioxidant administration against hypercholesterolemic poisoning with respect to body weight observed in the present study confirms previous results obtained by Manal *et al.,* (2012) who concluded that feeding rats with antioxidants could play an important role as a prophylactic against the toxic effects of high fat.

The relation between organs weight (liver and heart) and body weight might be based on fat accumulation in liver and heart cells. When organ weight was compared with final body weight, fat increased the hepatosomatic index causes liver damage. Uthandi and Ramasamy, (2011) reported that higher ingestion of potentially toxic components by the rat fed high fat lead to higher hepatosomatic index. These results were confirmed by histopathological examination (fatty changes of hepatocytes and granularity of the sarcoplasm focal cardiac myocytes), accordance with Sheyla *et al., (*2005), and the accumulation of intracellular lipid in cardiomyocytes in response to cholesterol diet (Puskas *et al.,* 2004). Meanwhile, there was no difference in relative organs weight in rats feed on hypercholesterolemic diet with soy lecithin, sumac powders and mixture (soy lecithin and sumac). While rats feed on high fat (H2 and H3) showed increased in relative organs weight (heart, liver and kidney). The obtained data are agreements with Rezq and El-Khamisy, (2011) which reported that relative organs weight was significantly increased after rats feed on high fat diets.

Effect of soy lecithin, sumac powder and their mixture on serum glucose levels and lipid profile levels of hypercholesterolemic rats:

Table (2) display the level of serum glucose in normal and experimental animals. The data revealed a significant increased elevation (44.68%) in blood glucose in H2 (P. control) rats compared to H1 (N. control) normal rats. Supplemented provision of soy lecithin, sumac and their mixture (soy lecithin and sumac) with hypercholesterolemic diet significantly decreased (19.85, 20.95and 24.63 %, respectively) the level of blood glucose compared to H2 (P. control) group. On the other hand, there were no significant variances in glucose among rats feed on soy lecithin and sumac groups throughout the feeding periods (60 days). The present results revealed that was non- significant in concentrations of serum glucose of rats given soy lecithin and sumac powder for 60 days while serum glucose significantly high in hypercholesterolemic rats H2 (P- Control) which agreement with (Labban *et al.,* 2014 and Sarah and Luma, 2018).

The hypercholesterolemic diet significantly increased elevation (89.28; 92.38 ;50 ; 50; 30.86 and 202.73 %, respectively) in serum total cholesterol; low- density lipoprotein- cholesterol; triglyceride; very low- density lipoprotein- cholesterol, total lipid and atherogenic index in H2 (P. control) rats compared to H1(N. control) normal rats. While, HDL- cholesterol was significantly declined (40. 23%) in H2 (P. control) rats compared to H1 (N. control) normal rats as *shown* in Table (2). Administration of the tested soy lecithin, sumac and its mixture (soy lecithin and sumac) improved or returned these values to the normal ones. On contrary, there were non-significant differences in serum total cholesterol, HDL- cholesterol, low density lipoprotein- cholesterol, triglyceride; very low-density lipoprotein- cholesterol and total lipid in among rats feed on soy lecithin and sumac groups throughout the feeding periods (60 days) but its mixture has significant improved results.

These results agree with that (Nagib, 2017). P-control (H2) diet increase oxidative stress and the presence of oxidized LDL-cholesterol and other lipoproteins. Oxidation converts LDL-cholesterol to a form that is rapidly taken up and degraded by macrophages and increased degradation of unoxidized LDL-cholesterol. Antioxidants are inhibiting metabolism of LDL-cholesterol and reduce toxicity of oxidized LDL-cholesterol. Oxidized lipoproteins may lead to development of atherosclerosis (Shafaeizade *et al.* 2011).

The increase in HDL-C may be a consequence of protection mechanism against the oxidative stress caused by the diet containing hypercholesterolemic and a mechanism to avoid oxidative changes in another lipoprotein such as LDL (Kareem *et al.,* 2009). HDL - C as mention in Table (2) showed that the rats of H4,H5 and H6 groups had lower concentrations of (HDL -C) compared with the H1 group rats but higher than H2 and H3 groups. Mohammadi and Oshaghi, (2014) found that antioxidant rich foods increased the response to the oxidative damage in the pathogenesis of many diseases and increased HDL-C.

Rats fed on hypercholesterolemic diet showed that the concentration of TC, LDL , LDL , VLDL, TG and TL increased significantly than non hypercholesterolemic diet . Meanwhile, the groups (H3,

Table 1: Effect of soy lecithin and sumac on net gain and % (liver, kidney and heart) organs in experimental of hypercholesterolemic rats.

Treatments	Initial (g)	Final (g)	Net gain (g)	Net gain (%)	Food consumption (g)	Liver %	Kidney %	Heart %
N. control (H1)	160[a]± 1	340.33[a] ±1.52	180.33[a] ±0.57	112.71	1175 [a]± 5	2.511[e] ±0.0046	0.3657[e]±0.0017	0.591[e]±0.0012
P. control (H2)	159.67[a]± 0.57	305.67[f]±1.52	146.33[e] ±1.53	91.84	961.67 [f]± 7.64	2.779[a]±0.0104	0.736[a]±0.0034	0.916[a]±0.0025
Drug (H3)	159.67[a]± 0.57	321[e]± 2	161.33[cd]±1.53	101.04	995 [e]± 5	2.472[b]±0.0092	0.5823[b]±0.0023	0.736[b]±0.0012
Lecithin (H4)	160.67[a]±1.15	320.67[b] ±1.15	160 [d]± 0.57	99.79	1091.67 [d]± 7.63	2.5723 [c]±0.0103	0.4987[c]±0.0029	0.699[c]±0.0012
Sumac (H5)	160.67[a]±1.15	324.33[c] ±0.57	163.66[c] ±1.52	101.86	1108.33 [c]± 7.63	2.5618[c]±0.0095	0.4967[c]±0.0012	0.696[c]±0.0012
Lecithin+Sumac (H6)	160.67[a]±1.15	328[b]±1	167.33[b]± 2.08	104.14	1125 [b]± 5	2.4723[d]±0.0085	0.3717[d]±0.0025	0.655[d]±0.0015
LSD	1.9215	1.7288	2.4807		11.4833	0.0138	0.0022	0.0042

Means, within the same column, followed by the same letter are not significantly different at <0.05.
Means are followed by the corresponding standard deviation.

Table 2: Effect of soy lecithin and sumac on glucose, total cholesterol, HDL-C, LDL-C, VLDL-C, triglycerides, total lipid and Atherogenicindex levels in experimental of hypercholesterolemic rats.

Treatments	Glucose mg/dl	TC mg/dl	HDL mg/dl	LDL mg/dl	VLDL mg/dl	TG mg/dl	TL mg/dl	Atherogenic Index
N. control (H1)	125.33[e]±1	105.66[e] ±1.53	30.67 [a]±0.58	65.70[e]±1	24.13[e]±1.21	120.66[e]±2.08	283 [e]±2	2.93[e]±0.008
P. control (H2)	181.33[a]±1.15	200[a]±2	18.33[e]±1.53	126.40[a]±1.5	36.2[a]±0.72	181[a]±1	370.33[a]±4.5	8.87[a]±0.010
Drug (H3)	152.66[b]±1.52	150.33[b]±1.53	21 [d]±1	101.60[b]±1.5	30.67[b]±0.51	153.33[b]±1.53	316 [b]±3	6.30[b]±0.010
Lecithin (H4)	145.33[c]±1.15	147.33[c]±1.53	23.8 [c]±1	87.80[c]±1.53	27.93[c]±1	139.66[c]±1.53	310.66[c]±1.15	4.86[c]±0.009
Sumac (H5)	143.33[c]±0.58	145[c]±1	25 [c]±1	84.50[c]±1	27.51[cd]±1.01	137.53[c]±0.58	306.66[c]±0.58	4.48[c]±0.009
Lecithin+Sumac (H6)	136.66[d]±1.53	141[d]±1	28 [b]±1	74.5 [d]±1	25.81[de]±0.53	129 [d]±1	300 [d]±1	3.58[d]±0.008
LSD	2.1380	2.6186	1.8752	2.2828	1.5486	2.4449	4.3576	0.8247

Means, within the same column, followed by the same letter are not significantly different at < 0.05.
Means are followed by the corresponding standard deviation.

H4, H5 and H6) fed on hypercholesterolemic diet and supplemented with different of treatments had lower value compared with H2 group (table 2) . These results are in agreement with (Doha *et al.,* 2010) and (Li *et al .,* 2016).

Effect of soy lecithin and sumac powders and their mixture on liver functions of hypercholesterolemic rats:

Administration of hypercholesterolemic diet produced significant adverse effects on the liver functions of the rats, which is evidenced by a significant increase elevation (47.15, 31.70 , 78.48 ; 84.93, 56.99 and 76.84 % respectively) in the actions of AST; ALT and ALP in H2 (P. control) and H3 (drug) compared to H1(N. control) normal. Treatment of hypercholesterolemic rats with soy lecithin and sumac exhibited improvement in the actions of AST, ALT and ALP compared to H2 (P. control) rats. On the other hand, there were non- significant variances in the actions of AST, ALT and ALP among rats feed on soy lecithin and sumac groups throughout the feeding periods (60 days) Table (3) but H6 has better improvement .

The Concentration of intracellular hepatic enzymes that have leaked into the circulation measured by alanine aminotransferase (ALT) and aspartate aminotrasferase (AST). Moreover, normal functioning of liver indicated by normal levels of ALT and AST . When the enzymes leak from the liver cytosol into the blood stream that lead to higher level of ALP in serum (Dauqan *et al .,* 2012), which is indicative of hepatotoxic of hypercholesterolemic rats. Also, one of the advantages of administration of soy lecithin and sumac to hypercholesterolemic rats was a reduction in ALP activity to its regular levels. High levels of ALP in serum indicate liver damage. In the present study, the decrease in ALP activity in hypercholesterolemic rats given soy lecithin and sumac shows that presented liver damage, as a result of metabolic changes such as administration of toxin, liver cirrhosis, hepatitis, and cancer of the liver, when the serum ALT, AST and ALP increased (Nagib, 2017) . Thus, they can be used as markers to estimate the extent of liver damage. In this concern, it was reported that oxidative stress mediates many of the effects caused by oxidized fats (Kunwar and Priyadarsini, 2011). The formation of reactive oxygen species (ROS) may increase the dietary oxidized fats and may cause an increased damage of proteins in the liver by enhancing lipid per oxidation of the cell membrane and increasing the generation of (ROS) which can lead to calcium homeostasis disturbances, increase membrane fluidity and cell death. On the other hand, (Abbass *et al .,* 2012 and Nagib, (2017) found that some natural substances that can delay the rate of oxidation by directing the breakdown of peroxides into stable substances that do not promote further oxidation or by sweeping free radicals away. Histopathological study of liver showed kuffer cells activation, vacuolar degeneration of hepatocytes and fatty degeneration of centro-lobular hepatocytes in the liver of hypercholesterolemic rats. Damaged membranes were recovered by the treatment with spices through enhancing antioxidants' status and decreasing lipid peroxidation (Labban *et al.,* 2014).

Effect of soy lecithin and sumac powder and their mixture on kidney functions of hypercholesterolemic rats:

It was clear from table (3) hypercholesterolemic diet produced significant adverse effects on the kidney functions of the rats, which is evidenced by a significant increase elevation (57.38,40.98; 63.33, 50 and 29.45,18.91%, respectively) in creatinine; urea and uric in H2 (P. control) and group drug compared to H1 (N. control) normal. Treatment of hypercholesterolemic rats with soy lecithin and sumac exhibited improvement in kidney functions compared to H2 (P. control) rats. Meanwhile, there were non- significant differences in creatinine, urea and uric in among rats feed on soy lecithin and sumac groups throughout the feeding periods (60 days).

Our study, demonstrated that hypercholesterolemic rats had a renal modification such as accumulation of fat in cells، increased kidney weight gain, glomerular sclerosis and inflammatory infiltrates, along with elevated levels of blood glucose, reinforce the idea that glycosylation of proteins, increased the release of pro inflammatory cytokines, oxidative stress, and the accumulation of lipid peroxidation products may be caused kidney damage (De Castro, 2013).Deterioration of the tissues and enlarged the kidneys of hypercholesterolemic rats (Amin *et al.,*2014) .

Effect of soy lecithin and sumac powder and their mixture on malonaldehyde , enzymatic antioxidants and non- enzymatic antioxidants activity in experimental of hypercholesterolemic rats:

Table (4) show the activity levels of Lactate dehydrogenase (LDH) enzyme malonaldehyde (MDA) enzyme in serum, enzymatic antioxidants, Catales , (GSH-Px) and non- enzymatic antioxidants(GSH) in blood, respectively, in normal and experimental rat groups. The activity of serum LDH enzyme was significantly increased (68.80 %) in H2 (P. control) hypercholesterolemic compared to H1 (N. control) normal (44.38) and also maloaldehyde (MDA) activity was significantly increased (474.38 %) in H2 (P. control) hypercholesterolemic compared to H1 (N. control) normal. While the activities of blood enzymatic antioxidants catales, (GSH-Px) and non- enzymatic antioxidants (GSH) were significantly decreased (52.55, 59.19 and 70.46 %, respectively) in H2 (P. control) compared to H1 (N. control) normal. Supplemented hypercholesterolemic rats groups with soy lecithin and sumac decreased the levels of LDH, malonaldehyde (MDA) and increased activities of enzymatic antioxidant, catales, GSH-Px and non-enzymatic antioxidants (GSH) . On contrary, there were non-significant differences in LDH, MDA, catales, (GSH-Px) and non- enzymatic antioxidants (GSH) among rats feed on soy lecithin and sumac groups but its mixture makes good improvement throughout the feeding periods (60 days).

A stressful condition leads to the excessive production of free radicals which results in oxidative stress an imbalance in the oxidant per antioxidant system. Generation of free radicals is an integral feature of normal cellular functions in contrast to excessive generation and/or inadequate removal of free radical results in destructive and irreversible damage to the cell (Sarah and Luma, 2018). Under normal conditions, there is a natural defense system provided by several enzymes such as Catalase (CAT), Glutathione Peroxidase (GSH-Px) and non- enzymatic antioxidants Glutathione reduced (GSH) which performs a vital role for detoxification of free radicals. The use of antioxidant rich food or antioxidant food supplements became immensely popular since many diseases have been associated with oxidative stress (Colares *et al.,* 2016, Nagib, 2017 and Sarah and Luma, 2018).

Pathological effects of different organs: Figure (1) shows the microscopic examination of the heart of the experimental rat groups. Microscopical examination of heart of rat from group H1 revealed the normal histological structure of cardiac myocytes (slide 1). In contrary, examined sections from H 2 (P. control) group showed vacuolation of the sarcoplasm of cardiac myocytes (slide 2), inter myocardial oedema (slide 3) and focal necrosis of cardiac myocytes (slide 4). However, heart of rats from drug group (H 3) revealed congestion of myocardial blood vessels (slide 5), slight inter myocardial oedema with few inflammatory cell's infiltration (slide 6) andvacuolation of the sarcoplasm of cardiac myocytes (slide 7). Meanwhile, heart of rats from group (H4) revealed no histopathological changes (slide 8). Heart of rats from group (H5) showed no histopathological changes (slide 9). On the other hand, heart of rats from group (H6) revealed no histopathological changes (slide 10).

Meanwhile, Figure (2) shows the microscopic examination of the liver of the experimental rat groups. Microscopical examination of liver of rat from group (H 1) revealed the normal histological structure of hepatic lobule (slide 1). On the other hand, liver of rats from P. control group (H 2) revealed steatosis of hepatocytes (vacuolar degeneration of hepatocytes) (slide 2) and oval cells proliferation in the portal triad (slide 3).

However, liver of rats from drug group (H3) revealed vacuolar degeneration of some hepatocytes (slide 4) and binucleation of hepatocytes (slide 5). Meanwhile, liver of rats from group (H 4) from revealed steatosis of some hepatocytes (vacuolar degeneration of hepatocytes) (slide 6). Liver of rats from group (H 5) showed steatosis of hepatocytes (vacuolar degeneration of hepatocytes) (slide 7). On the other hand, liver of rats from group (H6) revealed no histopathological changes (slide 8).

The present study cleared that the addition concentration of soy lecithin, sumac powders or their mixture in high fat diets could be declined the levels of lipid peroxidation, reducing the formation of atherogenic index which prevent tissue damage.

Table 3: Effect of soy lecithin and sumac on liver function and kidney function levels in experimental of hypercholesterolemic rats.

Treatments	AST U/L	ALT U/L	ALP U/L	Creatinine mg/dl	Urea mg/dl	Uric acid mg/dl
N. control (H1)	41 [e]±1	31 [e]± 1.07	150.33 [e]± 5.4	0.61 [e]±0.01	30 [e]±1	2.75 [e]±0.05
P. control (H2)	60.33 [a]±1.53	55.33 [a]±1.47	278[a]±10	0.96 [a]±0.01	49 [a]±1	3.56 [a]±0.058
Drug (H3)	54 [b]± 1.32	52.33 [b]±1.41	236[b]±8.5	0.86 [b]±0.01	45 [b]±1	3.27 [b]±0.025
Lecithin (H4)	51 [c]± 1.25	38 [c]±1.35	214.06 [c]±7.3	0.78 [c]±0.006	38.57 [c]±0.58	3.04 [c]±0.04
Sumac (H5)	49.5 [c]±1.21	36.96 [c]±1.28	217.33 [c]±7.8	0.77 [c]±0.006	37.07 [c]±0.58	3.11 [c]±0.058
Lecithin+Sumac (H6)	45.33 [d]±1.17	34.33 [d]±1.19	204.33 [d]± 6	0.69 [d]±0.01	34.66 [d]±0.58	2.87 [d]±0.058
LSD	1.8752	1.3259	2.8439	0.0156	1.4525	0.0883

Means, within the same column, followed by the same letter are not significantly different at <0.05.
Means are followed by the corresponding standard deviation

Table 4: Effect of soy lecithin and sumac on serum(MDA) and erythrocytes (Catalase, GSH-Px and GSH) levels experimental of hypercholesterolemic rats.

Treatments	Catalase (U/ml)	GSH-Px (U/ml)	GSH (mg/dl)	LDH U/L	MDA (nmol/ml)
N. control (H1)	175.8 [a]± 8.2	167.5 [a]± 7.4	37.54 [a]±1.02	827.66 [e]± 10.4	4.49 [e]± 0.4
P. control (H2)	83.41 [e]±6.5	68.35 [e]± 4.2	11.09 [e]±0.42	1463.66 [a]±15.8	25.79 [a]±0.84
Drug (H3)	127.51 [d]± 6.4	114.52 [d]±4.8	27.24 [d]±0.72	1195 [b]± 14.1	11.85 [b]±0.71
Lecithin (H4)	145.34 [c]± 7.2	120.35 [c]±5.6	29.15 [c]±0.85	980 [c]± 13.5	9.86 [c]±0.67
Sumac (H5)	148.51 [c]±7.4	124.59 [c]±5.9	28.54 [c]±0.93	962.66 [c]±12.9	9.23 [c]±0.62
Lecithin+Sumac (H6)	151.28 [b]±7.9	131.27 [b]±6.4	31.92 [b]±0.97	879.66 [d]±12.5	8.17 [d]±0.6
LSD	3.8547	5.3854	1.0574	23.7050	1.2187

Means, within the same column, followed by the same letter are not significantly different at <0.05.
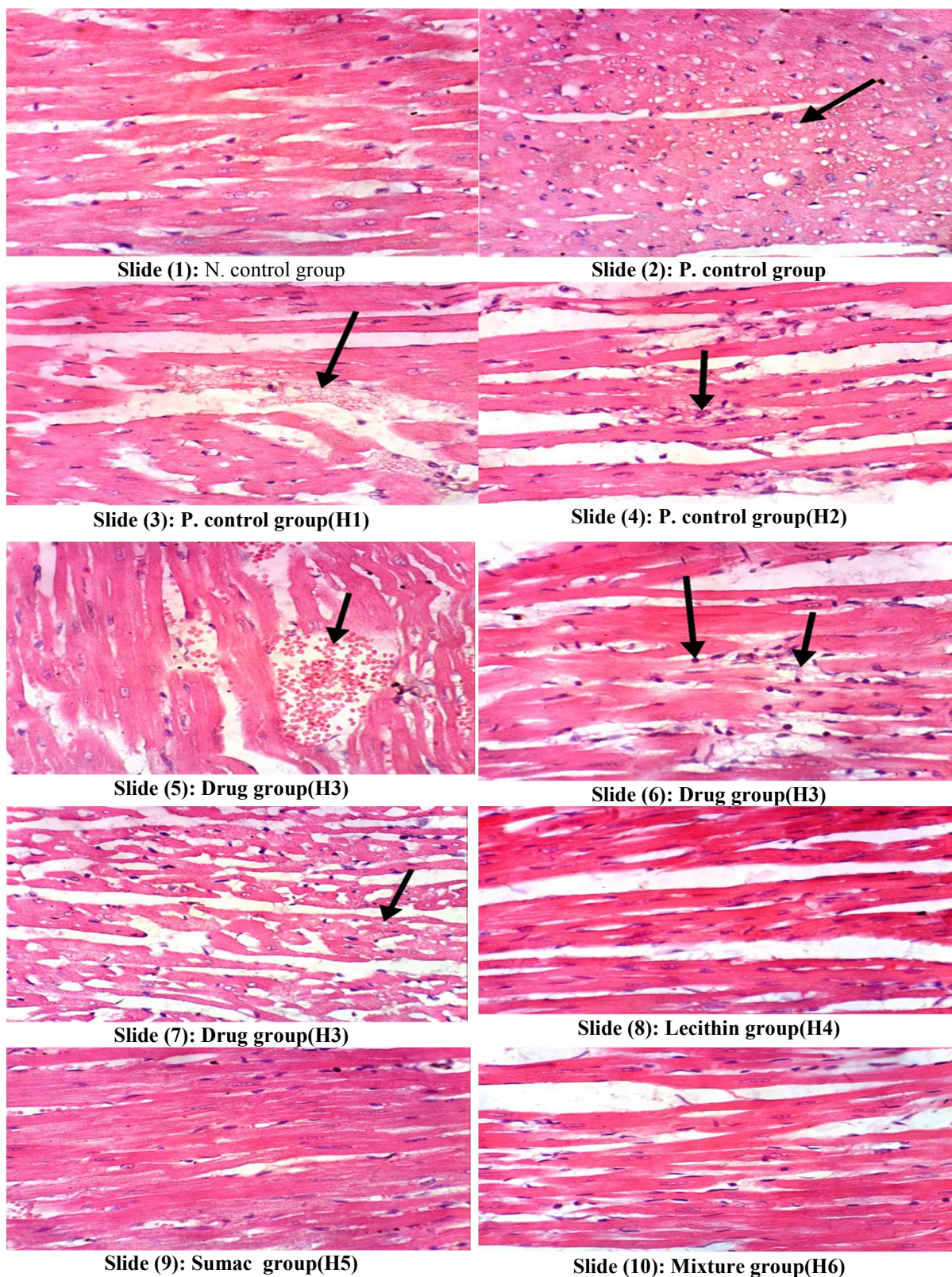Means are followed by the corresponding standard deviation.

Slide (1): N. control group

Slide (2): P. control group

Slide (3): P. control group(H1)

Slide (4): P. control group(H2)

Slide (5): Drug group(H3)

Slide (6): Drug group(H3)

Slide (7): Drug group(H3)

Slide (8): Lecithin group(H4)

Slide (9): Sumac group(H5)

Slide (10): Mixture group(H6)

Fig. 1: Histopathological changes in tissue sections of heart.

Slide (1): N. control group(H1)

Slide (2): P. control group(H2)

Slide (3): P. control group(H2)

Slide (4): Drug group(H3)

Slide (5): Drug group(H3)

Slide (6):Lecithine group(H4)

Slide (7): Sumac group(H5)

Slide (8): Mixture group(H6)
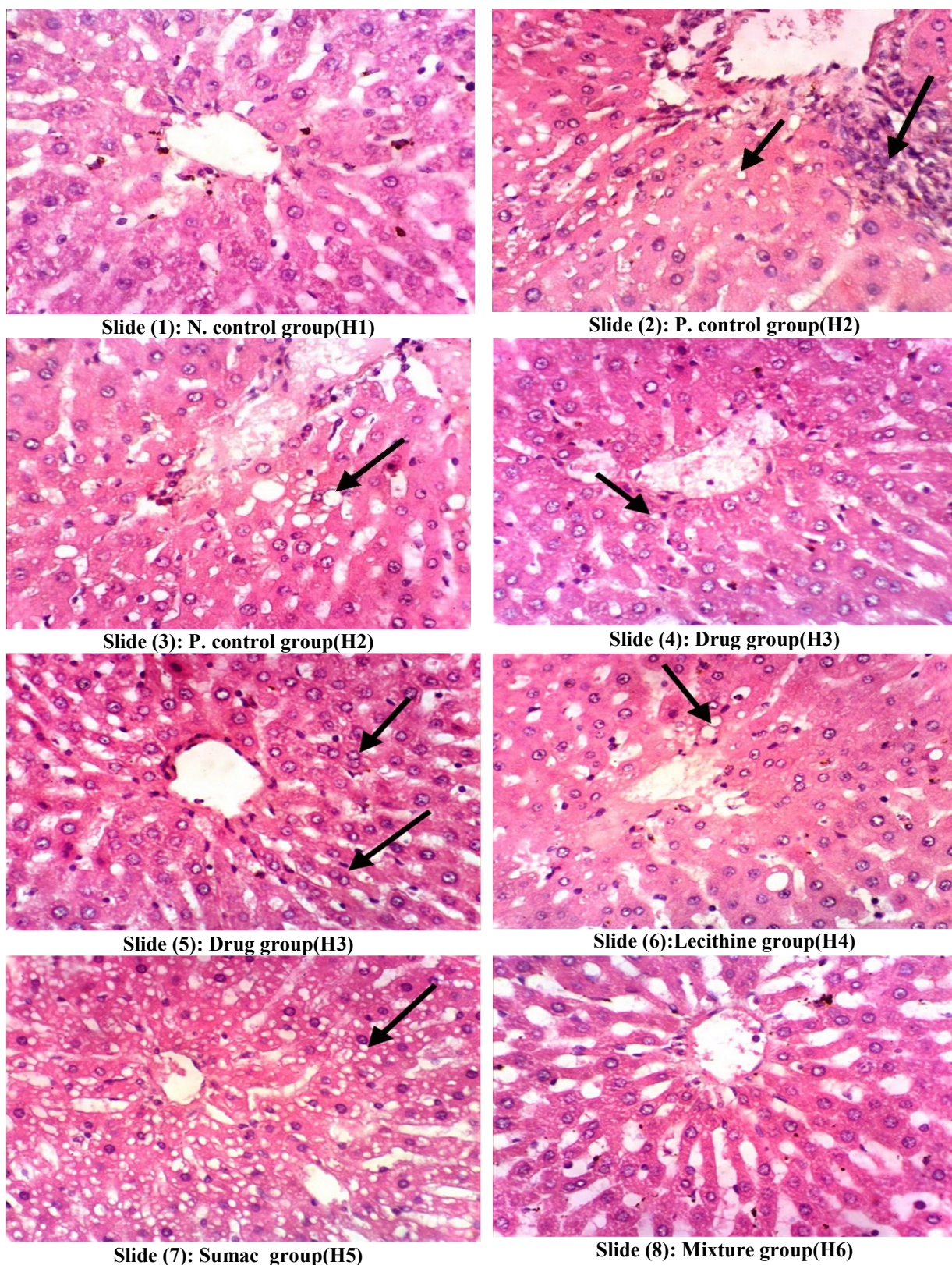
Fig. 2: Histopathological changes in tissue sections of liver.

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